

A Case of Bow Hunter's Stroke Caused by Non-Dominant Vertebral Artery

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Abstract- Rotational movements in the territory of vertebrobasilar artery of the head and neck can induce vertebrobasilar insufficiency (VBI) or infarction. The term "bow hunter's stroke" or "rotational VBI" has been used to describe this clinical syndrome. In most cases, symptoms were provoked because of involvement of a dominant vertebral artery (VA) with hypoplasia or occlusion of the contralateral VA. The author presented a case in which bow hunter's stroke was caused by occlusion of a non-dominant VA ending in the posterior inferior cerebellar artery (PICA). Diagnosis of rotational VBI was based on stereotypical clinical symptoms related to head rotation and hemodynamic study of the effects of head rotation. VA compression was documented in dynamic ultrasonography including the disappearance of end-diastolic flow in extracranial portion of VA and marked reduction in blood flow velocity (more than 50%) in the intracranial portion of VA upon head rotation. We emphasize that rotational occlusion of this anatomical variation is an important cause of VBI. This may cause permanent neurological deficits if left undiagnosed.

Key Words: Vertebrobasilar insufficiency, Bow hunter's stroke, Ultrasonography, Vertebral artery, Posterior inferior cerebellar artery

Acta Neurol Taiwan 2005;14:69-73

INTRODUCTION

Rotational vertebrobasilar insufficiency (VBI) or bow hunter's stroke⁽¹⁾ is used to describe the unique syndrome of hemodynamic VBI due to the compression of vertebral artery (VA) caused by rotational movements of the head⁽²⁾. VA is occluded temporarily by turning head

to the opposite side^(2,3), and this occlusion is usually asymptomatic due to sufficient contralateral flow. In most cases, symptoms are provoked when the dominant VA is compressed with a lack of sufficient collateral supply⁽⁴⁾. We presented a case in whom transient occlusion of the non-dominant left VA occurred during head turning to the right side.

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Received November 29, 2004. Revised January 5, 2005.

Accepted March 9, 2005.

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CASE PRESENTATION

A 67-year-old male was admitted to the hospital due to vertigo, double vision and unsteady gait. The medical history included hyperlipidemia and old stroke with right hemiparesis. He received anticoagulant therapy for one year. Symptoms aggravated when the patient turned his head to the right and resolved 3-5 minutes later after returning his head to the neutral position. His physical examination was unremarkable. Neurological examination revealed crossed hemiparesis, cerebellar dysfunction including left side dysmetria and ataxia. There were no limitations in extraocular movements and

no nystagmus. During hospitalization, there were two episodes of transient vertiginous attacks accompanied by exacerbating numbness over the left face and one episode of right side weakness for half an hour which was aggravated by head rotation to the right side.

Duplex sonography of VA showed high resistance flow in the left extracranial portion of VA (V2) (Fig. 1, velocity 23/5 m/s) in the neutral position. The absence of diastolic component in left V2 was demonstrated while turning to right side (Fig. 2, velocity 22/0 m/s). Transcranial color-coded imaging revealed markedly reduced flow in left intracranial portion of VA (V4) when the head was turned to right side (Fig. 3, velocity

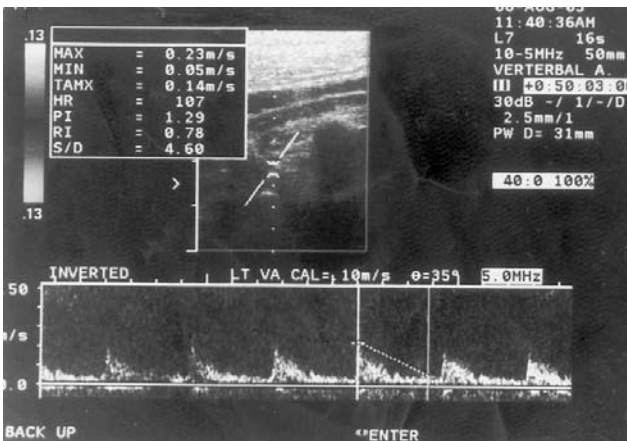


Figure 1. Left V2 in the neutral position.

