Case Report

Vertebral Artery Stenting for the Treatment of Bow Hunter's Syndrome: Report of 4 Cases

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Bow hunter's syndrome (BHS) is a rare condition resulting from vertebrobasilar insufficiency secondary to mechanical occlusion or stenosis of the vertebral artery (VA) due to head rotation. Traditionally, surgical intervention with C1-C2 fusion or VA decompression was the mainstay of therapy. Endovascular intervention was rarely performed to treat BHS. We reviewed the neurointerventional database from July 2005 to October 2010 to identify all cases of BHS treated with VA stenting. Here we report clinical, technical, and outcome data for 4 patients with BHS who were treated with VA stenting. In all 4 of these patients, stenting was performed in the V2 segment (C2-C6) of the VA without significant technical difficulties. All patients reported symptomatic relief, and only minor or no residual stenosis was detected by dynamic digital subtraction angiography. Our findings indicate that VA stenting for the treatment of BHS is feasible, safe, and clinically effective. Endovascular techniques might offer an alternative, minimally invasive therapy for the treatment of BHS. **Key Words:** Vertebrobasilar insufficiency—endovascular treatment—vertebral artery stenosis—cervical fusion—decompression.

Bow hunter's syndrome (BHS) is characterized by vertebrobasilar insufficiency (VBI) due to occlusion or stenosis of the vertebral artery (VA) caused by head movement.[1] The extracranial VA becomes occluded when the head is rotated while the artery is fixed by surrounding structures.[2] The hemodynamic effects of BHS vary from transient ischemic attack to stroke. The gold standard technique for diagnosing BHS is dynamic angiography with head rotation.[1,2]

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In 1933, DeKleyn and Versteegh[3] first described a syndrome of vertigo and nystagmus produced by head movement. In their postmortem studies, they noted compromised VA circulation with head rotation.[3] Ford then surgically treated rotational occlusion of the VA by cervical fusion in 1952.[4] Although these reports recognized this syndrome, it was not until 1978 that Sorenson introduced the term "bow hunter's syndrome."[5]

Patients with BHS have been treated conservatively and surgically. Conservative treatment involves restriction of head movement using a neck brace or collar or administration of anticoagulant medications.[6] Surgical options include fusion of C1-C2 or decompression of the occluded VA through a partial transversectomy at the site of occlusion.[1,7]

The role of endovascular techniques in the management of BHS has been mainly diagnostic. Only a few case reports describe endovascular techniques as a therapeutic option for BHS. Here we report 4 cases of BHS treated with VA stenting. To the best of our knowledge,

Table 1. *Patient characteristics*

	Patient 1	Patient 2	Patient 3	Patient 4
Sex	M	M	M	M
Age	66	55	85	70
Ethnicity	W	W	W	W
Symptoms	Vertigo, ataxia	Vertigo, headaches, tinnitus, diplopia	Vertigo, syncope	Confusion, diplopia
Risk factors				
Hypertension	No	Yes	No	Yes
Hypercholesterolemia	No	No	Yes	Yes
Diabetes	No	No	No	No
Smoking	Yes	No	No	No
Coronary artery disease	Yes	No	Yes	No

this is the largest case series involving endovascular management of BHS reported to date.

Methods

After acquiring Institutional Review Board authorization, we retrospectively reviewed the neurointerventional database to identify all cases of BHS treated with VA stenting. In the 4 cases identified, we collected data on demographics, clinical presentation, and outcomes, and reviewed and analyzed angiographic images. Here we report clinical, technical, and outcome data for these 4 patients.

All 4 patients were treated with daily aspirin 325 mg and clopidogrel 75 mg for 1 week before the procedure. All procedures were performed with the patient supine under conscious sedation with midazolam and fentanyl and local anesthesia with lidocaine. In accordance with stenting protocol, the patients were systemically heparinized to maintain an activated clotting time of 250-300 seconds.

Stenting was performed using a transfemoral approach in 3 of the 4 cases and a transradial approach in the remaining case. An ENVOY guide catheter (6 Fr for the transfemoral approach and 5 Fr for the transradial approach; Codman, Raynham, MA) was placed in the subclavian artery. Baseline intracranial images of the posterior circulation were obtained, and then dynamic digital subtraction angiography (DSA) with head positioning (neck flexion, extension, right turn flexion and extension, left turn flexion and extension) was performed to confirm the positional stenosis of the VA. The area of maximum dynamic stenosis on head turning was noted. The findings were confirmed clinically by asking the patient about the appearance of symptoms or by neurologic examination. If more than one area of stenosis was identified on positioning, the area of stenosis with the historical symptomatic position was chosen for treatment.

Once the area of symptomatic stenosis was confirmed, a roadmap with optimal angles was obtained. Subsequently, a microwire was advanced into the VA and positioned in the V4 segment. Then a self-expanding stent of the appropriate diameter and length (an Xpert biliary stent [Abbott Laboratories, Beringen, Switzerland] in 3 patients and a Wallstent [Boston Scientific, Natick, MA] in 1 patient) was deployed in the V2 segment covering the area of dynamic stenosis. In 3 of the 4 patients, angioplasty was performed to ensure good stent wall apposition. No embolic protection devices were used due to the lack of atherosclerosis in these areas. Final cervical DSA images were then obtained in different head positions to verify patency of the stent and flow, and final intracranial images were obtained to rule out any procedure-related complications. After the procedure, all patients were monitored in a neurologic intensive care unit. Dual antiplatelet therapy (aspirin 325 mg and clopidogrel 75 mg daily) was continued for 4 weeks, followed by life-long aspirin 325 mg daily.

Results

We report 4 patients, all white males aged 55-85 years, who presented to our facility between November 2009 and October 2010. The most common complaint was vertigo with head movement. Two patients also complained of visual changes and double vision (Table 1). All 4 patients underwent dynamic DSA. Three of the 4 patients exhibited a dominant left VA with a hypoplastic or stenosed contralateral VA; in the other patient, VBI was due to stenosis of the nondominant VA, which terminated in the posterior inferior cerebellar artery (PICA) and resulted in vertiginous episodes with head movement related to ipsilateral PICA ischemia. Dynamic DSA revealed non–hemodynamically significant stenosis with the head in the neutral position. With head turning, stenosis ranged from 75% to 99%, which was hemodynamically significant and produced symptoms in all 4 patients.

Once dynamic stenosis was confirmed, all 4 patients underwent stent placement in the V2 segment of the VA. One patient had an additional stent placed in the V1 segment because of severe VA-origin stenosis (Herculink Plus, Abbott Laboratories, Beringen, Switzerland).