Calcified Chronic Subdural Hematoma — Case Report

Hung-Zen Yang, Sheng-Hong Tseng[1], Yun Chen[1], Swei-Ming Lin[1], Jin-Cherng Chen[2]

Department of Surgery, Hualien Armed Forces General Hospital, Hualien, Taiwan; Department of Surgery[1], National Taiwan University Hospital and National Taiwan University, College of Medicine, Taipei, Taiwan; Department of Neurosurgery[2], Buddhist Dalin Tzu Chi General Hospital, Chiayi, Taiwan

ABSTRACT

Chronic subdural hematoma (CSH) is a well-known disease entity; however, calcified chronic subdural hematoma (CCSH) is uncommon. We report on a 59-year-old man who had sudden onset of left hemiplegia one year before admission. Brain CT scan showed right thalamic hemorrhage and one large calcified subdural hemaotma over the right fronto-temporo-parietal area. No surgical intervention was done then. He had a regular rehabilitation program and his muscle power improved gradually. However progressive deterioration in left side muscle power and left hemibody numbness was noted recently. He could not walk and needed to use a wheel chair. Unenhanced computed tomography and magnetic resonance imaging of the brain showed a CSH with calcified capsule over the right fronto-temporo-parietal area and the hematoma had a marked mass effect. He underwent surgery and the CCSH was excised totally. The patient recovered well. He could walk with a quadricane soon after surgery. He is improving after one year and 2 months of follow up. We feel surgical treatment for CCSH is feasible and often results in neurological improvement. (***Tzu Chi Med J*** 2004; **16**:261-265)

Key words: calcification, calcified chronic subdural hematoma, chronic subdural hematoma, surgery

INTRODUCTION

Chronic subdural hematoma (CSH) is a well known disease entity; however, calcified chronic subdural hematoma (CCSH) is uncommon [1-4]. The incidence of CCSH has been reported to be 0.3% to 2.7% of all CSHs [1-4]. There are about 100 cases reported in the literature [5]. Although surgical treatment for the CSH is widely accepted, there is still some controversy about whether it should be used [1,5-11]. In this report, we present a patient with symptomatic calcified chronic subdural hematoma who underwent successful excision and recovered well.

CASE REPORT

A 59-year-old man had had sudden onset of left hemiplegia one year before admission. Brain CT scan showed right thalamic hemorrhage and one large calcified subdural hemaotma over the right fronto-temporo-parietal area. No surgical intervention was done then. He had a regular rehabilitation program and his muscle power improved gradually. However progressive deterioration in left side muscle power and left hemibody numbness was noted recently. He could not walk and needed a wheel chair. On admission, he was conscious; however, mild left facial palsy was noted. The muscle power was grade 3/5 over the left upper limb and grade 3/5 over the left lower limb. His deep tendon reflex was increased in the left limbs, and he had an extensor plantar response on the left side, and plantar flexion on the right. No significant sensory or sphincter dysfunction was noted. Unenhanced computed tomography (CT) of

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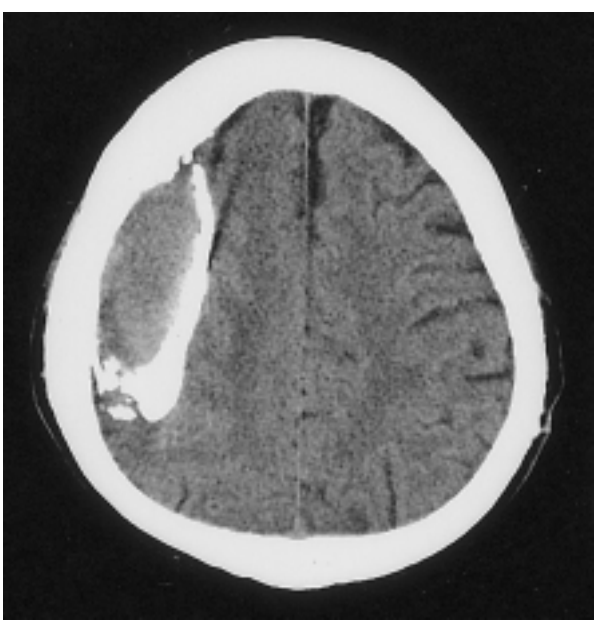
Address reprint requests and correspondence to: Dr. Jin-Cherng Chen, Department of Neurosurgery, Buddhist Dalin Tzu Chi General Hospital, 2, Min Sheng Road, Dalin, Chiayi, Taiwan

the brain revealed a calcified chronic subdural hematoma over the right fronto-temporo-parietal area, and the hematoma compressed the brain markedly (Fig. 1A). The lesion was hyperintense on T1 weighted magnetic resonance imaging (MRI) (Fig. 1B). The patient underwent craniotomy. An organized chronic subdural hematoma with calcified capsule was found over the right fronto-temporo-parietal area and the brain was markedly compressed by the hematoma. The dura was tightly adhered to the calcified capsule of the chronic subdural hematoma; however, the arachnoid membrane over the brain was intact and not adhered to the hematoma. The calcified CSH including the overlying dura was totally excised. The dura was repaired with a neuropatch. The postoperative course was uneventful and his muscle power improved. The pathology revealed an organized hematoma with calcification (Fig. 2). After one year and two months of follow up, he was doing well and his muscle power was nearly normal with some residual numbness and weakness due to previous right thalamic hemorrhage.

