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Case Report

Chronic calcified subdural hematoma masquerading as hemorrhagic extra-axial mass: A case report and brief review of the literature ☆,☆☆

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ABSTRACT

Calcified chronic subdural hematoma (CCSDH) is a rare condition characterized by the accumulation of calcified blood between the dura mater and arachnoid membrane, typically following remote trauma. These lesions often present as space-occupying, extra-axial masses over the cerebral convexity and can mimic extra-axial tumors, such as calcified meningiomas. A 73-year-old male with a history of prostate cancer, hypertension, and hyperlipidemia presented with vision changes and mild papilledema. CT imaging revealed a hyperdense, heterogeneous mass with localized mass effect, while MRI suggested a calcified meningioma due to minimal enhancement and a Dural tail. Given the mass effect and neurological symptoms, surgical evacuation was performed, and intraoperative findings confirmed a chronic subdural hematoma. Postoperatively, the patient's symptoms improved, and imaging confirmed complete hematoma evacuation. This case highlights the challenges in diagnosing CCSDH, as it can closely resemble other conditions like meningiomas, emphasizing the need for careful radiological assessment and timely surgical intervention.

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Background

A calcified chronic subdural hematoma is an extremely rare and infrequent diagnosis, with only a handful of cases reported in the literature [1]. The clinical presentation of a

chronic subdural hematoma differs significantly from that of a calcified subdural hematoma. Calcified or ossified chronic subdural hematomas typically present as space-occupying, extra-axial calcified masses over the cerebral convexity. These lesions are associated with signs and symptoms related to mass effect and may mimic extra-axial tumors. A history of

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remote trauma or the use of antiplatelet or anticoagulant medications is often noted [1–6].

On CT, calcified chronic subdural hematomas appear as extra-axial masses with a thick, calcified rim, often containing discontinuous chunks of calcium or ossification. They are frequently associated with localized mass effect. Layers of varying densities—lower, equal, or higher density than the surrounding brain tissue—may also be observed. Calvarial changes around the margin of the mass are not uncommon. On magnetic resonance imaging (MRI), the presentation can vary depending on the state of the hemorrhage, typically showing intrinsic septations and heterogeneous signal intensities, with little or no enhancement [1,7].

The differential diagnosis based on imaging includes calcified subdural hematomas, calcified epidural hematomas, meningiomas, calcified arachnoid cysts, or hemorrhagic intracranial metastases [2–5,8–10].

Management depends on the clinical presentation. Surgical removal is recommended for patients with acute or progressive neurological deficits. Asymptomatic calcified subdural hematomas should also be removed if they cause significant cerebral compression to prevent potential future brain damage [1,3,7].

Here, we present a case of a chronic calcified subdural hematoma that, on imaging, mimicked a calcified meningioma.

Case report

A 73-year-old man with a remote history of prostate cancer, hypertension, and hyperlipidemia presented with intermittent vision changes in the right eye. He reported no prior history of trauma or use of anticoagulants/antiplatelet agents. Vital signs and physical examination were unremarkable. Fundoscopic examination revealed mild bilateral papilledema. A subsequent CT scan was recommended for further evaluation.

Unenhanced CT of the head (Fig. 1) showed a large, left parietal convexity, heterogeneous, hyperdense collection with lo-

cal mass effect on the brain parenchyma, but without midline shift. There were no calvarial fractures or scalp contusions, and no subjacent parenchymal edema was noted. The differential diagnosis included chronic walled-off intracranial hemorrhage or a hemorrhagic extra-axial lesion. The extra-axial location made it an atypical presentation for a chronic walled-off hemorrhage. Additionally, the patient's lack of anticoagulant or antiplatelet use and absence of trauma made this diagnosis unlikely. As a result, further evaluation with MRI was recommended.

