

Encapsulated Acute Subdural Hematoma Mimicking Acute Epidural Hematoma on Computed Tomography

—Case Report—

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Abstract

An 87-year-old woman presented with an atypical case of acute subdural hematoma (ASDH) manifesting as disturbance of consciousness and left hemiparesis. Computed tomography revealed a high density lentiform lesion in the right convexity, which was thought to be acute epidural hematoma preoperatively. Emergent decompressive craniotomy revealed an encapsulated solid fresh clot in the subdural space and a bleeding small cortical artery under the clot. The arachnoid membrane and the brain parenchyma were intact, and no other abnormal feature such as aneurysm or arteriovenous malformation was observed. The encapsulated ASDH was removed en bloc and the patient fully recovered. Histological examination confirmed that both the outer thicker and the inner membrane were fibrinous single structures without vasculature. The red blood cells constituting the clot in the capsule maintained their cell structure. The reported pathological mechanisms of lentiform ASDH are adhesion of the arachnoid membrane and the dura mater or intracapsular bleeding from sinusoidal vessels in the outer membrane of the chronic subdural hematoma. However, in our case, the arachnoid membrane had not adhered to the dura mater and the capsule was a fibrinous single structural membrane without vasculature, which probably resulted from a previous hematoma due to initial bleeding from the cortical artery. The possible mechanism in our case was that the re-bleeding dissected and flowed into the fibrinous single structural membrane, resulting in formation of the lentiform ASDH.

Key words: acute subdural hematoma, lentiform, biconvex, acute epidural hematoma, encapsulated hematoma

Introduction

Acute epidural hematoma (AEDH) generally appears on computed tomography (CT) as a high density lentiform lesion, in contrast acute subdural hematoma (ASDH) generally appears on CT as a high density crescent-shaped lesion.⁴⁾ However, heterogeneous hematoma indicates ASDH rather than AEDH⁶⁾ although AEDH also occasionally appears as heterogeneous on CT.¹⁰⁾ ASDH appearing as a high density lentiform lesion on CT is rare, and little is known about the pathological mechanism of lentiform ASDH.¹⁵⁾ We describe a case of intracranial lentiform hematoma, which was thought to be AEDH preoperatively but was discovered to be encapsulated ASDH intraoperatively.

Case Report

An 87-year-old woman who had followed her usual daily routine the previous day was found lying on the floor at home and was brought to our emergency room with disturbance of consciousness. Physical examination found no traumatic findings such as abrasions or bruises. Her score on the Glasgow Coma Scale (GCS) was 13 points: scores for eye, verbal, and motor functions were 3, 4, and 6 points, respectively, and left Barre's arm sign was positive. She had been independent in daily life, although she had medical histories of mild dementia as well as hypertension, hyperlipidemia, and bronchial asthma. She had received neither antiplatelet nor anticoagulant medication. No abnormal value was found in blood tests including platelet count and coagulation function. Head CT revealed an intracranial high density lentiform lesion

Received July 28, 2011; Accepted November 15, 2011

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Fig. 1 Computed tomography scan on admission showing a massive intracranial high density lentiform lesion including low density areas (arrows) with small amounts of acute subdural hematoma (arrowhead).



Fig. 2 Photograph of the removed hematoma showing the encapsulated clot (arrow) with the extracapsular clot (arrowhead).

(average Hounsfield number: 77.2) in the right convexity with midline shift (Fig. 1). The lesion included some low-density areas, but the lentiform shape indicated a diagnosis of AEDH.

Emergent decompressive craniotomy discovered a large encapsulated ASDH instead of AEDH. The encapsulated ASDH was covered with thin fresh hematoma and was easily removed en bloc because the capsule had not adhered to the surrounding tissue. The capsule was whitish, smooth, and firm, and filled with solid fresh clot (Fig. 2). The outer membrane of the capsule (dural side) was thicker than the inner membrane (arachnoidal side). The arachnoid membrane and the brain parenchyma were intact, but a small cortical artery near the sylvian fissure was found to be bleeding, which was probably responsible for the ASDH. No other abnormal feature such as aneurysm or arteriovenous malformation was observed. Postoperative CT showed that the ASDH was completely removed and no delayed lesion such as brain contusion was appar-

