Enigmatic rapid "organization" of subdural hematoma in a patient with epilepsy: A case report

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Enigmatic rapid "organization" of subdural hematoma in a patient with epilepsy: A case report

Lv HT et al. An "organized" acute subdural hematoma

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Abstract

BACKGROUND

Determining a subdural hematoma (SDH) to be chronic by definition takes 3 weeks, whereas organized chronic subdural hematoma (OCSDH) is an unusual condition that is believed to form over a much longer period of time, which generally demands a large craniotomy. Therefore, it is genuinely a tardy process from the initial head trauma, if any, to the formation of an OCSDH. An acute subdural hematoma (ASDH) with incredible organization-like, membranaceous appearances has never been reported.

CASE SUMMARY

A 56-year-old female presented to our hospital with a seizure, and computed tomography (CT) on admission was negative for signs of intracranial hemorrhage. She had clear consciousness and unimpaired motor functions on arrival and remained stable for the following week, during which she underwent the necessary examinations. On the morning of the 10th day of hospitalization, the patient accidentally hit her head hard against the wall in the bathroom and promptly deteriorated into complete coma within 2 hours. Therefore, we performed an emergency CT scan and identified a left supratentorial subdural hematoma that was absolutely indicated for surgery. We performed the procedure as soon as possible. However, the intraoperative findings were astonishing: nothing liquefied was observed. Instead, a solid hematoma covered with a thick membrane was noted that strongly resembled an organized hematoma. Evacuation was successful, but the family stopped treatment the next day due to financial problems, and the patient soon died.

CONCLUSION

Neurosurgeons should address SDHs, especially ASDHs, with considerable discretion and individualization due to their highly diversified features.

Key Words: Organized chronic subdural hematoma; Acute subdural hematoma; Subacute subdural hematoma; Craniotomy; Encapsulation; Case report

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Core Tip: Through unknown mechanisms, a minority of chronic subdural hematomas tend to be organized in the end with the prerequisite of being chronic. Therefore, it will take a long time to form encapsulating membranes, as was traditionally thought. We hereby report this rare case of enigmatic rapid encapsulation of an acute subdural hematoma, which, according to the definition, cannot be considered organized. Such a rapid formation of thick membranes around an acute subdural hematoma is rare and will certainly make the procedure, if needed, unpredictable. We may also need to review the natural history of subdural hematomas.

INTRODUCTION

Subdural hematomas (SDHs) following traumatic head injury constitute an essential proportion of the cases seen in daily practice in the field of neurosurgery. An SDH is considered chronic (CSDH) when it is discovered more than 3 weeks after the initiating trauma incident^[1]. Organized chronic subdural hematoma (OCSDH) is a rare category of CSDH characterized by the formation of areas of solid consistency encapsulated by thick membranes^[2,3], which has been extensively reported regarding the diagnosis, treatment options and outcomes. Only a minority of CSDHs develop into OCSDHs, and OCSDHs have various clinical manifestations. Although the mechanisms underlying the development of OCSDHs in these cases remain unclear, the process of hematoma organization is undoubtedly slow (probably lasting at least 6 months)^[2,4,5]. It is conceivable that OCSDH develops only when the hematoma is chronic. By reviewing the literature, we found no previous reports of the rapid formation of membranes around a hematoma, causing it to appear organized, which is an interesting

presentation. In this article, we report a very rare case of "organized" acute subdural hematoma (ASDH) with an unusual appearance found in a deceptively simple craniotomy performed to evacuate a hematoma, and we believe this is the first report of this phenomenon.

2 CASE PRESENTATION

Chief complaints

A 56-year-old female farmer had a seizure 13 hours prior to presentation along with a slight fever.

History of present illness

The patient developed a cold approximately 5 days prior to presentation after having been exposed to rain. After a careful investigation of her history, we learned that the patient had two separate tonic-clonic seizures 2 and 3 hours respectively after the primary seizure characterized by oral and linguistic automatisms in sleeping.

History of past illness

Her major medical history included hypertension and diabetes for more than 10 years, both of which were marginally controlled by medications; a hysteromyomectomy 20 years prior; cerebral infarction 12 years prior; and renal dysfunction incidentally detected 3 years prior of unknown current extent.