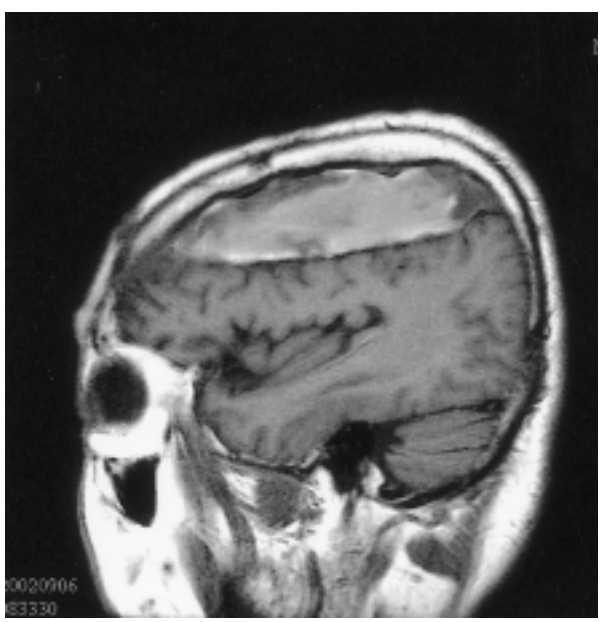
DISCUSSION

Calcified chronic subdural hematoma is uncommon [1-5] and occurs more frequently in children and young adults than in the aged [12-14]. Most CCSHs are located at the convexity [5] and the extent of the calcification varies widely, sometines involving the entire hemispheric surface [9]. The CCSH in our patient was at the convexity, although he was relatively older than most patients.

Most CCSHs can be diagnosed by CT or MRI [5, 15-19], and differentiated from the usual CSH by imaging studies and gross pathology. CCSHs have the following characteristics: (1) elliptical shape with the longest diameter in the frontotemporal direction; biconvex or flat shape on cross section, (2) the content is gelatinous or clay-like, but not liquefied, (3) the inner membrane is thick and has sinusoidal blood vessels, (4) the inner membrane is partially adhered to and evaginated into the cerebral cortex [12,20]. Our patient was diagnosed with CCSH because the CT and MRI showed a chronic subdural hematoma with calcified wall and the CCSH had all the characteristics mentioned above except the inner membrane was not adhered to the brain. Sometimes the CCSH may be confused with other calcified extra-axial space-occupying lesions, such as calcified epidural hematoma [17], calcified subdural empyema [21], meningioma [15,18], calcified arachnoid cyst [19], and calcified convexity dura mater with acute

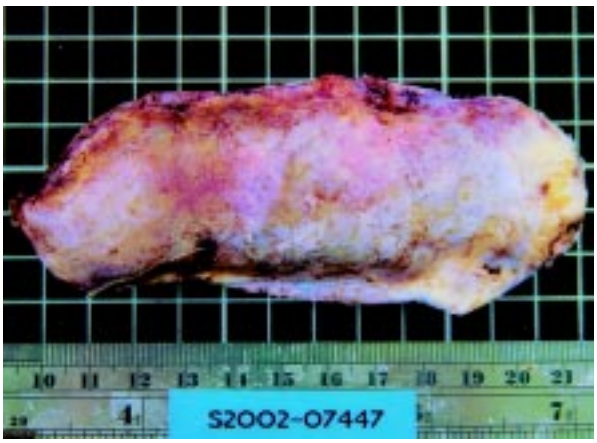


1A

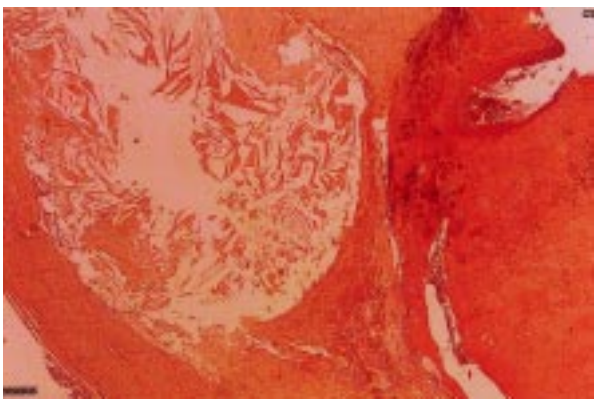


1B

Fig. 1. Computed tomography (CT) and magnetic resonance imaging (MRI) of the calcified chronic subdural hematoma. (A) Axial section of the unenhanced CT scan of the brain, showing a subdural lesion with calcified wall over the right fronto-temporo-parietal area with marked mass effect. (B) Sagittal section of the T1 weighted MRI, showing a hyperintense subdural lesion over the right fronto-temporo-parietal area with marked mass effect.



