Retinal Artery Occlusion : Ocular Stroke

Five minutes worth of advice from a colleague who knows as much about the topic as anyone in the world. What could be better?

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Occlusion of central retinal artery or its branches, severe enough to cause ischemia.

Patient profile:
Usually seen in early and mid-60s.
Males are affected slightly more than females. No racial predilection.

Risk Factors:
Diabetes, Hypertension, Lipid disorders, Cardiac disease, Systemic atherosclerotic disease

Mechanism of occlusion:
Younger patients: Thrombotic
>50 years of age: Embolic

A) Embolic: Most common cause (CRAO:1/3rd , BRAO: 2/3rd cases)

Sources of emboli-
Endogenous: Carotid Atheroma (80% cases), Cardiac valvular diseases, Cardiac tumour, Fat emboli, Amniotic fluid emboli, Leukoemboli (in pancreatitis), Septic emboli.

Exogenous: Talc emboli (i.v. drug abuse), injected steroids (nasal or periorbital), fragments of catheter tips or artificial heart valves, blood transfusion products.

Types of emboli:
Cholesterol (Hollenhorst plaque): Small glistening white. Usually carotid origin.
Platelet fibrin: Large, long and gray white.
Calcific: most severe occlusion, Large gray white.

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B) Thrombotic: Seen in hypercoagulable states. More common in younger patients.

C) Vasculitic: Most common condition is giant cell arteritis (>55 yrs age, associated with headache, scalp tenderness, jaw claudication, anorexia and fever) Other causes: SLE, RA, Behcet's disease, and localised vasculitis in toxoplasma and bartonella retinitis.

D) Other causes: Reflex vasospasm in migraine, sickle cell haemoglobinopathy and collagen vascular diseases may play a role; elevated IOP; Peripapillary capillary loop and external compression of central retinal artery.

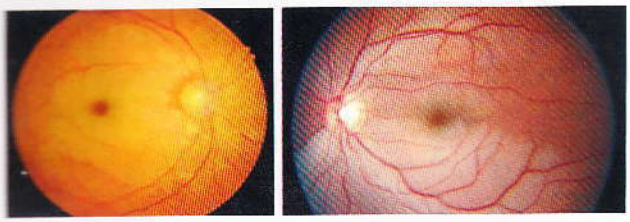
Pathophysiology: Ischemia due to vascular occlusion causes oedema of ganglion cells and they burst, imparting whitening/ opacification to transparent retina.

Presenting complaints:
1) Amaurosis fugax: prodromal symptom
   Transient mono-ocular visual loss
   Lasts 7-30 min
   Total resolution to normal
   Presence mandates complete ophthalmic examination and full systemic workup
2) Acute painless loss of Vision: CRAO
   Loss of Part of Visual field: BRAO
3) BRAO may also be asymptomatic.

Ophthalmic examination:
1) VA: CRAO: <3/60, BRAO: 3/60- 6/6
2) Pupil: CRAO: RAPD, BRAO: ±RAPD
3) Visual field: Field defects corresponding to the part affected
4) Fundus: Changes take 1-2 hrs to become evident clinically

CRAO: Ischemic retinal whitening of whole

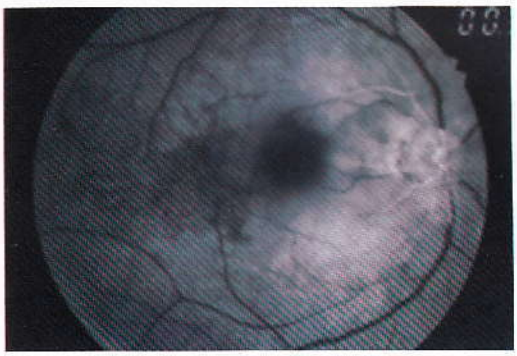
fundus with cherry red spot at fovea (no ganglion cells at fovea, no whitening, evident underlying vascular choroid). Boxcarring in retinal vessels.



BRAO: Retinal whitening in affected part

Less evident in nasal part (single layer ganglion cells).

5) FFA: Intact choroidal flush (c.f. Ocular ischemic syndrome). Absent, delayed or incomplete filling in retinal vessels with leading edge of dye.
6) ERG: Absent b wave (in ocular ischemic syndrome: both a & b wave are absent).



Management:

A) Manage acute occlusive event
- Goal is to restore blood flow.
- Manage aggressively if patients presents within 24 hrs.
- Lower IOP (Target IOP of 15 mm Hg): Digital ocular massage (because of simplicity should be tried in all patients, press globe for 5-15 seconds and sudden release), anterior chamber paracentesis, topical beta blockers, oral acetazolamide.
- Suspicion of giant cell arteritis: Start high dose steroids while biopsy report of temporal artery is awaited (saves other eye)

Other options with doubtful significance
- Medical vasodilatation: Sublingual nitroglycerine (10 mg). Other suggested options are calcium channel blockers, carbogen (inhaled) and pentoxifylline.
- Thrombolytic therapy
- Hyperbaric oxygen

B) Systemic work up
BP, Blood sugar, Lipid profile
Carotid ultrasound: especially older pts.

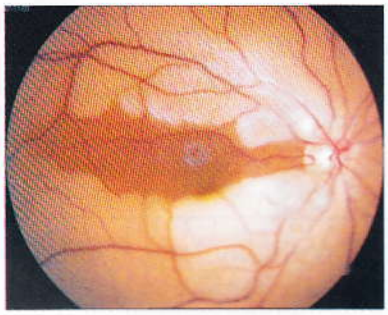
Echocardiography:
CBC, ESR, C - reactive protein, PT, PTT, Protein C, Protein S, Activated protein C, Factor V Leiden, Fasting plasma Homocysteine level, Anti phospholipid antibodies, Hb electrophoresis, VDRL

C) Complications
NVG: occurs in 2-16% cases (mainly CRAO). Most of the cases develop within 2 months. So patients of CRAO should be followed monthly for early diagnosis.

T/t: Anti VEGF, retinal laser photo-coagulation, cyclocryo therapy and anti-glaucoma drugs.

Prognosis
CRAO: Majority patients have final VA of finger counting in resolved phase. May be 6/6 in patients with intact cilioretinal artery.

BRAO: VA 6/12 or better in 80% cases. Fundus may return to near normal after months but field defects persist.



Chronic fundus changes are optic atrophy, attenuated vessels, atrophic retina.