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Case Report

Vertebrobasilar Artery Anomaly Presenting With Transient Bow Hunter's Syndrome

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Abstract

Vertebrobasilar artery anomaly can present with transient Bow Hunter's syndrome. A 37-year-old woman presented with a 1-year history of frequent fainting, dizziness and palpitations when turning her head to the right. Her physical examination showed no abnormalities except that the right Dix-Hallpike test, supine to head-lateral test and squat to stand test transiently induced subjective symptoms but not nystagmus. Time-of-flight magnetic resonance angiography revealed the following central vascular anomalies: (1) vertebrobasilar artery anomaly; (2) hypoplasia of the bilateral posterior communicating arteries; (3) hypoplasia of the posterior inferior cerebellar arteries; and (4) stenosis of the bilateral intracranial vertebral arteries. Diffusion weighted magnetic resonance imaging showed hyperintensity of the left-side midbrain and left-side vermis. One month after antiplatelet therapy with aspirin and recommended changes in head positioning, transient Bow Hunter's syndrome subsided. She had no repeat of symptoms over the following 1 year. [*Tzu Chi Med J* 2010;22(3): 149–152]

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1. Introduction

Bow Hunter's syndrome (BHS), also called rotational vertebral artery syndrome, results from vertebrobasilar insufficiency (VBI) secondary to mechanical stenosis or occlusion of the unilateral vertebral artery (VA) at the atlanto-axis during head rotation [1,2]. Diagnosis of BHS is sometimes difficult, especially when it is transient. Although vertebrobasilar artery anomaly is rare, it could contribute to BHS [1]. Herein, we report a rare case of vertebrobasilar artery anomaly with transient BHS.

2. Case Report

A 37-year-old woman came to our clinic because of a 1-year history of frequent episodes of fainting, dizziness, and palpitations with sudden head turning to the right side, both in the lying and standing positions and

when suddenly standing up. The symptoms always lasted several seconds. Symptoms subsided after she turned her head back to a natural position. There was no vertigo, nausea, vomiting, headache, tinnitus, diplopia, paresthesia, ataxia, or other neurological symptoms. She had visited other clinics but her symptoms were not relieved.

The patient was a right-handed housewife with a height of 160 cm, weight of 47 kg, and body mass index of 18.4 kg/m^2. She had no history of hypertension, diabetes mellitus or heart disease, and did not smoke, drink alcohol or chew areca nuts. Her physical examination showed no abnormalities except that the right Dix-Hallpike test and supine to head-lateral test induced subjective symptoms but not nystagmus.

Table 1 — Neck duplex scanning of extracranial vertebral arteries

	Diameter (cm)	Average velocity (cm/s)	Average flow (mL/min)	Resistance index
Right	0.332	21.0	109	0.70
Left	0.367	22.2	141	0.68