Personal and family history

Three of her family members, including the patient's mother and two brothers, died of "bronchitis". Of note, the brothers passed away at very young ages.

Physical examination

The patient exhibited clear consciousness and normal motor function (GCS 15) with occasional delirious speech and a positive Babinski sign on the right side. Both pupils

were 3 mm in diameter and sensitive to light. No other specific physical signs were found.

Laboratory examinations

Laboratory assessments showed a high level of C-reactive protein (CRP) (9.47 mg/L, reference range 0-8.0 mg/L); compromised renal function, with high serum creatinine (164 μmol/L, reference range 46-92 μmol/L) and high blood urea (13.53 mmol/L, reference range 2.90-8.20 mmol/L); and increased serum myoglobin (268.35 ng/ml, reference range <110.00 ng/ml). The test results for CSF obtained through a lumbar puncture were uneventful.

Imaging examinations

Computed tomography (CT) on admission showed multiple malacia sites, mainly in the right lobe, without any signs of hemorrhage (Figure 1). Magnetic resonance imaging (MRI) on the 3rd day after admission showed no signs of intracranial hemorrhage (Figure 2). On the morning of the 10th day after admission, a sudden deterioration of the patient prompted us to perform an emergency CT scan, which determined a left supratentorial subdural hematoma with considerable mass effect (Figure 3).

FINAL DIAGNOSIS

The main diagnoses were symptomatic epilepsy and ASDH.

TREATMENT

The process of treatment was rather dramatic. Immediate antiepileptic treatment as well as treatment for the comorbidities were administered on admission, and the patient seemed to remain stable over the course of the following week while necessary examinations were conducted. Just when we thought everything was going well, surprisingly, the patient experienced projectile vomiting and a severe headache on the morning on the 10th day. However, only 20 minutes later, she began to show arresting

weakness on her right side but she could still response to calling and her pupils were good. We ran an emergency CT scan (Figure 3). By the time the patient was returned to her room, she was in complete coma (GCS 5, E1V1M3), and her left pupil was dilated to 4 mm and fixed. After quickly debriefing her family members on the life-threatening situation, we operated as soon as possible with a large craniotomy. The intraoperative findings were astonishing. The hematoma strongly resembled an organized hematoma and assumed a form we not previously observed. We inclined to think the hematoma was acute according to the clinical course and CT scan despite some low-density components inside it. Therefore, the observation of the absence of a liquefied hematoma with only a modicum of clear CSF when we started to open the dura mater was an anomaly. The hematoma was better exposed after we cut the dura completely open in a cruciform fashion, and we found it under a solid covering of a thick membrane (Figure 4). This finding made us even more confused because we were confident that it was definitely not a CSDH based on strong evidence of recent CT and MRI scans. The flap range was believed to be sufficient for a typical ASDH but not necessarily for an "organized" hematoma, so a tentative tumorectomy-like technique was used to remove the hematoma in pieces. Fortunately, the attempt proved successful, which enabled us to evacuate the hematoma thoroughly without any unwanted damage. However, interestingly, the subarachnoid space macroscopically appeared dry, and a thinnish inner membrane of the hematoma was detected that did not adhere significantly over the brain surface (Figure 4). We washed the cavity with plenty of saline until it cleared, during which minor frontal lobe superficial arterial bleeding was easily controlled by bipolar electrocoagulation hemostasis. The procedure was generally successful. The brain tissue remained collapsed without emerging pulsations before closure, and the patient was then transferred to the ICU for further observation.

In fact, we performed an electroencephalogram on the second day after admission, but reexamination was required for conclusive findings. However, we were unable to perform the reexamination due to the unexpected craniotomy. On the 3rd day, an MRI revealed nothing to alert us to the hemorrhage. Another day later (the 4th day of

hospitalization), we tested her CSF by a regular lumbar puncture. The pressure was approximately 190 mm H₂O, and then approximately 7 ml of clear CSF was obtained and sent for evaluation. On the dawn of the 9th day, the patient suffered a gelastic seizure without warning that was later disassembled by injection of *diazepam* and *dilantin*, representing the only recurrence since admission. Then on the 10th day, it happened as above. However, the husband revealed that the patient hit her head against the wall in a bathroom rather heavily several minutes prior to deterioration. Carelessness was to blame for her injury, according to her husband, who was confident that it was not another seizure.

