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?
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?

Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Typical

Atypical

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

Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Noninflammatory

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?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Noninflammatory

Typical Optic Neuritis

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
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Noninflammatory

Typical (demyelinating)

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 dz process involves demyelination
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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%!
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Noninflammatory

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 process involves demyelination.
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%!

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

*Typical Optic Neuritis*
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

Noninflammatory

Typical Optic Neuritis

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

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Noninflammatory

Typical Optic Neuritis

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

Optic neuritis

Typical (demyelinating)

Atypical

Infectious

Immune

Name 3 infectious causes of atypical ON:
1) Lues (syphilis)
2) TB
3) Lyme

(There are many others, of course)
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) 
2) 
3) 

(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

(There are many others, of course)
Optic Neuropathy

Optic neuritis

- Typical (demyelinating)
  - Typical Optic Neuritis

- 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
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(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)

Why don’t we call it Wegener’s?

Because Dr. Wegener was a Nazi, and is suspected to have committed war crimes.
Typical Optic Neuritis

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Immune

Noninflammatory

Ischemic

Compressive

Toxic/nutritional

Congenital/hereditary

Traumatic

Typical Optic Neuritis
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…
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?
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A relative afferent pupillary defect (RAPD)
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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 two words
-- Abnormal two diff words
-- Impaired two diff diff words
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.

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 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 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.

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

No question—proceed when ready
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**What functional abnormalities are likely to be found in a pt with an optic neuropathy?**

- Decreased central acuity
- Abnormal visual fields
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---

No question—proceed when ready.
Typical Optic Neuritis

**Vision in typical optic neuritis**

If it hasn’t started improving by one month, it likely isn’t typical optic neuritis.

---

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-- **Decreased central acuity**
-- Abnormal visual fields
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Optic Neuropathy

Typical Optic Neuritis

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- Decreased central acuity
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Optic Neuropathy

What is typical optic neuritis?
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What functional abnormalities are likely to be found in a pt with an optic neuropathy?
- Decreased central acuity
- Abnormal visual fields
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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

Typical
(demyelinating)

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?

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Typical Optic Neuritis
<table>
<thead>
<tr>
<th>Types of Optic Neuropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noninflammatory</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>
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</tr>
<tr>
<td>Infectious</td>
</tr>
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**What functional abnormalities are likely to be found in a pt with an optic neuropathy?**

- Decreased central acuity
- Abnormal visual fields
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**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.

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**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?
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...
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 central scotoma
Central scotoma 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
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Dyschromatopsia in typical optic neuritis: Is it red-green, or blue-yellow?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

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Red-green
**Typical Optic Neuritis**

**Optic Neuritis**

- Typical (demyelinating)
- Atypical
  - Infectious
  - Immune

**Noninflammatory**

- Ischemic
- Compressive
- Toxic/nutritional
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- 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?**
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?
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Pretty unremarkable—only \( \% \) 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.
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When edema is present, is it usually mild, or florid (i.e., severe with associated hemorrhages)?
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 (i.e., severe with associated hemorrhages)? Mild.
Is typical optic neuritis associated with ocular pain?
Optic Neuropathy

Optic neuritis

Typical (demyelinating) Atypical

Typical Optic Neuritis

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

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.
Typical Optic Neuritis

Optic Neuropathy

Optic neuritis

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

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Typical Optic Neuritis

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?
Optic Neuropathy

Optic neuritis

Typical (demyelinating)

Atypical

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

**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.

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).
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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
- even one white matter change was present: 3/4
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Optic Neuritis

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What was the name of the study that followed typical optic neuritis patients 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.
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Optic Neuropathy

Optic neuritis

Noninflammatory
- Ischemic
- Compressive
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Typical optic neuritis (demylinating)

Atypical optic neuritis

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

...if even one white matter change was present:
Typical Optic Neuropathy

Optic neuritis

Typical

Atypical

Noninflammatory
- Ischemic
- Compressive
- Traumatic/nutritional/neonatal/hereditary

Optic Neuritis

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

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?
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To look for white-matter changes, the presence of which increases the likelihood of developing multiple sclerosis (MS)
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If a typical pt presents with what seems to be a typical case of typical optic neuritis, what sort of workup should be done?
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Because it influences decision-making vis a vis whether to initiate tx that can forestall MS onset (and may improve dz course).
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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?

