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Case Report

Microvascular proliferation in the clots: The key finding of acute subdural hematoma transforming into chronic subdural hematoma?

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ABSTRACT

Background: Despite extensive investigations, the exact etiology of chronic subdural hematoma (CSDH) remains elusive. Organized CSDHs are a distinct but less-understood type of CSDH.

Case Description: A 50-year-old hypertensive woman experienced headache without any previous head injury. At presentation, the patient showed no focal neurological deficits. Cranial computed tomography (CT) revealed a slightly compressive subdural hematoma that spontaneously regressed and no intracranial vascular lesions. Cerebral magnetic resonance imaging identified a non-enhancing nodular lesion in the subdural hematoma. After the patient presented disorientation and aphasia on post hospitalization day 14, CT showed a considerable enlargement of the subdural hematoma. Partial removal of the bi-layered hematoma was performed through a parietal craniotomy. Histological examination revealed microvascular proliferation in both the outer membrane and the nodular lesion. On postoperative day 35, CT demonstrated a remarkable resolution of the residual hematoma.

Conclusion: Development of microvascular proliferation in the clots of an acute subdural hematoma may lead to its rapid enlargement as an organized CSDH. Organized CSDH can be managed by partial removal of the outer membrane and hematoma through a craniotomy.

Keywords: Acute subdural hematoma, Chronic subdural hematoma, Craniotomy, Microvascular proliferation, Subdural clots

INTRODUCTION

Chronic subdural hematoma (CSDH) is one of the most common neurosurgical disorders. Typically, it is managed by burr hole drainage or an evacuation through a craniotomy. Despite extensive investigations, however, its exact etiology and optimal treatment strategy remain elusive.[1,7-9,11,13,14] A fraction of CSDHs has been reported to develop from acute subdural hematomas (ASDHs), which become symptomatic more rapidly than conventional CSDHs *de novo*.[5] Organized CSDHs are a distinct type of CSDHs that are not well-understood. Recently, less-invasive endoscopic techniques and hematoma removals through small craniotomies have been advocated as alternative approaches for such CSDHs.[1,2,12]

In contrast with trauma-associated ASDH, spontaneous or non-traumatic ASDH is thought to occur infrequently in association with a variety of pathological processes.[3,4,6] Here, we present

a unique case of organized CSDH that transformed from a non-traumatic ASDH and became symptomatic for 2 weeks, exhibiting microvascular proliferation in the subdural clots.

CASE PRESENTATION

A 50-year-old hypertensive woman presented to the emergency department of our hospital presenting headache while shopping. There was no history of preceding falls or head injuries. She had not been administered steroids, anticoagulants, or antiplatelet agents. At presentation, the patient was fully awake and did not exhibit any focal neurological deficits. The blood pressure was 176/113 mmHg, and blood examination revealed normal findings. Cranial computed tomography (CT) revealed a subtly compressive, apparently ASDH in the left cerebral convexity. It was 15 mm in thickness without displacement of midline structures and showed a regression in 1 day with resolution of the headache [Figure 1]. Three-dimensional CT angiography showed no vascular lesions in the intracranial dural sinuses or major cortical veins [Figure 2]. Cerebral magnetic resonance imaging (MRI) performed on post hospitalization day (PHD) 6 revealed a non-enhancing, nodular lesion in the subdural hematoma, and adjacent to the left parietal cortex [Figure 3]. The patient was conservatively managed based on a probable diagnosis of non-traumatic ASDH. However, the patient presented with disorientation and aphasia on PHD 14; CT showed a considerable enlargement of the subdural hematoma with better delineation of the nodular lesion on T2-weighted sequence [Figure 4]. Catheter angiography performed on PHD 14 did not reveal any intracranial vascular lesions. The patient underwent removal of the microsurgical hematoma including the nodular lesion through a 5 × 5-cm parietal craniotomy. The subdural hematoma showed a bi-layered structure comprising a thick outer membrane and inner semisolid clots. The cerebral cortex underneath the hematoma was intact. The nodular lesion identified previously on MRI, possessed a fibrous capsule, included clots, and adhered to the outer membrane of the hematoma and arachnoids. These attachments were bluntly dissected without injuring the cortical vessels coursing underneath [Figure 5]. Abnormal vasculature was not found between the lesion and surrounding tissues. The outer membrane of the hematoma and the semisolid subdural clots were partially removed. Microscopically, the resected outer membrane of the CSDH and the nodular lesion revealed areas of microvascular proliferation [Figure 6]. The patient's postoperative recovery period was uneventful. On postoperative day 35, CT showed a remarkable resolution of the residual hematoma [Figure 7].

