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ORIGINAL ARTICLE

Surgically treated symptomatic calcified chronic subdural hematoma

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Abstract

Calcified chronic subdural hematoma (SDH) or armoured brain is especially rare with only slight pathophysiology understanding. It happened after head trauma, subdural effusion, meningitis, or as a sequel of VP Shunt. But there is no definite mechanism of neither the pathogenesis nor the management. Because these patients have a thick calcified inner membrane, the optimal surgical procedure for armoured brain's patient has not been established. Moreover, it is also difficult to obtain good re-expansion of the brain after surgery. A calcified chronic SDH is less likely happened in adults or elder rather than children or young adults. Management of calcified chronic SDH is mostly individualized. With seizures, careful monitoring of anticonvulsant therapy is required. The indications of surgical procedure can be features of raised intracranial pressure, headache, or neurological deterioration. The surgical strategies depend on calcification's thickness and extension.

This study was a preliminary study of Achmad Adam's research project. This was a descriptive observational research with a case report design.

We report a rare case of calcified chronic SDH in the elderly, presenting with progressive neurological deficit and acute raised intracranial pressure, which was managed surgically to facilitate cerebral re-expansion and inhibit probable brain atrophy. Therefore, the authors intended to share a case report regarding a rare chronic SDH and its management, so hopefully, it could be acknowledged by other surgeons as something to learn from.

Keywords: *Armoured brain, calcified chronic subdural hematoma, cerebral re-expansion, seizures*

Introduction

Calcified chronic SDH or armoured brain is especially rare with only slight pathophysiology understanding.[1] It represented 0.3-2.7% of chronic SDHs,[2] since it was first explained in 1884.[3-5] It happened after head trauma, subdural effusion, meningitis, or as a sequel of VP Shunt.[1] Several authors also have reported calcification in traumatic SDH.[6-8] The development of calcified chronic SDH to form subdural hematoma varies from 6 months to years,[9,10] but no definite mechanism of neither the pathogenesis nor the management.[3] Because these patients have a thick calcified inner membrane, the optimal surgical procedure for armoured brain's patient has

not been established. Moreover, it is also difficult to obtain good re-expansion of the brain after surgery.[11] We describe an especially rare symptomatic armoured braincase with a history of preceding trauma several years before.

Case report

A 78-year-old man was brought to The Emergency Unit of Hasan Sadikin Hospital with one episode of generalized seizure on 12 hours before admission, for about 15-20 seconds. The complaint was preceded by progressive weakness of his right extremities since five days before. History of previous head trauma was admitted when the patient was 60 years old, with an episode of decreased consciousness, but he was not brought to seek any medical treatment until the consciousness was resolved. Since then, he was suffering intermittent headaches that could still be relieved by medication.

The patient came into our emergency room with a Glasgow Coma Score (GCS) 15, right hemiparesis, and grade 2 of motor strength. The computed tomography (CT) scans resulted in a large SDH of the left hemisphere, with calcified inner membrane (Figure 1). The patient's surgery was done within the next two days due to high blood pressure while the antiseizure medication was given throughout this time.

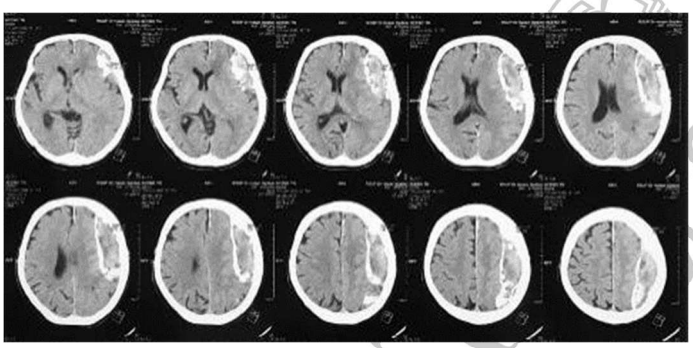

Figure 1. *The chronic calcified SDH was seen in the left frontoparietal convexity. Exposed "armour dura" with the mould of the underlying hematoma was shown during left frontoparietal craniotomy (Figure 2)*

The C-Shape manner was used to open the dura, exposing the capsule of the calcified chronic SDH, looking all white-yellowish, right next to the inner surface of the dura. The capsule seemed like a crab shell, so we decided to open it using Metzenbaum scissor and monopolar cauter. It was exposing gray mud-like contents, suggesting various stages of a subacute hematoma. After we remove the contents, the thick and very hard calcified inner membrane was exposed over the surface of parenchyma. Fortunately, the arachnoid membrane was intact and not adhered to the hematoma, therefore the hematoma was completely removed. We did a smooth dissection from the exposed temporal part of the cortex using a cottonoid pad. Some parts of the capsule tightly adhere to the large superficial cortical vein. Gross total removal of the mass was achieved with minimal damage to the underlying brain parenchyma and saline-filled the abandoned hematoma cavity. Dura mater was primarily sutured and the bone flap was finally done.

