Case Report

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Delayed Onset Acute Subdural Hematoma after Burr Hole Drainage in a Patient with Chronic Subdural Hematoma and Liver Cirrhosis

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Conflict of Interest

The authors have no financial conflicts of interest.

ABSTRACT

Although acute intracranial bleeding after burr hole drainage for chronic subdural hematoma (SDH) is rare, it could still occur and is associated with a poor clinical outcome. Although rare, most of them occur immediately or within a few days after drainage, especially in patients who are on antiplatelet drugs or anticoagulants. We report an unusual case of delayed-onset acute SDH that developed 14 days after burr hole drainage of chronic SDH in a 54-year-old man with liver cirrhosis and thrombocytopenia. The possible pathophysiological mechanisms of this rare entity are discussed, and the relevant literature is reviewed.

Keywords: Subdural hematoma; Delay; Liver cirrhosis

INTRODUCTION

Chronic subdural hematoma (SDH) is frequently encountered in the neurosurgical field and usually occurs in elderly patients. Burr hole trephination is one of the most commonly performed procedure for chronic SDH because of its simplicity, low recurrence rate, and low morbidity.

The patients usually have a good prognosis with simple burr hole trephination under local anesthesia.^{1,7,16)}

However, unexpected neurological deterioration caused by acute intracranial bleeding may complicate the postoperative course after burr hole trephination for chronic SDH. Most cases of acute intracranial bleeding develop immediately or within a few days after burr hole trephination. Bilateral or contralateral development of an acute SDH immediately after burr hole trephination has been reported to be a rare but serious and rapidly worsening complication.^{14,17} They usually are reported rarely, especially in patients who are on anticoagulants or antiplatelets.¹⁰ Liver cirrhosis (LC) and its related complications have been identified as higher risk factors for increased morbidity and mortality.^{2,8}

However, to the best of our knowledge, delayed onset acute SDH caused by LC and thrombocytopenia that is different from usual recurrence of chronic SDH has been extremely rare and only few cases have been reported.

Here, we report an unusual case of delayed onset acute SDH after evacuation of chronic SDH using burr hole trephination in a patient with liver cirrhosis and thrombocytopenia.

CASE REPORT

A 54-year-old man was admitted to our institute without any history of head injury with complaints of severe headache and left-side motor weakness. Neurologic examination revealed left hemiparesis (Grade V/IV). He had been under treatment for liver cirrhosis in local clinic since two years ago but he was not on antiplatelets or anticoagulants. He was afebrile and routine laboratory test results, which indicated severe thrombocytopenia, revealed a platelet count of 40,000 (normal range 150,000–400,000), prothrombin time of 16.4 seconds (normal range: 9.4–12.5 seconds), activated partial thromboplastin time of 37.2 seconds (normal range, 28–44.0), and prolonged international normalized ratio (INR) (1.46; normal range, 0.9–1.27). A brain computed tomography (CT) scan revealed chronic SDH in the right frontotemporoparietal (FTP) region with the loss of sulci markings (FIGURE 1A). A hemostatic agent, Tachosil[®] was used to close the burr hole site to prevent bleeding from the scalp into the intracranial cavity.

He underwent evacuation of hematoma through burr hole trephination using a 5-L catheter under local anesthesia. Dark old blood was removed and there was no evidence of active bleeding during the operation. The catheter was placed into the subdural space for further drainage (**FIGURE 1B**).

Postoperatively, the patient showed improvement in headache and motor weakness and he was able to ambulate independently without difficulty. Brain CT scan taken 2 days after surgery showed marked resolution of hematoma with improved midline shifting (**FIGURE 1C**). He was discharged 7 days after burr hole trephination without complaining of any symptoms. However, he was re-admitted to the emergency room 14 days after burr hole trephination. He denied any another injury after discharge. The patient's level of consciousness deteriorated to a stuporous state and a brain CT scan revealed newly developed acute SDH on Rt. FTP region (**FIGURE 2**).



FIGURE 1. Brain CT scans of the patient.

(A) Initial brain CT scan reveals a hypodense lesion in the right frontotemporoparietal region, suggesting chronic SDH with midline shift. (B) Postoperative CT scan taken the next day after burr hole trephination shows good positioning of drainage catheter without acute SDH. (C) Postoperative CT scan taken 2 days after burr hole trephination shows resolution of hematoma with improved midline shifing. CT: computed tomography, SDH: subdural hematoma.

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FIGURE 2. Brain computed tomography taken 14 days after trephination reveals acute subdural hematoma on Rt. F-T area with more midline shifting.



