CASE REPORT

Posttraumatic delayed cranio-orbital cerebrospinal fluid leakage: Case report

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Summary A 56-year-old man sustained subarachnoid haemorrhage, skull base fracture and multiple facial fractures in a traffic accident. Two weeks later, the patient developed a subperiosteal fluid collection into the orbit of the right side presenting with a progressive proptosis and an increased intraocular pressure. We performed drainage of the fluid on the superior part of the right orbit, followed by a surgical reduction of the facial fractures. The patient had no exophthalmos any longer, whose intraocular pressure was normalised. In conclusion, our case indicates that careful monitoring of clinical signs and a follow-up radiography would be mandatory for patients with craniocerebral trauma despite a lack of the definite symptoms. Clinicians should consider the possibility that the cerebrospinal fluid (CSF) leakage into the orbit might occur in these patients.

The main cause of cerebrospinal fluid (CSF) leakage is a meningeal fistula, and it occurs the most commonly because of head trauma. In addition, it also occurs at an incidence of 0.5–2% in cases of cranial injury without a facial involvement; its incidence may be as high as 25% in cases of midfacial injury.1 Dural fistula and CSF leakage result in rhinorrhoea and otorrhoea. This is occasionally accompanied by the fluid accumulation. Cranio-orbital CSF leakage, however, is very rare. To date, only fewer than 20 cases have been reported.2,3

Here, we present a case of posttraumatic orbital CSF leakage that developed 2 weeks after the accident.

Case report

A 56-year-old man with an intubation tube was transferred to us from a district general hospital. The patient sustained multiple injuries including subarachnoid haemorrhage, haemoperitoneum, elbow fracture, facial fracture and lacerations.

[Further details of the case report are provided in the full text of the article.]
On examination, the patient had a Glasgow Coma Scale (GCS) of 3. Physical examination showed systolic/diastolic blood pressure 150/70 mmHg and pulse rate 130 beats min⁻¹. However, visual acuity and extraocular movements could not be checked because the patient was not arousable. There was no periorbital ecchymosis, conjunctival haemorrhage, papilloedema or proptosis. The right pupil was of normal size with a sluggish reaction to the light. The left pupil was dilated without responses. Mental status was stuporous. The patient underwent a computed tomography (CT) scan of the head. This revealed bilateral orbital fractures of the medial wall and roof, bilateral temporal bone fractures, left-side zygomatic tripod fractures presenting those of the zygomaticofrontal suture, zygomatic arch, infraorbital rim and pterygomaxillary buttress with a combined subarachnoid haemorrhage and skull base fractures (Figure 1). However, the patient had no bleeding or fluid leakage from the ear or nose.

The patient underwent exploratory laparotomy for the haemoperitoneum, followed by small bowel and mesenteric resection and an open reduction of the elbow fracture. Following the administration of prophylactic intravenous antibiotics, the patient was admitted to the department of neurosurgery of our medical institution for the conservative treatment of subarachnoid haemorrhage, skull base fractures and orbital fractures.

Two weeks later, the patient presented with progressive proptosis, chemosis and lagophthalmos of the right eye (Figure 2). On follow-up CT scans, the patient had a subperiosteal fluid collection on the superior part of the right orbit (Figure 3). On ophthalmologic evaluation, the intraocular pressure was relatively higher on the right side than the left side. Ocular mobility and visual acuity could not be checked because the patient had a stuporous mentality.

The patient underwent surgical decompression, in which we performed an aspiration of 5 ml of transparent fluid from the superior part of the right orbit using a 10-cc syringe with an 18-G needle (Figure 4). This was followed by an open reduction of the zygomatic tripod fracture and impure blow-out fracture of the left side, bilateral medial wall blow-out fracture. A body fluid analysis confirmed the presence of CSF.

On the second postoperative day, the patient no longer had exophthalmos and proptosis of the right eye. On CT scans, there was a decreased fluid collection in the superior part of the right orbit (Figure 5). Then, the patient further received the conservative treatments with anti-oedematous medications, which was followed by the continuous use of the prophylactic intravenous antibiotics and eye drops. At 1-year follow-up, as well as on ophthalmologic examinations, the patient had no recurrence of the orbital CSF leakage. In addition, the intraocular pressure, ocular mobility and visual acuity were all normal.

**Discussion**

CSF fluid leakage is a well-known complication of head trauma or cranial surgery, whose common symptoms include rhinorrhoea, otorrhoea or lachrymal secretion. However, posttraumatic orbital CSF leakage is a very rare condition. It is manifested by an intermittent serous discharge from the eye, an inferolateral displacement of the globe and pulsatile proptosis. Metrizamide CT
Cisternography confirms a communication between the orbital cyst and subarachnoid space. Biochemical analysis of the fluid, including glucose or β2-transferrin, is also useful. Other conditions may cause similar clinical signs, and these include retrobulbar and subperiosteal haematoma, orbital abscess, cellulitis, mucocele and foreign body cyst.

We present a case of posttraumatic orbital CSF leakage occurring 2 weeks after the accident. On initial CT scans, the patient had orbital and skull base fractures with a minimal size of the defect due to the orbital roof fractures. At initial evaluation, the patient presented with no specific symptoms. Two weeks following the onset of injury, however, the patient had a delayed development of the proptosis. There were no evidence demonstrating meningitis, cellulitis and other infections. The patient had a higher intraocular pressure due to the CSF accumulation within the orbit. This might lead to the occurrence of optic nerve injury. The accumulated CSF was drained. Thus, the ocular symptoms were normalised. With the surgical operation, the fluid was confirmed to be CSF.

As shown in our case, patients with skull base fracture, particularly including those with orbital roof fractures, are at risks of developing delayed CSF leakage into the orbit although they initially presented with no definite symptoms. In addition, after a few weeks following the onset of traumatic head injury, these patients might present with such symptoms as progressive exophthalmos and other ophthalmic ones including eyeball pain, discharge or swelling. If the CSF accumulated for long, this would eventually cause ocular complications including optic nerve compression. Based on postoperative outcome at 1-year follow-up, we think that the drainage of the CSF collection was a sufficient procedure to prevent further CSF leakage. In conclusion, our case indicates that careful monitoring of clinical signs and a follow-up of radiography would be mandatory for patients with craniocerebral trauma despite lack of the definite symptoms. Clinicians should consider the possibility that the CSF leakage into the orbit might occur in these patients.

Disclosure

The authors have no commercial associations or financial interests that might pose or create a conflict of interest with information presented in this article.

References