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Serial Neuroimaging of a Growing Thrombosed Giant Aneurysm of the Distal Anterior Cerebral Artery

—Case Report—

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Abstract

An 81-year-old female presented with a giant aneurysm of the distal anterior cerebral artery (A_3) which grew from a small saccular aneurysm to a huge aneurysm within 36 months before manifesting as a mass lesion. The thrombosed portion of the aneurysm showed growth, whereas the aneurysmal cavity did not change in size. Computed tomography and magnetic resonance imaging showed new bleeding in the thrombosed portion. Hemorrhage into the thrombus and/or aneurysmal wall might have caused the aneurysmal growth. She refused surgery and was discharged with no deficits. Distal anterior cerebral artery aneurysm which shows neuroimaging signs of growth requires regular follow up as such lesions may become giant before manifesting clinical symptoms.

Key words: giant aneurysm, distal anterior cerebral artery, growing aneurysm, thrombosis, magnetic resonance imaging

Introduction

Giant aneurysm of the anterior cerebral artery (ACA) usually arises from the anterior communicating-ACA complex.[11] The incidence of aneurysms of the distal ACA is low, and giant aneurysms of the distal ACA are rare,[5] with only 11 cases reported previously.[2,4,5,7,9–12,15] We describe the serial neuroimaging findings of a distal ACA aneurysm which grew from a small saccular to a giant aneurysm in 36 months.

Case Report

An 81-year-old female with atrial fibrillation first complained of dizziness in January 1996. She consulted her physician. Computed tomography (CT) and magnetic resonance (MR) imaging indicated an unruptured aneurysm of the distal ACA. No further investigation was performed. The patient received medical treatment for atrial fibrillation and remained asymptomatic for 3 years. She complained of memory disturbance and consulted her physician in January 1999. MR imaging revealed enlargement of the lesion. She was then referred to our department for further examination. Neurological examination on admission revealed no deficit.

CT performed in January 1996 showed a partially calcified mass adjacent to the falx in the left frontal lobe (Fig. 1A). Repeat CT in February 1999 revealed a large isodense mass with calcification in the left paramedian frontal region (Fig. 1B), with partial rim-like enhancement around the periphery of the lesion (Fig. 1C). CT performed in April 1999 showed slight enlargement of the lesion and high density spotty areas in the thrombus suggesting new hemorrhage (Fig. 1D). CT obtained in July 1999 did not show the high density areas (Fig. 1E).

MR imaging performed in February 1996 showed a small lesion with the anterolateral part appearing as high intensity on T_1- and low intensity on T_2-weighted images, corresponding to thrombus, and

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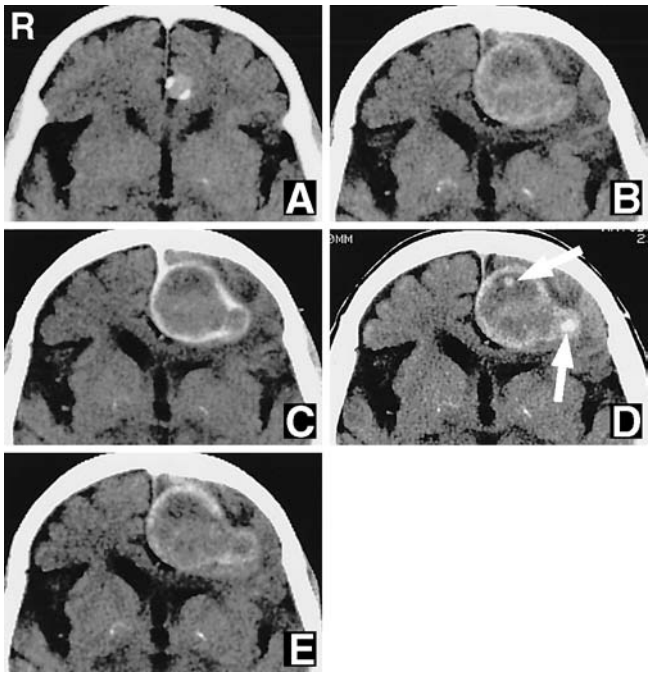

Fig. 1 Computed tomography scans (A) showing a small iso-density mass with calcification in January 1996, (B) showing enlargement of the lesion in February 1999, (C) with enhancement of the peripheral capsule with contrast medium, (D) showing spotty high density areas in the thrombus (*arrows*) in April 1999, and (E) showing no high density spotty area in July 1999.

