Case Report

A rare case of large bilateral epidural hematoma following ventriculoperitoneal shunt: a case report with review of literature

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Abstract

Epidural hematoma discovered several years after ventriculoperitoneal shunt procedure is a rare complication. It may be associated with ventricular decompression due to unregulated lowering of intra-cranial pressure. This leads to generation of suction force on structures between the cortex and the inner table of the skull resulting in bleeding from tearing of cortical bridging veins. We report a case of a 15-year-old female patient who underwent an uneventful ventriculoperitoneal shunt at the age of 6 months. 14 years later CT scan was done for the complaints of frequent headache that showed 2 large sized lesions that were partially calcified epidural hematomas. After surgical decompression of the larger of the two hematomas, patient's symptoms improved. We discuss the possible factors responsible for epidural hematoma, its calcification and ways to prevent or minimize such an avoidable complication.

Keywords: Epidural Hematoma, Hydrocephalus, Ventriculoperitoneal Shunt.

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Introduction

Ventriculoperitoneal (VP) shunt is a common procedure to achieve diversion of the cerebrospinal fluid (CSF). Despite being associated with several complications (infection, shunt malfunction, subdural hematoma, intraventricular or intracerebral hemorrhage, seizures, catheter migration, intra-abdominal pathologies) [1-4], a very delayed presentation of large epidural hematoma (EDH) due to subtle symptoms is a rare event. We report a 15-year-old female who was diagnosed to have two space occupying lesions (SOL) in left fronto-parietal and right frontal region. On craniotomy, SOL was noted to be organized EDH detected 14 years after VP shunt surgery.

Case History

A 15-year old female patient reported with history of increased headache off and on for the past three months. Patient had undergone an uneventful VP shunt placement at the age of 6-months. She gave history of occasional mild headaches over the past few years. There was no history of vomiting, loss of consciousness, fits, limb or facial weakness at any time during this period. Her vital signs showed a blood pressure of 100/62 mm Hg, pulse rate 96/minute, oxygen saturation of 99% on room air, with a peripheral temperature of 36.7^0C. Glasgow coma scale score was 15/15, vision was normal and pupils were normal in size and reacting to light, power in all limbs was 5/5 with intact sensations all over the body. CT of the head and neck done recently at a peripheral hospital was diagnosed to have SOL. She was referred to our tertiary care neurosurgery center. An urgent MRI was done that showed two extra-axial SOL with possible calcifications suggestive of EDH (Fig 1). One lesion was located in left fronto-parietal region measuring 5.30 X 9.28 X 17.40 cm and the other

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was seen in right frontal region measuring 2.95 X 2.82 X 14.0 cm. Both lesions were well defined causing mass effect on the brain cortex and effacement of lateral ventricles, left > right. There was a shift in midline structures to right side by about 1.70 cm. The tip of the VP shunt could be seen in the right lateral ventricle.

