Asymptomatic Calcified Chronic Subdural Hematoma

—Report of Three Cases—

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Abstract

Three adults with asymptomatic calcified chronic subdural hematoma are described. Plain skull x-ray films following head trauma disclosed calcified chronic subdural hematomas and computed tomography (CT) revealed large, frontoparietal, calcified hematomas in all cases. A 49-year-old male received hematoma removal because of his lower age and CT evidence of cerebral compression. An 83-year-old male was not operated on and remained asymptomatic. The third patient died of trauma-related injuries. In the non-elderly, surgery for asymptomatic calcified chronic subdural hematoma with significant cerebral compression should be considered for relief of cerebral compression and prevention of possible future brain damage.

Key words: calcification, chronic subdural hematoma, asymptomatic, ossification

Introduction

Calcification of chronic subdural hematoma is found more frequently in children and young adults than in the aged.^{7,10,17)} In children, mental retardation and epileptic seizures are common symptoms of calcified chronic subdural hematoma.^{23,31)} However, adults frequently present with milder neurological deficits. Asymptomatic cases have also been found incidentally.^{22,25,28-30)} We present here three patients with asymptomatic calcified chronic subdural hematoma and discuss the surgical indications for asymptomatic adults.

Case Reports

Case 1: A 49-year-old male was admitted on June 12, 1982, following a motor car accident during which he struck his forehead on the dashboard. There were no neurological signs or symptoms except for a slight headache and nausea. He had no past history of head trauma or alcohol abuse. Skull x-ray films revealed a large, right frontoparietal, calcified mass. The inner table of the skull directly overlying the mass was scalloped (Fig. 1). Computed tomographic

Fig. 1 Case 1. Plain skull x-ray films. *left:* Anteroposterior view, demonstrating a biconvex calcification in the right parietal region and scalloping of the inner table of the skull just over the calcification. *right:* Lateral view, showing a large frontoparietal calcification.

(CT) scans demonstrated a large, right frontoparietal, mixed hyper- and hypodense mass with a calcified rim. The midline structures were shifted slightly to the left (Fig. 2 *left*). Electroencephalographic findings were normal.

A large frontoparietal osteoplastic craniotomy exposed the calcified hematoma loosely adhering beneath the dura mater (Fig. 3). The inner capsule of the hematoma adhered slightly to the cerebral cortex. These loose adhesions were electrocauterized and cut. The hematoma was totally removed, and weigh-

Fig. 2 Case 1. *left*: Preoperative CT scan, demonstrating a mixed hyper- and hypodense mass with a calcified rim in the right frontal region. The midline structures are slightly shifted to the left. *right*: Postoperative CT scan, showing relief of cerebral compression.

ed 120 g. The hematoma contained a paste-like substance without fresh blood (Fig. 4 *upper*). Histological examination showed that both the inner and outer capsules were layers of connective tissue, characterized by hyalinization and scarcity of cells. Osteoid tissue was present in the outermost layer.

Fig. 3 Case 1. Operative view after dural opening, showing a large calcified mass situated in the subdural space. The cerebral cortex of the right temporal lobe appears normal.

There were no sinusoidal blood vessels within the capsule (Fig. 4 *lower*).

Convulsive seizures of the left face and upper extremity occurred in the immediate postoperative period but subsided within 1 day. He was discharged 1 month after surgery and could return to his previous occupation. Postoperative CT scans show-

Fig. 4 Case 1. *upper*: Lateral (*left*) and transectional views (*right*) of the surgical specimen. The outer capsule of the hematoma is extremely thick compared with the inner capsule. The paste-like hematoma is situated eccentrically. *lower*: Photomicrograph of the outer capsule, showing a thick layer of connective tissue and osteoid tissue with haversian canals. HE stain, $\times 100$.

Fig. 5 Case 2. Coronal CT scan, demonstrating a biconvex isodense mass bordered by a hyperdense calcified rim in the left frontal region. Bilateral cerebral hemispheres are markedly atrophic. The left temporal lobe is more atrophic than the right.

ed expansion of the cerebrum and relief of compression (Fig. 2 *right*). He has remained seizure-free for the last 7 years.

Case 2: An 83-year-old male was referred to our department on April 23, 1987 because plain skull x-ray films taken in a local hospital following a minor head injury revealed an abnormal intracranial calcification. He had no past history of head trauma. No neurological abnormalities or papilledema were

observed. Skull x-ray films demonstrated a faint biconvex calcification in the left frontoparietal region. CT scans showed a biconvex isodense hematoma with a hyperdense calcified rim in the left frontoparietal region (Fig. 5). Bilateral cerebral hemispheres were markedly atrophic. The left temporal lobe showed more atrophy than on the right. Extensive hypodense extracerebral fluid accumulation was noted around the hematoma. Electroencephalograms showed a diffuse α pattern, but no slow-wave focus. No surgical intervention was considered because of his age and good neurological condition. He has remained asymptomatic for the 5-year follow-up period.

