Ossified peripheral middle cerebral artery aneurysm in a 30-year-old man

doi: 10.1016/j.jocn.2008.07.084

Case Report; Journal of Clinical Neuroscience

Entirely Ossified Peripheral Middle Cerebral Artery Aneurysm in a 30-Year-Old Man

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A running title; Ossified Aneurysm in a 30-year-old man

Key Words: Aneurysm, Middle cerebral artery, Ossification, Thrombus

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Abstract

This 30-year-old male presented with several times of convulsion. A computed tomography scan showed a calcified or ossified focus in the right temporal lobe. Cerebral angiography yielded no abnormal findings. He underwent craniotomy, in which the M3 portion of the middle cerebral artery (MCA) was found to terminate with a blind end at the junction with the calcified mass. The pathological diagnosis showed a completely ossified cerebral aneurysm. The calcification of cerebral aneurysms is often found and considered as the ultimate result of intra-aneurysmal thrombus or degenerative changes in the aneurysmal wall. On the other hand, the complete mural ossification of cerebral aneurysm is rare, and ossified peripheral MCA aneurysms are still rarer in young individuals. This entirely ossified aneurysm suggests that it developed over a prolonged period and may have arisen during childhood.

1. Introduction

Calcified cerebral aneurysms are thought to derive from intra-aneurysmal thrombus or degenerative changes in aneurysmal wall, such as fibrosis, lipid deposition, hyalinization, and vaculorization.[1,2] However, cerebral aneurysms with entirely ossified walls are rarely seen in younger individuals. We report a 30-year-old man with a completely ossified peripheral middle cerebral artery (MCA) aneurysm. Although the precise developmental mechanisms underlying this aneurysm remain unclear, it is suggested that the ossification of this aneurysm developed over a prolonged period.

2. **Case report**

This 30-year-old male presented with several times of convulsion attacks. Although he tried some anti-convulsants, none of them worked effectively. On admission, neurological examination was normal. His serum levels of calcium, phosphorus and parathyroid hormone were normal. A computed tomography (CT) scan showed a high-density lesion suggesting a calcified mass in the right temporal lobe (Fig.1A). Its signal on magnetic resonance imaging was iso-intense with a hypo-intense rim on T1-weighted images; on T2-weighted images there was a mixed-intense signal with a hypo-intense rim (Fig.1B,C). Angiography revealed no abnormal findings. 3 dimensional-CT Angiography showed the peripheral branch of MCA connected to the mass (Fig.1D).

Based on these findings, we made a provisional preoperative diagnosis of thrombosed and calcified aneurysm and postulated that his convulsion attacks were attributable to it. Besides that, calcified cavernous angioma, sylvian fissure meningioma, parasite and tuberculoma were taken into differential diagnosis. We made an informed consent to him and his family. Consequently, they hope

removal of the mass and further detailed examination.

Upon right frontotemporal craniotomy, neither subarachnoid hemorrhage nor old hematoma was found in the Sylvian fissure. The branch of MCA (M3 segment) terminated with a blind end at the junction with the calcified mass in the insula and temporal lobe, which was removed completely after coagulation of the junction with the parent artery (Fig.2A). He discharged after a week without neurological deficits and his convulsion attacks have not appeared for five years.

Macroscopic inspection revealed an entirely calcified mass measuring 2 x 1.5 x 2 cm (Fig.2B), which was decalcified for light microscopic study. Histologically, the mass wall was composed of bone, calcification and connective tissue (Fig.3A). Bony tissue contained many osteocytes and fat tissue, but neither osteoblast nor osteoclast could be seen (Fig.3B). The luminal side of the wall harbored a thrombus with fibrosis and small vessels (Fig.3C). The histological diagnosis was cerebral aneurysm with mural ossification and organizing thrombosis.

3. **Discussion**

Although calcified intracranial aneurysms are not rare and incidental detection of calcified foci may lead to the diagnosis of aneurysms, calcification is usually partial and considered as degenerative changes in cerebral vasculature. While calcium deposits and lipid infiltration were reported in different layers of aneurysm wall, calcification is thought to be of thrombotic origin.[1,2)]

Intra-aneurysmal blood stasis and activating clotting factors such as thrombomodulin, prostacyclin, or thromboxane are thought to accumulate and result in the formation of mural thrombosis and eventually in calcification or ossification.[1)] In our case, an organizing luminal thrombus was detected just beneath the ossified wall, suggesting that ossification started at the thrombus. In addition, although bony tissue of the aneurysmal wall contained fat tissue and many osteocytes, neither osteoblasts nor osteoclasts were seen. These pathological findings indicate that the bony mass developed over a long period and is stable and inactive because there was no evidence of active turn over of bone matrix and collagen fibers.

