CASE REPORT

Vertex epidural hematomas caused by injury of the superior sagittal sinus

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Summary
We report on a case of vertex epidural hematomas with injury of the superior sagittal sinus due to a traffic accident. Imaging studies revealed skull fractures, subarachnoid hemorrhage, intracranial hemorrhage, epidural hematomas and injury of the superior sagittal sinus. On admission, his vital signs were normal except for pain at the site of fracture. Half an hour after admission, however, the patient’s Glasgow coma score suddenly dropped to E2M4V2. Emergent craniotomy with removal of hematomas was performed. Nine days after admission the patient had recovered well and was discharged with no neurological deficits.

Cases of traumatic brain injury have been reported before, and this type of clinical presentation with sudden change in consciousness is not rare. Vertex epidural hematomas with injury to the superior sagittal sinus requires special attention, however, because it has a varied clinical course. We present this case in the hope of helping others avoid misdiagnosing or delaying treatment in the future.

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1. Introduction

Vertex epidural hematoma (EDH) resulting from superior sagittal sinus injury is not rare, but may often escape detection. Head injuries are usually attended to in the emergency department. Brain computed tomography is a medical imaging method typically used to detect bleeding, brain injury and skull fractures. Unfortunately, sudden death also occurs in some head injury patients whose initial imaging studies and physical examinations show negative findings, and yet the subsequent autopsy may show unexpected vertex EDH. Diagnosis is often made by exclusion. We describe the features of a case of vertex EDH with superior sagittal sinus injury underlying a communicating skull fracture, and report our surgical technique during operation.

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2. Case report

A 14-year-old boy, previously healthy and robust, sustained a head injury and loss of consciousness following a motorcycle accident. He complained of a constant, and progressive, headache over 1 hour in the emergency department, and a growing soft mass was discovered on both sides of his frontotemporoparietal scalp. Initially, the patient was sent to Yee Zeen General Hospital where brain computed tomography (CT) revealed minimal intracerebral hemorrhage and EDH with skull fracture (Fig. 1A). Later, he was referred to our hospital.

On admission, his vital signs were normal and his Glasgow coma scale (GCS) was E4M6V5. Physical examination revealed considerable tenderness on both sides of his frontotemporoparietal scalp and deformity of the right lower leg. At first, 40 mg of ketorolac, a non-steroidal anti-inflammatory drug (NSAID), was administrated to relieve pain at the site of the fracture, the right distal fibula. The patient’s right leg pain improved, but nausea and headache recurred 10 minutes later and persisted. The GCS, when rechecked, was still E4M6V5. He obeyed our orders, but inattentively answered our questions. Isocoric pupils, 2.5 mm in size with prompt light reflex, were disclosed. Extraocular muscle functioning was normal. Muscle power and deep tendon reflexes were normal. Twenty-five minutes later, he started vomiting, and became lethargic. Then, a sudden change in consciousness occurred. The patient became drowsy, and his GCS dropped to E2M4V2 half an hour after admission. Isocoric pupils, 4.0 mm in size and lacking light reflex, were disclosed. He was intubated and had a repeated brain CT (Fig. 1B). The imaging studies revealed intracerebral hemorrhage and progressive EDH. About 40 minutes after admission, bradycardia and hypertension developed and increased intracranial pressure was noted that was attributed to the mass effect of hematomas. Emergent surgery was performed following a complete and detailed explanation of the therapeutic plans to his family.

During the operation, a comminuted fracture of the frontal and parietal bones at the high vertex was discovered (Fig. 2A). The EDH and intracerebral hematoma were

Figure 1  A: Epidural hematomas in the left frontotemporoparietal region. B: After admission, intracranial hemorrhage and epidural hematomas became larger, crossing the midline.
located in both frontal regions. Generalized subgaleal hematoma and soft tissue swelling in both frontotemporoparietal regions, being more severe on the right-hand side, were also noted. At this point, tear of the superior sagittal sinus caused by a hematoma overlaying the sinus was suspected (Fig. 2B). First, the bone fragments were gently removed and bone wax and Gelfoam were applied to stop blood oozing from the bone. Then the hematoma in the high vertex region was removed piece-by-piece to avoid active bleeding related to the superior sagittal sinus injury. A blood clot layer about 3 mm thick overlaying the superior sagittal sinus was left in place (Fig. 2C). Finally, Surgicel was used to cover the superior sagittal sinus region (Fig. 2D) No active bleeding occurred throughout the whole operation. The bone defect due to craniotomy was repaired with silk and a miniplate was applied for internal fixation (Fig. 2E). The patient recovered well and was discharged without neurological deficits 9 days after admission.