Case 3: A 78-year-old male was hit by a motor vehicle while riding a bicycle on January 25, 1992. He struck the occipital region of his head on the ground and was taken to our department immediately after the accident. He had previously been in good health and had no past history of head trauma. On admission, he was comatose and unresponsive to commands. The left pupil was dilated and unreactive to light. Plain skull x-ray films demonstrated a linear fracture of the right parietal bone and an intracranial biconvex calcification in the left parietal region (Fig. 6 *left*). CT scans demonstrated two biconvex hyperdense hematomas with calcified rims in the left frontal and parietal regions, and a diffuse subarachnoid hemorrhage without parenchymal lesion (Fig. 6 *right*). His neurological condition rapidly deteriorated and he died the next day.

Autopsy revealed primary brainstem injury and diffuse traumatic subarachnoid hemorrhage. Two

Fig. 6 Case 3. *left*: Plain skull x-ray film, Towne's view, demonstrating a biconvex calcification in the left parietal region and scalloping of the inner table of the skull over the calcification. A linear fracture of the right parietal bone is also present. *right*: CT scans, demonstrating two isodense masses bordered by calcified rims in the left frontal and parietal region. Diffuse traumatic subarachnoid hemorrhage is also present.

biconvex calcified subdural hematomas were located in the left frontal and parietal regions. The hematoma consisted of a paste-like substance. Histological examination found that both inner and outer capsules were layers of connective tissue, characterized by hyalinization, calcification, and scarcity of cells. There were no sinusoidal blood vessels within the capsule.

Discussion

Calcification and ossification occurs in 0.8–10% of chronic subdural hematoma patients.^{2,12,26,28} Our three calcified chronic subdural hematoma cases are 2.0% of the 153 adult cases of chronic subdural hematoma experienced in our department during the past decade.

Calcified chronic subdural hematomas generally preserve the original crescent configuration of the chronic subdural hematoma and cover a wide area of cerebral surface.^{1,4–6,11,16,21–24,27} The calcified chronic subdural hematomas in our patients were sausage-like in shape, with the greatest diameter in the frontoparietal direction. The hematomas were biconvex or round on transection. Many similar cases have been described.^{3,7–9,13–15,17–20,25,28–32} This characteristic shape suggests that chronic subdural hematoma, which initially covers a wide area of cerebral surface, shrinks in the craniocaudal direction during calcification. Scalloping of the inner table just over the hematoma without cranial asymmetry, characteristic in adult patients, may indicate localized compression toward the inner table during extended periods of calcification.

Regression of a calcified chronic subdural hematoma is difficult to ascertain. Sinusoidal blood vessels and fresh bleeding have been observed in calcified chronic subdural hematomas.^{15,18} Magnetic resonance imaging can differentiate fresh liquid hematoma from old deposits.³² Isu *et al.*¹⁵ reported a patient with an asymptomatic calcified subdural hematoma accompanied by a subacute subdural hematoma following minor head trauma. They presumed that damage to the abnormal vascular networks between the inner membrane and cerebral cortex had caused the subacute subdural hematoma. There have been two cases of initially asymptomatic calcified subdural hematoma presenting with sudden neurological deterioration due to subcortical hemorrhage subjacent to the calcified chronic subdural hematoma.^{8,13} Hypervascular granulation tissue around the calcified hematomas was considered a predisposing factor for subcortical hemorrhage. These findings indicate that regressive and pro-

gressive processes may develop concurrently in and around calcified chronic subdural hematoma.

Some authors consider that calcified subdural hematoma no longer expands and has already caused cerebral atrophy. From this viewpoint, removal of a calcified subdural hematoma provides no benefit, especially in children with long-standing symptoms.^{7,23} However, Mori *et al.*²⁴ reported a 5-year-old boy whose neurological condition deteriorated as a calcified subdural hematoma gradually expanded. Surgery is generally recommended for patients with progressive neurological disorders or increased intracranial pressure,^{24,30,31} but the surgical indications for asymptomatic cases remain controversial. Surgical removal of an asymptomatic calcified chronic subdural hematoma should be based on patient age and degree of cerebral compression shown by CT.²⁹ Surgery for asymptomatic chronic subdural hematoma in the elderly is not generally indicated.^{22,30} We did not operate on our second patient because of his advanced age and good neurological condition. CT showed marked diffuse cerebral atrophy, but this may have been due to aging. However, the left hemisphere, especially the temporal lobe, was more atrophic than the right, and possibly caused by the chronic subdural hematoma. A long-standing subdural hematoma, whether calcified or not, may facilitate cerebral atrophy. We operated on the first patient because of his lower age and CT evidence of cerebral compression. Postoperative CT confirmed expansion of the cerebrum and relief of compression.

In the non-elderly, surgery for asymptomatic calcified chronic subdural hematoma with significant cerebral compression should be considered for relief of cerebral compression and prevention of possible future brain damage.

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