

Acute epidural haematoma following ventriculoperitoneal shunt in an adult

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Abstract: The authors report a 50-year-old patient of pituitary macroadenoma with hydrocephalus, who developed a large left parieto-occipital epidural haematoma following placement of a ventriculoperitoneal shunt. This is the first case report describing this complication in an adult patient, and it is hypothesised that coagulation of the dura along with rapid lowering of intracranial pressure, may lead to its stripping from the bone with attendant tear in the emissary vein(s) resulting in the possibility of epidural haematoma formation in adults. Judicious dural coagulation should therefore avoid this uncommon and potentially life-threatening condition. (p118-120)

Key words: Ventriculoperitoneal shunt and extradural haematoma

Introduction

Ventriculoperitoneal (VP) shunt placement is associated with several complications, including intracranial haematomas. Although acute subdural haematomas (SDH) after ventricular decompression is well known, the formation of acute epidural haematoma (EDH) after a VP shunt is rarely seen and only six cases have been reported in English literature, all belonging to the paediatric age group.¹⁻⁷ We describe its occurrence in an adult and discuss the possible aetiology in this age group.

Case Report

A 50-year-old man was admitted with complaints of bilateral decreased vision for 3 months and headache along with vomiting for one month. On examination, he had a visual acuity of 6/24 in both eyes, with bitemporal field cuts. Rest of the neurological examination was normal. Routine biochemical investigations, including coagulation profile was normal. Contrast enhanced magnetic resonance imaging scan revealed a large sellar and suprasellar tumour, with retrosellar extension in the prepontine cistern and

hydrocephalus (Fig. 1). Due to large retrosellar extension which may not be completely removed by surgery, and associated symptomatic hydrocephaly, it was decided to place a VP shunt prior to surgery for pituitary macroadenoma.



Figure 1 - Contrast enhanced MRI scan revealing a large sellar and suprasellar tumour, with retrosellar extension and hydrocephalus

A medium pressure (Chabbra®) VP shunt was placed through left occipital burr hole, as the left ventricle was larger than the right one. The patient became progressively drowsy 4 hours after the surgery and developed vomiting. Computed tomography (CT) scan done at this time revealed a large acute EDH in the left parieto-occipital region, with midline shift and effacement of basal cisterns (Fig. 2a). He was immediately taken for surgery and EDH was evacuated by a trephine craniotomy. No specific bleeder was found. Diffuse dural oozing was controlled and dural hitch stitches were done. The shunt was changed to a “high pressure” shunt to prevent further EDH formation due to excessively

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low pressures. Postoperative CT head showed good evacuation of the haematoma (Fig. 2b). The patient improved after the surgery and was treated with surgery for pituitary macroadenoma on a later date.

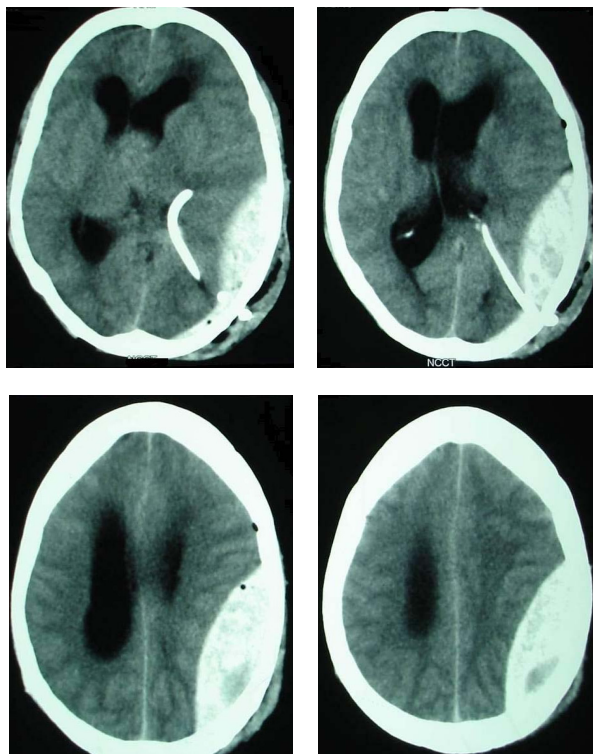


Figure 2a ↑ Non-contrast CT head showing the large parieto-occipital epidural haematoma in relation to the shunt site