Figure 2  A: Comminuted fracture at the high vertex. B: Blood clots covering the superior sagittal sinus. C: Epidural tack-up sutures placed from the dura to the craniotomy bone edge. A layer of blood clots were left on the superior sagittal sinus to prevent rebleeding. D: Pieces of Surgicel were inserted between the dura and the skull. Surgicel was used to avoid active bleeding. E: Surgicel was left in place by passing a few interrupted sutures between the epicranium and dura over the bone. Craniotomy was completed and the bone defect was repaired.
3. Discussion

Vertex EDH is frequently encountered in the emergency department. The incidence of vertex EDH has been reported to be 8%, with a reported mortality rate of 50% if these lesions are misdiagnosed. On account of its anatomical relation, if the vertex fracture line crosses the sagittal suture and causes diastasis, it usually involves the groove underlying the superior sagittal sinus and middle meningeal vessels, and then induces vertex EDH. Symptoms can be almost acute.2

In order to explore the cause of the rapid change in the consciousness of our patient, the literature with regard to vertex EDH and sagittal sinus injury was reviewed. Injury to the sagittal sinus and vertex EDH may change the intracranial pressure. It is believed that cerebrospinal fluid drains through the arachnoid granulations into the dural venous blood of the sagittal sinus.4 Vertex EDH may compress the sagittal sinus and compromise cerebral venous drainage, so the absorption of cerebrospinal fluid is impaired, which may be related to the presence of venous blood hypertension. Hydrocephalus and restricted blood supply then occur. Thus, patients may present features of increased intracranial pressure. If intracranial hypertension develops, it can induce uncal herniation and reflex bradycardia, which may then result in sudden death. Due to the impairment of venous drainage and diminished cerebral venous fluid resorption, vertex EDH caused by injury to the sagittal sinus requires aggressive surgical treatment, even with a relatively small hematoma.

Vertex EDH is easily misdiagnosed, even when a brain CT is performed immediately. Conventional axial plane CT is likely to miss the vertex EDH, and the presence of a small vertex EDH is commonly misdiagnosed as a bone artifact. Magnetic resonance imaging (MRI) venography is suggested when the vital signs of the patient are relatively stable. MRI venography is more sensitive and there is less chance of a misdiagnosis occurring, in terms of detecting obstruction of the superior sagittal sinus, than with conventional axial plane CT. Most cases of head injury with EDH are complicated, however, due to a critical condition and deteriorating consciousness. A time-consuming MRI venography may prevent these patients from obtaining a definitive diagnosis. Follow-up MRI venography is appropriate after surgery. If the patient’s vital signs are relatively stable and small vertex EDHs are difficult to diagnose in routine CT, MRI can be an excellent tool to solve this problem in patients with trauma to the skull vertex.6

The other challenging event in the operation our patient underwent was how to deal with profuse sinus bleeding. Massive hemorrhaging will disturb the operative exposure. The literature on venous sinus injury in the past 30 years was reviewed. Small holes or tears of the venous sinus can be managed with Surgicel or Gelfoam and gentle pressure. Temporary control of hemostasis is easily obtained by packing pledges of hemostatic materials. Direct closure of tears can be performed if it does not result in sinus stenosis. For larger ruptures, patch repair using a vein graft, pericranium or the fascia lata is required, with two hemi-running sutures. If the hemorrhage is out of control, ligation can be performed in non-critical areas (in the first quarter of the superior sagittal sinus). A temporary sinus—sinus shunt may be necessary to properly repair the sinus tear without compromising sinus blood flow. Massive red cell transfusions are required in such cases.

There are some respectable technical aspects, as follows, that may greatly facilitate proper management. Before elevation of the bone fragments, preparations for rapid hemorrhage and air embolism should be in place and an assistant should be ready to maneuver the operating table at a moment’s notice. Continuous generous irrigation over the sinus during elevation of the bone fragments reduces the chance of embolism. Wet swabs should be at hand to immediately cover the sinus. The semi-sitting (lounging) position allows a good venous return without increased intracranial pressure. The operative exposure should be as extensive as possible. The skin flap and craniotomy should extend across the midline to permit visualization of both sides of the sinus. The bridging veins, especially in the rolandic outflow area, should be preserved. To facilitate venous sinus patency after surgery, blood pressure, volume and viscosity must be carefully monitored.

Compared with EDH with no injury of the sagittal sinus, vertex EDH with injury of the sagittal sinus has a more varied clinical course. In this case, surgery was performed in a short time and the patient had a good outcome. In conclusion, vertex EDH with sagittal sinus injury requires keen attention and aggressive surgical treatment whenever possible to reduce the mass effect and to provide sufficient venous circulation in the brain.

References