Kobayashi reported an anterior communicating artery aneurysm that became calcified over 17 years, and speculated that blood stasis and formation of mural thrombosis in the aneurysm ultimately resulted in the calcification.[1] Others attributed degenerative changes such as fibrosis, thrombosis, calcification and ossification to non-specific responses to hemodynamic stress.[3] Meyers who detected calcification along the edges of tears in the internal elastic lamina of infant arteries suggested that flow factors play an etiologic role.[2,4]

Because aneurysmal calcification or ossification is primarily seen in elderly individuals, an alternative mechanism should be considered in our case. Its ossification suggests that his aneurysm developed over a prolong period and may have arisen during the congenital or pediatric stage. Pediatric aneurysms tend to occur in the peripheral portion of middle cerebral artery and are often mycotic.[4,5] However, our patient had no history of the bacterial endocarditis and cardiac echnograms were free of abnormal findings. Although the mechanisms underlying its development are unknown, our case is the first documentation of a completely ossified aneurysm in a young individual.

References

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Figure Legends

Figure 1

(A) Computed tomography scan showing a calcified, approximately 2-cm diameter mass in the left temporal region. (B) Magnetic resonance imaging was iso-intense with a hypo-intense rim signal on T1-weighted images. (C) There was a mixed-intense signal with a hypo-intense rim on T2-weighted images. (D) 3 dimensional-CT Angiography showed the peripheral branch of MCA (arrow) connected to the calcified mass.

Figure 2

(A) Intraoperative photograph showing that the mass was located in the insula and branched from the peripheral portion of middle cerebral artery (M3 segment). A, aneurysm; F, frontal lobe; M, branch of M2; SV, superficial sylvian vein; T, temporal lobe.

(B) The macroscopic image shows a cross section of the calcified mass. The bar indicates 1cm.

Figure 3

(A) Histological examination proved that the wall of the mass was composed of bone with fat tissue (arrows). (B) Many osteocytes (arrows) were seen. (C) The luminal side of the wall harbored a thrombus with fibrosis (arrows). The histological diagnosis was cerebral aneurysm with marked ossification and thrombosis. (A: Hematoxylin & Eosin X25, B: X150, C: X25)

Abbreviations:

CT: computed tomography, MCA: middle cerebral artery

Fig. 1

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(A)

(B)

(C)

(D)