MRI (Fig. 2) revealed that the left parietal convexity extra-axial lesion exhibited heterogeneous T1-hyperintense and T2-hypointense signal characteristics, with minimal enhancement within the lesion. There was also uplifting of the dura along the anterior and posterior aspects of the lesion, showing subtle heterogeneous enhancement, which was interpreted as a dural tail. Given the subtle heterogeneous enhancement within the lesion and presumed dural tail, the leading differential diagnosis was calcified meningioma, with less likely possibilities including walled-off extra-axial hemorrhage or a metastatic lesion.

Due to the presence of papilledema and local mass effect, the decision was made to proceed with surgical evacuation. Intraoperative images (Fig. 3) confirmed the diagnosis of a chronic walled-off subdural hemorrhage. Postoperative CT (Fig. 4) showed complete evacuation of the chronic hematoma. Following surgery, the patient reported symptom improvement and resolution of papilledema.

In this case, we present a chronic walled-off subdural hematoma that masqueraded as a calcified meningioma.

Discussion

Chronic subdural hematoma is a collection of blood between the dura mater and the arachnoid membrane, typically covering one or both cerebral hemispheres. It results from the tearing of the bridging veins near the sagittal sinus and persists for more than 21 days. Chronic subdural hematoma

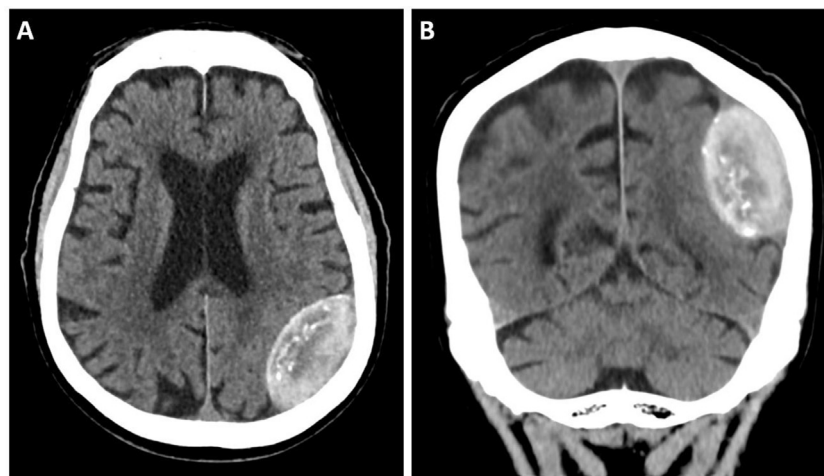


Fig. 1 – Unenhanced CT of the head, showing axial (A) and coronal (B) images, reveals a large, heterogeneous hyperdense collection in the left parietal convexity, without subjacent parenchymal edema. There is local mass effect on the brain parenchyma, but no midline shift. No calvarial fractures or scalp contusions are noted.