DISCUSSION

In the present case, the presumed non-traumatic ASDH initially showed spontaneous regression, but then transformed into an organized CSDH and markedly enlarged

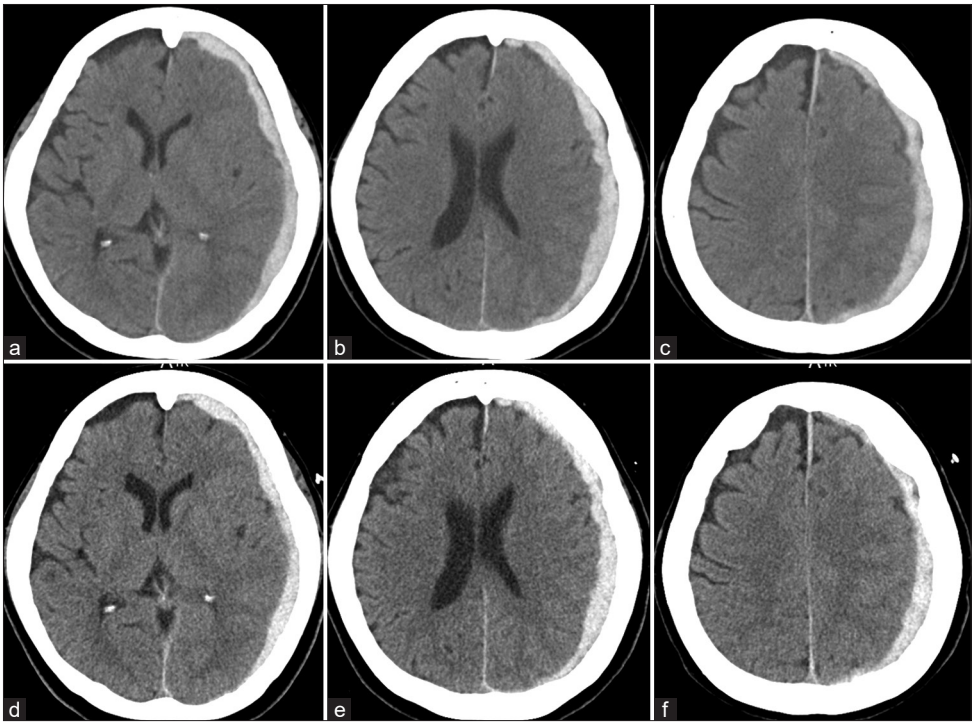

Figure 1: Axial computed tomography scans performed on the day of presentation (a-c) and the next day (d-f) showing a subtly compressive, the left subdural hematoma apparently in acute phase that underwent slight regression in 1 day.

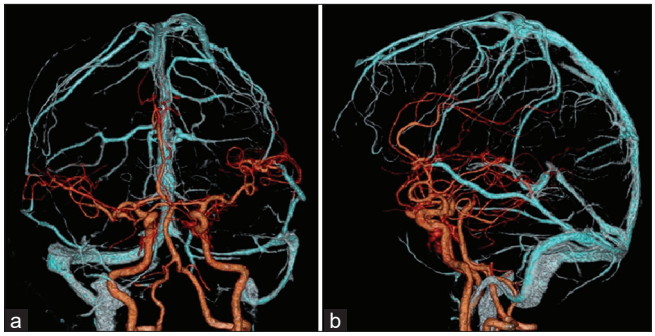

Figure 2: Three-dimensional computed tomography angiography, anteroposterior (a) and the left lateral view (b), showing intact intracranial dural sinuses and the major cortical veins distributed in the left cerebral hemisphere.

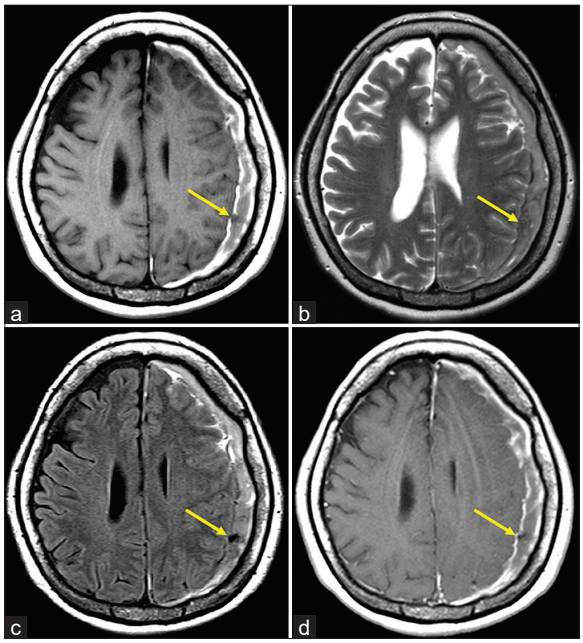

Figure 3: Non-contrast axial T1- (a), T2-weighted- (b), fluid attenuated inversion recovery-(c), and post-contrast axial T1-weighted magnetic resonance imaging (d), performed on post hospitalization day 6, showing a non-enhancing, nodular lesion in the subdural hematoma, adjacent to the left parietal cortex (a-d, arrow).

