Bow Hunter’s Stroke Caused by a Severe Facet Hypertrophy of C1-2

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INTRODUCTION

Head and neck rotation can cause mechanical stenosis or obstruction of vertebral artery (VA). This rare and unique symptomatic vertebrobasilar insufficiency or infarction is named “bow hunter’s stroke” after the first description by Sorensen1). Various pathologic conditions have been reported as causes of bow hunter’s stroke. We report a 71-year-old woman with rotatory occlusion of VA from rare massive facet hypertrophy at the C1-2 level treated by C1-2 posterior fixation and fusion.

CASE REPORT

A 71-year-old woman presented with a 3-year history of vertigo, hypoesthesia of left toes and occasional memory loss, with rotation of head to the right side. She had symptoms reversed by returning her head to a neutral position. A physical and neurological examination was normal. Plain X-rays and magnetic resonant imaging studies demonstrated cervical spondylosis. Contrast-enhanced computed tomography showed massive facet arthropathy. Various pathologic conditions have been reported as causes of bow hunter's stroke. We report a 71-year-old woman with rotatory occlusion of VA from rare massive facet hypertrophy at the C1-2 level treated by C1-2 posterior fixation and fusion.
and trajectory was oriented using anatomical landmarks, as described by Harms and Melcher\(^7\). A contoured horse-shoe shaped rod was secured to the C-1 arch using sublaminar cables. The rod was placed into the polyaxial screw heads and secured in position. Lateral arthrodesis was performed by decortication the exposed surfaces of the C1-2 joints with a high-speed drill and then packing cancellous iliac crest autograft over these joints (Fig. 3). The postoperative course was uneventful. The patient was neurologically completely asymptomatic in follow-up. Head rotation was reduced by approximately 30% compared to her preoperative status.

**DISCUSSION**

Bow hunter’s stroke is a symptomatic vertebrobasilar insufficiency caused by stenosis or occlusion of the VA with head rotation\(^12\). It is a common finding on angiography that head rotation produces stenosis or occlusion of a contralateral VA. However, temporary positional occlusion of one VA during daily activities rarely produces major effects on posterior circulation blood flow. In most reported cases, patients with ischemic attacks induced by rotational occlusion of one VA had an opposite VA that was hypoplastic, stenotic, or occluded. The VAs at the atlantoaxial level are particularly prone to mechanical compression during head and neck rotation because of its unique relationship to the surrounding transverse foramina, paravertebral muscles, and fibrous ligaments\(^5,6\). Axial rotation at the C1-2 level may cause VA occlusion within normal range of motion due to the relatively fixed position of the artery within the posterior arch of the atlas\(^5,14\). Head rotation to the right results in fixation of the right atlantoaxial joint, while the atlas moves forward on the axis on the left side. The segment of vertebral artery between C-1 and C-2 can be narrowed or occluded in the process\(^5\). Atlantoaxial instability, ossification or hypertrophy of the atlantooccipital membrane, tightness of the paravertebral musculature, or severe changes of spondylosis may also contribute to vertebral artery compression\(^3,5,6,8,13,14\). The second most site of VA compromise is its entrance into the C6 transverse foramen\(^8\). This is attributed to the VA being anchored by fibrous band just before its entrance into the transverse foramen and head rotation leads to stenosis of the VA at the tethered C6 level\(^10\). Rotational stenosis of the VA at its second cervical segment (from C3 to C6) is very rare and osteophyte formation seems to play major role in these cases\(^10\).

Treatment alternatives for rotational compression of the atlantoaxial portion of the VA have included verbal warn-
ings or braces to restrict head and neck rotation, surgical fusions to prevent atlantoaxial rotation, and decompression of the VA at the C1-2 level. Decompression of the VA at the C1-2 level by either anterior or posterior approach has been used for affected patients. Matsuyama et al. reported results for nine patients of bow hunter’s stroke treated with posterior decompression of VA. However, in three of these patients, recollection of the operated VA was recognized after surgery and two of them had more severe neurologic symptoms than preoperatively. Fox et al. reported spinal accessory nerve palsy as a complication of the anterolateral decompressive approach. The risks of direct surgical decompression of VA in the face of structural abnormalities such as massive facet hypertrophy in the present case are significant. Inadvertent vertebral artery injury during direct decompression could also have caused new neurological deficit or posterior fossa stroke. Posterior fixation of C1-2 to eliminate vertebral artery rotatory occlusion is a reasonable option in these patients.

CONCLUSION

We report the rare case of a bow hunter’s stroke from facet hypertrophy at the C1-2 level. C1-2 posterior fusion is a reasonable option considering significant risks of direct surgical decompression of VA in this case.

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