Bow Hunter's Stroke Associated with Atlantooccipital Assimilation

—Case Report—

Kyoji SAKAI and Takumi TSUTSUI

Department of Neurosurgery, Kochi Prefectural Aki Hospital, Aki, Kochi

Abstract

A 39-year-old male presented with bow hunter's stroke manifesting as repeated vertebrobasilar ischemic attacks induced by head rotation 45 degrees to the left. Three-dimensional computed tomography angiography clearly showed the occluded right vertebral artery (VA) between the axis and atlas. Single photon emission computed tomography study showed diffuse hypoperfusion of the brain stem and bilateral cerebellar hemispheres, suggesting hemodynamic compromise of these regions. He refused surgery and was treated conservatively. The most likely mechanism is that the affected VA was fixed by the ossification of the atlantooccipital membrane, vascular groove, and transverse foramen of the atlas, and therefore became elongated and compressed by head-turning.

Key words: bow hunter's stroke, vertebrobasilar insufficiency, single photon emission computed tomography

Introduction

Hemodynamic compromise of the vertebral artery (VA) induced by head rotation is an uncommon cause of vertebrobasilar insufficiency. Bow hunter's stroke results from vertebrobasilar insufficiency caused by mechanical occlusion or stenosis of the VA at the atlantoaxial level resulting from head rotation. Neurological symptoms seldom appear when the contralateral VA flow or distal vertebrobasilar system collateral flow from carotid circulation is sufficient. Here we present a case of bow hunter's stroke associated with atlantooccipital assimilation, in which cerebral blood flow (CBF) study clearly demonstrated diffuse hypoperfusion of the brain stem and bilateral cerebellar hemispheres.

Case Report

A 39-year-old male suffered from occasional episodes of unconsciousness when he rotated his head to the left. Neurological examination demonstrated no deficits with the head in the neutral position. Dizziness and nausea were reproducibly induced with his head rotated 30 degrees to the left, and

syncope was induced with his head rotated 45 degrees or more to the left. No nystagmus was seen. The symptoms disappeared rapidly after his head was returned to the neutral position.

Routine laboratory data including blood count, urinalysis, and chemistry showed only hyperlipidemia. Skull radiographs showed atlantooccipital assimilation (Fig. 1). Cervical spine radiographs revealed no narrowing or severe bony proliferative changes at the vertebral interspaces, nor any other abnormal findings such as atlantoaxial dislocation. Magnetic resonance (MR) imaging revealed no abnormality in the brain. Phase-contrast MR angiography demonstrated the dominant right VA and the hypoplastic left VA (Fig. 2).

Cerebral angiography with the head in the neutral position showed that the left VA terminated at the fourth segment (Fig. 3 left), whereas the normal right VA of moderate size supplied the territory of the distal end of the left VA (Fig. 3 center). Neither vertebral or carotid angiography visualized the bilateral posterior communicating arteries. Therefore, collateral flow to the posterior circulation from the carotid circulation seemed to be insufficient. Dynamic vertebral angiography with his head rotated 45 degrees to the left showed the right VA was occluded between the axis and atlas (Fig. 3 right).

Fig. 1 Lateral skull radiograph showing atlantooccipital assimilation.

Three-dimensional computed tomography (3D-CT) angiography with his head rotated to the left clearly showed the right VA occluded between the axis and atlas (Fig. 4).

CBF study using 740 MBq of technetium-99m-

Fig. 2 Phase-contrast magnetic resonance angiogram showing the dominant right vertebral artery (VA) and the hypoplastic left VA.

hexamethylpropyleneamine oxime with the patient's head rotated 45 degrees to the left showed diffuse hypoperfusion of the brain stem and bilateral cerebellar hemispheres suggesting the presence of hemodynamic compromise (Fig. 5).

The patient was treated conservatively with the administration of 81 mg of aspirin per day because

Fig. 3 *left:* Left vertebral angiogram with the head in the neutral position showing termination of the left vertebral artery (VA) at the fourth segment. *center:* Right vertebral angiogram with the head in the neutral position showing the normal right VA of moderate size. *right:* Right vertebral angiogram showing the right VA is completely occluded at the atlantoaxial level when the head is rotated 45 degrees to the left.

Fig. 4 Three-dimensional computed tomography angiogram with the head rotated 45 degrees to the left showing the occluded right vertebral artery (arrow) between the axis (C-2) and atlas (C-1). C-0: occipital bone.

Fig. 5 Single photon emission computed tomography scans with the head rotated 45 degrees to the left showing diffuse hypoperfusion of the brain stem and bilateral cerebellar hemispheres.

he refused any surgical intervention.