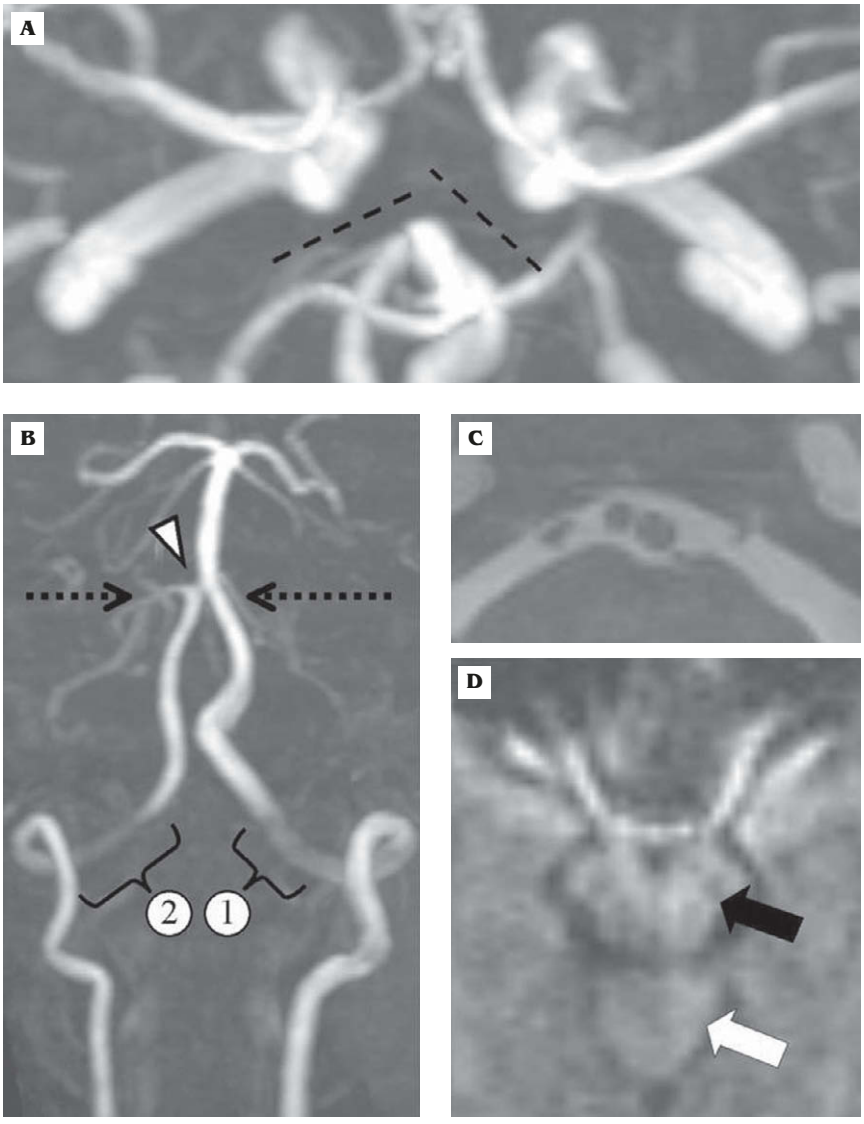

Fig. 1 — (A) Time-of-flight magnetic resonance angiogram shows hypoplasia of the bilateral posterior communicating arteries limiting circulatory communication of the anterior and posterior circulation (dotted lines). (B) The right vertebral artery (VA; diameter, 3.8 mm) directly flows into the right anterior inferior cerebellar artery, and is located near the basilar artery (diameter, 4.3 mm) (arrowhead) but does not connect with it. The left intracranial VA (diameter, 3.7 mm) has a stenotic segment with decreased blood flow (bracket 1), as does the right VA (bracket 2). In addition, the bilateral posterior inferior cerebellar arteries are hypoplastic. (C) T2-weighted axial view of the area indicated by the dotted arrows in (B) shows that the right VA and right anterior inferior cerebral artery are not connected to the basilar artery. (D) Diffusion weighted magnetic resonance imaging shows hyperintensity of the left-side midbrain (black arrow) and left-side vermis (white arrow).

The Epley (canalith repositioning) maneuver was attempted several times (rollover test), but did not resolve the symptoms. In addition, the squat to stand test induced similar symptoms. Cervical spinal extension and flexion radiographs did not show any bony structural deformity. Twenty-four-hour Holter monitoring, blood examinations and an awake electroencephalogram showed no abnormalities.

Neck duplex scanning simply revealed that the right VA had a slightly higher vascular resistance than the left (Table 1). Time-of-flight magnetic resonance angiogram (TOF MRA) showed: (1) hypoplasia of the bilateral posterior communicating arteries (Fig. 1A); (2) a vertebrobasilar artery anomaly (Figs. 1B and 1C); (3) stenosis of the bilateral intracranial VA (Fig. 1B); and (4) hypoplasia of the posterior inferior cerebellar arteries (Fig. 1B). Diffusion weighted magnetic resonance imaging showed hyperintensity of the left-side midbrain and left-side vermis (Fig. 1D).

An antiplatelet agent, aspirin (Bokey 100 mg daily, oral), was recommended. Furthermore, the patient was asked to turn her head slowly and not keep her head in the right-rotation position for too long. Her symptoms subsided over the following month, and the aspirin was discontinued. She had no repeat of symptoms over the following year.

