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Case Reports & Case Series

A rare case of depressed skull fractures at the anterior cranial fossa associated with communicating hydrocephalus resulting a progressive vision loss

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ABSTRACT

Although an open depressed skull fractures at the anterior cranial fossa is relatively common, comorbidity with a progressive vision loss at initial presentation, with acute communicating hydrocephalus is rare. Here we report a rare case of a progressive vision loss presented with communicating hydrocephalus following open depressed skull fractures at a mid-frontobasal. Fragments of skull were extracted and the dural tear was repaired. Within the first post-operative day, the patient already gained full consciousness and his headache was completely resolved. Unfortunately, the ophthalmologic *examinations revealed* his vision remains impaired. We discussed the pathological mechanisms of a progressive vision loss and acute communicating hydrocephalus in a patient who was diagnosed with traumatic open depressed skull fracture at a mid-frontobasal, with focus on surgical management of this intriguing case. This case makes a strong argument for an early neurosurgical intervention in frontobasal fractures.

1. Introduction

Depressed skull fractures in cases of traumatic brain Injury (TBI) are resulted from direct high-energy blows to a small surface area of the skull by a blunt object [1]. Patients with this diagnosis can be presented with various symptoms, depending on the area of the brain that becomes the focus of the impact [2]. According to Ponsford et al., common symptoms that are usually presented in patients following TBI are headaches, fatigue, drowsiness, excessive sleepiness, inability to concentrate, clouding of consciousness, hyperacusis and visual impairment [3,4]. Post-traumatic hydrocephalus (PTH) is a potentially significant sequelae of TBI [5]. Despite it is a treatable condition, it increases the rates of morbidity and mortality in TBI patients. The treatment of PTH is CSF diversion through ventricular shunting. Published reports acknowledged improvement in clinical outcome of patients after ventricular shunting, proven by imaging findings and neurophysiological functions [6,7]. An alternative mechanism of how hydrocephalus can occur in patients with TBI is through meningitis which can happened after fracture of cranial vault or basilar skull fracture [7,8]. Recently, a paper suggesting late neurosurgical intervention for frontal depressed skull fractures [9] that we will discuss further.

The mechanism that leads to a progressive vision loss and acute communicating hydrocephalus in traumatic open depressed skull fractures at mid-frontobasal is yet to be unrevealed. Herewith, we report our experience of treating a case of open depressed skull fractures at the anterior cranial fossa (mid-frontobasal) that is suspected to be associated with communicating hydrocephalus, accompanied with a progressive vision loss.

2. Case report

A 19-year-old male was admitted to our emergency unit with a progressive vision loss of his both eyes as the chief complain, accompanied with moderate intermittent headache. Three weeks prior to admission, the patient had a motorcycle accident, causing decrease of consciousness and vomiting. His medical history did not acknowledge any visual impairment prior to the accident. History of bleeding from nose, blurred vision and smelling disturbance were all denied. When initially admitted just after the accident, it was identified that he had an open skull fracture at mid-frontobasal (Fig. 1A), and had undergone local debridement that was performed by a general surgeon at another

