

CASE REPORT

Postoperative hematoma involving brainstem, peduncles, cerebellum, deep subcortical white matter, cerebral hemispheres following chronic subdural hematoma evacuation

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ABSTRACT

Among the intracranial hematomas, chronic subdural hematomas (CSDH) are the most benign with a mortality rate of 0.5-4.0%. The elderly and alcoholics are commonly affected by CSDH. Even though high percentage of CSDH patients improves after the evacuation, there are some unexpected potential complications altering the postoperative course with neurological deterioration. Poor outcome in postoperative period is due to complications like failure of brain to re-expand, recurrence of hematoma and tension pneumocephalus. We present a case report with multiple intraparenchymal hemorrhages in various locations like brainstem, cerebral and cerebellar peduncles, right cerebellar hemisphere, right thalamus, right capsulo-ganglionic region, right corona radiata and cerebral hemispheres after CSDH evacuation. Awareness of this potential problem and the immediate use of imaging if the patient does not awake from anesthesia or if he develops new onset focal neurological deficits, are the most important concerns to the early diagnosis of this rare complication.

Key words: Chronic SDH evacuation, intra-axial hemorrhage, intraparenchymal hemorrhage, postoperative hemorrhage

Introduction

Among the intracranial hematomas, chronic subdural hematomas (CSDH) are the most benign with a mortality rate of 0.5-4.0%.^[1-3] The elderly and alcoholics are commonly affected by CSDH. Even though high percentage of CSDH patients improves after the evacuation, there are some unexpected potential complications altering the postoperative course with neurological deterioration. Spontaneous intracerebral hematoma (ICH) following evacuation of the CSDH is rare, but potentially lethal complication with a reported incidence of 0.7-4.0%.^[2-5] Review of literature showed 28 cases. We present a

case report with multiple hemorrhages in brainstem, cerebral and cerebellar peduncles, right cerebellar hemisphere, right thalamus, right capsulo-ganglionic region, right corona radiata and cerebral hemispheres after CSDH evacuation. This case is unique in that it involved almost all the territories of the brain.

Case Report

A 48-year-old man known hypertensive with blood pressure under control was admitted to our institution 6 weeks after minor head trauma caused by fall. He had complained of progressive frontal headache and became confused. There was no history of vomiting, weakness, sensory complaints and bowel or bladder disturbances. Glasgow Coma Scale (GCS) of the

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patient was E4M6V4 (14/15). A computerized tomography (CT) scan showed bilateral fronto-temporo-parietal large, mixed dense, CSDHs with multiple septations. There was ventricular compression on left side with shift of the midline structures to right [Figure 1]. The subdural collections were treated through left fronto-temporo-parietal craniotomy, excision of membranes with evacuation of subdural hematoma on left side and evacuation of the subdural hematoma with burr holes on right side followed by closed-system drainage. During the surgery he had transient increases in arterial pressure up to 180/100 mmHg with fluctuations. In the immediate post operative period,

patient could not be extubated with GCS of E1M2VT. Patient had spontaneous respirations with stable blood pressure but showed right-sided pupil dilatation without reaction to light. Immediately patient shifted to magnetic resonance imaging (MRI) which showed multiple large mixed intensity areas involving brainstem, cerebral and cerebellar peduncle, right cerebellar hemisphere, right thalamus, right capsulo-ganglionic region, right corona radiata and adjacent frontal lobe causing effacement of 3rd and 4th ventricles [Figure 2] with blooming of all these areas in gradient images [Figure 3] suggestive of intraparenchymal hemorrhages. MRI also showed right

