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Case Report

Chronic calcified subdural hematoma: Case report and review of the literature

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Abstract

Background: Calcified chronic subdural hematoma is a rare but known entity, estimated to represent 0.3-2.7% of chronic subdural hematomas. Although surgical treatment is unanimous for chronic subdural hematomas, therein lies some doubt on it being applied to calcified chronic subdural hematomas.

Case Description: We report a case of a 73-year-old male, presenting with deterioration of motor function in his right limbs since 18 months, with computed tomography (CT) scans and magnetic resonance imaging (MRI) documenting a large subdural collection of the left hemisphere, with calcified inner membrane, which was successfully and completely removed, with progressive clinical and radiological improvement.

Conclusions: We report a case where this type of rare lesion was removed with a progressive and complete resolution of the patient's symptoms, restoring his previous neurological condition. From the cases described in the literature and our own experience with this case, we think surgical treatment in these patients, when symptomatic, is necessary and viable, frequently resulting in the patient's improvement.

Key Words: Calcified chronic subdural hematoma, outcome, surgical treatment

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INTRODUCTION

Calcified chronic subdural hematoma is a rare but known entity, estimated to represent 0.3-2.7% of chronic subdural hematomas, [5,7] since it was first described in 1884. [1,4,6]

There are about 100 cases published. [2,7] Although surgical treatment is unanimous for chronic subdural hematomas, therein lies some doubt on it being applied to calcified chronic subdural hematomas. [1,5,7] The optimal surgical procedure for this type of lesion, classically referred to as

"armored brain", [3] has not been established due to the limited reexpansion of the brain after surgery. [2] This is probably related with the presence of a thick calcified inner membrane, which is frequently adherent to the cortical surface of the parenchyma, limiting the dissection from the brain, which may cause brain contusion, bleeding, or the appearance of new neurologic deficits. [3]

Several authors report that there is no improvement of long-standing symptoms with surgery, thus recommending surgery only when acute or progressive neurological symptoms occur.^[3,6]

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CASE REPORT

We report a case of 73-year-old male, presenting with deterioration of motor function in his right limbs since 18 months, transferred from a foreign institution without access to neurosurgical care or evaluation. The patient was admitted to our emergency room, in Glasgow coma score (GCS) 14, right hemiparesis with motor strength grade 3. The computed tomography (CT) scans and magnetic resonance imaging (MRI) documented a large subdural collection of the left hemisphere, with calcified inner membrane [Figure 1]. The patient's surgery was delayed due to a pulmonary infection, and throughout this time, his clinical condition progressively deteriorated. When he finally had surgery on his subdural hematoma, the patient was in GCS 9, with no motor function to the right limbs. Left frontoparietal craniotomy was performed [Figure 2], exposing an "armored dura", with the mould of the underlying hematoma. The dura was opened in an arcuate manner, exposing the calcified capsule of the chronic subdural hematoma, which was tightly adherent to the inner surface of the dura. After dissecting it, the capsule was incised, exposing various stages of subacute hematoma. After total removal of these partially calcified gray mud-like materials, the thick partially calcified inner membrane was exposed overlying the parenchymal surface. However, the arachnoid membrane was intact and not adhered to the hematoma, allowing for its complete removal without injuring the underlying brain. The postoperative period was uneventful, and at his 2 month follow-up, the patient was in GCS 15, maintaining some gate disturbances, with right hemiparesis more noticeable in his lower limb (grade 3 of 5). At his 3 month follow-up, he was able to deambulate with support of a clutch, only with slight monoparesis of the right lower limb (grade 4 of 5). His postoperative CT scans documented progressive reexpansion of the brain and resolution of subdural collection [Figure 3].

DISCUSSION

The calcified chronic subdural hematoma is manifested mainly by seizure, dementia, mental retardation, growth retardation or headache, hemiparesis but sometimes incidentally found without any symptom. [4,7] It is a rare entity and occurs more frequently in children and young adults than in the aged, [7] with calcification and ossification occurring in 0.8-10% of chronic subdural hematoma patients. [1,5]

The course of the development of calcification in a calcified chronic subdural hematomas is unclear. However, the hematoma may progress gradually from hyalinization to calcification, and finally ossification through irritation of the tissue. After hemorrhage calcification usually takes 6 months to many years to develop. The proposed mechanism of calcification is that poor circulation and absorption in the subdural space together with intravascular thrombosis, T.4.7 prolonged existence of the hematoma in the subdural space, stagnant blood due to

