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Case Report

Acute epidural vertex hematoma with good hemostasis using delayed surgery after monitoring of coagulation and fibrinolytic parameters: A case report

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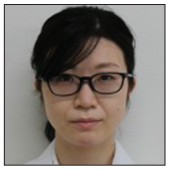

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ABSTRACT

Background: The appropriate timing and method of surgery for vertex epidural hematoma (VEDH) are uncertain due to the presentation and slow symptomatic exacerbation caused by bleeding from a venous origin involving the injured superior sagittal sinus (SSS). Coagulation and fibrinolytic disorders that occur after traumatic brain injury also worsen bleeding. For these reasons, it is challenging to decide the surgical procedure and timing of surgery.

Case Description: A 24-year-old man involved a car accident and was transported to our emergency department. He was unconscious but not lethargic. Computed tomography showed VEDH overlying the SSS, and hematoma increased temporarily. Due to abnormal coagulation and fibrinolysis at admission, he underwent intentionally delayed surgery after control of coagulation and fibrinolysis. Bilateral parasagittal craniotomy was chosen to ensure hemostasis from the torn SSS. The patient improved without complications and was discharged with no neurological deficit. This case indicates that this surgical strategy is favorable for VEDH with slow symptomatic progression.

Conclusion: VEDH is mostly caused by bleeding from the injured SSS secondary to diastatic fracture of sagittal suture. Intentionally delayed surgical intervention using bilateral parasagittal craniotomy after stabilization of coagulation and fibrinolysis is favorable for prevention of further hemorrhage and good hemostasis.

Keywords: Coagulation, Epidural vertex hematoma, Fibrinolysis, Parasagittal craniotomy, Traumatic brain injury

INTRODUCTION

Vertex epidural hematoma (VEDH) is a rare condition that accounts for 1–8% of all intracranial extradural hematomas.[1] The surgical procedure and timing of surgery are not established. Conservative treatment is chosen because most cases initially have no symptoms, but delayed neurological deterioration frequently occurs and many cases eventually require surgical intervention. Since the injured superior sagittal sinus (SSS) is the source of bleeding, a surgical technique is required that avoids further tearing and bleeding of the SSS. Coagulation and fibrinolytic disorders that occur after traumatic brain injury also worsen bleeding, which causes intraoperative hemorrhage. In the case reported here, we performed intentionally delayed VEDH evacuation by bilateral parasagittal craniotomy while monitoring coagulation and fibrinolytic parameters, and obtained a successful outcome with good hemostasis.

CASE REPORT

Clinical presentation

A 24-year-old man had an accident involving a vehicle while walking and was transported to our emergency department. On arrival, his level of consciousness had deteriorated (Glasgow Coma Scale: 13 [E3V4M6]). He was not lethargic, and his pupils were equal and round, and reactive to light and movement. Computed tomography (CT) showed traumatic subarachnoid hemorrhage, a 1-cm thick VEDH overlying the SSS, and a parietal skull fracture reaching a diastatic fracture of the sagittal suture [Figure 1]. Head CT 1 h (hour) later showed increased hematoma, but there was no enlargement another hour later and the patient had no exacerbation of neurological symptoms [Figures 2]. Plasma fibrinogen concentration at admission (304 mg/dL) was within the normal range, but D-dimer concentration was high (56.1 μg/mL). Since abnormal coagulation and fibrinolysis are likely in the acute phase, and delayed neurological deterioration might be caused by a general increase in VEDH, we planned intentionally delayed surgery after coagulation and fibrinolytic factors stabilized. Six units of fresh frozen plasma (FFP) were administered and fibrinogen concentration at 3 and 6 h after injury was maintained at >150 mg/dL [Figure 3]. There was no coagulation abnormality during the course and the decision was made to evacuate the hematoma 6 h after injury.

Surgical procedure

Under general anesthesia, the patient was placed in the supine position with the head in a neutral position and fixed in a Mayfield clamp. A horseshoe skin incision overlying the lesion was sketched [Figure 4]. Because the SSS was thought to be injured by diastatic fracture over the sagittal suture, we performed bilateral parasagittal craniotomy, while leaving a midline island of bone including the peritoneal fracture and diastatic fracture. Dural tenting sutures and Surgicel® (Johnson and Johnson, New Brunswick, NJ, USA) were used to avoid bleeding from the torn SSS. The bone flaps were removed and a large bifrontal extradural hematoma was identified. The hematoma was sucked out and evacuated with suction and irrigation. The bone flaps were replaced and secured with titanium miniplates. The skin flap was closed in two layers and a subcutaneous wound drain was placed.

Postoperative course

After surgery, the patient was returned to the intensive care unit with intubation and sedation. Postoperative head CT showed no residual hematoma and no recurrence of hemorrhage and contusion [Figure 5]. This suggested that sufficient decompression had been obtained by evacuation. The plasma fibrinogen concentration was within the normal postoperative range [Figure 3]. The patient woke from anesthesia uneventfully and was extubated on the day after surgery. His level of consciousness was clear and the postoperative course was uneventful. He was discharged to home 21 days after surgery with no neurological deficits.