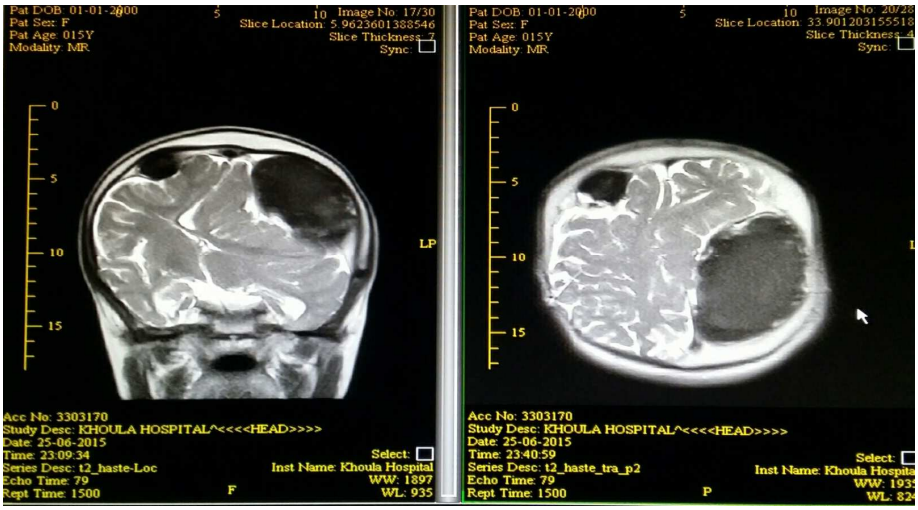

Fig 1: Showing two extra-axial SOL with possible calcifications

Patient was kept under observation as according to our clinical assessment patient no longer required VP shunt and plan was to remove the shunt during elective craniotomy. Patient was scheduled for surgery and shunt was ligated 3 days prior to surgery.

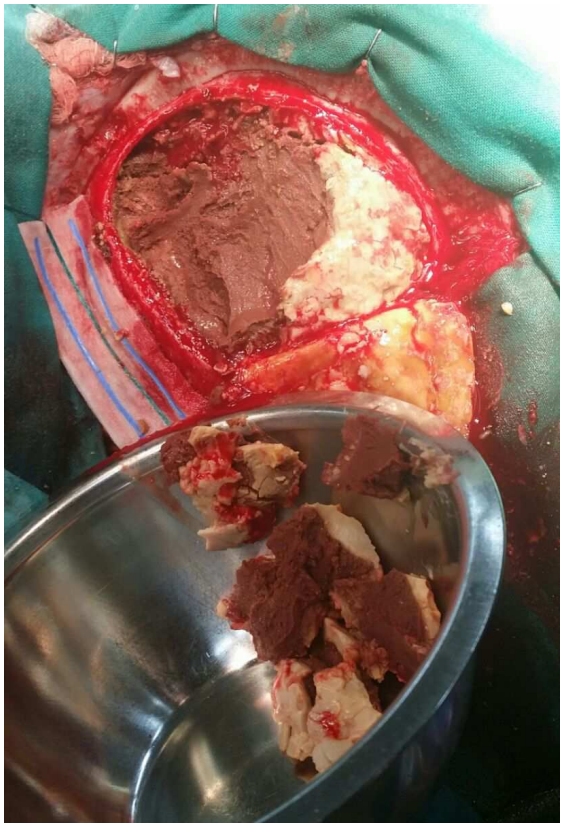

Fig 2: Showing partly excised sandy texture chronic epidural hematoma material

An elective craniotomy was performed under general anesthesia. Patient was initially positioned for right-sided VP shunt removal. The shunt was easily identified and its ventricular end came out freely. Thereafter, patient was positioned for left sided craniotomy and excision of lesion. The incision was marked with the help of Brain Lab. Skin flap was elevated and a three-burr hole large craniotomy was performed. Following elevation of bone flap, the outer layer of the lesion was opened. Underneath this layer, sandy texture chronic epidural hematoma material was found and evacuated as shown in Fig 2. On evacuating this sandy material, another thick covering was noted underneath it. This was gently removed (Fig 3). The dura present below this layer was left intact. After securing hemostasis, bone flap was carefully fixed in place and skin closed in layers. It was decided to leave the right side lesion for the present time.

Gross specimen included multiple sandy greyish material and yellow membranous tissue that measured 8.5 X 6.5 X 4.0 cm with a wall thickness of around 0.1 cm (Fig 4). On microscopic examination of the excised material, necrotic substance admixed with hemorrhagic material and cholesterol cleft was noted. Scattered inflammatory cells were seen including macrophages. Immunohistochemcal stains highlighted the scattered lymphocyte (CD 45 positive), and few macrophages (CD 68 positive).