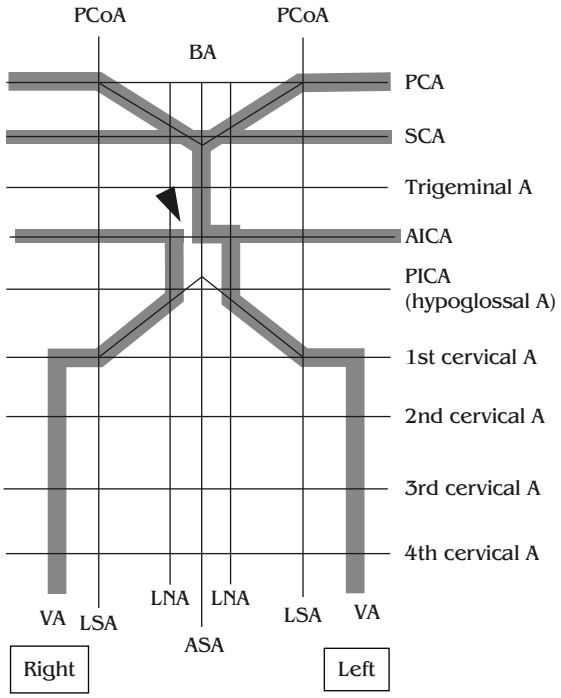

Fig. 2 — It is probable that the vertebrobasilar arterial system (thin net) degenerated abnormally in the 5th week of embryologic development, resulting in the vertebrobasilar artery anomaly (thick lines) in our patient. The bilateral longitudinal neural arteries (LNA) did not completely fuse into the basilar artery (BA). The right vertebral artery (VA) was directly connected to the right anterior inferior cerebral artery (AICA) via a segment of the LNA, but did not connect to the BA (arrowhead). The bilateral posterior communicating arteries (PCoA) and posterior inferior cerebellar arteries (PICA) were hypoplastic. PCA=posterior cerebral artery; SCA=superior cerebellar artery; LSA=lateral spinal artery; ASA=anterior spinal artery.

3. Discussion

The negative Dix-Hallpike test, negative supine to head-lateral test and negative rollover test in this patient implied that benign paroxysmal positional vertigo was not likely. These positioning tests simply induced subjective episodes of fainting and dizziness, so a diagnosis of hemodynamic changes in the central nervous system when changing position or turning the head was preferred. Despite the normal 24-hour Holter monitoring, cardiogenic syncope was possible, but this condition seldom occurs secondary to simple head turning to one side.

Although digital subtraction angiography is the gold standard for diagnosing VA stenosis [3], it has a slight morbidity associated with mortality. Extracranial duplex scanning is the initial noninvasive investigation of choice for extracranial VA disease, as is transcranial duplex scanning for intracranial VA disease, but these scans cannot evaluate VA at the atlanto-axis [3]. TOF MRA relies on flow-related enhancement to depict vessels and flow characteristics. When used alone, it can detect intracranial and extracranial VA, and even VA at the atlanto-axis, but low flow is occasionally misdiagnosed as stenosis [3]. TOF MRA and extracranial duplex scanning are preferred over digital subtraction angiography or transcranial duplex scanning because of convenience, safety and accuracy. Furthermore, extracranial duplex scanning is performed over one side of the neck plane when the examinee's head is rotated to the other side; therefore, the data (Table 1) accurately depict a stable hemodynamic condition of the extracranial VA, rather than a transient hemodynamic state of mechanical stenosis at the atlanto-axis.

Among 18–89-year-old adults, 74.6% have defects of the posterior circle of Willis and, of these, 57.4% have hypoplasia of the bilateral posterior communicating arteries [4], limiting circulatory communication of the anterior and posterior circulation [5]. This results in VBI. Vertebrobasilar artery anomaly is rare, and might result from embryologic maldevelopment (Fig. 2). This means that the posterior circulation in our patient was separated into two independent distributions (Figs. 1B and 1C). Furthermore, the bilateral posterior inferior cerebellar arteries were hypoplastic, and the right anterior inferior cerebellar artery and right VA supply the right-side brainstem and cerebellum, and the left anterior inferior cerebellar artery and left VA supply the left-side brainstem and cerebellum.

Fine branches of the basilar artery also supply the brainstem. Therefore, her posterior circulation region was susceptible to VBI, or even transient hemodynamic change.

Head rotation is known to induce mechanical stenosis or occlusion of the contralateral VA at the atlanto-axis, while in the ipsilateral VA, vascular resistance is reduced and it becomes unobstructed. With rightward head rotation, VBI in the left anterior inferior cerebellar artery and left VA is worse than on the right side. It is probable that atherosclerosis has gradually restricted the wall compliance in the bilateral intracranial VAs. According to the clinical condition and imaging studies, we suggest a transient VBI, which could not be seen on duplex scanning, occurring secondary to mechanical stenosis of the left VA at the atlanto-axis when the head is turned to the right side. Transient VBI did lead to fainting, but fortunately not to stroke. Dizziness might occur secondary to mild ischemia of the left-side brainstem and left-side vermis (Fig. 1D). Palpitations followed a compensatory increase in the cardiac output. As a whole, the fainting and dizziness from transient VBI secondary to head rotation were diagnosed as transient BHS.

We could do nothing for the vertebrobasilar artery anomaly; therefore, an antiplatelet agent and changes in head positioning were recommended to reduce transient BHS, or even prevent Bow Hunter's stroke (6). In the following month, the transient BHS subsided and the medication was discontinued. There was no repeat of symptoms over the following year.

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