Central Retinal Arterial Occlusions

Director Pakistan Institute of Community Ophthalmology 8 **Professor of Vitreo-retinal Ophthalmology** Hayatabad Medical Complex Peshawar, Pakistan

No financial disclosures

PROF. DR. SANAULLAH JAN FRCS (Edinburgh, UK), FRCS (Glasgow, UK), FCPS (Pak) Fellowship in Vitreo-Retina (Germany & India)

Most important people in Life









PDR and DME

YJ 70/M, 06/02/2019

PDR (NVD) No DME

PRP versus PRP plus Anti-VEGF (Avastin) Earlier & high rate of regression of neovessels in combination group

Mushtaq M, Sanaullah jan. Comparison between Pan-retinal photocougulation and Pan-retinal photocoagulation plus intravitreal bevacizumab in Proliferative diabetic retinopathy. Journal of Ayub Medical College 2012; 24:3-4.

12/06/2014

PDR / No DME but Extensive NVD/NVE

Anti-VEGF? PRP?

Sudden Painless Loss of Vision

- Retinal vascular Occlusions
- Retinal Detachment
- Eale's disease), PVD, Valsava retinopathy, Raised BP etc

• AION

Vitreous /Pre-retinal bleed due proliferative retinopathy (DR, RVO,

Most important people in Life

- Central RAO:
- 50%: idiopathic
- 33% carotid artery disease
- 10% Giant cell arteritis
- Blockage is within optic nerve substance (obstruction site not visible on ophthalmoscope)
- Branch RAO Obstruction distal to Lamina Cribrosa
- Visible blockage.

- Atherosclerosis Most common cause of CRAO --- 80% cases Mostly by thrombus formation (Localized intimal damage due to Artherosclerosis - incites thrombus)
- Embolization: Common cause of CRAO Ophth artery-first branch of internal carotids.
 - Carotids---emboli from atheromatous plaques at carotid bifurcation. Heart:
 - Thrombus
 - **Calcific** Aortic/mitral valves, **Vegetations** Bacterial endocarditis, MI (Lt side of heart), M.stenosis with Atrial fibrillation Myxomatous - Atrial myxoma

Retinal Artery Occlusions Types of emboli

- Carotid artery disease: Leading cause of morbidity and mortality. Carotid bifurcation-Vulnerable to atheromatous ulceration and stenosis
- Cholesterol (Hollenhorst Plaques) Rarely causes significant obstruction. Frequently symptomatic Refractile golden to yellow orange crystals-usually at bifurcation of arteries.

• Fibrinoplate Emboli: Dull grey elongated particles-usually multiple-may fill entire luman.

Causes:

occasionally complete obstruction Retinal TIA-Painless unilateral loss of vision-few min – Recovery

- **Retinal Transit Ischemic Attacks Amaurosis fugax:** usually but
- May be associated ipsilateral cerebral TIAs with contralateral signs.

- Calcific: From atheromatous plaques in ascending aorta/carotid arteries, calcific heart valves- usually single white-usually close to disc more dangerous.
- Periarteritis: Systemic vasculitis, polyarteritis nodosa, S.L.E, Optic neuritis, Behcet's, syphilis, G.C.A and other collagen diseases, mucormycosis.
- Blood disorders: Protein S or Protein C deficiency, sticky platelet syndrome.
- Hypercoagulative states- ploycythemia, sickle cell disease.

- **COMPRESSIVE:** External compression by tumor, hemorrhage, inflammation.
- **TRAUMA:** Direct damage to O.N and vessels.
- Retinal Migraine: Rare Exclude other causes.
- MISCELLANEOUS: BP, Diabetes, Retro bulbar anesthesia etc.

Central Retinal Artery Occlusion:

Size / Location of obstructed vessel. Severity / Duration of obstruction. Usually atheroma – but can be calcific emboli.

FEATURES: within seconds

- Acute / profound visual loss
- Relative APD Marcus Gun pupil
- White retina + cherry red spot within minutes

• 20 % individuals Cilio-retinal arteries from ciliary circulation – spares macula

Retinal vessels – Narrowing – sludging and segmentation of blood column(wks)

FA of central retinal artery occlusion

Early filling of cilioretinal artery

Non-filling of other vessels

Late staining of vessel walls

Branch retinal artery occlusion (BRAO)

- VA variable
- APD mild or absent
 - Retina whitening
- Arteriolar narrowing

White cloudy swelling clears (Permanent sectorial visual field defect-atrophy of inner retinal layers). **Re-canalization of obstructed** vessel – only subtle or absent ophth signs

FA of branch retinal artery occlusion

Early masking

Extreme delay of Arterial phase Late staining of arterial walls

EVALUATION: Clinical

F.F.A

- **Color Doppler U/S evaluation** orbital circulation degree of obstruction differentiate ophthalmic artery obstruction from CRAO
 - BRAO/CRAO- Pts at risk to have obstruction in other eye 10%-bilaterality Evaluate for embolic sources:

Cardiovascular exam

Carotid artery exam

(Echocardiogram, carotid non-invasive testing)

Rule out G.C.A- Pts above 50 years

Do E.S.R-Biopsy if needed.

Cherry red spot (CRAO)- Ischemic visual loss, age, associated systemic disease, check surrounding vessels and retina, other causes-storage diseases

TREATMENT

Retina-Highly metabolic organ -very sensitive to ischemia

Ischemia - 90 min- cell death due to hypoxia No proven Rx available

Try with in 48/72 hours

Dislodging emboli distally:

>A/C Paracentesis, ocular massage, medications to lower IOP

➢ CRA is end artery -No true normal anastomosis - No regeneration -

Dissolving thrombi: Clot dissolving medications streptokinase, urokinase, heparin, tPA Systemic-I/V infusion Local -via ophth artery with catheter Initial reports-encouraging-risks only reserved for cases and only CRAO not BRAO.

Increasing oxygenation to retina: Carbogen - 95% O2 + 5% Co2 For 10 min every 2 hours for 1-2 days. Avoid in chronic obstructive lung disease.

Protecting surviving retinal cells from ischemic damage?

within 48 hours

Treatment options for carotid disease

Antiplatelet therapy

- Aspirin 75 mg daily
 Aspirin + dipyridamole (Persantin)
- Clopidorel (Plavix) 75 mg daily

Anticoagulants

- if antiplatelet therapy ineffective
- **Carotid endarterectomy**
- Patients with other risk factors for stroke
 Symptomatic carotid stoposis > 70%
- Symptomatic carotid stenosis > 70%

Neo-vascularization is uncommon compared to venous occlusions

Proliferative retinopathy: NVE /NVD

Laser

• Anti VEGF

S

Thanks