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An early presentation of a large calcified chronic subdural hematoma presenting as an inner eggshell in an elderly patient: A case report

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ABSTRACT

INTRODUCTION: Calcified chronic subdural hematoma which is usually found in children and takes several months for calcification to be seen on imaging is presented in an elderly patient in which the calcification could be visible early after trauma.

PRESENTATION OF CASE: An elderly man, 83, living with caregiver, was brought to our hospital due to dysarthria and suffocation for 2 days. Three months ago, he experienced his head injury from a fall. Four days ago, he developed progressive left hemiparesis. Because of clinically progressive deterioration and computed tomography scan revealing acute epidural hematoma, the patient was transferred to operating room. Gross findings and pathologic reports unfolded that the lesion was consistent with calcified chronic subdural hematoma. Despite a successful operation, the patient cannot survive.

DISCUSSION: In addition to the presentation in an elderly patient, there was scarcely any paper reporting an early presentation of calcium deposition. Nevertheless, according to the lack of previous data recorded for his earlier performance, deviation on walking or sitting, before the accident, we cannot actually know whether there has been calcium deposition in his brain before or not.

CONCLUSION: Calcified chronic subdural hematoma could present in elderly group of patient and early presentation of calcium deposition following trauma is possible. However, surgery in this group of patient entails a risk of fatality, despite the successful operation.

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1. Introduction

Calcification of chronic subdural hematoma is an extremely rare condition. The incidence is about 0.3–2.7% of all chronic subdural hematoma [1]. The lesion most commonly occurred in children [2]. Generally, the calcification takes several months to a year to occur on imaging. An appearance on computed tomography (CT) scan can mimic meningioma, epidural hematoma or even acute epidural hematoma with calcification, therefore, definite diagnosis is difficult. Surgery is not recommended for asymptomatic and elderly patients unless the patients are clinically progressively deteriorated. We did observe an elderly male patient with a large calcified chronic subdural hematoma at his right hemisphere. The calcified lesion take only a few months to be seen and the definite diagnosis

was made after surgery. Finally, surgery that cannot be avoided entails a risk of death in this case.

This work is compliant with the SCARE checklist, and also, has been reported in line with the SCARE criteria [3].

2. Presentation of case

An 83 year-old male patient daily cared for by members of staff at a nursing home was brought to our emergency department due to dysarthria and suffocation for two days. Previously, he was conscious and the only his underlying disease was benign prostatic hyperplasia which was treated with medication. With an unknown cause, he always deviated to the left side while he walk or sit. In the past three months, he experienced his head injury from a fall where his head hit the ground. He had, however, no additional neurological deficits, so he did not come to hospital. But for 4 days earlier, he had developed progressive left hemiparesis. His caregiver noticed that he had a weakness on his left side, could not move his left arm as normal, and had increased left-sided deviation on walking or sitting down.

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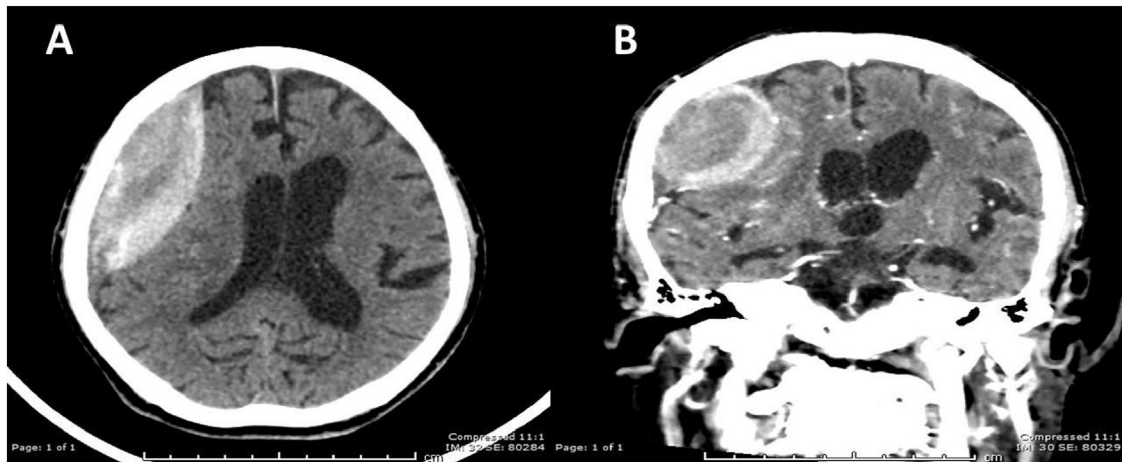


Fig. 1. CT scan revealed biconvex heterogenous hypodensity extra-axial collection about $3.4 \times 8.2 \times 4$ cm with thickened rim calcification along right frontoparietal convexity and right coronal suture in transverse (A) and coronal (B) view.

