ISSN 2005-7881
Journal of Neurosonology 6(1):16-19, 2014

CASE REPORT

보우-헌터 증후군 의심환자에서 경두개초음파검사로 확인한 추골동맥 순환부전증 1예

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Bedside Approach of Vertebral Artery Insufficiency by Transcranial Doppler Sonography in Clinically Suspected Bow Hunter's Syndrome

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ABSTRACT

Bow hunter's syndrome (BHS) is clinical manifestation of dynamic compromise of vertebral artery. Dynamic imaging with provocative digital subtraction angiography (DSA) is a preferred modality but non-invasive bed side screening tool for a dynamic compression of the vertebral artery is needed.A 56-year-old man developed cervicalgia and syncope while manipulating his neck. Cervical spine CT and MR angiography demonstrated osteophyte with uncovertebral hypertrophy at left C5/6 level and right vertebral artery hypoplasia. Transcranial doppler (TCD) sonography on midbasilar artery revealed sudden disappearance of basilar flow while the patient moved his neck extended and left twisted. The patient was instructed not to move his neck to provoking position and discharged without any medication. This is a report of bedside, non-invasive and early approach of clinically suspected BHS with TCD.

Journal of Neurosonology 6(1):16-19, 2014

Key Words: Bow hunter's syndrome, Transcranial doppler sonography, Osteophyte, Vertebrobasilar insufficiency

The vertebrobasilar system is composed of paired vertebral arteries, which may allow for compensation in the instance of unilateral disease. However, one of the vertebral arteries, more commonly the left, is often found to be significantly larger than the contralateral side and provides the majority of inflow to the posterior circulation. Bowhunter's syndrome (BHS) is the clinical manifestation of posterior circulation ischemia provoked by dynamic compromise of the dominant vertebral artery. Clinical symptoms are dizziness, nausea, presyncopal sensation, syncope, and even ischemic stroke. When BHS is suspected, dynamic imaging with provocative digital subtraction angiography (DSA) is the preferred modality for definitive diagnosis.[1] It demonstrates vertebral artery compromise secondary to compression of dominant or bilateral vertebral arteries.[2] But invasive technique with some complications can be a limitation. In this report we present a case

Received August 29, 2014 / Accepted December 30, 2014

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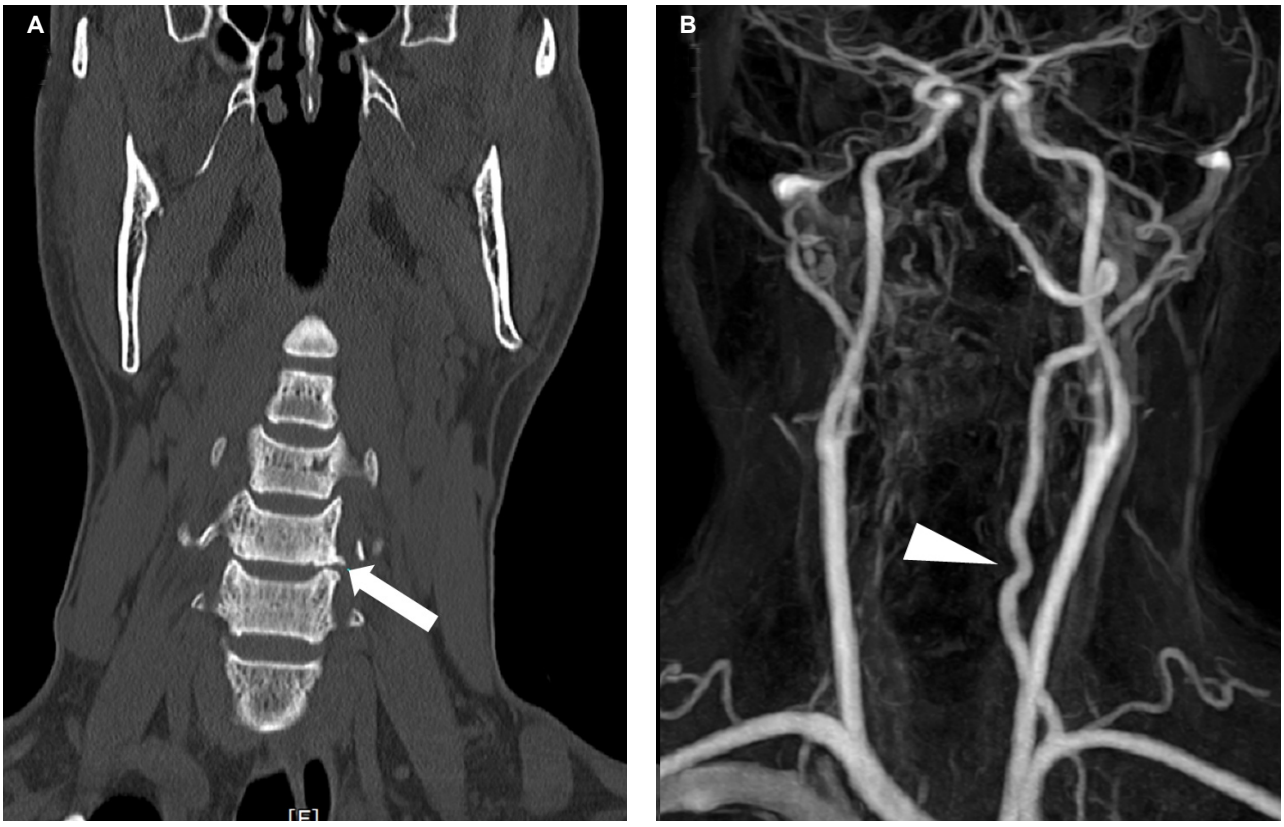

