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Armoured brain. An unusual presentation of chronic subdural haematoma

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ABSTRACT

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 debate on surgical evacuation of calcified chronic subdural hematomas. We report a case of an 18-year-old male presenting after 17 years of VP shunt with calcified chronic SDH. He presented with only mental retardation and no other neurological deficit and as such he was managed conservatively and discharged with close follow-up. We believe that surgical treatment should only be used in circumstances when it is symptomatic, essential, and viable, and will eventually lead to the patient's neurological improvement based on the cases mentioned in the literature. Our personal experience with this case is that the non-surgical procedure sometimes is able to provide good outcomes, especially when the extracranial problems are managed carefully and the patient doesn't complain of any neurological deficit.

Keywords
brain injury, calcification, chronic subdural haematoma

INTRODUCTION

Chronic calcified subdural hematoma was originally described in 1884, though uncommon it is a well-known condition that accounts to 0.3-2.7% of chronic subdural hematomas.[1,2,3,4] The mechanism of calcified chronic subdural hematoma, sometimes known as "armoured" brain, is poorly understood. The ideal surgical technique for patients with armored brains has not been determined because these patients have a thick calcified inner membrane.[5] Additionally, it is challenging to get a successful re-expansion of the brain following surgery.[6] There have been about 120 cases of chronic calcified subdural hematoma published, and most cases of calcified chronic SDH are managed on an individual basis.[4] The need for surgery may be indicated by signs of increased intracranial pressure, headache, or neurological decline.[1,2] The surgical approaches depend on the extent and thickness of the calcification. Here, we present a case study of an 18-year-old male with an armored brain who was conservatively handled.

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Case report

The patient is an 18-year-old male, who has a history of Dandy Walker Malformation and underwent right sided parietal MPVP shunt at the age of 1 year. Following shunt surgery, he was lost to follow up, and he presented again in 2023 for follow-up with mental retardation. NCCT brain was done and it was suggestive of right sided calcified chronic SDH. (Figure 1). Even though there was significant midline shift, it did not cause any neurological symptoms. As such he was managed conservatively. He has continued to do well after 8 months of post discharge.

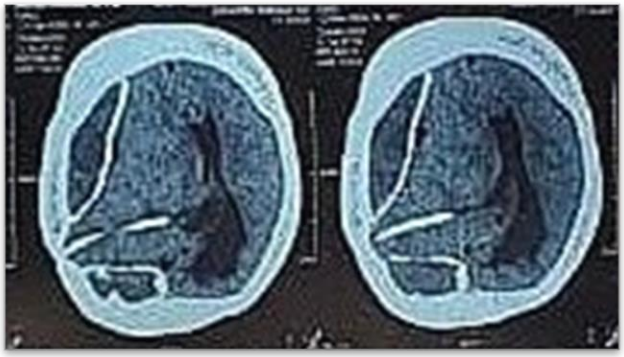

Figure 1. NCCT Brain showing the calcified membranes with shunt with left deviated lateral ventricles with midline shift.

Discussion

Chronic subdural hematoma is one of the common pathologies encountered in neurosurgical practice, but its calcification or ossification has been rarely published in the literature. Although the exact incidence of calcified or ossified CSDHs is unknown, it has been reported to range from 0.3 to 2.7%.[1,2] The calcified or ossified CSDHs due to various etiologies occur more frequently in children and young adults. They generally present with seizures, headache, memory deficit, difficulty in walking, or deficit in the level of consciousness.[7] In our case, he presented with only mental retardation and no other complain.

Calcified chronic SDH has been observed as late complication of head injury or late sequel of post meningitis subdural effusion. It has also been reported as delayed complication of VP shunt. Systematic review studied by Turgut et al.showed that there were 78 men and 29 women from 25 countries, ages ranging from 4 months to 86 years (mean 33.7 years), with etiologies of head trauma in 33.3%, shunting for hydrocephalus in 27.2%, or following cranial surgery in 4.4%. The duration of symptoms ranged from acute onset to 20 years, with a mean of 24.1 months.[8]

The pathogenesis of calcification in CSDH is not very well understood. Some popular postulates regarding its formation are [9, 10, 11]

1) Dense collagen depositions occur on the membranes forming fibrotic capsule, eventually calcifies from progressive mineralization
2) Inadequate circulation and lack of absorption in the subdural cavity
3) Thrombosis in the bloodstream, clotting blood in the subdural cavity
4) Poor circulation and poor venous return
5) Thick layers of connective tissue prone to easy calcification.

Even though surgical treatment for chronic SDHs is universal, there is some question over whether it can be used for calcified chronic SDHs. Due to the restricted reexpansion of the brain following surgery, the ideal surgical treatment of "armored brain," has not been determined. This is most likely connected to the existence of a thick, calcified inner membrane that is frequently adhered to the cortical surface. Surgical removal of the calcification is difficult, and it could damage the underlying cortex. [12, 13] For calcified chronic SDH that is increasing in size, surgical intervention is preferred. Various surgical methods for calcified chronic SDH have been documented. Twist drill aspiration, burr hole aspiration, or microsurgical dissection are techniques frequently used. [12, 13] Brain contusion, bleeding, and the development of fresh neurological deficits can occur as a result of micro surgically removing a calcified hematoma from the surface of the brain. On the other hand, other authors argue that by reducing the detrimental effects of compression and inflammation as well as normalizing cerebral blood flow, evacuation of the hematoma will enhance neurological status.5 According to our experience; patient management is decided on an individual basis. Patients who have no symptoms, are elderly, or don't have any neurological deficits can be handled conservatively with constant monitoring.

Conclusions

Despite the rarity and indolent character of chronic calcified subdural hematomas, which can have remarkable radiologic features but no clear clinical

correlate, these rare entities are well tolerated. We describe a case of known DWM who underwent VP shunt and then developed chronic calcified membranous SDH 18 years later. This patient was managed conservatively because surgery is useful in only a small number of patients with a steadily worsening neurological status. We believe that surgical treatment should only be used in circumstances when it is symptomatic, essential, and viable, and will eventually lead to the patient's neurological improvement based on the cases mentioned in the literature. Our personal experience with this case is that the non-surgical procedure sometimes is able to provide good outcome, especially when the extra cranial problems are managed carefully and the patient doesn't complain of any neurological deficit.

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