Before you begin: This is a big topic, and big topics beget big slide-sets. There’s natural breaks in a couple of spots (@slides 152- and 355-ish); I placed *break time!* slides to mark them.
There's no single correct way to divide up the optic neuropathies. That said, there's a compelling argument that you should think of them in terms of these two subgroups. What are they?
There’s no single correct way to divide up the optic neuropathies. That said, there’s a compelling argument that you should think of them in terms of these two subgroups. What are they?
What is the common name for an optic neuropathy secondary to an inflammatory process?

Typical Optic Neuritis

Optic Neuropathy

Inflammatory

Noninflammatory
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Noninflammatory

What is the common name for an optic neuropathy 2ndry to an inflammatory process?
Optic neuritis
Again, no single correct answer (and several viable options). But there’s a compelling argument that you should think of them as belonging to one of two subgroups. What are they?
Again, no single correct answer (and several viable options). But there’s a compelling argument that you should think of them as belonging to one of two subgroups. What are they?
What does it mean to say an optic neuritis is typical?

It means the underlying disease process involves demyelination.
What does it mean to say an optic neuritis is typical?
It means the underlying dz process involves demyelination.
What does it mean to say an optic neuritis is typical?
It means the underlying dz process involves demyelination

Note: In common clinical parlance, the term *typical* is reserved for demyelination that is either idiopathic or related to MS
What does it mean to say an optic neuritis is typical?
It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
What does it mean to say an optic neuritis is typical?
It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
A woman between 15 and 45
What does it mean to say an optic neuritis is typical?
It means the underlying dz process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
A woman between 15 and 45 (average age #)
What does it mean to say an optic neuritis is typical?
It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
A woman between 15 and 45 (average age 32)
What does it mean to say an optic neuritis is typical?
It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
A woman between 15 and 45 (average age 32)

What proportion of typical optic neuritis pts are women?
Almost 80%!
What does it mean to say an optic neuritis is typical?
It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient?
A woman between 15 and 45 (average age 32)

Almost 80%!
Optic neuritis

Typical (demyelinating)

Noninflammatory

Typical Optic Neuritis

As the title implies, we will have much more to say about typical optic neuritis later in the set.

What does it mean to say an optic neuritis is typical? It means the underlying disease process involves demyelination.

Demographically speaking, who is the typical typical optic neuritis patient? A woman between 15 and 45 (average age 32).

What proportion of typical optic neuritis pts are women? Almost 80%!
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Noninflammatory

No single correct answer, yada yada yada. What are these two groups?
Optic Neuropathy

- Optic neuritis
  - Typical (demyelinating)
  - Atypical
    - Infectious
    - Immune

No single correct answer, yada yada yada.
What are these two groups?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

Name 3 infectious causes of atypical ON:
1)
2)
3)

(There are many others, of course)
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Typical Optic Neuritis

Name 3 infectious causes of atypical ON:
1) Syphilis
2) Bartonella
3) Lyme

(There are many others, of course)
Optic Neuropathy

Optic neuritis

- Typical (demyelinating)
- Atypical
  - Infectious
  - Immune

Name 3 immune-related causes of atypical ON:
1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis (formerly known as Wegener's)

(There are many others, of course)
Optic Neuropathy

Optic neuritis

Typical
( progressive demyelinating)

Atypical

Infectious

Immune

Name 3 immune-related causes of atypical ON:
1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis

(There are many others, of course)
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Name 3 immune-related causes of atypical ON:
1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis (formerly known as Wegener's)

(There are many others, of course)
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Name 3 immune-related causes of atypical ON:
1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis (formerly known as Wegener’s)

(There are many others, of course)
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

**Typical Optic Neuritis**

Name 3 *immune-related* causes of atypical ON:

1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis

(There are many others, of course)

*Why don’t we call it Wegener’s?*

(formerly known as Wegener’s)
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Name 3 immune-related causes of atypical ON:
1) Sarcoid
2) SLE or some other vasculitic process
3) Granulomatosis with polyangiitis (formerly known as Wegener’s)

Why don’t we call it Wegener’s?
Because Dr Wegener was a Nazi, and is suspected to have committed war crimes

(There are many others, of course)
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

Typical Optic Neuritis
Optic Neuropathy

Optic neuritis
- Typical (demyelinating)
- Atypical
  - Infectious
  - Immune

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic
What is far-and-away the most common type of optic neuropathy? 

Hint…
What is far-and-away the most common type of optic neuropathy?

*Hint*...It’s not listed on this slide!

*Hint*...
What is far-and-away the most common type of optic neuropathy?

Hint…It’s not listed on this slide!

Hint…It’s so common, it gets its own ophthalmic subspecialty!

It’s…

Glaucoma
What is far-and-away the most common type of optic neuropathy?

*Hint* …It’s not listed on this slide!

*Hint* …It’s so common, it gets its own ophthalmic subspecialty!

It’s… **Glaucoma** (don’t forget—glaucoma is an optic neuropathy!)
What exam finding is the sine qua non of unilateral or asymmetric bilateral optic neuropathy?
What exam finding is the sine qua non of unilateral or asymmetric bilateral optic neuropathy? A relative afferent pupillary defect (RAPD)
What exam finding is the sine qua non of unilateral or asymmetric bilateral optic neuropathy? **A relative afferent pupillary defect (RAPD)**

What should you do if a presumptive unilateral/asymmetric bilateral ON pt doesn't have an RAPD?
What exam finding is the sine qua non of unilateral or asymmetric bilateral optic neuropathy?

A relative afferent pupillary defect (RAPD)

What should you do if a presumptive unilateral/asymmetric bilateral ON pt doesn't have an RAPD?

You should question the diagnosis
What functional abnormalities are likely to be found in a pt with an optic neuropathy?

--Decreased central acuity
--Abnormal visual fields
--Impaired color vision
What functional abnormalities are likely to be found in a pt with an optic neuropathy?
-- Decreased central acuity
-- Abnormal visual fields
-- Impaired color vision
Typical Optic Neuritis

What is the typical pattern of vision loss in typical optic neuritis?

Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?

VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What is the long-term VA prognosis?

Very good—about 90% will be 20/40 or better at one year.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision
Typical Optic Neuritis

Optic Neuropathy

What is the typical pattern of vision loss in typical optic neuritis?
- Vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later
- VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range
- Very good—about 90% will be 20/40 or better at one year

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
- Decreased central acuity
- Abnormal visual fields
- Impaired color vision
Optic Neuropathy

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

What is the typical pattern of vision loss in typical optic neuritis?

Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

What is the long-term VA prognosis?

Very good—about 90% will be 20/40 or better at one year.
What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a day, week, or two later.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

Optic Neuropathy

Typical Optic Neuritis
What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

Optic Neuropathy

Typical Optic Neuritis

- Optic neuritis
  - Noninflammatory
  - Typical (demyelinating)
  - Ischemic
  - Compressive
  - Toxic/nutritional
  - Congenital/hereditary
  - Traumatic

Atypical Infectious Immune
What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

What is the typical pattern of vision loss in typical optic neuritis?

Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

This pattern of vision loss and recovery over time in typical optic neuritis bears repeating for emphasis.

No question—proceed when ready
What is the typical pattern of vision loss in typical optic neuritis? Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

This pattern of vision loss and recovery over time in typical optic neuritis bears repeating for emphasis.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

No question—proceed when ready.
Typical Optic Neuritis

**Vision in typical optic neuritis**

VA loss will stop progressing, leveling off for a week or two.

**What is the typical pattern of vision loss in typical optic neuritis?**

Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

This pattern of vision loss and recovery over time in typical optic neuritis bears repeating for emphasis.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

No question—proceed when ready
What is the typical pattern of vision loss in typical optic neuritis? Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

This pattern of vision loss and recovery over time in typical optic neuritis bears repeating for emphasis.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

No question—proceed when ready
What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

This pattern of vision loss and recovery over time in typical optic neuritis bears repeating for emphasis.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

No question—proceed when ready.
What is the typical pattern of vision loss in typical optic neuritis? Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision

How profound is the vision loss?
VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What is the long-term VA prognosis?
Very good—about 90% will be 20/40 or better at one year.
Optic Neuropathy

Typical Optic Neuritis

What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?
VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision
Optic Neuropathy

What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?
VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision

Typical Optic Neuritis
What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?
VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What is the long-term VA prognosis?
Very good—about 90% will be 20/40 or better at one year.

What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision
What are the functional abnormalities likely to be found in a patient with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

What is the typical pattern of vision loss in typical optic neuritis?
Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?
VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What is the long-term VA prognosis?
Very good—about \( \% \) will be 20/40 or better at one year.
What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

What is the typical pattern of vision loss in typical optic neuritis?

Unilateral vision loss which develops and nadirs over a few days, with spontaneous recovery beginning a week or two later.

How profound is the vision loss?

VA can be anywhere from 20/20 to NLP; however, most cases are in the 20/40 – 20/200 range.

What is the long-term VA prognosis?

Very good—about 90% will be 20/40 or better at one year.
What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision

What pattern(s) of VF loss occur in typical optic neuritis?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

What pattern(s) of VF loss occur in typical optic neuritis?

It can be anything, but is most commonly a...
What functional abnormalities are likely to be found in a pt with an optic neuropathy?
--Decreased central acuity
--Abnormal visual fields
--Impaired color vision

What pattern(s) of VF loss occur in typical optic neuritis?
It can be anything, but is most commonly a central scotoma
Central scotoma in typical optic neuritis
What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

**Dyschromatopsia in typical optic neuritis: Is it red-green, or blue-yellow?**
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

Dyschromatopsia in typical optic neuritis: Is it red-green, or blue-yellow?

Red-green
**Optic Neuropathy**

- Optic neuritis
  - **Typical** (demyelinating)
  - **Atypical**
    - Infectious
    - Immune

**Noninflammatory**
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

---

**What functional abnormalities are likely to be found in a pt with an optic neuropathy?**
- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

**Dyschromatopsia in typical optic neuritis:** Is it **red-green**, or **blue-yellow**?
- **Red-green**

**How common is it?**
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

What functional abnormalities are likely to be found in a pt with an optic neuropathy?

- Decreased central acuity
- Abnormal visual fields
- Impaired color vision

Dyschromatopsia in typical optic neuritis: Is it red-green, or blue-yellow?

Red-green

How common is it?

Per the Neuro book, it is “nearly universal”
What is the usual appearance of the ONH in typical optic neuritis?
What is the usual appearance of the ONH in typical optic neuritis? Pretty unremarkable—only ___% of cases present with disc edema.
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

- Typical
  - (demyelinating)
- Atypical

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic
- Infectious
- Immune

What is the usual appearance of the ONH in typical optic neuritis?
Pretty unremarkable—only 1/3 of cases present with disc edema
What is the usual appearance of the ONH in typical optic neuritis? Pretty unremarkable—only 1/3 of cases present with disc edema.

When edema is present, is it usually mild, or florid (ie, severe with associated hemorrhages)?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary

What is the usual appearance of the ONH in typical optic neuritis? Pretty unremarkable—only 1/3 of cases present with disc edema

When edema is present, is it usually mild, or florid (ie, severe with associated hemorrhages)? Mild
Optic Neuropathy

Optic neuritis

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

Atypical

Typical (demyelinating)

Is typical optic neuritis associated with ocular pain?

Yes—over 90% will complain of pain, especially during eye movements.

While it doesn't have to, it often precedes it.
Is typical optic neuritis associated with ocular pain?
Yes—over \% will complain of pain
Is typical optic neuritis associated with ocular pain?
Yes—over 90% will complain of pain
Is typical optic neuritis associated with ocular pain? Yes—over 90% will complain of pain provoked by eye movements.
Is typical optic neuritis associated with ocular pain?
Yes—over 90% will complain of pain provoked by eye movements.
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Infectious
- Immune
- Congenital/hereditary
- Traumatic

Is typical optic neuritis associated with ocular pain?
Yes—over 90% will complain of pain provoked by eye movements.

Does the onset of pain typically precede, follow, or coincide with the loss of vision?
Is typical optic neuritis associated with ocular pain? Yes—over 90% will complain of pain **provoked by eye movements**

Does the onset of pain typically precede, follow, or coincide with the loss of vision? While it doesn’t have to, it often precedes it
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

- Typical (demyelinating)
- Atypical

Noninflammatory

- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That's it.

The purpose of the MRI is to look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?
MRI brain and orbits, with contrast. That’s it.
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI?
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing two diff words
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)
Typical Optic Neuritis

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI?

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS?

The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if…

...there were no white matter changes on MRI: 1/4

...if even one white matter change was present: 3/4
Typical Optic Neuritis

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT).

What proportion of typical optic neuritis pts develop MS by 15 years if there were no white matter changes on MRI: 1/4. If even one white matter change was present: 3/4.
Typical Optic Neuritis

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT)

Is the ONTT one of those trials I’m expected to know by name? Yes it is

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

Optic Neuropathy

Optic neuritis

Typical

Atypical

Noninflammatory

Ischemic

Compressive

Toxic/nutritional

Congenital/hereditary

Traumatic

Infectious

Immune
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Noninflammatory
  - Ischemic
  - Compressive
  - Toxic/nutritional
  - Congenital/hereditary
  - Traumatic

Atypical

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT)

Is the ONTT one of those trials I'm expected to know by name? Yes it is

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if...

- there were no white matter changes on MRI: 1/4
- if even one white matter change was present: 3/4
Optic Neuropathy

Typical Optic Neuritis

Optic neuritis

Typical (demyelinating)

Atypical

Noninflammatory

- Ischemic
- Compressive
- Traumatic/nutritional
- Congenital/hereditary

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if there were no white matter changes on MRI:

- 1/4 if even one white matter change was present: 3/4

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)
What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if…
…there were no white matter changes on MRI: 1/4

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)
Optic Neuropathy

Optic neuritis

Typical

Atypical

Noninflammatory

- Ischemic
- Compressive
- Traumatic/nutritional
- Congenital/hereditary

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI?

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS?
The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if…

…there were no white matter changes on MRI: 1/4

…if even one white matter change was present:

1/4

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI?

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS?
The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if…
…there were no white matter changes on MRI: 1/4
…if even one white matter change was present: 3/4
What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS? The Optic Neuritis Treatment Trial (ONTT).

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if…
…there was no white-matter change on MRI?
…if even one white-matter change was present?

Why is knowing the likelihood of developing MS important?

Because it influences decision-making vis a vis whether to initiate tx that can forestall MS onset (and may improve dz course).
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI?

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS?
The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if:

...there were no white matter changes on MRI:

1/4

...if even one white matter change was present:

3/4

Why is knowing the likelihood of developing MS important?

Because it influences decision-making vis a vis whether to initiate tx that can forestall MS onset (and may improve dz course)

**The likelihood of developing multiple sclerosis (MS)**
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis – noninflammatory

Typical

(demyelinating)

Atypical

Infectious

Immune

Ischemic

Compressive

Toxic/nutritional

Congenital/hereditary

Traumatic

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI?

To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)

What was the name of the study that followed typical optic neuritis pts over many years, and (among other things) assessed their risk of developing MS?

