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Case Report

Early Acute Spontaneous Giant Extradural Haematoma Following Ventriculoperitoneal Shunt Surgery in an Adult with Chronic Communicating Hydrocephalus: A Case Report

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Abstract

Background: Ventriculoperitoneal shunt surgery for chronic hydrocephalus with raised intracranial pressure has expectant subdural haematoma among other possible complications. This is due to rupture of the bridging veins between the dura mater and brain especially with rapid decompression of the intracranial pressure. This haematoma mechanism is clearly understood and sudden deterioration of the patient with neurologic deficits clearly indicate need for urgent neuroimaging to confirm diagnosis. Although a rare complication, epidural haematoma can occur following cerebrospinal fluid diversion for chronic hydrocephalus.

Purpose: To report a case of acute extradural haematoma following ventriculoperitoneal shunt in an adult with chronic hydrocephalus and the review of related literatures.

Methods: Literatures regarding occurrence of acute extradural haematoma, pathophysiology and management were reviewed.

Case Presentation: A 43-year-old female patient who had right sided ventriculoperiotoneal shunt for communicating hydrocephalus with raised intracranial pressure. She deteriorated on the second day post-surgery which necessitated urgent cranial computed tomography scan for which finding of acute epidural haematoma was noted.

Conclusions: Ventriculoperitoneal shunt insertion for patients with hydrocephalus may be associated with either common or rare complications and the incidence of rare complications are higher with chronic hydrocephalus with raised intracranial pressure. A high index of suspicion, meticulous surgical skill set and close monitoring are key to early intervention and good outcome.

Keywords: Ventriculoperitoneal Shunt; Hydrocephalus; Intracranial Pressure; Extradural Haematoma

Abbreviations

VP: Ventriculo-Peritoneal; EDH: Extradural Haematoma; CSF: Cerebrospinal Fluid; ICP: Intracranial Pressure; CT: Computed Tomography

Introduction

Ventriculoperitoneal shunt insertion is the most common neurosurgical intervention and has become the mainstay for the treatment of hydrocephalus since it was reported in 1898 [1,2]. Complications such as shunt infection and malfunction commonly result in a significant number of prolonged admission and readmissions in the hospitals [3]. Subdural hematoma was discussed in detail for the first time by Dandy as a common complication following ventriculoperitoneal shunt [4]. Generally, it is observed in the first days of shunt placement or at a later period when compared to non-traumatic extradural haematoma which always occurs few hours following shunt surgery [5]. Extradural haematoma is a life-threatening indication for neurosurgical intervention [6]. This can result following traumatic brain injury, ventriculoperitoneal shunt for chronic hydrocephalus and decompressive craniectomy for acute subdural haematoma [7]. Different mechanisms have been postulated to be responsible for the development of the extradural haematoma following ventriculoperitoneal shunt. When this occur at the burrhole site, it may be due to vascular injury in the dura or inadequate control of cranial bleeding. When it occurs at a site distant from the burrhole, it can be associated with the dura mater detaching from the osseous tissue (anterior part of the cranium are more loosely attached compared to the posterior part) following changes in intracranial pressure [8]. This can result in rupture of the middle meningeal arteries embedded in the skull.

Case Presentation

A 43-year old female presented to the Neurosurgery clinic with weakness of both upper and lower limbs of 3years duration. The weakness started in the right limbs and progressed to involve the left limbs. She was initially able to ambulate with a limp, later with walking aid after which she became unable to ambulate. The patient also had headache which was moderate in intensity, holocranial, worse in the morning and associated with some episodes of vomiting which relieved the headache. There was associated blurring of vision in both eyes. No history of seizures, change in personality. Patient experienced weight loss due to reduced intake and prolonged confinement in bed. She initially presented to a tertiary health facility where initial neuroimaging was done and was lost to follow-up for 3years. She had no comorbidities and not on any anticoagulant. She had reduced muscle bulk, power of grade 4 in all limbs and marked long tract signs. Brain magnetic resonance imaging done showed pan-ventriculomegaly with periependymal transudation of cerebrospinal fluid, caudal descent of the brainstem, subdural hygroma with uneffaced sulci and gyri. A diagnosis of Chronic Communicating Hydrocephalus was made.

She was counseled and had emergency ventriculoperitoneal shunt with medium pressure shunt. Analgesics, antibiotics and fluid therapy were commenced.

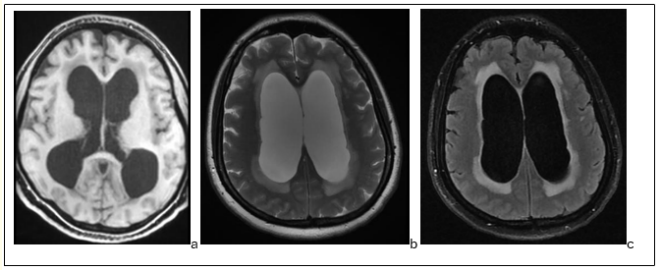

Figure 1: Preoperative axial sections of Cranial MRI; a T1WI, b. T2WI, c. FLAIR.

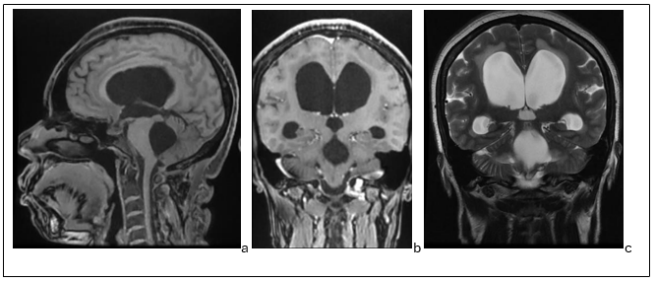

Figure 2: Pre operative cranial MRI sections; a. Sagittal T1WI, b. Coronal T1WI + contrast, c. Coronal T2WI.

