Retinal Manifestations in Systemic Infection – A Ready Reckoner

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Bacterial Infections

OCULAR TUBERCULOSIS

Focal choroiditis (Fig 1) in a 28 year old lady who presented with mild blurring of vision OD with multiple systemic problems including cranial nerve palsies. HRCT was done which revealed the presence of miliary tuberculosis.

Figure 1

Subretinal abscess with optic disc granuloma in a 38 year old female who presented with periorbital swelling and pain on extraocular movements. (Fig 2a)

Mantoux was done which showed necrotising reaction. Resolution was seen following ATT and supportive systemic steroids. (Fig 2b)

Figure 2a

Figure 2b
Focal retinochoroiditis (Fig 3a) with vitreous haze in a 19 year old boy who presented with recent onset blurring of vision with a significant history of weight loss and evening rise of temperature. Systemic evaluation showed caseating hilar lymphadenopathy, following which he was put on ATT. The retinochoroidal lesion subsequently scarred (Fig 3b).

Multifocal choroiditis with optic nerve head granuloma and papillitis (Fig 4) in a 40 year old male, who presented with painful, sudden onset decrease in vision, lid edema and erythema, a picture simulating orbital cellulitis. Systemic workup showed presence of tuberculosis.

Solitary choroidal granuloma (Fig 5) discovered in a 50 year old male of pulmonary tuberculosis who had no eye symptoms but came for routine ophthalmic evaluation.

SYPHILIS

Ocular syphilis is relatively uncommon and can have myriad presentations like focal or multifocal choroiditis, chorioretinitis, neuroretinitis, occlusive vasculitis (Fig 6), optic neuropathy and even extensive pigmentary in late stages changes simulating retinitis pigmentosa. It should essentially be suspected in any case of uveitis resistant to conventional therapy. Ocular syphilis should be equated with tertiary syphilis and managed like neurosyphilis.

PARASITIC INFECTIONS

OCULAR TOXOPLASMOSIS

Focal necrotizing lesion adjacent to an old chorioretinal scar. (Fig 7a, b) representing
congenital infection. This is a classical presentation of Toxoplasmosis as compared to tuberculosis where fresh activity is seen within an area of old scar. Systemic toxoplasmosis can present with multiple lymphadenitis and pyrexia of unknown origin and can even be mistaken for lymphoma.

Focal necrotizing retinochoroiditis patch without an adjacent chorioretinal scar (Fig8) – active toxoplasma retinochoroiditis representing an acquired infection.

Dense vitreous haze with underlying active focal retinitis patch (Fig 9a) - Head light in Fog appearance

Inactive vasculitis with an adjacent retinochoroidal scar (Fig 9b)

Kyrelias arterialitis - non occlusive non inflammatory, non leaking arterial plaque (Fig 10)
Franceschetti’s syndrome- traction band extending from old scar to disc. These bands can also extend between two scars (Fig 11)

Inactive lesion- Punched out chorioretinal scar at posterior pole (Fig 12)

**OCULAR TOXOCARIASIS**

Ocular toxocariasis is caused by infestation with the toxocara canis more commonly known as the intestinal roundworm of dogs. It typically presents in healthy adults as a solid peripheral or posterior pole granuloma with associated traction bands resulting in disc drag (Fig 13a, b), vascular distortion, macular heterotopias, retinal stress lines and even retinal detachment. All these findings can be seen in the absence of uveitis. A much less common presentation as chronic endophthalmitis in a younger age group.

**OCULAR GNATHOSTOMIASIS**

Gnathostoma spinigerum- unusual intraocular worm presenting as anterior uveitis with secondary glaucoma, subretinal track with adjacent vasculitis (Fig 14b), in a 44 year old male. Repeat examination 1 week later showed the presence of worm on the iris and iris holes (Fig 14a).

**OCULAR DIROFILARIASIS**

Intraretinal dirofilarial worm with diffuse retinal pigmeny changes (Fig 15a, b)-(Fundus photo courtesy Dr Biju Raju.)

**INTRA OCULAR CYSTICERCOSIS**

Cysticercosis is caused by the pork tapeworm, it is a relatively rare infection, and presents as a cystic lesion inside the eye (Fig 16). Ultrasound demonstrates a cystic lesion with central
hyperechoic, highly reflective scolex. Surgical removal is the treatment of choice. Here we can see a subretinal cysticercosis pre and post op pictures and USG. CT scan showed that there were cerebral cysticercosis as well. Always look for neurocysticercosis in cases of ocular cysticercosis.

48 year old male patient of alcoholic liver disease and suspected hepatocellular carcinoma presented with diminution of vision both eyes. Fundus examination showed vitreous exudation with foci of retinitis in both eyes (Fig 18a,b). He was diagnosed to have metastatic endophthalmitis. Vitreous biopsy culture was positive for Candida.

MUCORMYCOSIS

Mucormycosis- in an HIV positive Manipuri lady who later succumbed to infection. (Fig 19)

VIRAL DISEASES

CMV RETINITIS

43 year old male, known HIV positive with low CD 4+ counts who presented with floaters. Fundus examination showed whitish area of retinal opacification with adjacent haemorrhage along the retinal blood vessels(Fig 20a,b) suggestive of CMV infection.
The same patient after treatment with intravenous gancyclovir-inactive CMV retinitis characterized by decreased haemorrhages, sheathing of vessels and a more granular appearance (Fig 20 c,d)

In the absence of treatment, these lesions show a relentless extension along the retinal blood vessels with eventual involvement of the optic nerve head.

**ACUTE RETINAL NECROSIS**

Acute retinal necrosis (Fig 21a,b,c) typically affects healthy individuals and is characterized by retinal necrotic infiltrates with periarteritis which begin in the retinal periphery and eventually result in full thickness retinal necrosis. These may be accompanied by vitritis, retinal haemorrhages and optic disc edema. The posterior pole is involved in late stages.

This 45 year old gentleman came with history of lump in the throat, fever and diminution of vision. Biopsy from the neck revealed lymphocytic infiltration, further investigations proved that he had systemic CMV infection (Fig 22)