

# **Acute Epidural-Like Appearance of an Encapsulated Solid Non-organized Chronic Subdural Hematoma**

## **—Case Report—**

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### **Abstract**

**We report the exceptional case of an encapsulated solid non-organized chronic subdural hematoma (SDH) in a 67-year-old woman that was admitted with acute hemiplegia followed by rapid deterioration in consciousness 5 months after a minor head trauma. Computed tomography (CT) showed an extracerebral biconvex shaped hyperdense mass that led to the misdiagnosis of an acute epidural hematoma. Urgent craniotomy revealed an encapsulated mass filled with solid fresh clot in the subdural space. Complete evacuation of this SDH, including both its inner and outer membranes, was achieved, and the patient recovered successfully. Histological analysis confirmed that the content of the hematoma corresponded to a newly formed clot that was enclosed between an inner membrane, composed of two collagen layers, and an outer membrane with a three layered structure. Chronic SDH may seldom present as an encapsulated solid non-organized lesion that consists of a fibrous capsule enclosing a fresh clot and lacking the thick fibrous septations that typically connect the inner and outer membranes of organized chronic SDH. This entity mimics the clinical course and radiological appearance of acute epidural hematomas and should be considered in the differential diagnosis of extracerebral hyperdense biconvex shaped lesions.**

Key words: acute epidural hematoma, biconvex hematoma, chronic subdural hematoma, encapsulated hematoma, organized subdural hematoma

### **Introduction**

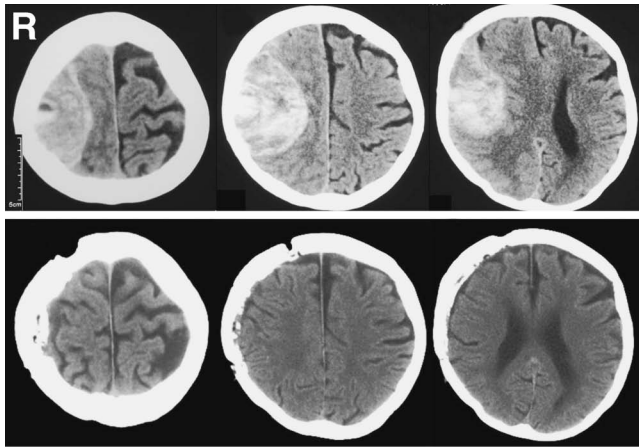
Chronic subdural hematoma (SDH) is a common type of intracranial hemorrhage characterized by an insidious onset that usually involves elderly people with a history of minor head trauma.<sup>12,20,31</sup> Chronic SDH consists of a fibrous capsule filled, in the majority of cases, with bloody fluid.<sup>7</sup> The fibrous capsule is composed of an outer membrane under the dura mater and an inner membrane over the arachnoid. Generally, these typical or liquid chronic SDHs are successfully evacuated through burr holes. Nevertheless, about 0.5–2% of them develop thick membranes and/or multiple septations turning into encapsulated masses of predominantly solid consistency that require a craniotomy to be successfully removed.<sup>1,9,12,13,17,21,23,25</sup> This latter uncommon SDH subtype is known as organized chronic SDH. A crescent shape and a density usually similar or lower to that of the adjacent cerebral parenchyma are the usual computed tomography (CT) features of both liquid and organized chronic SDHs.<sup>6,19</sup> In contrast, acute epidural hematomas are biconvex or lentiform shaped lesions with high-density content on CT without

contrast medium.<sup>5</sup>

We report on the rare case of an extracerebral hyperdense hematoma with a biconvex shape on CT that was diagnosed in an aged person who had suffered a rapid neurological deterioration and had no history of a recent head trauma. The lesion was thought to correspond to an acute epidural hematoma but an encapsulated solid non-organized chronic SDH was actually found during the surgical exploration of the mass. The diagnosis of an encapsulated and solid non-organized chronic SDH that mimics the radiological appearance of an acute epidural hematoma is exceptional with only one similar case previously reported. Here we provide the neuroradiological and surgical findings of this atypical epidural-like chronic SDH as well as a detailed histological analysis of the lesion.

