Organized Chronic Subdural Hematoma
Requiring Craniotomy
—Five Case Reports—

Shigeki IMAIZUMI, Takehide ONUMA, Motonobu KAMEYAMA, and Hiroshi NAGANUMA*

Departments of Neurosurgery and *Pathology, Sendai City Hospital, Sendai

Abstract
Two child and three elderly patients underwent craniotomy for organized and/or partially calcified chronic subdural hematomas (CSHs). The characteristic feature of magnetic resonance imaging was a heterogeneous web-like structure in the hematoma cavity. Both children had undergone one side subduroperitoneal shunt for bilateral CSHs when infants. As a result, the opposite hematoma cavities persisted and developed into calcified CSHs after a couple of years. All three elderly patients with senile brain atrophy showed various systemic complications such as cerebral infarction, diabetes mellitus, leg ulceration, cirrhosis, and bleeding tendency. Craniotomy for removal of the hematoma and calcification achieved good results in all patients. Subdural space created by shunt, craniotomy, or brain atrophy and persisting for a certain period, and additional various brain damage such as microcirculatory disorder, meningitis, encephalitis, or premature delivery may be important in generating calcified or organized CSH.

Key words: calcified chronic subdural hematoma, organized chronic subdural hematoma, craniotomy, magnetic resonance imaging

Introduction
Treatment for chronic subdural hematoma (CSH) has tended to change from craniotomy to burr hole trepanation in recent years. However, cases with organized and/or partially calcified CSHs may require craniotomy. The incidence of organization or calcification of CSH is 0.5–2%. Calcified CSH was first described as a finding at autopsy in 1884. The first operation on a large, calcified, cystic subdural hematoma occurred in 1930. Thereafter, numerous reports concerning calcified or organized hematoma have been published. Nevertheless, the etiology, imaging diagnosis, and management have remained obscure, partly because few studies have used modern imaging methods such as magnetic resonance (MR) imaging and brain computed tomography (CT). Moreover, the calcification process has been little described. This study describes two child and three elderly patients with calcified or organized CSH treated by craniotomy.

Case Reports

Case 1: A 3-year, 8-month-old boy was born prematurely weighing 1820 g without distinctive head trauma. The patient was referred to our department because of a syncopal attack lasting for 5 minutes at 7 months after birth. CT demonstrated bilateral classic CSHs (Fig. 1A). Bilateral burr hole trepanations were performed, but no improvement was obtained. Right subduroperitoneal (SP) shunt was then performed (Fig. 1B) and the patient was followed as an outpatient. He was walking at 1 year and 3 months. However, the left CSH began to enlarge 18 months after the right SP shunt. Three years after the first SP shunt, left SP shunt was performed (Fig. 1C) and temporarily decreased the left CSH. One month later, the left subdural tube became infected and an abscess expanded subcutaneously. The infected tube was removed but one month later the left CSH enlarged and calcified on the subdural membrane (Fig. 1D). Three years and 3 months had elapsed from the initial right SP shunt to the appearance of the left calcified CSH. Preoperative symptoms were fever due to meningitis and progressive disturbance...
Fig. 1 Case 1. Computed tomography scans showing the growth process of calcified chronic subdural hematoma (CSH). Bilateral CSHs were initially seen (A). After right subduroperitoneal (SP) shunt indicated by the arrow, the left hemisphere did not reexpand (B). Since the left CSH remained and enlarged, left SP shunt was performed (C) followed by extraction due to infection of the subdural tube. Calcified neomembrane was identified 39 months after the initial right SP shunt (D). Postoperative course was uncomplicated with no recurrence (E).

The patient underwent left frontoparietal craniotomy to remove the calcified CSH. The dura mater was thick and adhered tightly to the calcified outer membrane of the hematoma. Removal of the outer membrane together with the dura mater revealed a dark-gray paste-like hematoma. A hard, calcified, and organized layer was found at the bottom of the hematoma. This calcified inner membrane did not tightly adhere to the brain surface, and could be removed without damaging the brain. The hematoma and the membrane weighed 160 g.