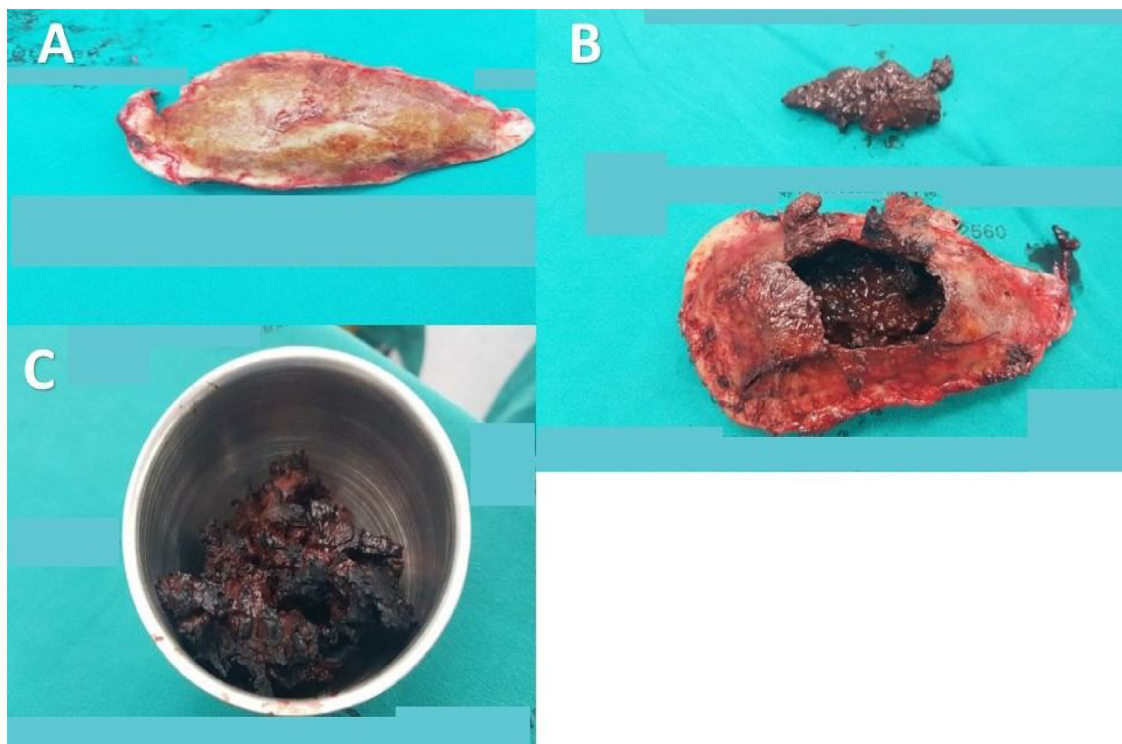


Fig. 2. The appearance looked like double yellowish thick walls of an egg shell stuffed (A and B) with soft clay-like content (C).

At our emergency room, his vital signs were stable and Glasgow coma scale (GCS) showed 15/15 (E4V5M6) but slowly responsive. Some degree of dysarthria was observed. His right pupil was 3 mm in diameter reactive to light while on the right side could not be evaluated due to its abnormal shape. He had left-sided hemiparesis grade I equally in both upper and lower limbs. According to desaturation with pulse oximetry dropping to 91% and the presence of wheezing in both lungs, the patient was therefore intubated at that time. Subsequently, non-contrast computed tomography (CT) scan was performed, revealing biconvex heterogenous hypodensity extra-axial collection about $3.4 \times 8.2 \times 4$ cm (width \times anteroposterior length \times height) with thickened rim calcification about 8 mm along right frontoparietal convexity and right coronal suture. Inward displacement of adjacent enhancing cortical vessels were seen. And also mild swelling of right cerebral hemisphere with compression to ipsilateral lateral ventricle and midline structure

shifting to the left about 5 mm were noted (Fig. 1). Following such findings which could explain his progressive left hemiparesis, the provisional diagnosis of acute epidural hematoma has been made and the patient was then transferred to the operating room.