Discussion

Extracranial narrowing or occlusion of the VA resulting from head-turning may occur a) at the first segment of the VA originating from the subclavian

artery and entering into the transverse C-6 foramen, b) at the second segment of the VA passing through the C-5 to C-2 transverse foramina, and c) at the atlantoaxial level. Powers' syndrome is rotational VA compromise at the first segment resulting from constriction by the longus colli and scalenus muscles. It is not clear whether the affected VA is ipsilateral or contralateral to the side of neck rotation.^{4,7,8,12} Spondylotic vertebrobasilar insufficiency occurs at the middle and lower cervical levels due to compression by cervical osteophytes. Usually, the affected VA is ipsilateral to the side of neck rotation.¹⁵ Bow hunter's stroke is caused by narrowing or occlusion of a dominant VA between the axis and atlas with head rotation. The affected VA is contralateral to the side of neck rotation.¹ In the present case, phase-contrast MR angiography revealed the dominant right VA and the hypoplastic left VA, and ischemic symptoms repeatedly occurred when the patient rotated the head to the left. Skull radiography showed atlantooccipital assimilation, and cervical spine radiography showed no severe changes of spondylosis or spinal instability. For this reason, bow hunter's stroke is the most likely diagnosis. Phase-contrast MR angiography, a less invasive examination performed prior to the dynamic vertebral angiography with head rotation, was very useful for identifying the dominant VA.

Bow hunter's stroke was named after a patient who suffered vertebrobasilar insufficiency when he practiced archery.¹⁶ This clinical entity can be divided into three types of etiologies.^{17,18} Patients with type 1 present with several types of VA injuries resulting from chiropractic neck manipulations,¹³ bow hunting,¹⁶ painting,¹¹ swimming,¹⁹ or spontaneous head-turning.^{1,5,13} Repeated neck hyperextension and hyperrotation movements sometimes cause intimal damage, mural dissection, vascular spasm, and subsequent thrombo-embolism. Cerebral angiography shows narrowing or occlusion of the VA at the atlantoaxial level even in the neutral head position. Patients with type 2 have associated rheumatoid atlantoaxial dislocation or rotational subluxation of a facet that may compress the VA and cause vertebrobasilar insufficiency.³ Patients with type 3 suffer repeated ischemic attacks following voluntary neck rotation. Patients always complain of occasional vertigo, dizziness, or syncope on rotation of the head within the usual range. These symptoms disappear immediately after the head is returned to the neutral position. Dynamic cerebral angiography demonstrates that head rotation induces stenosis or occlusion of the VA contralateral to the side of neck rotation. Usually the opposite VA is hypoplastic, stenotic, or occluded.

Many developmental and degenerative factors contribute to rotational occlusion, such as atherosclerosis and kinking of the VA, stenosis of transverse foramen, and the depth of the vascular groove of the atlas, hypertrophy of the atlantooccipital membrane, fibrous band, and tightness of the paravertebral muscles.^{1,4,6,9,14,17,18)} The most likely mechanism in the present case of bow hunter's stroke with atlantooccipital assimilation is that the affected VA was fixed by the ossification of the atlantooccipital membrane, vascular groove, and transverse foramen of the atlas, and therefore became elongated and compressed by head-turning.

Thirty cases of bow hunter's stroke type 3 have been reported since 1985.^{2-4,6,9,10,14,15,17,21)} Including our case of type 3, there were 17 males and 14 females, aged from 37 to 75 years (mean 58.3 years). Dynamic vertebral angiography showed rotational VA occlusion or narrowing at the atlantoaxial level occurred in 11 right VAs and 20 left VAs.

3D-CT angiography and single photon emission computed tomography studies of six cases of bow hunter's stroke clearly revealed stretched and occluded VA at the atlantoaxial level and the presence of hemodynamic compromise because of head-turning.^{9,10)} Decreased CBF was detected in the lower portion or most of the unilateral cerebellar hemisphere contralateral to the side of neck rotation. However, the CBF study in our case showed very unusual diffuse hypoperfusion of the brain stem and bilateral cerebellar hemispheres.

Treatment for bow hunter's stroke can be divided into conservative and surgical therapies. Conservative therapy includes a verbal warning and/or neck brace to prevent neck rotation to the affected side and the administration of antiplatelet agents. Surgical intervention includes posterior fusion of C1-2^{3,4,9,10,21)} and decompression of the affected VA at the atlantoaxial level by transversectomy.^{2,4,6,9,14,17)} Twelve of 30 patients with bow hunter's stroke type 3 underwent posterior fusion, and 17 underwent the transversectomy. Posterior fusion provides certain and complete relief of preoperative symptoms, but markedly reduces the range of head rotation. The reduction of head rotation was approximately 60% compared to the preoperative range.⁹⁾

Major axial rotation occurs between C-1 and C-2 in the occipital-atlanto-axial (C-0-C-1-C-2) complex. One side axial rotation between C-0 and C-1 is 3 to 8 degrees, and unilateral C-1-C-2 axial rotation is 34 to 41.5 degrees. Therefore, the C-0-C-1-C-2 joints can rotate approximately 45 degrees to one side. Thus approximately 60% of the axial rotation of the entire cervical spine and occiput occurs in the C-0-C-1-C-2 complex.²⁰⁾ In contrast, VA decompression by

transversectomy does not limit physiological neck movements. However, three of nine patients treated with transversectomy experienced reocclusion of the affected VA with head rotation several months postsurgery. One patient suffered from cerebellar infarction postoperatively.⁹⁾

Posterior fusion seems to be preferable to the transversectomy to be certain of avoiding postoperative reocclusion of the affected VA. Our patient had associated with atlantooccipital assimilation, so required, but did not accept, posterior fixation from the occiput to the upper cervical spine. However, this procedure could cause severe restriction of head rotation.