FIGURE 3. Follow-up computed tomography scan taken 6 months after emergent craniotomy and hematoma removal shows complete removal of acute subdural hematoma without midline shifting.

The hematoma was removed through emergent craniotomy and hematoma removal under general anesthesia. In the surgical field, there was no evidence of active bleeding, but large subdural clot suggesting acute bleeding was observed. After emergent surgery, the patient's mental status recovered to near-alert, and a follow-up CT scan revealed hematoma removal with improved midline shifting (**FIGURE 3**). At the final follow-up 6 months after emergent craniotomy, his mental status recovered to alert. The patient recovered fully without any neurologic sequelae except intermittent headache.

DISCUSSION

Chronic SDH is a common pathology that tends to occur in elderly patients. Burr hole trephination under local anesthesia is the simplest and one of the most commonly performed procedure for the initial treatment of chronic SDH.^{11,13)}

Chronic SDH usually has a favorable outcome and good prognosis, and many reports suggest that gradual drainage of hematoma by placing a drainage catheter in the subdural space can further reduce the recurrence rate. However, possible complications related to this type of drainage include acute intracranial hemorrhage, infection, recurrence, and hydrocephalus.¹¹

Moreover, chronic SDH patients' comorbidities can result in adverse clinical outcomes due to the development of postoperative acute intracranial bleeding. Acute intracranial bleeding such as ipsilateral or bilateral acute SDH after burr hole trephination may occur immediately after surgery and is usually associated with direct cortical injury or rapid brain parenchymal shift after drainage of hematoma.^{13,17}

Obviously, antiplatelet and anticoagulant medications are a potential risk factor for acute hemorrhage.¹⁰⁾ Hematological diseases as an underlying comorbidity induce thrombocytopenia, and coagulopathy can also be a risk factor for the development of acute intracranial bleeding.³⁾

Liver cirrhosis-related comorbidities may be linked to acquired thrombocytopenia and coagulopathy resulting from hypersplenism, impaired liver function with decreased fibrinogen and increased fibrinolysis, damaged systemic vessel walls, and deficient platelet aggregation and activation of the clotting cascade.^{4,9)}

The exact pathophysiologic mechanisms of delayed onset acute SDH after evacuation of chronic SDH remain to be elucidated but it is already conceived that many pathological factors contribute to the development of delayed acute SDH. For instance, involving weakness of vessel walls, damage to the cerebral vasculature secondary to perioperative parenchymal shift, a sudden and rapid increase in cerebral blood flow combined with defective vascular autoregulation, and hemorrhage into previously undetected contusions have been suggested as possible causes of delayed acute SDH.^{5,6}

Furthermore, thrombocytopenia and aggravated coagulopathy might have a profound influence on delayed acute SDH in our patient.

Even though we could not find any bleeding focus in our case, a torn bridging vein might have damaged the cerebral vasculature and triggered delayed developed acute SDH which became clinically apparent only after more than 14 days following minimal burr hole trephination. Schmidt, et al.¹⁵) reported that the recurrence risk of subdural hematoma is largely limited to the first year, and the characteristics of patients including co-morbidities greatly influence the recurrence risk of SDH. The cumulative risk of recurrent SDH was 9% at 4 weeks and stabilising at 14% after one year. Predictors related to recurrence were male sex, older age, alcohol addictions diabetes mellitus and trauma diagnoses.¹⁵)

In our patient, despite severe thrombocytopenia and impaired coagulation function caused by liver cirrhosis, the deteriorated mental status resulted from delayed development of acute SDH required open craniotomy and hematoma removal to minimize mortality rather than repeated burr hole trephination. Regrettably, due to the improved symptoms before discharge, the CT scan was not performed at 7 days after surgery, which is known to one of the predictable factors of recurrence rate of chronic SDH to evaluate brain re-expansion rate.¹²

Coagulopathy and poor prognosis may be linked to the severity of liver cirrhosis, which is caused by related systemic vascular injuries and multiple organ failures.

Therefore, we believe that patients who could undergo open craniotomy for delayed onset acute SDH after burr hole trephination must be carefully selected because the procedure requires favorable conditions, normal coagulation function, and strong support of the whole family in spite of good surgical result in our patient.

CONCLUSION

Delayed onset acute SDH that occurred 14 days after evacuation of chronic SDH in a patient with liver cirrhosis is an unusual complication. Intensive and aggressive monitoring over time accompanying careful therapeutic intervention appears to be mandatory to prevent poor outcomes in patients with chronic SDH and liver cirrhosis.

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