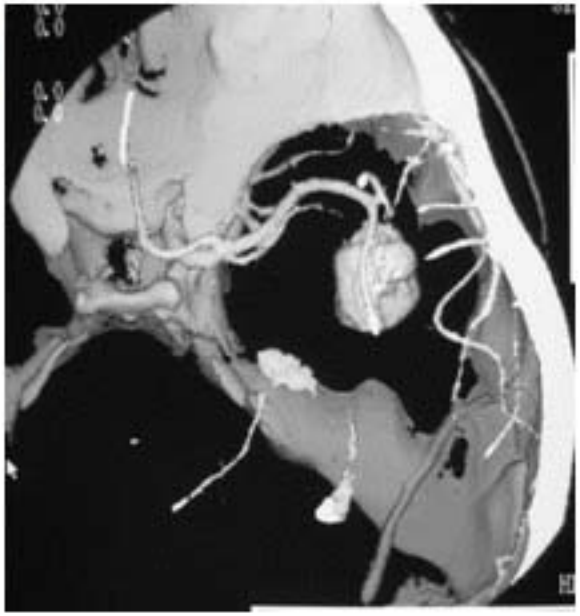

Fig. 2

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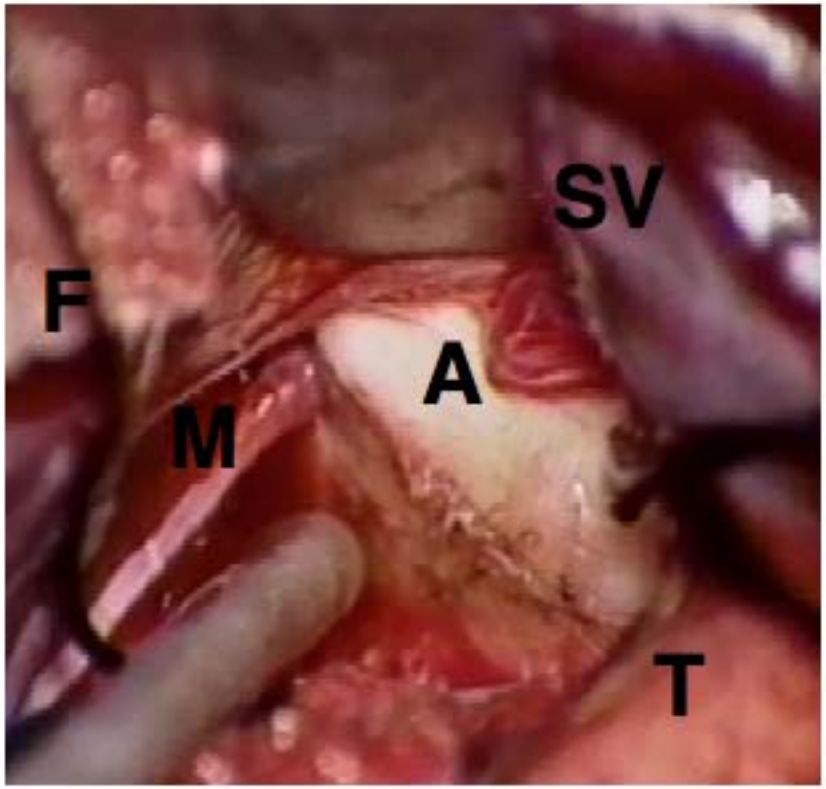

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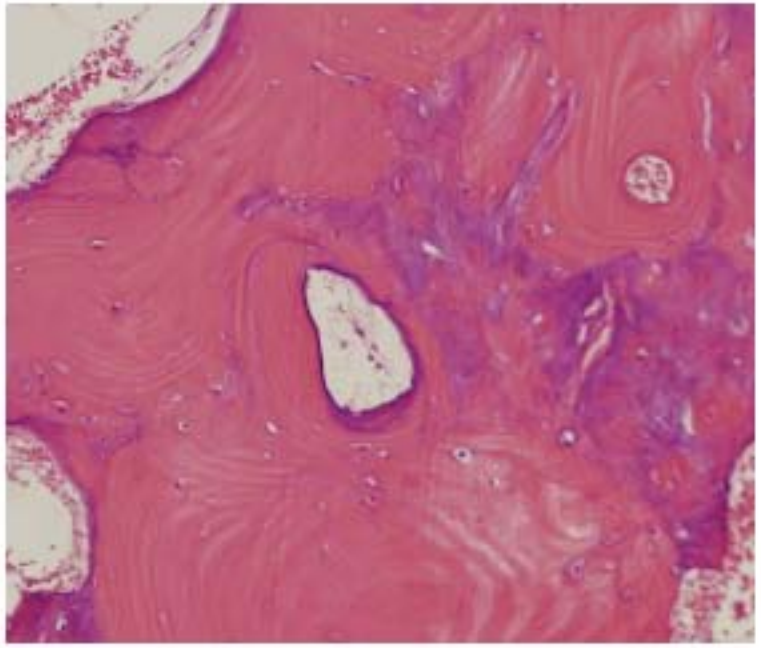

