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Fusiform Basilar Artery Aneurysm Associated with Pontine Lacunar Infarctions

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Case Description

A 47-year-old woman with hypertension and chronic kidney disease (CKD) was admitted for clinical syndrome of hypertensive urgency. On initial examination, she was found to have right-sided weakness and gait ataxia, while rest of the neurological examination was within normal limits. The patient reported that these symptoms gradually started 6 month ago, for which she never seek medical attention. Magnetic resonance angiography (MRA) showed aneurysmal distention of the basilar artery. (Fig. A) The artery measured 58 mm in length and 16 mm in diameter, pressing on the right pons. (Fig. B) Lacunar infarcts were also revealed in left pons, most likely caused by perforator occlusion within the aneurysm. Antiplatelet, antihypertensive, and lipid-lowering therapy was initiated, and patient was discharged with close follow-up by neurology and neurointervention divisions.

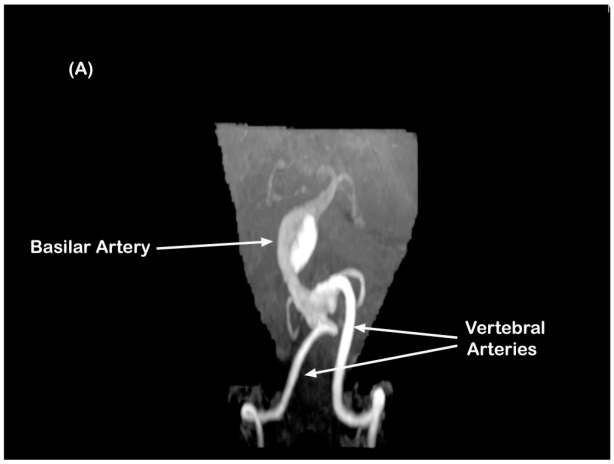

Figure A.

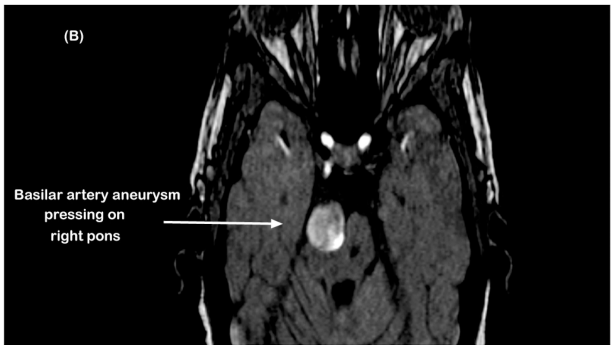

Figure B.

Pathogenesis and Proposed Mechanism of Stroke

The most common complication of cerebral dilative arteriopathy appears to be caused by hemodynamic disturbance, especially cerebral ischemia, mainly in the area supplied by the basilar artery. Intraluminal thrombus, local embolism, atherosclerosis, and obstruction of paramedian branches seems to be most likely mechanism [1]..Different theories have been put forth to explain the etiology and pathogenesis of basilar artery aneurysm. Genetic diseases such as Marfan's syndrome, Ehlers–Danlos syndrome, Fabry's disease, sickle cell disease, and autosomal dominant polycystic kidney disease have been associated with the development of dilative arteriopathy and thrombotic complications in children and young adults [2]. In the elderly people, advanced age, male gender, hypertension, previous history of myocardial infarction, and lacunar infarct have been postulated as possible risk factors [3]. There is a fair amount of uncertainty regarding atherosclerosis being associated with DIA. Sudhir et al., [4] reported higher prevalence of coronary ectasia in patients with familial hypercholesterolemia while Pico et al., [3] chal-

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Table 1. Estimated risks of ischemic and nonischemic events in 5 years [6]

Clinical Symptoms.	%
TIA.	10
Stroke	7.6
Brainstem compression	10%
Progressive hydrocephalus	3.3
Subarachnoid hemorrhage	2.6
Intraparenchymal hemorrhage	4.7
Case fatality	36.2

lenged this association. Few recent studies have shown low levels of serum matrix metalloproteinases-3 (MMP-3) and 5A polymorphism of the promoter region of MMP-3 as a possible mechanism of aneurysm formation [5].

Clinical Symptoms and Therapeutic Considerations

Transient ischemic attack (TIA) can remain asymptomatic or can present with symptoms ranging from stroke, compression of the cranial nerves, headache, hydrocephalus, brain stem compression, trigeminal neuralgia, tinnitus and vertigo [2]. Clinical course has recently been summarized by the Wolters et al., [6]. Estimated risks of ischemic and nonischemic events occurred in 5 years are shown in Table 1.

No prospective, randomized trials are available to define best medical or interventional treatment of the cerebral dilatative arteriopathy. Our knowledge of natural history of the disease and treatment options are largely based on case series and cohort studies. The use of antiplatelet is controversial, and aspirin alone for primary prevention has not been shown to be beneficial. Symptoms caused by the compression of cranial nerves (trigeminal neuralgia or hemifacial spasm) may be alleviated by decompressive surgery. However, due to its potential complications, surgical treatment or interventional therapy of the aneurysm is limited, high risk, compassionate, and investigational. Treatment of patients with vertebrobasilar dolichoectasia (VBD) needs to be individualized by patient characteristics, symptoms, the risk of treatment and compliance [7].

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