



An Unusual Presentation of Chronic Subdural Hematoma—“Subdural Mud”

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Abstract

Chronic subdural hematoma (SDH) is widely seen in neurosurgical practice, however, the incidence of calcification or ossification in chronic SDH is a rare finding with an incidence of 0.3 to 2.7%. We report a case of an 85-year-old male after having undergone a parietal burr hole craniectomy for left-sided chronic SDH at a hospital in his locality, presented with a drop in conscious level, Glasgow Coma Scale of 9/15 (E3V1M5) with left-sided hemiparesis. A computed tomography scan revealed a thick left subdural collection with multiple densities along with a right capsuloganglionic bleed with intraventricular extension, which was successfully and completely removed, with progressive clinical improvement. Intraoperatively, as the SDH had the consistency and color, like that of mud/clay with thick membranes, it is referred to as “subdural mud.” In our case, surgical intervention did improve the neurological and functional outcome of the patient which supports the fact that surgery is indicated in patients with calcified chronic SDH with acute or progressive neurological deterioration.

Keywords

- ▶ calcification
- ▶ chronic subdural hematoma
- ▶ subdural mud

Introduction

Chronic subdural hematoma (SDH) is widely seen in neurosurgical practice. It is said to be chronic when the clinical manifestations appear 21 days after the trauma and usually appear hypodense to the surrounding brain on a computed tomogram. The annual incidence of chronic SDH is approximately 1 to 5.3 cases per 100,000 population.¹

Calcification or ossification is rare in patients with chronic SDH,^{2–4} but was first reported at autopsy in 1844 by pathologist Carl von Rokitansky. The incidence of calcification typically ranges from 0.3 to 2.7%.^{5–7} Organized chronic SDH can be diagnosed when the hematoma is encapsulated with thick inner and outer membranes with multiple septations and usually has a solid or clay-like consistency.

This presentation of calcified chronic SDH is pathognomonic and is referred to as “armored brain” or “Matrioska head.”⁸ As the chronic SDH in our patient had a consistency and color, like that of mud/clay with thick membranes, we referred it to

as “subdural mud.” This is a case of a large calcified chronic SDH with neurological deterioration who had improved significantly after the evacuation of the subdural collection and excision of the membranes.

Case Report

Presentation

An 85-year-old male patient had already undergone a left parietal burr hole craniectomy after having been diagnosed with left-sided chronic SDH at a hospital in his locality. Prior to the burr hole surgery, patient had history of recurrent falls at home for past 4 to 5 months and 1 day prior to surgery he had multiple episodes of vomiting and headache. As the patient’s condition further deteriorated on postoperative day 2, he was referred to our hospital for further management. At presentation to the emergency unit, the patient had a Glasgow Coma Scale (GCS) of 9/15 (E3V1M5) with pupils bilateral equal and sluggishly reactive and weakness of left

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upper and lower limbs (grade $\frac{3}{5}$ Medical Research Council grading). The results of routine laboratory examinations were unremarkable. A noncontrast computed tomography (CT) scan of the head (**Fig. 1**) at admission, revealed a thick left subdural collection with multiple densities and a thick inner membrane. CT also demonstrated a right capsuloganglionic bleed with intraventricular extension.

Surgery

The patient was planned for emergency evacuation of the subdural collection because of low GCS and midline shift with mass effect after obtaining consent from patient relatives. Left frontotemporoparietal craniotomy was performed with the patient under general anesthesia. The dura was opened in C-shaped fashion and could be easily separated and removed from the outer membrane. The outer membrane was thickened and calcified and approximately 5 to 10 mm thick which was removed (**Fig. 2A**). Yellowish orange mud-like materials were found under the outer membrane, but no fluid was noted (**Fig. 2B**). After the total evacuation of these materials, a thick, hard inner membrane was seen over the cortical surface (**Fig. 2C**). An attempt was made to excise the inner membrane and it was excised off without any cortical surface injury or arachnoid breach. After evacuation the cortical surface was pulsating and with strict hemostasis (**Fig. 2D**). The dura was closed in a water-tight fashion, the bone flap was placed back, and the incision closed in layers with an extradural drain. The entire surgery took approximately 3 to 3.5 hours.

