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# Huge ossified crust-like subdural hematoma covering the hemisphere and causing acute signs of increased intracranial pressure

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**Abstract** We report the successful removal of an ossified crust-like chronic subdural hematoma (SDH) covering the hemisphere in a 16-year-old boy. In this article, the

importance of the surgical approach is stressed, and the rarity of this condition in the neurosurgical literature is also outlined.

**Key words** Ossified chronic subdural hematoma · Epilepsy · Computed tomography

## Introduction

A review of the literature has revealed that calcified chronic subdural hematoma (SDH) is relatively common, but an ossified one is extremely rare, representing 0.3–2.7% of chronic SDHs [5, 6, 14, 16]. There are only sporadic reports of successful surgical treatment of such SDHs. We document an additional histologically verified case of ossified chronic SDH here.

## Case report

This 16-year-old boy was admitted to our hospital on 29 December 1992 with complaints of epileptic seizures and dysphasia. In the past, he had sustained a head trauma with a short period of unconsciousness (10 years before). He was well, and did not have any symptoms until the last 4 years. Since that time he had suffered from very frequent epileptic attacks which began on the right, and unconsciousness. Because of the epileptic seizures he had been hospitalized in a local hospital and antiepileptic medication consisting of phenytoin was begun. Six days before the admission his convulsive seizures

became worse with gradually developing headache, nausea, and vomiting. On admission he was alert but had dysphasia and a right-sided hemiparesis.

On neurological examination tendon reflexes on the right were found to be increased and Babinski's sign was positive on the right. There was a right-sided spastic hemiparesis, most characteristic in the hand. There were no disturbances of sensation, but papilledema and central paresis of the VII cranial nerve were present.

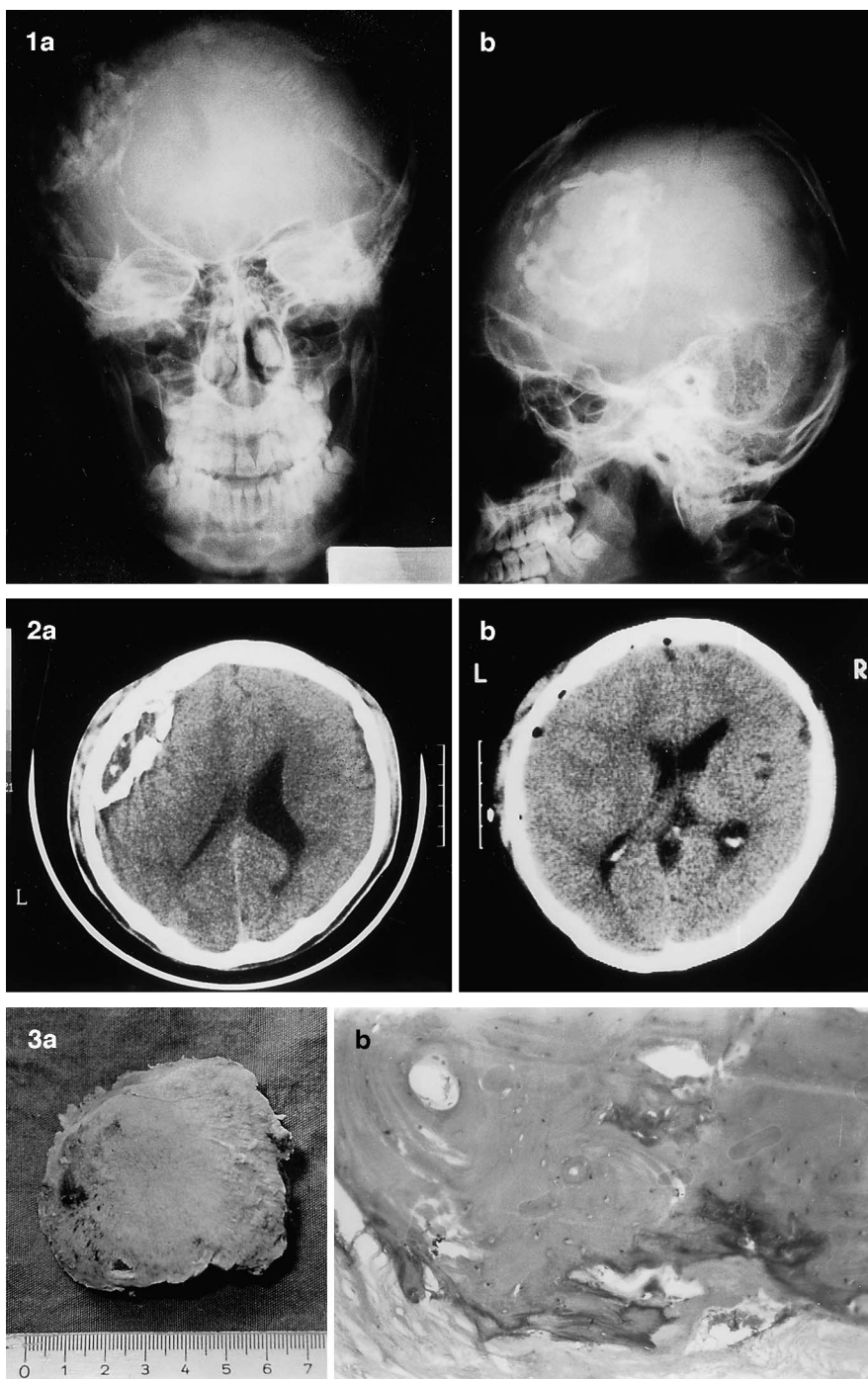
Routine roentgenogram revealed a plate of calcification beneath the inner table on the right, extending from the frontal to the parietal region (Fig. 1a, b). The sella turcica was normal in size and shape, and there was no erosion of the clinoids. Electroencephalography (EEG) was not remarkable apart from some slow wave activity over the left frontoparietal area. Computed tomography (CT) scan showed a hyperdense lesion with a hypodense central part (Fig. 2a).