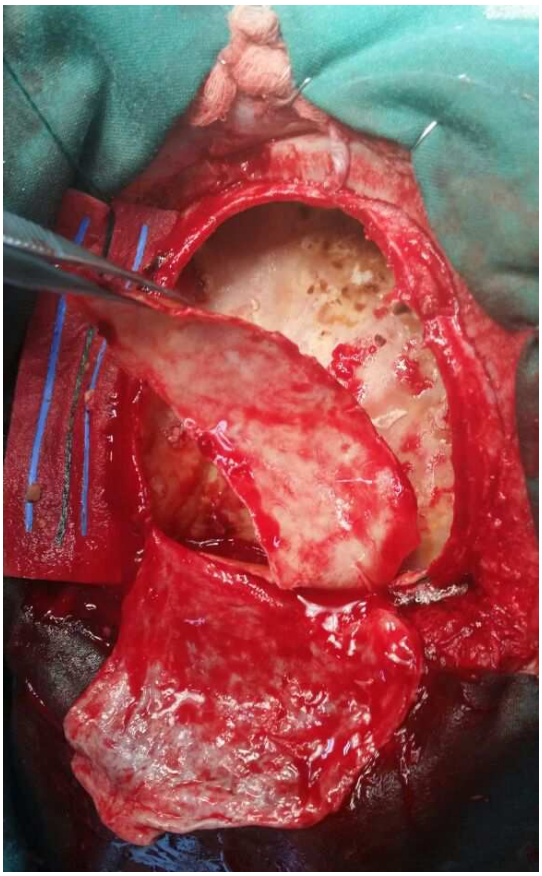

Fig 3: Showing an elevated craniotomy flap and the incised outer layer of the lesion. After evacuation the hematoma, underneath which yellow colored avascular lower layer of the lesion can be seen.

Section showed strips of fibro-collagenous tissue. This represented an outer and inner membrane of the organized hematoma. The outer membrane consisted of acute and chronic inflammatory cells while the inner membrane was composed of loose fibrous tissue with calcifications and lymphocyte collection. These findings were consistent with acute on chronic hematoma.

The patient made an uneventful recovery. Follow up CT done on the first postoperative day and repeated on the 16th day after the surgery showed significant resolution of the soft tissue fluid collection at the operative site along with reduction in mid line shift. Right frontal lesion with calcifications remained unchanged from preoperative CT (Fig 5). Patient was discharged 18 days after the surgery with GCS of 15/15 without any complications.

