Recurrent intracranial epidural hematoma following ventriculoperitoneal shunt in a child

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Abstract

Intracranial hematoma is commonly observed in neurosurgical practice. However, recurrent intracranial epidural hematoma following ventriculoperitoneal (VP) shunt is more of an exception than the norm. It is a rare but serious cause of morbidity and mortality in patients with VP shunt. However, treatment is very promising especially with surgical intervention in time. Here we report a case of a ten-year-old girl who presented with chronic headache for a couple of years whose imaging features suggested a hydrocephalus with tonsillar herniation. Initially, she developed right frontotemporal hematoma and then bilateral frontal epidural hematoma following a VP shunt. Emergency decompression was done.

Keywords: Epidural hematoma, Hydrocephalus, intracranial, Ventricular-peritoneal shunt

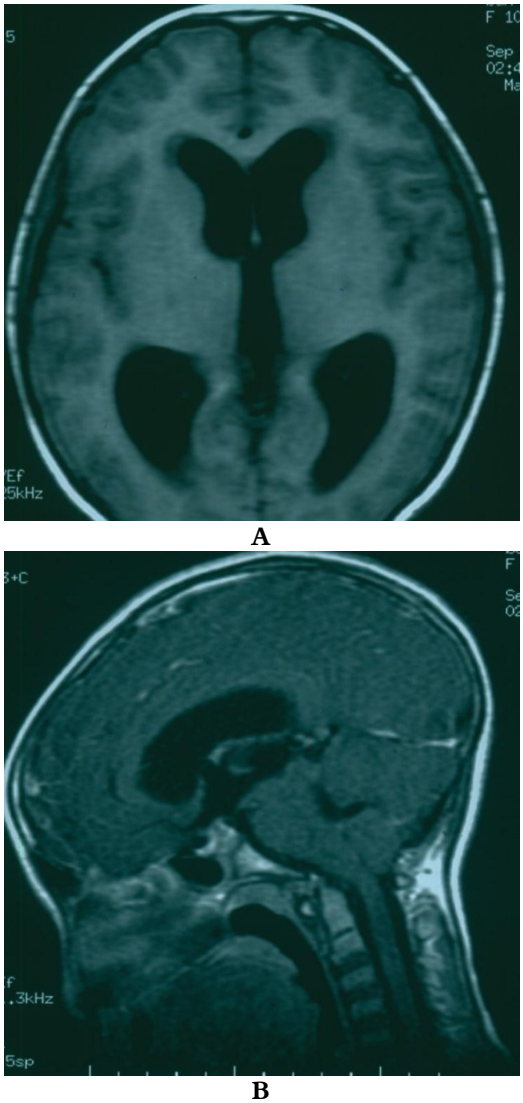
Introduction

Intracranial epidural hematomas (EDHs) accounts for approximately 2% of patients following head trauma and 5-15% of patients with fatal head injuries. 65-90% cases are associated with skull fractures4. EDH following cerebrospinal fluid (CSF) diversion for hydrocephalus is rare. We report our experience with EDH after ventriculoperitoneal shunt (VPS), management aspects and review of literature.

Case report

A ten-year-old female child presented with intermittent headache for 2 years. She was diagnosed with and treated for sinusitis at a local hospital. However, it was severe with repeated vomiting for a couple of months. She was referred to our hospital for further management. There was no history of trauma, fever, any anticoagulation drug intake, or any other co-morbid disease seemingly responsible for such event as elaborated from history. On examination, there was no neurological deficit. Magnetic resonance imaging revealed obstructive hydrocephalus as well as cerebellar tonsil herniation (Figure 1 A and B). Her blood profile was within the normal range. Immunological investigations were also within normal limit. Patient underwent right ventricular peritoneal shunting procedure. Post operative computed tomography (CT) scan was performed as her GCS was altered and showed right fronto-parieto-temporal epidural hematoma (Figure 2A). Emergency fronto-parieto-temporal craniotomy was done and dural tacked up sutures along the margin of craniotomy. 50 ml dark red blood clots, some of which was liquefied was found and evacuated. Patient was put under close monitoring in ICU.

