



Case Report

Basilar artery dissection with rupture 6 years after accidental detection: A case report

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ABSTRACT

Background: Chronic basilar artery dissection (BAD) is a rare pathology, and only a few reports have been mentioned in the literature. The imaging features of chronic BAD, especially those that develop into a subarachnoid hemorrhage (SAH), are unknown.

Case Description: We report a unique case of a chronic BAD with a split intimal flap that developed into an SAH. A 74-year-old man was diagnosed with BAD. After considering all treatment options, conservative treatment was selected for the patient. We continued imaging follow-up of the patient in our outpatient clinic once a year. The BA gradually dilated and the internal flap split. The patient and his family refused surgical treatment; therefore, conservative treatment was continued. Six years and 2 months from the first diagnosis, he developed a massive SAH and died.

Conclusion: In the case of this patient, the weakened condition of the internal elastic lamina may have caused dissection of the intimal flap between the intima and media. Furthermore, the dissection may have developed and connected the true lumen to the pseudolumen and induced BA rupture. Thereby, split of the internal flap could be a risk factor for rupture in chronic BAD.

Keywords: Basilar artery, Dissection, Intimal flap

INTRODUCTION

Basilar artery dissection (BAD) is a rare, but significant disorder. Ruecker *et al.* reported that BAD accounted for 1% of all cases of subarachnoid hemorrhage (SAH), and no <10.5% and 4.5% of posterior circulation and overall cranial vessel dissections, respectively.^[1,4] BAD starts with a tear of the intima and internal elastic lamina. Because the artery is filled with fast moving blood under arterial pressure, blood can find its way into the tear and starts to dissect or split the layers of the artery wall.

The clinical presentations of BAD are various. Large dissections may narrow the artery and can cause intra-arterial clots. Potentially, the intra-arterial clots and arterial stenosis caused by BAD can interrupt blood flow and cause ischemic stroke.^[10,12] Dissections with tears of the internal elastic lamina accompanied by rupture of the adventitia lead to subarachnoid hemorrhage.^[5,6]

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Furthermore, the enlargement of the arterial diameter due to dissection can cause brainstem compression.^[17]

Patients presenting with ruptured BAD have high mortality.^[8] By contrast, patients with unruptured BAD tend to have a favorable long-term outcome and become stable, whereas the BAD becomes chronic.^[5] However, it is known that some of the chronic BAD develops into SAH.^[3,7] It is challenging to decide the right time for surgical or endovascular intervention since the risk factor of chronic BAD developing into SAH is not well-understood.

Here, we report a unique case of a chronic BAD with a split intimal flap that developed into a massive SAH and discuss the risk factors for rupture in chronic BAD cases.

CASE DESCRIPTION

A 74-year-old man complaining of mild right hemiparesis was referred to our hospital. He had a history of atrial fibrillation, multiple neuromas, and an infarction in the right corona radiata due to severe stenosis of the right middle cerebral artery and had left hemiparesis since then. Neither computed tomography nor magnetic resonance imaging (MRI) demonstrated any ischemic or hemorrhagic lesions in the brainstem. However, MRI demonstrated thickened vessel wall of the BA with isointense appearance on T1-weighted and hyperintense appearance on T2-weighted images, indicating intramural hematoma [Figure 1a and b]. Time-of-flight MR angiography and basi-parallel anatomical scanning images indicated focal dilatation of both inner and outer diameters of the BA [Figure 1c and 1d]. From these morphological features, it was diagnosed as a BAD. Since there were no new ischemic changes in the MRI, we thought that the mild right hemiparesis was due to the compression of the BAD against the brainstem. Surgical treatment was a choice, but he and his family chose conservative treatment mainly because they wanted to avoid the risks of the surgical procedure.