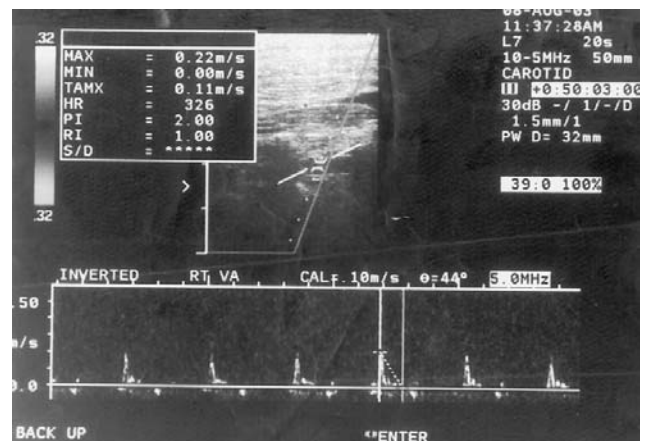


Figure 2. Left V2 after turning to the right side.

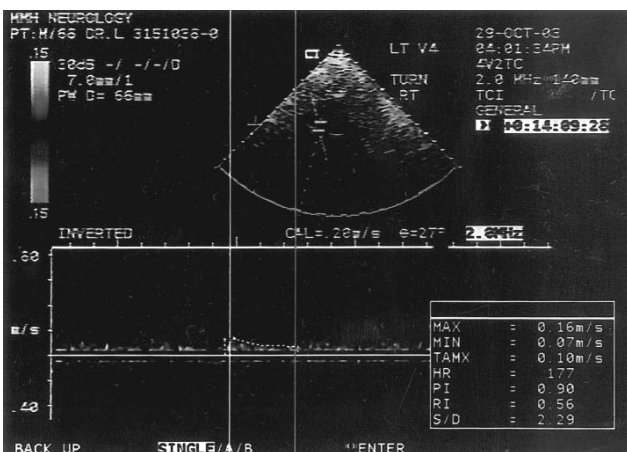


Figure 3. Left V4 after turning to the right side. Right V4 is not shown here.

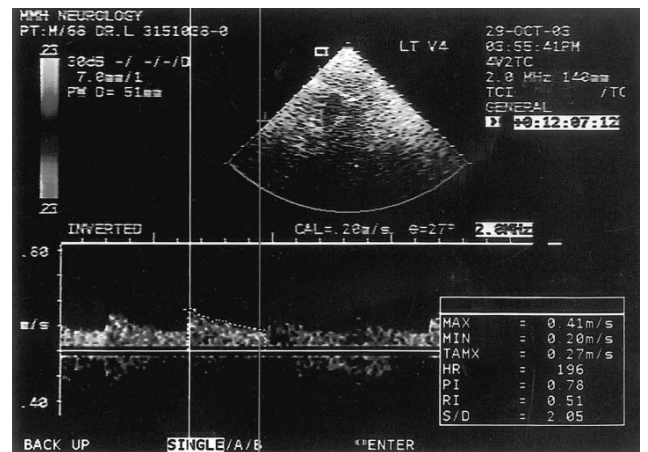


Figure 4. Left V4 in the neutral position.



Figure 5. Cerebral angiography in the lateral view showed the ending of Left VA in left PICA.

16/7 m/s) compared with that when the head was in the neutral position (Fig. 4, velocity 41/20 m/s). However, there was a normal flow profile in the basilar artery and the bilateral posterior cerebral arteries (PCA). Magnetic resonance imaging (MRI) study of the brain confirmed left lateral medullary infarction.

Cerebral angiography study displayed several characters of vessel architecture, including stenosis in bilateral VAs in atlantoaxial portion of VA (V3). The left VA was smaller than right VA and terminated in PICA (Fig. 5). The muscular branch of right VA and neck vessels were acting as collaterals for the basilar artery, with bilateral carotid system acting as collaterals for bilateral PCAs and basilar artery via posterior communicating artery.

Presenting symptoms remitted after the patient was instructed to refrain from head turning to the right side and we increase the dosage of anticoagulation therapy to reach an international normalized ratio (INR) of 2.5-3.5.

DISCUSSION

The initial findings of neurological examinations in this case were suggestive of Wallenberg's syndrome due to left VA insufficiency. In order to find the mechanism of recurrent stroke in this patient who was already receiving anticoagulant therapy, we arranged cerebral

angiography and dynamic ultrasonography to find the initial site of hemodynamic compromise relating to head posture.

“Bow hunter's stroke” is described in the literature as a unique syndrome presenting in an episodic manner, characterized by stereotypic symptoms including dizziness, vertigo, double vision, unsteady gait, a feeling of faintness often leading to loss of consciousness upon head rotation, and can be relieved after returning the head to the neutral position⁽³⁾.