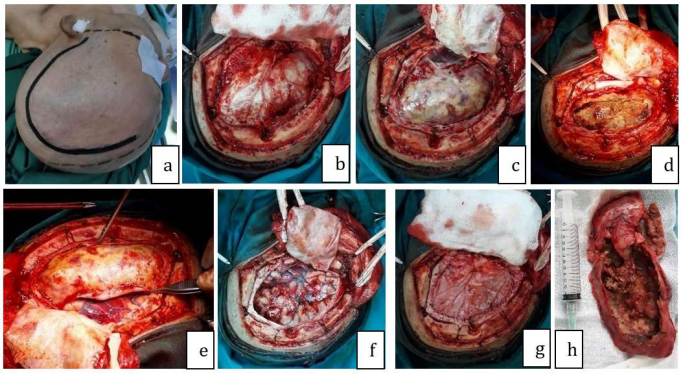

Figure 2. *Intraoperative images. (a) left frontoparietal incision, (b) craniotomy was performed, exposing hard and tense dura, (c) the dura was opened C-shape, exposing underlying calcified chronic SDH capsule, (d) the capsule was opened, showing gray mud-like contents inside the capsule, (e) & (f) complete removal was allowed because the inner capsule was not adherent to the parenchymal surface, (g) dura mater was primarily sutured, (h) gross appearance of calcified chronic SDH after total removal*

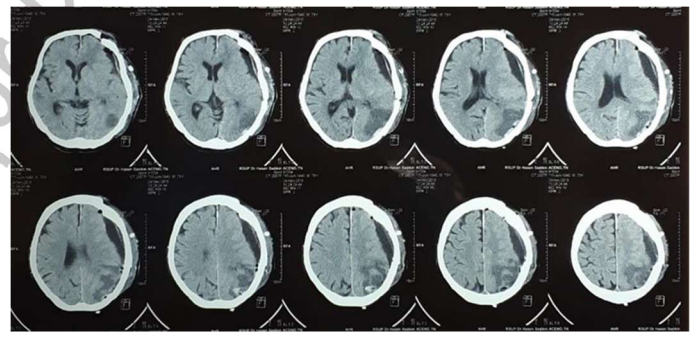

Figure 3. *Post-operative CT-Scan showed no residual hematoma and significant relief of compression, with a subdural fluid collection.*

Post-operatively, it was uneventful and the patient maintains his consciousness. The motor strength of his right extremities was increased from 2 to 4 after three days post-operatively. During post-operative care, the patient was given 100 mg intravenous phenytoin, and no seizure was observed. On the 7th day postoperative, the patient underwent control head CT-Scan before discharge. CT-Scan showed no residual hematoma with significant relief of compression (Figure 3). However, there was a subdural fluid collection with a small infarct at

the left occipital area. The patient came to our outpatient clinic about 1-month postoperatively. The headache and motoric strength were improved (motoric strength scored as 4). The histopathological result showed hyalinized tissue with some dystrophic calcification and congested blood vessels, suggesting calcified hematoma (Figure 4).

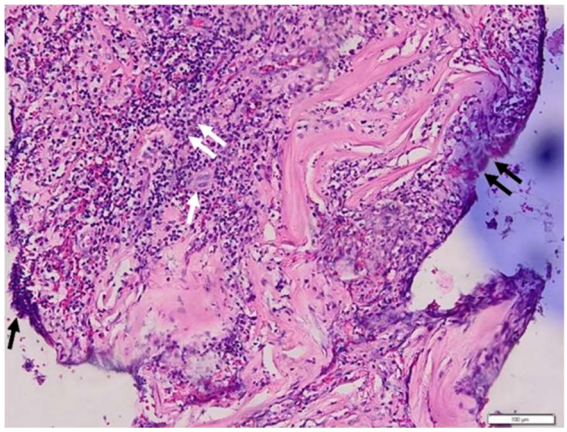

Figure 4. *Histopathological examination of the inner membrane showed densely hyalinized tissue with some dystrophic calcification (black arrow) and congested blood vessels (white arrow). Focal interstitial fresh hemorrhage (double black arrow) and small neovascular proliferation with scattered chronic inflammatory cells are also noted (double white arrow)*

Discussion

Calcified chronic SDH or as one may have called "Armoured Brain" or Matrioska head is a huge calcified chronic SDH covering the brain. Matrioska head itself derives from a well-known Russian doll.[12]

Calcified chronic SDH or armoured brain is especially rare with only slight pathophysiology understanding.[1] It represented 0.3-2.7% of chronic SDHs,[2] since it was first explained in 1884.[3-5] Calcified chronic SDH is less likely to happen in adults or elder rather than children or young adults.[13] Although calcification is said to be found in some cases of chronic SDHs, a hematoma as large as ours is unusually found. Chronic SDH happened after to head trauma, subdural effusion, meningitis, or as a sequel of VP Shunt.[1] Disorders such as coagulopathy (sepsis, liver insufficiency, therapeutic anticoagulant use), intracranial hypotension (occurs after over-drainage causing bridge veins traction in shunted patients), chronic alcoholism, malformations of vascular, and tumors (both primer and metastatic) may have a role in etiology.[3] It is also usually one of the complications of minor head trauma.[3-5] Several authors have reported calcification in traumatic SDH,[6-8] which is relatively commoner than other causes of SDH, as we found in our patient.