At the theatre, a fronto-parieto-temporal craniotomy was performed. Intra-operatively, a mass, approximately $12.5 \times 6 \times 2$ cm in size, made up of a huge calcified chronic subdural hematoma was found under dura mater. There was no any bloody content above the dura. The appearance looked like double yellowish thick walls of an egg shell stuffed with soft clay-like content. The outer and inner membranes could be easily peeled off from dura mater and underlying brain surface without cortical injury (Fig. 2). Pathologic examination showed red blood cells accumulated as hematoma which was encapsulated by dense fibrocollagenous tissue within the sac-like structure. The fibrous wall also revealed scattered dystrophic calcifications and some hemosiderin-laden macrophage

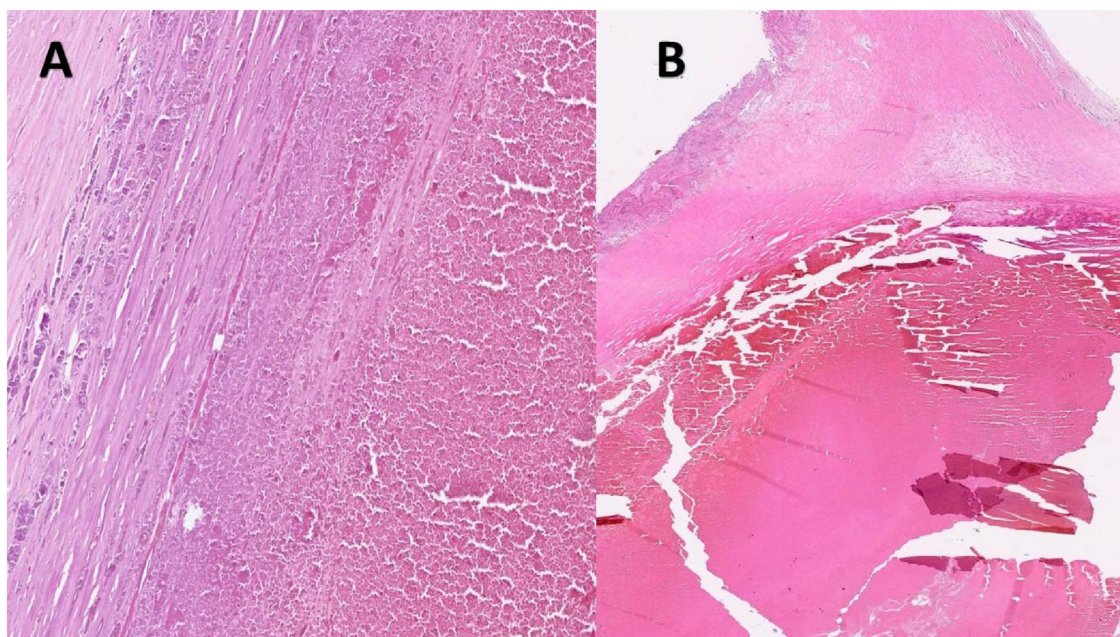


Fig. 3. Pathologic examination (A, $\times 100$) and (B, $\times 40$) showed red blood cells accumulated as hematoma which was encapsulated by dense fibrocollagenous tissue within the sac-like structure. The fibrous wall also revealed scattered dystrophic calcifications and some hemosiderin-laden macrophage infiltration.



Fig. 4. A follow-up CT scan showed hypodensity lesion at right fronto-parietal area without midline shift suggesting cerebral infarction possibly due to brain compression from the calcified chronic subdural hematoma.

infiltration. Neither atypical cells nor malignant cells were seen (Fig. 3).