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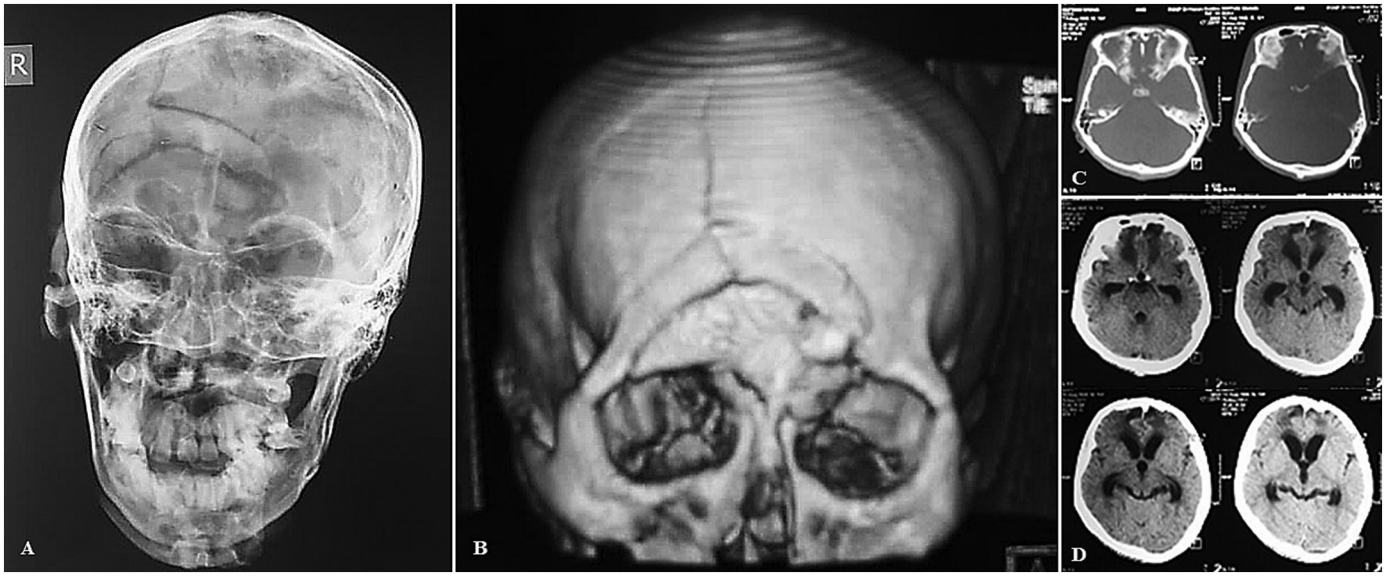

Fig. 1. Skull X-ray performed immediately after the accident shows multiple fracture lines at a mid-frontobasal area with double contour, suggesting depressed skull fractures (A). CT bone window and 3D reconstruction showed depressed fractures at a mid-frontobasal area greater than the thickness of the skull (B), occupying frontal sinus with multiple fracture lines and bone fragmentation (C). CT scan images taken 3 weeks after the accident, showed enlargement of all ventricles with periventricular hypodensity.

hospital. He was hospitalized for 17 days, and interestingly, developing gradual decrease of visual acuity. Five days prior to his admission, his vision kept worsened until he has no light perception any longer, thus he immediately referred to our centre. Upon arrival at our centre, he seemed confused (GCS 14), complained the occurrence of disturbing headache, despite the eyes' movement is normal to all directions. Further examinations on both eyes revealed that the pupils are round and equal in both eyes, with both pupils have diameter of 5 mm, with no light reflexes. Through visual acuity examination, the examiner confirmed that there was no light perception on both eyes. Ophthalmoscopy examination showed bilateral secondary optic atrophy. Other neurological tests on other cranial nerves, motor and sensory functions were all declared within normal limit.

A non-contrast head CT scan revealed fragments of the fractured skull, with depression and interlocking at mid-frontobasal, involving more than one lamina (Fig. 1B). These fractures were located on the frontal sinus (Fig. 1C). Furthermore, we identified a communicating hydrocephalus with periventricular hypodensity in both temporal horns, sized > 2 mm in width. Consequently, cerebral sulci, sylvian and interhemispheric fissure are all compressed (Fig. 1D). Contrast CT scan revealed meningeal enhancement and isohypodense mass at mid-frontobasal area with enhancement, which was suspected as an abscess (Fig. 2). After the initial evaluation, we performed craniectomy and debridement using full coronal incision. Skin flap was made by exposing frontal area to superior orbital rim, thus reaching nasofrontal junction and pericranium are preserved. After removing pericranium around fracture edges, we identified the depressed fracture at frontobasal bone sized 10 × 6 cm, with interlocking segments. Both sinus and air cells were all exposed, with pus accumulated on its surroundings. We performed the removal of all depressed-fragments of bones, pus, debris and air cells. The exposed duramater was white, tense and not intact, with lacerated area sized approximately 3 × 1 cm (Fig. 3).

