Reminder of important clinical lesson

A minor lid laceration concealing a potentially life-threatening injury

Lamis Abdelaziz,¹ Ramez Barbara,² David Scullion,¹ Edel Cosgrave,¹ Timothy Metcalfe¹¹Ophthalmology Department, Harrogate District Hospital, Harrogate, UK;²Ophthalmology Department, Bnai Zion Medical Centre, Haifa, Israel**Correspondence to** Dr Lamis Abdelaziz, lamisabdelaziz@doctors.org.uk**Summary**

A 29-year-old man presented with an injury to his left upper lid, having stumbled onto the tip of a pool cue. He presented with minimal signs and symptoms, initially thought as a minor lid laceration and was managed with lid suturing. Two months later, he presented with vertical diplopia on upward gaze and showed a picture consistent with traumatic Brown's syndrome. Subsequent imaging revealed the extent of orbital injury and confirmed a retained foreign body embedded in the frontal lobe of the brain. This was surgically removed and found to represent the tip of the pool cue. Postoperatively, the patient made a good recovery, although he still has a slight residual diplopia on upward gaze.

BACKGROUND

Our case is unusual as it presents a rare case of what appeared to be a minor injury concealing an otherwise potentially serious and life-threatening injury. It emphasises the importance of thorough assessment of patients with ocular injuries with penetrating objects, and highlights the need for considering appropriate radiological investigations early on in the presentation to minimise the risk of undesirable outcomes for the patient. Most reported cases^{1–12} of retained intracerebral foreign bodies re-presented with signs and symptoms related to the foreign body in the brain. Our case however, re-presented with signs and symptoms related to the damage of the extraocular muscle structures sustained during the injury.

CASE PRESENTATION

A 29-year-old man presented to the accident and emergency department with a left upper eyelid injury. Few hours before, he stumbled and fell directly onto the tip of the pool cue causing a laceration of the left upper eyelid. The patient recalled sudden severe pain and profuse bleeding at the time of injury. On attendance in casualty, visual acuity in the right eye was 6/5 and in the left eye was 6/18. The upper eyelid laceration was thought to be superficial, and was glued. Orbital x-rays were reported as normal. The patient was reassured, advised to take oral analgesia and referred to the eye clinic for further assessment and management.

The patient was seen in the eye clinic 3 days later. On examination, he had a partial-thickness 1.5 cm left upper lid superonasal laceration with upper eyelid swelling. The globe was intact. He reported pain and blurred vision. Unaided visual acuity was; 6/4 right and 6/18 left. There was a subconjunctival haemorrhage covering the inferior nasal sclera. The cornea was clear and the anterior chamber was quiet. Pupils were equal and normally reactive. Funduscopy was normal. It was thought that there was a slight skin loss and exposure of the subcutaneous tissues at the site of the laceration and therefore further surgical

repair was planned, which was done the following day under local anaesthetics. Five days postoperatively, his left visual acuity improved to 6/4 in both eyes. He had a left upper lid ptosis thought to be secondary to postoperative lid swelling.

Two months following the injury, the patient attended the eye clinic with vertical diplopia on upward gaze. Clinically he had a 2 mm left ptosis. Formal assessment of ocular movements showed restriction in dextro-elevation of the left eye, a pattern consistent with Traumatic Brown's syndrome. There was a compensatory head posture and a chin tilt to avoid diplopia in the primary position. The patient was otherwise well.

INVESTIGATIONS

An MRI scan of the orbits was requested to evaluate the trochlea apparatus, (figures 1 and 2). Surprisingly, this demonstrated disruption of the left superomedial orbital roof with intense oedema in the left frontal brain substance. Centrally within the oedema was a discoid area of signal void on all sequences. There was intense enhancement around this area but the discoid abnormality failed to enhance. These findings were interpreted as a foreign body consistent in size and shape with the tip of the pool cue. The enhancement was felt to represent adjacent active cerebritis. A CT scan was also performed in order to optimally evaluate the extent of the bony disruption of the orbital roof (figure 3) and this showed that the left superomedial orbital wall fracture was close to the site of insertion of the trochlear with trochlear avulsion.