Histological examination of the dura mater showed granulomatous inflammation. Postoperative course was uncomplicated. Five-year follow-up study revealed no deficits, complaints, or recurrence (Fig. 1E).

Case 2: A 2-year, 6-month-old boy presented with emesis after slight head trauma resulting from a fall at home. Bilateral CSHs appeared 3 weeks later (Fig. 2A). The patient underwent SP shunt for the larger left CSH. The left CSH healed, but the right CSH cavity persisted for over a year. Right organized CSH together with clouding of consciousness was noticed 2 years after the left SP shunt. T1-weighted MR imaging showed the subdural hematoma as a web-like heterogeneous high intensity area (Fig. 2B).

A right temporooccipital craniotomy was performed. Incision of the dura mater exposed a dark-red thick outer membrane, which was removed together with the paste-like subdural hematoma. The center of the hematoma consisted of old, yellow, organized, and partially calcified hematoma. The hard calcified inner membrane adhered tightly to the brain. Removal of the inner membrane was incomplete because of the danger of damaging the brain surface.
The patient was discharged on the 11th postoperative day and continues to do well 6 years after surgery with no recurrence.

**Case 3**: A 78-year-old male presented with disturbance of intelligence. He had a history of insulin-controlled diabetes mellitus for 28 years and lacunar infarction 1 year before. He had suffered no distinctive head trauma recently. CT demonstrated left CSH with heterogeneous net-like high density structure in the hematoma cavity (Fig. 3A). T₁- and T₂-weighted MR imaging demonstrated irregular web-like structures in the hematoma cavities described as high intensity areas (Fig. 3B–D).

Burr hole trepanation was first tried, but left temporoparietal craniotomy was necessary to remove the organized CSH with many trabeculae.

Both surgery and postoperative course were uncomplicated, and the patient is presently healthy with no recurrence 6 years after surgery.

**Case 4**: A 69-year-old male presented with left hemiparesis and urinary incontinence. He had suffered from serious diabetes mellitus for 10 years, and cirrhosis, leg ulceration, and bleeding tendency due to thrombocytopenia. He had no history of head trauma. CT demonstrated right CSH with a thickened calcified inner membrane (Fig. 4A). T₂-weighted MR imaging showed calcification on the inner membrane as a low intensity curve and heterogeneous net-like structure in the hematoma cavity appearing as a high intensity area (Fig. 4B).

Burr hole trepanation was first tried, but only the localized hematoma under the burr hole could be absorbed. The operative scalp wound became infected and subcutaneous pyorrhea developed one week after burr hole surgery. Subcutaneous drainage was placed for 2 weeks but since a large amount of the CSH remained, a right frontotemporal craniotomy was performed one month later to totally remove the hematoma and subtotally remove the inner or-
Table 1  Clinical features of patients with organized chronic subdural hematoma (CSH) requiring craniotomy

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/sex</th>
<th>Past history/Complication</th>
<th>Head trauma</th>
<th>Previous treatment</th>
<th>Duration of calcification/Side of the calcified CSH</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3/M</td>
<td>immature infant/lt tube infection, meningitis</td>
<td>–</td>
<td>bil burr hole trepanation, bil SP shunt, lt CSH</td>
<td>3 yrs 3 mos after rt SP shunt/lt CSH</td>
</tr>
<tr>
<td>2</td>
<td>2/M</td>
<td>–/–</td>
<td>+</td>
<td>lt SP shunt</td>
<td>2 yrs after SP shunt/rt CSH</td>
</tr>
<tr>
<td>3</td>
<td>78/M</td>
<td>cerebral infarction, diabetes mellitus</td>
<td>–</td>
<td>lt burr hole trepanation for the present CSH</td>
<td>unknown/lt CSH</td>
</tr>
<tr>
<td>4</td>
<td>69/M</td>
<td>diabetes mellitus, cirrhosis, leg ulceration, thrombocytopenia/rt wound infection, meningitis</td>
<td>–</td>
<td>rt burr hole trepanation for the present CSH</td>
<td>unknown/rt CSH</td>
</tr>
<tr>
<td>5</td>
<td>77/M</td>
<td>diabetes mellitus, bil CSHs (burr holes)</td>
<td>+</td>
<td>–</td>
<td>unknown/lt CSH</td>
</tr>
</tbody>
</table>

SP: subduroperitoneal.

