



# Recurrent vertebrobasilar insufficiency—bow hunter's syndrome

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**Abstract:** Vertebrobasilar insufficiency manifests with vertigo, tinnitus, bulbar weakness and gait ataxia. Rotational compression of vertebral artery can occur at its atlantoaxial or subaxial segment. Bow hunter's syndrome or rotational vertebrobasilar insufficiency is a rare condition with potential to cause posterior circulation infarction. We report a case of a 52-year-old male patient who presented with sudden onset vertigo, vomiting and gait ataxia. His magnetic resonance imaging (MRI) brain showed acute infarction in right cerebellar vermis with multiple gliotic areas in left cerebellar hemisphere suggestive of recurrent posterior circulation infarction. Dynamic MR angiography showed rotational compression of vertebral artery.

**Keywords:** Bow hunter's syndrome; rotational vertebrobasilar insufficiency; dynamic angiography

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## Introduction

Bow hunter's syndrome or rotational vertebrobasilar insufficiency syndrome was first described by Sorensen in 1978 in a patient who developed lateral medullary syndrome during archery practice (1). The rarity of this condition is exemplified by the fact that it has been described in the form of case reports and few case series. Jost and Dailey as well as Rastogi *et al.* have carried out systematic review of literature providing information regarding the characteristics of this condition (2,3).

The paired vertebral arteries are the first and largest branches of the subclavian arteries. They ascend through the transverse processes of the upper six cervical vertebrae, pass behind the lateral masses of the atlas, enter the dura mater behind the occipital condyles. It winds behind the superior articular process of the atlas and enters the cranium through the foramen magnum where it unites with the opposite vertebral artery to form the basilar artery (at the lower border of the pons) (4). This course through soft tissue and bony canal subjects the artery to strain caused by neck movements.

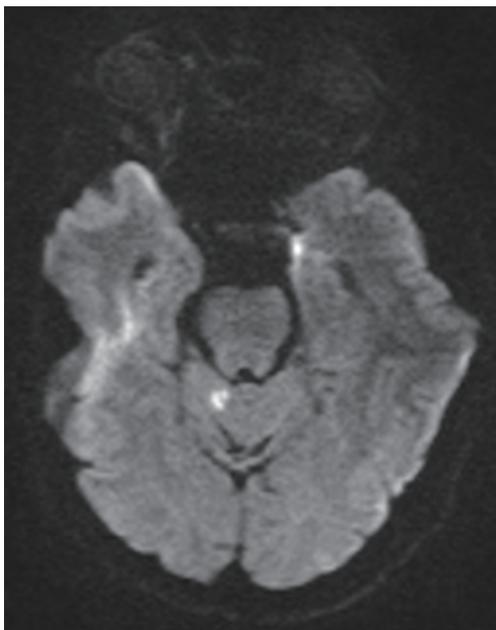
Bow hunter's syndrome is a clinical manifestation arising due to dynamic compression of vertebral arteries during

extension and rotational neck movement. The underlying pathology is usually a herniated disc, degenerative conditions such as spondylosis, an osteophyte or thickened ligament (5-9). At the level of atlantoaxial joint, the lesion may be caused by an ossified or thickened atlanto-occipital membrane, a dural fold in the foramen magnum, occipitalised atlas, an accessory ossicle behind the atlanto-odontoid junction, erosive rheumatoid arthritis of C1-C2 or C1-C2 facet hypertrophy (10-14). At the subaxial level compression is caused by a bony spur or herniated disc (15).

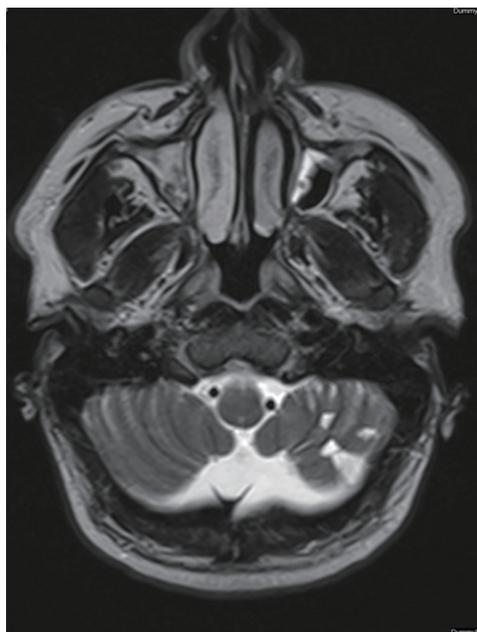
The underlying pathogenesis is due to endothelial damage by repetitive shear stress or stasis of blood leading to increased propensity for thromboembolism (16).