Yearly follow-up MR examinations demonstrated gradual aneurysmal enlargement of the BA [Figure 2]. Furthermore, recanalized blood flow in the pseudolumen, enhancement in the BA wall, and changes in the imaging features of the intimal flap were observed in black-blood T1-weighted volumetric isotropic turbo spin echo acquisition images 6 years after the onset [Figure 3]. The intimal flap looked like it split into two. We recommended surgical treatment again to the patient; however, he and his family refused surgical treatment. Thus, we continued with conservative treatment and follow-up. Two months from the diagnosis of the duplicating intimal flap, the patient developed a massive SAH (Hunk and Kosnik, Grade V) and died [Figure 4].

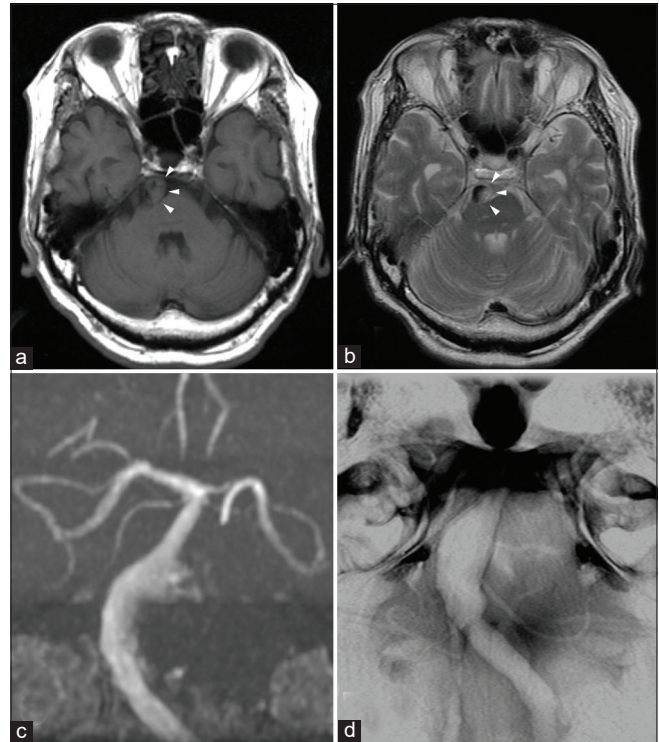


Figure 1: Magnetic resonance imaging (MRI) findings obtained on admission. Axial T1-weighted (a) and T2-weighted (b) MRI showing a narrowed eccentric flow void surrounded with intramural hematoma (arrowhead) that expands the basilar artery diameter. MR angiography (c) and basi-parallel anatomical scanning (d) showing focal dilatation of the basilar artery.

DISCUSSION

In the present case, the patient was stable in the acute phase and was symptomatically stable; however, he developed SAH in the chronic phase and died. The imaging features of chronic BAD that develops into SAH are not well-known. In our case, gradually dilating BA, enhancement in the BA wall, and intimal flap appearance of a split were observed. These imaging features may be considered as risk factors for rupture in chronic BAD.

Mizutani described the tortuous dilating basilar artery as a risk factor for poor prognosis.^[7] He postulated the substantial mechanism of the giant tortuous dilation as a two-stage dissection. The first stage is macroscopic dissection within a thickened intima and thrombus formation in this pseudolumen. The second stage is repeated hemorrhage into this thrombosed pseudolumen, combined with microscopic multiple mural dissections and wall distention based on the weakened condition of the internal elastic lamina.