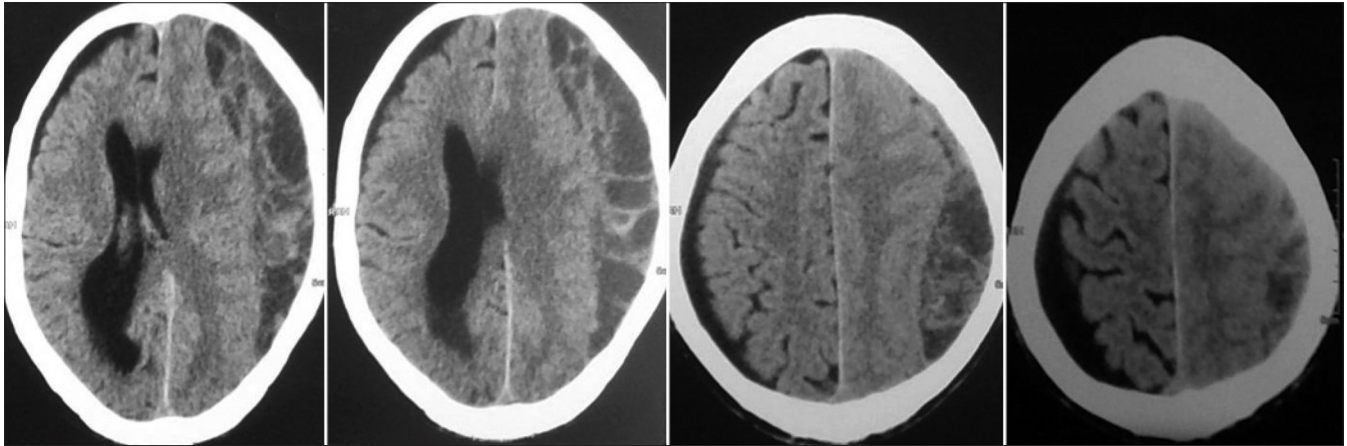


Figure 1: Preoperative computerized tomogram showing bilateral fronto-temporo-parietal subdural hematomas with multiple septations

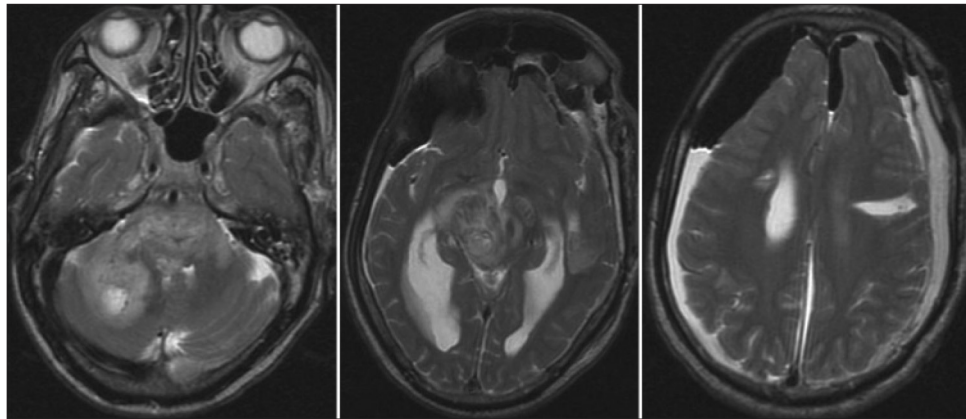


Figure 2: Immediate postoperative MRI showing multiple mixed intensity lesions with edema and pneumocephalus with ventricular compression

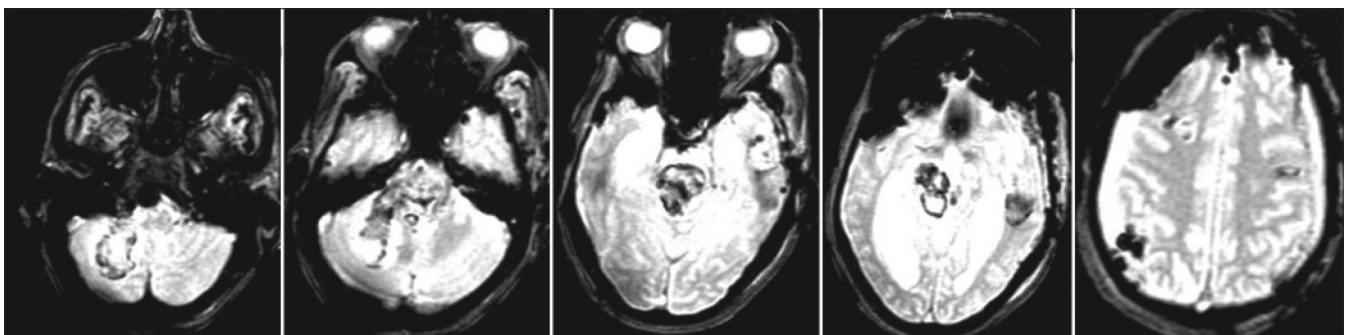


Figure 3: Postoperative MRI gradient images showing blooming of the lesions in Figure 2 suggestive of hemorrhages

