The retina - retinal diseases
The eye

Forrás: http://www.virtualmedicalcentre.com
The layers of the retina

X. Retinal pigment epithelium (single cubic layer of pigmented epithelial cells)
IX. Layer of rods and cones (photoreceptors, supporting of Müller cells)
VIII. Outer limiting membrane (process of glial cells)
VII. Outer nuclear layer (cell nuclei of the rods and cones – first neuron)
VI. Outer plexiform layer (synapses between the axons of the first neuron and dendrites of the second neuron)
V. Inner nuclear layer (cell nuclei of the bipolar nerve cells of the second neuron, horizontal cells, amacrin cells)
IV. Inner plexiform layer (synapses between the axons of the second neuron and dendrites of the third neuron)
III. Ganglion cell layer (cell nuclei of the multipolar ganglion cells of the third neuron)
II. Nerve fiber layer (axons of the third neuron)
I. Internal limiting membrane (glial cell fibers separating the retina from the vitreous body)
Histology of the retinal layers

I. Internal limiting membrane
II. Nerve fiber layer
III. Ganglion cell layer
IV. Inner plexiform layer
V. Inner nuclear layer
VI. Outer plexiform layer
VII. Outer nuclear layer
VIII. Outer limiting membrane
IX. Layer of rods and cones
X. Retinal pigment epithelium
Light pass through the retinal layer

1. The visible light is absorbed by the photopigments of the outer layer
2. The signals are created in a multiple step photochemical reaction
3. They reach the photoreceptor synapsis as action potentials where they are relayed to the second neuron
4. The signals are relayed to the 3. or 4. neurons
5. The impulse is transmitted through the fibres of the optic nerve
   → visual cortex
Examination of the retina

Helmholz was the first who invented and Graefe was a pioneer who examined and described the changes of the fundus.
Examination of the retina

Visual acuity examination
Visual field test
Direct ophthalmoscopy
Indirect binocular ophthalmoscopy
Ultrasonography
Fluorescein angiography
Optical coherence tomography (II, III. gen)
Normal fundus

Retina: transparent, bright red coloration from the vasculature of the choroid
Optic disk: sharply edged yellowish-orange, may be exhibit a central depression
Macular area: flattened oval area, 3-4 mm, temporal to and slightly below (15°) the optic disk
Arterioles: terminal
Venules
Direct ophthalmoscopy

- is positioned close to the patient’s eye
- 16 power magnified images
- Shows only a short portion
- Contains +/- lenses
  - Direct images, 2 dimensional
  - Suitable technic for less experience examiner
- Using to measure prominence of retinal changes
Indirect ophthalmoscopy

Using the slitlamp
90 D lens
Patient is sitting
3 dimensional images
Virtual, inverted images
Binocular
Stereoscopic overview
Indirect binocular ophthalmoscopy

- 2-6 magnification
- virtual, inverted images
- binocular
- good stereoscopic overview
- 3 dimensional
- It is a screening system (digital)

Forrás: http://www.gheg.de
Ultrasonography

Indication: cloudy ocular media (cataract, corneal haze, vitreous haemorrhages)
Special ocular transducer
Retina is highly reflective
Vitreous body is anechoic
- Using: retinal detachment, intraocular tumor, trauma
Fluorescence angiography

5.0 ml of 5% fluorescence sodium injected into the cubital vein
Fluoresceine is binding to the blood protein in the arterioles and veins
Shows the vasculature
Digital imagine system is showing imagines
Hyperfluorescency hypofluorescency
Using: diagnose AMD, tumor, retinal disorders, retinal vascular disorder, diabetic retinopathy, inflammatory retinal processes
Optical coherence tomography

II. generation

Near infrared light passing through the retina is compared interferometrically with a reference beam at each level within the tissue. The strength of the interferometric signal depends upon the optical reflectivity of each retinal structure, allowing construction of high-resolution cross-sectional images.

- resolution of 8 to 10 μm,
- in vivo measurements, three-dimensional
Optical coherence tomography

III. generation

- in vivo measurements
- resolution of 2 μm,
- fast image acquisition
Vascular supply to the retina

Retinal Arterial Occlusion

Definition: Retinal infarction due to occlusion of an artery.
Forms: central/branch
Etiology: embolisation, arteriosclerosis
Symptoms: sudden, painless, unilateral blindness
Ophthalmoscopy: retina is grayish-white due to edema and loss of its transparency
Fovea centralis is cherry red spot (not contain any fibers)
Column of blood will be seen to be interrupted
Rarely will be observe an embolus
Retinal Arterial Occlusion
Treatment

Emergency treatment (6 h) is often unsuccessful. Ocular paracentesis, massage, reduce IOP. Hemodilution is improved vascular supply. Work up to identify the source of the embolus is important. To treat the underlying disease and prevent another embolization.