Fig. 5 Case 5.  Computed tomography scan showing double chronic subdural hematomas with a thickened calcified inner membrane (A). Axial T1-weighted magnetic resonance image showing heterogeneous structures in the hematoma cavity and calcified inner membrane as a low intensity curve (B).

Histological examinations

The dura mater, membrane, and clot were examined in all cases. The characteristic findings of infection are presented for Cases 1 and 4 (Table 1). Common findings were a subdural neomembrane consisting of thick, fibrous collagen tissue composed of numerous vessels and many spindle-shaped cells. There were deposits of calcium, hemosiderin, and cholesterol in the neomembranes and trabeculae. The dura mater was thickened, but no histological changes were observed except in one case with calcification. Hematoma clot was old, looked like thombus, and consisted of mostly fibrin and partially organized hematoma.

Discussion

I. Surgical management

The pathogenesis and surgical treatment of classic CSH are controversial, and remain obscure. However, in the past 20 years or so, most neurosurgeons have considered that burr hole trepanation is sufficient for the management of CSH. Burr hole trepanation offers at least equivalent efficacy to craniotomy, with much lower mortality and morbidity and a shorter operative time and ease. Burr hole treatment may interrupt the self-perpetuating...
cycle of microhemorrhages from ectatic subdural neocapillaries by the removal of the hematoma that contains the anticoagulation factors, so that hemostasis and fibrosis can occur.\[10\]

Craniotomy should be reserved for patients in whom the hematoma reaccumulates or residual hematoma membranes prevent reexpansion of the brain.\[10\] Craniotomy is generally accepted as the optimum approach when CSH reaccumulates, there is solid hematoma, the brain fails to expand, or there is marked cerebral swelling subjacent to the hematoma.\[10\] We agree since our five cases of organized or partially calcified CSH correspond to the first three of these conditions and craniotomy achieved good results. The initial burr hole treatment failed in our two elderly patients, and craniotomy was necessary after 1 week (Case 3) and 50 days (Case 4). Previous surgery in the two children (Cases 1 and 2) involved SP shunt for CSHs and one elderly patient (Case 5) with a history of bilateral CSHs treated by burr holes (Table 1). All these patients finally required craniotomy. Preoperative information from CT and MR imaging were useful. We propose that craniotomy is the best choice of treatment for calcified or organized CSH associated with progressive symptoms.

Endoscopic removal of organized CSHs has obtained good results.\[10\] Advantages of the endoscopic approach include access to virtually the entire hematoma cavity through a Perneczky’s \[7,15\] “keyhole” under local anesthesia. This less invasive method provides an alternative technique with similar results.

Surgery for calcified CSH has no effect on longstanding irreversible symptoms caused by cerebral atrophy. The symptoms are dependent on the accompanying underlying brain damage rather than the calcified mass, and removal of such lesions may be neither necessary nor beneficial.\[5,11\] However, calcified or organized CSH should not be surgically ignored.\[20\] Patients with acute or progressive neurological disorders should be evaluated and craniotomy considered.

II. Possible etiology based on the clinical course

Poor circulation, vascular thrombosis, and parathyroid disorder are all causes of calcification, but the mechanism of calcification is still unclear. Three of our five cases (Cases 1, 2, and 5) had histories of bilateral CSHs treated by SP shunt or burr hole trepanation (Table 1). Bilateral CSHs in the two children (Cases 1 and 2) are thought to be due to craniofacial disproportion. The patients had undergone single SP shunt when they were infants. As a result the opposite hematoma cavities remained and developed into calcified CSHs after a few years (Table 1). Two boys with CSH and histories of ventriculo-atrial shunt for hydrocephalus developed calcification of the CSHs after 2 years and 4 years, respectively.\[18\] A 5-year-old boy who underwent ventriculoperitoneal (VP) shunt for hydrocephalus 5 months after birth experienced meningitis at the age of 2 years and bilateral calcified CSHs at 3 years before undergoing bilateral cranietomies.\[12\] A 39-year-old female with an organized CSH and Crouzon disease had a history of VP shunt one year previously and underwent burr hole surgery, but removal of the organized hematoma failed and craniotomy was refused by the family.\[14\] Six children aged from 9 to 15 years with calcified CSH had all undergone bilateral or left craniotomy for subdural hematoma and suffered meningitis or encephalitis in their infancy.\[11\] Three of the six children were prematurely born.