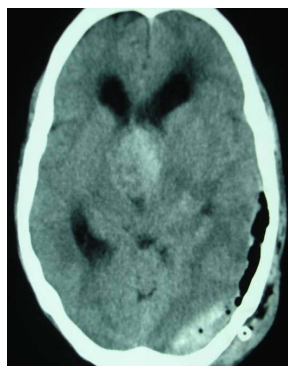


Fig 2b ← Postoperative CT head showing good evacuation of the epidural haematoma

Discussion

Formation of acute SDH after ventricular decompression is well known in neurosurgical practice.¹ As the brain slackens after decreasing ventricular pressure, the subdural bridging veins are stretched and may get torn, causing subdural haematoma. However, pulling away of dura from the skull after decreased intracranial pressure is much less

common and is thought to be due to the firm attachment of the dura to the skull in adults. Indeed, most cases of EDH after VP shunt reported in English literature have been in children.^{1,3,4,7} The case reported by Fujimoto, et al had congenital Factor X deficiency and post haemorrhagic hydrocephalus, who underwent a VP shunt. In this patient coagulopathy was also responsible for postoperative spontaneous EDH formation.² Our patient did not have any evidence of coagulopathy either before or after surgery. Kalia, et al reported a case of multiple epidural haematomas in a patient with hydrocephalus treated by VP shunt.⁴ Their patient became symptomatic 5 days after shunting and required craniotomy for evacuation of one of the haematomas. Isolated cases have also been described after posterior fossa exploration, air ventriculogram, endoscopic biopsy.^{3,5,6}

The probable mechanism for formation of EDH after ventricular decompression is sudden lowering of intracranial pressure, which causes separation of dura from the skull. This is supported by the fact that most reported cases occur in children, in whom the dura is less tightly adhered to the skull than in adults. Kalia, et al proposed that in some patients, discrepancy between cranial and brain volumes, or craniocerebral disproportion may be responsible.⁴ However, in adults these causes are considered unlikely. We hypothesise that coagulation of the dura may lead to its stripping from the bone with attendant tear in the emissary vein(s) in some adults whose dura is not tightly stuck to the bone, resulting in the possibility of EDH formation in adults. This dural separation is further aided by rapid lowering of intracranial pressure.

Coagulopathy, if present, may contribute to EDH formation.² In case of EDH directly adjoining the burr hole site of VP shunt, excessive coagulation of dura of the burr hole before dural incision, may in some cases cause enough shrinkage, to result in dural separation from the skull and EDH formation. This dural separation is further aided by rapid lowering of intracranial pressure.

Meticulous surgical technique with avoidance of excessive dural shrinkage at the burr hole site, minimal cerebrospinal fluid drainage at the time of ventricular catheter insertion, use of high or medium pressure valves if possible, use of anti-syphon device in shunt and slow return to upright position after VP shunt may further reduce the incidence of this uncommon and possibly under reported shunt complication.

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Answer Neuro-Radiological Quiz

Giant dilated perivascular spaces

Discussion

Perivascular spaces (PVS) are also known as Virchow–Robin spaces that accompany penetrating arteries for variable distances as they penetrate the brain substance. These PVS are routinely noted in MRI in all ages. Occasionally, they may enlarge and appear as a cystic bizarre shaped lesion mimicking cystic tumors, parasitic cysts, cystic infarction, non neoplastic neuro epithelial cysts.

The most common site of PVS is along the lenticulostriate arteries adjacent to the anterior commissure. Less common sites are the sub insular region, dentate nuclei and cerebellum.

The typical MR imaging features of dilated PVS are rounded well defined smooth outline cystic areas laying almost the pathway of penetrating arteries, isointense to cerebrospinal fluid (CSF) in all sequences with no enhancement. Giant dilated PVS may be surrounded by hyper intensity in the fluid-attenuated inversion recovery (FLAIR) sequences (Fig. 2). Several theories were suspected, among these were chronic ischemic changes caused by the mass effect of the PVS. Another possibility is related to chronic mechanical stress caused by high blood pressure on the brain arterial. The last possibility is likely in this case since the patient is known to have polycystic kidney disease with chronic hypertension.

Giant dilated PVS has been reported earlier.¹⁻³ All reported cases was found to have mass effect over the related structures of variable severity, occasionally associated with obstructive hydrocephalus if located in the mesencephaly.

The differential diagnosis of giant dilated PVS is from other lesions having CSF signal intensity, such as neuro-epithelial cysts, cystic infarction, mucopoly-scharridosis. Differentiation is made on the basis of the neuroimaging findings, the location of the lesions and the clinical history.

Suggested reading:

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