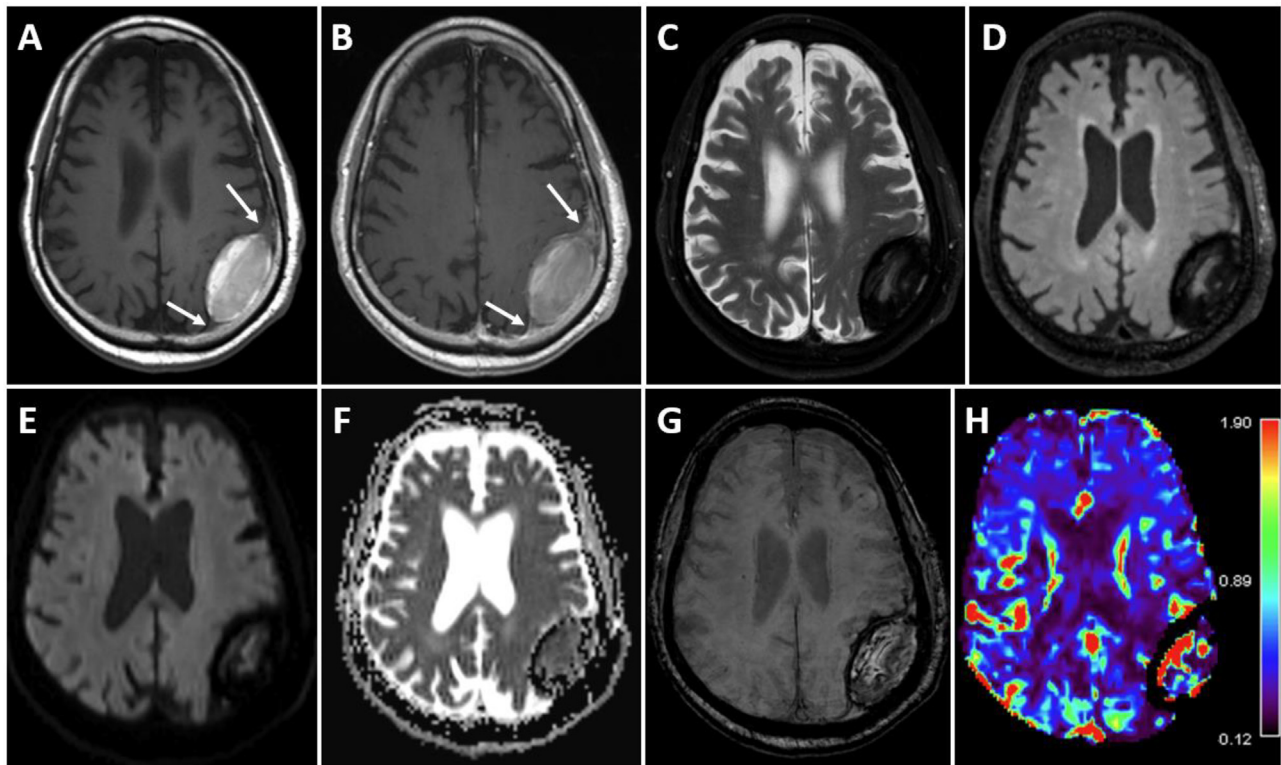


Fig. 2 – Axial T1-weighted (A), axial T1-weighted post-contrast (B), axial T2-weighted fat-suppressed (C), axial T2-weighted fluid-attenuated inversion recovery (FLAIR) (D), axial diffusion-weighted (E), axial apparent diffusion coefficient (F), axial susceptibility-weighted (G), and axial leakage-corrected cerebral blood volume maps demonstrate an extra-axial focus in the left parietal convexity. The lesion exhibits heterogeneous T1-hyperintense and T2-hypointense signal characteristics, with minimal enhancement on the post-contrast T1-weighted sequence. Uplifting of the dura along the anterior and posterior aspects of the lesion is noted, with subtle enhancement (A and B, white arrow). The lesion also shows susceptibility artifacts. Dynamic susceptibility contrast (DSC) perfusion imaging indicates increased relative cerebral blood volume (rCBV); however, due to the presence of susceptibility artifacts, this finding is likely artifactual. Given the subtle heterogeneous enhancement within the lesion and along its anterior and posterior aspects, the leading differential diagnosis includes calcified meningioma or a hemorrhagic extra-axial lesion, with other possibilities including a calcified walled-off extra-axial hemorrhage or a metastatic lesion.

usually follows an injury, although the trauma may be minor, and the patient may not recall the event. The first report of calcified chronic subdural hematoma came from von Rokitsky during an autopsy. Calcification typically develops after an interval of more than 6 months. Whether the development of calcified chronic subdural hematoma follows a regressive or progressive course remains controversial [1,5,7,11].

The calcification process may result from “regressive” changes, such as poor absorption of the hematoma in the subdural space, calcium deposition, and hyalinization of connective tissue, rather than from an “active” process. However, the exact mechanism of calcification remains unclear. Certain predisposing factors, such as coagulopathies, are thought to contribute to the development of these lesions. Occasionally, the calcified hematoma may tightly adhere to the dura mater and cerebral cortex, making dissection difficult and potentially causing brain contusion or bleeding. This has led to the view that removal may not always be necessary or beneficial. However, in some cases, a calcified chronic subdural hematoma can behave like an active lesion that grows similarly to a neoplasm, and there is a risk of hem-

orrhage due to vascular proliferation observed in the capsule of the calcified hematoma. Consequently, surgical intervention is generally recommended for progressively enlarging lesions [4,5].