- **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.)
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    - IV steroids delayed the onset of MS in pts who had 2+ white-matter lesions at presentation.
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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?

Like, 1000 mg a day

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?

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)

[gender]
[age]
Optic Neuropathy

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

Atypical
- Infectious
- Immune
- 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)

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

Female
Young adult
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)

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

How does a case of typical optic neuritis typically present?

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

Laterality
Amount of time
Amount of time

Optic Neuropathy

Noninflammatory

Ischemic
Compressive
Toxic/nutritional
Congenital/hereditary

Typical (demyelinating)

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)

How does a case of typical optic neuritis typically present?

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

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

Optic Neuropathy
Optic Neuritis
Noninflammatory
Ischemic
Compressive
Toxic/nutritional
Congenital/hereditary

Typical (demyelinating)
Atypical

347x385
347x385
513x355
513x355
42x163
42x163
If the pt deviates from the typical pattern...

Optic Neuropathy

Optic Neuritis

Noninflammatory

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)

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

- Female
- Young 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

No question—proceed when ready
Optic Neuropathy

Optic neuritis
- Noninflammatory
- 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).

Typical Optic Neuritis

Typical vs. Atypical Presentation
- 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 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.

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

No question—proceed when ready
If the pt or the presentation deviates from the typical pattern...
You should question the dx of typical (demyelinating) optic neuritis

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? That's it.

MRI brain and orbits, with contrast.

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?

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

Typical case

Atypical

Compressive

Toxic/nutritional
Congenital/hereditary
Traumatic

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

How does a case of typical optic neuritis typically present?
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

Optic neuritis

Typical

(demyelinating)

Atypical

Infectious

Immune

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

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.
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) 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?
Syphilis
Bartonella
Lyme testing (if endemic)
Sarcoid
SLE

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

Compressive
- Toxically/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.

Typical Optic Neuritis

Optic neuritis

Typical (demyelinating) but

Not idiopathic or MS-related

Infectious

Immune

Atypical

Neuropathy

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: ?
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.

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
  - Not idiopathic or MS-related
  - Infectious
  - Immune

Atypical

(Cont)
- Compressive
  - Toxic/nutritional
  - Congenital/hereditary
  - Traumatic
- Syphilis: Serum and CSF RPR/TPPA
- Bartonella
- Lyme testing (if endemic)
- Sarcoid
- SLE
- 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.

Neuropathy

Optic neuritis

Typical (demyelinating but)

- Not idiopathic or MS-related

Atypical

Infectious

- Infectious

Immune

- Immune

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

Compromise
- Toxins/nutritional
- Congenital/hereditary
- Traumatic

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

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

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)
Sarcoid
SLE

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

(Cont)
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

- 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): ?
- Sarcoid
- SLE

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)
- 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

Typical (demyelinating) 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

Compressive
– Toxic/nutritional
– Congenital/hereditary
– Traumatic

Meningeal process

NMO(SD)

MOGAD

What etiologies? What studies?

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

(Cont)
Granulomatosis w/ polyangiitis
LHON
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

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: ?
SLE

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

Atypical

Typical (demyelinating but not idiopathic or MS-related)
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

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

Typical (demyelinating but)

- 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

(Cont)
- 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.

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

Typical (demyelinating but Not idiopathic or MS-related)

Atypical

Immune

Infectious

Neuropathy

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 neuritis
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

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.

