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Sir,

Posterior scleritis mimicking orbital cellulitis

Posterior scleritis is a common cause of diagnostic confusion because of its variable clinical signs and symptoms.[1] We discuss a case of posterior scleritis that presented with classical signs and symptoms of orbital cellulitis. To our knowledge this clinical picture has not been previously reported.

Case report

A 63-year-old lady presented with a 7-day history of a painful, red left eye and periorbital oedema. There was no history of trauma, precipitating lid lesions or sinusitis. On examination her visual acuity was 6/9 OD, 6/12 OS. She had periorbital oedema and conjunctival injection with chemosis (Figure 1). Left eye movements were restricted horizontally and on downgaze. There was no proptosis. Intraocular pressures and fundal examination were normal, and she was apyrexial. A clinical diagnosis of orbital cellulitis was made and sinusitis was excluded by the otolaryngologist. The patient was admitted and started on intravenous antibiotics.

The following day the chemosis had worsened and the anterior chamber was shallow. Intraocular pressure was 25 mmHg OS and fundal examination showed 360° choroidal effusions. Ultrasound scan showed a choroidal ring detachment and scleral thickening posteriorly (Figure 2). The diagnosis was revised to one of posterior scleritis and the patient was started on oral anti-inflammatories, topical steroids, and mydriatics. Within 24 h the lid oedema and conjunctival chemosis resolved. Systemic investigations showed no abnormality.

Figure 1 Left eye showing upper lid erythema, oedema, and conjunctival injection with chemosis.

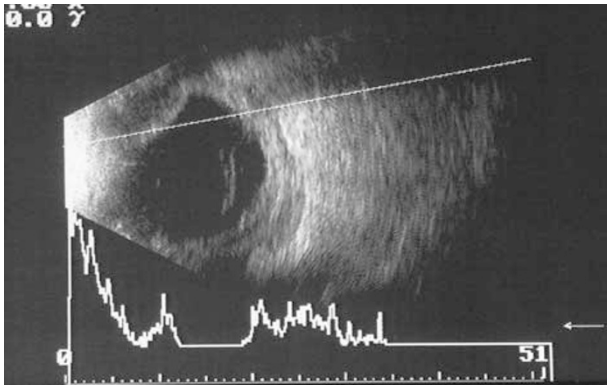

Figure 2 Ultrasound scan showing posterior scleral thickening and an anterior choroidal detachment.

Comment

Orbital cellulitis and posterior scleritis are both potentially life-threatening conditions that require urgent management. The patient described appeared clinically to have orbital cellulitis but was apyrexial with no obvious infective source. This illustrates that caution should be exercised when making a diagnosis of orbital infection in the absence of any obvious cause for or indicators of infection.

Posterior scleritis often presents a diagnostic challenge as it can frequently mimic other pathologies[1–5] and is almost certainly an underdiagnosed condition. It is commonly misdiagnosed because the presenting signs and symptoms are determined by the location and severity of the inflammation and its relationship to surrounding structures.[5] The inflammation appears to have spread anteriorly, involving the upper lid structures causing lid swelling and simulating cellulitis.

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References

1 Benson WE, Shields JA, Tasman W, Crandall AS. Posterior scleritis. A cause of diagnostic confusion. *Arch Ophthalmol* 1979; **97**(8): 1482–1486.
2 Dodds EM, Lowder CY, Barnhorst DA, Lavertu P, Caravella LP, White DE. Posterior scleritis with annular ciliochoroidal detachment. *Am J Ophthalmol* 1995; **120**: 677–679.

3 Quinlan MP, Hitchings RA. Angle-closure glaucoma secondary to posterior scleritis. *Br J Ophthalmol* 1978; **62**: 330–335.
4 McCluskey PJ, Watson PG, Lightman S, Haybitle J, Restori M, Branlay M. Clinical features, systemic associations, and outcome in a large series of patients. *Ophthalmology* 1999; **106**(12): 2380–2386.
5 Benson WE. Posterior scleritis. *Surv Ophthalmol* 1988; **32**: 297–316.

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Sir,

Endoscopic visualization to aid deep anterior lamellar keratoplasty

We welcome the interesting work by Drs Moore *et al* [1]and want to applaud the authors for their novel ideas. However, we must take exception to the statement that 'an adequate air bubble is not commonly seen'. Both Dr Anwar and myself are able to produce the 'big bubble' consistently: in 40–60% of eyes on the first try, in 80–90% of eyes on the aggregate of the first two tries, and in nearly all eyes when various additional manoeuvers (such as additional air injections, or preliminary anterior keratectomy followed by fluid injection and further air injection) are used in the initially resistant cases.

We maintain that the 'Big Bubble' technique[2,3] is the most efficient way of performing maximum depth lamellar keratoplasty—an essential feature of which is the baring of the host's Descemet's membrane over the central region of the cornea. Two prerequisites for successful use of the 'Big Bubble' technique are

(a) that a 'big bubble' has actually been generated, and
(b) that the surgeon is aware of this fact.

Success of (a) depends on close observation of several details, several of which were ignored in this experimental work:

(1) In their paper, Dr. Moore and coauthors do not mention that they trephined the cornea prior to injecting the air. This important first step of the 'Big Bubble' technique serves to 'isolate' the central cornea (to a large extent) from the peripheral cornea. Failure to perform this step may aid excessive spread of air into the corneal periphery, to the trabecular meshwork and into the anterior chamber instead of deep spread towards Descemet's membrane.
(2) The authors made an opening into the eye (to insert the endoscope) *before* injecting the air. Again, this would facilitate air entry into the anterior chamber. Air inside the anterior chamber directly competes for space with the 'big bubble'. The more air that is present in the anterior chamber (and the higher the pressure), the smaller will be the room available for the bubble of Descemet's detachment.
(3) A 26 gauge needle was used for injecting air into the cornea instead of a 27 or 30 gauge needle. (At this time, the relevance of this difference in technique is uncertain.)
(4) The force of the initial air injection may not have been sufficient.
(5) As the authors of the paper conceded, it is possible that cadaver eyes react different from live eyes. Further, it is conceivable that the pathological conditions for which this surgery is performed actually predispose these eyes to the formation of a central detachment of Descemet's membrane.
(6) Finally, we want to stress that here too, as in other skills, a certain learning curve is natural.
(7) Despite the differences in technique listed above, the authors did record the formation of several small bubbles of air between Descemet's membrane and deep stroma. Hence, it seems that some areas of detachment were generated, albeit not a confluent central region.

Regarding point (b) above, we diagnose a 'big bubble' by several characteristic features: the first indication is that the air (the blanching of the corneal stroma) spreads in a wave-like manner—like waves spreading over water when a drop falls on a calm surface—*in a circular fashion*. A completed bubble frequently exhibits a feathery white band at its (circular) periphery—offset, by a band of darker cornea, from the whitened region of air-insufflated stroma near the needle tip. (In some very rare cases, a 'big bubble' can be achieved without any air infiltrating/whitening the corneal stroma.) The anterior