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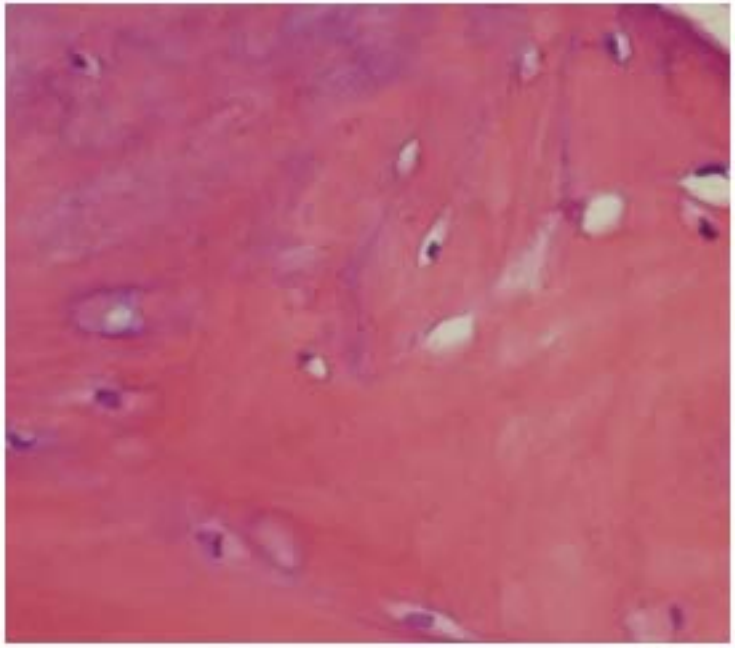

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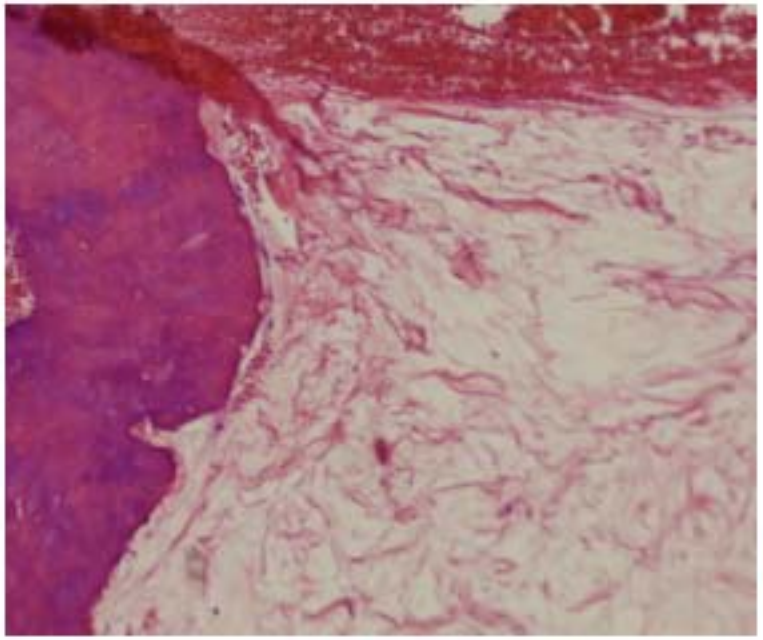

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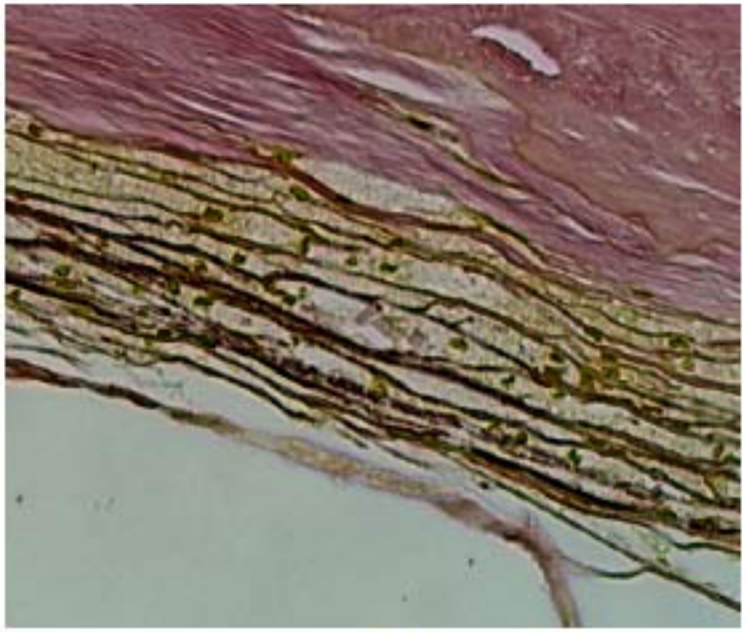

(D)

Fig. 3 Hayashi ↑