The Optic Neuritis Treatment Trial (ONTT)

Per the ONTT, what proportion of typical optic neuritis pts develop MS by 15 years if...

…there were no white-matter changes on MRI?

1/4

…if even one white-matter change was present?

3/4

Why is knowing the likelihood of developing MS important?

Because it influences decision-making vis a vis whether to initiate tx that can forestall MS onset (and may improve dz course)

We will have much more to say about MS later in the set
The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Typical Optic Neuritis

Steroids

Two steroids were used—what were they? How were they dosed?

– IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
– PO prednisone. 1 mg/kg/d x 14 days, then tapered off.

With respect to vision, to what extent did steroids provide a long-term benefit?

None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?

The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have any positive effects on MS risk?

IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?

Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis. So PO pred @1 mg/kg/d doesn’t help, and may harm, optic neuritis pts.
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

- IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
- PO prednisone. 1 mg/kg/d x 14 days, then tapered off.

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have any positive effects on MS risk?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

So PO pred @1 mg/kg/d doesn't help, and may harm, optic neuritis pts.
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they?
--?
--?
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they?
--IV methylprednisolone
--PO prednisone
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. ?
--PO prednisone

IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation.
But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

So PO pred @1 mg/kg/d doesn’t help, and may harm, optic neuritis pts.
**Typical Optic Neuritis**

The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have any positive effects on MS risk?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

So PO pred @1 mg/kg/d doesn’t help, and may harm, optic neuritis pts.
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. ?

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have any positive effects on MS risk?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

So PO pred @1 mg/kg/d doesn’t help, and may harm, optic neuritis pts.
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?

--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?

None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision faster than the control group.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.
So PO pred @1 mg/kg/d doesn't help, and may harm, optic neuritis pts.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?
- IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
- PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had white-matter lesions at presentation
**Typical Optic Neuritis**

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?

--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?

None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?

The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?

IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation.
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by # years post-event, there was no difference in the rate of MS development between these groups.
**Typical Optic Neuritis**

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

**Steroids**

Two steroids were used—what were they? How were they dosed?

--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of...
Optic Neuropathy

Optic neuritis

Noninflammatory

Typical (demyelinating)

Atypical

Infectious

Immune

Ischemic

Compressive

Toxic/nutritional

Congenital/hereditary

Traumatic

113

Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?

--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)

--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?

None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?

The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?

IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?

Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.
The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis. So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
Optic Neuropathy

Optic neuritis
- Noninflammatory
- Typical (demyelinating)
- Atypical
- Infectious
- Immune
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

Does this mean PO pred is contraindicated in typical optic neuritis?

So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
- IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
- PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed MS development in pts with 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

Does this mean PO pred is contraindicated in typical optic neuritis?
No, subsequent studies found that megadose PO steroids hasten VA recovery without increasing the risk of recurrence.

So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis?

Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

Does this mean PO pred is contraindicated in typical optic neuritis?
No, subsequent studies found that megadose PO steroids hasten VA recovery without increasing the risk of recurrence.

So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
Typical Optic Neuritis

The ONTT evaluated what sort of drug as tx for typical optic neuritis? Steroids

Two steroids were used—what were they? How were they dosed?
---IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
---PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

Does this mean PO pred is contraindicated in typical optic neuritis?
No, subsequent studies found that megadose PO steroids hasten VA recovery without increasing the risk of recurrence.

‘Megadose’? How much pred are we talking about here?

So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
The ONTT evaluated what sort of drug as tx for typical optic neuritis?
Steroids

Two steroids were used—what were they? How were they dosed?
--IV methylprednisolone. 250 qid x 3 days (then pred 1 mg/kg/d x 11d, then tapered off)
--PO prednisone. 1 mg/kg/d x 14 days, then tapered off

With respect to vision, to what extent did steroids provide a long-term benefit?
None. The final VA outcome of the Steroid group was no different than that of the control group.

Did steroids have any positive effects on vision?
The IV group regained their final (best) vision a week or two faster than the control group—although to reiterate for emphasis, their final VA was not better than that of the controls. (The PO steroid group did not enjoy even this modest benefit.)

Did steroids have a positive impact on the risk of developing MS?
IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation. But as was the case with VA, eventually this outcome difference between the IV steroid and control groups disappeared—by 3 years post-event, there was no difference in the rate of MS development between these groups.

Did steroids have any negative effects?
Indeed they did—the PO pred group had an increased risk of recurrence of optic neuritis.

Does this mean PO pred is contraindicated in typical optic neuritis?
No, subsequent studies found that megadose PO steroids hasten VA recovery without increasing the risk of recurrence

‘Megadose’? How much pred are we talking about here?
A gram a day (same as the IV dose of methylprednisolone in the ONTT)

So PO pred @1 mg/kg/d doesn’t help—and seems to harm—optic neuritis pts.
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That's it.

As a review: Who is the typical typical optic neuritis pt?

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what is the workup that should be done?

**As a review: Who is the typical typical optic neuritis pt?**

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)

**Typical Optic Neuritis**

- Female
- Young adult

Optic Neuropathy

Noninflammatory
- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary

Atypical

Infectious

Immune

Traumatic

Toxic/nutritional

Congenital/hereditary

Ischemic

Compressive

Toxic/nutritional

Congenital/hereditary
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

Typical Optic Neuritis

Female
Young adult
VA loss
Nadirs over
Recovery starts
Pain with
Disc edema

Laterality
Amount of time
Amount of time

As a review: Who is the typical typical optic neuritis pt? How does a case of typical optic neuritis typically present?
Typical Optic Neuritis

Optic Neuropathy

- Noninflammatory
  - Ischemic
  - Compressive
  - Toxic/nutritional
  - Congenital/hereditary

Optic Neuritis

Atypical

- Infectious
- Immune

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?

MRI brain and orbits, with contrast. That's it.

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)

As a review: Who is the typical typical optic neuritis pt?

Female
Young adult
VA loss unilateral
Nadir over several days
Recovery starts <1 month
Pain with eye movement
Disc edema absent or mild

How does a case of typical optic neuritis typically present?
If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI? To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

As a review: Who is the typical typical optic neuritis pt?

Typical (demyelinating)
- Female
- Young adult
- VA loss unilateral
- Nadirs over several days
- Recovery starts <1 month
- Pain with eye movement
- Disc edema absent or mild

Atypical
- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

If the pt deviates from the typical pattern…

How does a case of typical optic neuritis typically present?

No question—proceed when ready
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...

<table>
<thead>
<tr>
<th>Optic Neuropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noninflammatory</td>
</tr>
<tr>
<td>Atypical</td>
</tr>
<tr>
<td>Infectious Immune</td>
</tr>
<tr>
<td>Ischemic</td>
</tr>
<tr>
<td>Compressive</td>
</tr>
<tr>
<td>Toxic/nutritional</td>
</tr>
<tr>
<td>Congenital/hereditary</td>
</tr>
<tr>
<td>Traumatic</td>
</tr>
</tbody>
</table>

If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done? MRI brain and orbits, with contrast. That’s it.

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS).

As a review: Who is the typical optic neuritis pt?
Female
Youth adult
VA loss unilateral
Nadirs over several days
Recovery starts ≤1 month
Pain with eye movement
Disc edema absent or mild

How does a case of typical optic neuritis typically present?
Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

If a typical pt presents with what seems to be an atypical case of typical optic neuritis, what sort of workup should be done?
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

No question—proceed when ready.
Optic Neuropathy

**If the pt or the presentation deviates from the typical pattern...**

You should question the dx of typical (demyelinating) optic neuritis

---

As a review: **Who is the typical optic neuritis pt?**

**Typical (demyelinating)?**
- Female
- Young adult
- VA loss unilateral
- Nadirs over several days
- Recovery starts <1 month
- Pain with eye movement
- Disc edema absent or mild

**Atypical?**
- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

---

**How does a case of typical optic neuritis typically present?**

**Typical case**
- Female
- Young adult
- VA loss unilateral
- Nadirs over several days
- Recovery starts <1 month
- Pain with eye movement
- Disc edema absent or mild

**Atypical case**
- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

---

What is the purpose of the MRI?
To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)

---

No question—proceed when ready
Optic Neuropathy

Optic neuritis

Typical (demyelinating) but not idiopathic or MS-related

Atypical

Infectious

Immune

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

No question—proceed when ready
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Optic neuritis

- Typical
  - Not idiopathic or MS-related
  - Infectious
  - Immune

- Atypical
  - Ischemic
  - Compressive
  - Toxic/nutritional
  - Congenital/hereditary
  - Traumatic

What etiologies?

- ?
- ?
- ?
- ?
- ?

(Cont)

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid
If the pt or the presentation deviates from the typical pattern,...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

**Typical Optic Neuritis**

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

**Optic neuritis**

- Typical (demyelinating) **but**
- Not idiopathic or MS-related
- Infectious
- Immune

**Atypical**

- Toxins/nutritional
- Congenital/hereditary
- Traumatic

**What etiologies?**

- Syphilis
- *Bartonella*
- Lyme testing (if endemic)
- Sarcoid
- SLE

(Cont)

- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Not idiopathic or MS-related

Infectious

Immune

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

What etiologies? What studies?
Syphilis: ?
Bartonella
Lyme testing (if endemic)
Sarcoid
SLE

(Cont)
Granulomatosis w/ polyangiitis
LHON
Meningeal process
NMO(SD)
MOGAD
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

Not idiopathic or MS-related

Infectious

Immune

Atypical

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

If the pt or the presentation deviates from the typical pattern...

You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies

What etiologies? What studies?

Syphilis: Serum and CSF RPR/TPPA
Bartonella
Lyme testing (if endemic)
Sarcoid
SLE

(Cont)
Granulomatosis w/ polyangiitis
LHON
Meningeal process
NMO(SD)
MOGAD
Optic Neuropathy

Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Optic neuritis

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

Atypical

- Traumatic
- Compressive
- Toxic/nutritional
- Congenital/hereditary

Typical

(demyelinating but not idiopathic or MS-related)

- Infectious
- Immune

What etiologies? What studies?
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: ?
- Lyme testing (if endemic)
- Sarcoid
- SLE

(Cont)
- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD
Optic Neuropathy

Optic neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis,
and institute a workup for infectious/autoimmune etiologies.

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic)
Sarcoid
SLE

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

(Cont)
Granulomatosis w/ polyangiitis
LHON
Meningeal process
NMO(SD)
MOGAD

Typical Optic Neuritis

Typical (demyelinating but not idiopathic or MS-related)

Atypical

Infectious

Immune

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

No

Neuropathy
Typical Optic Neuritis

If the patient or the presentation deviates from the typical pattern, you should question the diagnosis of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Optic neuritis

- Typical (demyelinating)
  - Not idiopathic or MS-related

- Atypical
  - Infectious
  - Immune

What etiologies? What studies?
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): ?
- Sarcoid
- SLE

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

(Cont)
- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD

Compressive
- Traumatic
- Toxic/nutritional
- Congenital/hereditary
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Neuropathy

Optic neuritis

Typical (demyelinating) but

Not idiopathic or MS-related

Infectious

Immune

Atypical

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

(Cont)

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid
SLE

Granulomatosis w/ polyangiitis
LHON
Meningeal process
NMO(SD)
MOGAD
Optic Neuropathy

**Typical Optic Neuritis**

If the pt or the presentation deviates from the typical pattern... You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies

Typical (demyelinating) but Not idiopathic or MS-related

Atypical

- Infectious
- Immune

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: ?

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

Granulomatosis w/ polyangiitis
LHON
Meningeal process
NMO(SD)
MOGAD
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Optic neuritis

- Typical
- Atypical

Typical (demyelinating)
- Not idiopathic or MS-related

Atypical
- Infectious
- Immune

What etiologies? What studies?
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE

What studies?
- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD

Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

- Compressive
  - Toxic/nutritional
  - Congenital/hereditary
  - Traumatic

(Cont)
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern... You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Optic neuritis

- Typical (demyelinating)
  - Not idiopathic or MS-related
  - Male
  - Older
  - VA loss bilateral
  - Progressive VA loss
  - No recovery after a month
  - Lack of pain
  - Disc edema severe/florid

- Atypical
  - Infectious
  - Immune

What etiologies? What studies?

- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE: ?

(Cont)
- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD
Optic Neuropathy

Optic neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

What etiologies? What studies?

- **Syphilis**: Serum and CSF RPR/TPPA
- **Bartonella**: IgM titers
- **Lyme testing (if endemic)**: Serum/CSF ELISA
- **Sarcoid**: Chest XR or CT; +/- Gallium/PET
- **SLE**: ESR, ANA, Anti-DNA

(Cont)
- Granulomatosis w/ polyangiitis
- LHON
- Meningeal process
- NMO(SD)
- MOGAD
Optic Neuropathy

**Typical Optic Neuritis**

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis,
and institute a workup for infectious/autoimmune etiologies.

Typical (demyelinating) optic neuritis is not idiopathic or MS-related.

Atypical optic neuritis can be infectious or immune-related.

**What etiologies? What studies?**

- **Syphilis**: Serum and CSF RPR/TPPA
- **Bartonella**: IgM titers
- **Lyme testing (if endemic)**: Serum/CSF ELISA
- **Sarcoid**: Chest XR or CT; +/- Gallium/PET
- **SLE**: ESR, ANA, Anti-DNA
- **Granulomatosis w/ polyangiitis**: ?
- **LHON**
- **Meningeal process**
- **NMO(SD)**
- **MOGAD**

**Male**
**Older**
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

**Compressive**
- Toxic/nutritional
- Congenital/heriteditary
- Traumatic
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern... You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

What etiologies? What studies?
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE: ESR, ANA, Anti-DNA

(Cont)
- Granulomatosis w/ polyangiitis: ANCA
- LHON
- Meningeal process
- NMO(SD)
- MOGAD
If the pt or the presentation deviates from the typical pattern…
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

**Typical Optic Neuritis**

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

**Atypical**

- Toxic/nutritional
- Congenital/hereditary
- Traumatic
- Ischemic
- Compressive

**Optic neuritis**

- **Typical**
  - Not idiopathic or MS-related
  - Infectious
  - Immune

- **Atypical**
  - (demyelinating *but* not idiopathic or MS-related)

*What etiologies? What studies?*

- **Syphilis:** Serum and CSF RPR/TPPA
- **Bartonella:** IgM titers
- **Lyme testing (if endemic):** Serum/CSF ELISA
- **Sarcoid:** Chest XR or CT; +/- Gallium/PET
- **SLE:** ESR, ANA, Anti-DNA

*(Cont)*

- **Granulomatosis w/ polyangiitis:** ANCA
- **LHON:** ?
- Meningeal process
- NMO(SD)
- MOGAD
Optic Neuropathy

Optic neuritis

Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

What etiologies? What studies?
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE: ESR, ANA, Anti-DNA

(Cont)
- Granulomatosis w/ polyangiitis: ANCA
- LHON: Genetic testing
- Meningeal process
- NMO(SD)
- MOGAD

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic
Optic Neuropathy

Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Neuropathy

Optic neuritis

- Typical (demyelinating but not idiopathic or MS-related)
- Infectious
- Immune

Atypical

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

Meningeal process: ?