Najli in 1870 and Gegenbauer in 1899 were the first to describe such a phenomenon. Because of anatomical relationship with neighboring bone, muscles and ligaments, the vertebral arteries are susceptible to mechanical compression during head rotation⁽³⁾. Cadaver studies have demonstrated that the head rotation caused narrowing of the contralateral VA⁽⁵⁾, especially at C1-C2 level. The ipsilateral atlantoaxial articulation is fixed during rotation of the head, and the opposite atlas moves both downward and forward in relation to the axis. This results in stretching and narrowing of the contralateral VA at the C1-C2⁽⁶⁾. Mitchell reported that the blood flow of V4 was reduced significantly in either ipsilateral or contralateral side during head rotation, with a greater compromise on the contralateral side⁽⁷⁾.

Intrinsic lesions (atherosclerosis most common) or extrinsic compression of VA can cause VBI after head turning. The extrinsic lesions include cervical spondylosis (most common), cervical disc herniation, craniovertebral junction malformation, tumor, fibrous band, apophyseal joint subluxation, atlantoaxial arterial anomaly^(8,9). In our case the cause of hemodynamic compromise is due to the anatomical variation of non-dominant VA ending in PICA.

Mechanical compression may not be the primary cause; rapid and marked stretching of the arteries can cause vasospasm, arterial dissection, or intramural hemorrhage. These repeated microtraumas lead to thrombi, atherosclerosis and decreased blood flow in the vessels⁽¹⁰⁾.

The rotational compression of VA is usually asymptomatic because of adequate collateral flow from contralateral VA. In Husni's study, VBI was induced in their

patients because of hypoplasia and narrowing of the opposite VA⁽¹¹⁾. Struzengger et al. also reported preexisting anomalies or diseased contralateral VA and functionally absence of posterior communicating arteries on both sides in patients with rotational VBI⁽¹²⁾. Therefore, as a result of insufficient collateral flow, these patients are more susceptible to hemodynamic change when their dominant VAs are compressed during head turning to the opposite side^(6,9,12).

In our case, repetitive symptoms of left lateral medullary syndrome developed when the left non-dominant VA was compressed during the head was turned to the right side. The left VA was considered non-dominant because it was smaller than the right side and terminated in the PICA. Therefore, we attributed his vertebrobasilar ischemic symptoms to this anatomical variation, where the left VA occlusion could not be compensated by flow from the contralateral VA during head turning.

Frisoni et al pointed out a greater incidence of a smaller VA ending in PICA in bow hunter's stroke patients (8%) versus general population (< 1%)^(13,14). If the presence of this anatomical variation is known, chiropractic neck maneuvers are contraindicated.

Dynamic cerebral angiography is the established method of diagnosis. Axial contrast computed tomography is used to determine the relationship between the affected VA and the surrounding structures. Three-dimensional computed tomography angiography (CTA) and magnetic resonance angiography (MRA) proved rotational changes in the morphology of the VA. Single-photon emission computed tomography (SPECT) with 99m Tc-HMPAO could document decreased cerebral blood flow noted in the cerebellum and occipital cortex after head rotation⁽⁹⁾.

Dynamic ultrasound findings are characteristic and the diagnostic criteria are defined as end-diastolic velocity decline to zero in V2^(15,16), or marked fall in blood flow velocity (more than 50%) in V4, basilar artery or PCA after rotation followed by a transient increase in blood flow velocity (reactive hyperemic response) upon returning to neutral position^(12,17). In our case, the absence of end-diastolic velocity of V2 and the marked fall in velocity of V4 (more than 50% reduction) at left VA

were demonstrated after head rotation to the right side but not to left side. The dynamic ultrasonography of other intracranial vessels was unremarkable because this relationship was irrelevant to the anatomical variation.

Conservative treatment included neck immobilization, either by instructing patient to refrain from head turning or to restrict head turning mechanically by the use of a collar and careful spinal manipulative therapy^(5,18). Aspirin and anticoagulation had been used as in our patient^(3,19).

In summary, the anatomical variation of nondominant VA ending in PICA increases the risk of VBI (especially ipsilateral lateral medullary syndrome) and it needs further evaluation and management to prevent permanent neurological deficits. Dynamic ultrasonography is a simple, noninvasive method to document a hemodynamic etiology in patients with suspected rotational vertebrobasilar insufficiency.

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