Fig. 1. (A) Osteophytes anduncovertebral hypertrophy at left side of C5/6 vertebra in cervical computed tomography coronal view (white arrow). (B) Laterally curved left vertebral artery at C5/6 level in magnetic resonance carotid angiography (arrow head).

of BHS diagnosed with transcranial Doppler (TCD) sonography.

CASE

A 56-year-old man without any medical and surgical history presented with symptoms of cervicalgia and paresthesia from his left shoulder to elbow and syncope while manipulating his neck. The patient reported no cervicalgia, dizziness or paresthesia while sitting in upright position. On physical examination, the patient was found to have positive Spurling's test on left side rotation of his neck.

Neurological examination, vestibular function tests (including caloric test, Frenzel goggle test), bilateral auditory evoked potential study, nerve conduction study, 3-position blood pressure test, Valsalva maneuver test, elecrocardiography, transthoracic echocardiography, and 24-hour holter monitoring were normal. Brain magnetic resonance image (MRI) and magnetic resonance angiography (MRA) of cerebral and cervical area revealed hypoplasia of right vertebral artery. Left vertebral artery and bilateral carotid arteries were normal. No signal changes were found in MR diffusion weighted imaging. Computed tomography scan of cervical spine demonstrated osteophytes with uncovertebral hypertrophy at left C5/6 vertebra (Fig. 1A), resulting lateral deviation on the left vertebral artery at same level (Fig. 1B). In support of these findings, the patient was suspected to have bow hunter's syndrome. Instead of conventional angiography, we used transcranal doppler sonography (ST3 Power M-mode Transcranial Doppler. Spencer Technologies, Redmond, WA, USA). After placing diagnostic probe to midbasilar artery in depth of 85mm, the patient was asked to move his neck to flex, extend, rotate, tilt and twist side to side. There was sudden disappearance of basilar flow and patient felt dizzy when he rotated and sustained his neck to left side (45 degrees extension and 70 degrees twist in detail) (Fig. 2). There was no positive finding in contralateral side. The patient was instructed not to move his neck to the typical position and discharged without any medication.

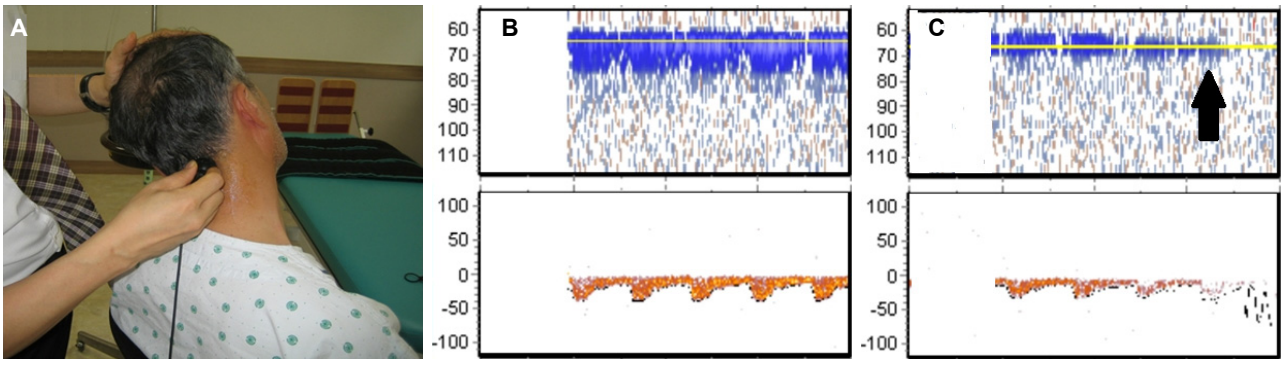

Fig. 2. Midbasilar artery flow rapidly disappeared while the patient rotated and sustained his neck to the left. (A) After placing transcranial doppler (TCD) probe at midbasilar artery, the patient is asked to rotate his neck to left side (45 degrees extension and 70 degrees rotation in detail). (B) Normal TCD waves of midbasilar artery. (C) Rapid disapperance of TCD waves while the patient rotates and sustained his neck (black arrow)