Post-operatively, the patient regained some consciousness and could obey orders or requests. Voluntary movement on the left hemiparesis, however, still was at the same grade (grade I). A follow-up CT scan showed hypodensity lesion at right fronto-parietal area without midline shift suggesting cerebral infarction possibly due to brain compression from the calcified chronic subdural hematoma (Fig. 4). Although infarction of right cerebral artery greatly concerned us that it might be another cause of the lesion, antiplatelet could not be administered due to immediate postoperative period following a major surgery. The only management we could perform was conservative treatment. A few days later, CT scan revealed no increase in hypodensity of the lesion and swelling of the brain. Unfortunately, the patient developed ventilator-associated pneumonia, atrial fibrillation, and heart failure in which the diagnosis of Takotsubo cardiomyopathy had been made. Although medications covering all medical problems including infection and atrial fibrillation were immediately given and

tracheostomy was also performed due to prolonged intubation, the patient finally passed away from severe pneumonia.

3. Discussion

Chronic subdural hematoma is not uncommon; however, calcified chronic subdural hematoma is extremely uncommon with an incidence about 0.3–2.7% of all cases of chronic subdural hematoma [1]. Generally, the conditions have more affected children than adults [2], but in this presentation, we presented this rare condition in an elderly patient.

Various etiologies of the calcification have been reported. These included traumatic subdural hematoma, secondary to ventricular shunting procedure, post-meningitis or encephalitis, and tumors of central nervous system [2,4–7]. It seems that our patient's lesion was caused rather by trauma than other causes. A plausible explanation introduced in previous reports suggested that a number of factors including poor circulation and absorption within subdural space, stasis and long standing of hematoma, vascular thrombosis and adequate arterial supply but with poor venous outflow of the membrane might play a role for the development of calcification. In the reports, the calcium deposition usually take at least 6 months to many years to be visible on roentgenography in most of the patients; however, in our presentation, the calcification was seen on CT scan only after 3 months following the patient's fall. This early calcium deposition had been reported only once in the literature [2,8–11]. Nevertheless, according to the lack of previous data recorded for his earlier performance, deviating to the left side while he walk or sit, before the falling, we cannot actually know whether there has been calcium deposition in his brain before and later this fall caused acute bleeding inside.

In addition to the hemiparesis and gait disturbance presented in our patient, other clinical manifestations include dementia, deteriorated vision and headache, vomiting, seizure or alteration of conscious secondary to increased intracranial pressure [8,9,12]. Frequently, failure to thrive or mental retardation occurred in children [13]. However, the definite diagnosis is made by CT scan or magnetic resonance imaging and pathology. Although the imaging characteristics in this patient as previ-

ously aforementioned suggested that acute epidural hematoma and epidural hematoma or meningioma with calcification could not be excluded, the lesion found at surgery and pathologic report confirmed the diagnosis of calcified chronic subdural hematoma.

Management of the condition is currently controversial. Conservative treatment is usually advocated for the patients who are asymptomatic, elderly, or without progressive neurologic deterioration [5,12–15]. While surgical intervention is recommended in patients who are progressively deteriorated and have neurologic symptoms that cannot be explained by the lesion [5,12,14]. As our patient was not clinically stable, although the patient was elderly, surgery was indispensable. Total removal of the calcified mass is usually technically difficult since the inner membrane may tightly adhere to the brain cortex and trying to detach can cause the underlying brain injury. Unfortunately, despite the success removal of lesion without difficulty, the patient cannot survive the post-operative medical conditions.

4. Conclusion

As we have shown, calcified chronic subdural hematoma could present in elderly group of patient and early presentation of calcium deposition following trauma is possible. However, surgery in this group of patient should be avoided and should the surgery is imperative it entails a risk of fatality, despite the successful operation.

Declaration of Competing Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Ethical approval

The consent form and information sheet using in the process of obtaining a consent were approved by IRB at our institution.

Consent

The patient's wife has been informed prior to the conduction of this manuscript and informed consent has also been obtained. A copy of the written consent is available for review by the editor-in-chief of the journal on request.

Author contribution

Piyaporn Songnatsiri collected data and wrote manuscript.

Tanawat Ounahachok, Taywin Atikankul and Juthamas Thananon contributed to conceptualization.

Paiboon Sookpotarom contributed to conceptualization, data curation, supervision and editing of the manuscript.

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