### **Case Report**

A 67-year-old woman presented at the emergency room with acute onset of left hemiplegia and slurred speech. The patient had no indication of recent head trauma. On admission, she was confused and disoriented to time and space, opened her eyes to voice stimulation, and obeyed simple orders. The patient scored 13 points on the Glas-

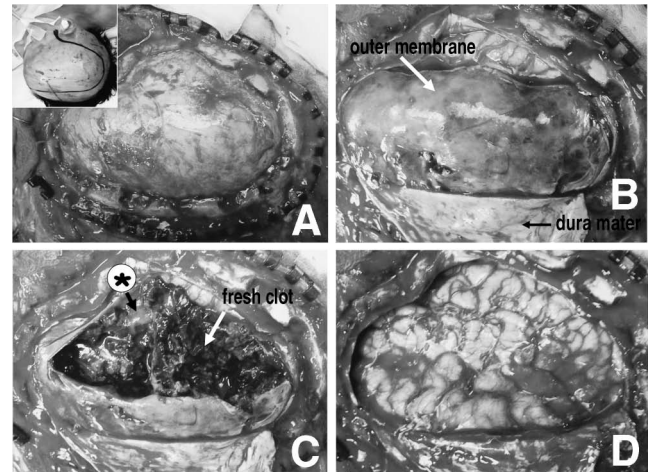


**Fig. 1** Preoperative axial computed tomography scans at different levels showing a right extracerebral biconvex-shaped subdural hematoma, hyperdense relative to the adjacent brain parenchyma that mimics the appearance of an acute epidural hematoma (upper row), and 3 months after the surgery showing a laminar hyperdense area under the craniotomy flap suggestive of calcification of the dura mater (lower row).

gow Coma Scale (GCS; eye: 3, verbal: 4, motor: 6). Neurological examination revealed left hemiplegia and facial weakness. The patient was receiving anticoagulation medication (acenocoumarol) because of a history of paroxysmal atrial fibrillation. She had also a history of arterial hypertension, chronic renal insufficiency, and protein-caloric malnutrition. Relatives of the patient indicated that she had suffered a minor head trauma 5 months before the admission, caused by an accidental fall in which she fractured her hip. CT performed at that time was unremarkable and the patient was operated on for the hip fracture.

Blood tests performed at the emergency room showed that her international normalized ratio was 1.6 (normal values: 0.8–1.2) and her creatinine level was 2.8 mg/dl (normal values: 0.6–1 mg/dl). Emergent head CT revealed an extracerebral and mostly hyperdense right frontoparietal mass with a biconvex morphology (Fig. 1 upper row). These signs led us to the wrong diagnosis of an acute epidural hematoma. The low density areas observed within the hematoma were considered a swirl phenomenon caused by unclotted blood secondary to active bleeding, a phenomenon typically observed in acute epidural hematomas.<sup>38)</sup>

An urgent large right fronto-temporo-parietal craniotomy was performed on the day of admission. Contrary to our expectations no epidural hematoma was found (Fig. 2). Once the dura mater was opened, an encapsulated solid lesion was observed. The dura mater was easily dissected from the encapsulated lesion that had a glistening blue-yellowish appearance. After opening the fibrous membrane under the dura mater (outer membrane), a fresh solid dark-red clot containing scattered xanthochromic fluid spots was disclosed. The clot was totally evacuated allowing inspection of a thin transparent fibrous membrane over the brain surface (inner membrane). Then, this inner mem-

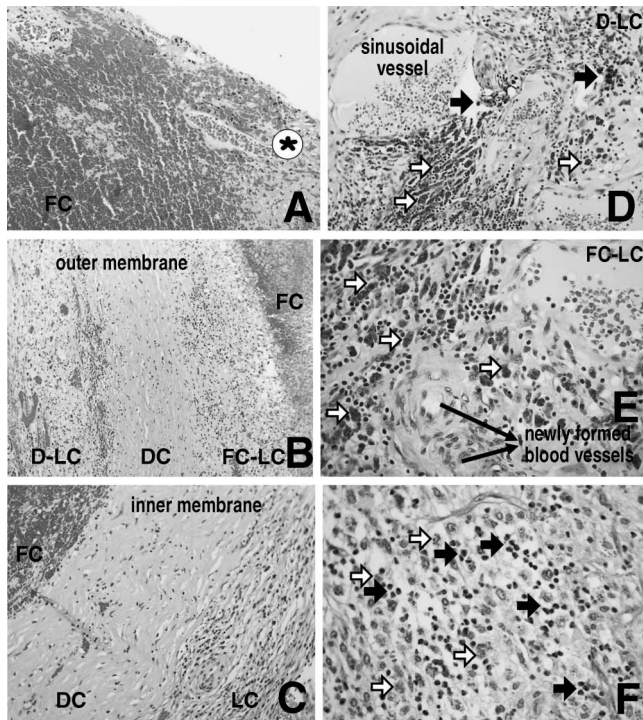


**Fig. 2** Intraoperative photographs showing the skin incision for a large fronto-temporo-parietal craniotomy (A, inset), removal of the frontoparietotemporal craniotomy flap and exposure of the dura mater with absence of epidural lesions (A), opening of the dura mater and reflection toward the sagittal sinus disclosing a subdural encapsulated lesion (B), opening of the fibrous capsule exposing a fresh dark-red clot with some yellowish spots (asterisk) (C), and after removal of the capsule demonstrating absence of vascular anomalies on the brain surface under the hematoma (D).

brane was separated from the underlying arachnoid layer using water jet dissection. Complete removal of the hematoma and its surrounding membranes demonstrated the absence of vascular anomalies on the brain surface.