in the following 2 weeks. Despite careful observation, the exact cause of the ASDH and its original site were not identified in this case, although the existing condition of hypertension might have acted as a risk factor for the development of the hemorrhage.[3,4] Alternatively, based on intraoperative findings, Pacchionian granulation was thought to be a possible cause of the ASDH. Spontaneous resolution in a short period is known to be an infrequent but distinct phenomenon of ASDH, which is represented by dissemination and redistribution of the subdural hematoma.[10] In addition, the period for the transformation of the ASDH into the symptomatic CSDH in our case was consistent with that seen in a previous study.[5] Although not evident on neuroimages, intraoperative and histological findings showed that the nodular lesion presented microvascular proliferation, typically a characteristic finding of the outer membrane of CSDHs, indicating production of hematoma fluid.[13] Therefore, we deduced that the microvascular proliferation found in the subdural clots and that in the outer membrane might attribute to the transformation of ASDH into CSDH and its rapid enlargement thereafter. This notion should be validated in a larger sample in the future studies.

In the present case, the organized CSDH was managed by partial removal of the outer membrane and the associated subdural clots through a craniotomy. The size of the craniotomy was much smaller than the whole extension of the hematoma because we predicted that the subdural hematoma was in a fluid state and could be evacuated through the small cranial window. However, similar to a previous study, the residual hematoma resolved spontaneously following the partial removal.[2] There may be undetected factors for spontaneous regression of CSDHs after craniotomy and partial removal of the hematomas. In this case, the nodular lesion buried in the subdural hematoma was targeted for investigation, because it was a distinct structure and was expected to provide clues to the underlying etiology. Therefore, we chose a craniotomy for accurately removing the lesion, instead of burr hole drainage aiming at decompression.

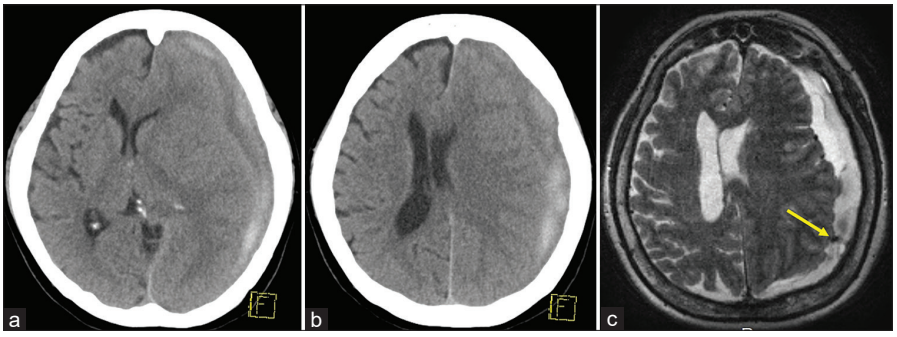

Figure 4: Axial computed tomography scans (a and b) and T2-weighted magnetic resonance imaging (c), performed on post hospitalization day 14, showing a considerable enlargement of the subdural hematoma with the nodular lesion better circumscribed from the surrounding hematoma (c, arrow).

