Right Vertebral Artery Stenosis with Left Hemispheric TIA: A Perplexing Etiology

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Introduction

Vertebral Artery (VA) atherosclerosis is a seldom-discussed disease that contributes significantly to posterior circulation ischemic events. Demonstrated in up to 40% of individuals with cerebrovascular disease, VA atherosclerosis accounts for nearly 30% of all transient ischemic attacks (TIA) or stroke [1]. Historically, less than 5% of surgeries performed for cerebrovascular disease address lesions of the VA origin where atherosclerotic disease is most prevalent [2].

First studied in the 1960’s, the natural history of VA occlusive disease was felt to be benign when compared with carotid distribution ischemic events [3]. It is not until recently that current investigators are beginning to challenge this notion. In a literature review, Flossmann and Rothwell found no evidence that patients presenting with posterior circulation ischemic events were at lower risk of subsequent stroke or death when compared to those presenting with carotid TIA or minor stroke. Furthermore, their risk of stroke was slightly higher in the acute time period following initial symptom onset [4]. A complicating issue is the fact several other medical conditions may produce symptoms mimicking posterior circulation ischemic events such as use of antihypertensive medications, cardiac arrhythmias, anemia, and benign vertigo.

Unlike carotid intervention, where well-defined roles for secondary prevention of ischemic events exist, the benefit of surgical or endovascular revascularization for proximal VA atherosclerosis remains yet to be clearly defined. Appropriate patient selection for intervention requires individualized assessment of risks balanced with potential benefits of revascularization [5]. This report presents a patient with recurrent left hemispheric TIA as a result of right VA stenosis managed via endovascular intervention.

Case Report

A 59-year-old female was evaluated in the in-patient setting for recent complaints of intermittent right upper and lower extremity weakness. Symptoms had increased in frequency and were also associated with new onset speech difficulties. Pertinent medical history included hypertension well controlled with use of carvedilol and losartan. She endorsed no history of dyslipidemia, coronary artery disease, or diabetes. Social history was remarkable for 49 pack-year nicotine use and a family history of maternal Peripheral Arterial Disease (PAD) was noted. Upon evaluation by the vascular surgery service, symptoms had completely resolved and physical examination was unremarkable. Pertinent serum laboratory analysis was also unremarkable for anemia, hyperglycemia, renal insufficiency, and electrolyte abnormalities. Additionally, non-fasting lipid panel results were as follows: Total cholesterol 122 mg/dL; Triglycerides 82 mg/dL; HDL 59 mg/dL; LDL 47 mg/dL. Prior to consultation, imaging studies obtained included Magnetic Resonance (MR) and Computed Tomography (CT) angiography of the head and neck. MR imaging revealed high-grade stenosis of the left Internal Carotid Artery (ICA) and small bilateral subcortical infarctions (Figure 1). Additionally, CT displayed complete left ICA occlusion, 50% right ICA stenosis, and multi-segment disease of the left Vertebral Artery (VA) (Figure 2). Carotid duplex ultrasonography was performed which confirmed left ICA occlusion, moderate right ICA stenosis, and visualized bilateral VA antegrade blood flow (Figure 3). The patient was discharged home on clopidogrel, statin therapy, and previous anti-hypertensive medications.

Figure 1: Magnetic resonance (MR) angiography of the head depicting acute left hemispheric subcortical infarction.
Post-operative right VA duplex surveillance at three, six, and seven months demonstrated increasing Peak Systolic Velocities (PSVs) in excess of 300 cm/sec consistent with in-stent restenosis. Re-intervention was performed by placement of a 5 mmx39 mm iCast (Atrium, Hudson, NH, USA) stent with use of an embolic protection device. To date, outpatient follow-up with arterial duplex surveillance at three months displays PSVs of <100 cm/sec and the patient remains symptom free. She has been provided with smoking cessation materials and education however unfortunately continues to smoke.