Surgical specimens, that included separately fresh clots and fragments of the outer and inner membranes, were fixed in 10% formalin, embedded in paraffin, stained with hematoxylin and eosin, and prepared for routine light microscopy. Histological examination evidenced that the hematoma consisted of a fresh or newly formed clot enclosed by two thin membranes (Fig. 3). The inner membrane of the hematoma was thinner than the outer and was composed of two layers, a dense collagenous layer right over the hematoma and a loose collagenous layer next to the arachnoid. In the sections corresponding to the outer membrane of the hematoma, three layers could be identified, with a middle dense collagenous layer bounded by two loose collagenous layers (one just behind the dura mater and the other over the hematoma). The loose collagenous layer on the side of the dura mater showed abundant sinusoidal thin-walled blood vessels with a large lumen. In the vicinity of blood vessels within the loose collagenous tissue of both the inner and outer membranes, abundant eosinophils, macrophages, and deposits of iron pigment were observed. In addition, scattered areas of recent hemorrhage were observed within the outer membrane.

Immediately after the surgery, the GCS score of the patient improved to 15 and neurological examination showed mild left-side weakness. The patient completed rehabilitation therapy and eventually recovered from left-



**Fig. 3** Representative photomicrographs of the operative specimens. **A:** Fresh or newly formed clot (FC) and fibrin meshwork (asterisk). Hematoxylin and eosin (HE) stain, original magnification  $\times 20$ . **B:** The outer membrane has a three-layer structure: a middle dense collagenous layer (DC) bounded by two loose collagenous layers (LCs), the most superficial beneath the dura mater (D-LC) and the deepest adjacent to the FC (FC-LC). HE stain, original magnification  $\times 10$ . **C:** The inner membrane had a two-layer structure, with a DC adjacent to the FC and LC over the arachnoid. HE stain, original magnification  $\times 20$ . **D:** The D-LC contains thin-wall large sinusoidal vessels, and was infiltrated by hemosiderin-laden macrophages (white arrows) and eosinophils (black arrows). HE stain, original magnification  $\times 40$ . **E:** The FC-LC enclosed newly formed blood vessels and hemosiderin-laden macrophages (white arrows). HE stain, original magnification  $\times 40$ . **F:** The LC within the outer membrane contained abundant infiltration of eosinophils (black arrows) and hemosiderin-laden macrophages (white arrows). HE stain, original magnification  $\times 40$ .

side weakness. Three months after the surgery, the patient was healthy and had no neurological deficits. Follow-up CT showed expansion of the right cerebral hemisphere (Fig. 1 lower row).

## Discussion

The present case of atypical chronic SDH occurred in an aged patient with sudden and rapid neurological derangement, and appeared as an extracerebral hyperdense biconvex shaped lesion on CT. These features led us to the radiological misdiagnosis of an acute epidural hematoma. However, a fibrous capsule filled with a fresh-solid clot between the dura mater and arachnoid space was the unexpected lesion found at the surgical intervention. This case

was also unusual for the absence of recent head trauma or significant coagulopathy in a patient presenting with such important acute bleeding. Given the striking surgical finding, we thought that histological analysis of both the capsule and the content of this hematoma could provide some insight into the pathological mechanisms involved in its development.

Most chronic SDHs consist of a fibrous capsule enclosing bloody fluid. After undergoing encapsulation, chronic SDHs usually experience gradual enlargement through repetitive episodes of bleeding from the outer membrane.<sup>16,28</sup> Thickness and composition of the neomembranes varies greatly from case to case depending on the age of the hematoma, although complete formation of the fibrous capsule usually requires 3 to 4 weeks.<sup>7,30</sup> A majority of chronic SDHs are liquid when diagnosed and can be treated by evacuation-aspiration through burr holes. However, a few cases develop a solid structure due to the penetration of fibroblasts that replace the fibrin meshwork of the hematoma by collagen fibrils. Whole organization of a chronic SDH occurs when the outer and inner membranes fuse completely, a process that takes a period of 6 to 12 months. These solid or organized chronic SDHs usually require a craniotomy to be adequately evacuated.<sup>17,25</sup>