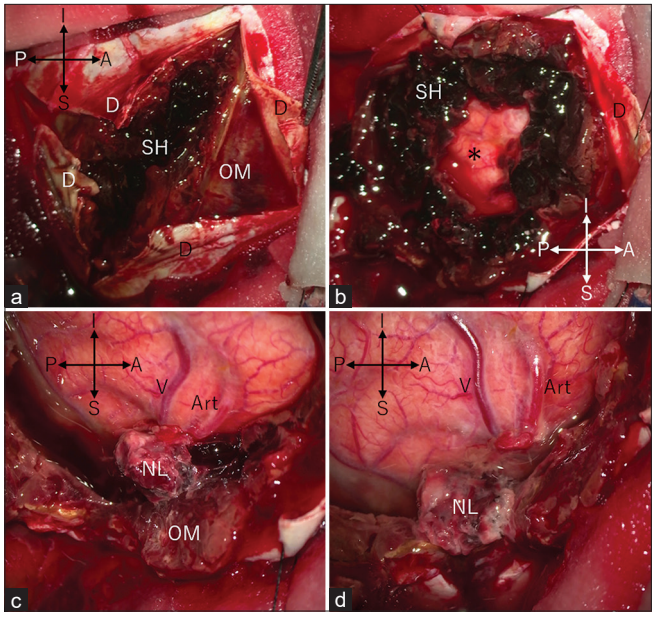

Figure 5: Intraoperative photos showing, on reflection of the dura mater, a bi-layered subdural hematoma comprising a thick outer membrane and inner semisolid clots (a), intact cortical surface exposed after hematoma evacuation (b), nodular lesion adhering to the outer membrane of the hematoma and the arachnoids (c), and nodular lesion separate from the arachnoids with intact cortical vessels coursing underneath (d). A: anterior; Art: cortical artery; D: dura mater; I: inferior; NL: nodular lesion; OM: outer membrane of hematoma; P: posterior; S: superior; SH: semisolid hematoma; V: cortical vein; and Asterisk: cortical surface.

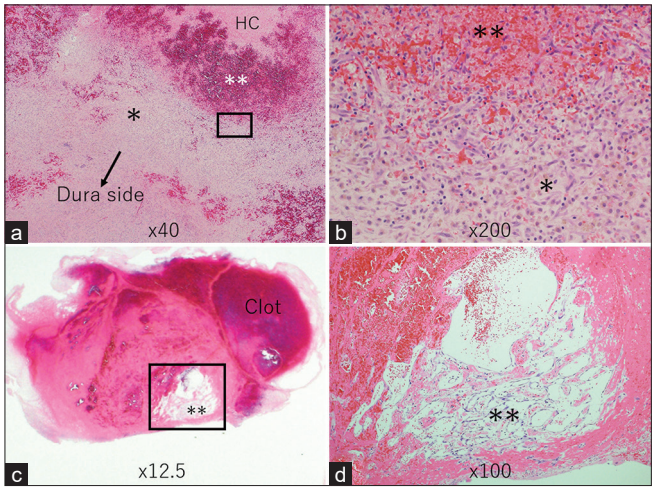

Figure 6: Photomicrographs of the resected outer membrane (a and b) and nodular lesion (c and d) showing microvascular proliferation present in the outer membrane, adjacent to the hematoma cavity, and within the nodular lesion. (b and d) represent magnified views of the square areas in (a and c), respectively. (a-d) Hematoxylin and eosin stain. HC: Hematoma cavity; *Fibrous connective tissue; **Microvascular proliferation.

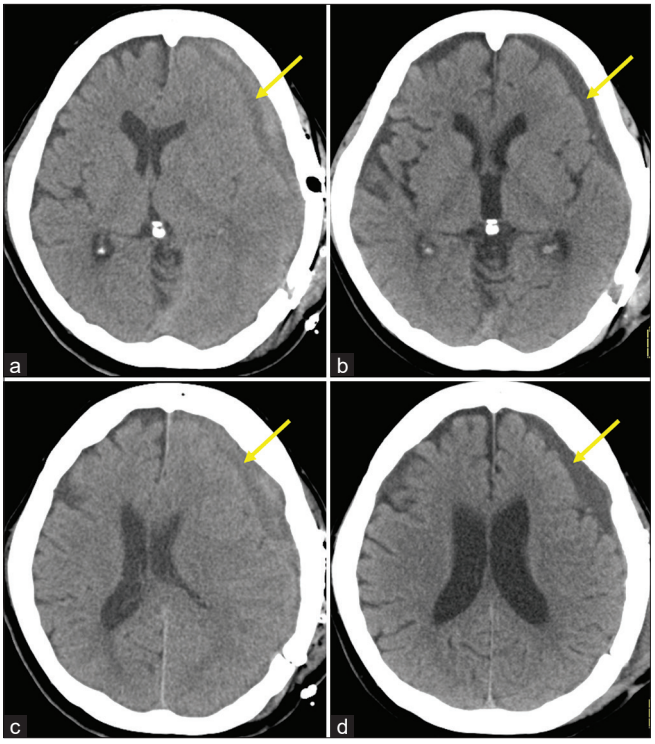

Figure 7: Axial computed tomography scans performed on postoperative day 1 (a and b) and day 35 (c and d) showing resolution of the residual subdural hematoma.

CONCLUSION

Development of microvascular proliferation in the clots of an ASDH may lead to its rapid enlargement as an organized CSDH. Organized CSDHs can be managed by partial, instead of extensive, removal of the outer membrane, and the included hematoma through a craniotomy.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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