- Systemic/local lysis is no longer performed due to the poor prognosis.
Retinal Vein Occlusion

Definition: Circulatory dysfunction of CRV/BRV

Etiology: local factors (a-v crossing)
- arteriosclerosis,
- cardiovascular/thrombo-philic risk factors

Symptoms: slowly loss of vision (difference power)

Ophthalmoscopy: linear, intraretinal hemorrhages, cotton-wool spots, optic disk and macular edema
Retinal Vein Occlusion

Forms: Branch/central/hemiretinal

Pathogenesis: ischemic/non ischemic forms are diagnosed by FA. (larger than 10 disk diameter the non perfusion area)

Treatment (acute): Hemodilution, crystalloid infusion, low molecular weight heparin, decreasing retinal edema, radical scavengers (Vitamin-C), antithrombotic/antiplatelet therapy

Decrease risk factors!

Treatment (later): laser, vitrectomy
Hypertensive Retinopathy stage I.

Definition: is a retinopathy with arterial changes caused by HT.

Stages: I-IV

Symptoms: signs of HT

Ophthalmoscopy: different changes of I-IV, loss of VA, hypertensive vascular changes

Treatment: blood pressure should be reduce (120/90 mmHg)

Stage I: widening of arteriole reflexes, (tailspin) arterioles changes
Hypertensive Retinopathy stage II.

Stage II:
- vascular constriction
- veins tortousity
- Gunn’s a-v crossing
Hypertensive Retinopathy stage III-IV.

Stage III:
- copper-wire arterioles
- Retinal hemorrhages
- Hard exudates
- Cotton-wool spots
- Retinal edema

Stage IV: Stage III + disc swelling
- silver-wire arterioles
Diabetic Retinopathy

Definition: is an ocular microangiopathy in DM, it results the thickening the basement membrane of the vessels and loss of pericytes and endothelial cells.

Pathogenesis: capillary closure resulting an ischemia and hypoxy. Angiogenetic factors are produced, resulting breakdown in the blood-retina barrier→NV, macular edema

Stages:0-4

Symptoms: asymptomatic for a long time, only late stages and macular involvement occur decreasing VA

Ophthalmoscopy: different at stages

Treatment: DM, decreasing risk factors, laser, vitrectomy end-stage untreatable
Diabetic Retinopathy
background

Stage „0”:- without any angiopathy/retinopathy

Stage 1. background:
- microaneurysm
- retinal hemorrhages
- hard exudates
- cotton-wool spots
- microvascular abnormalities (2-3 quadrants)
Diabetic Retinopathy
pre-proliferative

Stage 2.: all of these signs in 4 quadrants and intraretinal NV

Treatment: severe retinopathy + NV→may be laser
Diabetic Retinopathy
proliferative

Stage 3.

- papillary/elsewhere
- NV and connective tissue
- Vitreous hemorrhages

Treatment: laser/vitrectomy
Diabetic Retinopathy
proliferative

Stage 4.:
- end-stage with traction retinal detachment
Treatment: vitrectomy but the result is poor

PREVENTION!
Focal, direct, laser treatment
Panretinal laser treatment
Diabetic maculopathy

Definition: It is independent disorder which is developed in all stages resulting the breakdown of the blood-retina barrier

CSMO: hard exudates, thickening the retina within 500µm of the center of the macula

Ischemic maculopathy: with enlargement of foveal avascular zone caused by capillary closure (leads to visual impairment/blind)

Treatment: focal/grid laser
Periferal Retinal Degenerations I.

Definition: degenerative changes lie parallel to the ora serrata of the retina

Forms: harmless / Precursors of retinal detachment

Symptoms: asymptomatic

3 mirror lens examination: depend on forms of retinal degenerations

Treatment: may be laser

Prognosis: long term risk of retinal detachment is only 1%
Periferal Retinal Degenerations II.

Harmless
- Vitreoretinal adhesions
- Pigment hypertrophy (clumping)
- Chorioretinal atrophy
- Retinoschisis
- White without pressure degeneration

Precursors of retinal detachment
- Lattice
- Snail-track
Retinoschisis I.

Definition: is splitting of the sensory retina into two layers at level of the outer plexiform layer (VI-IV).
Symptoms: asymptomatic (VA ↓)
Ophthalmoscopy: reveal bullose separation of the split inner layer of the retina
Treatment: surgical or laser
Retinoschisis III.
III. generation OCT
Retinal break

Definition: is a full-thickness defect (hole) in the sensory retina
Symptoms: flashes of light, floaters,
Ophthalmoscopy: PVD, hemorrhages,
Treatment: surgical or laser
Age-related Macular Degeneration

Definition: progressive degeneration of the macula in elderly patients

Pathogenesis: genetic factors, smoking, intensive sunlight, aging, hypoxia → VEGF ↑

Stages: early/late (choroidal NV)

Symptoms: gradual loss of VA, image distorsion, macropsia/micropsia, color-vision disturbing
Age-related Macular Degeneration

Stages

**Early**

Ophthalmoscopy: drusen, atrophy and proliferation of retinal pigment epithelium

Treatment: non-specific, lutein-vitamin products

**Late (Wet)**

Ophthalmoscopy: signs of early stage + geographical atrophy, serous fluid, lipids, intraretinal hemorrhage, serous PE detachment

End stage: fibrovascular scar

Treatment: laser, intravitreal VEGF inhibitors (ranibizumab)
Retinal Inflammatory Disease I.

