Surgical or Conservative Treatment For Calcified Chronic Subdural Hematomas: 
Report of Two Cases

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Summary

Calcification occurs in the membranes of chronic subdural hematoma in the range of 0.3% to 
2.7%. Although usually seen in cases with posttraumatic subdural hematoma, few patients 
with hydrocephalus after ventriculoperitoneal shunt have also been reported. We aimed to 
describe two patients presented with calcified chronic subdural hematoma after 
ventriculoperitoneal shunt used for hydrocephalus.

Key words: Ventriculoperitoneal shunt, calcified, subdural hematoma, treatment

Two asymptomatic incidentally discovered 
male patients, aged 30 and 41 years, were 
admitted to our clinic for routine control. 
The patients received ventriculoperitoneal 
shunt for their congenital hydrocephalus 8 
and 10 years ago. Neurological 
examinations revealed normal findings. 
Symptoms of increased intracranial pressure were not observed. Computed 
tomography of the brain revealed a 
calciﬁed subdural hematoma over the left 
fronto-temporo-parietal area (Figure 1a, 
1b). The lesions were hypointense on T1 
weighted magnetic resonance imaging
(Figure 1c). As the hematoma did not compress the brain markedly and cause neurological deficits, conservative treatment was chosen. At one-year follow up, the patients showed no symptoms or signs of increased intracranial pressure.

Calcified chronic subdural hematomas may rarely be a complication of chronic shunting for hydrocephalus. The course of the development of calcification is unclear. It has been suggested that metabolic factors play an important role in the development of calcification. Still others suggest that several vascular factors lead to calcification, such as poor circulation and absorption in the subdural space and vascular thrombosis. The usual interval between hemorrhage and development of calcification varies between 6 months and many years. Thus, whether the development of calcification follows a regressive or progressive course is still controversial. Calcified chronic subdural hematoma has been associated with brain atrophy, thus a hematoma may not cause a mass effect. In addition, a calcified hematoma may sometimes tightly adhere to duramater and cortex and dissection from the brain may cause brain contusion or bleeding. Therefore, removal of this lesion had not been considered necessary or beneficial. Surgical intervention should be limited to patients who have progressive neurological deficits or evidence of increased intracranial pressure. Niwa declared an asymptomatic case, which the calcified mass has been totally removed at the operation, but at the follow-up after 3 months the shift was resumed and the calcification reappeared 10 months after the operation. The surgical intervention for calcified subdural hematoma has no effect on the long term brain atrophy and the symptoms are related to that brain damage. Moreover, the commonly atrophic parenchyma after discharged from its armor is not liable to expand completely so the increased brain volume will not be enough to prevent accumulation of subdural hygroma.

In summary we did not believe that removal of such lesions were necessary or beneficial for our patients. Surgical removal of the calcification is difficult and could damage the underlying cortex, and hence should not be performed routinely. Surgical intervention may be necessary in young patients or in situations where patients are symptomatic.

Figure 1: Ventricular catheter (white arrow) and calcified chronic subdural hematoma (black arrows) is seen on axial CT image (a), ventricular catheter (white arrow) and calcified chronic subdural hematoma (black arrows) is seen on axial CT image (b), and calcified chronic subdural hematoma (white arrows) is seen on T1 weighted coronal MR image (c).
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