

RETINAL VASCULAR OCCLUSIONS

RETINAL VEIN OCCLUSION (RVO)

Types

1) Central Retinal Vein Occlusion (CRVO)

- Non-ischaemic (venous stasis retinopathy)
- Ischaemic

2) Branch Retinal Vein Occlusion (BRVO)

3) Hemi-Retinal Vein Occlusion (HRVO)

Aetiology / Predisposing Factors

Common:

- Age >55 yrs (20% cases)

- Arteriolar sclerosis → compression at AV crossings or common sheath posterior to lamina cribrosa
- Systemic hypertension (2/3 cases BRVO)
- Diabetes mellitus (~10% cases)
- Raised intraocular pressure (CRVO more common in POAG)
- Drugs: oral contraceptives
- Smoking

Uncommon:

- Hyperviscosity: leukemia, polycythemia
- Thrombophilia, hyperhomocysteinemia
- Inflammatory: Eales disease, sarcoidosis
- Local: orbital cellulitis, cavernous sinus thrombosis

Investigations 

- Blood pressure
- CBC, ESR
- Fasting & random blood sugar

- Lipid profile
 - Plasma protein electrophoresis
 - Autoantibodies: ANA, Anti-DNA, ANCA
 - Fundus exam, FFA, OCT for macular assessment
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CENTRAL RETINAL VEIN OCCLUSION (CRVO)

Non-Ischaemic CRVO (75%)

Symptoms:

- Sudden, unilateral, painless vision loss

Signs:

- Mild-moderate visual loss
- No/mild afferent pupillary defect
- Retinal veins: dilated, tortuous
- Dot-blot & flame-shaped hemorrhages (all quadrants)
- Mild cotton-wool spots, mild optic disc & macular edema

Investigations:

- FFA: capillary perfusion
- OCT: macular edema assessment

Visual Prognosis

- Haemorrhages resolve within 6-12 months.
- Vision returns near normal in ~50% cases.
- Persistent visual loss is mainly due to cystoid macular oedema.

Complications:

- Cystoid macular edema (CME)
- Conversion to ischaemic type (15% in 4 months, 30% in 3 yrs)

Treatment:

- Systemic: control HTN, DM, IOP; stop OCPs; aspirin/antiplatelets
- Ocular: anti-VEGF (Avastin, Lucentis, Eylea), intravitreal triamcinolone

- Laser: not effective for vision improvement
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Ischaemic CRVO

Features:

- Severe, sudden unilateral vision loss (counting fingers or worse)
- Marked afferent pupillary defect
- Retinal veins: very tortuous & engorged
- Extensive deep blot & flame-shaped hemorrhages ("tomato-soup fundus")
- Severe disc edema, cotton-wool spots

Investigations:

- FFA: extensive non-perfusion
- OCT: macular edema

Prognosis:

- Poor due to macular ischemia

Complications:

- Macular ischemia, chronic CMO
- Rubeosis iridis → neovascular glaucoma (>50% cases in 3 months)

Treatment:

- Macular edema: same as non-ischaemic
- Rubeosis iridis: PRP; cryotherapy if view is obscured

BRANCH RETINAL VEIN OCCLUSION (BRVO)

Types:

- Quadrant vein occlusion (major branch away from disc at AV crossing)
- Hemiretinal vein occlusion (main branch at disc margin)

Clinical Features:

- Sudden blurred vision if macula involved

- Fundus: hemorrhages & edema in affected area only
- Secondary glaucoma: rare

Prognosis:

- Generally good; chronic macular edema is main cause of poor vision
- Neovascularization develops in ~10%

Treatment:

- Observation if visual acuity good
- Anti-VEGF for macular edema
- Laser photocoagulation:
 - Grid for macular edema with good perfusion
 - Scatter for neovascularization
- Intravitreal steroid (triamcinolone) for persistent macular edema

RETINAL ARTERY OCCLUSION (RAO)  

Types

1. Central Retinal Artery Occlusion (CRAO)
 2. Branch Retinal Artery Occlusion (BRAO)
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CENTRAL RETINAL ARTERY OCCLUSION (CRAO) ⚡

Aetiology / Causes

- Thrombosis at lamina cribrosa → ~75% cases
 - Embolism (~20%): from carotid atherosclerosis, aortic arch, cardiac valves
 - Cholesterol emboli (commonest)
 - Platelet-fibrin emboli
 - Calcific emboli
 - Inflammation: giant cell arteritis, SLE, PAN, Wegener
 - Vasospasm: retinal migraine
 - Raised IOP: post-retinal surgery
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Clinical Features

Symptoms:

- Sudden, painless, monocular vision loss
- Amaurosis fugax ("curtain" over vision)
- Visual acuity: profound loss (may be light perception only)

Signs:

- Fundus: pale, whitish retina due to intracellular edema
- Cherry-red spot at fovea (choroid visible through thin retina)
- Fovea remains red because it is supplied by the underlying choroidal circulation (via short posterior ciliary arteries) rather than the occluded central retinal artery
- Narrowed retinal arteries, mild venous narrowing
- "Cattle-tracking" of blood in veins (segmentation)
- Later: optic atrophy, thread-like arteries

Investigations:

- OCT: may show embolic plaque at optic nerve head
- FFA: delayed arterial filling, masked choroidal fluorescence
- Electroretinography

Prognosis:

- Irreversible neural damage after 90 minutes of ischemia
 - Depends on cause, severity, and duration
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Treatment (Emergency) 🚓

1) Reduce IOP & improve perfusion:

- Acetazolamide 500 mg IV stat or 250 mg qid × 24 h
- Mannitol 20% IV 1.5–2 g/kg over 30–60 min
- Anterior chamber paracentesis
- Digital ocular massage (15–20 min)

2) Dislodge / dissolve embolus:

- Sublingual isosorbide nitrate 10 mg
- Breathing high oxygen + 5% CO₂ mixture
- Nd:YAG laser embolysis (0.5–10 mJ shots)

3) Reduce retinal edema:

- IV methylprednisolone 1 g

BRANCH RETINAL ARTERY OCCLUSION (BRAO)

Aetiology

- Usually embolus-related
- Accounts for ~38% of acute retinal arterial occlusions

Clinical Features

Symptoms:

- Sudden, painless, altitudinal or sectoral visual field loss
- Visual acuity variable

- RAPD often present

Signs:

- Retina distal to occlusion: opaque, whitish, edematous
- Attenuated arteries & veins
- Cherry-red spot may be seen if macula involved

Investigations:

- Visual field testing → confirms defect
- FFA → shows non-perfusion of occluded area

Treatment:

- Same protocol as CRAO
- Nd:YAG laser embolysis if embolus visible (0.5-1 mJ per shot)

-> The End <-