DISCUSSION

VEDH accounts for 1–8% of intracranial extradural hematomas.[1] The clinical symptoms and course of VEDH are atypical.[3] This makes it difficult to determine the optimal timing and surgical technique, and a surgical strategy for VEDH has not been established. In our case, VEDH of the SSS was treated successfully with good hemostasis using bilateral parasagittal craniotomy after coagulation and fibrinolysis were stabilized. Our experience may help in defining the appropriate timing of surgery and surgical procedure for VEDH.

VEDH is usually caused by venous bleeding from the SSS and slowly progresses. Most cases initially have no symptoms, but delayed neurological symptoms frequently appear. Kim *et al.* found that 71.4% of cases were initially managed with observation without surgery, but that delayed neurological deterioration then occurred in 44% of these patients and that 40% eventually required surgical intervention.[3] Therefore, VEDH has a high rate of required surgery, but intentionally

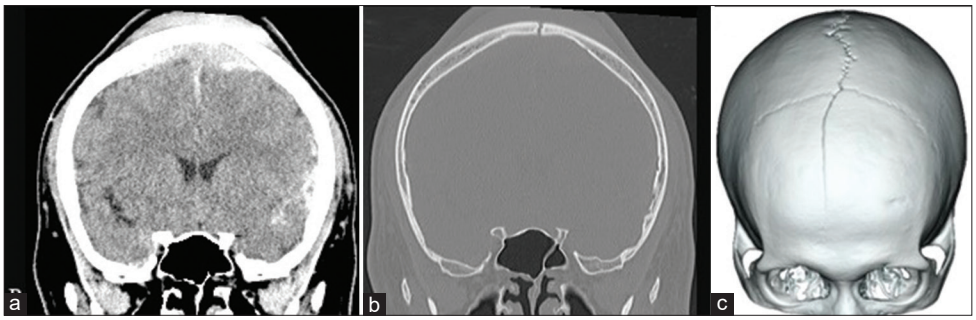

Figure 1: Head computed tomography on admission. (a) Traumatic subarachnoid hemorrhage and a 1-cm thick vertex epidural hematoma overlying the superior sagittal sinus. (b and c) Parietal skull fracture reaching a diastatic fracture of the sagittal suture.

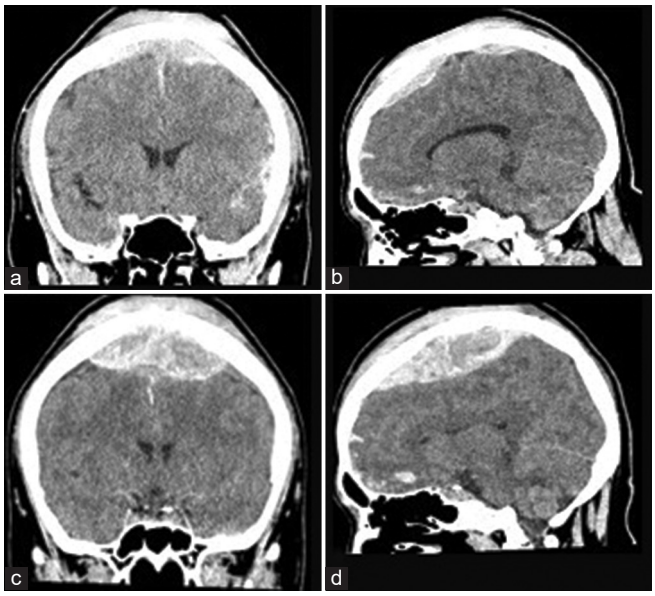

Figure 2: Coronal (a, c) and sagittal (b, d) head computed tomography. (a and b) Hematoma overlying the superior sagittal sinus at arrival. (c and d) The hematoma increased 1 h later.

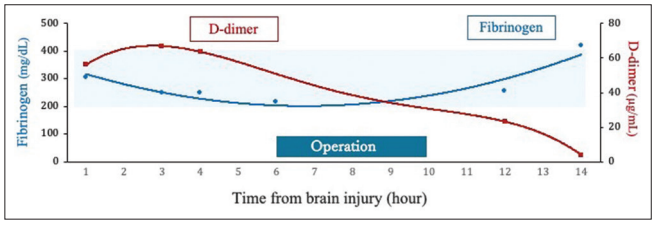

Figure 3: Time course of coagulation and fibrinolytic parameters. D-dimer increased progressively between admission and 3 h after injury and decreased after 3 h. Plasma fibrinogen decreased between admission and 3 h after injury but was maintained at >200 mg/dL.

delayed surgical decompression can be used due to the slow symptomatic exacerbation.