TREATMENT

The patient was referred urgently to the neurosurgical team, who advised surgical removal of the foreign body. The patient underwent left frontal craniotomy and the pool cue tip was identified and removed 3 months following the injury. During the operation there was extensive inflammatory tissue in the left frontal lobe which was debrided and a large free bone fragment originating from the

Figure 1 Coronal T2 weighted MRI scan demonstrated oedema within the left frontal lobe of brain (asterisk). There is a central discoid area of black signal void (arrow) close to the left superomedial orbital margin.

Figure 2 Coronal T1 weighted MRI image with gadolinium enhancement. An intense area of circumferential enhancement is seen, reflecting reactive cerebritis (arrow). The central discoid area remains black.

orbital roof was dissected and removed. Postoperatively, he received prophylactic flucloxacillin and metronidazole

Figure 3 Coronal CT image on bony windows clearly demonstrates the injury to the orbital roof. The elevated bony fragment projects into adjacent bone. The discoid area can only be barely appreciated on CT as a subtle area of increased attenuation. This would be almost impossible to appreciate without the MRI images for comparison.

and pneumococcal vaccination. Postoperative CT angiography did not reveal any intracranial vascular injury. Six weeks later, the patient made a good recovery but he had some residual paraesthesia involving the distribution of the left supraorbital nerve which was expected to resolve in time. There were no reported symptoms indicating any neuropsychiatric sequelae.

OUTCOME AND FOLLOW-UP

Two months following surgery, slight diplopia was still present on elevation and dextro-elevation. There was a 1 mm residual ptosis. There was a compensatory head posture with a slight chin elevation and a head tilt to the right. Ocular movements demonstrated restricted elevation on right gaze with corresponding over action of the right inferior oblique muscle simulating a Brown's type pattern. No prism correction was required. Three months later ocular movements remained stable. The patient maintained his compensatory head posture and diplopia was only appreciated briefly on elevation and dextro-elevation. Fresnel prisms were suggested to alleviate the head posture but the patient declined this and he was discharged from the clinic.

DISCUSSION

Brown syndrome is an oculomotility disorder of the superior oblique tendon characterised by restriction of elevation in adduction.¹³ It is usually congenital but can be acquired as a result of inflammation or trauma.¹⁴ In our case, the most likely explanation for the traumatic Brown's syndrome is the trochlear avulsion secondary to the penetrating injury.

In this case, initially, there was a failure to recognise the extent of the injury because there were minimal signs and symptoms. There was a communication failure at the time of the lid surgical repair as the operating surgeon thought that the patient had a normal CT scan of the orbit when in fact he only had orbital x-rays reported as normal. Postoperatively, the patient had lid ptosis attributed to lid swelling and therefore was subsequently discharged. The patient remained well throughout this presentation and the MRI findings were a surprise to everyone involved in his care. Luckily the foreign body in the brain was eventually

removed with no major complications sustained and the outcomes were satisfactory at the end, of an otherwise potentially life threatening presentation.

Cases of intracranial and/or orbital retained foreign bodies, initially presenting as minor eyelid lacerations, have been reported.^{1–12} The more serious injuries were often missed at presentation because they were thought as trivial lid injuries, appearing with minimal signs and symptoms, thus thorough assessment and investigations were often delayed and many cases were initially managed with lid suturing.^{1–12} The bone of the orbital roof is thin, can be easily fractured and is vulnerable in any penetrating lid injury inflicted in an upward direction and it provides an easy entry route into the brain.^{1–3} The transorbital entry route is a well-recognised route into the frontal lobe.¹⁵ Freeman used this approach for thousands of transorbital prefrontal lobotomies as a form of psychosurgery.¹⁵