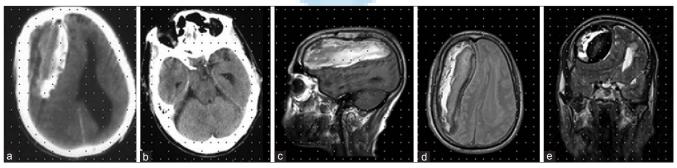


Figure I: Chronic calcified subdural hematoma of the frontoparietal right convexity, with calcified inner membrane, determining a significant midline shift, with uncal herniation. (a and b) axial CT scan, (c) Sagital TI MRI, (d) axial DP MRI, (e) Coronal T2 MRI

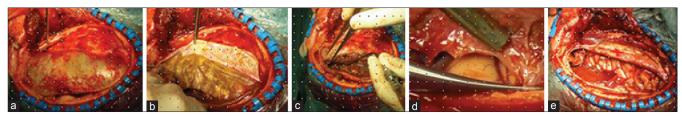


Figure 2: Intraoperative images. (a) Right fronto-parietal craniotomy, exposing the dura with the mould of the underlying calcified chronic subdural hematoma's capsule, (b) dissection of the inner surface of the dura from the underlying calcified capsule, (c) after opening the capsule, aspiration of the contents of the liquid content, with various stages of subacute organized hematoma, (d) the inner membrane, which was not adherent to parenchymal surface, allowing for its complete removal without injuring the underlying brain, (e) the lack of brain reexpansion after complete removal of the hematoma

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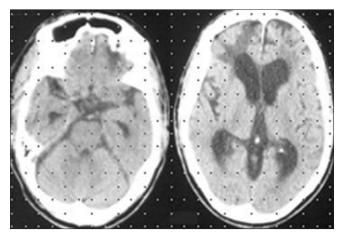


Figure 3: Postoperative CT scan at 90 days, showing reexpansion, without any residual subdural collection

insufficient arterial supply and inadequate venous return, thick connective tissue membrane, and other local factors are considered to contribute to the development of calcification of the chronic subdural hematoma.^[7]

Additionally, abnormal inherent metabolic tendency to calcification can play a role in calcification. However, the mechanism of calcification is still unclear and the periods of calcification are quite different.^[1,4,7]

Although there are many different views of the treatment for the calcified chronic subdural hematoma, observation is recommended for asymptomatic ones without acute or progressive neurological disorders in the elderly. Nonetheless, surgical procedure should be considered for the infants or young patients, or the patients having acute or progressive neurological disorders (due to the increased hemorrhage risk as evidenced by the vascular proliferation in the capsule of calcified chronic subdural hematoma) [7] or with intracerebral hematoma in order to prevent additional brain damage. [4,5]

Removal of the calcified chronic subdural hematoma reduces the mass effect and cerebral irritation, and increases the cerebral blood flow, thus patients can improve neurologically after surgery.^[7]

Confirming the reports from some previous publications, [1,7] the postoperative neurology recovery in our patient, attests to the efficacy of a surgical treatment of patients with symptomatic calcified chronic subdural hematoma, especially for those with clinical deterioration.

From the analysis of the available reports in the literature, we think that it is also a plausible conclusion to say that if the inner layer is thicker and compressing the brain seriously, fluid drainage by itself may be insufficient to improve the symptoms and helping the reexpansion of

the brain. Therefore, in such cases, the inner layer should be carefully dissected if feasible.^[1]

One of the most frequent complications that may be observed after chronic subdural hematoma operations is recurrent hemorrhage. It is thought that insufficient brain expansion following hematoma drainage, developing following prolonged compression in recurrent hemorrhage is the basic factor. However, since ossified subdural hemorrhages are rather rare, there is insufficient information regarding the recurrence rate in the literature. In chronic subdural hemorrhage cases, recurrent hemorrhage, and residual subdural fluid collection should be differentiated from each other. Disappearance of residual fluid may sometimes last for weeks or even months. Therefore, unless there is presence of clinical deterioration, no intervention should be carried out regarding the residues in the control CT.^[1]

In our patient, such issue was raised frequently, due to the deficient re-expansion in the first 2 weeks in the postoperative period, with collection of isodence fluid in the subdural space, which progressively diminished in size, as the brain reexpanded to its normal position.

CONCLUSIONS

Chronic calcified subdural hematomas are rare entities, which are well tolerated due to their indolent nature even though the radiologic findings might be quite impressive and without direct clinical correlation.

We report a case where this type of lesion was removed with a progressive and complete resolution of the patients symptoms, restoring his previous neurological condition, deeming him independent.

From the cases described in the literature and our own experience with this case, we think surgical treatment in these patients, when symptomatic, is necessary and viable, frequently resulting in the patient's improvement.

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