Among patients with traumatic brain injury (TBI), 63% have coagulopathy on admission, and this is associated with progressive intracranial brain contusions and contributes to a poor outcome.[4] The mechanisms of coagulopathy in isolated TBI are thought as follows: Tissue factor released from the damaged brain generates thrombin, and fibrinogen is consumed and converted to fibrin, which promotes a hypercoagulable state. D-dimer is generated during degradation of fibrin. Furthermore, tissue-type plasminogen activator released from damaged brain tissue results in an increase in plasmin converted from plasminogen and promotes hyperfibrinolysis.[4]

Hemostatic disorders that occur following TBI increase within 3 h after brain injury and a hypercoagulable stage follows 6 h after injury.[5,7] The time course of coagulation and fibrinolytic parameters such as D-dimer and plasma fibrinogen indicates the prognosis in the acute phase of TBI.

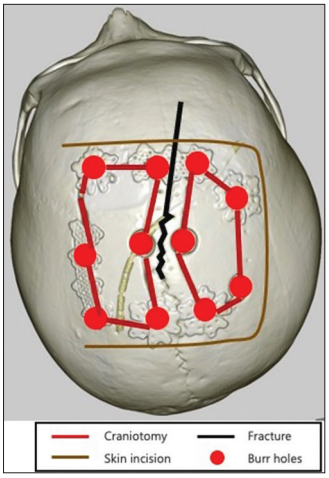

Figure 4: Illustration of the operative view. A horseshoe skin incision overlying the lesion was sketched. Burr holes were placed on each side and parasagittal craniotomy was performed, leaving a midline island of bone including the peritoneal fracture and diastatic fracture.

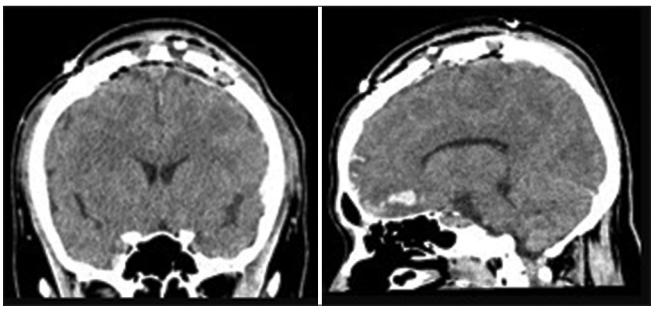

Figure 5: Coronal (a) and sagittal (b) head computed tomography. Postoperatively, there was no residual hematoma, no recurrence of contusion, and decompression of brain tissue.

D-dimer as a fibrinolytic parameter increased progressively between admission (within 1 h after injury) and 3 h after injury and remained elevated for at least 12 h. Plasma fibrinogen significantly decreased between admission and 3 h after injury and, then, showed a non-significant increase by 6 h before significantly increasing from 6 to 12 h.[5] These results indicate that patients presenting within 3 h after injury have a high risk of hemorrhage expansion due to hyperfibrinolysis. Nakae *et al.* found that outcomes of isolated TBI were significantly better in cases with high fibrinogen than in those with low fibrinogen and emphasized the importance of maintaining plasma fibrinogen at ≥150 mg/dL at 3 h after injury during FFP transfusion.[6] To achieve this target, it is important to predict possible reduction of fibrinogen from admission to

3 h after injury. An abnormal D-dimer level occurs earlier than the decrease in plasma fibrinogen,[7] and D-dimer levels are significantly negatively correlated with fibrinogen levels between admission and 3 h after injury.[5] This suggests that, to maintain plasma fibrinogen at ≥150 mg/dL, FFP transfusion should be considered if D-dimer is elevated at admission, even if fibrinogen at admission is within the normal range.[6]

Surgical intervention in the acute phase increases the risk of hemostasis and progression of intracranial brain contusions due to coagulation and fibrinolytic disorders. In a case of VEDH with slow symptomatic exacerbation, it is preferable to perform intentionally delayed surgery after stabilizing coagulation and fibrinolysis. There are two main options for the surgical technique: single-flap vertex craniotomy or bilateral parasagittal craniotomy. Kim *et al*. reported that there was no significant difference between these methods in 40 cases.[3] Fernandes-Cabral *et al*. emphasized that the most important step in evacuation of a VEDH is to spare the midline anatomy from craniotomy to ensure a stable anchor point for tacking-up the underlying displaced dura and SSS, preventing further extensive bleeding from the diastatic fracture, and eliminating the risk of further tearing of the injured sinus during bone flap elevation.[2] Kim *et al*. concluded that bilateral parasagittal craniotomy should be regarded as the initial treatment option, because the midline bony island can be easily removed in a case needing direct repair of the SSS, and this technique can be safely performed and easily changed to vertex craniotomy.[3] A case of a VEDH with an injured SSS is likely to have further bleeding from diastatic fracture and tearing of the SSS, and bilateral parasagittal craniotomy is the preferred choice for good hemostasis.

CONCLUSION

VEDH is mostly caused by bleeding from the injured SSS secondary to diastatic fracture of sagittal suture. Surgical management is difficult due to exacerbation of hemorrhage in the perioperative period. Intentionally delayed surgical intervention using bilateral parasagittal craniotomy after stabilization of coagulation and fibrinolysis is favorable for prevention of further hemorrhage and good hemostasis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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