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

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 man presented with a history of several convulsion episodes. A computed tomography (CT) scan showed a calcified or ossified focus in the right temporal lobe. Cerebral angiography yielded no abnormal findings. At craniotomy, the M3 portion of the middle cerebral artery (MCA) was found to terminate with a blind end at the junction with the calcified mass. A pathological diagnosis of completely ossified cerebral aneurysm was made. Calcified cerebral aneurysms are not rare; they are thought to be the result of intra-aneurysmal thrombi or degenerative changes in the aneurysmal wall. However, the complete mural ossification of cerebral aneurysms is seldom seen, and ossified peripheral MCA aneurysms are still extremely rare in young individuals. We suggest that the entirely ossified aneurysm we encountered developed over a prolonged period and may have arisen during childhood.

1. Introduction

Calcified cerebral aneurysms are thought to derive from intra-aneurysmal thrombi or degenerative changes in the aneurysmal wall, e.g. fibrosis, lipid deposition, hyalinization, and vaculorization.[1,2] However, cerebral aneurysms with completely ossified walls are rare 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, we suggest that the ossification of his aneurysm occurred over a prolonged period.

2. **Case report**

This 30-year-old man presented with a history of several convulsion episodes. Anti-convulsant medication was ineffective. 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). On T1-weighted magnetic resonance images (MRI), the mass was iso-intense with a hypo-intense rim; on T2-weighted images there was a mixed-intensity signal with a hypo-intense rim (Fig. 1B, C). Angiography revealed no abnormal findings. Three-dimensional-CT angiography showed that the peripheral branch of the MCA connected with the mass (Fig. 1D).

Based on these findings we made a provisional preoperative diagnosis of thrombosed and calcified aneurysm and postulated that his convulsive episodes were attributable to the lesion. We also considered calcified cavernous angioma, Sylvian fissure meningioma, parasites, and tuberculoma in our differential diagnosis. After obtaining prior written informed consent from the patient and his

family, we proceeded to carry out further detailed examination and removal of the mass.

At right frontotemporal craniotomy neither subarachnoid hemorrhage nor old hematoma was found in the Sylvian fissure. The M3 segment of the MCA terminating with a blind end at the junction with the calcified mass in the insula and temporal lobe was removed completely after coagulation of the junction with the parent artery (Fig. 2A). He was discharged after a week without neurological deficits and he has experienced no convulsions in the past 5 years.

Macroscopic inspection revealed an entirely calcified mass measuring 2 x 1.5 x 2 cm (Fig. 2B). The mass was decalcified for light-microscopic study. Histologically, its wall was composed of bone, calcification and connective tissue (Fig. 3A). The bony tissue contained many osteocytes and fat tissue but neither osteoblasts nor osteoclasts could be seen (Fig. 3B). The luminal side of the wall harbored a thrombus with fibrosis and small vessels (Fig.3C). Elastica van Giesson staining showed that the wall included fragmented internal elastic lamina (Fig. 3D). The histological diagnosis was cerebral aneurysm with mural

ossification and organizing thrombosis.

3. **Discussion**

Although calcified intracranial aneurysms are not rare and the incidental detection of calcified foci may lead to the diagnosis of aneurysms, calcification is usually partial and considered as degenerative changes in the cerebral vasculature. While calcium deposits and lipid infiltration were reported in different layers of the aneurysmal 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 patient we detected an organizing luminal thrombus was detected just beneath the ossified wall. This suggested that the 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 remained stable and inactive because there was no

evidence of active turnover of bone matrix and collagen fibers.

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

Because aneurysmal calcification or ossification is primarily seen in elderly individuals, alternative mechanisms must be considered in our 30-year-old patient. The ossification suggested that his aneurysm developed over a long period of time and may have arisen during the embryonic or pediatric stage. Pediatric aneurysms tend to arise in the peripheral portion of the MCAmiddle and are often mycotic.[4,5] However, our patient had no history of bacterial endocarditis and cardiac echnograms disclosed no abnormal findings. Based on pathological evidence of mural hemorrhage and patchy calcification, Yasui et al.[6] suggested

that in their patient with a fusiform vertebral artery aneurysm, vessel dissection may have been attributable to mechanism underlying the development of mural calcification. In our patient there was no neuroradiological or intraoperative evidence of dissection; as he was quite young, we ruled out the likelihood of dissection. Although the developmental mechanisms remain unknown, ours is the first documented case of a completely ossified aneurysm in a young individual.

References

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Figure Legends

Figure 1

(A) CT scan showing a calcified, approximately 2-cm diameter mass in the left temporal region. (B) On T1-weighted MRI the mass was iso-intense and manifested a hypo-intense rim signal. (C) On T2-weighted MRI the mass showed a mixed-intensity signal with a hypo-intense rim. (D) On 3D-CTA, the peripheral branch of the MCA (arrow) was 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 the MCA (M3 segment). A, aneurysm; F, frontal lobe; M, M3 branch; SV, superficial Sylvian vein; T, temporal lobe. (B) Macroscopic image showing a cross-section of the calcified mass. The bar indicates 1 cm.