We continued opening the duramater and acknowledged gliotic tissue with abscess formation with an approximate size of 3 × 1 cm at mid-frontobasal. 5 cc of pus was aspirated from the abscess and submitted for culture examination. As the policy at our centre permits an immediate administration of broad spectrum antibiotics after a patient being suspected of having brain abscess, we then gave the patient a triple drug combination, including intra-venous of Ceftriaxone, Metronidazole and Gentamycin. Afterwards, we performed craniectomy debridement and placed an external ventricular drain (EVD). CSF was submitted for further examination to dismiss any possibilities that the mass was caused by an abscess. Although the patient regained consciousness and the headache completely resolved, liquor cerebrospinal (LCS) diversion did not reverse his vision loss. One week after the surgery, the LCS culture was proven to be sterile, thus we decided to convert the EVD to ventriculo-peritoneal shunt (VP Shunt). Antibiotics therapy was administered for 6 weeks, and the patient was discharged in good condition, despite his visual was never reversed. After 3 months follow-up, the patient underwent cranioplasty to close the bone defect.

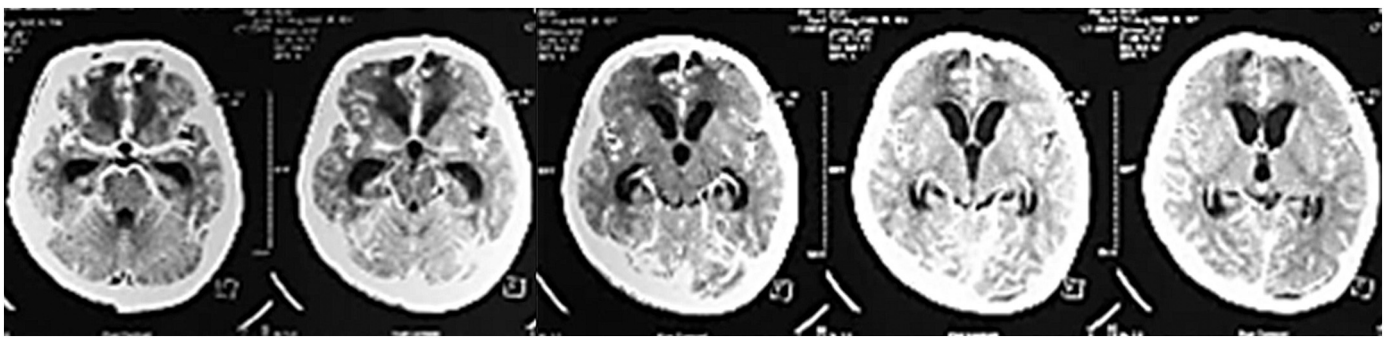

Fig. 2. Contrast CT images showed meningeal enhancement and isohypodense mass with enhancement at a mid-frontobasal.

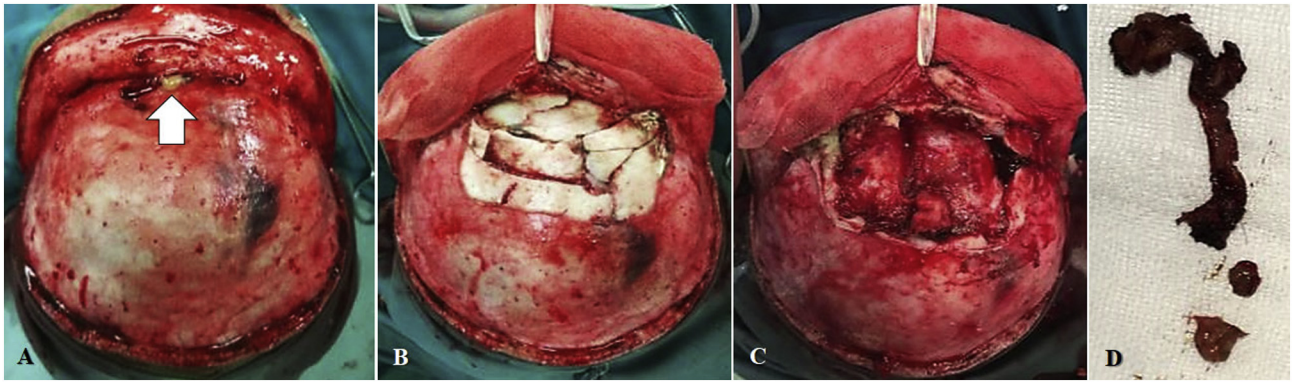

Fig. 3. Skin was flapped anteriorly after full coronal incision, and we identified pus accumulation (*arrow*) around the fractures' area (A). Pericranium around the fracture edges was then removed, thus the depressed fractures at frontal bone, frontal sinus and frontal air cells were all exposed, with pus accumulation surrounding and within them. Fragmented bones, pus and debris were all removed, along with air cells (B). After bone fragments were removed, we revealed laceration of duramater (C). Gliosis tissue mixed with abscess in mid- frontobasal sized 3 × 1 cm in width with 5 cc of pus were then identified (D).