DISCUSSION

Bow hunter's syndrome (BHS) describes a positional vertebrobasilar arterial insufficiency in the setting of head rotation. The name originated from meaning of rotational position of the head that a bow hunter assumes when properly aiming his game with his bow. The association between head rotation and vertebrobasilar insufficiency had been suggested in 1933. The phenomenon was first suggested in cadaveric studies in 1952. The term "bow hunter's syndrome" was first used as "bow hunter's stroke" describing a patient who developed Wallenberg syndrome during archery practice in 1978.[3]

The vertebral artery occlusion occurs at the atlantoaxial level and also subaxial cervical spines. Physiologically, during normal physiologic head rotation, the vertebral artery at the atlantoaxial joint is progressively stretched between the two transverse foramina, resulting in narrowing and flow limitation of the vessel. When ligamentous bands and osteophyte develops, the stretching of the artery can be exaggerated.[4] The most common leading cause is hypertrophic osteophyte arising from the uncovertebral joints and other following causes are thickened atlantoaxial membrane or fibrous bands, ligamentous hypertrophy, disc herniation, cervical spondylosis, surgical fixation, chiropractic manipulation and rheumatoid arthritis.

Fleming et al.[5] reported all patients with lesions above C3 and accompanying V3 or V4 segment of vertebral artery had contralateral presentation, with contralateral vertebral artery compromised. In contrast, patients with lesions below C3 showed ipsilateral presentation. The authors assumed stretching as a mechanism of vertebral artery compromise above C3 and compression below C3. But there are still many controversies.[6] According to these findings, osteophyte of C5/6 as in our case can be considered as a cause of ipsilateral presentation even though limitation still remains without confirmatory conventional angiography.

Depending on the amount of compensatory flow, symptoms vary widely and include dizziness, nystagmus, nausea and vomiting, frank hemiparesis, sensory changes, Horner's syndrome, swallowing difficulties, and loss of consciousness. The compromised flow symptoms are commonly transient, but permanent deficits are still reported.

Conventionally, most patients present with baseline occlusion or hypoplasia of one vertebral artery and contralateral compression will result in insufficient perfusion. But there are rare cases with bilateral vertebral artery compression in the setting of normal neutral vascular imaging.[7] Other mimical syndrome is stylocarotid artery syndrome that is rare condition results from compression of the internal carotid artery by the elongated styloid process of ligament of the temporal bone.[8]

Digital subtraction angiography (DSA) is the most preferred method compared to ultrasonography, CT and MRA. DSA provides dynamic, simultaneous images of flow change and precise localization. Ultrasonographic study has relatively low sensitity compared to conventional angiography (36.4% versus 100%).[9] But conventional cerebral artery angiography has a few but critical complications such as air embolism, thromboembolism, dye allergic reaction, puncture site hematoma. There are about 1% overall incidence of neurologic deficit and 0.5% incidence of persistent deficit. Complication rate in-

creases with age and vessel procedural difficulty.[10] In consideration of increased cervical vertebral degeneration, vascular tortuosity in aged person and unilateral hypoplastic vertebral artery in bow hunter's syndrome, conventional vertebral artery angiography can be more risky compared to transcranial Doppler sonography. So in practical point of view, Iguchi et al.[11] and our report demonstrated TCD as a non-invasive and accurate bed side screening test for dynamic hemodynamic changes of vertebral artery.

Treatment options for this condition begin with conservative measures such as neck immobilization or a cautious effort not to rotate the head to the affecting side. Surgical procedure or contralateral side stent deployment are needed when it is symptomatic to the point of interfering with usual activity of daily life or conservative measures fail. In case of atlantoaxial instability, surgical fixation would be reasonable, whereas decompression is treatment of choice in vertebral artery compression cases.[5]

We admit there is no evidence of the real focus of dynamic vertebral artery flow change. So it would be advisable to use TCD as a bedside noninvasive tool for initial approach of clinically suspected bow hunter's syndrome before undergoing invasive study. This report highlights TCD as a safe and accurate screening test for a dynamic compression of the vertebral artery when patient present with syncope or stroke-like symptoms.

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