Although the pathogenesis of calcification in chronic subdural hematoma is not fully understood, local, metabolic, and vascular factors are believed to play a major role in calcification and ossification. Calcification typically occurs between 3 to 12 months after the onset of the hematoma. Ossification, which is a terminal phase of the process, is less common than calcification. The extent of calcification in calcified chronic subdural hematoma can vary widely, involving the convexity and sometimes the entire hemispheric surface. The process may progress gradually from hyalinization to calcification and, eventually, ossification due to tissue irritation. After hemorrhage, calcification usually takes anywhere from 6 months to several years to develop. The proposed mechanism includes poor circulation and absorption in the subdural space, intravascular thrombosis, prolonged hematoma existence, stagnant blood due to insufficient arterial supply, inadequate venous return, thick connective tissue

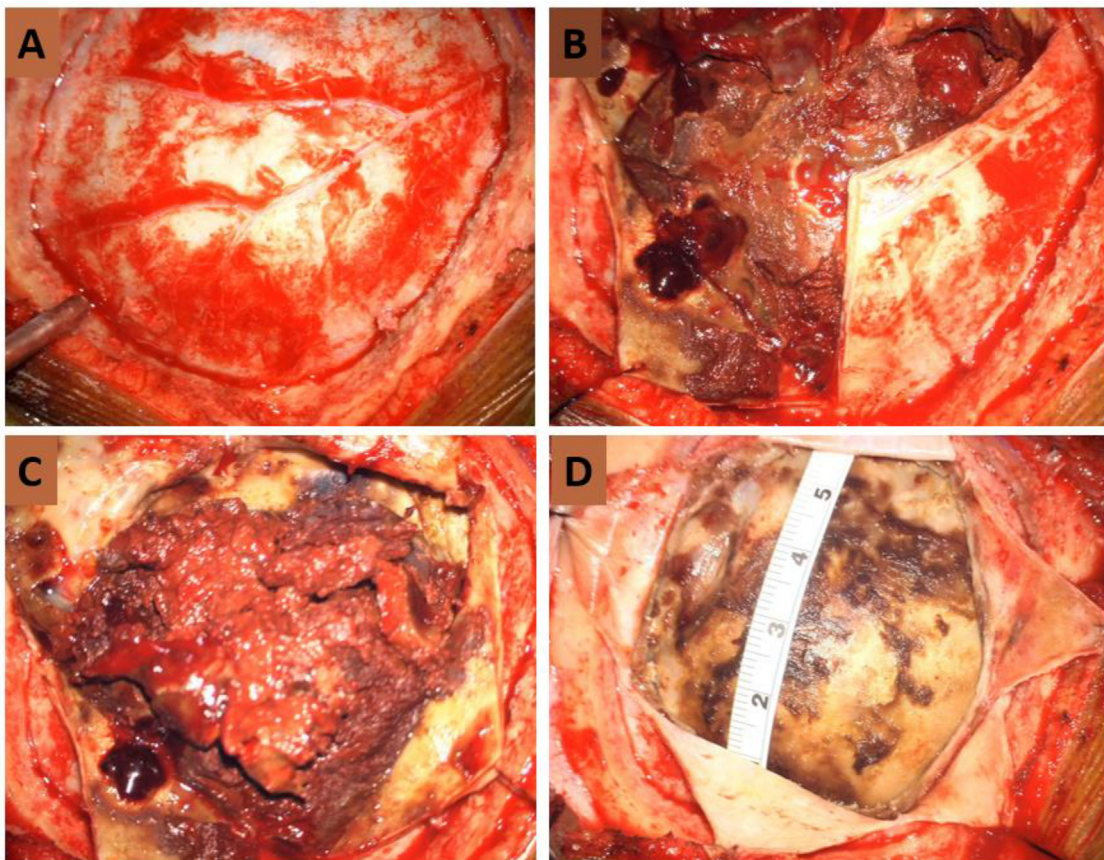


Fig. 3 – Intraoperative images during the evacuation of a chronic calcified subdural hematoma. Figure A shows the dural exposure after bone removal. Figure B depicts the dural opening, revealing both acute and chronic blood. Figure C illustrates partial clot evacuation, with some of the thick white inner membrane exposed at the 12 o'clock position. Figure D shows complete evacuation, with the inner membrane elevated to the level of the craniotomy.