What etiologies? What studies?

- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE: ESR, ANA, Anti-DNA

(Cont)
- Granulomatosis w/ polyangiitis: ANCA
- LHON: Genetic testing
- Meningeal process: ?
- NMO(SD)
- MOGAD
Optic Neuropathy

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

**Typical Optic Neuritis**

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

Optic neuritis

- Typical (demyelinating) but
- Atypical
  - Not idiopathic or MS-related
  - Infectious
  - Immune

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: Chest XR or CT; +/- Gallium/PET
SLE: ESR, ANA, Anti-DNA

(Cont)
Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD)
MOGAD
Optic Neuropathy

Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies

Typical (demyelinating) but not idiopathic or MS-related

Atypical

Not idiopathic or MS-related

Infectious

Immune

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: Chest XR or CT; +/- Gallium/PET
SLE: ESR, ANA, Anti-DNA

(Cont)
Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD): ?
MOGAD
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern…
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: Chest XR or CT; +/- Gallium/PET
SLE: ESR, ANA, Anti-DNA

(Cont)
Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD): Serum AQP4-IgG, spinal MRI
MOGAD
Optic Neuropathy

If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Typical Optic Neuritis

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

Optic neuritis

Typical (demyelinating but not idiopathic or MS-related)

Infectious

Immune

Atypical

Neuropathy

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: Chest XR or CT; +/- Gallium/PET
SLE: ESR, ANA, Anti-DNA

(Cont)
Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD): Serum AQP4-IgG, spinal MRI
MOGAD: ?
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern... You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies

Neuropathy

Optic neuritis

Typical (demyelinating) but

Not idiopathic or MS-related

Infectious

Immune

Atypical

Male

Older

VA loss bilateral

Progressive VA loss

No recovery after a month

Lack of pain

Disc edema severe/florid

What etiologies? What studies?

Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA
Sarcoid: Chest XR or CT; +/- Gallium/PET
SLE: ESR, ANA, Anti-DNA

(Cont)

Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD): Serum AQP4-IgG, spinal MRI
MOGAD: Serum MOG-IgG
If the pt or the presentation deviates from the typical pattern…
You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

**Typical Optic Neuritis**

- Male
- Older
- VA loss bilateral
- Progressive VA loss
- No recovery after a month
- Lack of pain
- Disc edema severe/florid

**Atypical**

- Ischemic
- Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

**What etiologies? What studies?**
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella: IgM titers
- Lyme testing (if endemic): Serum/CSF ELISA
- Sarcoid: Chest XR or CT; +/- Gallium/PET
- SLE: ESR, ANA, Anti-DNA
- Granulomatosis w/ polyangiitis: ANCA
- LHON: Genetic testing
- Meningoal process: LP with cytology
- NMO(SD): Serum AQP4-IgG, spinal MRI
- MOGAD: Serum MOG-IgG

*We will address these conditions in considerable detail later in the set*
Typical Optic Neuritis

If the pt or the presentation deviates from the typical pattern... You should question the dx of typical (demyelinating) optic neuritis, and institute a workup for infectious/autoimmune etiologies.

Neuropathy

Optic neuritis

Male
Older
VA loss bilateral
Progressive VA loss
No recovery after a month
Lack of pain
Disc edema severe/florid

But first let’s take a minute to drill down on MS

Typical (demyelinating but not idiopathic or MS-related)

Atypical

Infectious

Immune

What etiologies? What studies?
Syphilis: Serum and CSF RPR/TPPA
Bartonella: IgM titers
Lyme testing (if endemic): Serum/CSF ELISA

We will address these conditions in considerable detail later in the set

(Cont)
Granulomatosis w/ polyangiitis: ANCA
LHON: Genetic testing
Meningeal process: LP with cytology
NMO(SD): Serum AQP4-IgG, spinal MRI
MOGAD: Serum MOG-IgG
(This is a good point in the set to take a break)
What does CDMS stand for in this context?
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
What does CDMS stand for in this context? 
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in M vs F
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in life stage
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)
What does CDMS stand for in this context? 
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?
What does CDMS stand for in this context?  
Clinically-definite multiple sclerosis

In a nutshell, what is MS?  
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?  
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?  
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?  
Yes, it is more common in [ ]
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?
Yes, it is more common in Whites
What does CDMS stand for in this context? 
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?
Yes, it is more common in Whites

There is a geographic predilection—what is it?
What does CDMS stand for in this context? 
Clinically-definite multiple sclerosis

In a nutshell, what is MS? 
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection? 
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection? 
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection? 
Yes, it is more common in Whites

There is a geographic predilection—what is it? 
It is more prevalent among people who live closer to the equator
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?
Yes, it is more common in Whites

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator
What does CDMS stand for in this context?
Clinically-definite multiple sclerosis

In a nutshell, what is MS?
An inflammatory neurodegenerative disorder of the CNS that produces progressive
disability over time

Is there a gender predilection?
Yes, it is more common in women (2-3 times more common, in fact)

Is there an age predilection?
Yes, it is more common in young adults (age 25-40)

Is there a racial predilection?
Yes, it is more common in Whites

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

What is the classic two-word description of the typical clinical course in MS?
CDMS: *Basics*

*What does CDMS stand for in this context?*
Clinically-definite multiple sclerosis

*In a nutshell, what is MS?*
An inflammatory neurodegenerative disorder of the CNS that produces progressive disability over time

*Is there a gender predilection?*
Yes, it is more common in women (2-3 times more common, in fact)

*Is there an age predilection?*
Yes, it is more common in young adults (age 25-40)

*Is there a racial predilection?*
Yes, it is more common in Whites

*There is a geographic predilection—what is it?*
It is more prevalent among people who live farther from the equator

*What is the classic two-word description of the typical clinical course in MS?*
‘Relapsing-remitting’
Are ocular manifestations common in MS?
Are ocular manifestations common in MS? Indeed they are—optic neuritis occurs in 75% of MS cases.
Are ocular manifestations common in MS? Indeed they are—optic neuritis occurs in 75% of MS cases.
CDMS: Manifestations

Nonocular

Ocular

Are ocular manifestations common in MS?
Indeed they are—optic neuritis occurs in 75% of MS cases (and is the presenting symptom in ___%.)
CDMS: Manifestations

Nonocular  Ocular

Are ocular manifestations common in MS? Indeed they are—optic neuritis occurs in 75% of MS cases (and is the presenting symptom in 25%.)
Are ocular manifestations common in MS? Indeed they are—optic neuritis occurs in 75% of MS cases (and is the presenting symptom in 25%)

Three non-neuritis ocular manifestations are often encountered as well—what are they?
CDMS: Manifestations

Nonocular

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Are ocular manifestations common in MS? Indeed they are—optic neuritis occurs in 75% of MS cases (and is the presenting symptom in 25%)

Three non-neuritis ocular manifestations are often encountered as well—what are they?
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

Optic neuritis S/S

Nystagmus/oscillations

Diplopia

Uveitis

In a nutshell, what is a nystagmus and/or oscillation?
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.
In a nutshell, what is a nystagmus and/or oscillation? Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.
In a nutshell, what is a nystagmus and/or oscillation? Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation? In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.
In a nutshell, what is a nystagmus and/or oscillation? Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

But I thought jerk nystagmus was fast, and pendular nystagmus was slow. What’s the deal?
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

But I thought jerk nystagmus was fast, and pendular nystagmus was slow. What’s the deal?
You thought correct—jerk is fast, pendular slow. But these terms refer to the speed of the refixation movement—the initial displacement is slow in both.
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

Is nystagmus/oscillations a common, or rare occurrence in MS?
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

In a nutshell, what is a nystagmus and/or oscillation? Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation? In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

Is nystagmus/oscillations a common, or rare occurrence in MS? Common (especially nystagmus)
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

Is nystagmus/oscillations a common, or rare occurrence in MS?
Common (especially nystagmus)

Is there a particular direction (ie, horizontal, vertical, rotary) in which the nystagmus tends to manifest?
In a nutshell, what is a nystagmus and/or oscillation?
Both are involuntary eye-movement patterns that involve displacement of gaze off of its intended target, followed by a refixation movement.

What is the difference between a nystagmus and an oscillation?
In a nystagmus, the velocity of the displacement movement is by definition slow, whereas in an oscillation it’s by definition fast.

Is nystagmus/oscillations a common, or rare occurrence in MS?
Common (especially nystagmus)

Is there a particular direction (ie, horizontal, vertical, rotary) in which the nystagmus tends to manifest?
No—it can be any direction (and either jerk or pendular)
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

Is diplopia a common manifestation of MS?

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
CDMS: Manifestations

Typical Optic Neuritis

Nonocular

Ocular

Is diplopia a common manifestation of MS?
Indeed it is

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.
**Is diplopia a common manifestation of MS?**

Indeed it is

**Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?**

3, 4 and 6

**Step-back II: The EOM control pathway has four levels or subsections. What are they?**

--?

--?

--?

--?
CDMS: **Manifestations**

**Nonocular**

Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

**Step-back II: The EOM control pathway has four levels or subsections. What are they?**

-- The *Supranuclear* pathways
-- The *Internuclear* pathway
-- The *Nuclear* level: The CN3, 4 and 6 nuclei themselves
-- The *Infranuclear* pathway
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

- **The Supranuclear pathways**
  - The Internuclear pathway

**Broadly speaking, what constitutes the supranuclear pathways?**
CDMS: Manifestations

Nonocular

Is diplopia a common manifestation of MS?
Indeed it is

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?
-- The Supranuclear pathways
-- The Internuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?
Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei.

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
   - The Internuclear pathway

Broadly speaking, what constitutes the supranuclear pathways? Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

---The **Supranuclear pathways**

- The Internuclear pathway

_Broadly speaking, what constitutes the supranuclear pathways?_ Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei. Examples of supranuclear dysfunction include **Parinaud syndrome**, PSP, and **convergence** or **divergence excess/insufficiency**.
Is diplopia a common manifestation of MS?
Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
  - The Internuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?
Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei. Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

What does PSP stand for in this context?
**Is diplopia a common manifestation of MS?**
Indeed it is.

**Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?**
3, 4 and 6.

**Step-back II: The EOM control pathway has four levels or subsections. What are they?**

---
**The Supranuclear pathways**
- The Internuclear pathway

**Broadly speaking, what constitutes the supranuclear pathways?**
Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei. Examples of supranuclear dysfunction include Parinaud syndrome, **PSP**, and convergence or divergence excess/insufficiency.

What does PSP stand for in this context? Progressive **supranuclear** palsy.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

-The Supranuclear pathways
-The Internuclear pathway
-The Nuclear level: The CN3, 4 and 6 nuclei themselves
-The Infranuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?

Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are 'supra' in that they carry signals to the nuclei.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

It should be noted that, generally speaking, diplopia is not a feature of supranuclear pathway lesions.

This is because most supranuclear-pathway lesions affect both eyes symmetrically. Notable exceptions are lesions of the convergence and divergence control mechanisms.

CDMS: Manifestations
CDMS: Manifestations

Nonocular

Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

- The Supranuclear pathways
- The Internuclear pathway
- The Nuclear level: The CN3, 4 and 6 nuclei themselves
- The Infranuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?

Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

 Typical Optic Neuritis

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

It should be noted that, generally speaking, diplopia isn’t a feature of supranuclear pathway lesions.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

- The supranuclear pathways
- The internuclear pathway
- The nuclear level: The CN3, 4 and 6 nuclei themselves
- The infranuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?

Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

It should be noted that, generally speaking, diplopia isn’t a feature of supranuclear pathway lesions. This is because most supranuclear-pathway lesions affect three words.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

--- The Supranuclear pathways

- Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are 'supra' in that they carry signals to the nuclei.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

It should be noted that, generally speaking, diplopia isn’t a feature of supranuclear pathway lesions. This is because most supranuclear-pathway lesions affect both eyes symmetrically.
Typical Optic Neuritis

CDMS: **Manifestations**

Nonocular

Is diplopia a common manifestation of MS? Indeed it is

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- **Diplopia**
- Uveitis

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

--- **The Supranuclear pathways**

- The Supranuclear pathways
  - Inputs to the nuclei from centers in the cortex, cerebellum, vestibular system, etc. These locations are ‘supra’ in that they carry signals to the nuclei.
  - Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.

It should be noted that, generally speaking, diplopia isn’t a feature of supranuclear pathway lesions. This is because most supranuclear-pathway lesions affect both eyes symmetrically. Notable exceptions are lesions of the convergence and divergence control mechanisms.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?

3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

--- The Supranuclear pathways
- The Internuclear pathway
- The Nuclear level: The CN3, 4 and 6 nuclei themselves
- The Infranuclear pathway

It should be noted that, generally speaking, diplopia isn’t a feature of supranuclear pathway lesions. This is because most supranuclear-pathway lesions affect both eyes symmetrically. Notable exceptions are lesions of the convergence and divergence control mechanisms.

Examples of supranuclear dysfunction include Parinaud syndrome, PSP, and convergence or divergence excess/insufficiency.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections.

-- The **Supranuclear** pathways
-- The **Internuclear** pathway
-- The **Nuclear** level: The CN3, 4 and 6 nuclei themselves
-- The **Infranuclear** pathway

**Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance?**

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections.

--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathways
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6

What is the name of the internuclear connection shared by these two nuclei? The medial longitudinal fasciculus (MLF).
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6 nuclei themselves
-- The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance?
3 and 6

What is the name of the internuclear connection shared by these two nuclei?
The medial longitudinal fasciculus (MLF)
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections.

-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6
-- The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.

What is the name of the internuclear connection shared by these two nuclei? The medial longitudinal fasciculus (MLF).

Damage to the MLF results in what clinical condition?
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections.
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.

What is the name of the internuclear connection shared by these two nuclei? The medial longitudinal fasciculus (MLF).

Damage to the MLF results in what clinical condition? An internuclear ophthalmoplegia (INO).
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections:
--The supranuclear pathways
--The internuclear pathway
--The nuclear level: The CN3, 4 and 6 nuclei themselves
--The infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.

What is the name of the internuclear connection shared by these two nuclei? The medial longitudinal fasciculus (MLF).

Damage to the MLF results in what clinical condition? An internuclear ophthalmoplegia (INO).

Typical Optic Neuritis

In a nutshell, how does a unilateral INO manifest?

Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a bilateral INO manifest?

With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for "whole bilateral INO").

If you see a young person with a WEBINO, think MS first.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

In a nutshell, how does a unilateral INO manifest?
Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus.

Bilateral INO manifest?
With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for "w"all-e" b"ilateral INO).

If you see a young person with a WEBINO, think MS first.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?
-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6 nuclei themselves.
-- The Infranuclear pathway.

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance?
3 and 6.

What is the name of the internuclear connection shared by these two nuclei?
The medial longitudinal fasciculus (MLF).

Damage to the MLF results in what clinical condition?
An internuclear ophthalmoplegia (INO).

