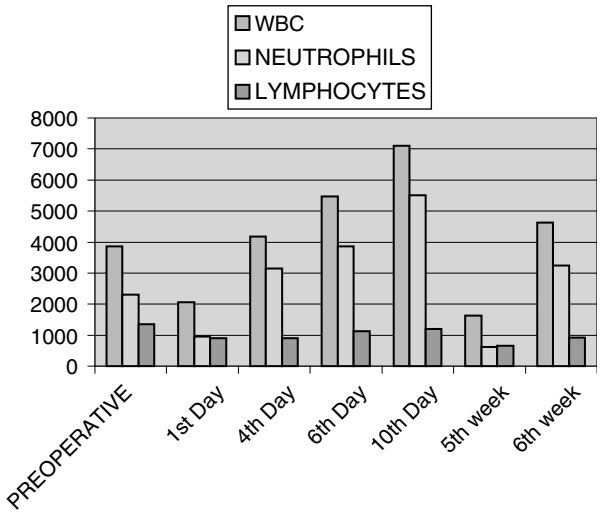

Figure 1 Evolution of white blood cell count (WBC). Changes in neutrophils and lymphocytes per cubic millimetre. Days are counted after diagnosis of endophthalmitis. On fifth week, gliclazide had been introduced for 2 weeks. It was discontinued the same day and 1 week later WBC count had partially recovered.

has been previously noticed with other drugs of the same family like tolbutamide in 0.18% of cases; in half of them, WBC count returned to normality without discontinuation of the drug.[3–6]

WBC are responsible for the inflammatory response to infection, neutrophils and lymphocytes being involved in the formation of hypopyon.[7] Neutropenia has been associated to a higher risk of surgical endophthalmitis.[8,9]

To our knowledge, this is the first case of antidiabetic drug-induced leukopenia related to endophthalmitis in a diabetic patient. Patients with low preoperative WBC counts on oral antidiabetic drugs undergoing eye surgery might need to discontinue these for insulin in order to improve their immunologic response to infection.

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Sir,

Traumatic and postoperative hyphaema in a patient with sickle cell trait

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Following traumatic hyphaema in sickle cell trait patients, the sickled red blood cells occupy the corneoscleral meshwork, juxtacanalicular connective tissue, and the inner wall of Schlemm's canal resulting in a resistance to aqueous outflow.[1]

This is thought to be a cause for a significant rise in intraocular pressure (IOP) that is frequently associated with sickle cell hyphaemas. A reported series of 99 patients with hyphaema found that 92% of sickle cell-positive patients had an IOP greater than 22 mmHg within 48 h of admission.[2] This compared to only 19% of sickle cell trait-negative patients.

We present a case with traumatic hyphaema and elevated IOP in a patient with sickle cell trait who failed to respond to medical therapy. Anterior chamber washout normalised the IOP despite resulting in a hyphaema similar in magnitude to the initial hyphaema caused by trauma.

Case report

A 15-year-old boy of apparent Caucasian origin presented to clinic accompanied by his mother 2 days after blunt injury to his left eye (LE). Examination of the LE revealed a visual acuity of 6/12, hyphaema <1 mm, and an IOP of 48 mmHg. No disc or retinal abnormalities were detected.

He was admitted, and oral acetazolamide, topical prednisolone 0.5% q.i.d., and topical timolol 0.5% b.d. were administered.

After 8 h, the IOP was 36 mmHg with no change in the hyphaema. At this point, further questioning revealed that his father was of Afro-Caribbean origin, raising the suspicion of sickle cell trait.

Haemoglobin electrophoresis was carried out and confirmed sickle cell trait. Acetazolamide, which can alter the pH of aqueous and hence precipitate sickling, was therefore withheld. Oral glycerol and intravenous mannitol was subsequently given with caution.

IOP remained high at 39mm Hg with no change in the level of hyphaema. Owing to the persistent raised pressure, an anterior chamber washout was carried out the following day (3rd day post-trauma). Since the hyphaema was small, balanced salt solution alone was used to irrigate the anterior chamber via a paracentesis. This was complicated by intraoperative bleeding with a resulting post-operative hyphaema of 1 mm.

Despite the hyphaema, his IOP remained <16 mmHg for a week following surgery. His visual acuity at 1 week was 6/12 LE and had improved to 6/6 at 1-month follow-up.

Comment

Patients of Afro-Caribbean and Mediterranean origin have a higher prevalence of sickle cell disease and this case emphasises the need to obtain a detailed history of ethnic origin from all patients with hyphaema. These patients tend to have higher IOPs that respond poorly to medical intervention. As acetazolamide is contraindicated and both glycerol and mannitol can precipitate sickling via dehydration, the options of medical treatment remain limited. Earlier diagnosis of sickle cell trait and aggressive IOP control is therefore crucial. We discuss the possible mechanisms for the rise in IOP.

Experimental studies showed that hyphaemas (both normal and sickle cell) occupying <25% of the anterior chamber did not show a significant change in aqueous outflow facility.[3] This does not explain the IOP rise associated with a small hyphaema seen in our case and suggests that there may be other factors involved in raising the IOP.