the posteromedial part appearing as a signal void corresponding to blood flow (Fig. 2A, B). T_1-weighted MR imaging with gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) showed aneurysmal enhancement (Fig. 2C). MR imaging on admission in February 1999 showed a large, lobulated, mixed signal intensity mass in the left frontal region. The posteromedial portion appeared as a signal void with Gd-DTPA enhancement corresponding to blood flow in the aneurysm. The lesion was remarkably enlarged, but the aneurysmal cavity did not change in size over the course of 36 months (Fig. 2D–F). MR imaging performed in April 1999 also showed a lobulated, mixed signal intensity mass. The signal intensity of the thrombosed portion was almost the same as 2 months previously. However, the small lobulus was slightly enlarged and a small portion appeared as high intensity on T_1- and low intensity on T_2-weighted images (Fig. 2G, H). This small region was considered to indicate new hemorrhage.

Digital subtraction angiography (DSA) performed in March 1999 indicated that the A_1 segment of the left ACA was aplastic, and the bilateral A_2 originated from the right A_1. DSA also showed a small aneurysmal opacification at the A_3 portion of the left ACA (Fig. 3 *left*). Three-dimensional CT angiography clearly demonstrated the anatomical relationship between the aneurysm and the surrounding structures (Fig. 3 *right*).

The patient refused surgery because of her advanced age. She has since had no neurological problems and remains asymptomatic.

Discussion

Cerebral aneurysms occur in patients with various congenital circulatory defects.[13] Unusual anatomical variations of the cerebral vessels may cause flow disturbance leading to aneurysm formation.[3] The incidence of aneurysm of the distal ACA is less than 5%,[7] but may be higher in patients with azygos ACA.[6] In our case, the ACA was not the azygos type, but the A_1 segment of the left ACA was aplastic and the bilateral A_2 originated from right A_1. This congenital vascular defect might have caused hemodynamic stress to the distal ACA, leading to formation of the aneurysm.

Enlargement of a small aneurysm and presentation as a mass lesion is rare.[1] Giant aneurysms sometimes arise from the vertebrobasilar artery and associate with thrombus. Some of these thrombosed aneurysms continue to grow and present as mass lesions.[8] Giant aneurysms may grow by recurrent hemorrhage into the wall.[14] The wall of a thrombosed aneurysm becomes a highly vascular structure containing a rich network of vasa vasorum,[9] and this is considered to be the origin of hemorrhage in the thrombus. The aneurysm in our case was small when first identified, with a partial thrombus and calcification. Our findings indicate long-standing aneurysmal thrombosis and clot organization. In the following 36 months, the total volume of the mass apparently increased, but the aneurysmal cavity did not change in size. Therefore, intraaneurysmal thrombus is suspected to be responsible for growth of this distal ACA aneurysm. CT and MR imaging indicated new bleeding into the thrombus and adjacent aneurysmal wall. These findings can be considered to show that repeated small hemorrhage into the thrombus and/or aneurysmal wall might lead to aneurysmal growth.

In our case, serial CT showed growth of the aneurysm 3 years after the first examination. Intraaneurysmal hemorrhage was observed 2 months later. The aneurysm might have grown gradually in the first 3 years, although the patient had no symp-