OUTCOME AND FOLLOW-UP

Upon arrival at the ICU, the patient relied on a ventilator with GCS 5 (E1V1M3), and the left pupil was still dilated (d=4.5 mm) and fixed. The family members decided to give up further treatment right on the second day after surgery due to financial problems. So, the patient was released from the hospital, and follow-up was not performed, as it would have been unavailable and pointless. She died shortly thereafter because she had still been on a ventilator when the decision to terminate treatment was made.

DISCUSSION

First, the diagnosis is of particular interest given that the most recent scan with the exception of that performed on the day of surgical intervention was an MRI performed exactly 7 days prior. Even if the head injury did not immediately cause hemorrhage, it was a subacute subdural hematoma (SSDH) at most with no chance of being a CSDH. Despite the husband's statement, did the patient have another seizure when she hit the wall? We will never know. Currently, we believe that the essence of CSDH is a fibrous capsule enclosing bloody fluids that requires 3-4 weeks to complete encapsulation^[2,6]. No previous study has reported such a rapid development of an organized SDH, as noted in our case, and we regret that a pathological examination of the specimen, which

could have revealed the structural features of the membranes, was not accomplished in the end. Even if it was subacute, which it was likely not, we cannot explain the almost complete absence of a liquefied hematoma, which conflicts with both our experience and historical reports. Cai et al.^[7] and Bosma et al.^[8] reported surgical treatment for several SSDH cases with a transcranial neuroendoscopic approach and traditional burr holes, respectively. These studies reported major liquefaction of the hematomas (subacute), as the liquid was easily drained by regular suction. Making the boldest assumption that CT findings on the operative day were due to fresh bleeding secondary to an SSDH, fresh clotted blood derived from recent bleeding within a CSDH or an SSDH usually undergoes rapid liquefaction^[2]. However, the hematoma was as solid as organized but with marked differences from fresh clotted blood. Thus, we were put in a predicament because we had to evacuate the hematoma like resecting a convexity meningioma. Youn et al. reported a tumor-like presentation of an organized SDH, but the hematoma was chronic^[9]. Moreover, the capsular membranes of SSDHs are usually yellowish, whereas those in our case were light in color.

During the initial treatment, oral *aspirin* was given to the patient who was not a routine user of the drug in the past for ischemic considerations in the nine consecutive days before this unexpected operation. We are not sure whether this treatment played a role in the hemorrhagic event. However, some authors suggest that an elevated risk of SDH (SSDH/CSDH) is related to the use of antiplatelet and/or anticoagulation agents^[10,11].

CSDH organization involves the formation of a solid hematoma to replace the primary liquefied bloody contents when the inner and outer membranes completely fuse due to a slowly increased volume of fibrous material by unknown mechanisms^[12]. Killeffer et al. noted that the liquid characteristic of CSDHs is linked to prevalent local hyperfibrinolytic activities within them^[13], so fibrinolytic abnormalities may be related to the strange organized appearance of the hematoma in our case. However, solid evidence is lacking.

This hematoma did not have characteristics consistent with those of ASDH, CSDH or even SSDH with acute rebleeding. We cautiously present a vagarious theory of any possibility it may be a "subarachnoid" hematoma that the outer membrane of the hematoma we saw was actually the arachnoid which was thickened for some reason and what's left on the brain surface was just pia mater (it looked very dry, unlike arachnoid covering rich CSF). Regardless of the true nature of this hematoma, a large craniotomy was the only correct choice for the patient.

CONCLUSION

The proportion of cases of CSDHs and SSDHs that are classified as organized SDHs is low, whereas no cases of ASDHs with organizational characteristics have been reported. There is still no feasible explanation of the complete formation of encapsulating membranes around this hematoma in such a short time, and even an educated guess cannot be made. The general principles of neurosurgical practice shall not be compromised even under peculiar circumstances. More discreetness and more individualized treatment should be administered by neurosurgeons when dealing with SDHs, especially acute or subacute SDHs, as they can be very diverse with regard to their features. On the other hand, we should reconsider and further explore the natural history of SDHs.

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