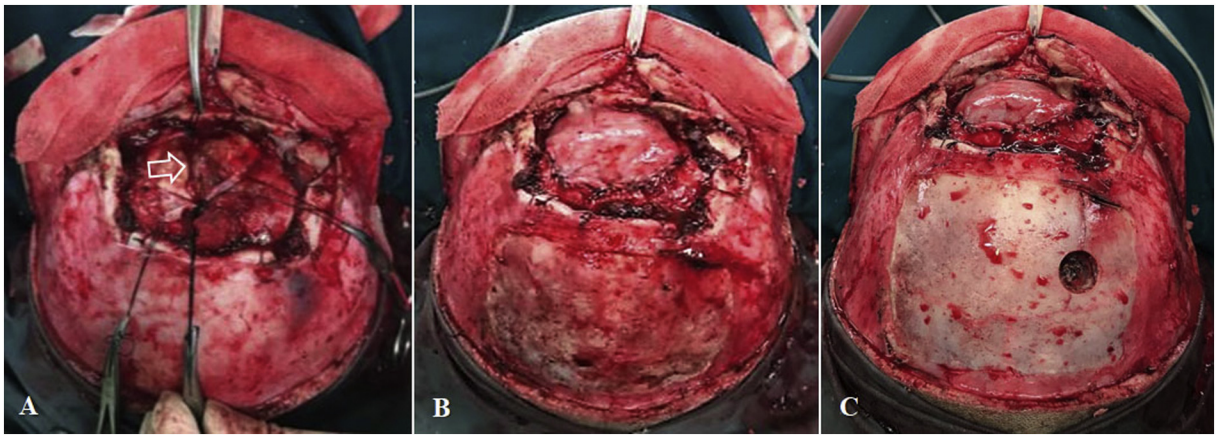

Fig. 4. Gliotic tissue with abscess formation were found at left superior frontal gyrus (*arrow*) (A). Duraplasty was performed using pericranial graft, followed by obliteration of frontal sinus ostium and cranialization using pericranial graft (B). The procedure was continued with EVD insertion at right Kocher point (C).

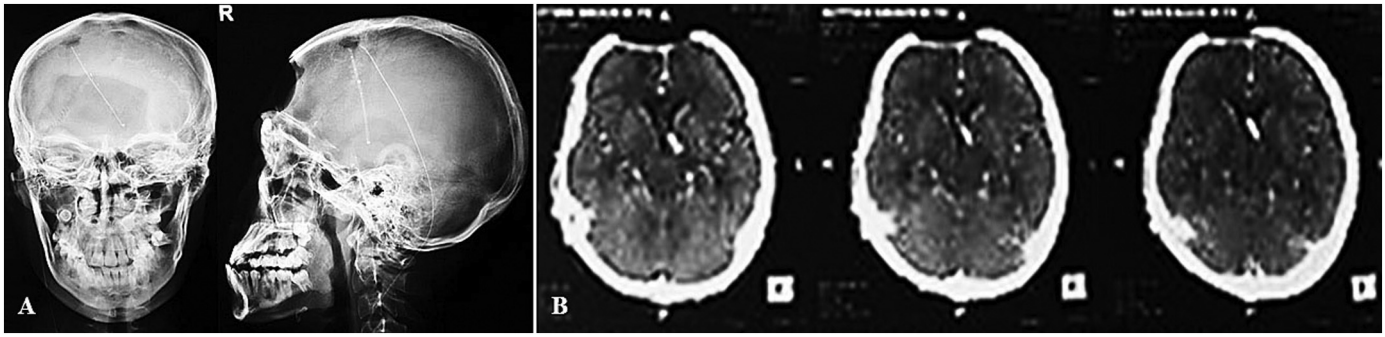

Fig. 5. Follow-up Skull X-ray (A) and contrast CT image (B) after VP shunting and 6-week administration of antibiotics. As seen on the images, there was no sign of hydrocephalus and no enhancement mass were identified. We also noted that the meningeal enhancement could not be acknowledged any longer when compared with the previous CT images.

