# Eales: An Disease Overview

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### *Introduction*

Eales Disease is an idiopathic peripheral retinal vasculopathy characterised by inflammation ,ischemia,retinal neovascularisation and hallmark is recurrent vitreous hemorrhage.

### *Epedemiology*

Eales disease has been reported predominantly in india although it has been found in Europe and North America.The incidence in india is 1/200.Commonly it occurs in male in their second decade.

### *Risk factor*

The disease has been related to tuberculosis exposure and hypersensitivity to tuberculo protein,It has been linked to stroke, demyelination,intranuclear ophthalmoplegia, haematological abnormality, vestibule auditory dysfunction and focal sepsis.

### *General pathology*

Eales disease was described as combination of recurrent vitreous hemorrhage in young men with constipation,epistaxis and headache. Basic pathology includes retinal periphlebitis,ischemia and neovascularisation. Peripheral retinal vessels first become inflamed and sheathed and then become occluded.Loss of perfusion leads to retinal neovascularisation that causes recurrent vitreous haemorrhages. It is a bilateral disease in 90% cases though symptoms present unilaterally.

### *Pathophysiology*

Infiltration of Tcell lymphocytic infiltration hasbeen found in epiretinal and subretinal membrane.There is association of retinal s antigen ,HLA B5,DR1,DR4 of patients with autoimmune disease.

### *Clinical picture*

Hallmark sign of Eales disease are retinal phlebitis,peripheral nonperfusion and retinal neovascularisation.

### *Retinal phlebitis*

Retinal periphlebitis is characterised by mid peripheral venous dilatation,perivascular exudates along the peripheral vein,superficial retinal hemorrhage.Vascular sheathing ranges from thin white lines limiting the blood column on both sides to segmental heavy exudates sheathing.

### *PERIPHERAL NONPERFUSION*

Fine solid white lines representing the remains of obliterated large vessels are commonly observed in the area of nonperfusion.These fine lines retain normal configuration of retinal vasculature.The junction between anterior peripheral nonperfusion and posterior perfused retina is usually sharply demarcated.The vascular abnormality at the junction between perfused and nonperfused area include microaneurysm,vevo venous shunt,venous beading occasional ly hard exudates and cotton wool spots.

### *Neovascularisation*

Neovascularisation of retina occurs in up to 80% cases.New vessels form either on the disc(NVD) or elsewhere(NVE) on the retina. NVE usually located at the junction of perfused and nonperfused retina.Bleeding from neovascularisation is common and usually recurrent and is major cause of vision loss. In recurrent bleeding fundus will show evidence of old blood with signs of fibrous organisation, retinitis proliferans or tractional retinal detachment. Untoward sequel includes

uveitis,complicated cataract,rubeosis iridis and secondary neovascular glaucoma in the later stage of disease.

### *FFA FEATURE*

| | | | | |
|---|---|---|---|---|
| Angiopathy | Grade1 | Gradell | Gradelll | GradelV |
| Venous changes | 1/12 | <2/12 | <3/12 | >3/12 |
| Retinal hemorrhages | <1/12 | <2/12 | <3/12 | >3/12 |
| Proliferative retinopathy | ..... | | | |
| New vessels | <1/12 | <1/12 | <2/12 | >2/12 |
| Fibroustissue | <2/12 | <2/12 | <3/12 | >3/12 |
| Vitreous hge | | <4/12 | <8/12 | >8/12 |

Active vasculitis is characterised by staining of vessel wall or frank extravasation.Vascular sheathing due to gliosis without active inflammation does not stain.Inflammation of venous segment causes obstruction to venous flow that causes venous engorgement and tortuosity distal to obstruction.Newly formed blood vessels become distinctly outlined during arteriovenous phase with their abnormal branching patterns and dye leak.Dye leakage and later dye staining stop after resolution of venous inflammation.

### *Treatment:*

A. Corticosreroid is main form of treatment.Oral and periocular corticosteroids are used to control retinal vasculitis.

B. Antitubercular treatment(ATT)

C. Anti-VEGF therapy

D. Photocoagulation

E. Vitrectomy

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