The two major structural features of the present hematoma were the presence of mature thin fibrous outer and inner neomembranes, usually observed in encapsulated liquid chronic SDH, in addition to the solid fresh-clot content. Histological features of the membranes were similar to those of typical chronic SDHs<sup>27,30,34</sup> (Fig. 3). This hematoma lacked the thick membranes or fibrous septations present in organized chronic SDHs. Consequently, the term encapsulated solid non-organized chronic SDH was chosen to define this particular lesion. Histological analysis of the surgical specimen suggested that acute massive bleeding had occurred within an unnoticed pre-existing encapsulated chronic SDH. The outer membrane had a three-layered structure, with a middle layer of compact or dense collagenous tissue bounded by two layers of loose collagenous tissue. This arrangement corresponded to the type III membrane described in the histological classification of the structure of the outer membrane of chronic SDHs according to the time of maturation since the traumatic event.<sup>22</sup> Hence, the period of maturation of the fibrous capsule in this case was probably several months. Besides, rich infiltration by hemosiderin-laden macrophages and eosinophils was observed in the loose collagenous tissue of both the inner and outer membranes. Eosinophilic infiltration occurs in up to 80% of the neomembranes of chronic SDHs with a duration of more than 3 months,<sup>10,26,34,35</sup> but is almost absent in both fresh hemorrhages and completely organized hematomas.<sup>10</sup> Presence of hemosiderin-laden macrophages within the neomembranes supported previous and repeated bleeding episodes.<sup>4</sup> Therefore, the three-layered structure of the outer membrane and the abundant infiltration of hemosiderin-laden macrophages and eosinophils within the fibrous capsule are the fundamental findings supporting the long evolution of the chronic SDH in the present

case.

Fresh clotted blood secondary to recent bleeding within a chronic SDH usually undergoes a rapid liquefaction. In contrast, the present case harbored a recently formed clot which remained solid and filled the cavity of the fibrous capsule. Only one case of a chronic SDH with a septated compartment containing an organized solid clot has been reported.<sup>37)</sup> Biochemical conditions that determined the absence of clot liquefaction in both the previous and our own cases are unknown. Bloody fluid of chronic SDHs usually remains liquid because its chemical composition prevents the function of hemostatic mechanisms. Failure of coagulation is due to the consumption of clotting factors within the chronic SDH, secondary to over-activation of both the intrinsic and extrinsic clotting pathways.<sup>15,33,34)</sup> Moreover, the hematoma fluid has an anticoagulant effect when added to normal blood due to its high content of fibrinogen degradation products that inhibit the fibrin polymerization.<sup>18,32,34)</sup> No measures of coagulation proteins and inhibitors were performed in the hematoma contents of our case, but we believe that an imbalance of the normal relationships between coagulant/anticoagulant factors could explain the absence of liquefaction of the subdural clot within the chronic SDH.

Regardless of the consistency of the hematoma, whether solid or liquid, both acute and chronic SDHs that expanded at the virtual subdural space generally show a crescent shape on CT. Only a few cases of biconvex-shaped subdural blood collections mimicking the morphology of acute epidural hematomas have been reported, and most of them corresponded to acute SDHs diagnosed in patients with hemorrhagic diathesis.<sup>3,11,14,24)</sup> Only one previous case of a chronic SDH displaying the same epidural-like biconvex shape has been reported, in which acute bleeding had presumably superimposed on earlier chronic encapsulated content.<sup>24)</sup> The epidural-like appearance of our chronic SDH was likely related to the acute bleeding into the cavity of a previously encapsulated chronic SDH. Expansion of the clot against the wall of the capsule offering the least resistance, which is the inner membrane, may have caused stretching and ballooning of this wall, and so resulted in the biconvex epidural-like appearance of the hematoma.<sup>3,29)</sup>

This solid non-organized chronic SDH appeared mostly as hyperdense indicative of a recent bleeding. Definite causes of the acute massive bleeding in this case remain elusive, but the sinusoidal fragile vessels identified within the loose collagenous layer of the outer membrane may represent the original source (Fig. 3D). Blood vessels of this type are a constant finding within the neomembranes of mature chronic SDHs and are considered to be the main source of repetitive bleedings into these hematomas.<sup>8,16,36)</sup> The elevated amount of eosinophils in the neomembranes of this hematoma could also have been involved, because degranulation of these cells releases angiogenic and fibrinolytic factors involved in both the formation of fragile sinusoidal vessels and their tendency to spontaneous bleeding.<sup>2)</sup> Finally, the patient's history of hypertension could have favored the bleeding tendency of these fragile vessels.

In conclusion, an encapsulated solid non-organized chronic SDH consists of a fibrous capsule enclosing a fresh clot and lacking the thick membranes and fibrous septations that typically connect the inner and outer membranes of organized chronic SDHs. Such hematomas may appear as a hyperdense biconvex-shaped mass on CT without contrast medium, mimicking the morphology of an acute epidural hematoma. These hematomas may cause abrupt and severe neurological derangement as result of acute bleeding within the previously developed chronic SDH. History of hypertension and coagulopathy may be factors contributing to such massive bleeding within a chronic SDH in the absence of a recent head trauma.

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