Figure 3

(A) On histological examination proved that the wall of the mass was composed of bone with fat tissue (arrows) (H&E x 25). (B) Many osteocytes (arrows) are seen

(H&E x150). (C) The luminal side of the aneurysmal wall harbored a thrombus with fibrosis (arrows) (H&E x 25). (D) The aneurysmal wall contained fragmented the internal elastic lamina (elastic van Giesson stain X 150). The histological diagnosis was cerebral aneurysm with marked ossification and thrombosis.

Abbreviations:

CT computed tomography;, MCA middle cerebral artery

Fig. 1

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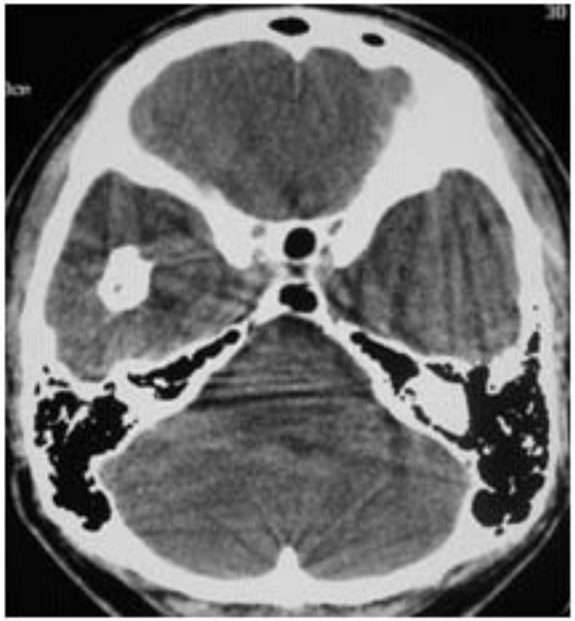
(A)



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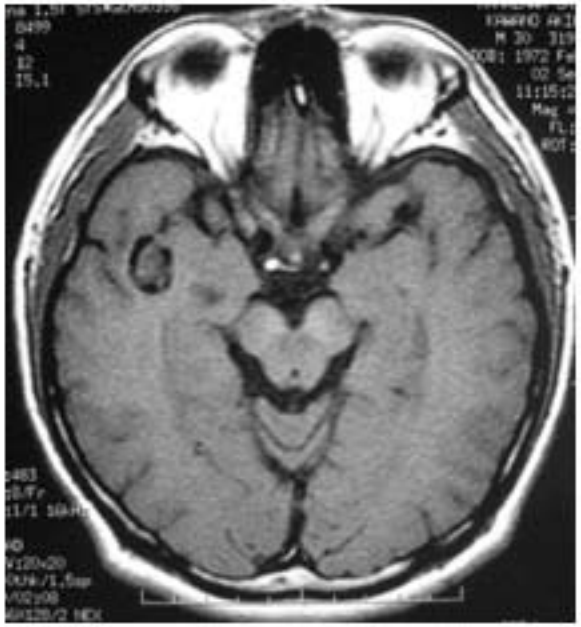

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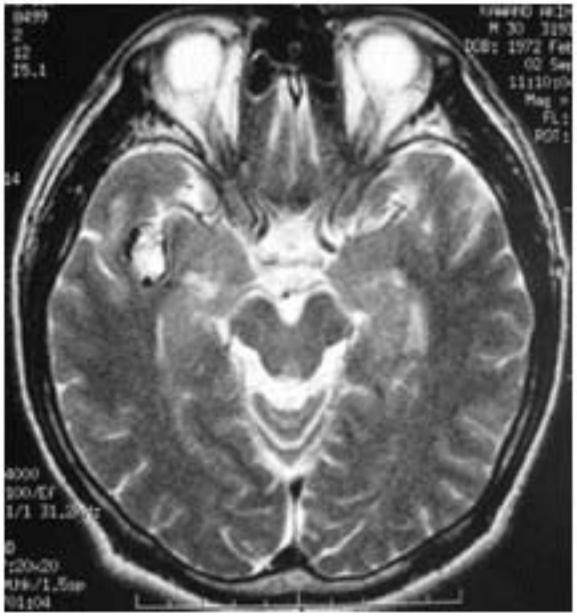