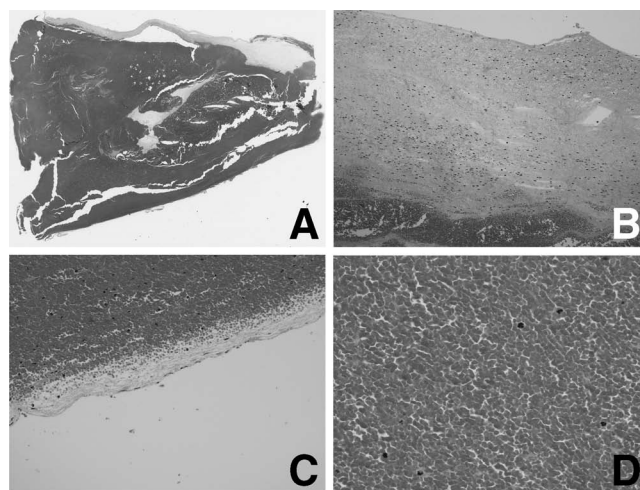


Fig. 3 Photomicrographs of the resected specimen of the encapsulated hematoma (A: hematoxylin and eosin stain, loupe) showing a fibrinous single structural membrane that was thicker on the dural side (B: hematoxylin and eosin stain, original magnification $\times 100$) than the arachnoidal side (C: hematoxylin and eosin stain, original magnification $\times 100$). The red blood cells constituting the clot in the capsule maintained their cell structures (D: hematoxylin and eosin stain, original magnification $\times 400$).

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Histological examination of the surgical specimens showed the outer membrane was thicker than the inner membrane, and both were fibrinous single structures without vasculature, which indicated old hematoma (Fig. 3). The hematoma inside the capsule consisted of only freshly clotted red blood cells that maintained their cell structure.

Her GCS score improved to 15 points soon after surgery, and her postoperative course was uneventful. She left our hospital without neurological deficit 2 weeks after surgery.

Discussion

Analysis of 64 cases of AEDH found that 94% of these cases were caused by external force, with 75% ($n = 48$) of the cases involving skull fracture, 44% ($n = 28$) involving brain contusion, and a few cases involving subdural hematoma, subarachnoid hemorrhage, and intraventricular hemorrhage.¹⁰ In our case, physical examination found no evidence of trauma indicating external force, radiography showed no skull fracture, and CT demonstrated lentiform hematoma including low density areas associated with a small but apparently ASDH (Fig. 1). These findings are atypical of AEDH, but the lentiform appearance of the hematoma led us to a preoperative diagnosis of AEDH.

Since she was at home alone and found lying on the floor by her family, the details of the situation at the onset are not known. Although traumatic ASDH is mostly associated with brain contusion and/or intracerebral hemorrhage, neither of these signs was recognized on pre- and

postoperative CT or intraoperatively in our case.¹⁹⁾ In addition, no traumatic findings were observed at the physical examination. Thus, the ASDH of our case was considered to be spontaneous rather than traumatic. Several criteria have been proposed for acute spontaneous subdural hematoma (ASSDH) of arterial origin: No history of head trauma; no damage to the underlying cortex; no aneurysm or arteriovenous malformation around the affected artery; and identification of an arterial origin of the hemorrhage at operation.²⁰⁾ The bleeding source has been identified as laceration of the arterial wall at the bifurcation or adhesion to the dura mater of small cortical arteries, especially within 3 cm of the sylvian fissure. Major re-bleeding can occur 5 hours to 2 weeks after minor initial bleeding.²⁰⁾ Advanced age, hypertension, coagulopathy, and alcohol abuse are the reported risk factors.^{2,3,6,9,11,12,14,20,21)} In the present case of an elderly woman with hypertension, the encapsulated ASDH, considered to have the same bleeding source from the cortical artery as the initial bleeding, satisfied the criteria of ASSDH of arterial origin.

Although six reports of lentiform ASDH have been published to date, only one was a case of encapsulated ASDH, in which fragile sinusoidal vessels identified in the outer membrane of a chronic subdural hematoma (CSDH) were considered to be the source of bleeding.¹⁵⁾ The other cases of lentiform ASDHs without capsules resulted from adhesion of the arachnoid membrane and dura mater; some thought to be due to previous neurosurgical interventions or radiotherapy.^{1,7,8,16,18)}

Coronal magnetic resonance imaging might be useful for discriminating lentiform ASDH from AEDH because the relationships between the dura mater, hematoma, and subarachnoid space can be clarified.⁹⁾ Of course, emergent decompressive craniotomy takes precedence over the differential diagnosis in a patient with massive hematoma. In our case, the capsule was not the membrane of CSDH because of the finding of a fibrinous single structural membrane without vasculature, probably resulting from a previous hematoma due to the initial bleeding.^{5,13,17,22)} The possible mechanism in our case was that the re-bleeding dissected and flowed into the fibrinous single structural membrane, resulting in formation of the massive encapsulated ASDH, although some of the blood flowed outside the capsule.

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