Rotational vertebrobasilar ischemia due to vertebral artery dynamic stenoses complicated by an ostial atherosclerotic stenosis

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Abstract: We describe a patient with rotational vertebrobasilar ischemia (RVBI) due to vertebral artery (VA) compressive stenoses during neck rotation, complicated by an ostial atherosclerotic stenosis (OAS). Referred for ‘near-syncopal spells’, inquiry revealed a symptom-complex consistent with vertebrobasilar transient ischemic attacks (TIAs) provoked by head rotation. VA dynamic angiography with imaging via prevertebral subclavian injections in neck-rotated positions while reproducing symptoms, demonstrated two compressive stenoses not present in the neck-neutral position, establishing the diagnosis of RVBI due to CT-demonstrated cervical spondylosis. There was an occluded contralateral VA, isolated posterior circulation, and absent vertebral collateral flow. Disabling symptoms persisted despite using a cervical collar. Surgical decompression of the dynamic stenoses would not address the OAS, was considered high risk, and absence of a suitable donor artery precluded distal VA reconstruction. RVBI resolved with ostial stent placement by improving perfusion pressure across the compressive stenoses. To our knowledge, this is the first report of RVBI in which the affected VA had an obstructive atherosclerotic stenosis in addition to the characteristic rotation-induced dynamic stenoses, and the first report of stent placement in the culprit artery to treat this disorder. Diagnosis depends on recognizing the association of symptoms with positional neck changes and VA dynamic angiography demonstrating the compressive stenosis while reproducing symptoms. This case illustrates the management complexities when there are coexisting abnormalities, emphasizing the need to individualize treatment. RVBI is a potentially correctable cause of TIAs and particularly relevant due to the aging population which has a significant incidence of both degenerative cervical and atherosclerotic cerebrovascular disease.

Keywords: atherosclerosis, cerebral angiography, cervical osteophytes, dynamic angiography, imaging – diagnostic, posterior-circulation transient ischemic attack, rotation-induced compressive stenosis, rotational vertebrobasilar ischemia, stents, vertebral artery

Presentation

A 76-year-old man was referred for debilitating ‘near-syncopal spells’ for 6 months. Further inquiry revealed episodic light-headedness, ataxia, diplopia, bilateral blurred vision with flashes of light, binocular vibratory sensation, dysarthria, bilateral leg weakness, and falling. Symptoms occurred when turning his head to the right, occasionally with upward gaze as when shaving and reaching to his cabinets, resolving within 10 minutes of lying down. Generalized weakness persisted for hours. History included carotid, subclavian, and coronary artery stents, diabetes mellitus, hypertension, and dyslipidemia.

Assessment

Vital signs were normal without orthostatic hypotension. Bilateral brachial systolic pressures were equal, there was a systolic murmur, and carotid
sinus massage was normal. Neurologic exam was unremarkable. Laboratory, ECG, and cardiac rhythm monitoring were normal. An echocardiogram demonstrated aortic valve sclerosis and normal ejection fraction. A brain MRI, to assess for mass or infarct, was normal.

Cerebral angiography performed 2 months earlier demonstrated a patent left internal carotid artery stent, complete filling of the right anterior circulation via the anterior communicating artery, an occluded right internal carotid artery, and external carotid 80% stenosis. There was a normal dominant right vertebral artery (RVA), although the ostium was suboptimally visualized, an occluded intracranial left vertebral artery, and a patent left subclavian artery stent. There was no communication between the anterior and posterior cerebral circulation.

**Diagnosis**

Evaluation excluded orthostatic hypotension, carotid sinus hypersensitivity, cardiac arrhythmia, and intracranial mass. Recognizing this patient’s symptom complex as vertebrobasilar transient ischemic attacks occurring with head turning, strongly suggested rotational vertebrobasilar ischemia (RVBI).

The prior angiogram delineated the cerebrovascular anatomy. To look for compression of the lone patent vertebral artery (VA) during head turning, RVA angiography was performed in head neutral and rotated positions. An ostial atherosclerotic 75% eccentric stenosis, not seen in the prior study, was evident in the neutral position (Figure 1). Imaging with progressive rotation to the right, with extension, demonstrated a 75% stenosis at the C5-C6 level and a 50% stenosis at the C4-C5 level that were not present in the neutral position (Figures 2 and 3). Rotation-induced dynamic stenoses with reproduction of symptoms confirmed the diagnosis as RVBI. A cervical spine CT showed diffuse degenerative disc disease and facet hypertrophy without fixed compromise of the transverse foramina.