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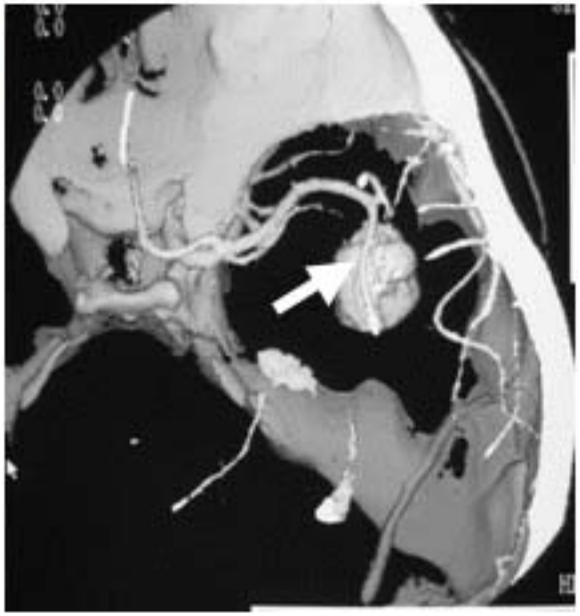

Fig. 2

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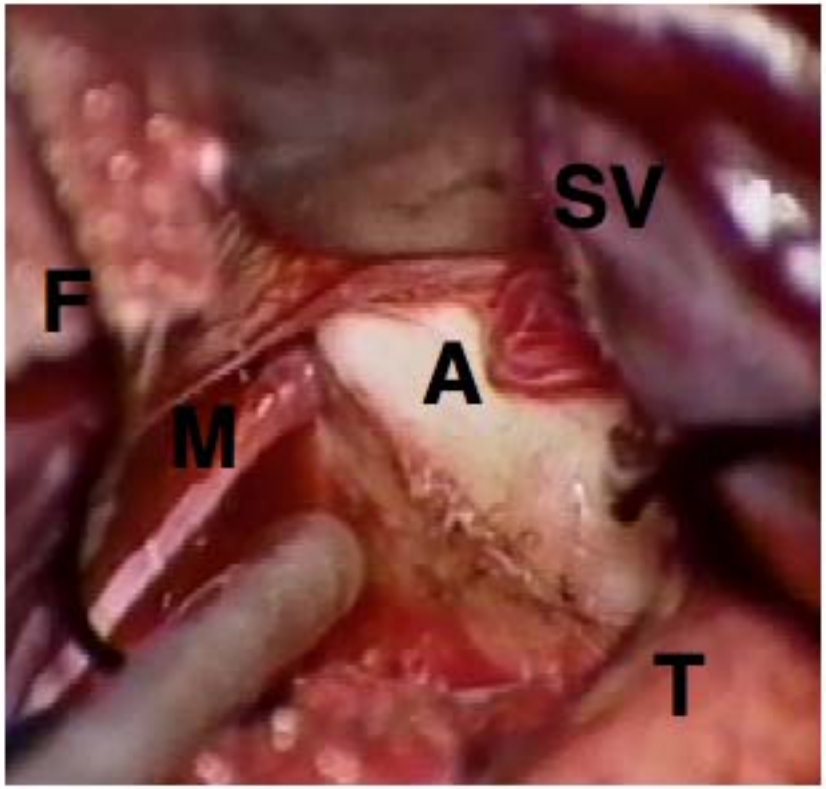

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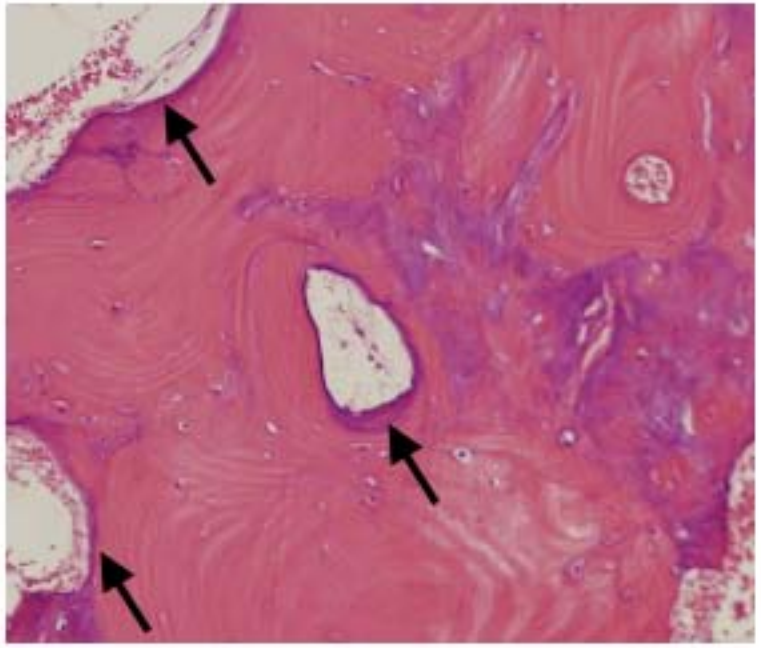