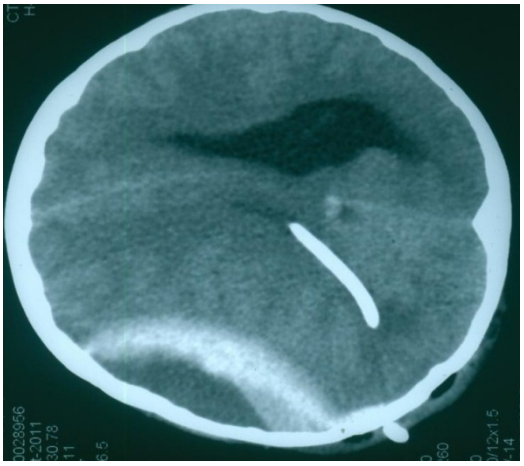
Post operative CT scan of craniotomy two days later showed just EDH (Figure 2B) but in a small amount so patient underwent strict observation. Patient complained of headache a week after craniotomy. CT scan showed bilateral frontal huge epidural hematoma (Figure 2C).



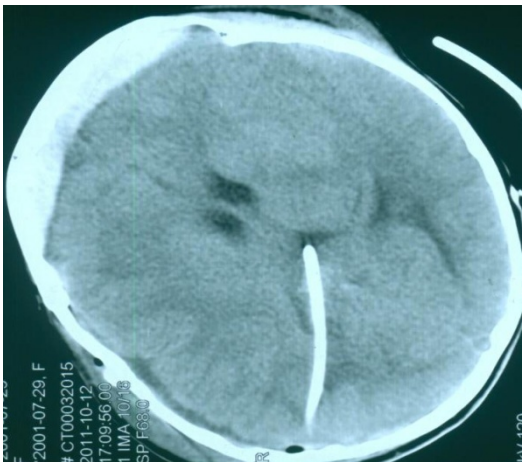
A

B

Figure 1 Preoperative Axial **(A)** and sagittal **(B)** section of MRI showing hydrocephalus and tonsillar herniation



A



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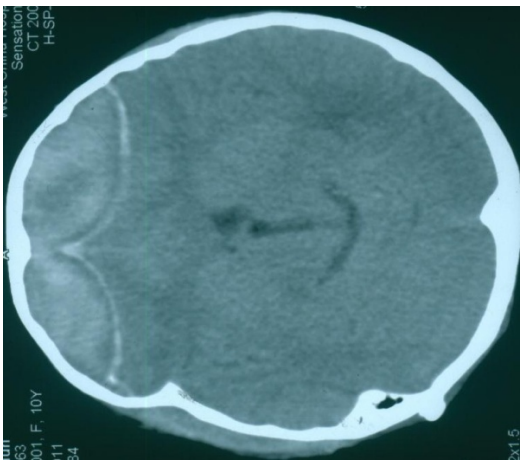

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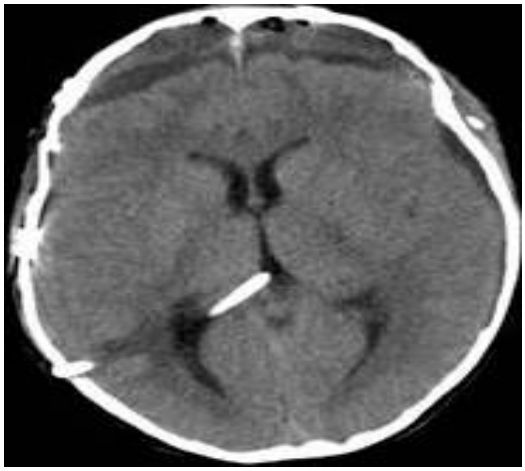

D

Figure 2 (A) Non-contrast axial head CT after VPS revealing right fronto- parietto-temporal hypo-hyperdensity-epidural hematoma and a portion of the shunting apparatus with mass effect. **(B)** Non-contrast head CT obtained after 2 days of second operation, revealing hematoma frontal region **(C)** Head CT after 7 days of VPS, revealing huge bilateral frontal epidural hematoma. **(D)**Head CT after third operation showing normal ventricle with shunting apparatus

Bilateral frontal craniotomy was done and dura was tight sutured along site of craniotomy. About 70 ml dark calcified black blood, adhesion with dura was found and evacuated. This time patient recovered smoothly. Post operative CT scan showed normal shaped ventricle without EDH (Figure 2D).

