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Nodular Anterior Scleritis Associated with Ocular Trauma

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ABSTRACT

It is accepted that ocular trauma may be the precipitant of non-infectious uveitis. We report the case of a patient who developed unilateral nodular anterior scleritis following ocular trauma. Infection and systemic inflammatory diseases were excluded as causes of the inflammation. Our observations suggest the possibility that ocular trauma may act as a trigger for scleritis.

Keywords: scleritis, ocular trauma

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INTRODUCTION

Although scleritis may occur in the absence of apparent etiological factors, a systemic condition, most commonly connective tissue disease or herpes zoster ophthalmicus, is associated in half of cases[1]. Scleritis is also a well-recognised, but rare complication of ocular surgery[2]. We describe a case of unilateral nodular anterior scleritis which followed minor non-surgical ocular trauma.

CASE

A 24-year-old Bangladeshi construction worker presented complaining of left ocular pain and redness of two weeks duration. The patient reported that one month previously, he had suffered a self-limiting foreign body injury to the left eye while welding. There was no medical history.

Visual acuities were right 20/20 and left 20/25. Several granulomata at 3 o'clock on the left bulbar conjunctiva adjacent to the limbus suggested a healed conjunctival laceration. Between 1 and 5 o'clock, the anterior sclera was swollen with markedly congested vessels. Adjacent corneal stroma was edematous, with infiltrate and haemorrhage from deep vessels, and the aqueous contained small numbers of cells. No foreign body was seen. Right ocular examination was unremarkable.

Left scleritis was diagnosed. Due to suspicion of an infectious etiology, treatment was instituted with topical ciprofloxacin 0.30% hourly and oral ciprofloxacin and ketoconazole, as well as oral flurbiprofen. No micro-organisms were isolated from a conjunctival swab.

Although the condition initially stabilised four days later, the patient reported increasing pain, consistent with extending scleral inflammation and development of a nodule at 1 o'clock. Stains and cultures of biopsied sclera detected no bacteria, including mycobacteria, or fungi. C-reactive protein, rheumatoid factor, anti-nuclear, anti-double stranded DNA and anti-neutrophil cytoplasmic antibodies, and syphilis serology were either normal or negative. The Mantoux response measured 18 mm with blistering. In view of negative microbiological investigations, normal chest X-ray and systemic examination, and high endemicity of tuberculosis in the patient's homeland, his reaction was considered unrelated to the scleritis.

Oral prednisolone, 80 mg daily for one week, and prophylactic anti-tuberculous treatment were administered. Clinical improvement was apparent within 48 hours, with marked reduction of scleral edema and congestion. Prednisolone was tapered over two months without recurrence of the inflammation.

DISCUSSION

We conclude that ocular trauma was a likely precipitating factor in this case of scleritis. The temporal relationship and inflammation at the site of trauma strongly suggest the association. Infection, particularly tuberculosis, was excluded, and there was no evidence of systemic inflammatory disease.

Trauma has been cited as both a trigger and an aggravating factor for systemic inflammatory diseases including rheumatoid arthritis and psoriasis[3]. Sympathetic ophthalmia is a dreaded complication of penetrating eye injury[4], and recently, even trivial blunt trauma has been recognised as a precipitant of uveitis[5]. An association between ocular surgery, particularly multiple procedures, and necrotising scleritis is established[2]. However, we are not aware of previous reports linking non-necrotising scleritis and accidental trauma.

The mechanism by which injury might initiate scleritis remains uncertain. When necrotising scleral

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inflammation follows ocular surgery, disturbance of the vasculature and local ischaemia may be important[2]. In the case we report, the preceding trauma was mild, without clinical vascular damage. Clinical findings were indistinguishable from those of non-necrotising "idiopathic" scleritis or scleritis associated with systemic disease, and presumably the same pro-inflammatory cells and molecules were involved. As highlighted elsewhere[5], trauma may release critical mediators which trigger an abnormal positive feedback inflammatory loop in persons predisposed to ocular inflammation.

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