## Case presentation

A 52-year-old male patient presented to emergency department with complaint of sudden onset giddiness, nausea, vomiting, slurring of speech and imbalance of gait. On examination, he was conscious and vitals were normal. His higher mental functions like attention, executive function, judgement, language and praxis did not show any impairment. He had dysarthria and right sided gaze evoked nystagmus. Examinations of cranial nerves did not



**Figure 1** Diffusion-weighted imaging (DWI) showing diffusion restriction in right cerebellar vermis suggestive of acute infarction.



**Figure 2** T2 weighted image showing areas of hyperintensity in left cerebellar hemisphere suggestive of chronic ischemic changes.

show any deficit. Motor system examination was normal. There was no sensory deficit. He had intention tremor and impaired finger—nose test in right upper limb. Gait was



**Figure 3** T2 weighted sagittal images showing occipitalised atlas with basilar indentation in extension.

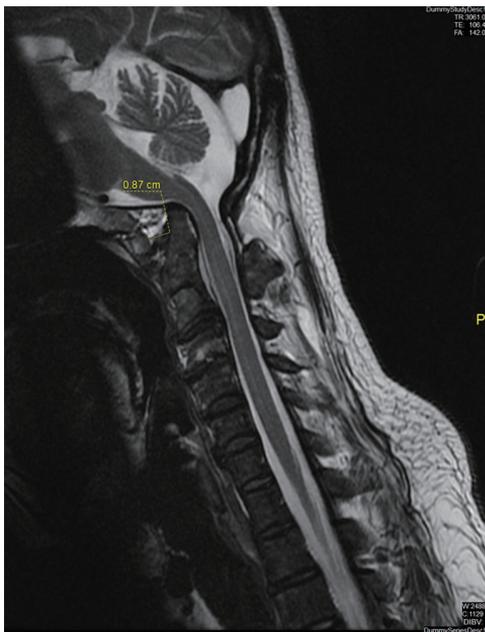
severely ataxic with tendency to fall on either side. There was no history of headache, trauma to head, fever, loss of consciousness, neck pain or seizure.

Magnetic resonance imaging (MRI) brain showed diffusion restriction in right cerebellar vermis suggestive of acute infarction (*Figure 1*). There were foci of gliosis in left cerebellar hemisphere (*Figure 2*). He was diagnosed as a case of ischemic stroke involving posterior circulation and treated with oral antiplatelet and statin. He was advised gait training exercises and rehabilitation for ataxia. Magnetic resonance angiography of neck and brain vessels did not show any vessel stenosis. He was evaluated for cardiac causes of stroke. Thrombophilia work up was negative.

In view of chronic left cerebellar infarcts evident on MRI, the case history was reviewed with the patient. He described intermittent episodes of dizziness and nausea which were self-limiting. He was unclear on the factors that had precipitated these symptoms. Review of MRI images showed presence of craniovertebral junction anomaly with occipitalised atlas and mild basilar invagination indenting the cervicomedullary junction (*Figures 3 and 4*).

Dynamic magnetic resonance angiography was done which showed compressive occlusion of right vertebral artery on extending the neck (*Figure 5*).