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

Atypical

Compressive
- Toxic/nutritional
- Congenital/hereditary
- Traumatic

Typical (demyelinating but not idiopathic or MS-related)

Infectious

Immunee

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
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 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**: Chest XR or CT; +/- Gallium/PET
- **SLE**: ESR, ANA, Anti-DNA

**(Cont)**
- **Granulomatosis w/ polyangiitis**: ?
- **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

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
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
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

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

Typical Optic Neuritis

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
LNHON: Genetic testing
Meningeal process
NMO(SD)
MOGAD

Infectious

Immune

Atypical

Not idiopathic or MS-related
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**

- Optic neuritis
  - Typical
    - 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
    - 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: 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
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)
- MOGAD

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)
  - Not idiopathic or MS-related
- Atypical
  - Infectious
- Immune
  - Compressive
    - Ischemic
    - 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.
Optic Neuropathy

Optic neuritis

Typical
(demyelinating)
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

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

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.
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

**Atypical**

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

**Optic neuritis**

- **Typical (demyelinating but 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): 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.

### Optic neuritis

- **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**: 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
Optic Neuropathy

- Optic neuritis
  - Atypical
    - Infectious
    - Immune
  - Typical (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
- Granulomatosis w/ polyangiitis: ANCA
- LHON: Genetic testing
- Meningeal 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:

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

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

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
(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?
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
CDMS: *Basics*

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

*In a nutshell, what is MS?*
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*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
CDMS: Basics

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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)
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Is there an age predilection?
Yes, it is more common in life stage
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Yes, it is more common in young adults
CDMS: *Basics*

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

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Yes, it is more common in Whites
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Yes, it is more common in Whites

There is a geographic predilection—what is it?
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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

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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?
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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?
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

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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’
CDMS: Manifestations

Typical Optic Neuritis

Nonocular

Ocular

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

Nonocular

Ocular

- Optic neuritis S/S
- ?
- ?
- ?

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

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

*In a nutshell, what is a nystagmus and/or oscillation?*
**CDMS: Manifestations**

**Nonocular**

*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.

**Ocular**

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

**Typical Optic Neuritis**
CDMS: Manifestations

Nonocular

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?

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.
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.

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Typical Optic Neuritis

CDMS: Manifestations

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  - 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.

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?
In a nutshell, what is a nystagmus and/or oscillation?
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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).
CDMS: **Manifestations**

Typical Optic Neuritis

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

CDMS: **Manifestations**

- **Typical Optic Neuritis**
  - Nonocular
  - Ocular
    - Optic neuritis S/S
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    - 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
Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

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

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
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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?
--?
--?
--?
--?
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
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?**
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
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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.
**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

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.
CDMS: Manifestations

Nonocular

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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.

Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis
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

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- Optic neuritis S/S
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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.

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What does PSP stand for in this context? Progressive supranuclear palsy.
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- 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.

Typical Optic Neuritis

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

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

**Nonocular**

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

**Ocular**

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

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- The Supranuclear pathway

Broadly speaking, what constitutes the supranuclear pathways?

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

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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 three words.

Typical Optic Neuritis

CDMS: Manifestations

Nonocular

Ocular

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

CDMS: Manifestations

Nonocular

*Is diplopia a common manifestation of MS?*
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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?

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--- The **Internuclear pathway**

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

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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.

This is because most supranuclear-pathway lesions affect both eyes symmetrically.

Optic neuritis S/S
Nystagmus/oscillations
Diplopia
Uveitis

Typical Optic Neuritis

**Ocular**
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- The Internuclear 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.
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**CDMS: Manifestations**

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---

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

---

**Typical Optic Neuritis**
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
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Which two cranial nerve nuclei share an internuclear connection of well-established clinical importance? 3 and 6.
CDMS: *Manifestations*

Nonocular

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

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

-- The *Infranuclear* pathway

Ocular

*Optic neuritis S/S

Nystagmus/oscillations

Diplopia

Uveitis

Typical Optic Neuritis

**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?**
**CDMS: Manifestations**

- **Nonocular**
- **Ocular**

**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)?
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-- 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)
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

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

Ocular

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

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. What are they?

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

What are the two cranial nerve nuclei that 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)

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

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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 \textit{w}all-e\textit{b}ilateral INO). If you see a young person with a WEBINO, think MS first.
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

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-- The Supranuclear pathways
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Ocular

Optic neuritis S/S
Nystagmus/oscillations
Diplopia

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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.