Previous cases suggest that one side shunting continuously drains cerebrospinal fluid or hematoma adjacent to the ipsilateral brain and prevents reexpansion of the displaced brain, causing the hematoma cavity to persist. Microcirculatory disorder caused by arteriosclerosis or diabetes mellitus in the hematoma cavity may cause calcium deposition in the chronic necrotizing connective tissue.\[4,11,12\] Meningoencephalitis associated with the postoperative hematoma cavity could induce generation of calcification. Calcium deposition could also occur in the degenerated inner membrane. Meningitis after SP shunt or burr hole trepanation was seen in two (Cases 1 and 4) of our five patients. The three elderly patients (Cases 3–5) had various complications such as diabetes mellitus, cirrhosis, leg ulceration, thrombocytopenia, and cerebral infarction. All these metabolic or circulatory disorders could have caused microcirculatory disturbance resulting in chronic necrosis with calcium deposition in the inner membrane.

III. Process of calcification

Most cases of calcified or organized CSHs have been reported without description of the patients’ histories before calcification. Our Cases 1 and 2 are the first to document the growth of calcified CSHs. The calcification in our cases took a couple of years (Table 1). Hematoma should exist for at least 3 years before calcification is possible, but in one case calcification became visible on skull roentgenography after only 3 months.\[3\]

IV. Imaging diagnosis

Preoperative diagnosis of organized or partially calcified CSH is important for the selection of the therapeutical strategy. Few reports\[12,14,17\] have de-
scribed CT findings and only one paper\(^1\) showed MR imaging findings. Combination of the CT and MR imaging is very useful for making the diagnosis preoperatively. Firm calcification on the subdural neomembrane appears as bone on CT (Figs. 1 and 4) and MR imaging (Figs. 4 and 5). However, organized or partially calcified hematomas appear as heterogeneous moderate high density areas on CT (Figs. 3 and 5), and as mixed areas consisting of slight low and high intensity lesions on T\(_1\)-weighted MR imaging, with heterogeneous web- or net-like appearances in the hematoma cavities shown as high intensity areas (Figs. 2–5). Since subdural effusion appears as a homogeneous low intensity area, differentiation is possible. T\(_2\)-weighted MR imaging also shows the calcification as characteristic heterogeneous structures in the hematomas (Figs. 3 and 4).

V. Conclusion

Our five cases of progressive organized or partially calcified CSH show an association with failure of cerebral reexpansion with or without additional brain damage such as infection and/or microcirculatory disturbance. In the child patients, SP shunt prevented reexpansion of the opposite side of the brain and maintained the subdural space for a certain period. In the elderly patients, senile cerebral atrophy or preceding surgery may have prevented reexpansion of the brain with various complications (Table 1). These conditions can cause calcification or organization on the subdural neomembrane. T\(_1\)-weighted MR imaging showed organized or partially calcified subdural hematoma as a characteristic heterogeneous web-like appearance. We propose that craniotomy is the best choice of treatment for calcified or organized CSHs manifesting as progressive symptoms.

References

2) Araujo JF, Gracia Iafigliola M, Jose Balbo R: [Chronic subdural hematoma: analysis of 35 cases]. Arq Neuropsiquiatr 54: 71–74, 1996 (Port, with Eng abstract)

Address reprint requests to: S. Imaizumi, M.D., Department of Neurosurgery, Sendai City Hospital, 3–1 Shimizukoji, Wakabayashi-ku, Sendai 984–8501, Japan.