frontal pneumocephalus. Patient was evaluated for coagulation disorders, which were negative. The patient was further managed conservatively. On postoperative day 3 imaging was repeated which showed no increase in hematoma and decrease of pneumocephalus. After a complicated postoperative course he expired on postoperative day 7.

Discussion

Review of literature showed spontaneous ICHs complicating surgical evacuation of CSDHs. Following the evacuation of subdural hematoma intra-axial hemorrhages were noted in the following sites: Brainstem,^[6] cerebellum,^[7] cerebral hemispheres^[8] ventricles.^[9] Symptoms of the ICH appeared in immediate postoperative period^[4,10-13] or several days after the event.^[2,4,5,11,14] In all the cases the hematoma developed in the ipsilateral hemisphere, but in our case the hematomas were multiple and appeared bilaterally with large part on the contralateral side. This complication usually lead to the poor outcome in these patients^[4,5,10-12] with a fatal outcome reported in one-third of the patients, with another third severely disabled.^[4,5,11,14,15]

The theory of rapid perioperative parenchymal shift causing direct vascular damage fit well with the phenomenon of ICH formation in our patient, who had previous bilateral subdural collections with shift of the midline structures. The pathogenic mechanism that seems most likely to be responsible for these hemorrhages may involve a sudden increase in cerebral blood flow combined with defective vascular autoregulation. Labile hypertension and wide swings in blood pressure during operation, as in our patient, may be contributory. Preoperative cerebral blood flow in patients with CSDH is known to be uniformly decreased over the compressed brain. Surgical decompression allows cerebral blood flow to return to normal values.^[16] We hope that sudden restoration of normal perfusion pressure in areas of defective cerebral vascular autoregulation due to subcortical swelling underlying surface compression,^[17] focal impedance of the venous drainage, or ischemic loss of CO₂ reactivity^[18] might in turn lead to the vascular damage that resulted in intraparenchymal hemorrhage.

Those previous theories are supported by recent Single positron emission computed tomogram (SPECT) studies, which showed there is ipsilateral cortical and subcortical hyperemia especially in the elderly age group.^[19] Ogasawara *et al.*, showed postoperatively, there is a progressive normalization of the blood flow to these areas.^[19] Physiological aging of the vessels with increased fragility may not able to sustain the rapid alterations in the blood pressures in the immediate postoperative period. Seizures are an important factor in these patients. Grunwald *et al.*^[20] revealed an actually increased uptake in the epileptic focus following a Tc99 ECD SPECT scan.

Poor prognosis of the patient in the postoperative period is due to complications like failure of brain to re-expand, recurrence of hematoma and tension pneumocephalus. Previous reports showed the occurrence of postoperative hematoma in one of the location of the previously mentioned sites. The unique feature in this case report is occurrence of the hematoma in all the above-mentioned locations. This may be due to the large amount of SDH, long duration of symptoms along with massive midline shift before evacuation, in turn those further leads to changes in the perfusion of the brain. In this regard we stress the need for slow decompression of CSDHs, possibly with controlled re-expansion, careful control of the blood pressure lability with a gradual emergence from anesthesia.

Conclusions

Awareness of this potential problem and the immediate use of imaging if the patient does not awake from anesthesia or if the patient develops new onset focal neurological deficits are the most important concerns to the early diagnosis of this rare complication. Slow decompression of CSDHs, possibly with controlled re-expansion is advised and careful control of the blood pressure lability with a gradual emergence from anesthesia.

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Conflicts of interest

There are no conflicts of interest.

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