In a nutshell, how does a unilateral INO manifest?
Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a bilateral INO manifest?
With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for 'w' all-e 'x' 'torted b'ilateral INO).

If you see a young person with a WEBINO, think MS first.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections.
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

In a nutshell, how does a unilateral INO manifest? Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

Damage to the MLF results in what clinical condition? An internuclear ophthalmoplegia (INO).

If you see a young person with a WEBINO, think MS first.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?
-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6 nuclei themselves
-- The Infranuclear pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.

What is the name of the internuclear connection shared by these two nuclei? The medial longitudinal fasciculus (MLF).

Damage to the MLF results in what clinical condition? An internuclear ophthalmoplegia (INO).

In a nutshell, how does a unilateral INO manifest?
Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a bilateral INO manifest?
With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for "w"all-e" b"ilateral INO).
**CDMS: Manifestations**

**Nonocular**

Is diplopia a common manifestation of MS? Indeed it is.

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?

-- The **Supranuclear** pathways
-- The **Internuclear** pathway
-- The **Nuclear** level: The CN3, 4 and 6 nuclei themselves
-- The **Infranuclear** pathway

Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance?

3 and 6

What is the name of the internuclear connection shared by these two nuclei?

The **medial longitudinal fasciculus** (MLF)

Damage to the MLF results in what clinical condition?

An **internuclear ophthalmoplegia (INO)**

**Typical Optic Neuritis**

In a nutshell, how does a unilateral INO manifest?

Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a **bilateral** INO manifest?

With the same motility difficulties, but on attempted lateral gaze in **either** direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for **W**all-e**d** **b**ilateral **I**nternuclear **O**phthalmoplegia **N**). If you see a young person with a WEBINO, think MS first.
Is diplopia a common manifestation of MS?
Indeed it is

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?
3, 4 and 6

Step-back II: The EOM control pathway
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

In a nutshell, how does a unilateral INO manifest?
Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a bilateral INO manifest?
With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for wall-eyed bilateral INO).

Damage to the MLF results in what clinical condition?
An internuclear ophthalmoplegia (INO)
Typical Optic Neuritis

Right gaze  Primary  Left gaze

WEBINO
Is diplopia a common manifestation of MS? Indeed it is

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Step-back II: The EOM control pathway:
-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6 nuclei themselves
-- The Infranuclear pathways

What is the name of the internuclear connection shared by these two cranial nerve nuclei? The medial longitudinal fasciculus (MLF)

Damage to the MLF results in what clinical condition? An internuclear ophthalmoplegia (INO)

In a nutshell, how does a unilateral INO manifest? Upon attempted lateral gaze, the adducting eye adducts slowly (or not at all), while the abducting eye abducts fully, but displays an end-point nystagmus. Additionally, the eye on the abducting side may be exotropic in primary gaze.

How does a bilateral INO manifest? With the same motility difficulties, but on attempted lateral gaze in either direction. Both eyes are often exotropic in primary, resulting in a WEBINO (acronym for wall-eyed bilateral INO).

If you see a young person with a WEBINO, think MS!

Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Typical Optic Neuritis

CDMS: Manifestations

What constitutes the infranuclear pathway?

--The Infranuclear pathway

The Nuclear level: The CN3, 4 and 6 nuclei themselves

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?

3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, i.e., the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a 'nerve').

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn't come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated EOM abnormalities; i.e., the ophthalmoparesis is almost always accompanied by nonocular signs and symptoms of CNS damage—that is, a stroke-like presentation.
Typical Optic Neuritis

CDMS: Manifestations

What constitutes the infranuclear pathway?
Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the EOMs themselves.

The Nuclear level: The CN3, 4 and 6 nuclei themselves

--The Infranuclear pathway
What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space, the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the EOMs themselves.

Note that the only portion of the infranuclear pathway that is located within the CNS is the \textit{portion}, ie, the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem.

---

**Typical Optic Neuritis**

CDMS: \textit{Manifestations}

Manifestations

- Nonocular
- Ocular

Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?

3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

---

- The \textit{Supranuclear} pathways
- The \textit{Internuclear} pathway
- The \textit{Nuclear} level: The CN 3, 4 and 6 nuclei themselves
- The \textit{Infranuclear} pathway

Typical Optic Neuritis S/S
- Uveitis S/S
- Nystagmus/Oscillation

---

The Nuclear level: The CN 3, 4 and 6 nuclei themselves

---

The \textit{Infranuclear} pathway
Typical Optic Neuritis

CDMS: Manifestations

What constitutes the infranuclear pathway?
Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space, the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the EOMs themselves.

Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, ie, the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem.

--The Infranuclear pathway
Typical Optic Neuritis

Aqueduct

3rd nerve nucleus

Red nucleus

Cerebral peduncle

Third nerve fascicle

Cranial nerve fascicle
What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the EOMs themselves. Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, ie, the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve.’)

---

The Intracranial level: The CNs, 3 and 6 nuclei themselves

---

The Infranuclear pathway
CDMS: Manifestations

What constitutes the infranuclear pathway?
Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the EOMs themselves. Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, ie, the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve.’)

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated EOM abnormalities; ie, the ophthalmoparesis is almost always accompanied by nonocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

--The Infranuclear pathway
CDMS: Manifestations

What constitutes the infranuclear pathway? Everything after the nuclei: the axons running through the subarachnoid space; the ‘cranial nerve’ portion passing through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the extraocular muscles themselves.

Note that the only portion of the infranuclear pathway that is located within the CNS is the **fascicular portion**, i.e., the axon bundle that has left the CN nucleus but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve’.)

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not produce isolated EOM abnormalities; i.e., the ophthalmoparesis is almost always accompanied by **nonocular** signs and symptoms of CNS damage—that is, a stroke-like presentation.

---

**The Infranuclear pathway**
CDMS: Manifestations

What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the Extraocular Muscles themselves. Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, i.e., the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. Only after these fibers have entered the subarachnoid space are they formally designated a ‘nerve.’

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated Extraocular Muscles (EOM) abnormalities; i.e., the ophthalmoparesis is almost always accompanied by non-ocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

--The Infranuclear pathway

What term is used to describe conditions presenting with motility dysfunction secondary to fascicle damage + non-ocular CNS findings? Fascicular syndrome
CDMS: Manifestations

What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the extraocular muscles themselves. Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, i.e., the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve.’

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated EOM abnormalities; i.e., the ophthalmoparesis is almost always accompanied by nonocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

--The Infranuclear pathway

Typical Optic Neuritis

What term is used to describe conditions presenting with motility dysfunction secondary to fascicle damage + non-ocular CNS findings? Fascicular syndrome

Four fascicular syndromes involve the CN3 fascicle—what are they?

--?

--?

--?

--?

Three fascicular syndromes involve the CN6 fascicle—what are they?

--Millard-Gubler syndrome

--Foville syndrome

--Raymond syndrome
CDMS: **Manifestations**

**Nonocular Manifestations**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?

- 3, 4 and 6

Step-back II: The EOM control pathway has four levels or subsections. What are they?

- The **Supranuclear** pathways
- The **Internuclear** pathway
- The **Nuclear** level: The CN3, 4 and 6 nuclei themselves
- The **Infranuclear** pathway

**Typical Optic Neuritis**

What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the extraocular muscles themselves.

Note that the only portion of the infranuclear pathway that is located within the CNS is the **fascicular portion**, i.e., the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve’.)

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that generally speaking, lesions of the nuclei and/or fascicles do not present with isolated extraocular muscle abnormalities; ophthalmoparesis is almost always accompanied by nonocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

---

**Fascicular syndrome**

Four fascicular syndromes involve the CN3 fascicle—what are they?

- Weber syndrome
- Benedikt syndrome
- Claude syndrome
- Nothnagel syndrome

---
What constitutes the infranuclear pathway?
Everything after the nuclei: the axons as they run through the brainstem to enter the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the extraocular muscles themselves. Note that the only portion of the infranuclear pathway that is located within the CNS is the fascicular portion, that is, the axon bundle that has left the CN nucleus, but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve.’)

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated extraocular muscle abnormalities; that is, the ophthalmoparesis is almost always accompanied by non-ocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

--The Infranuclear pathway

**Typical Optic Neuritis**

**CDMS: Manifestations**

What term is used to describe conditions presenting with motility dysfunction secondary to fascicle damage + non-ocular CNS findings?

**Fascicular syndrome**

Four fascicular syndromes involve the CN3 fascicle—what are they?
--Weber syndrome
--Benedikt syndrome
--Claude syndrome
--Nothnagel syndrome

Three fascicular syndromes involve the CN6 fascicle—what are they?
--?
--?
--?
What constitutes the infranuclear pathway? Everything after the nuclei: the axons as they run through the subarachnoid space; the ‘cranial nerve’ portion as it passes through the subarachnoid space into the cavernous sinus and then the orbit to the neuromuscular junction; the junction itself; and finally the extraocular muscles themselves. Note that the only portion of the infranuclear pathway that is located entirely within the CNS is the fascicular portion, i.e., the axon bundle that has left the CN nucleus but is still within the substance of the brainstem. (It is only after these fibers have entered the subarachnoid space that they are formally designated a ‘nerve’.)

The cranial-nerve nuclei and their fascicles are located within the brainstem. Given this, it shouldn’t come as a surprise that, generally speaking, lesions of the nuclei and/or fascicles do not present with isolated EOM abnormalities; i.e., the ophthalmoparesis is almost always accompanied by nonocular signs and symptoms of CNS damage—that is, a stroke-like presentation.

-- The Infranuclear pathway
CDMS: Manifestations

Nonocular

Is diplopia a common manifestation of MS?
Indeed it is

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

At last: Which of these portions of the EOM control pathway can be affected in MS?
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Is diplopia a common manifestation of MS?
Indeed it is

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)?
3, 4 and 6

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Step-back II: The EOM control pathway has four levels or subsections. What are they?
-- The Supranuclear pathways
-- The Internuclear pathway
-- The Nuclear level: The CN3, 4 and 6 nuclei themselves
-- The Infranuclear pathway

At last: Which of these portions of the EOM control pathway can be affected in MS?
Recall that MS was defined as a neurodegenerative disorder of the CNS. The last portion of the EOM pathway that is located within the CNS is the nerve .
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

At last: Which of these portions of the EOM control pathway can be affected in MS? Recall that MS was defined as a neurodegenerative disorder of the CNS. The last portion of the EOM pathway that is located within the CNS is the nerve fascicles.
Is diplopia a common manifestation of MS? Indeed it is.

Taking a step back: Which three cranial nerves are responsible for controlling eye position (and thus are implicated in diplopia)? 3, 4 and 6.

Step-back II: The EOM control pathway has four levels or subsections. What are they?
--The Supranuclear pathways
--The Internuclear pathway
--The Nuclear level: The CN3, 4 and 6 nuclei themselves
--The Infranuclear pathway

At last: Which of these portions of the EOM control pathway can be affected in MS? Recall that MS was defined as a neurodegenerative disorder of the CNS. The last portion of the EOM pathway that is located within the CNS is the nerve fascicles. Thus, MS damage can (and does) occur in the supranuclear, internuclear, and nuclear portions, as well as the fascicular section of the infranuclear portion.
MS conveys an increased risk of uveitis. How much?

Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
MS conveys an increased risk of uveitis. How much?
MS pts are \# times more likely to experience uveitis than are non-MS individuals!
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point?
CDMS: *Manifestations*

Typical Optic Neuritis

**Nonocular**

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

*What proportion of MS pts will develop uveitis at some point? About 1/3*

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point? About 1/3

In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point?
About 1/3

In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?
To follow

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point?
About 1/3

In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?
To follow, but: As many as % of MS pts will manifest uveitis up to prior to their eventual MS diagnosis!

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point?
About 1/3

In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?
To follow, but: As many as 25% of MS pts will manifest uveitis up to 10 years prior to their eventual MS diagnosis!

Typical Optic Neuritis

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis
MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

What proportion of MS pts will develop uveitis at some point?
About 1/3

In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?
To follow, but: As many as 25% of MS pts will manifest uveitis up to 10 years prior to their eventual MS diagnosis!

MS has several HLA associations, one of which conveys a higher risk of developing uveitis. Which one?
**Typical Optic Neuritis**

**CDMS: Manifestations**

**Nonocular**

MS conveys an increased risk of uveitis. How much?

**MS pts are 10 times more likely to experience uveitis than are non-MS individuals!**

What proportion of MS pts will develop uveitis at some point?

About 1/3

**In those MS pts who develop it: Does it tend to precede, or follow, their MS diagnosis?**

To follow, **but**: As many as 25% of MS pts will manifest uveitis up to 10 years prior to their eventual MS diagnosis!

**MS has several HLA associations, one of which conveys a higher risk of developing uveitis. Which one?**

HLA-DR15

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- **Uveitis**
**CDMS: Manifestations**

**Nonocular**

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

_Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they?_

--?

--?

--?

--?


**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- **Uveitis**
CDMS: Manifestations

Nonocular

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

Taking a step back: *There are four types of uveitis, based on the location of the inflammation. What are they?*

--- Anterior
--- Intermediate
--- Posterior
--- Panuveitis

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Typical Optic Neuritis
**CDMS: Manifestations**

**Nonocular**

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

**Ocular**

--- Optic neuritis S/S
--- Nystagmus/oscillations
--- Diplopia

**Uveitis**

--- Anterior: ?
--- Intermediate
--- Posterior
--- Panuveitis

--- Typical Optic Neuritis
**CDMS: Manifestations**

**Nonocular**

*MS conveys an increased risk of uveitis. How much?*  
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

_Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?_

--Anterior: The primary location of inflammation is the _antior chamber_ and/or _anterior vitreous_

--Intermediate: The primary location of inflammation is the _main vitreous cavity_ , +/- the _peripheral retina_

--Posterior: _the site of inflammation is the retina_ and/or _choroid_ (the _optic nerve head_ can be involved too)

--Panuveitis

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia

_Uveitis_

**Typical Optic Neuritis**
**CDMS: Manifestations**

**Nonocular**

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- **Uveitis**

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

*Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?*

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate

--Posterior

--Panuveitis
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: ?

--Posterior

--Panuveitis

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

Typical Optic Neuritis
CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate: The primary location of inflammation is the two or three words, +/- the

--Posterior

--Panuveitis

Ocular

Optic neuritis S/S

Nystagmus/oscillations

Diplopia

Uveitis
**CDMS: Manifestations**

**Nonocular**

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia

**Uveitis**

*Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?*

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

--Posterior

--Panuveitis
**CDMS: Manifestations**

**Nonocular**

*MS conveys an increased risk of uveitis. How much?*  
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

**Ocular**

- **Optic neuritis S/S**
- **Nystagmus/oscillations**
- **Diplopia**
- **Uveitis**

*Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?*

- **Anterior**: The primary location of inflammation is the anterior chamber and/or anterior vitreous
- **Intermediate**: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
- **Posterior**: ?
- **Panuveitis**
MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis
MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid
--Panuveitis
**CDMS: Manifestations**

**Nonocular**

*MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!*

*Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?*

--- Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--- Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--- Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--- Panuveitis

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis
**CDMS: Manifestations**

**Nonocular**

- MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

**Ocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

*Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?*

--- Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--- Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

--- Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

--- Panuveitis: ?
CDMS: Manifestations

Nonocular

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

Typical Optic Neuritis

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved
 Trails of inflammation: Optic neuritis S/S, Nystagmus/oscillations, Diplopia, Uveitis

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS? Intermediate uveitis

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis
MS conveys an increased risk of uveitis. How much?

MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?

Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition?

It is bilateral in almost all (>95%) cases

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?

Yes, about 15% of intermediate-uveitis pts will develop MS

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?

--Vitritis, which tends to be mild

--Periphlebitis
CDMS: Manifestations

- Nonocular
  - Optic neuritis S/S
  - Nystagmus/oscillations
  - Diplopia
  - Uveitis

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

- **Anterior**: The primary location of inflammation is the anterior chamber and/or anterior vitreous
- **Intermediate**: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
- **Posterior**: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
- **Panuveitis**: All three locations are equally involved

Which form is most likely to occur in MS?
Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral.

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, i.e., are individuals with intermediate uveitis at higher risk of developing MS?
Yes, about 15% of intermediate-uveitis pts will develop MS.

Two specific manifestations (i.e., signs) of intermediate uveitis are classically associated with MS—which ones?
- Vitritis, which tends to be mild
-Periphlebitis

Typical Optic Neuritis
CDMS: Manifestations

Nonocular Manifestations

Ocular Manifestations

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

MS conveys an increased risk of uveitis. How much?
MS pts are 10 times more likely to experience uveitis than non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?
Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral in almost all (>95%) cases.

Typical Optic Neuritis

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral in almost all (>95%) cases.
CDMS: Manifestations

Nonocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

- **Anterior**: The primary location of inflammation is the anterior chamber and/or anterior vitreous
- **Intermediate**: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
- **Posterior**: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
- **Panuveitis**: All three locations are equally involved

Which form is most likely to occur in MS?

Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition?

It is bilateral in almost all (>95%) cases

MS conveys an increased risk of uveitis. How much?

MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?

- Vitritis, which tends to be mild
-Periphlebitis

MS conveys an increased risk of uveitis S/S

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

- **Anterior**: The primary location of inflammation is the anterior chamber and/or anterior vitreous
- **Intermediate**: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
- **Posterior**: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
- **Panuveitis**: All three locations are equally involved

Which form is most likely to occur in MS?

Intermediate uveitis
CDMS: Manifestations

**Typical Optic Neuritis**

MS conveys an increased risk of uveitis. How much?

MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous

--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?

Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition?

It is bilateral in almost all (>95%) cases

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?

Yes, about 15% of intermediate-uveitis pts will develop MS

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?

--Vitritis, which tends to be mild

--Periphlebitis
**CDMS: Manifestations**

**Optic Neuritis S/S**
- Nystagmus/oscillations
- Diplopia
- Uveitis

**MS conveys an increased risk of uveitis. How much?**
MS pts are 10 times more likely to experience uveitis than non-MS individuals!

**Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?**

- **Anterior:** The primary location of inflammation is the anterior chamber and/or anterior vitreous.
- **Intermediate:** The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina.
- **Posterior:** The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too).
- **Panuveitis:** All three locations are equally involved.

**Which form is most likely to occur in MS?**
Intermediate uveitis

**Is intermediate uveitis in MS a unilateral, or bilateral condition?**
It is bilateral in almost all (>95%) cases.

**MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?**
Yes, about 15% of intermediate-uveitis pts will develop MS.

**Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?**
- Vitritis, which tends to be mild
-Periphlebitis
**CDMS: Manifestations**

**Nonocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

**Ocular**

- **Optic neuritis**
- **Uveitis**

MS conveys an increased risk of uveitis. How much? MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

- **Anterior:** The primary location of inflammation is the anterior chamber and/or anterior vitreous
- **Intermediate:** The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
- **Posterior:** The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
- **Panuveitis:** All three locations are equally involved

Which form is most likely to occur in MS? Intermediate uveitis

Is intermediate uveitis in MS a unilateral, or bilateral condition? It is bilateral in almost all (>95%) cases

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS? Yes, about 15% of intermediate-uveitis pts will develop MS.

Typical Optic Neuritis
CDMS: Manifestations

Typical Optic Neuritis

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral in almost all ( >95% ) cases

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?
Yes, about 15% of intermediate-uveitis pts will develop MS

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?
--?
--?

--Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?
Intermediate uveitis
CDMS: Manifestations

Typical Optic Neuritis

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral in almost all (>95%) cases

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?
Yes, about 15% of intermediate-uveitis pts will develop MS

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?
--Vitritis
--Periphlebitis

Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved

Which form is most likely to occur in MS?
Intermediate uveitis
CDMS: Manifestations

Is intermediate uveitis in MS a unilateral, or bilateral condition?
It is bilateral in almost all (>95%) cases.

MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?
Yes, about 15% of intermediate-uveitis pts will develop MS.

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?
--Vitritis, which tends to be mild vs severe
--Periphlebitis

Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina.
--Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--Panuveitis: All three locations are equally involved.

Which form is most likely to occur in MS?
Intermediate uveitis.
CDMS: Manifestations

**Nonocular**

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

**Ocular**

*MS conveys an increased risk of uveitis. How much?*

MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

-- **Anterior:** The primary location of inflammation is the anterior chamber and/or anterior vitreous

-- **Intermediate:** The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

-- **Posterior:** The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

-- **Panuveitis:** All three locations are equally involved

Which form is most likely to occur in MS?

Intermediate uveitis

*Is intermediate uveitis in MS a unilateral, or bilateral condition?*

It is bilateral in almost all (>95%) cases

*MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?*

Yes, about 15% of intermediate-uveitis pts will develop MS

Two specific manifestations (ie, signs) of intermediate uveitis are classically associated with MS—which ones?

-- Vitritis, which tends to be mild

-- Periphlebitis

---Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina

---Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)

---Panuveitis: All three locations are equally involved

**Typical Optic Neuritis**
**MS conveys an increased risk of uveitis. How much?** MS pts are 10 times more likely to experience uveitis than are non-MS individuals!

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--- Anterior: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--- Intermediate: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--- Posterior: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--- Panuveitis: All three locations are equally involved

**Which form is most likely to occur in MS?** Intermediate uveitis

--- Typical Optic Neuritis

*Is intermediate uveitis in MS a unilateral, or bilateral condition?*

It is bilateral in almost all (>95%) cases

*MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?* Yes, about 15% of intermediate-uveitis pts will develop MS

--- Two specific **manifestations (ie, signs) of intermediate uveitis** are classically associated with MS—which ones?

-- Vitritis, which tends to be mild
--Periphlebitis

--- Speaking of dz severity: In general, does MS-associated intermediate uveitis tend to be milder, or more severe than the idiopathic version? Milder
**CDMS: Manifestations**

**Nonocular**: Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

**Ocular**

**MS conveys an increased risk of uveitis. How much?**

**MS pts are 10 times more likely to experience uveitis than are non-MS individuals!**

Taking a step back: There are four types of uveitis, based on the location of the inflammation. What are they? How is each defined?

--- **Anterior**: The primary location of inflammation is the anterior chamber and/or anterior vitreous
--- **Intermediate**: The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina
--- **Posterior**: The site of inflammation is the retina and/or choroid (the optic nerve head can be involved too)
--- **Panuveitis**: All three locations are equally involved

**Which form is most likely to occur in MS?**

Intermediate uveitis

Is intermediate uveitis in MS a unilateral or bilateral condition?

It is bilateral in almost all (>95%) cases

**MS is a risk factor for developing intermediate uveitis. Is the reverse true as well, ie, are individuals with intermediate uveitis at higher risk of developing MS?**

Yes, about 15% of intermediate-uveitis pts will develop MS

**Two specific manifestations (ie, signs) of intermediate uveitis** are classically associated with MS—which ones?

--- Vitritis, which tends to be mild
---Periphlebitis

Speaking of dz severity: In general, does MS-associated intermediate uveitis tend to be milder, or more severe than the idiopathic version?

Milder
The Neuro book divvies the nonocular S/S of MS into five groups—what are they?
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Motor

Sensory

Motor

Cerebellar

Sphincter

Mental

Optic neuritis S/S

Nystagmus/oscillations

Diplopia

Uveitis

The Neuro book divvies the nonocular S/S of MS into five groups—what are they?
What motor symptoms are commonly encountered in MS?
What motor symptoms are commonly encountered in MS? Weakness of the extremities or facial musculature can occur, as can hemi- or paraplegia.
CDMS: Manifestations

Nonocular
- Motor
- Cerebellar

Ocular
- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Sensory
- Mental
- Sphinctor

What sensory symptoms commonly occur in MS?
What sensory symptoms commonly occur in MS?
Paresthesias of the face and/or body
What sensory symptoms commonly occur in MS?

Paresthesias of the face and/or body (classic presentation: paresthesia of the trunk described as 'bandlike')

**CDMS: Manifestations**

- **Nonocular**
  - Motor
  - Cerebellar
  - Sensory
    - Mental
  - Sphincter

- **Ocular**
  - Optic neuritis S/S
  - Nystagmus/oscillations
  - Diplopia
  - Uveitis

**Typical Optic Neuritis**
**What sensory symptoms commonly occur in MS?**

Paresthesias of the face and/or body *(classic presentation: paresthesia of the trunk described as ‘bandlike’)*
What sensory symptoms commonly occur in MS? Paresthesias of the face and/or body (classic presentation: paresthesia of the trunk described as ‘bandlike’)

Speaking of sensory symptoms in MS: What is Lhermitte’s sign? Shock-like sensations precipitated by a movement
What sensory symptoms commonly occur in MS?
Paresthesias of the face and/or body (classic presentation: paresthesia of the trunk described as ‘bandlike’)

Speaking of sensory symptoms in MS: What is Lhermitte’s sign?
Shock-like sensations precipitated by neck flexion
Which sphincter are we talking about here?
CDMS: Manifestations

Nonocular
- Motor
- Cerebellar
- Sensory
- Mental

Ocular
- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Sphincter

Which sphincter are we talking about here?
The bladder sphincter
Which sphincter are we talking about here?
The bladder sphincter

What bladder sphincter-related symptoms occur in MS?
CDMS: Manifestations

Nonocular

- Motor
- Cerebellar
- Sensory
- Mental
- Sphincter

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis

Typical Optic Neuritis

*Which sphincter are we talking about here?*
The bladder sphincter

*What bladder sphincter-related symptoms occur in MS?*
Incontinence, frequency, and/or urgency
What mental *manifestations are commonly encountered in MS?*
What mental *manifestations are commonly encountered in MS?* Primarily emotional issues: Lability; depression; irritability
What sorts of cerebellar-related symptoms do MS pts experience?
What sorts of cerebellar-related symptoms do MS pts experience?
Ataxia, dysarthria, and intentional tremor (among others)
What lab test cinches a diagnosis of MS?
Typical optic neuritis

CDMS: Evaluation

Labs

Imaging

What lab test cinches a diagnosis of MS?
There ain’t none
Typical Optic Neuritis

CDMS: *Evaluation*

Labs

Imaging

*What lab test cinches a diagnosis of MS?*
There ain’t none

*What imaging finding is pathognomonic for MS?*
Typical Optic Neuritis

CDMS: *Evaluation*

Labs

Imaging

*What lab test cinches a diagnosis of MS?*
There ain’t none

*What imaging finding is pathognomonic for MS?*
Same as labs—no such thing exists
Typical Optic Neuritis

CDMS: *Evaluation*

What lab test cinches a diagnosis of MS?
There ain’t none

Remember, MS is a *clinical* diagnosis—labs and imaging are contributory, but of themselves cannot make it!

Same as labs—no such thing exists
OK, so what lab(s) are contributory vis a vis diagnosing MS?
OK, so what lab(s) are contributory vis a vis diagnosing MS?
The finding of **two words** in the **a body fluid**
OK, so what lab(s) are contributory vis a vis diagnosing MS?

The finding of oligoclonal bands in the CSF
OK, so what lab(s) are contributory vis a vis diagnosing MS?
The finding of oligoclonal bands in the CSF

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
OK, so what lab(s) are contributory vis a vis diagnosing MS?
The finding of oligoclonal bands in the CSF

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
IgG
OK, so what lab(s) are contributory vis a vis diagnosing MS? The finding of oligoclonal bands in the CSF

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
IgG

What proportion of CDMS pts manifest these CSF bands?
OK, so what lab(s) are contributory vis a vis diagnosing MS?
The finding of oligoclonal bands in the CSF

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
IgG

What proportion of CDMS pts manifest these CSF bands?
Over 90%
OK, so what lab(s) are contributory vis a vis diagnosing MS? The finding of oligoclonal bands in the CSF.

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
IgG

What proportion of CDMS pts manifest these CSF bands? Over 90%

Are similar bands found in the serum of CDMS pts?
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

OK, so what lab(s) are contributory vis a vis diagnosing MS?
The finding of oligoclonal bands in the CSF

Which immunoglobulin form (IgA, IgE, IgG, etc) do the bands take?
IgG

What proportion of CDMS pts manifest these CSF bands?
Over 90%

Are similar bands found in the serum of CDMS pts?
No
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI? MRI
Typical Optic Neuritis

CDMS: Evaluation

Imaging

Labs

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

*Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?*

MRI

*In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?*

FLAIR
Typical Optic Neuritis

CDMS: *Evaluation*

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?

**FLAIR**

What does FLAIR stand for in this context?
**Typical Optic Neuritis**

**CDMS:** Evaluation

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?

MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?

**FLAIR**

What does FLAIR stand for in this context?

Fluid-attenuated inversion recovery
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI? MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it? FLAIR

Do MS lesions enhance with gadolinium?
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/wo gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Do MS lesions enhance with gadolinium?
Yes
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series ($T1; T2; w/w/o$ gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
uni- vs multifocal; white- vs gray matter lesions
Typical Optic Neuritis

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white–matter lesions
Typical Optic Neuritis

CDMS: *Evaluation*

Labs

Imaging

*Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?*

MRI

*In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?*

FLAIR

*Other than optic-nerve changes, what imaging findings are typically encountered in MS?*

Multifocal white-matter lesions, usually in shape
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?

MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?

FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?