Acknowledgments

We would like to thank Mr. Thomas D. Bauer for editorial assistance.

References

- 1) Barton JW, Margolis MT: Rotational obstruction of the vertebral artery at the atlantoaxial joint. *Neuroradiology* 9: 117-120, 1975
- 2) Fox MW, Piepgras DG, Bartleson JD: Anterolateral decompression of the atlantoaxial vertebral artery for symptomatic positional occlusion of the vertebral artery. Case report. *J Neurosurg* 83: 737-740, 1995
- 3) Fujimoto S, Yoshino K, Itoh T, Terai Y: [Rotational stenosis of the vertebral artery at the atlantoaxial joint: report of three cases]. *No Shinkei Geka* 17: 861-865, 1989 (Jpn, with Eng abstract)
- 4) Fujita T, Ohnishi H, Goda K, Yamada T: [Vertebrobasilar insufficiency due to mechanical occlusion of extracranial vertebral artery]. *No Socchu No Geka* 20: 143-149, 1992 (Jpn, with Eng abstract)
- 5) Grossmann RI, Davis KR: Positional occlusion of the vertebral artery: a rare cause of embolic stroke. *Neuroradiology* 23: 227-230, 1982
- 6) Hanakita J, Miyake H, Nagayasu S, Nishi S, Suzuki T: Angiographic examination and surgical treatment of bow hunter's stroke. *Neurosurgery* 23: 228-232, 1988
- 7) Kojima N, Tamaki N, Fujita K, Matsumoto S: Vertebral artery occlusion at the narrowed "scaleno-vertebral angle." Mechanical vertebral occlusion in the distal first portion. *Neurosurgery* 16: 672-674, 1985
- 8) Mapstone T, Spetzler RF: Vertebrobasilar insufficiency secondary to vertebral artery occlusion from a fibrous band. *J Neurosurg* 56: 581-583, 1982
- 9) Matsuyama T, Morimoto T, Sakaki T: Comparison of C1-2 posterior fusion and decompression of the vertebral artery in the treatment of bow hunter's stroke. *J Neurosurg* 86: 619-623, 1997
- 10) Matsuyama T, Morimoto T, Sakaki T: Bow hunter's stroke caused by a nondominant vertebral artery occlusion: case report. *Neurosurgery* 41: 1393-1395, 1997

- 11) Okawara S, Nibbelink D: Vertebral artery occlusion following hyperextension and rotation of the head. *Stroke* 5: 640-642, 1974
- 12) Powers SR, Drislane TM, Nevins S: Intermittent vertebral artery compression. A new syndrome. *Surgery* 49: 257-264, 1961
- 13) Sherman DG, Hart RG, Easton JD: Abrupt change in head position and cerebral infarction. *Stroke* 12: 2-6, 1981
- 14) Shimizu T, Waga S, Kojima T, Niwa S: Decompression of the vertebral artery for bow-hunter's stroke. Case report. *J Neurosurg* 69: 127-131, 1988
- 15) Shinohara N, Kohno K, Takeda S, Ohta S, Sakaki S: [A case of bow hunter's stroke caused by bilateral vertebral artery occlusive change on head rotation to the right]. *No Shinkei Geka* 26: 417-422, 1998 (Jpn, with Eng abstract)
- 16) Sorensen BF: Bow hunter's stroke. *Neurosurgery* 2: 259-261, 1978
- 17) Takahashi I, Kaneko S, Asaoka K, Harada T: [A case of bow hunter's stroke]. *No To Shinkei* 46: 183-187, 1994 (Jpn, with Eng abstract)
- 18) Takahashi I, Kaneko S, Asaoka K, Harada T: [Angiographic examination of the vertebral artery at the atlantoaxial joint during head rotation]. *No Shinkei Geka* 22: 749-753, 1994 (Jpn, with Eng abstract)
- 19) Tramo MJ, Hainline B, Petito F, Lee B, Caonna J: Vertebral artery injury and cerebellar stroke while swimming: case report. *Stroke* 16: 1039-1042, 1985
- 20) White A, Panjabi M: *Clinical Biomechanics of the Spine*, ed 2. Philadelphia, JB Lippincott, 1990, pp 92-94
- 21) Yang PJ, Latack JT, Gabrielsen TO, Knake JE, Gebarski SS, Chandler WF: Rotational vertebral artery occlusion at C1-C2. *AJNR Am J Neuroradiol* 6: 98-100, 1985

Address reprint requests to: K. Sakai, M.D., Department of Neurosurgery, Kochi Prefectural Aki Hospital, 1-32 Hoei-cho, Aki, Kochi 784-0027, Japan.