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

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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
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

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

What constitutes the infranuclear pathway?

Typical Optic Neuritis

What constitutes the infranuclear pathway?

CDMS: 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 supranuclear pathways
- The internuclear pathway
- 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 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.
CDMS: Manifestations

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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.

Typical Optic Neuritis

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.

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

--The \textit{Infranuclear} pathway
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-- The Infranuclear pathway
Typical Optic Neuritis

Aqueduct

3rd nerve nucleus

Red nucleus

Cerebral peduncle

Third nerve fascicle

Cranial nerve fascicle
CDMS: Manifestations

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--The Infranuclear pathway
Typical Optic Neuritis

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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.

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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 still 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’.)

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--- The **Infranuclear pathway**
CDMS: Manifestations

Typical Optic Neuritis

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--- 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

- 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 Supranuclear pathways
- The Internuclear pathway
- 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 **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.

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

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 to the orbit to the neuromuscular junction; the junction itself; and finally the eye 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

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?
-- Millard-Gubler syndrome
-- Foville syndrome
-- Raymond 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.

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 (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

**Typical Optic Neuritis**

**CDMS: Manifestations**

*What term is used to describe conditions presenting with motility dysfunction 2ndry to fascicle damage + non-ocular CNS findings?*  
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--Weber syndrome  
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--Claude syndrome  
--Nothnagel syndrome

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

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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.

Typical Optic Neuritis

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--Foville syndrome
--Raymond syndrome

---

The cranial-nerve nuclei and their fascicles are located within the brainstem.
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*?
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

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--The Supranuclear pathways
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--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.
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
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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.
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**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.
CDMS: Manifestations

Nonocular

Ocular

Typical Optic Neuritis

MS conveys an increased risk of uveitis. How much?

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

Nonocular

MS conveys an increased risk of uveitis. How much? MS pts are # times more likely to experience uveitis than are non-MS individuals!

Ocular

- Optic neuritis S/S
- Nystagmus/oscillations
- Diplopia
- Uveitis
**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!"
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?

Ocular

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

Nonocular

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What proportion of MS pts will develop uveitis at some point?
About 1/3

Ocular

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

Typical Optic Neuritis
CDMS: Manifestations

Nonocular

**Typical Optic Neuritis**

Ocular

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

*MS conveys an increased risk of uveitis. How much?*

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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?*

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*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**
**Typical Optic Neuritis**

**CDMS: Manifestations**

**Nonocular**

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*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 \( \text{amount of time} \) 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?
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MS has several HLA associations, one of which conveys a higher risk of developing uveitis. Which one?
**CDMS: Manifestations**

- **Nonocular**
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**Typical Optic Neuritis**

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**HLA-DR15**
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--- Anterior

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### CDMS: Manifestations

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**CDMS: Manifestations**

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Which form is most likely to occur in MS?
Intermediate uveitis

Ocular

Optic neuritis S/S
Nystagmus/oscillations
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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

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Two specific manifestations (i.e., signs) of intermediate uveitis are classically associated with MS—which ones?

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Nonocular S/S

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Nystagmus/oscillations

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CDMS: Manifestations

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Intermediate uveitis
**CDMS: Manifestations**

**Nonocular Manifestations**

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Speaking of dz severity: In general, does MS-associated intermediate uveitis tend to be milder, or more severe than the idiopathic version?

Milder
Nonocular:

- Optic neuritis S/S
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- 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

Typical Optic Neuritis

Nonocular

Ocular

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

The Neuro book divvies the nonocular S/S of MS into five groups—what are they?
The Neuro book divvies the nonocular S/S of MS into five groups—what are they?

Nonocular
- Motor
- Sensory
- Cerebellar
- Mental
- Sphincter

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

Typical Optic Neuritis

CDMS: Manifestations
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

**Typical Optic Neuritis**
What sensory symptoms commonly occur in MS?

CDMS: Manifestations

Nonocular
- Motor
- Cerebellar
- Sensory
- Mental

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
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')
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’).