Multifocal white–matter lesions, usually ovoid in shape
Typical Optic Neuritis

CDMS: **Evaluation**

Labs

Imaging

*Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?*

MRI

*In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?*

FLAIR

*Other than optic-nerve changes, what imaging findings are typically encountered in MS?*

Multifocal white-matter lesions, usually ovoid in shape and in location
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white-matter lesions, usually ovoid in shape and periventricular in location
MRI of a patient with multiple sclerosis (MS) shows demyelinating plaques. A, T1-weighted, postgadolinium MRI scan demonstrates enhancing white matter lesions bilaterally, as well as “black holes” (arrows). B, T2-weighted MRI scan shows periventricular, multifocal, hyperintense white matter lesions consistent with demyelination. C, FLAIR scan confirms periventricular ovoid white matter lesions.
Typical Optic Neuritis

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white-matter lesions, usually ovoid in shape and periventricular in location

What is the eponymous name for these ovoid lesions?
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white–matter lesions, usually ovoid in shape and periventricular in location

What is the eponymous name for these ovoid lesions?
Dawson’s fingers
Typical Optic Neuritis

MS: Dawson’s fingers
Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white-matter lesions, usually ovoid in shape and periventricular in location

What proportion of CDMS pts manifest these findings?
Typical Optic Neuritis

Which (if either) is the preferred imaging modality for detecting MS-associated abnormalities—CT, or MRI?
MRI

In addition to the usual series (T1; T2; w/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Other than optic-nerve changes, what imaging findings are typically encountered in MS?
Multifocal white-matter lesions, usually ovoid in shape and periventricular in location

What proportion of CDMS pts manifest these findings?
About 90%
Do steroids still have a role in managing MS?
Do steroids still have a role in managing MS?
Mos def—they are the go-to therapy for acute exacerbations
Do steroids still have a role in managing MS?
Mos def—they are the go-to therapy for acute exacerbations

Is the dose low, or high?
CDMS: Treatment

Steroids

Do steroids still have a role in managing MS?
Mos def—they are the go-to therapy for acute exacerbations

Is the dose low, or high?
High
Do steroids still have a role in managing MS?
Mos def—they are the go-to therapy for acute exacerbations

Is the dose low, or high? Is the preferred route PO, IM, or IV?
High.

Typical Optic Neuritis

CDMS: Treatment

Steroids

DMT
CDMS: Treatment

Steroids

Typical Optic Neuritis

Do steroids still have a role in managing MS?
Mos def—they are the go-to therapy for acute exacerbations

Is the dose low, or high? Is the preferred route PO, IM, or IV?
High. IV.
Typical Optic Neuritis

CDMS: Treatment

Steroids

DMT

What does DMT stand for in this context?
Typical Optic Neuritis

CDMS: Treatment

Steroids → DMT

What does DMT stand for in this context?
Disease-modifying therapy
What does DMT stand for in this context?
Disease-modifying therapy

There are three classes of DMTs—what are they?
--?
--?
--?
CDMS: *Treatment*

**Steroids**

**DMT**

*What does DMT stand for in this context?*

Disease-modifying therapy

*There are three classes of DMTs—what are they?*

-- Interferons
-- Monoclonal antibodies
-- Immunomodulators
What does DMT stand for in this context?
Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
**CDMS: Treatment**

What does DMT stand for in this context?
Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
Fingolimod
CDMS: **Treatment**

**What does DMT stand for in this context?**
Disease-modifying therapy

*There are three classes of DMTs—what are they?*
-- Interferons
-- Monoclonal antibodies
-- Immunomodulators

*One immunomodulator is notorious for causing macular edema— which one?*
Fingolimod

*By what name is fingolimod-associated macular edema known?*
What does DMT stand for in this context? Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
Fingolimod

By what name is fingolimod-associated macular edema known? It is called ‘fingolimod-associated macular edema’ (FAME)
What does DMT stand for in this context?
Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
Fingolimod

By what name is fingolimod-associated macular edema known?
It is called ‘fingolimod-associated macular edema’ (FAME)

What is the tx for FAME?
What does DMT stand for in this context?
Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
Fingolimod

By what name is fingolimod-associated macular edema known?
It is called ‘fingolimod-associated macular edema’ (FAME)

What is the tx for FAME?
Cessation of the medication
What does DMT stand for in this context? Disease-modifying therapy

There are three classes of DMTs—what are they?
-- Interferons
-- Monoclonal antibodies
-- Immunomodulators

One immunomodulator is notorious for causing macular edema— which one?
Fingolimod

By what name is fingolimod-associated macular edema known? It is called ‘fingolimod-associated macular edema’ (FAME)

What is the tx for FAME? Is it effective?
Cessation of the medication.
What does DMT stand for in this context? Disease-modifying therapy

There are three classes of DMTs—what are they?
--Interferons
--Monoclonal antibodies
--Immunomodulators

One immunomodulator is notorious for causing macular edema—which one?
Fingolimod

By what name is fingolimod-associated macular edema known?
It is called ‘fingolimod-associated macular edema’ (FAME)

What is the tx for FAME? Is it effective?
Cessation of the medication. Yes.
(This is a good point in the set to take a break)
Optic neuritis

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself:
Typical Optic Neuritis

*Does this pt have ‘clinically isolated’ Optic neuritis’?*

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…

*aka clinically isolated syndrome (CIS)*
Does this pt have ‘clinically isolated Optic neuritis’?

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?’

aka clinically isolated syndrome (CIS)

No question—proceed when ready
Does this pt have ‘clinically isolated Optic neuritis’?

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?'

But the Neuro book places great emphasis on asking two additional questions: 1) ‘Does this pt have clinically isolated optic neuritis, or...
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…does she have MS?’

But the *Neuro* book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or…does she have

   what *NMO* stands for

   (or

   what *NMOSD* stands for

   )?'
(or) NMOSD?

Typical Optic Neuritis

MS

Does she have NMO?

Does this pt have ‘clinically isolated Optic neuritis’?

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…does she have MS?'

But the Neuro book places great emphasis on asking two additional questions: 1) ‘Does this pt have clinically isolated optic neuritis, or…does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?'
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?"

But the *Neuro* book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or...does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?

2) ‘Does this pt have clinically isolated optic neuritis, or...does she have NMO or MOGAD?'

**Typical Optic Neuritis**

**MS**

*Does she have NMO?*

*Does she have MOGAD?*

*Does this pt have ‘clinically isolated Optic neuritis’?*
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…does she have MS?’

But the Neuro book places great emphasis on asking two additional questions:  
1) ‘Does this pt have clinically isolated optic neuritis, or…does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?'  
   **And:**  
2) ‘Does this pt have clinically isolated optic neuritis, or…does she have myelin oligodendrocyte glycoprotein IgG-associated disorder?’
Does this pt have ‘clinically isolated Optic neuritis’?

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?’

But, the Neuro book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or...does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?

2) ‘Does this pt have clinically isolated optic neuritis, or...does she have myelin oligodendrocyte glycoprotein IgG-associated disorder?’

Why the emphasis on these two questions/conditions?
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…does she have MS? The Neuro book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or…does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?

2) ‘Does this pt have clinically isolated optic neuritis, or…does she have myelin oligodendrocyte glycoprotein IgG-associated disorder (MOGAD)?

Why the emphasis on these two questions/conditions?

For several reasons related to dz management:
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or…does she have MS?"

The Neuro book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or…does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?

2) ‘Does this pt have clinically isolated optic neuritis, or…does she have myelin oligodendrocyte glycoprotein IgG-associated disorder?"

Why the emphasis on these two questions/conditions?
For several reasons related to dz management:
--Differences in pathophysiology means tx for NMO(SD), MOG and MS differ; and
--?
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?"

The Neuro book places great emphasis on asking two additional questions:

1) ‘Does this pt have clinically isolated optic neuritis, or...does she have neuromyelitis optica (or neuromyelitis optica spectrum disorder)?

2) ‘Does this pt have clinically isolated optic neuritis, or...does she have myelin oligodendrocyte glycoprotein IgG-associated disorder (MOGAD)?

Why the emphasis on these two questions/conditions?
For several reasons related to dz management:
--Differences in pathophysiology means tx for NMO(SD), MOG and MS differ; and
--Some MS txs are ineffective in one—and worse, are deleterious in the other.
As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself: ‘Does this pt have clinically isolated optic neuritis, or...does she have MS?’

**Typical Optic Neuritis**

Does this pt have ‘clinically isolated Optic neuritis’?

Does she have **NMO**?

Does she have **MOGAD**?

---

**the Neuro book places great emphasis on asking two additional questions**

**Why the emphasis on these two questions/conditions?**

For several reasons related to dz management:

---

1) Differences in pathophysiology means tx for NMO(SD), MOG and MS differ; and
2) Some MS txs are ineffective in MOG —and worse, are deleterious in NMO(SD)
By what eponymous name is NMO also known?
By what eponymous name is NMO also known?
Devic's dz
By what eponymous name is NMO also known? Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
By what eponymous name is NMO also known?
Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Longitudinally extensive transverse myelitis
By what eponymous name is NMO also known? Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis aka Devic’s dz

What is transverse myelitis? Inflammation of the spinal cord.

How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss.

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years.

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging.
Typical Optic Neuritis

By what eponymous name is NMO also known? Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis aka Devic’s dz

What is transverse myelitis? Inflammation of the spinal cord.

How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss.

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years.

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging.
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?

What is the other?
Longitudinally extensive transverse myelitis (NMO)

What is NMO also known as?
Devic's disease

What is the other inflammatory process involved in NMO?
Longitudinally extensive transverse myelitis
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriplegia, often with sensory loss
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

But MS can also present with paresis + sensory loss. How is this any different?

What is the other?
Longitudinally extensive...
Typical Optic Neuritis

NMO
aka Devic's dz

NMOSD

MS

MOGAD

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a **symmetric** para- or quadriplegia, often with sensory loss

But MS can also present with **paresis + sensory loss. How is this any different?**
The difference is the symmetry. Whereas NMO presents with bilaterally symmetric motor and/or sensory loss, **symmetric deficits are distinctly uncommon in MS.**
What is transverse myelitis? Inflammation of the spinal cord.

How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss.

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years.

What is the other? Longitudinally extensive transverse myelitis.
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within \( \text{unit of time} \) to \( \text{unit of time} \) of each other

What is the other?
Longitudinally extensive
What is transverse myelitis? Inflammation of the spinal cord

How does transverse myelitis present clinically? As a symmetric para- or quadripareisis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years.

What is transverse myelitis?
Longitudinally extensive transverse myelitis.
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriplegia, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years
**Typical Optic Neuritis**

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?

What is the other?
Longitudinally extensive...
Typical Optic Neuritis

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriplegia, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging

What is the other?
Longitudinally extensive

transverse myelitis
Sagittal T2-weighted MRI of the spinal cord in a patient with NMOSD depicting a hyperintense lesion
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging

Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions?
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging

Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions?
2-3 vertebral segments or so
Sagittal T2-weighted MRI of the spinal cord in a patient with NMOSD depicting a hyperintense lesion **over more than 3 vertebral segments**
Typical Optic Neuritis

By what eponymous name is NMO also known?
Devic's dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Longitudinally extensive transverse myelitis aka Devic's dz

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?
2-3 vertebral segments or so

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?

How posterior are we talking about here?
They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis)

What does chiasmal involvement portend vis a vis exam findings in NMO?
It raises the possibility that bitemporal and/or homonymous hemianopic VF defects might be found
Typical Optic Neuritis

What is transverse myelitis?

- *So, the spinal cord lesions in NMO are longitudinally extensive...Perchance, is the same true of the optic nerve lesions in NMO?*
- *Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.*

Longitudinally extensive **transverse myelitis**

How extensive (ie, long) are these lesions?

- 2-3 vertebral segments or so
Typical Optic Neuritis

What is transverse myelitis?
- So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?
- Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

What is the other?
- Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so
MRI findings in NMOSD-associated optic neuritis: Enhancement is *bilateral*, *extensive*, and *posterior*.

MRI findings in MS-associated optic neuritis: Enhancement is *unilateral*, *short*, and *anterior*.
What is transverse myelitis?

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?

Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

How posterior are we talking about here?

Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so
By what eponymous name is NMO also known?

Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Longitudinally extensive transverse myelitis aka Devic’s dz

What is transverse myelitis?

Inflammation of the spinal cord

How does transverse myelitis present clinically?

As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?

They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?

As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?

2-3 vertebral segments or so

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?

Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

How posterior are we talking about here?

They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis)

What is the other? Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so
Typical Optic Neuritis

What is transverse myelitis?

1. So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?
2. Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

How posterior are we talking about here?

They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis)

How extensive (ie, long) are these lesions?
2-3 vertebral segments or so
Thirteen-year-old girl presenting with bilateral visual loss due to NMO-associated optic neuritis. Axial FLAIR brain imaging showed optic chiasm involvement (white arrow).
Typical Optic Neuritis

NMO
aka Devic's dz

MS

NMOSD

MOGAD

What is transverse myelitis?

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?

Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

How posterior are we talking about here?
They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis)

What does chiasmal involvement portend vis a vis exam findings in NMO?

How extensive (ie, long) are these lesions?
2-3 vertebral segments or so
What is transverse myelitis?
- So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?
- Indeed it is! Further, in addition to longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.
- How posterior are we talking about here?
  - They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis)
- What does chiasmal involvement portend vis a vis exam findings in NMO?
  - It raises the possibility that bitemporal and/or homonymous hemianopic VF defects might be found.

How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so
By what eponymous name is NMO also known? Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis aka Devic’s dz

What is transverse myelitis? Inflammation of the spinal cord

How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so

What is the other? Longitudinally extensive transverse myelitis

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so

What does chiasmal involvement portend vis a vis exam findings in NMO? It raises the possibility that bitemporal and/or homonymous hemianopic VF defects might be found
Typical Optic Neuritis

What is transverse myelitis?

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?

Indeed it is! For NMO, the optic nerve lesions tend to be more posterior than those found in typical optic neuritis.

How posterior are we talking about here?

They can extend to the optic chiasm (which is almost unheard of in typical optic neuritis).

What does chiasmal involvement portend vis a vis exam findings in NMO?

It raises the possibility that bitemporal and/or homonymous hemianopic VF defects might be found.

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?

No, it tends to be worse.

Does it exhibit spontaneous recovery a la typical optic neuritis?

It does not.

Is long-term visual prognosis good a la typical optic neuritis?

It is not—in fact, it is common for at least one eye to end up with VA < 20/200.
By what eponymous name is NMO also known? Devic's dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis aka Devic's dz

Transverse myelitis? Inflammation of the spinal cord. How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss. How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years.

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging. How extensive (ie, long) are these lesions? 2-3 vertebral segments or so.

Longitudinally extensive transverse myelitis

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis? No it tends to be worse. Does it exhibit spontaneous recovery a la typical optic neuritis? It does not. Is long-term visual prognosis good a la typical optic neuritis? It is not—in fact, it is common for at least one eye to end up with VA < 20/200.
Typical Optic Neuritis

By what eponymous name is NMO also known?
Devic's dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Longitudinally extensive transverse myelitis aka Devic's dz

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?
2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?
No it tends to be worse

What is the Optic Chiasm?
What is transverse myelitis?

- Inflammation of the spinal cord

How does transverse myelitis present clinically?

- As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?

- They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?

- As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?

- 2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?

- No it tends to be worse

Does it exhibit spontaneous recovery a la typical optic neuritis?

- It does not

Is long-term visual prognosis good a la typical optic neuritis?