RVBI is caused by compression of a dominant extracranial VA, producing a transient flow-limiting stenosis or occlusion, induced by head turning. Typically, the contralateral VA is hypoplastic or occluded and compensatory flow from the anterior circulation is absent or inadequate, as in this patient. VA occlusion can occur normally during head turning; however, basilar system flow from the contralateral VA, or the circle of Willis through the posterior communicating arteries (PCoAs), prevents ischemia. Common variations of the PCoAs, such as hypoplastic or absent portions, and atherosclerosis, can preclude compensatory circulation critical in the setting of compromised bilateral VA flow.

**RVBI symptoms** are characteristically transient ischemic attacks, induced by a specific head position, resolving after returning the head to neutral position, and are reproducible. They reflect the location and severity of ischemia, occur in combination, and are rarely isolated. Common symptoms include vertigo, ‘dizziness’ (in reference to vertigo, lack of mental clarity, unsteadiness, and lightheadedness), visual abnormalities, ataxia, dysarthria, and bilateral motor and/or sensory changes. Syncope, near-syncope, and occasionally drop attacks are often described. Orthostatic hypotension can also precipitate symptoms. Recurrent ischemia may impair autoregulation such that even mild hypotension can result in critically reduced cerebral perfusion.

The affected VA can be an embolic source for stroke because of stasis, chronic vascular injury, and thrombus formation at the site of repeated compression, even in the absence of rotational symptoms. Emboli, hypoperfusion, and thrombotic occlusion, alone or in combination, may also result from a severe fixed compressive
stenosis. Stroke from RVBI is occasionally reported; however, the incidence is unknown.

Rotational VA compression is usually caused by musculotendinous insertions or fascial bands at the VA proximal portion, an osteophyte encroaching the transverse foramen at the C3–C7 levels, and several mechanisms at the C1–C2 level; however, osteophytic compression is the most common, as occurred in this patient. Other causes can include disc herniation and subluxation. Musculotendinous compression can cause RVBI in association with thoracic outlet syndrome. The affected VA and direction of head rotation are typically ipsilateral when compression is due to an osteophyte, contralateral when at the C1–C2 level, and either when compression occurs at the VA proximal portion. Additionally, compression can occur at multiple levels in both arteries and, although rare, in both VAs simultaneously.

Transcranial Doppler with head rotation has been used to determine the need for angiography. Cerebral angiography delineates the vascular anatomy, anterior to posterior circulation through the PCoAs, commonly occurring VA anatomic variations, and the presence of VA collateral flow from branches of the ipsilateral external carotid and subclavian arteries. VA dynamic angiography with imaging performed via prevertebral subclavian injections with the neck in neutral and the symptom-provoking positions, most importantly while reproducing symptoms, identifies the presence, site, and degree of compression, establishes the diagnosis and guides surgical planning. Angiography has been described with head rotation to each side (often with extension), progressive rotation to the symptomatic side, and, most recently, in the provoking position while eliciting symptoms. The contralateral VA is similarly imaged if patent and co-dominant. Symptoms rarely result from a non-dominant VA. Manually exerting mild axial pressure to simulate the weight of the head can aid in eliciting symptoms and demonstration of the stenosis. Dynamic angiography performed in mild Trendelenburg’s position or with the patient in sitting or upright positions (injections via the brachial arteries) has been described, although not generally utilized with more recent angiographic systems. Fine-cut cervical CT and MRI, at the site of rotational stenosis, help ascertain the cause of compression. Three-dimensional CT angiography with head
rotation can show both the rotational stenosis and mechanism of compression.\textsuperscript{7}

**Management**

The natural history of RVBI is unknown. Treatment strategies are based on case reports and non-randomized surgical series. Management includes limiting head and neck movement, or surgery. Voluntarily restricting head movement or cervical collar immobilization can be suboptimal in relieving symptoms, uncomfortable, and impractical. Furthermore, a review of the literature revealed that nearly 50\% of non-surgically treated patients sustained a stroke or had residual neurological deficits.\textsuperscript{8} Surgery is highly effective for symptom relief,\textsuperscript{3,6–9,12} has been advocated to reduce the risk of stroke,\textsuperscript{6,8} and is also considered for ineffective voluntary or cervical collar immobilization,\textsuperscript{7} and to avoid injury, such as from falls or when driving.\textsuperscript{7} The patient’s operative suitability is also an important consideration.