CDMS: Manifestations

Nonocular
- Motor
- Sensory
- Mental

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

Typical Optic Neuritis

Sphincter

Which sphincter are we talking about here?
CDMS: Manifestations

Nonocular

- Motor
- Cerebellar
- Sensory
- Mental
- Sphincter

Ocular

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

Which sphincter are we talking about here?
The bladder sphincter
CDMS: Manifestations

Typical Optic Neuritis

Nonocular

- Motor
- Cerebellar
- Sensory
- Mental
- Sphincter

Ocular

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

Which sphincter are we talking about here?
The bladder sphincter

What bladder sphincter-related symptoms occur in MS?
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)
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

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
CDMS: Evaluation

Typical Optic Neuritis

Labs

What lab test cinches a diagnosis of MS?
There ain’t none

Imaging

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
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?
Typical Optic Neuritis

CDMS: Evaluation

Labs

Imaging

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?
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

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?
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
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

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/w/o gadolinium), there is an image-type that is especially useful—what is it?
FLAIR

Do MS lesions enhance with gadolinium?
Yes
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?
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

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
**Typical Optic Neuritis**

**CDMS: Evaluation**

**Imaging**

**Labs**

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*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
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
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.
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**

**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 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
CDMS: **Treatment**

- **Steroids**
- **DMT**

*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?*
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.
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.
What does DMT stand for in this context?
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?
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
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.
Typical Optic Neuritis

50 Ways to Take a Break

(This is a good point in the set to take a break)
Typical Optic Neuritis

Optic neuritis

As we have seen, when assessing a typical optic neuritis pt it is vital to ask oneself:

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…

aka clinically isolated syndrome (CIS)
Typical Optic Neuritis

Does she have MS?

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 **neuromyelitis optica** (or **neuromyelitis optica spectrum disorder**)?’

*what shorter abb. stands for* (or *what longer abb. stands for*)
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)?’

And: 2) ‘Does this pt have clinically isolated optic neuritis, or... does she have myelin oligodendrocyte glycoprotein IgG-associated disorder?’

What abb. stands for NMO? NMOSD?
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?’
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?'

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*

**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?"

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?"

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?'

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 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
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?

What is the other?

Longitudinally extensive transverse myelitis
Typical Optic Neuritis

What is transverse myelitis?
Inflammation of the spinal cord

What is the other?
Longitudinally extensive transverse myelitis
What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?

What is the other?
Longitudinally extensive

transverse myelitis
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
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

But MS can also present with paresis + sensory loss. How is this any different?

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

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 the other?
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 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, but can be separated by several years

How does transverse myelitis manifest on MRI? As a hyperintense signal on T2 imaging
By what eponymic 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 unit of time to unit of time of each other

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

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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?
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Inflammation of the spinal cord

How does transverse myelitis present clinically?
As a symmetric para- or quadripareisis, often with sensory loss

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They usually occur within weeks to months of each other, but can be separated by several years.
Typical Optic Neuritis

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

What is the other?
Longitudinally extensive

NMO
aka Devic’s dz

What is transverse myelitis?
Inflammation of the spinal cord

How does transverse myelitis present clinically?
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NMOSD

MS

MOGAD
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As a hyperintense signal on T2 imaging
Typical Optic Neuritis

Sagittal T2-weighted MRI of the spinal cord in a patient with NMOSD depicting a hyperintense lesion
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 quadriplegia, often with sensory loss

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How extensive (ie, long) are these lesions?
**Typical Optic Neuritis**

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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

NMOSD

MS

NMO

aka Devic’s dz

MOGAD
Sagittal T2-weighted MRI of the spinal cord in a patient with NMOSD depicting a hyperintense lesion **over more than 3 vertebral segments**
NMO (aka Devic's dz) involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Longitudinally extensive transverse myelitis.

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?

How extensive (ie, long) are these lesions? 2-3 vertebral segments or so.

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 the other?

Longitudinally extensive transverse myelitis.
By what eponymous name is NMO also known?

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What is transverse myelitis?

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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.

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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
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*. 