Several studies indicated that aneurysm wall enhancement could identify unstable aneurysms.^[4,11,16] The exact mechanism of vessel wall enhancement in an aneurysm is

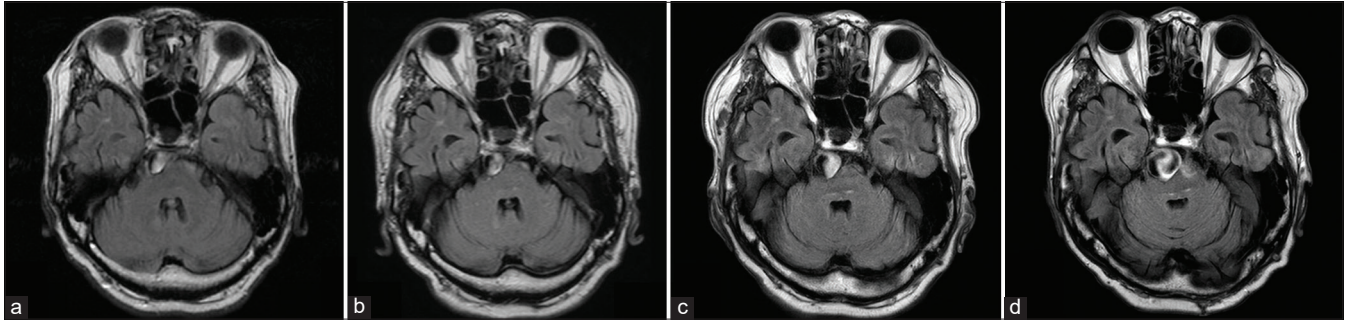


Figure 2: Time course images of fluid-attenuated inversion recovery. Magnetic resonance imaging on admission (a), 2 years later (b), 4 years later (c), and 6 years later (d) slow dilatation of the basilar artery.

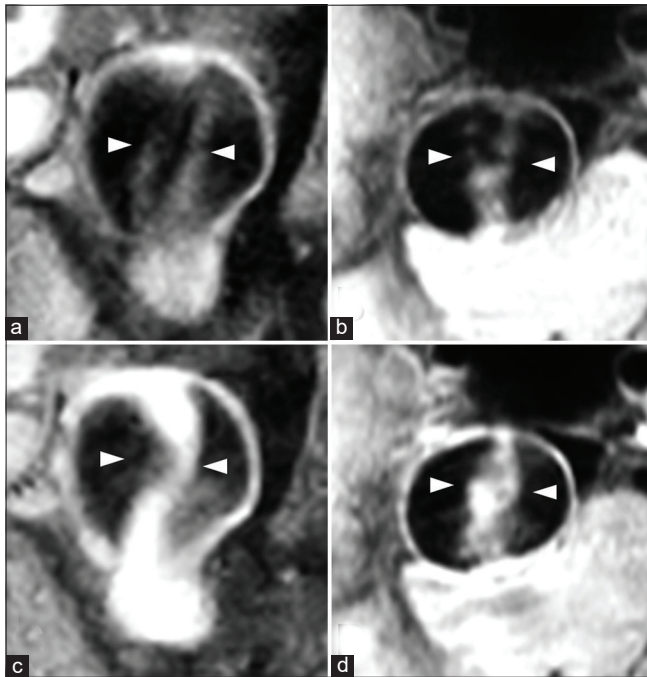


Figure 3: Magnetic resonance imaging (MRI) findings 6 years from the first diagnosis. Coronal (a) and axial (b) noncontrast volumetric isotropic turbo spin echo acquisition (VISTA) MRI and coronal (c) and axial (d) contrast VISTA MRI showing blood flow in the pseudolumen and changes in image findings of the intimal flap with enhancement. The intimal flap looks like it split into two (arrowhead).

unclear, although an inflammation is thought to be involved. Inflammation plays an important role in the pathophysiology of an aneurysm. The inflammatory process is initiated by a hemodynamic stress and leads to apoptosis of smooth muscle cells, which are the predominant matrix synthesizing cells of the vascular wall.^[2] These processes weaken the arterial wall progressively, resulting in dilatation, aneurysm formation, and ultimately rupture.