3. Discussion

According to Neville, et al., in 2014, time to surgery (early surgery) does not seem to be crucial to improve prognosis of the patients with frontal depressed skull fractures, allowing surgical correction could be scheduled after patient's stabilization without compromising neurological recovery. Although 71.6% showed good recovery, half of these patients have smell and taste disturbance, which may significantly compromise the quality of life [9]. However, our case opposing the view of above paper that suggesting late neurosurgical intervention for frontal depressed skull fractures. In our case, if the definitive intervention perform earlier, the progression of visual loss could be avoid as well as the diffuse meningitis and the development of the hydrocephalus that most likely the cause of the blindness. Ideally a comprehensive thin slice CT scan of the skull base suggests to be performed in order to clarify the extent of the injury. Somehow, limited resources as well as national insurance for the treatment of such patients in our country are the true obstacle.

It is generally accepted that the inflammatory reaction, whether acute or chronic, and the ensuing fibrosis process impede the fluency of

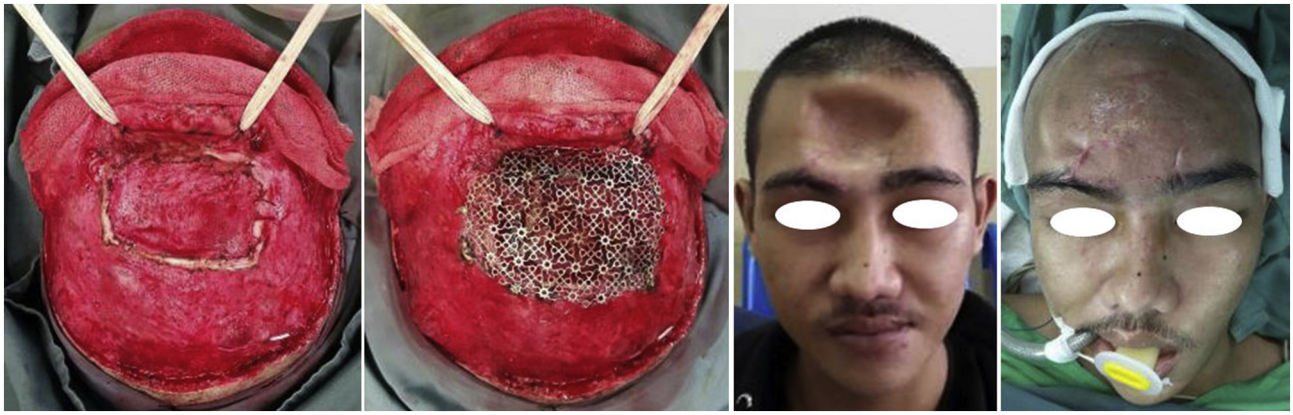

Fig. 6. Cranioplasty was performed using titanium mesh three months after the patient being discharged.

CSF flow outward to sinus. Beside the proliferation of leptomeningeal cells, present studies primarily target the pathological obstruction of arachnoid granulations (AGs), including the mechanical blockage and fibrosis of AGs. Both meningitis and subarachnoid hemorrhage (SAH) can initiate this pathological process through the accumulation of CSF resulting in communicating hydrocephalus. Furthermore, fibrosis and adhesions of the leptomeningeal arachnoid granulations could also be resulted from meningitis and SAH [10,11].