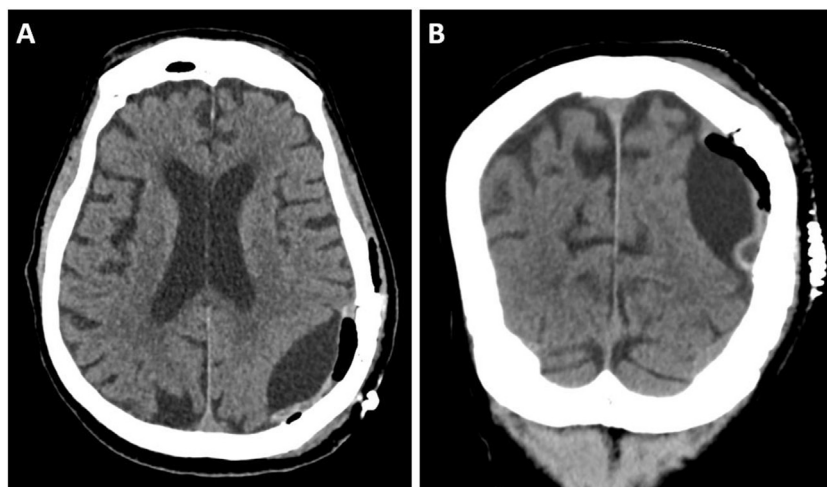


Fig. 4 – Unenhanced CT of the head, showing axial (A) and coronal (B) images, demonstrates immediate postoperative changes following left parietal craniotomy and chronic subdural hematoma evacuation. There is expected small postoperative pneumocephalus and fluid within the resection cavity.

membranes, and other local factors that contribute to calcification [5,12,13].

Calcified chronic subdural hematoma is often associated with brain atrophy, which may reduce its mass effect. Additionally, a calcified hematoma may tightly adhere to the dura and cortex, making dissection riskier and potentially leading to brain contusion or bleeding [5,14–16].

Most calcified chronic subdural hematoma can be diagnosed through imaging. They are typically elliptical in shape with the longest diameter in the anteroposterior direction, and they have a biconvex or flat shape on cross-section. The content is gelatinous or clay-like but not liquefied, and the inner membrane is thick with sinusoidal blood vessels. This membrane is often partially adhered to and evaginated into the cerebral cortex. The differential diagnosis of calcified chronic subdural hematoma includes other calcified extra-axial lesions, such as calcified epidural hematomas, calcified meningiomas, calcified arachnoid cysts, and metastases. Among these conditions, calcified chronic subdural hematoma is most commonly confused with a calcified meningioma. In our case, the patient exhibited upliftment of the dura along its anterior and posterior aspects, with minimal heterogeneous enhancement, which was more suggestive of meningioma than chronic calcified subdural hematoma [4,5,8,17–19].

Here, we describe a rare case of calcified chronic subdural hematoma and briefly review the existing literature.

Conclusion

In conclusion, calcified chronic subdural hematoma, often resulting from minor trauma, presents diagnostic challenges due to its potential to mimic other lesions, such as calcified meningiomas. The calcification process, which typically occurs 6 to 12 months after the hematoma forms, is influenced by various local and vascular factors. While some cases remain stable, others may grow and require surgical intervention to prevent complications. Careful imaging and assessment are essential in differentiating this condition and determining the need for treatment based on lesion progression.

Patient consent

Informed consent was obtained by MK granting permission to publish this case report. A copy of the informed consent can be provided to the Editor-in-Chief of the journal upon request.

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