A CASE OF BOW HUNTER’S STROKE PRESENTING SYNCOPE AS AN INITIAL SYMPTOM WHILE DRIVING

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Abstract: Bow Hunter’s Stroke is a consequence of vertebrobasilar insufficiency as a result of mechanical occlusion or stenosis of the vertebral artery at the C1-C2 level by head rotation. It is rarely symptomatic in daily activities. We describe a case of Bow Hunter’s Stroke (BHS) presenting syncope as an initial symptom while driving a car.

A 59-year-old male patient suddenly lost consciousness while driving and his car dropped into a ravine 20 meters deep. On admission he was conscious, but suffering Central Cord Syndrome (CCS).

We examined the cause of syncope. At the time of the accident, he turned his head to the rear in order to back his car and lost consciousness. Magnetic resonance angiography demonstrated the dominant vertebral artery (VA) in the left and the hypoplastic VA in the right. Cerebral angiography on turning the head 90 degrees to the right revealed the left VA occluded at the C1-C2 level. Therefore we diagnosed the patient with BHS. The vertigo symptom was intractable with conservative treatment, and we therefore performed C1-C2 posterior fusion. The post-operative course was uneventful and he does not have the symptoms anymore.

Syncope sometimes induces traffic accidents, but it is rare that BHS is detected on examination of common trauma. Therefore when vertigo, dizziness, or syncope is found in common trauma, BHS must be considered as a potential cause.

Key words: Bow Hunter’s Stroke, syncope, traffic accident, posterior fusion

INTRODUCTION

There are various causes of traffic accidents and some cases result from the occurrence of endogenous disease. On examination of the causes of traffic accidents, we sometimes can’t discover exactly when the patient hit his head and fell unconscious. We describe a case of
Bow Hunter’s Stroke (BHS) which was detected with a traffic accident as the opportunity.

CASE REPORT

A 59-year-old male patient, who had had lumbar spondylolisthesis, suddenly lost consciousness while driving. His car dropped into a ravine 20 meters deep and he was admitted to our emergency trauma center. On admission he was conscious, but complained of pain and numbness in the distal part of his upper extremities and a weakened grasping strength. His deep tendon reflex was weakened in the upper extremities. His lumbar spondylolisthesis changed for the worse, numbness in the left lower extremity grew severely, and his sense of touch was weakened. Cervical magnetic resonance imaging (MRI) revealed spondylosis with osteophyte at the C4-5 level. Considering his symptoms and the radiological findings, we diagnosed him with Central Cord Syndrome (CCS). CCS symptoms disappeared after conservative therapy.

We next examined the cause of syncope. Before the traffic accident, vertigo and faintness had occurred when the patient looked over his right shoulder. At the time of the accident he turned his head to the rear in order to back his car and lost consciousness. Magnetic resonance angiography (MRA) demonstrated the dominant left Vertebral Artery (VA) and the hypoplastic right VA (Fig. 1). Cerebral angiography with the head in the neutral position revealed sufficient left VA blood flow. But on turning the head 90 degrees to the right under sedation, the left VA was occluded at the C1–C2 level (Fig. 2). Therefore we diagnosed the case as BHS. After conservative therapy with a neck brace and antiplatelet drugs, the patient

Fig.1. Magnetic resonance angiogram showing the dominant left vertebral artery (VA) and the hypoplastic right VA.
Fig. 2. a): Left vertebral angiogram with the neck in a neutral position showing no abnormal findings.
b): Left vertebral angiogram showing occlusion in the third segment of the vertebral artery when the neck is fully rotated to the right (about 90°).

Fig. 3. Lateral X-ray demonstrates posterior cervical fusion with titanium instrumentation and iliac bone graft.
was discharged. But he continued to experience vertigo and faintness, and we therefore performed C1-C2 posterior fusion with titanium instrumentation and the iliac bone graft (Fig. 3). The post-operative course was uneventful and he was discharged. The patient’s lifestyle was not disturbed by head rotation, and he does not have the symptoms he had preoperatively.

DISCUSSION

In the emergency center, CCS is sometimes found but it is rare that BHS is detected on examination of common trauma. We described a case of Bow Hunter’s Stroke, whose syncope episode led to a motor vehicle crash with resultant CCS.

BHS is a symptomatic vertebrobasilar insufficiency caused by stenosis or occlusion of the VA with head rotation. In most cases, it is caused by occlusion of a dominant VA due to mechanical stretching or compression and dislodgement between C1–2 with head rotation. It is rarely symptomatic in daily activities, but if unilateral VA blood flow is poor or absent, vertebrobasilar ischemic attacks will be induced during head and neck rotation.

Patients always complain of occasional vertigo, dizziness or syncope on rotation of the head, but these symptoms disappear immediately after the head returns 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.

In the present case, MRA revealed the hypoplastic right VA and the dominant left VA, and ischemic symptoms repeatedly occurred when the patient rotated the head to the right, making BHS the most likely diagnosis.

Treatments for BHS can be divided into conservative and surgical therapies. Conservative therapy includes verbal warnings or use of a neck brace to limit head and neck rotation and the administration of antiplatelet agents. Surgical treatment may be indicated to preclude life-threatening accidents or when patients complain that their lifestyle is restricted by conservative treatments.

There are several different surgical procedures, including posterior fixation of C1-C2 and decompression of VA at the atlantoaxial level by transversecotomy. Posterior fusion provides complete relief of preoperative symptoms, but markedly reduces the range of head rotation. However, patients with C1–2 posterior fusion do not always complain of inconvenience in daily life. In contrast, VA decompression by transversecotomy does not limit physiological neck movements, although postoperative reocclusion has been reported.

We performed posterior fixation of C1-C2 using titanium instruments with iliac bone graft in the present case. Physiological rotation of the head is sacrificed by this procedure to some extent, but life-threatening neurological sequelae can be eliminated.

Syncope sometimes induces traffic accidents, and in this case, the patient’s syncope episode led to a motor vehicle crash with resultant CCS. With continuously closer examination of the cause of syncope, we could diagnose BHS, and posterior fusion allowed the patient to become symptom-free. Therefore when vertigo, dizziness, or syncope is found in the examination of common trauma, we should pay careful attention to endogeneous disease, including BHS.
A case of Bow Hunter’s Stroke

disease, including BHS.

REFERENCES