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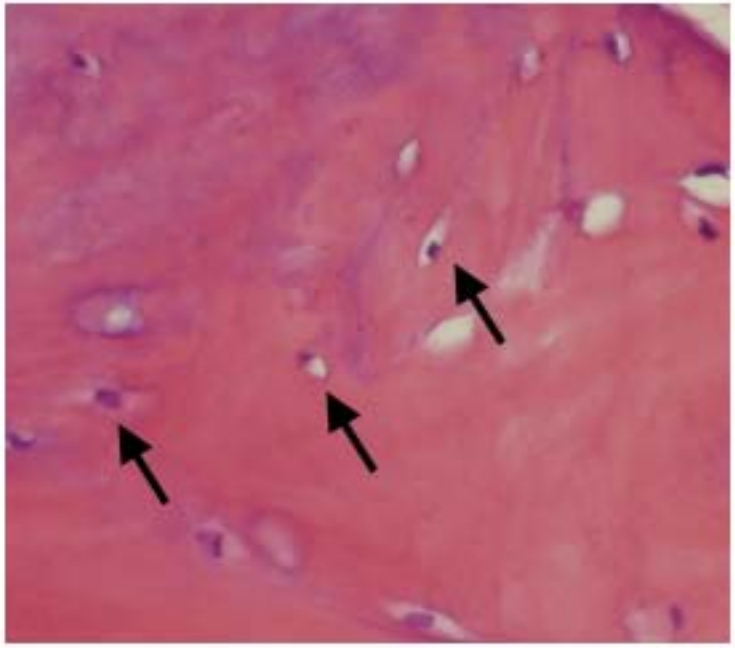

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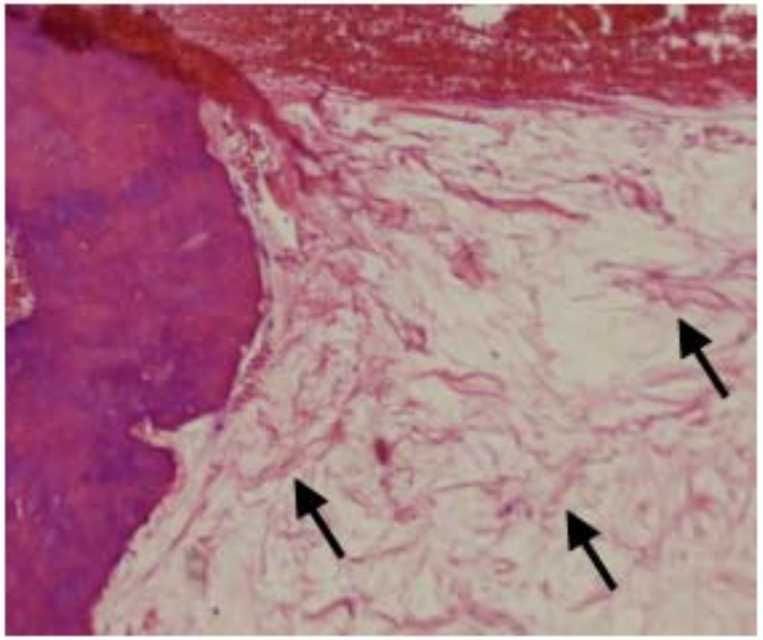

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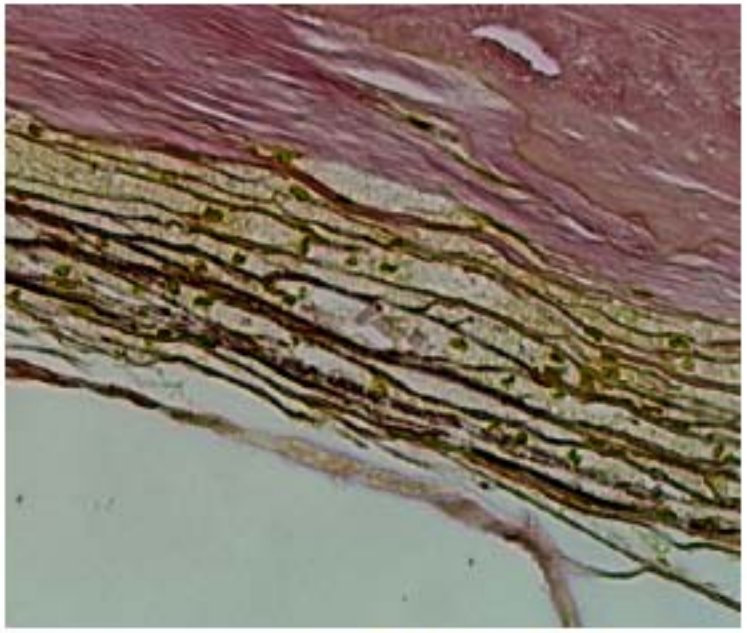

(D)

Fig. 3 Hayashi ↑