**Typical Optic Neuritis**
Typical Optic Neuritis

What is transverse myelitis?
- Longitudinally extensive transverse myelitis

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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).
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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 2 to 3 weeks to months of each other, but can be separated by several years

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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 4 weeks to 4 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

What is the Optic Chiasm? Almost unheard of in typical optic neuritis

What does chiasmal involvement portend vis à 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
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better vs worse
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What is the Optic Chiasm involved in NMO(SD)?

- Yes, that's true of the optic nerve lesions as well

How posterior are we talking about here?

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What is the Optic Chiasm? A very small structure that may be found at the base of the brain near the beginning of the optic nerve. It is considered a midline structure.

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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?

- The point at which the optic nerves come together

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NMOSD

aka Devic’s dz

MS

NMO

MOGAD
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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 (aka Devic’s dz) stands for aquaporin-4.
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?
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NMO is an antibody-mediated autoimmune condition. What is the target of the antibodies? The protein aquaporin-4 (AQP4) is targeted.

What does this protein do? It is the main water channel protein in astroglial cells.

In the present context, what do astrocytes do?
NMOSD

MS

NMO
aka Devic's dz

MOGAD

+ transverse myelitis

Optic neuritis

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?**
Typical Optic Neuritis

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What do oligodendrocytes do?
They provide myelin in the CNS—oligodendrocyte loss = demyelination
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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)

What does this protein do?
It is the main water channel protein in astroglial cells

In the present context, what do astrocytes do?
Their loss results in 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?
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NMOSD involves three separate and specific inflammatory processes. Two are optic neuritis and longitudinally extensive transverse myelitis. What is the third?
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Area postrema syndrome

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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?
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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

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How does area postrema syndrome present clinically?
With intractable episodes of one or both of the following:
--Hiccups
--Nausea/vomiting
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How long do hiccups have to last to be considered ‘intractable’?
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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. How long do hiccups have to last to be considered 'intractable'?
At least 30 days or so.
Typical Optic Neuritis

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Area postrema syndrome

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Speaking of intractable hiccups…Only two conditions addressed in the BCSC can present with them. One is NMOSD; what is the other?

Wallenberg syndrome

What is the noneponymous name for Wallenberg syndrome?

Lateral medullary syndrome

What is the classic ocular finding in Wallenberg syndrome?

Horner syndrome

Wallenberg’s hallmark nonocular symptom is sensory. What is it?

Loss of pain and temperature sensation to the ipsilateral face and contralateral body

What other signs/symptoms are common in Wallenberg syndrome?

--Cerebellar signs: Disequilibrium, ataxia, nystagmus, skew deviation
--Speech and swallowing difficulties

Occlusion of what vessel is implicated in Wallenberg syndrome?

The ipsilateral vertebral or (less commonly) posterior inferior cerebellar artery
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Wallenberg syndrome
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Horner syndrome
Wallenberg’s hallmark nonocular symptom is sensory. What is it?
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- Cerebellar signs: Disequilibrium, ataxia, nystagmus, skew deviation
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Occlusion of what vessel is implicated in Wallenberg syndrome?
The ipsilateral vertebral or (less commonly) posterior inferior cerebellar artery.
NMOSD + area postrema syndrome

NMO aka Devic's dz

Speaking of intractable hiccups…Only two conditions addressed in the BCSC can present with them. One is NMOSD; what is the other?

Wallenberg syndrome

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Area postrema syndrome

What is the area postrema?
A portion of the posterior medulla

How does area postrema syndrome present clinically?
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- Hiccups
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How long do hiccups have to last to be considered ‘intractable’?
At least 30 days or so

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sensory vs motor

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NCOSD in NMOSD

Optic neuritis and area postrema syndrome

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NMOSD is NMO (aka Devic's dz) + transverse myelitis + area postrema syndrome.

For more on Wallenberg syndrome, see slide-set N3

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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?