- It is not—in fact, it is common for at least one eye to end up with VA < 20/200
By what eponymous name is NMO also known?
Devic’s dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Longitudinally extensive transverse myelitis aka Devic’s dz

What is transverse myelitis?
Inflammation of the spinal cord
How does transverse myelitis present clinically?
As a symmetric para- or quadriparesis, often with sensory loss
How are the optic neuritis and transverse myelitis episodes related temporally?
They usually occur within weeks to months of each other, but can be separated by several years
How does transverse myelitis manifest on MRI?
As a hyperintense signal on T2 imaging
How extensive (ie, long) are these lesions?
2-3 vertebral segments or so

So, the spinal cord lesions in NMO are longitudinally extensive…Perchance, is the same true of the optic nerve lesions in NMO?
Indeed it is! Further, in addition to being longer, the optic nerve lesions in NMO tend to be more posterior than those found in typical optic neuritis.

What is the Optic Chiasm? What does chiasmal involvement portend vis a vis exam findings in NMO?
It raises the possibility that bitemporal and/or homonymous hemianopic VF defects might be found

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?
No it tends to be worse

Does it exhibit spontaneous recovery a la typical optic neuritis?
It does not

Longitudinally extensive transverse myelitis
By what eponymous name is NMO also known? Devic's dz

NMO involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis aka Devic's dz

What is transverse myelitis? Inflammation of the spinal cord

How does transverse myelitis present clinically? As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally? They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis? No it tends to be worse

Does it exhibit spontaneous recovery a la typical optic neuritis? It does not

Is long-term visual prognosis good a la typical optic neuritis? It is not—in fact, it is common for at least one eye to end up with VA < 20/200
Typical Optic Neuritis

What is transverse myelitis?
- Inflammation of the spinal cord
How does transverse myelitis present clinically?
- As a symmetric para- or quadriparesis, often with sensory loss
How are the optic neuritis and transverse myelitis episodes related temporally?
- They usually occur within weeks to months of each other, but can be separated by several years
How does transverse myelitis manifest on MRI?
- As a hyperintense signal on T2 imaging
How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so

How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?
- No it tends to be worse

Does it exhibit spontaneous recovery a la typical optic neuritis?
- It does not

Is long-term visual prognosis good a la typical optic neuritis?
- It is not

Longitudinally extensive transverse myelitis

What is the Optic Neuritis spectrum disorder (ONSD)?
- Also known as NMO, Devic’s disease

NMOSD

NMO
aka Devic’s dz

MS

MOGAD
**Typical Optic Neuritis**

What is transverse myelitis?
- Inflammation of the spinal cord

How does transverse myelitis present clinically?
- As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
- They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
- As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side *à la* typical optic neuritis?
- No it tends to be worse

Does it exhibit spontaneous recovery *à la* typical optic neuritis?
- It does not

Is long-term visual prognosis good *à la* typical optic neuritis?
- It is not—in fact, it is common for at least one eye to end up with VA < Snellen VA
**Typical Optic Neuritis**

What is transverse myelitis?
- Inflammation of the spinal cord

How does transverse myelitis present clinically?
- As a symmetric para- or quadriparesis, often with sensory loss

How are the optic neuritis and transverse myelitis episodes related temporally?
- They usually occur within weeks to months of each other, but can be separated by several years

How does transverse myelitis manifest on MRI?
- As a hyperintense signal on T2 imaging

How extensive (ie, long) are these lesions?
- 2-3 vertebral segments or so

Speaking of VA loss in NMO(SD)—does it tend to be on the mild-to-moderate side a la typical optic neuritis?
- No it tends to be worse

Does it exhibit spontaneous recovery a la typical optic neuritis?
- It does not

Is long-term visual prognosis good a la typical optic neuritis?
- It is not—in fact, it is common for at least one eye to end up with VA < 20/200
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein AQP4 (also known as aquaporin-4) is the target of antibodies in NMO.
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4)
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4)

What does this protein do?
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in CNS cell type cells
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in astroglial cells
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
The protein aquaporin-4 (AQP4)

**What does this protein do?**
It is the main water channel protein in astroglial cells

*In the present context, what do astrocytes do?*
**NMO** is an antibody-mediated autoimmune condition. **What is the target of the antibodies?**
The protein aquaporin-4 (AQP4)

**What does this protein do?**
It is the main water channel protein in astroglial cells

**In the present context, what do astrocytes do?**
They maintain oligodendrocyte viability—so, astrocyte loss ➞ oligodendrocyte loss
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
They maintain oligodendrocyte viability—so, astrocyte loss → oligodendrocyte loss

What do oligodendrocytes do?
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
They maintain oligodendrocyte viability—so, astrocyte loss \( \rightarrow \) oligodendrocyte loss

What do oligodendrocytes do?
They provide myelin in the CNS—oligodendrocyte loss = demyelination
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
They maintain oligodendrocyte viability—so, astrocyte loss $\rightarrow$ oligodendrocyte loss

What do oligodendrocytes do?
They provide myelin in the CNS—oligodendrocyte loss $\rightarrow$ demyelination. Thus, loss of AQP4 channels $\rightarrow$ astrocyte loss $\rightarrow$ demyelination.
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4)

What does this protein do?
It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
The maintain oligodendrocyte viability—so, astrocyte loss → oligodendrocyte loss

What do oligodendrocytes do?
They provide myelin in the CNS—oligodendrocyte loss = demyelination. Thus, loss of AQP4 channels → astrocyte loss → demyelination.
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies?
The protein **aquaporin-4 (AQP4)**

**Is lab testing available to detect antibodies against AQP4?**
Yes, and they form part of the diagnostic criteria for NMO.

It is the main water channel protein in astroglial cells.

**In the present context, what do astrocytes do?**
They maintain oligodendrocyte viability—so, astrocyte loss \(\rightarrow\) oligodendrocyte loss.

**What do oligodendrocytes do?**
They provide myelin in the CNS—oligodendrocyte loss = demyelination. Thus, loss of AQP4 channels \(\rightarrow\) astrocyte loss \(\rightarrow\) demyelination.
NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein **aquaporin-4 (AQP4)**

*Is lab testing available to detect antibodies against AQP4? Yes, and they form part of the diagnostic criteria for NMO*

It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
They maintain oligodendrocyte viability—so, astrocyte loss → oligodendrocyte loss

What do oligodendrocytes do?
They provide myelin in the CNS—oligodendrocyte loss = demyelination. Thus, loss of AQP4 channels → astrocyte loss → demyelination.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

*What is the area postrema?*
**NMOSD**

+ area postrema syndrome

**NMO**

aka Devic’s dz

+ transverse myelitis

**Optic neuritis**

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

*What is the* area postrema?

A portion of the posterior medulla
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:
--?
--?
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:
--Hiccups
--Nausea/vomiting
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome

What is the area postrema? A portion of the posterior medulla

How does area postrema syndrome present clinically? With intractable episodes of hiccups and/or nausea/vomiting:

- Hiccups
- Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’?
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following: hiccups or nausea/vomiting. How long do hiccups have to last to be considered ‘intractable’?
At least 30 days or so.
Like NMO, does NMOSD involve antibodies against the AQP4 protein?

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:

--Hiccup
--Nausea/vomiting

How long do hiccup have to last to be considered ‘intractable’?
At least 30 days or so
Like NMO, does NMOSD involve antibodies against the AQP4 protein? Indeed it does.

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.

What is the area postrema? A portion of the posterior medulla.

How does area postrema syndrome present clinically? With intractable hiccups and vomiting. How long do hiccups have to last to be considered ‘intractable’? At least 30 days or so.
Like NMO, does NMOSD involve antibodies against the AQP4 protein? Indeed it does.

Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD?

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:

--Hiccups
--Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’?
At least 30 days or so
Like NMO, does NMOSD involve antibodies against the AQP4 protein? Indeed it does.

Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD? Indeed it is.

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.

What is the area postrema?
A portion of the posterior medulla.

How does area postrema syndrome present clinically?
With intractable: --Hiccups
--Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’?
At least 30 days or so.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?

Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:
-- Hiccups
-- Nausea/vomiting

How long do hiccups have to last to be considered 'intractable'?
At least 30 days or so

Like NMO, does NMOSD involve antibodies against the AQP4 protein? Indeed it does

Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD? Indeed it is

The AQP4 water channel membrane protein is found mainly in three locations—what are they?
-- ?
-- ?
-- ?
(In retrospect, this should not be surprising)

NMOSD is a syndrome that involves three separate and specific inflammatory processes: optic neuritis, longitudinally extensive transverse myelitis, and area postrema syndrome. It is also referred to as Devic's disease.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.

What is the area postrema?
A portion of the posterior medulla.

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:
--Hiccups
--Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’?
At least 30 days or so.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome

What is the area postrema? A portion of the posterior medulla

How does area postrema syndrome present clinically? With intractable episodes of one or both of the following:

- Hiccups
- Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’? At least 30 days or so

Like NMO, does NMOSD involve antibodies against the AQP4 protein? Indeed it does

The AQP4 water channel membrane protein is found mainly in the area postrema, spinal cord, and optic nerve. (In retrospect, this should not be surprising)

Is NMOSD a common cause of demyelinating diseases? Not in North America, but it accounts for about half the cases in Asia and the West Indies.
NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome.

What is the area postrema? A portion of the posterior medulla.

How does area postrema syndrome present clinically? With intractable episodes of one or both of the following:

--Hiccups
--Nausea/vomiting

How long do hiccups have to last to be considered 'intractable'? At least 30 days or so.
**Typical Optic Neuritis**

Optic neuritis

**NMOSD**

aka Devic’s dz

+ transverse myelitis

**Optic neuritis**

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome

What is the area postrema? A portion of the posterior medulla

How does area postrema syndrome present clinically?

With intractable episodes of one or both of the following:

--Hiccups

--Nausea/vomiting

How long do hiccups have to last to be considered ‘intractable’? At least 30 days or so

Like NMO, does NMOSD involve antibodies against the **AQP4 protein**? Indeed it does

The AQP4 water channel membrane protein is found mainly in three locations—what are they?

- Area postrema
- Spinal cord
- Optic nerve

(In retrospect, this should not be surprising)

Like NMO, does NMOSD involve antibodies against the **AQP4 protein**? Indeed it does

Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD? Indeed it is

The AQP4 water channel membrane protein is found mainly in three locations—what are they?

- Area postrema
- Spinal cord
- Optic nerve

(In retrospect, this should not be surprising)

Is NMOSD a common cause of demyelinating dz? Not in North America, but it accounts for about half the cases in Asia and the West Indies.
**NMOSD**

Is NMOSD a common cause of demyelinating dz?
Not in North America, but it accounts for about half the cases in Asia

**NMO**
aka Devic's dz

+ transverse myelitis

**Optic neuritis**

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:

--- Hiccups
--- Nausea/vomiting

How long do hiccups have to last to be considered 'intractable'? At least 30 days or so

Like NMO, does NMOSD involve antibodies against the AQP4 protein?
Indeed it does

The AQP4 water channel membrane protein is found mainly in three locations—what are they?
- Area postrema
- Spinal cord
- Optic nerve
(In retrospect, this should not be surprising)

Like NMO, does NMOSD involve antibodies against the AQP4 protein?
Indeed it does

Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD?
Indeed it is

The AQP4 water channel membrane protein is found mainly in three locations—what are they?
- Area postrema
- Spinal cord
- Optic nerve
(In retrospect, this should not be surprising)

+ area postrema syndrome

Is NMOSD a common cause of demyelinating dz?
Not in North America, but it accounts for about half the cases in Asia

NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third? Area postrema syndrome
**NMOSD**

**Is NMOSD a common cause of demyelinating dz?**
Not in North America, but it accounts for about half the cases in Asia and the West Indies.

**NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?**
Area postrema syndrome

**What is the area postrema?**
A portion of the posterior medulla

**How does area postrema syndrome present clinically?**
With intractable episodes of one or both of the following:
- Hiccups
- Nausea/vomiting

**How long do hiccups have to last to be considered ‘intractable’?**
At least 30 days or so

**Like NMO, does NMOSD involve antibodies against the AQP4 protein?**
Indeed it does

**The AQP4 water channel membrane protein is found mainly in three locations—what are they?**
- Area postrema
- Spinal cord
- Optic nerve

(In retrospect, this should not be surprising)

**Also as with NMO, is Ab positivity a diagnostic criteria for NMOSD?**
Indeed it is

**The AQP4 water channel membrane protein is found mainly in three locations—what are they?**
- Area postrema
- Spinal cord
- Optic nerve

(In retrospect, this should not be surprising)
NMOSD

**Typical Optic Neuritis**

Optic neuritis gifts you with two symptoms:

- Transverse myelitis
- Area postrema syndrome

**Area postrema**

A portion of the posterior medulla

How does area postrema syndrome present clinically?

With intractable episodes of one or both of the following:

- Hiccups
- Nausea/vomiting

How long do hiccups have to last to be considered "intractable"?

At least 30 days or so

Like NMO, does NMOSD involve antibodies against the AQP4 protein?

Indeed it does

The AQP4 water channel membrane protein is found mainly in these locations—what are they?

- Postreema
- Spinal cord
- Optic nerve

(Area postrema syndrome isn’t too surprising)

Is NMOSD a common cause of demyelinating dz?

Not in North America, but it accounts for about half the cases in Asia and the West Indies
How are NMO and NMOSD treated?
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids aka Devic’s dz.
Typical Optic Neuritis

How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—dose, and duration
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, and/or should be considered.
Typical Optic Neuritis

**NMOSD**
- + area postrema syndrome

**NMO**
- aka Devic’s dz
- + transverse myelitis

How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, plasma exchange and/or IVIG should be considered.
Typical Optic Neuritis

**NMOSD**

+ area postrema syndrome

**NMO**

aka Devic’s dz

+ transverse myelitis

**Optic neuritis**

How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, plasma exchange and/or IVIG should be considered.

What class of medicine has been shown to reduce the risk of recurrence?
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, plasma exchange and/or IVIG should be considered.

What class of medicine has been shown to reduce the risk of recurrence?
Immunosuppressives
How are NMO and NMOSD treated?
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, plasma exchange and/or IVIG should be considered.

What class of medicine has been shown to reduce the risk of recurrence? Immunosuppressives

What happens if an NMO(SD) pt is misdiagnosed as having MS and is started on DMT?
**Typical Optic Neuritis**

**NMOSD**
- + area postrema syndrome

**NMO**
- aka Devic’s dz
- + transverse myelitis

**MS**

**MOGAD**

**Optic neuritis**

**How are NMO and NMOSD treated?**
Acute exacerbations are treated with steroids—1 g/d for 3-5 days. If ineffective, plasma exchange and/or IVIG should be considered.

**What class of medicine has been shown to reduce the risk of recurrence?**
Immunosuppressives

**What happens if an NMO(SD) pt is misdiagnosed as having MS and is started on DMT?**
These meds will increase the risk of recurrence.
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)
Typical Optic Neuritis

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM?

In a nutshell, what is ADEM?

An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?

Children

Is there a gender predilection?

Yes, it is more common in males

There is a geographic predilection—what is it?

It is more prevalent among people who live farther from the equator
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord
NMOSD + area postrema syndrome

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM? An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Typical Optic Neuritis

MS

+ area postrema syndrome

NMOSD

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator.
NMOSD + area postrema syndrome

Typical Optic Neuritis

MS

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM).

