

In the case reported, thrombosis of the central retinal vein may have been related to the vascular abnormality, or to the effects of chronic hypertension or both. Alternatively it may have been a coincidental event. However, CRVO is uncommon in young adults, and when it does occur it is commonly mild with good visual recovery. This makes this coincidence an unlikely event. In this patient, associated arterial abnormality may have contributed to the severity of vascular decompensation.

Pathophysiological factors that contribute to central retinal vein thrombosis include inflammatory or degenerative disease of the venous wall, compression by an adjacent sclerotic artery, haemodynamic alterations predisposing to stagnation and clotting abnormalities. Arteriosclerosis and hypertension can affect any of these factors, and indeed they are the most common systemic associations of CRVO. Vascular developmental anomalies can also promote thrombosis further. Walters and Spalton,¹⁷ in a study of 17 young patients with CRVO, suggested that inflammation of the venous wall is an unlikely cause and that an alternative explanation for the development of CRVO in young patients is a congenital anomaly of the central retinal vein.

In conclusion, a rare case of CRVO and retinal arteriolar tortuosity associated with coarctation of the aorta is reported. The vascular tortuosity persisted 4 years after successful treatment.

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Sir,

Pseudohypopyon due to Synchrony Scintillans

Synchrony scintillans (cholesterolosis bulbi) is a rare ocular condition in which cholesterol crystals form in the vitreous, usually following a traumatic vitreous haemorrhage. The clinical appearance is of multiple crystalline flakes, golden or coloured when illuminated, in a fluid vitreous. Most of the crystals settle in the lower vitreous, to be briefly resuspended when the eye moves. If the integrity of the lens or zonule is disrupted, vitreous with synchrony scintillans may enter the anterior chamber and present a striking appearance. In the case documented here, many of the crystals settled to the bottom of the anterior chamber, giving the appearance of a pseudohypopyon.

Case Report

A woman with Down's syndrome (trisomy 21), had a history of self-injury, in that she had habitually punched her own eyes since childhood. At age 24 years she was found to have early cataracts, but no other ocular abnormality. At age 30 years vision had deteriorated significantly, and a dense cataract was removed from the left eye. Because of posterior synechiae and calcification of the lens capsule, the procedure was technically difficult, resulting in the complete removal of the lens.

Post-operatively, the anterior chamber was found to contain vitreous, with a suspension of crystalline bodies which sparkled pink, green and gold in the slit-lamp beam (Fig. 1). Some of the crystals remained suspended, but most settled to the bottom of the anterior chamber, forming a 2 mm pseudohypopyon. With saccadic eye movements, many crystals became resuspended, and swirled for few seconds before resettling.



Fig. 1. The eye with synchysis scintillans in the anterior chamber, 1 week after cataract extraction. Note the pseudohypopyon made up of cholesterol crystals, and the colours reflected by those crystals which remain suspended in the vitreous.

The vitreous cavity itself showed a similar appearance, with most of the crystals settling inferiorly. There was minimal evidence of post-operative uveitis, and intraocular pressure was normal.

The intraocular crystals did not prevent a good view of the retina, which was completely detached with advanced proliferative vitreoretinopathy. Retinal reattachment was not felt to be indicated and, when last reviewed 2 months later, the appearance of the eye was unchanged.

Discussion

Post-traumatic cholesterolosis bulbi may take many forms. Cholesterol crystals may present in the anterior chamber alone following a hyphaema, or in the vitreous as a late sequel of vitreous haemorrhage. Cholesterolosis of the subretinal space may follow a subretinal haemorrhage.¹

Eagle and Yanoff² reviewed the literature on anterior chamber cholesterolosis, and found that most of the eyes were blind from old trauma. More than half of patients had raised intraocular pressure, though this was attributed in most cases to rubeosis iridis and chronic angle closure. We are unaware of any case in which cholesterol crystals were found to be actually blocking the trabecular meshwork and directly affecting the intraocular pressure, though a 1937 report described a clinical case in which 'glaucoma was controlled by removal of the material from the anterior chamber'.³

We can find only one case in which anterior chamber cholesterolosis itself was blamed for a severe inflammatory reaction,⁴ and even here, supporting evidence for a direct effect is lacking. These eyes may of course become inflamed via other pathological processes, related to the original trauma. In the presence of a pseudohypopyon, assessment of inflammatory signs may be more difficult, and misdiagnosis by non-ophthalmologists may result in an emergency referral. In our own case, the patient's general practitioner was rightly concerned that the patient had a post-operative endophthalmitis.

Synchysis scintillans of the anterior chamber is an unusual and interesting condition. It is seen in eyes which have suffered previous physical insult, and is often associated with other pathological mechanisms which may threaten the function or comfort of the eye. Though synchysis scintillans itself appears to be relatively benign, its presence should direct the clinician to assess the eye fully with regard to other sequelae of ocular trauma.

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