EVALUATION OF PATIENTS WITH EALES' DISEASE AT SHREE BIRENDRA HOSPITAL

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Abstract: Six patients with documented Eales' disease followed-up at Shree Birendra Hospital for a period of three years were investigated. The association of Eales' disease with tuberculoprotein hypersensitivity was confirmed in all six cases.

Introduction

In 1880 Henry Eales, an ophthalmologist described a syndrome of recurrent vitreous haemorrhages in young men with epistaxis and constipation.[1] He termed this new entity primary recurrent retinal haemorrhage. In 1887, Wadsworth described associated inflammation and neovascularization with this disease.[2] Since then, numerous authors have grouped periphlebitis retinae and idiopathic recurrent vitreous haemorrhages with or without retinal perivasculitis under the term Eales' disease. Duke - Elder thought that Eales' disease represented the clinical manifestation of many diseases.[3] Refined diagnostic tests have demonstrated that many of the so-called idiopathic haemorrhages are the result of diseases with known causes, such as sarcoidosis, systemic lupus erythematosus, diabetes mellitus, scikle-cell disease and collagen vascular diseases. However, after elimination of these causes, a group of patients remains with idiopathic peripheral non perfusion and perivasculitis of the retina. Many investigators now agree that Eales' disease is a distinct entity comprising characteristic funduscopic and fluorescent angiographic features. Although this disease has been called periphlebitis retinae, emphasizing the abnormalities of retinal venules, evidence suggests that the inflammation in this disease affects both arterioles and venules.

Methods

Six patients with Eales' disease confirmed by ocular examination and followed-up at Shree Birendra Hospital underwent extensive medical and ophthalmologic examination. Ophthalmologic evaluation included determination of visual acuity, applanation tonometry, silt-lamp biomicroscopic examination of the anterior segment, and direct and indirect ophthalmoscopy, including examination of the posterior and peripheral retinal with a Goldmann three-mirror contact lens after pupillary dilatation. The investigations done were CBC including ESR, BT, CT, Prothrombin time, Platelet Count, Blood Sugar Fasting and Post Prandial, Rheumatoid Factor, LE Cell, Antinuclear antibody, VDRL, FTAbs, Chest X-ray, Mantoux test and Fundus Fluorescein Angiography.

Results

Patient Profile

The results of examination of eyes of 6 patients with documented Eales' disease were included in the study. Patients with unilateral or bilateral idiopathic obliterative peripheral retinal vasculopathy were considered for inclusion in the study and all patients with a known and definite cause for retinal vascular disease (eg. branch vein occlusion, diabetes, sarcoidosis, identifiable collagen vascular disease, systemic lupus erythematosus, sickle-cell haemoglobinopathies) were excluded. Included in this study were six young male soldiers ranging in age from 21 to 30 years.

Ocular Involvement

Four of the six patients presented with abrupt painless unilateral blurring of vision with a visual acuity ranging from 2/60 to 6/60. The other two patients complained of painless unilateral diminished visual acuity with floaters. Their visual acuity being 6/24 and 6/18. All the patients developed visual symptoms in the other eye a few months later.

The earliest signs of ocular involvement was nonperfusion of the capillaries of the peripheral retina. With progression of the disease retinal capillary closure extended contiguously in a posterior direction. Sheathing of peripheral retinal vessels suggested perivascular inflammation. With increasing retinal nonperfusion and ischemia, neovascularization appeared at the junction of perfused and nonperfused retina.

Vitreous haemorrhage was the cause of most of the diminished visual acuity. Two of the cases with mi vitreous haemorrhage improved with time. The neovascularization in some of the eyes was treated wi peripheral scatter photocoagulation. Three patients ultimately became uniocular blind. One due to capilla closure that extended into the macula, the other due to tractional retinal detachment with unsuccessful retin reattachment surgery and the third due to anterior segment neovascularization with secondary glaucoma. O patient had retinal phlebitis with central vein occlusion of left eye. This patient had active pulmona tuberculosis. His visual acuity at the time of presentation was 6/60 the anti tubercular treatment institute resulted in a dramatic recovery. There was marked clearing of the retinal haemorrhages and venous engorgemen After three months the visual acuity improved to 6/12 with almost complete resolution of the retinal change

Association with Tuberculosis

Previous investigations have emphasized the relation between Eales' disease and tuberculo prote hypersensitivity. In this study two patients had evidence of previously undiagnosed active pulmona tuberculosis. The Mantoux test was positive in all the six cases. Chest radiographs were normal, with t exception of the x-rays of the patients with active pulmonary tuberculosis.

Discussion

There is a belief in a group of patients with idiopathic retinal vasculopathy with or without vitreal haemorra who have the clinical entity of Eales' disease. It is not clear whether all these patients have the sam pathophysiologic process or simply manifest a final common stage of many different etiologic entities. Not a patients with retinal vascular inflammation and nonperfusion, however, have Eales' disease. Some of the patients clearly have a systemic disorder or identifiable vasculopathy (such as diabetes, sarcoidosis, identifiab collagen vascular disease, systemic lupus erythematosus, sickle-cell haemoglobinopathies), not Eales' disea However one can rule out these conditions with a careful history and appropriate laboratory tests.

Every patients with Eales' disease should have a PPD placed. Many laboratory and clinical studies ha pointed to a relationship between retinal vasculitis and systemic tuberculosis. Axenfeld and Stock 1911, initiall suggested this association. Furthermore, some ophthalmologist treat patients with Eales' disease with systemi steroids, and patients on these medicines with a positive PPD should have concomitant anti tuberculos therapy. Gilbert demonstrated histopathologically tubercle bacilli around a retinal vein in a case of retin perivasculitis. In 1924, Finnoff was able to produce a perivasculitis in rabbit eyes after injecting tuberculos bacilli into a common carotid artery.[4] In 1940, Verhoeff and Simpson demonstrated histopathologically tuberculosis granuloma within the central vein of an eye presenting with central retinal vein occlusion. T other eye of the same patients had presented earlier with a retinal periphlebitis.[5] Further evidence of a li between tuberculosis and retinal phlebitis was presented by Elliott.[6] Lyle and Wybar emphasized that retin phlebitis can affect the central retinal vein as well as peripheral venules.[7] The mechanism by which acti systemic tuberculosis could cause retinal vasculitis remains unsettled.

Key words: Eales' disease, periphlebitis retinae, tuberculoprotein hypersensitivity, vasculitis.

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