After review of case history, a diagnosis of vertebrobasilar

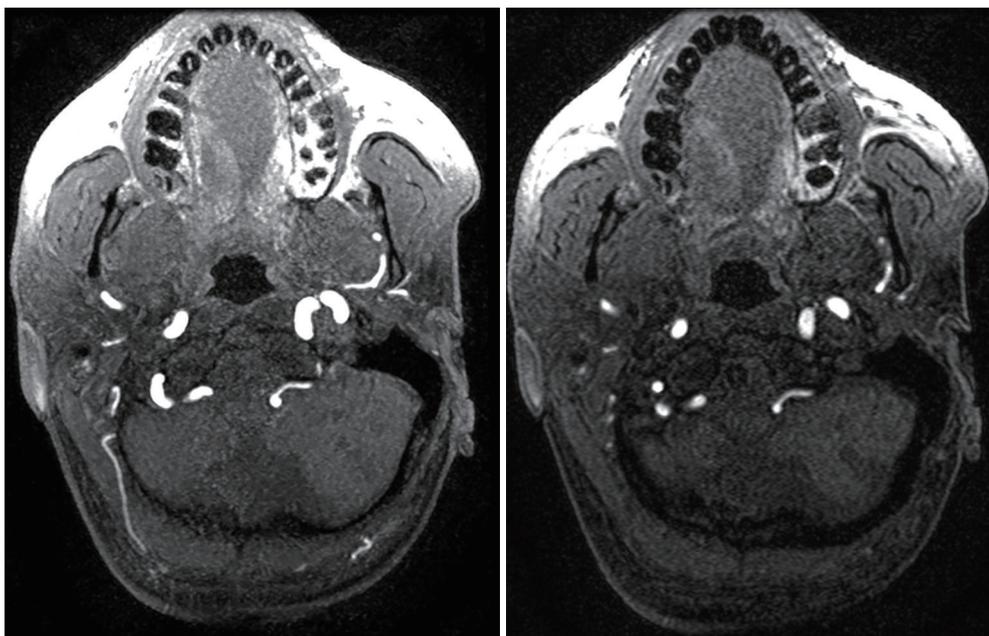


**Figure 4** T2 weighted sagittal images showing flexion position of neck.

insufficiency (bow hunter’s syndrome) was made. Neurosurgical opinion was obtained. The potential risk of recurrent posterior circulation stroke was discussed with the patient in detail. He was provided information and counseling regarding the available treatment modalities. He chose to be on conservative management and deferred any kind of intervention at present.

**Discussion**

Jost and Dailey reviewed 128 patients of bow hunter syndrome and determined that the occlusion was subaxial (between C3 and C7) in 58% cases, 36 % at C1-C2 level and in 6% cases proximal to C7 and distal to C1. The mean age at presentation was 58±11 year with male predominance (54% vs. 46%) (2). According to review by Rastogi *et al.*, the mean age at presentation was 53 years and the level of the atlantoaxial (C1-C2) joint was most commonly involved (99/142) (3). In our case, the dynamic compression was at the level of atlantoaxial joint and symptoms were



**Figure 5** Gadolinium enhanced magnetic resonance angiography showing compressive occlusion of vertebral artery by occipitalised atlas vertebrae.

precipitated with neck extension.

In these had dominant vertebral artery compression at the atlantoaxial or subaxial level with the non-dominant vessel being hypoplastic, stenotic or occlusive by arteriosclerosis. The posterior communicating arteries are generally hypoplastic or aplastic leading to poor communication with circle of Willis and inadequate collateral blood flow from anterior circulation.

Husni and Storer reported 23 patients with vertebrobasilar insufficiency induced by the rotational occlusion of one vertebral artery with the opposite artery being hypoplastic or absent in 22 patients and narrowed at its origin in 1 patient (17).

Repeated sheering forces and compression by external compression like that by a bony spur can lead to intimal and/or medial tear and increased platelet aggregation. Arterial dissection and thrombus formation are prominent causes of artery to artery embolism leading to this syndrome (18,19). There was no evidence of dissection or thrombosis in our patient.

Clinical manifestation consists of a spectrum ranging from transient ischemic attacks (TIA) to posterior circulation stroke. Symptoms can be repeatedly precipitated by rotation or extension of the head and neck and resolve immediately as the head returns to a neutral position. The classic presentations of this syndrome were summarized by Velat *et al.* include dizziness, vertigo, nystagmus, nausea with associated emesis, Horner's syndrome, syncope, and motor or sensory deficits that occur with head rotation (15).

Diagnostic evaluation includes computed tomography (CT) and MRI to visualize the bony and soft tissue structures leading to vascular compression. CT angiography/MR angiography sequences are required to characterize the vascular occlusion. The gold standard diagnostic test is digital subtraction angiography (DSA). It evaluates vessel patency in neutral and in the rotated position and thus supplements diagnosis, localization and planning of treatment (20).

There are still no standard treatment options for bow hunter syndrome owing to the rarity of this condition (16). In their study, Choi *et al.* analyzed the outcome of patients diagnosed with dynamic compression of vertebral artery and managed with conservative measures. The median follow-up period was 37.5 months with 2 patients out of 21 treated with surgical fusion of C1-C2 vertebrae (21). The remaining 19 patients did not show any fresh deficit during the period of follow up. This study led to conclusion that conservative treatment protocol might be safe and

might be considered as a first-line treatment modality. In other studies patients who were treated conservatively with medication may require surgery because of repeated, aggravated symptoms (22).

Surgical treatments, often involving surgical decompression of the VA or C1-C2 fusion, were performed in the majority of cases resulting from abnormal bony structures, disc herniation, instability of the joint and others. in the setting of contralateral VA stenosis, angioplasty with or without stent placement of the stenotic portion will provide a sufficient effect (23).

## Conclusions

Recurrent vertebrobasilar insufficiency or bow hunter's syndrome presents with recurrent symptoms like vertigo, tinnitus and imbalance. Many cases are treated as inner ear disease. Though the incidence of this condition is rare, it can potentially cause infarction and irreversible brain injury. Possibility of this condition should be considered in patients with recurrent symptoms of posterior circulation.

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None.

## Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

*Informed Consent:* We confirm that a written informed consent was taken from the patient for publication of this case report and images.

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