On the second post operative day, she was noted to have an altered consciousness with tonic seizures affecting all extremities. She also had lateralizing signs (left hemibody weakness and anisocoria). The shunt was ascertained to be functional. Urgent cranial computed

tomography scan was done and showed a hyperdense biconvex lesion (with swirl sign) in the right frontotemporoparietal region with significant midline shift, effacement of the ventricles and cisterns. Volume of haematoma was estimated at 400mls.

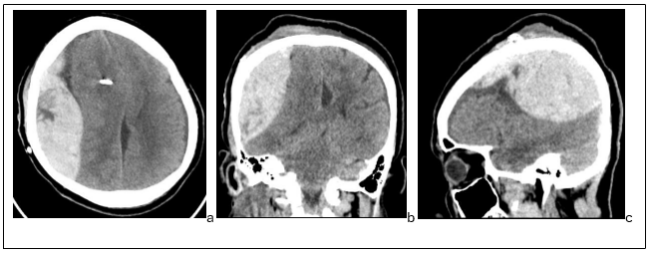

Figure 3: Post operative cranial CT scan; a. Axial view, b. Coronal view, c. Sagittal view.

Emergency craniotomy and evacuation of extradural haematoma was done. Intraoperatively, an avulsed actively bleeding middle meningeal artery was cauterized. No bleeding was observed in the subdural space. There was marginal improvement in consciousness in the immediate post operative period. Patient was closely monitored until her demise a day after craniotomy.

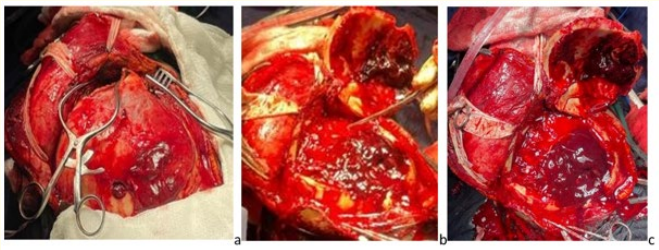

Figure 4: Perioperative findings at Craniotomy.

Discussion

Rare complications like subdural haematoma, extradural haematoma and cerebellar haemorrhages following ventriculoperitoneal shunt surgery may result from overdrainage of cerebrospinal fluid [9].

Fukamachi., *et al.* reviewed the postoperative cranial computed tomography images of 1,055 patients and found that extradural haematoma rate was 0.4% following ventriculoperitoneal shunt [10]. There is no consensus on the mechanism of this extradural haematoma. Some authors have suggested that the reduction in the intracranial pressure causes the cortex to collapse, renting vessels attached to the dura [11]. This most probably explains the course in our index patient. Some other postulated causes of extradural hematoma to include head trauma after surgery, coexisting bleeding disorder, bleeding from an occult dural vascular malformation and improper haemostasis[10]. There was no derangement in the clotting profile parameters, no overt vascular anomaly during hematoma evacuation and haemostasis was adequately secured.

Yucel., *et al.* presented a case of a 7year old female who had myelomeningocele repair at birth. This child with paraparesis had undergone shunt revision for the third time at the age of 3 years. Last shunt hardware was a high pressure shunt. She presented to emergency department at 7th year of life with left upper limb weakness and altered consciousness for which urgent cranial computed tomography scan revealed large right sided acute extradural haematoma for which emergency craniotomy was done [12]. Seyithanoglu., *et al.* documented occurrences of non-traumatic haemorrhages despite use of moderatehigh pressure shunts and adjustable pressure or flow control devices which were recommended to reduce the risk of this complication [14]. Our index case had a medium pressure shunt inserted in accordance with our unit protocol. This did not seem to reduce the rate of ventricular decompression. A high pressure shunt may have reduced the speed of decompression but there is no evidence to suggest that this will prevent an extradural haematoma formation.

Sengupta and Hankinson reported three cases of extradural haematoma as complications of ventricular drainage among 22 patients and associated the underlying reasons as young age and chronic hydrocephalus [11]. Our index case shared similar characteristics in terms of being young and chronicity of the hydrocephalus. Supratentorial extradural haematoma has been discussed in 43 cases after ventricular decompression in terms of cerebrospinal fluid by Odake., *et al.* [14]. Delay in symptoms and diagnosis of frontoparietal convexity hematoma results from the spread throughout the entire hemisphere [10]. Cases of chronic, calcified epidural hematomas after ventriculoperitoneal shunting have been reported [15]. Odake., *et al.* also reported that mortality rate of extradural haematoma following ventriculoperitoneal shunt was 44.2% while those who recovered with deficit are 11.6%. Our index patient presented with a supratentorial extradural haematoma and never recovered fully till death.

The proposition of using programmable valve to prevent haematoma following surgery for chronic hydrocephalus was suggested by Louzada., *et al.* [16]. However, cases of extradural haematoma occurrence following programmable-valve shunt insertion has been reported by Power., *et al.* and Harkness [17,18]. In our index case, the extradural haematoma was evacuated and the shunt removed as the craniotomy incision site was in the shunt territory. There are few cases of extradural haematoma reported after endoscopic third ventriculostomy. This may be a viable option where patient characteristics suggest a susceptibility towards haematoma formation.

Conclusion

Ventriculoperitoneal shunt insertion for patients with hydrocephalus may be associated with either common or rare complications and the incidence of rare complications are higher with chronic hydrocephalus with raised intracranial pressure. A high index of suspicion, meticulous surgical skill set and close monitoring are key to early intervention and good outcome. More research is required to elucidate the aetiopathogenesis and predisposing factors which will greatly enhance the prevention and management of this condition.

Declaration of Interest

The authors have no conflict of interest to declare.

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