The pathogenesis of calcification remains unclear. Afra stated that things that lead to calcification might be poor circulation and absorption in the subdural space and vascular thromboses.[6] Dense collagen deposits occur on the membrane in chronic SDH, further forming a fibrotic capsule. At a very late stage, due to progressive mineralization, this fibrotic capsule calcifies.[14,15] The calcifications have been reported mostly along the inner surface of the dura mater. However, calcification of both outer and inner layers in bilateral chronic SDH has also been reported.[3] Regarding the interval of calcified hematoma development, Nakamura studied the cycle of hematoma and concluded that the etiology was delayed absorption process of the hematoma.[16] The interval between the original SDH and the growing calcified chronic SDH varies from 6-months to many years.[9,10] Moreover, Boyd *et al*., also stated that the hematoma must exist at least 3-years before it becomes calcified.[7] Ossification develops further a few years after calcification and thought that it was the result of this process.[17] There are also bilateral chronic SDH cases developing into unilateral calcification. This suggests that there are roles of local factors in the development of calcification.[8] As in our patient, SDH collection may exist after head trauma 28 years prior to admission. The hematoma may be trapped in subdural space and calcification occurred. A long time interval could also lead to the ossification of the capsule.

Histopathology result of calcified chronic SDH is structurally distinct from the chronic SDH, and the inner membrane is as thick as, or thicker than, the outer membrane and has densely hyalinized tissue with some dystrophic calcification and congested blood vessels,[5] as we found in our case.

Clinical presentation of calcified SDH can be asymptomatic and varies widely[9,14,18,19] to acute raised intracranial pressure in others.[18,20] The patient may be asymptomatic and the symptoms present after a history of trauma or seizure which can speed up the problem.[18] This patient had a chronic intermittent headache since the episode of trauma, which was probably due to the existence of SDH that caused raised intracranial pressure. Once the inner membrane adhered to the cortex was calcified and ossified, it might irritate the cortex and causing seizures. Seizures might further cause edema on the underlying brain and led to compression of the motor cortex.

Management of calcified chronic SDH is mostly individualized. Careful monitoring of anticonvulsant therapy is even required in patients with seizures.[10] Successful surgical removal has been reported occasionally.[6,9,18,21] The indication of the surgery consists of raised intracranial pressure's features, headache, or neurological

deterioration.[3,14,18] Some suggest that asymptomatic, old patients, may just be observed.[3]

The surgical techniques regarding calcified chronic SDH were variedly reported. Authors performed twist drill aspiration, burr hole aspiration or microsurgical dissection.[12] Microsurgical dissection of calcified hematoma from brain surface can badly result in brain contusion, bleeding, and appearance of new neurological deficit.[20,22,23] The surgical strategies depend on the thickness and extent of calcification.[12] It was stated by Kaplan *et al*., that if the inner layer is thicker and compressing the brain progressively, fluid drainage by itself may not be sufficient to improve the symptoms and help the re-expansion of the brain.[3] In this case, it was decided to perform a craniotomy to remove the hematoma and its membrane, as the clinical manifestation showed progressive neurological deficit and the head CT-scan showed thick calcification and compression of the brain.

McLaurin and McLaurin[8] reported six pediatric patients who had calcified SDHa surgically removed. A long-term postoperative observation showed insignificant symptoms improvement.[8] The main reason for those failures was thought to be due to underlying brain damage from the initial injury, also form brain atrophy which already occurred.[9] In our case, the progressive neurological deficits were improved postoperatively, but still, there was an insignificant benefit in the patient who had chronic stable neurological deficits. The sign of brain compression was shown on CT-scan even if the hematoma was densely calcified. Regarding the patients with symptoms or deficits,[8] if there's already neurological deficits due to brain atrophy, it was unlikely to obtain improvement, even if a surgical procedure is taken.[9] In our case, the decompression itself might prevent brain atrophy, thus, beneficial for this patient. We suggest surgical removal for the calcification over the cortex despite the absence of symptoms, particularly when prominent cerebral compression is seen, to allow cerebral re-expansion and inhibit probable brain atrophy development.

Conclusion

The calcified chronic SDH is rare entities and the pathogenesis and treatment are still unclear. We report a case of calcified chronic SDH in the elderly, presenting with progressive neurological deficit and acute raised intracranial pressure, which surgical procedure was done to allow cerebral re-expansion and inhibit probable brain atrophy development.

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