Discussion

Low-flow hemodynamics and embolic disease are the two mechanisms responsible for vertebrobasilar circulation ischemic events. Although fewer patients suffer from embolic disease than from hemodynamic mechanisms, actual infarctions in the posterior circulation are most commonly the result of embolic events [6]. There is a lack of randomized controlled data regarding the medical management of asymptomatic VA atherosclerosis. Current guidelines from several vascular, neurosurgical, and multidisciplinary medical societies recommend best medical management to include those practices established for carotid artery disease [7]. Intervention for disease, whether surgical or endovascular, takes into account symptomology, concurrent carotid artery disease, circle of Willis patency, and contralateral posterior circulation anatomy in order to guide treatment strategies. In addition to carotid artery atherosclerosis affecting anterior circulation, Ciccone and colleagues have extensively studied carotid artery dolichoectasias in terms of clinical significance. This rare entity, found in 2-6% of the general population, was studied for association with neurological symptomatology in patients without concomitant carotid artery atherosclerotic disease. Investigators found carotid echo-color Doppler to be both sensitive and specific for diagnosis. Additionally, five year follow up revealed carotid artery tortuosity and kinking to be a progressive pathology with tortuosity specifically, associated with an increased risk of cardiovascular death in the study population [8]. Similar research demonstrated a positive correlation between carotid artery dolichoectasia and arterial hypertension in a study population with non-ischemic dilated cardiomyopathy [9].

Established indications for intervention of extracranial VA atherosclerosis include symptomatic high-grade stenosis, as well as
progressive, high-grade stenosis in the presence of compromised contralateral VA flow [10]. No randomized controlled studies comparing open versus endovascular treatment of VA atherosclerosis currently exist. The largest retrospective study available comparing the two modalities found open intervention (VA to common carotid artery transposition) to provide better symptomatic relief in those presenting with transient symptoms attributed to low-flow hemodynamics. Although not statistically significant, trends toward superior long-term patency along with decreased need for secondary intervention were also observed in the open group. Open intervention, as expected, was associated with higher rates of perioperative morbidity [11]. More recently however, treatment strategies have shifted toward endovascular approaches to include VA stenting.

A large review of over 700 patients with proximal VA atherosclerosis undergoing VA stenting demonstrated the efficacy and safety of this growing treatment modality. Technical success and perioperative event rates of 99% and 1.5% respectively, were observed in symptomatic patients treated. With follow up of nearly two years, many case series included in this review demonstrated In-Stent Restenosis (ISR) as the primary limitation of endovascular intervention when compared with the durable outcomes of open surgical intervention [12].

Atherosclerotic lesion length has been well recognized and validated as an important predictor of ISR in cardiac literature. Currently, smaller studies evaluating lesion length in the VA have also found it an independent predictor of ISR [13]. Higher elastic recoil intrinsic to VA anatomy has been another hypothesized explanation for such high rates of ISR observed with endovascular intervention. As many case series and reviews in the literature allude to, VA atherosclerosis can be managed safely and efficaciously with endovascular techniques. Investigators have now turned attention to technical aspects to improve the durability of endovascular intervention.

Balloon expandable coronary stents appear to be the most common stent employed in several case series for treatment of proximal VA atherosclerosis. A large prospective study from 2003 to 2011 evaluated Drug Eluting Coronary Stents (DES) versus Bare Metal Coronary Stents (BMS) for superiority when used to treat proximal VA atherosclerosis. Over 200 patients were treated for symptomatic lesions of greater than 70% stenosis. With long term follow up of more than 3.5 years, both DES and BMS use was found to be safe and technically feasible. While both effective for symptomatic treatment of VA atherosclerosis, DES use significantly decreased the need for later endovascular intervention in the long term [14].

In summary, VA atherosclerosis is an underappreciated disease process that contributes significantly to cerebrovascular disease. In general, treatment with open intervention carries a higher morbidity and mortality in the acute setting while appearing to be more durable in terms of long-term patency. While the literature supports the safety and feasibility of VA stenting, it is plagued by ISR in those with long atherosclerotic lesions. When stenting is performed, use of coronary drug eluting stents appears to be favored. Decisions to intervene, whether open or endovascular takes into account contralateral VA flow, symptomatology, and concomitant anterior circulation occlusive disease.

This case demonstrates a scenario in which a patient suffered TIA secondary to proximal VA atherosclerosis. Aberrant anatomy complicated the case leading to a delayed diagnosis prior to endovascular treatment. Her post-operative course was complicated by ISR mandating further endovascular therapy. This review highlights the need for increased awareness of posterior circulation ischemic events as well as treatment modalities employed for intervention.

References