A study by Goldberg[4] shows that the percentage of sickling was higher in aqueous humour than in a control salt solution, suggesting that aqueous humour may have a deleterious effect on cells with a sickling propensity. On the other hand, it may be the initial sickling that makes the aqueous a non-favourable medium for its reversal to occur. Studies supporting this show that sickled erythrocytes injected into rabbit anterior chambers reduced the pH and pO_2 of the aqueous,[5] and hyperbaric oxygen administration reduced the percentage of sickling.[6] This may suggest a vicious circle of sickling, aqueous changes and further precipitation of sickling.

In our case, since the pre- and postoperative hyphaemas were similar, it suggests that the latter being in a more favourable medium (balance salt solution) prevented the initiation of the vicious circle, and hence IOP was normal postoperatively.

The chemical and erythrocyte changes that occur in the aqueous at the time of initial injury lead to a rise in IOP. This case provides evidence that these changes do not occur in a surgically induced hyphaema. Patients with sickle cell hyphaemas respond poorly to the limited medical options available and achieve poorer visual outcome compared to trait-negative patients.[2] We feel that early surgical intervention and understanding the exact sequence and nature of these changes is important as this may lead to efficient management with subsequent improvement of outcome in these patients.

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Sir,

Bilateral periocular swelling in Sweet's syndrome
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Sweet's syndrome is an uncommon skin disorder of unkown aetiology characterised by painful erythematous plaques, and associated with pyrexia, marked leucocytosis and elevated erythrocyte sedimentation rate (ESR). Although originally described as affecting the arms and legs, it has been reported on the head and neck. Periocular presentations are unusual and particularly associated with escharotic lesions. We report a case with marked bilateral periocular swelling.

Case report

A 72-year-old man developed bilateral periocular swelling overnight. He gave no history of facial trauma, sinus disease, or allergy. He denied diplopia, or visual loss. His past medical history included ischaemic heart disease for which he took oral diltiazem, bumetanide, elantan LA, aspirin, and sublingual glyceryl trinitrate. On admission, the patient was pyrexial with a temperature of 38.8°C and dyspnoeic with ankle swelling. He had gross bilateral periocular swelling and vesicular exudative lesions of the eyelids (Figure 1). There was no lymphadenopathy, or skin involvement other than the periocular area.

The corrected near vision was N8 right and left. Pupil reactions were brisk and normal. Motility was restricted particularly on the left and in all positions of gaze with mild proptosis. The anterior and posterior segments were healthy.

Clinically he was treated for probable Herpes Zoster cellulitis with secondary bacterial infection. In view of his pyrexia and likely cardiac failure, a medical opinion was sought and the following investigations were requested: ESR 100 mm/h (<20), elevated white cell count (WCC) 13.9 × 10/l (4–16), C-reactive protein 638 mg/l (<10), random glucose 25.6 mmol/l (4.5–5.6), urea 17 mg/dl (3–7), creatinine 172 μ/l (60–110), sodium 124 mmol/l (135–146), potassium 4.6 mmol/l (3.5–5.0), blood cultures that subsequently were negative and a chest X-ray (CXR) showed an enlarged heart and pulmonary oedema.

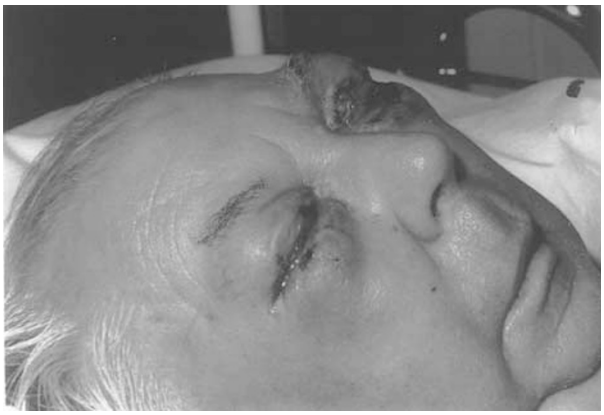

Figure 1 Bilateral periocular swelling with exudtive vesicles more marked on the left.

Intravenous insulin and frusemide were commenced to control his diabetes and heart failure. His cellulitis was treated with intravenous acyclovir and benzylpenicillin with flucloxacillin.

After 24 h, computed tomography (CT) was arranged of the orbits, paranasal sinuses, and brain in order to exclude orbital cellulitis, subperiosteal abscess, cavernous sinus thrombosis, or mucormycosis. The CT demonstrated only preseptal ocular and facial swelling (Figure 2).

Over the next 48 h the diffuse swelling subsided but the crusting became haemorrhagic and necrotic particularly over the left upper lid (Figure 3). A dermatology opinion suggested a dermatosis, and a skin biopsy was taken of the left upper lid.

Pathologic examination demonstrated that the epidermis was uninvolved but there was a dense polymorph infiltration of the entire thickness of the dermis (Figure 4). The blood vessels demonstrated swelling of the endothelial cells but no evidence of a true vasculitis (Figure 5, arrow). This suggested a diagnosis of Sweet's syndrome, or acute neutrophilic dermatosis.

He was commenced on oral prednisolone 30 mg daily and made a rapid recovery.

Comment

Sweet's syndrome, also known as acute neutrophilic dermatosis, was first described by Dr Robert D Sweet in 1964.[1] It is characterised by abrupt onset fever, painful erythematous skin papules and plaques, and