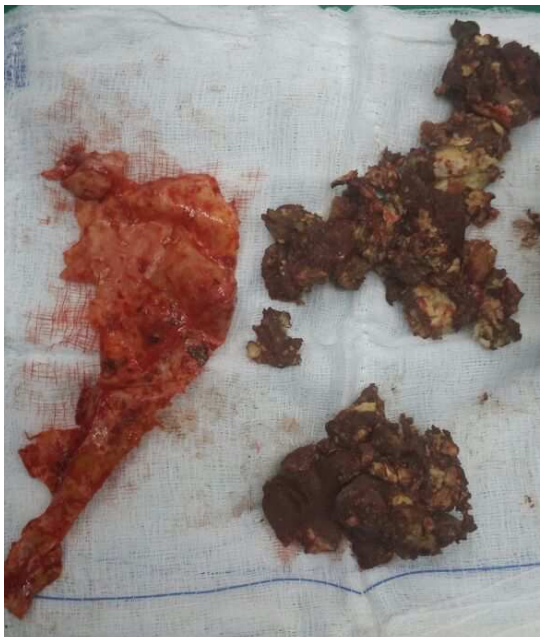

Fig 4: Showing multiple excised brownish materials with yellow membranous tissue

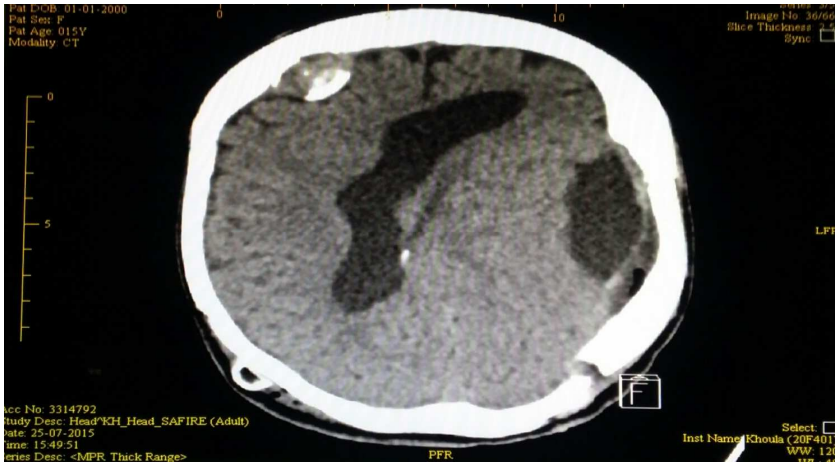

Fig 5: Follow up CT done 16 days after the surgery

Discussion

There are very few case reports of large chronic epidural hematoma following VP shunt surgery in literature [5, 6]. Such EDH, secondary to VP shunt, generally causes symptoms like headaches, vomiting, and somnolence. Surprisingly, our patient had a very large chronic EDH but with very subtle symptom of intermittent mild headache. This was considered a casual headache by the attending physician in the peripheral hospital and was treated with simple analgesic leading to missed diagnosis.

When a pediatric patient with a large hydrocephalous undergoes VP shunt, the size of the head does not necessarily decrease at the same pace as the brain does due to drainage of CSF especially when non-programmable VP shunts with uncontrolled drainage have been used like in the present case. This results in creation of space between the inner surface of the skull and the outer surface of the brain. This in turn generates a suction force on structures between the cortex and the inner table of the skull leading to bleeding due to

tearing of cortical bridging veins [5, 7, 8]. A similar hypothesis for the EDH after VP shunt is the stripping of dura from the inner surface of the skull as a result of drop in intracranial pressure with subsequent accumulation of epidural blood [6]. This may rarely occur spontaneously or may be associated with relatively trivial injury. The interesting finding in our patient was the absence of history of trauma and minimal symptoms despite the hematoma reaching a substantial size. The possible cause of the EDH in this patient may be attributed to over-functioning of the non-programmable shunt valve leading to disproportionate changes in head size and the brain and the resultant EDH.It is difficult to postulate the exact period during which the over-functioning of the VP shunt valve occurred in this patient. Unfortunately, the VP shunt that was removed could not be sent to the biomedical division for its functional assessment.

EDH may develop within days or weeks or even months after performing VP shunt [9]. The incidence of EDH following VP shunt has been quoted as 0.4% [10]. The largest series of EDH following ventricular drainage procedure reported in literature till date has been three cases by Sen and Hankinson [11].

The exact mechanism of calcification of the hematoma is poorly understood but it may be attributed to an inflammatory response to damaged vascularized tissue such as the dura [12]. There is another hypothesis that calcium is deposited under conditions of poor circulation or malabsorption of the hematoma [13].

In our patient, who underwent VP shunt surgery in 2001, programmable shunt valve was not used since it was not available at our center at that time. Use of this newer programmable shunt may help to reduce the problem of over-functioning VP shunts resulting in this complication. Subtle symptoms and delayed detection of post VP shunt hematoma like in the present case may be attributed to a reduction in CSF volume in the ventricles via the shunt leading to a slow rise in intracranial pressure and related symptoms.

Several suggestions have been made to prevent hematoma formation following VP shunt. These include: meticulous surgical technique, minimize spillage of CSF during VP shunt placement, use programmable pressure valves to prevent over drainage, and gradual placement of the patient in upright position [14].

Conclusion

Close follow-up of even subtle signs and symptoms with early postoperative CT scan following VP shunt surgery using programmable shunt is essential to detect and treat EDH in the early stages before it presents with a large epidural hematoma.

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