2A



2B

Fig. 2. Gross pathology and histopathology of the calcified chronic subdural hematoma. (A) Photograph of the calcified chronic subdural hematoma. (B) Histopathology of the calcified chronic subdural hematoma, showing old blood clots with organized changes and fibrocalcified capsule. (H & E 100×)

epidural hematoma [16]. Among these diseases, CCSH is most often confused with calcified subdural empyema and intraoperative aspiration of pus is often needed to confirm the diagnosis [5,21]. Our patient was afebrile and was in good general condition; in addition, the operative findings confirmed the lesion was a CCSH, not a calcified subdural empyema.

The course of the development of calcification in a CCSH is unclear. However, the hematoma may progress gradually from hyalinization to calcification, and finally ossification through irritation of the tissue. After hemorrhage calcification usually takes 6 months to many years to develop [1,3,6,22]. It is difficult to understand the mechanisms of the development of a calcified CSH, because it takes a long course. Poor circulation and absorption into the subdural space and vascular thromboses, inherent metabolic tendency to calcification, prolonged existence of the hematoma in the subdural space, stagnant blood due to sufficient arterial supply and inadequate venous return, thick connective tissue membrane, and other local factors are considered to contribute to the development of calcification of CSH [6,10]. We had no evidence to indicate any of these mechanisms contributed to the development of the CCSH in our patient.

CCSHs may manifest in seizures, mental and physical retardation, hemiparesis, and gait disturbance [5]; however, some patients are asymptomatic in spite of a large hematoma [3,23]. Thus whether the development of CCSH follows a regressive or progressive course is still controversial. The CCSH has been associated with brain atrophy, thus a hematoma may not cause a mass effect [3,10]. In addition, a calcified hematoma may sometimes tightly adhere to the dura mater and cortex and dissection from the brain may cause brain contusion or bleeding [3,6]. Therefore, removal of this lesion had not been considered necessary or beneficial [10]. However, a CCSH can be an active lesion which grows like a neoplasm. There is also a risk of hemorrhage as evidence of vascular proliferation has been noted in the capsule of calcified chronic subdural hematoma [3,8, 20,24]. Thus, surgical intervention is favored for a progressively enlarging CCSH. In recent years, successful removal of calcified CSH with good neurological recovery had been reported in sporadic cases [1,5-9,11]. Removal of the CCSH reduces the mass effect and cerebral irritation, and increases the cerebral blood flow, thus patients can improve neurologically after surgery [5]. The good postoperative neurological recovery in our patient confirmed that surgery is beneficial for patients with symptomatic CCSH, especially for those with clinical deterioration [1,5-9,11]. On the other hand, there is no consensus about surgical treatment for asymptomatic CCSHs. However, good outcomes in some asymptomatic cases [3,23] and fears of an actively growing lesion suggestthat surgery might also be considered in asymptomatic patients.

In summary, we presented a patient with an uncommon calcified chronic subdural hematoma, which was successfully excised resulting in a good recovery. From the literature review and the experience in our patient, we feel surgical treatment for CCSH is feasible and often results in neurological improvement.

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鈣化性慢性硬腦膜下血腫一病例報告

楊宏仁　曾勝弘[1]　陳　芸[1]　林瑞明[1]　陳金城[2]

國軍花蓮總醫院外科　台大醫院暨台灣大學醫學院外科部[1]　佛教大林慈濟綜合醫院神經外科[2]

摘要

慢性硬腦膜下血腫為常見之疾病，然而鈣化性硬腦膜下血腫並不常見，我們報告一例59歲男性患者，住院前一年曾突發左側肢體乏力，經頭部電腦斷層影像檢查，顯示有右側視丘腦出血及右側大片鈣化硬腦膜下血腫。當時並未進行手術治療，患者接受規則藥物及復健治療，病患肌力日漸改善。近來，病患左側肌力退步，且有麻木現象，無法行走，需倚賴輪椅代步。頭部電腦斷層及核磁共振掃描檢查，顯示右側鈣化硬腦膜下血腫併明顯腫塊效應，病患接受開顱完全切除鈣化硬腦膜下血腫，術後恢復良好，很快即可柱杖行走，麻木感亦緩解。經過一年兩個月之追蹤，並無惡化現象，依據文獻回顧，及治療此病人之臨床經驗，我們認為，慢性硬腦膜下血腫之手術治療確實可行，且通常可造成神經功能之改善。(**慈濟醫學** 2004; **16**:261-265)

關鍵語：鈣化，鈣化性硬腦膜下血腫，慢性硬腦膜下血腫，外科手術

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