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord.

Is it more common in children, or adults?
Children.

Is there a gender predilection?
Yes, it is more common in M v F.
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males
464

Typical Optic Neuritis

NMOSD

MS

+ area postrema syndrome

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

NMO

MOGAD

Devic’s dz or adults?
Is it more commonaka
in children,

Children

+ transverse myelitis

Is there a gender predilection?
Yes, it is more common in males

+ ADEM

Optic neuritis

There is a geographic predilection—what is it?
is more prevalent
amongand
people
who live
farther fromprocesses.
the equator One is optic neuritis.
MOG Itinvolves
two separate
specific
inflammatory

What is the other?
Acute disseminated encephalomyelitis (ADEM)


MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live closer to the equator
In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator
Typical Optic Neuritis

In a nutshell, what is ADEM?
An acute **autoimmune demyelinating condition affecting the brain and/or spinal cord**

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
**It is more prevalent among people who live farther from the equator**

Like MS

NMOSD

+ area postrema syndrome

MoG

What is the other?
Acute disseminated encephalomyelitis (ADEM)

MS

Like MS
NMOSD

+ area postrema syndrome

MS

Typical Optic Neuritis

What is the other?
Acute disseminated encephalomyelitis (ADEM)

**In a nutshell, what is ADEM?**
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

Not like MS
**Typical Optic Neuritis**

**NMOSD**
+ area postrema syndrome

**MS**

**In a nutshell, what is ADEM?**
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

**Is it more common in children, or adults?**
Children

**Is there a gender predilection?**
Yes, it is more common in males

**There is a geographic predilection—what is it?**
It is more prevalent among people who live farther from the equator

**MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?**
Acute disseminated encephalomyelitis (ADEM)

**How does it present clinically?**
In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

How does it present clinically?
With multifocal neurologic deficits in concert with encephalopathic signs/symptoms
Typical Optic Neuritis

MoG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM) aka Devic's dz.

In a nutshell, what is ADEM? An acute autoimmune demyelinating condition affecting the brain and/or spinal cord.

Is it more common in children, or adults? Children.

Is there a gender predilection? Yes, it is more common in males.

There is a geographic predilection—what is it? It is more prevalent among people who live farther from the equator.

How does it present clinically? With multifocal neurologic deficits.

What are the more common neurologic deficits? --?

--?
NMOSD
+ area postrema syndrome

Typical Optic Neuritis

MS

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

MOG involves two separate and specific inflammatory processes. One is optic neuritis.
What is the other?
Acute disseminated encephalomyelitis (ADEM)

How does it present clinically?
With multifocal neurologic deficits

What are the more common neurologic deficits?
--Extremity weakness
--Ataxia
Typical Optic Neuritis

NMOSD

+ area postrema syndrome

MS

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

What is the other?
Acute disseminated encephalomyelitis (ADEM)

What are the S/S of encephalopathy?
Encephalopathic signs/symptoms
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

What are the S/S of encephalopathy?
Stupor (or even frank coma); irritability; confusion

encephalopathic signs/symptoms
NMOSD
+ area postrema syndrome

Typical Optic Neuritis

MS

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord.

Is it more common in children, or adults?
Children.

Is there a gender predilection?
Yes, it is more common in males.

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator.

What is the other?
Acute disseminated encephalomyelitis (ADEM).

How does it present clinically?
With multifocal neurologic deficits in concert with encephalopathic signs/symptoms.

How does it present radiologically?
NMOSD + area postrema syndrome

MS

Typical Optic Neuritis

In a nutshell, what is ADEM?
An acute autoimmune demyelinating condition affecting the brain and/or spinal cord

Is it more common in children, or adults?
Children

Is there a gender predilection?
Yes, it is more common in males

There is a geographic predilection—what is it?
It is more prevalent among people who live farther from the equator

What is the other?
Acute disseminated encephalomyelitis (ADEM)

How does it present clinically?
With multifocal neurologic deficits in concert with encephalopathic signs/symptoms

How does it present radiologically?
With large, bilateral, diffuse lesions involving both gray and white matter structures including the brainstem
(A) **ADEM.** Axial FLAIR showing bilateral, globular, hyperintense lesions in cortical gray matter (among other locations)
(A) **ADEM.** Axial FLAIR showing bilateral, globular, hyperintense lesions in cortical gray matter (among other locations). (B) For comparison, FLAIR showing Dawson’s fingers typical of MS.
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?
Optic neuritis

The ONH in MOG-associated optic neuritis—is it normal-to-mildly edematous, as is typical in typical optic neuritis?
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

The ONH in MOG-associated optic neuritis—*is it normal-to-mildly edematous, as is typical in typical optic neuritis*? No, it tends to be much worse
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically?
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically? Unlike the location, and tissue type (color) lesions of MS, MOG presents with tissue type (color) lesions
**MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?**

Acute disseminated encephalomyelitis (ADEM)

**What is the most common presenting sign of MOG?**
Optic neuritis

**How does MOG present radiologically?**
Unlike the periventricular white-matter lesions of MS, MOG presents with **gray**-matter lesions.
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically? Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically? Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?
Optic neuritis

How does MOG present radiologically?
Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem. However, it has no pathognomonic radiographic features, and often cannot be differentiated from ADEM.
Typical Optic Neuritis

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically? Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)? Severe

Indeed it does

How about long-term visual prognosis: good, or nah? Good
Typical Optic Neuritis

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG? Optic neuritis

How does MOG present radiologically? Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)? Severe

How does MOG present radiologically? Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?

Optic neuritis

How does MOG present radiologically?

Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)?

Severe

Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?

Good
Typical Optic Neuritis

NMOSD
+ area postrema syndrome

MS

NMO
aka Devic’s dz

MOGAD

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)?
Severe

Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?
Spontaneous recovery is the rule

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?
Optic neuritis

How does MOG present radiologically?
Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem

Good
MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?

Acute disseminated encephalomyelitis (ADEM)

What is the most common presenting sign of MOG?

Optic neuritis

How does MOG present radiologically?

Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)?

Severe

Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?

Spontaneous recovery is the rule

How about long-term visual prognosis: good, or nah?

Good
Typical Optic Neuritis

NMOSD
+ area postrema syndrome

MOG aka Devic's dz

MOGAD

Regarding VA loss in MOG—does it tend to be on the mild-to-moderate side a la typical optic neuritis, or severe as in NMO(SD)?
Severe

Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?
Spontaneous recovery is the rule

How about long-term visual prognosis: good, or nah?
Good

How does MOG present radiologically?
Unlike the periventricular white-matter lesions of MS, MOG presents with gray-matter lesions, as well as diffuse lesions involving the brainstem.
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies?
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies?
It’s all there in the name—a glycoprotein on myelin oligodendrocytes
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It’s all there in the name—a glycoprotein on myelin oligodendrocytes.

What does this protein do?
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It’s all there in the name—a glycoprotein on myelin oligodendrocytes.

What does this protein do?
At the time of this writing, this has yet to be elucidated. But whatever the protein does, it is mission-critical to maintaining oligodendrocyte viability.
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It’s all there in the name—a glycoprotein on myelin oligodendrocytes.

What does this protein do? At the time of this writing, this has yet to be elucidated. But whatever the protein does, it is mission-critical to maintaining oligodendrocyte viability, because like NMO (and typical optic neuritis), MOG is a demyelinating dz.
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It’s all there in the name—a glycoprotein on myelin oligodendrocytes.

What does this protein do? At the time of this writing, this has yet to be elucidated. But whatever the protein does, it is mission-critical to maintaining oligodendrocyte viability, because like NMO (and typical optic neuritis), MOG is a demyelinating dz.
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It is all the way in the name—the glycoprotein on myelin oligodendrocytes.

Is lab testing available to detect these antibodies? Yes, and they form part of the diagnostic criteria for MOG.

What does this protein do? At the time of this writing, this has yet to be elucidated. But whatever the protein does, it is mission-critical to maintaining oligodendrocyte viability, because like NMO (and typical optic neuritis), MOG is a demyelinating dz.
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies? It's all there in the name—a glycoprotein on myelin oligodendrocytes. What does this protein do? At the time of this writing, this has yet to be elucidated. But whatever the protein does, it is mission-critical to maintaining oligodendrocyte viability, because like NMO (and typical optic neuritis), MOG is a demyelinating dz.

**Is lab testing available to detect these antibodies?** Yes, and they form part of the diagnostic criteria for MOG.
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--?
--?
--?
--?
--?
--?
--?
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

- Bilateral presentation
- Laterality
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

-- Bilateral presentation
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

- Bilateral presentation
- Severe vision loss
- ?
- ?
- ?
- ?
- ?
- ?
- ?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

-- Bilateral presentation
-- Severe vision loss
-- ?
-- ?
-- ?
-- ?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Chronicity episodes
To recap: The following findings push you *away* from typical optic neuritis/MS and *toward* MOG or NMO(SD), but do not help differentiate between the two:

-- Bilateral presentation
-- Severe vision loss
-- Recurrent episodes
-- ?
-- ?
-- ?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--?
--?
--?

two words

ON enhancement
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--?
--?
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of location and tissue type lesions on MRI
--?
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--- Bilateral presentation
--- Severe vision loss
--- Recurrent episodes
--- Longitudinally extensive ON enhancement
--- A lack of periventricular white matter lesions on MRI
--- ?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
These findings push you **away** from MOG and **towards** NMO(SD): 
--MRI brain 
--? 
--? 
--? 
--? 
--?

To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two: 
--Bilateral presentation 
--Severe vision loss 
--Recurrent episodes 
--Longitudinally extensive ON enhancement 
--A lack of periventricular white matter lesions on MRI 
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you **away** from MOG and **towards** NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--?
--?
--?
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--- Bilateral presentation
--- Severe vision loss
--- Recurrent episodes
--- Longitudinally extensive ON enhancement
--- A lack of periventricular white matter lesions on MRI
--- A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
Typical Optic Neuritis

To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--?
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

-- Bilateral presentation
-- Severe vision loss
-- Recurrent episodes
-- Longitudinally extensive ON enhancement
-- A lack of periventricular white matter lesions on MRI
-- A lack of oligoclonal bands in the CSF
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you **away** from MOG and **towards** NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

Warning: Don’t misinterpret the meaning of this list! If a listed characteristic is present, it greatly increases the likelihood of NMO(SD) over the other two entities. But if the characteristic is not present, this shouldn’t be taken to exclude NMO(SD).

--A lack of oligoclonal bands in the CSF

No question—proceed when ready
Typical Optic Neuritis

NMOSD

NMO (aka Devic’s dz)

These findings push you **away** from MOG and **towards** NMO(SD):

-- MRI brain unremarkable
-- No spontaneous VA recovery
-- No pain with eye movements
-- Hx transverse myelitis
-- Hx area postrema syndrome
-- Poor visual outcome

Warning: Don’t misinterpret the meaning of this list! If a listed characteristic is present, it greatly increases the likelihood of NMO(SD) over the other two entities. But if the characteristic is **not** present, this shouldn’t be taken to exclude NMO(SD). Consider pain with eye movements—a sizeable minority (~1/3) of NMO(SD) optic neuritis pts c/o such pain. It’s just that of the three, NMO(SD) is vastly more likely than the others to present w/o pain.

-- A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

- Bilateral presentation
- Severe vision loss
- Recurrent episodes
- Longitudinally extensive ON enhancement
- A lack of periventricular white matter lesions on MRI
- A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

- MRI brain unremarkable
- No spontaneous VA recovery
- No pain with eye movements
- Hx transverse myelitis
- Hx area postrema syndrome
- Poor visual outcome

Warning: Don’t misinterpret the meaning of this list! If a listed characteristic is present, it greatly increases the likelihood of NMO(SD) over the other two entities. But if the characteristic is not present, this shouldn’t be taken to exclude NMO(SD). Consider pain with eye movements—a sizeable minority (~ 1/3) of NMO(SD) optic neuritis pts c/o such pain. It’s just that of the three, NMO(SD) is vastly more likely than the others to present w/o pain.
These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

Warning: Don’t misinterpret the meaning of this list! If a listed characteristic is present, it greatly increases the likelihood of NMO(SD) over the other two entities. But if the characteristic is not present, this shouldn’t be taken to exclude NMO(SD). Consider pain with eye movements—a sizeable minority (~1/3) of NMO(SD) optic neuritis pts c/o such pain. It’s just that of the three, NMO(SD) is vastly more likely than the others to present w/o pain. So interpret the presence of a listed characteristic as strongly indicative of NMO(SD), but interpret the absence of one much more circumspectly in that regard. (The same caution will be true regarding the items on the MOG list you are about to encounter.)

--A lack of oligoclonal bands in the CSF

No question—proceed when ready
To recap: The following findings push you **away** from typical optic neuritis/MS and **toward** MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:
--MRI brain with gray-matter changes

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

Note: The text contains some abbreviations and acronyms which might require additional context for full comprehension.
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from NMO(SD) and towards MOG:
--MRI brain with gray-matter changes
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:

--MRI brain with gray-matter changes
--Perineural enhancement on MRI

--?
--?
--?
To recap: The following findings push you *away* from typical optic neuritis/MS and *toward* MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you *away* from MOG and *towards* NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you *away* from NMO(SD) and *towards* MOG:

--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx [abbrev.]
--?
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:
--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx ADEM
--?
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:

--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx ADEM
--Disc edema
--?
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:

--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx ADEM
--Severe disc edema
--?
Typical Optic Neuritis

To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:

--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:

--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx ADEM
--Severe disc edema
--Steroid responsive/dependence

These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome
To recap: The following findings push you away from typical optic neuritis/MS and toward MOG or NMO(SD), but do not help differentiate between the two:
--Bilateral presentation
--Severe vision loss
--Recurrent episodes
--Longitudinally extensive ON enhancement
--A lack of periventricular white matter lesions on MRI
--A lack of oligoclonal bands in the CSF

These findings push you away from MOG and towards NMO(SD):
--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
--Hx transverse myelitis
--Hx area postrema syndrome
--Poor visual outcome

These findings push you away from NMO(SD) and towards MOG:
--MRI brain with gray-matter changes
--Perineural enhancement on MRI
--Hx ADEM
--Severe disc edema
--Steroid responsive/dependence
Can typical optic neuritis present bilaterally? Yes. Can it be chronic? Yes. But you (speaking to errbody who isn’t a fellowship-trained neuro-oph) shouldn’t make that call, because such cases are zebras, if not unicorns.
(Warning: Soapbox speech ahead)
Can typical optic neuritis present bilaterally? Yes. Can it be chronic? Yes. But you (speaking to errbody who isn’t a fellowship-trained neuro-oph) shouldn’t make that call, because such cases are zebras, if not unicorns. So don’t select ‘bilateral typical optic neuritis’ or ‘chronic typical optic neuritis’ as answers on the OKAP or WQEs, don’t utter those words when taking the Boards, and most importantly, don’t write them on a pt’s chart until and unless Neuro-Oph has written them first.