Postoperative Period

It was uneventful and there was no incidence of postoperative seizures or any evidence of rebleed. Adequate control of hypertension and conservative management of right capsuloganglionic bleed with intraventricular bleed was done as the patient showed significant improvement in the postoperative period. At

discharge, wound was healthy and patient had GCS 15/15 (E4V5M6) with left-sided hemiparesis.

Discussion

Organized chronic SDH can be diagnosed when the hematoma is encapsulated with thick inner and outer membranes with multiple septations and usually has a solid or clay-like consistency. It usually presents with headache, hemiparesis, seizure, dementia, or can be seen incidentally. It is a rare presentation and is seen in children and young adults than in the aged, in our case it was an elderly patient. More than half of the patients have a history of minor head injury, while in the remaining patients, the history of trauma to the head is unclear and in children the etiology is usually post-shunt (ventriculoatrial/peritoneal) procedure with over drainage. Our patient had recurrent history of self-falls at home for the last 4 to 5 months.

The exact mechanism by which they occur is difficult to explain but most likely there is decreased circulation beneath the SDH and, as a result, it leads to calcium deposition and connective tissue hyalinization. As a result, over the course of approximately 6 months to years after initial hemorrhage, the calcium deposits accumulation leads to significant calcification.

The ideal surgical procedure for patients with calcified chronic SDH is still a matter of debate,⁶ because of the rarity of the condition and the presence of a thick adherent inner membrane, thus limiting the dissection and reexpansion of the brain after surgery.

Petraglia et al⁹ noted that surgery is infrequently indicated for calcified chronic SDH and beneficial in only a few patients.

However, a systematic review study by Turgut et al¹⁰ showed that surgical removal of symptomatic calcified chronic SDH, did reduce the mass effect and increased cerebral perfusion and, as a result, improved neurological status in the 83 operated patients.

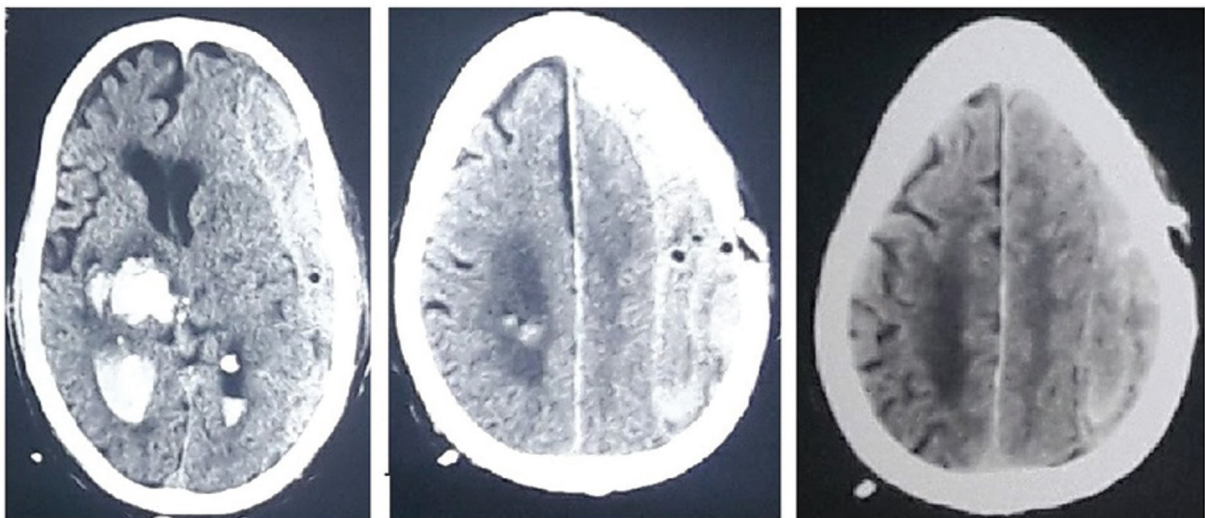


Fig. 1 Noncontrast computed tomography (CT) head showing left side thick subdural collection with burr hole defect and right capsuloganglionic bleed with intraventricular extension.