Definition: inflammation which affects the retinal choroidal layer

Forms:
- acute/chronic, exogenous/endogenous,
- focal/disseminated, vasculitis/retinitis,

Symptoms: VA↓, floaters,

Signs: vitreous changes: flare, cells opacities, PVD, tissue destructions, snow-ball opacities,

Ophthalmoscopy: choroiditis with yellow/greyish patches, retinitis
Retinal Inflammatory Disease II.  
Endogenous-focal chorioretinitis

1. Idiopathic (do not fall into any categories-25%)
2. Associated with systemic disease (spondilitis)
3. Infections (bacteria-tuberculosis, viruses-h.zoster CMV, fungi-candidiasis, protozoons-toxoplasmosis, rouhworms-toxocariasis

Retinal Inflammatory Disease III.
Endogenous-multifocal chorioretinitis

Ophthalmoscopy: white necroting infiltrates of the inner retina, hemorrhages, retinal exudation

Treatment: systemic steroids, antiviral drugs (acyclovir, gancyclovir), chlorambucyl

Forrás: http://www.aao.org
Retinal vasculitis

Definition: inflammation of the retinal vasculature
Epidemiology: one of the clinical sy-s
Symptoms: VA ↓, black dots, Slit lamp: cells in the vitreous body, whitish preretinal infiltrates, vascular occlusion, intraretinal bleeding, edema↓
Treatment: may be specific
Central Serous Chorioretinopathy

Definition: serous detachment of the retina and/or retinal PE
Epidemiology: 3-4. decade of life, men
Etiology: physical/psychological stress → local factors → defect of outer retinal blood-barrier
Symptoms: VA↓, hyperopia, central dark spots, image distortion, macropsia/micropsia
Ophthalmoscopy: serous RD in the macula, fine brown/white PE scar
Treatment: is requires for the first occurens. NSAID (indometacinum)
Central Serous Chorioretinopathy
II. and III. generation OCT
Retinal Detachment

Definition: degenerative retinal disorder, with the detachment of the neurosensory retina from the underlying retinal PE

Types: 1. rhegmatogenous, 2. tractional, 3. exudative, 4. tumor related

Symptoms: flashes of the light, floaters, dark shadow in the visual field, VA↓

Ophthalmoscopy/stereoscopy: detached, white retina, and loss of its transparency

Treatment: surgical
Retinal Detachment
II. and III. generation OCT
Retinopathy of prematurity I.

Definition: Retinal disorder of the preterm infants with the disruption of the normal development of the retinal vasculature. Normally after birth the growth of the retinal vasculature continues for 4 weeks. Exposure to oxygen disturbs the normal development of the retinal vasculature. Vessels obliteration, NV,

Zones: divided 3 zones in the fundus of preterm infants

The most severe changes can be seen in zone I and II.

Stages 1-5 based on severity of the disease
Retinopathy of prematurity II.

Stages

1. Demarcation

2. Formation of the ridge

3. Ridge with extraretinal proliferation

Retinopathy of prematurity III. Stages

4. Subtotal retinal detachment

5. Total retinal detachment

Toxic retinopathy

Definition: the retinal changes in this retinopathy caused by medications, are often bilateral and symmetrical.

Probably toxic agents are: chloroquin, quinin, tamoxifen, thioridazin

Ophthalmoscopy: initially macular edema → later punctuate PE changes → bull’s eye maculopathy → end stage maculopathy

Treatment: there is no treatment
Retinal dystrophies I.

Best’s Vitelliform dystrophy

Definition: disorder of the macula, usually bilateral

Inheritance: autosomal dominant trait with variable penetrance and expressivity

Stages: 1-5

Symptoms: VA↓,

Ophthalmoscopy: morphologic findings are remarkable: yellowish central pigment changes→like a fried egg→scar

Treatment: there is no treatment
Retinal dystrophies II.

Stargardt disease

Definition: macular dystrophy with changes in the retinal PE
Inheritance: autosomal recessive disease
Symptoms: progressive loss of VA
Ophthalmoscopy: slight, white fleck lesions of the macula, later these are developed at the periphery too
Treatment: no treatment

Forrás: http://retinagallery.com
Retinal dystrophies III.

Retinitis pigmentosa

Definition: Pigment deposits characterize this disorder which progress from the periphery to the center of the retina. It is a rod-cone dystrophy.

Inheritance: autosomal recessive (60%), autosomal dominant (25%) and X-linked (15%),

Symptoms: glare, night blindness, VA↓, visual field defects, color vision defects

Ophthalmoscopy: bone-cell like brown pigmentation, atrophy of the optic nerve

Treatment: no treatment
Thanks for your attention!