Herr Doktor Fuchs left quite an ophthalmic legacy. Can you identify these eponymous diseases and signs?

Fuchs

Seen mainly by pathologists

Ernst Fuchs
(1851-1930)
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**Fuchs adenoma**

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Seen every day at the slit lamp

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**Fuchs** two words-itis

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An occasional DFE finding
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Fuchs crypts
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Fuchs marginal keratitis
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Dalén - Fuchs nodule
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Fuchs one word

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What are the two noneponymous names for Fuchs adenoma?

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What are the two noneponymous names for Fuchs adenoma?
- Pseudoadenomatous hyperplasia of the ciliary body
- Coronal adenoma

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**What extremely important function is served by the nonpigmented epithelium of the ciliary body?**

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There’s another benign (but locally very aggressive) tumor of the nonpigmented epithelium of the ciliary body. What is it?

**Medulloepithelioma (aka diktyoma)**
- Rare
- Presents in childhood
- Usually requires enucleation
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There's another benign (but **locally very aggressive**) tumor of the nonpigmented epithelium of the ciliary body. What is it?

Medulloepithelioma (aka diktyoma)

How 'locally aggressive' is it?
- Aggressive enough to cause death

**Fuchs endothelial dystrophy**

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How does it present?

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No, it presents in childhood

*How does it present?*

As an iris mass along with one or more of the following:

- Glaucoma
- Hyphema
- Sectoral cataract

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*Medulloepithelioma (aka diktyoma)*

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*Enucleation is usually required*
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**Fuchs crypts**
- Craterlike openings in surface of the anterior

**Fuchs endothelial dystrophy**

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**Ernst Fuchs**
(1851-1930)

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**Fuchs colloidal keratopathy**
Ernst Fuchs
(1851-1930)

Herr Doktor Fuchs left quite an ophthalmic legacy. Can you identify these eponymous diseases and signs?

Fuchs adenoma
--Very common (~25% of eyes post-mortem)
--A small (<1mm) nodular hyperplasia of the nonpigmented epithelium of the ciliary body
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Fuchs crypts
--Craterlike openings in surface of the anterior iris

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**Why is the ONH tilted in this manner?**
- Abnormal closure of the embryonic optic fissure at the optic-nerve/globe junction results in an oblique (read: tilted) orientation of the ONH. The abnormal closure also creates a modest staphyloma in the inferonasal region of the globe.

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OK, but why on earth do these pts get a VF defect?

It's actually pretty simple. As indicated below, these eyes are staphyloma-ish inferonasally. Thus, the 'axial length' inferonasally is longer than it is in other regions. Because of this extra axial length, the refractive correction used during the performance of a visual-field test--a correction based on the non-staphylomatous fovea--is not myopic enough for the inferonasal retina. The subsequent uncorrected refractive error produces a refractive scotoma in the superotemporal VF. This implies that the VF defect will resolve if the 'proper' refractive correction is employed. Does it? Indeed it does

This implies also that the bitemporal VF loss shouldn't respect the vertical midline. Does it? Indeed it doesn't
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There is another optic-nerve condition that presents with nonprogressive bitemporal inferior loss that doesn’t respect the vertical midline. What is it?
Superior segmental optic nerve hypoplasia (SSONH)

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**Superior segmental optic nerve hypoplasia (SSONH)**

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What is the appearance of the nerve head in SSONH? Pretty much what you would expect based on the name—a normal-appearing nerve save for a thin superior rim, with associated thinning of the superior nerve fiber layer.

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**Superior segmental optic nerve hypoplasia (SSONH)**
- There is another optic-nerve condition that presents with nonprogressive bitemporal inferior loss that doesn’t respect the vertical midline. What is it?
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**What is the appearance of the nerve head in SSONH?**
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**Is SSONH an acquire, or congenital condition?**
- Congenital

**What is the classic causal association? (Hint: It concerns the status of the pt's mother.)**
- Maternal DM

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**4 Ds of optic-nerve hypoplasia**
- Diabetes
- Drink (ie, heavy maternal EtOH consumption during gestation)
- Drugs, especially Dilantin (ie, maternal use during gestation)
- de Morsier syndrome

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**Ernst Fuchs**
(1851-1930)

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--Area of RPE hyperplasia overlying regressed CNVM in pathologic myopia

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**Fuchs endothelial dystrophy**

**Fuchs heterochromic iridocyclitis**
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Fuchs coloboma
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- The ONH is elevated superotemporal
- Associated with high myopia
- Progressive pathologic myopia
- Pseudovitreous plaque overlying nonpigmented epithelium of the ciliary body
- Looks like a tiny pearl onion on the pars plicata
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Dalén–Fuchs nodule
- Highly focal aggregate of inflammatory cells beneath the RPE
- Seen in granulomatous conditions, esp. sympathetic ophthalmia
- Classic trigeminal neuralgia
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- Diminished visual acuity
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- Can cause secondary glaucoma

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What is the exception; ie, under what circumstances is the darker eye the one with FHI?

In individuals with light-blue eyes...the iris atrophy stemming from the FHI process will make visible the darkly-pigmented epithelium of the posterior iris, thus making the eye appear darker.
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Besides being stellate, what else is unusual about the KP in FHI?
1) They can be interconnected via very fine tendrils or filaments
2) They are evenly distributed across the entire endothelium

How are KP usually distributed?
In an area of the cornea known as Arlt’s triangle

Where/what is Arlt’s triangle?
It’s an equilateral triangle with its apex at the corneal center and base near the inferior border of the cornea

Name 4 other uveitides associated with stellate KP.
1) Herpes simplex
2) Herpes zoster
3) CMV retinitis
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