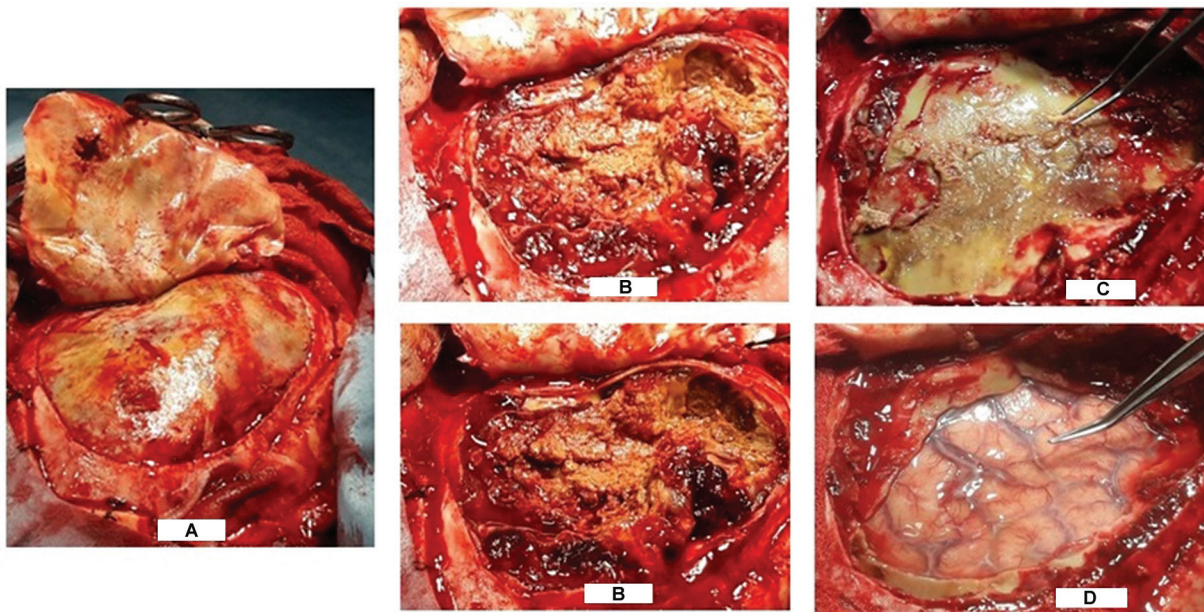


Fig. 2 (A) Dura separated from outer membrane of the organized chronic subdural hematoma (SDH). (B) Organized chronic SDH with yellowish mud/clay-like consistency, referred to as "subdural mud." (C) Thick inner membrane over the cortical surface after evacuation of the chronic SDH. (D) Visible cortical surface after excision of the thick inner membrane.

Surgery should be considered in:

1. Infants, children, and young patients because of the increased hemorrhage risk
2. Patients with acute or progressive neurological deterioration
3. Patients with intracerebral hematoma to avoid further cerebral damage

The different surgical options advocated are:

1. Craniotomy with full/partial membranectomy. Although many neurosurgeons do not advocate the full excision of inner calcified membrane because of its technical difficulties.
2. Shunt revision, in whom the cause of the calcified chronic SDH is overdrainage of ventriculoperitoneal shunt for hydrocephalus in children.
3. Recently, multiple crossing/tenting sutures have been suggested to obliterate the dead space between the residual rigid inner and outer membranes.

Surgery for these cases is problematic, as even though the outer membrane and mud-like organized hematoma are removed, it is difficult to excise the thick, calcified inner membrane⁶ that can injure the cortical surface of the brain causing contusion, hematoma, or onset of fresh neurologic deficits. However, in our case, it was possible to excise the inner membrane without any cortical injury or arachnoid breach. Very rarely it has been reported that those who undergo partial membranectomy, there is a possibility of herniation of brain into the subdural space.

Conclusion

Although the imaging findings in these types of patients are alarming, they are tolerated well. In our case, surgical

intervention did improve the neurological and functional outcome of the patient which supports the fact that surgery is indicated in patients with calcified chronic SDH with acute or progressive neurological deterioration.

Conflict of Interest

None declared.

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