On the other hand, splitting of the intimal flap has never been reported as a risk factor. In our case, the initial intimal flap

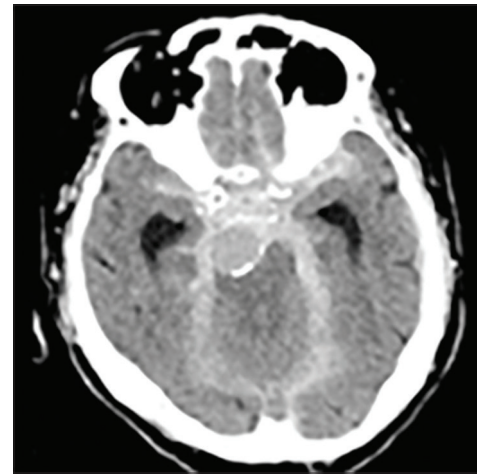


Figure 4: Computed tomography showing a massive subarachnoid hemorrhage in the posterior fossa.

may have been formed within the intima. The reason why the intimal flap split is unknown. However, the weakened condition of the media may have caused dissection between the media and adventitia [Figure 5]. Therefore, the intimal flap was imaged as if it was split into two. Furthermore, the dissection may have developed and ruptured. Thus, splitting of the internal flap could be a risk factor for rupture in chronic BAD.

Surgical clipping and wrapping, endovascular treatment, and conservative treatment have been used for the management of BAD.^[1,5,9,13,15] However, currently, there is no established treatment. Since the mortality rate of ruptured BAD is high, we should actively consider proceeding surgery against cases with this distinctive image even in chronic BAD.

CONCLUSION

We found a split of the intimal flap in a case of chronic BAD followed by lethal SAH. This imaging feature may be a new risk factor for rupture in chronic BAD. Further studies are

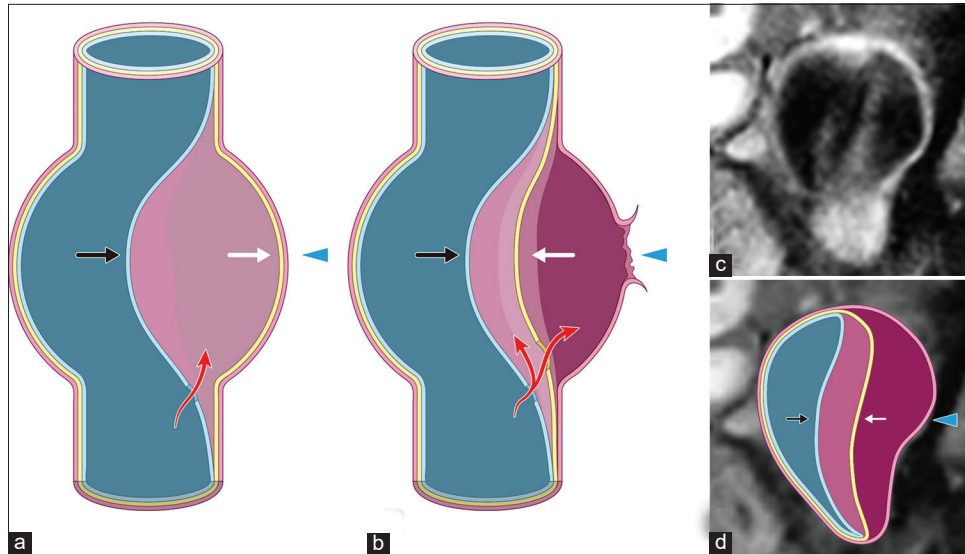


Figure 5: Illustration of the hypothesis of the split intimal flap (a and b). First, the blood flow into the space between the adventitia (arrowhead), media (white arrow), and intima (black arrow). Next, the weakened condition of the media may cause dissection between the media and adventitia and induce rupture. Coronal noncontrast volumetric isotropic turbo spin echo acquisition magnetic resonance imaging (c) with the illustration of the hypothesis in this case (d) image of the adventitia (white arrowhead), media (white arrow), and intima (black arrow).

needed to reveal the significance of this imaging feature of chronic BAD.

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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