Aulino *et al*⁶ described a case of a 35-year-old man presenting with a generalised seizure. An MRI scan showed a left temporal lobe mass thought to be associated old blood products (hemosiderin) with surrounding oedema.³ Surgical removal revealed a wooden foreign body and on further questioning the patient remembered a fight 16 years previously where he received a blow to the orbit with a pool cue.³ Liu *et al*⁴ reported a case of an 18-month-old boy with a penetrating upper lid injury with a bamboo stick, managed initially as a superficial lid trauma.⁴ A month later he presented with headaches, severe orbital swelling, and an MRI scan confirmed a foreign body in the left frontal lobe within a localised brain abscess which was subsequently removed and the patient made a full recovery.⁴

Wood can be difficult to detect on plain x-ray films and CT scans as it has a similar density to air and fat and can be interpreted as such.^{5 6} Therefore, if a wooden foreign body is suspected following a periorbital trauma, an MRI scan is the investigation of choice,^{5 6} although it should not be used if a metallic foreign body is suspected.^{3 7} Lakshmanan *et al*⁶ reported a case of a 10-year-old boy with orbital cellulitis following a lid injury. CT orbit did not show any foreign bodies.⁵ Two days later, the symptoms quickly resolved following a spontaneous extrusion of a wooden foreign body through the upper lid.⁵ Prazere *et al*⁶ described three separate cases of orbital injuries with wooden objects. In all cases, there were retained orbital wooden foreign bodies not detected with CT scans but were evident on MRI scans.⁶

Shag and Decock² reported a case of a 2-year-old boy with an upper eyelid injury while playing with colour pencils. During the surgical lid repair, clear fluid was noted to be coming out through the lid wound, which was then analysed showing a β -tracer protein, suggestive of cerebrospinal fluid (CSF) leak.² Neuroimaging confirmed a retained foreign body in the right frontal lobe which was subsequently removed.² They recommend this laboratory test as a screening procedure for penetrating orbital injuries as it is simple, rapid and inexpensive.²

Early detection of these retained foreign bodies is paramount in preventing serious and life-threatening complications.^{1–12} Therefore, for any lid injury with a penetrating object, the clinician should have a high index of suspicion for retained orbital and/or intracerebral foreign bodies and a low threshold for imaging.^{1–12} A detailed and systematic assessment of the patient's presentation and

a thorough questioning of the mechanism of injury are essential to evaluate the nature and extent of injury.^{1–12}

Surgical removal of retained orbital and intracranial foreign bodies is almost universally recommended^{1–12} even if there are minimal signs and symptoms as the risks of leaving the foreign body in the orbit or the brain is greatly higher than the risks involved with the operation to remove the foreign body.^{1 3 5 6} Reported risks of these retained foreign bodies include; orbital cellulitis, epilepsy, permanent personality change, meningitis, hemiparesis, encephalitis, cerebral abscess, skull osteomyelitis, cerebral granuloma and death.^{1 3 5 6}

Our case is unusual as it presents a rare case of what appeared to be a minor injury concealing an otherwise potentially serious and life-threatening injury. It emphasises the importance of thorough assessment of patients with ocular injuries with penetrating objects, and highlights the need for considering appropriate radiological investigations early on in the presentation to minimise the risk of undesirable outcomes for the patient. Most reported cases^{1–12} of retained intracerebral foreign bodies re-presented with signs and symptoms related to the foreign body in the brain. Our case however, re-presented with signs and symptoms related to the damage of the extraocular muscle structures sustained during the injury.

Learning points

- Any case of penetrating injuries to the lids and/or orbit should be thoroughly assessed even if it appears to be minor, taking into account the nature, type and mechanism of injury.
- Always have high index of suspicion of retained foreign bodies and low threshold for radiological investigations.
- Appreciate the limitations of some radiological investigations in detecting intraorbital/intracerebral foreign bodies.
- Ask the patient about the offending object and enquire if it was whole and intact following the injury.

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Competing interests None.

Patient consent Obtained.

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