CASE REPORT

Seizure control following surgical resection 17years after a mismanaged compound depressed skull fracture: case report

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ABSTRACT
Compound depressed skull fracture is a discontinuity in the skull in which one (or more) of the discontinuous edges is (are) displaced below the inner table of the surrounding intact skull in conjunction with a breach of the epidermis and the meninges or in communication with the paranasal sinuses and middle ear structures resulting in a connection to the exterior environment. The sequelae of such lesions when not properly managed could be quite significant in the outcome of the injury and the life of the patient. We report a case of a 19-year old Nigerian male who presented with post-traumatic epilepsy 17years after sustaining a compound depressed skull fracture at 8months of age, following a fall from height, but, which was not properly managed. It was a case of missed opportunity for an initial proper diagnosis and management, buttressing the need for an early expert surgical management of compound depressed skull fractures and the efficacy of resective surgery in the management of this type of epilepsy.

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INTRODUCTION

Skull fractures and their management were recognized and described in Edwin Smith's papyrus; the oldest known published surgical paper.[1] Smith's papyrus described a conservative and expectant approach to skull trauma. The 15th century management of paediatric skull fractures was illustrated by a Turkish physician of the Ottoman Empire, Serefeddin Sabuncuoglu (1385-1468), in his textbook Cerrhiyyetu'l Haniyye (Imperial Surgery).[2]

Depressed skull fractures are those fractures in which the fragments are displaced inwards (intracranially) below the inner table of the

surrounding intact skull; it is usually associated with traumatic brain injury.

The depressed fragment of a fractured skull if it breaches or compresses brain tissue could lead to gliotic changes of the underlying brain. Gliosis is a non-specific reactive change of glial cells in response to damage to the central nervous system. It is the universal response of the central nervous system (CNS) to injury, and results from traumatic, ischaemic and haemorrhagic assaults.

Gliosis is characterized by the proliferation or hypertrophy of glial cells including astrocytes, microglia and oligodendrocytes. In its most extreme form, the proliferation associated with gliosis could lead to the formation of a scar. In any of its forms at all, there is an alteration in cellular activity with the potential to create widespread effects on neurons and non-neuronal components causing either a loss of normal functions or gain of abnormal ones like seizures.[3]

Depressed skull fractures account for 7-10% of children admitted to hospital with head injury and 15-25% of children with skull fractures.[4] Radiological modalities used in the diagnosis include skull x-rays which may show an overt displacement, concentric fracture lines, stellate fracture lines and /or bone-in-bone appearance, each of which is individually diagnostic of depressed skull fractures. Computed tomographic scan, however, remains the imaging modality of choice.

Compound depressed skull fractures should be surgically elevated in most cases except in a few cases where the fragment may be tamponading a dural venous sinus and elevation may lead to severe loss of blood.[5] They are surgical emergencies regarded as contaminated wounds and unless treated promptly and properly, complications like meningitis, cerebral abscess, osteomyelitis or post-traumatic seizures, may supervene.[6]

We report the case of a 19-year old male student who was admitted into our service with post-traumatic epilepsy occurring 17years after a compound depressed skull fracture which was not properly managed. We also seek to highlight the need for expert management of skull fractures even when the initial post-traumatic neurological status of the patient appeared to be normal.

CASE REPORT

The 19-year old male was referred to our service in Nnamdi Azikiwe University Teaching Hospital Nnewi, Anambra State with complaints of recurrent seizures of one year duration. Our hospital is a federal tertiary health facility which serves as a referral centre with a potential catchment of up to 20% of Nigeria's population.

The seizures were of sudden onset, focal and tonic-clonic in character, involving just the right upper and lower extremities. Each episode lasted about one minute and occurred two times per month. There was an associated sensory aura preceding each episode, with subsequent loss of consciousness and post-ictal sleep that last for about one hour, following each seizure.

The seizures were not controlled despite treatment with anti-epileptic drugs (AEDs) – phenobarbitone, carbamazepine and phenytoin sodium, at different times.

The patient had a positive history of a fall from the elder brother's back at 8months of age. He had struck his head on the concrete stairs at the time, and had a seizure, immediately after. He was, subsequently, taken to a hospital where skull x-rays were done and his parents were reassured that there were no major problems. The detected skull fracture from x-ray studies at the time

was managed conservatively by the doctor (a general practitioner) and the patient was discharged in good health with no repeat seizures until 17years later. He was placed on multiple AEDs without resolution, necessitating his referral to our service in view of the history of previous head injury.

Clinically, he was a healthy looking male with normal vital signs and in no obvious distress. He was not pale, not jaundiced and not cyanosed. There were no gross cranial nerve, sensory or autonomic deficits but there was a slight increase in tendon stretch reflexes in the right upper and lower extremities, the same side where he had his seizures.

Cranial computed tomography (CT) scan revealed a healed/ossified left parietal depressed skull fracture with an inwardly displaced fracture fragment into the parietal lobe and surrounding area of cerebral atrophy, *see Figures 1 and 2*.