Discussion

In 1902, Cushing H. introduced ventricular drainage (VD) as a means of reducing intracranial pressure. Since then, VD procedure has been frequently performed in neurosurgical practice. Several intracranial subdural hematomas (SDH) after VPS have been reported in the literature as a complication. To our knowledge, the first reported complication of VD was mentioned by Schorste in 1942 (9). There are only few published reports of intracranial EDH as a complication of VPS. Intracranial EDH following CSF diversion for hydrocephalus is an important cause of immediate deterioration and contributes to morbidity and mortality. Their presentation is according to the size and location of hematoma, elevated intracranial pressure and midline shift. The computed tomography scan has played a significant role in the early detection and proper treatment of post operative intracranial hematoma.

EDHs are contact injuries resulting from blunt trauma to the skull and meninges. Fractures, most often linear, are present in 30 to 91 per cent of patients with epidural hematomas (1). It is thought that the initial impact, with deformation or fracturing of the cranium, produces detachment of the dura directly beneath the site of the blow and injures blood vessels (most commonly branches of the middle meningeal artery). Hemodynamic factors like vascular malformations of the dura mater and preoperative administration of anticoagulation or disorders of blood coagulation (spontaneous or iatrogenic), hypertension, effect of operative position to venous outflow are the mechanisms of EDH. J. F. Sanchis et al (1975) explained that neighborhood infection is also one of the causative factors for EDH. In the literature, authors mentioned that mechanical factors like bridging vein tearing, dural detachment because of brain parenchyma displacement induced by CSF (5, 6, 7). Our case is interesting as EDH developed adjacent and distant to the VP shunt in 24 hours and one week respectively. In our knowledge, the duramater is firmly attached at the cranial sutures in infants. Sudden lowering of

intracranial pressure (ICP) or rapid drainage of ventricular CSF or gravitation flow of CSF result in brain shrinkage from the skull. Ultimately, detaching the collagenous fixations of the dura from the inner table of the skull may initially cause dural and diploic veins to bleed into the extradural pocket (7, 8). Once bleeding has begun, the blood fills the pocket. Experimental evidence indicates that arterial bleeding into the resulting pocket creates a hydraulic "water press" effect, progressively stripping away the dura from the skull and widening the perimeter of the hematoma (2).

Post shunt EDH can be managed surgically and conservatively. The choice between a surgical or a nonsurgical treatment of post shunt EDH requires the evaluation of various factors: volume, thickness, midline shift and amount of fresh blood present on CT scan (4), the age of the patient, and the clinical picture. Huge acute or subacute collections in adults or in children with closed fontanelles usually require surgical treatment. However, in a series concerning a pediatric population, it was stated that asymptomatic EDH may become symptomatic later on and that it is safer to treat all post shunt EDH, whether symptomatic or not (1). Our case is quite unique where a patient had presented primarily with extradural hematoma after VPS and again gradually developed EDH within one week after craniotomy. We performed craniotomy twice and evacuation of hematoma after dural tenting sutures along the margin to help hemostasis (3). She did well post operatively even after three consecutive operations and was discharged from the hospital in good condition.

Conclusion

VPS is a common neurosurgical interventional procedure. Patients should be monitored closely post shunt, so intervention can be done immediately if any evidence of deterioration like SDH, EDH and others. Neurosurgeons must keep in mind that ICP raised patients may present with such potentially fatal complication which can be successfully treated if diagnosed in time.

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