In this case, the communicating hydrocephalus was resulted from an open depressed fracture at a mid-frontobasal that pressed frontal sinus. He had undergone local debridement approximately three weeks prior the admission to our centre, while hydrocephalus had already developed at the time of his admission (Figs. 1D and 2). Duraplasty was performed by using pericranial graft, while frontal sinus ostium was obliterated, followed by cranialization [12–14] (Fig. 4). The procedure was completed with EVD insertion at right Kocher point, where shunt was inserted to 6 cm. The diverted CSF was transparent and yellowish, with an opening pressure of 18 cmH_2O. Sample of CSF was then submitted for profiling and culture examination. The CSF profiling identified the existence of leukocytes (PMN 24%, MN 76%), while Nonne and Pandy were both positives and the concentration of glucose reached 58 mg/dl, and the protein 162 mg/dl. These findings suggest that the most possible cause of hydrocephalus in our patient was subarachnoid hemorrhage, not bacterial meningitis [8,10]. Nonetheless, broad spectrum antibiotics were still given since pus and abscess formation were revealed during intra-operative. The regiment of antibiotics that were administered including Ceftriaxone, Gentamicin and Metronidazole [11,15]. The administration of broad spectrum antibiotics was also due to the fact that we could not completely exclude the possibilities of bacterial meningitis as the cause of hydrocephalus [8,16]. Inadequate debridement of open depressed fracture at the initial treatment prior patient's admission to our centre, exposed frontal sinus, along with laceration of the duramater could all lead to CSF and arachnoid layer infection, leading to meningitis and formation of abscess. The patient already gained full consciousness after his surgery and his headache was completely resolved. However, his vision remains impaired, suggesting that irreversible tissue damage have occurred.

To our surprise, the results from cultures of both pus and CSF samples were sterile. We suspected the administration of broad spectrum antibiotics regiment prior to the collection of samples causing the negative cultures of pus and CSF. Moreover, results from previous publications described the negative results from 14 to 34% culture of samples that are collected from abscess. In those cases, administration of antibiotics prior to collection of abscess material was also suspected to be the cause of sterility of the cultured samples [17,18]. In another study, causative organism was only successfully isolated in 4.4% of total CSF samples tested [20]. Additionally, it has been shown that antibiotic exposure can render sterility of CSF within hours after intravenous medications [19]. Unfortunately the patient could not tolerate EVD clamp trials. The failure of EVD weaning led to VP shunting [21,22]. PTH is a treatable TBI complication and most of the patients underwent shunt surgery, achieve favourable outcomes [8]. The duration of antibiotic therapy for brain abscess patient is 6–8 weeks [15,17,18], while in patients who are suspected with bacterial meningitis but no pathogen is identified in cultures should be given empiric antibiotics regiment for a minimum duration of 2 weeks [16]. In our patient, empiric antibiotics administration was continued to 6 weeks; follow-up CT scan showed the expected improvement (Fig. 5). The patient was discharged in good clinical condition and showed no symptoms of infection, despite his visual loss that seemed permanent.

After 3-month follow-up, the patient underwent cranioplasty using titanium mesh to close his bone defect (Fig. 6). Although the optimal timing of cranioplasty remains debatable, a systematic review written by Yadla et al., that analyzed 18 studies to determine the effect of early surgery (within 3 months of craniectomy) on rates of infection and other complications showed no significant differences in rates of infection rates or overall complication between early and late surgery, or between allograft and autograft cranioplasties [23]. In another recent meta-analysis performed by Xu et al., it was concluded that early (< 12 weeks) and late (> 12 weeks) cranioplasty result in no differences in overall complications [24]. Quah et al. reported that there were no significant differences in infection rates between early and late cranioplasty [25]. Morton et al., reported that cranioplasty that is performed between 15 and 30 days after the initial craniectomy might minimize infection, seizure and bone flap resorption, whereas cranioplasty that is done in > 90 days would minimize hydrocephalus, but may increase the risk of seizure. Regardless, the cranioplasty surgery in our patient went well and we found no complications at 3- and 6-month follow-up.

4. Conclusions

Further exploration in patients with a progressive vision loss, post-traumatic depressed skull fractures at the anterior cranial fossa and hydrocephalus is suggested to unravel the underlying problem. Actually, this case makes a strong argument for an early neurosurgical intervention in frontobasal fractures.

Consent

Informed consent was obtained from the patient for publication of this case report and any accompanying images. The patient's family was present at the time.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

AF, CBK, DH and MZ had examined, treated, observed and followed up the patient. All authors participate in writing the manuscript, has read and approved the final manuscript.

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