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Not in North America, but it accounts for about half the cases in Asia and the West Indies.
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**Typical Optic Neuritis**

**NMOSD**

- + area postrema syndrome

**NMO**

aka Devic’s dz

- + transverse myelitis

**Optic neuritis**

**MS**

**MOGAD**
How are NMO and NMOSD treated? Acute exacerbations are treated with steroids.
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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?
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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
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- + area postrema syndrome

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**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

---

**MOG**

-NMOSD

**What is the other?**
Acute disseminated encephalomyelitis (ADEM)
Typical Optic Neuritis

NMOSD

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MS

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MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other?
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It is more prevalent among people who live closer to the equator

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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) 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 prevalent among people who live farther from the equator?

Like MS

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How does it present clinically?
With multifocal neurologic deficits in concert with encephalopathic signs/symptoms
NMOSD

+ area postrema syndrome

MS

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How does it present clinically?
With multifocal neurologic deficits

What are the more common neurologic deficits?
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Typical Optic Neuritis

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+ area postrema syndrome

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Yes, it is more common in males.

There is a geographic predilection—what is it?
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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**

+ Optic neuritis

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What are the S/S of encephalopathy?
Encephalopathic signs/symptoms

Stupor (or even frank coma); irritability; confusion
**Typical Optic Neuritis**

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**MOG**

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NMOSD
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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?
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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)

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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 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)

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NMOSD + area postrema syndrome

NMO aka Devic's dz

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Optic neuritis

MOG involves two separate and specific inflammatory processes. One is optic neuritis. What is the other? Acute disseminated encephalomyelitis (ADEM)

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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)

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
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Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?

Good
Typical Optic Neuritis

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Does it exhibit spontaneous recovery a la typical optic neuritis, or nah like NMO(SD)?
Spontaneous recovery is the rule
**Typical Optic Neuritis**

**NMOSD**
- + area postrema syndrome

**NMO** (aka Devic's dz)

**MOG**

**MOGAD**

**MS**

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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.
Typical Optic Neuritis

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How about long-term visual prognosis: good, or nah?

Good
MOG is an antibody-mediated autoimmune condition. What is the target of the antibodies?
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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.

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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.

**Is lab testing available to detect these antibodies?**
Yes, and they form part of the diagnostic criteria for MOG.

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**Typical Optic Neuritis**

**MOGAD**

**MS**

**Optic neuritis**

**NMO**

aka Devic’s dz

+ transverse myelitis

**MOG**

+ ADEM

**NMOSD**

+ area postrema syndrome

+ 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:

--?
--?
--?
--?
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
- ?
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--?
--?
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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

--Severity vision loss

--?
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--Bilateral presentation
--Severe vision loss
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NMOSD → NMO

aka Devic’s dz

NMO → MOGAD

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
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--Bilateral presentation
--Severe vision loss
--Recurrent episodes

ON enhancement

Two words
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
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--A lack of lesions on MRI
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--Bilateral presentation
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--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
--three words
--three words
--three words
--three words

NMOSD

Typical Optic Neuritis

NMO aka Devic’s dz

MOGAD

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:

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These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
--No spontaneous VA recovery
--No pain with eye movements
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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:

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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
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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:

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--MRI brain unremarkable
--No spontaneous VA recovery
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--Hx transverse myelitis
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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  [two words] syndrome

--?
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:

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These findings push you away from MOG and towards NMO(SD):

--MRI brain unremarkable
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--No pain with eye movements
--Hx transverse myelitis
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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:

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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 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).
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
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-- 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
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--A lack of periventricular white matter lesions on MRI
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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
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--A lack of oligoclonal bands in the CSF

No question—proceed when ready
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These findings push you away from NMO(SD) and towards MOG:
--MRI brain with possibly changes
--?
--?
--?
--?
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:

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These findings push you **away** from NMO(SD) and **towards** MOG:

- MRI brain with gray-matter changes

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:

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Typical Optic Neuritis

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These findings push you away from NMO(SD) and towards MOG:

--MRI brain with gray-matter changes on MRI
--?
--?
--?

---

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MOGAD

NMO akas Devic's dz

Optic Neuritis

MS
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--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:

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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
--- ?

These findings push you away from MOG and towards NMO(SD):

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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
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(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.
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.