On the basis of the neurological and the radiological findings the diagnosis of a calcified or ossified chronic SDH was made and we decided upon an operation. On 31 December 1992, a craniotomy was made in the left frontoparietal region. When the bone flap was turned down the dura mater appeared to be normal, but when the dura mater was opened it revealed a stony crust-like hard mass covering the convexity. The dura mater was carefully dissected back, so that this subdural mass was exposed in its entirety. Although the mass was partially adherent to the cerebral cortex, it was easily separated from the surrounding tissue. Total removal of the mass, which measured 7×6 cm, was accomplished (Fig. 3a). Pathological evaluation of the specimen after decalcification revealed a hard material with a typi-

**Fig. 1a, b** Skull X-ray. **a** Anteroposterior and **b** lateral projections of plain X-ray film, showing calcification extending from frontal to parietal region in the left side. Note that there was a highly brachycephalic cranium in addition to the obvious abnormality

**Fig. 2a, b** CT scan. **a** Preoperative CT scan showing a huge mass with high density over the left hemisphere. The central part of the lesion is low density. The lateral ventricle and the sulci are almost completely obliterated on the affected side. There was midline shift from left to right. **b** Postoperative CT scan demonstrating some decrease in midline shift with complete disappearance of the mass shown in **a**

**Fig. 3** **a** Photograph of specimen removed at operation (scale in cm). **b** Photomicrograph of subdural mass removed at operation, showing a typical structure of bone and Haversian canals (haematoxylin-eosin,  $\times 40$ )



cal bone structure and with formation of Haversian canals surrounded by osteoblasts (Fig. 3b).

A postoperative CT scan 4 days after the initial scan demonstrated complete disappearance of the mass (Fig. 2b). By the time of discharge from hospital there were signs of rapid recovery from the focal neurological deficits. With an antiepileptic drug, phenytoin 200 mg/day orally, no epileptic seizures have occurred since the operation. When the patient was seen in the outpatient clinic 13 months later, he was neurologically intact.

## Discussion

Although many techniques for the management of chronic SDH have been advocated in the literature, the pathogenesis of calcification or ossification of chronic SDH remains unclear. According to some authors, metabolic, vascular and/or some local factors play a part in this process [1, 13].

The interval between the initial traumatic event and the development of calcification is generally longer than 6 months [1, 5, 6]. After hyalinization and calcification, the irritation of tissue probably results in the process of ossification, which takes many years in most of the published cases [1, 5, 7, 10]. Calcification of a chronic SDH is more frequent than its ossification, as ossification may be considered a terminal phase of the process. Fortunately, the ossified mass was removed with ease without causing bleeding in our case and in all those documented in the literature.

Clinically, chronic SDH usually presents in one of three ways [2, 3, 8–11, 15]: (1) symptoms of raised intracranial pressure (ICP); (2) fluctuating drowsiness; and (3) progressive dementia. However, in some cases no special pattern is produced by this lesion, as in our case. In our case there were symptoms of elevated ICP and progressive neurological deficits suggesting a cerebral stroke. We think that, in this case, either pressure or vascular compromise secondary to the initial trauma or epileptic seizures was responsible for the neurological symptoms. By contrast, Kotwica and Brzezinski [8] reported that there was no brain damage in their patients with chronic SDHs. They suggest that the capsule of the hematoma plays an important part in the incidence of epilepsy in chronic SDH, and it was developed in 7% of them [8]. McKissock et al. [12] also stated that such a clinical presentation is uncommon and usually associated with a poor prognosis. Our result is at odds with these conclusions; our patient was well after the total removal of the subdural ossified mass.

At present, CT is the method of choice for determination of the location and age of any chronic SDH [3, 7, 11]. The CT appearance of our case was highly characteristic for an ossified chronic SDH: CT revealed a huge ossified crust-like mass covering the hemisphere, discrediting the clinical diagnosis of cerebral stroke made because of the rather sudden onset of the symptoms. In fact, the patient's symptoms are possibly related to Todd's palsy, a focal deficit after seizures, which usually resolves completely with time. Focal neurological deficits may result from the reduction in cerebral blood flow within the area of the brain compressed by a chronic SDH [4, 8]. Recently, Kotwica and Brzezinski [9] also reported that young patients had a higher incidence of such neurological syndromes than do older patients. On the other hand, İplikçioğlu et al. [7] suggest that the removal of a calcified or ossified hematoma is neither necessary nor beneficial in these patients. Our experience is the opposite: progressive focal deficits in a young patient sometimes need a surgical procedure. During the operation, moreover, care had to be taken to prevent a new SDH that could have arisen from an injury to the vascular structures situated between the ossified hematomas and the cortex.

The results of our own and others' experience with chronic SDHs are as follows. The most interesting fact is that the rather acute onset of clinical symptoms in our case suggested a false diagnosis of cerebral stroke initially. Second, the CT scan is always sufficient to determine the correct preoperative diagnosis in the cases of chronic SDHs. Third, it is stressed that early diagnosis and proper surgical treatment are vital, especially in some younger patients.

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