Figure 1: Soft tissue window of the computed tomography showing the area of left parietal cerebromalacia (gliosis) with significant cerebral oedema

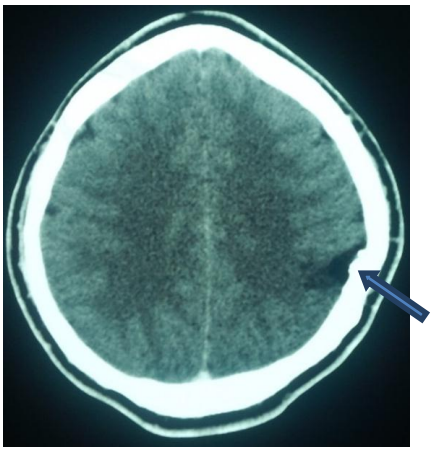
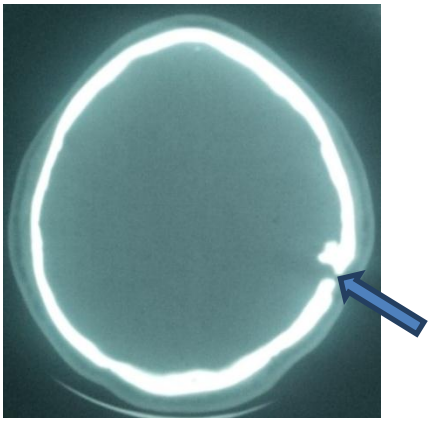


Figure 2. Cranial computed tomography bone window showing the left parietal depressed fracture with malunion and cranial defect



A diagnosis of post-traumatic epilepsy, secondary to a mismanaged compound depressed left parietal skull fracture with parietal lobe gliosis, was made.

He was fully worked up and subsequently, underwent operative treatment by elevation, craniectomy and excision of gliotic brain with pericranial duroplasty. Intraoperative findings were a 5cm×3cm dural defect, a depressed and inwardly displaced fracture fragment traversing the dural defect and lodging inside the underlying parietal lobe with gliotic brain tissue filling the circumference of the dural defect and surrounding the displaced fracture segment. There were briskly hemorrhagic dural and cerebral vessels in and around the wound bed

The patient was placed on post-operative antibiotics and an AED, sodium valproate, and was finally discharged home after 2weeks, with no neurological deficits or breakthrough seizures, even after the drugs were stopped 3months after discharge. He has had eight scheduled follow-up visits in the following 18months with no repeat seizure.

DISCUSSION

The association of epilepsy and head injury has been recognized since antiquity. Given the description of skull fractures in the Edwin papyrus, written in the 17th century BC but referring to observations made as far back as

3000BC, it is almost certain that patients surviving severe head injuries went on to develop epilepsy.[7]

Epidemiological studies have found that post-traumatic epilepsy accounts for approximately 20% of symptomatic epilepsy in the general population, and 5% of all patients seen at specialized epilepsy centres.[8] Acute trauma such as that which occurs following depressed skull fractures can cause penetrating head injury like in the index case. It is pertinent to note that brain damage resulting from penetrating head injury may increase the risk of epilepsy, nearly 600-fold.[9]

There are several patient and injury characteristics that increase the likelihood of developing post-traumatic seizures e.g. reduced level of consciousness (GCS <10), early post-traumatic seizure, cortical contusion, depressed skull fracture, epidural hematoma, intracerebral hematoma, and wounds with dural penetration, like in the index patient.[10] Most of these features are applicable in the index patient who had an untreated depressed skull fracture as well as immediate post-traumatic seizures after the head trauma, only to develop epilepsy after 17yrs.

More importantly, depressed skull fractures were the most common abnormal CT scan finding in patients with post-traumatic seizures (50%). This is in keeping with the aetiologic pattern of paediatric head trauma in our environment as shown in some previously published studies with falls accounting for 15% and 48.1% of pediatric head trauma, respectively.[11,12]

The inability to make a prompt and proper diagnosis and to deliver expert surgical treatment in the index case could be due to a poor knowledge of the diagnostic indices of compound depressed skull fracture, its definitive treatment modality and its complications, on the part of the clinician who initially attended to him. More than 83% of cases of compound depressed skull fractures in our environment had delayed definitive management with 79.6% actually coming on referral from other clinicians/facilities, in a previous study.[13]

Epilepsy surgery has been established as an effective treatment option in pharmaco-resistant focal epilepsies like in the index case.[14] This is made possible by the continued advances in pathologic and imaging technology, facilitating the easier detection of structural lesions in patients with chronic focal seizures. According to the German Epilepsy Register, 6.8% of histology specimens in 2009 were diagnosed as gliosis.

Some studies have reported a decrease in seizures following resective surgeries among a group of selected patients.[15] Our index patient was seizure-free on a single AED for three months after the surgery, contrary to his uncontrolled seizures on multiple AEDs prior to his resective surgery. He has remained seizure-free without any AED for more than 18monhs now.

CONCLUSION

Depressed skull fractures could be a cause of uncontrolled post-traumatic seizure as well as lead to other complications following traumatic brain injuries. This case report underscores the grave implications of mismanagement of these fractures and the value of appropriate neuroimaging evaluation and expert surgical management, when complications supervene.

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