Site and cause of the rotational stenosis determine the surgical approach. When at the proximal portion, surgery involves resection of the offending musculotendinous insertions\textsuperscript{9} or fascial bands\textsuperscript{17}; when at the C3-C7 levels, unroofing of the foramina (partial transversectomy) with osteotysectomy\textsuperscript{6} and occasionally discectomy\textsuperscript{3,8}; and if at the C1-C2 level, either partial transversectomy\textsuperscript{3,8,12} or cervical fusion.\textsuperscript{3,7} Contrasted with decompression at C1-C2, fusion at this level significantly limits range of motion\textsuperscript{7} and has an increased risk of surgical injury to the VA.\textsuperscript{3} Fusion is necessary for subluxation at any level and can salvage an unsatisfactory decompression at C1-C2.\textsuperscript{3} Few operative complications are reported.\textsuperscript{6} Distal VA reconstruction is also highly effective for relieving symptoms of rotational\textsuperscript{16,19–21} and fixed compressions\textsuperscript{21} and can be considered in lieu of decompressive surgery unless there is accompanying nerve impingement.\textsuperscript{16} Reconstruction should be considered for a VA embolic source\textsuperscript{16,19,20} or occlusion with patency above the C2 transverse process.\textsuperscript{16,19} Antiplatelet therapy should be used as recommended in the AHA/ASA stroke prevention guidelines.

The most common lesion of the VA is ostial atherosclerosis\textsuperscript{19} and the most common abnormality of the VA second segment is osteophytic compression.\textsuperscript{20} Our patient is unique in that both abnormalities affected the patent VA and RVBI was due to rotation-induced compressive stenoses complicated by the OAS. A cervical collar was too restrictive and symptoms, although improved, remained significant. Surgical decompression of the two dynamic stenoses would not address the OAS, was prohibitive due to the absence of collateral circulation in the event of surgical injury to the VA, and distal VA reconstruction was not feasible due to the absence of a suitable donor artery.

We postulated that restoring proximal flow by stent placement across the OAS would increase perfusion pressure across the dynamic stenoses. The increased intraluminal distending pressure\textsuperscript{22} could in part resist the extrinsic compressive force, enough to render the dynamic stenoses less significant, thereby resolving symptoms. Also, even a modest improvement in perfusion might be beneficial in the probable event of impaired cerebral autoregulation attributable to recurrent ischemia, diabetes mellitus, hypertension, and the patient’s age. The patient would, however, remain at risk of recurrent RVBI due to potentially worsening osteophytic hypertrophy and in-stent restenosis, as well as thromboembolic stroke from repeated rotation-induced compression.\textsuperscript{12,13} Additionally, bony overgrowth could possibly result in a fixed compressive stenosis with risk of emboli,\textsuperscript{14} hypoperfusion,\textsuperscript{15} and thrombotic occlusion.\textsuperscript{6} Acknowledging these factors and considering the patient’s disabling symptoms, stent placement across the ostial stenosis was thought to be the best option and was performed uneventfully (Figure 4). The patient remains asymptomatic with unlimited activity at 14 months.

To our knowledge, this is the first report of RVBI in which the affected VA had an obstructive atherosclerotic stenosis in addition to the characteristic rotation-induced dynamic stenoses; also, the first report of stent placement in the culprit artery

**Figure 4** Right vertebral arteriogram, shallow oblique view. Resolution of the ostial atherosclerotic stenosis after deployment of a drug-eluting stent (arrow); an embolic protection device was used.
to treat this disorder. Stent placement in a VA OAS contralateral to the affected VA has been reported.23

This report illustrates the importance of obtaining an accurate history, recognizing the association of vertebrobasilar ischemia with positional head changes, and the diagnostic approach to RVBI. This case demonstrates the management complexities when there are coexisting abnormalities and emphasizes the need to tailor treatment to the individual patient. Although uncommon, RVBI is particularly relevant due to the aging population, which has a significant incidence of both degenerative cervical and atherosclerotic cerebrovascular disease.24

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References