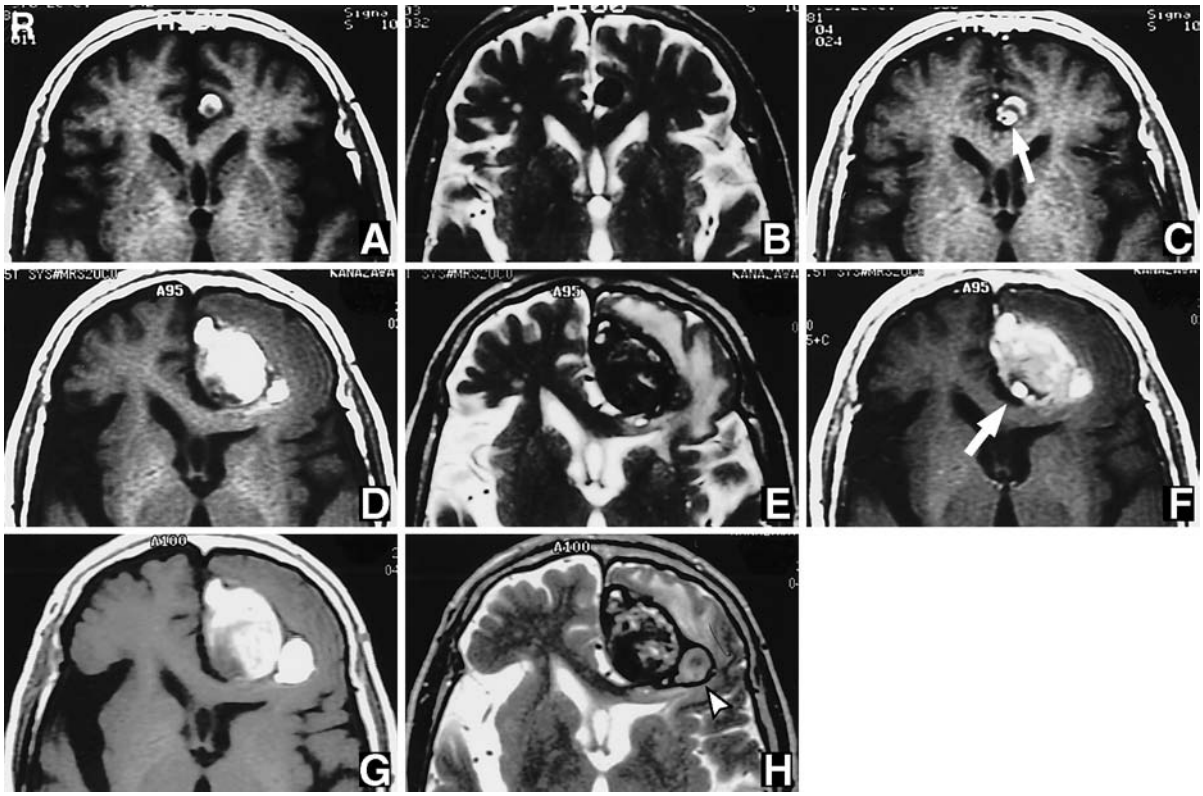

Fig. 2 T_1- (A) and T_2-weighted (B) magnetic resonance (MR) images showing a mass lesion with a thrombus and aneurysmal cavity in February 1996, and (C) the aneurysmal cavity enhanced by gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) (*arrow*). T_1- (D) and T_2-weighted (E) MR images showing a mixed signal intensity in February 1999, and (F) the aneurysmal cavity enhanced by Gd-DTPA (*arrow*) and not changed in size. T_1- (G) and T_2-weighted (H) MR images showing slight enlargement of the lesion and a small region of high (G) and low (H) intensity in the thrombus (*arrowhead*).

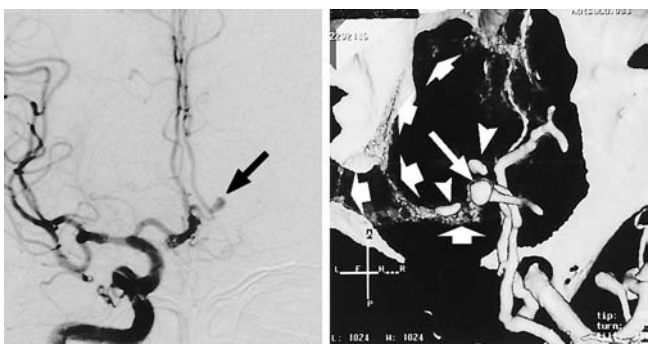

Fig. 3 *left*: Right internal carotid angiogram showing a small saccular aneurysm on the A_3 portion of the left anterior cerebral artery (*arrow*). *right*: Three-dimensional computed tomography angiogram showing the bilateral A_2 segments originating from the right A_1. The aneurysm cavity (*arrow*), calcifications (*arrowheads*), and enhanced capsule (*thick arrows*) are clearly demonstrated spatially.

toms, because her intracranial pressure was not high due to brain atrophy. Review of 23 cases of growing giant aneurysms found that the interval between the first diagnosis and the detection of growth ranged from 5 to 216 months.[1] Clinically silent growth must have occurred in most cases of giant aneurysm, and the course is unpredictable even after a long period of clinical silence unless the aneurysm is totally calcified. Therefore, once growth of the lesion has been observed, frequent neuroradiological examination (once every 3–6 months) is necessary, even if the patient remains without symptoms.

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