Clinical Outcome of Patients with Acute Posterior Circulation Stroke and Bilateral Vertebral Artery Occlusion

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Abstract

Background and Introduction: Patients presenting with posterior circulation acute ischemic events are occasionally noted to have occlusion of bilateral vertebral arteries with basilar artery blood flow entirely dependent from the anterior circulation. There is limited data about prognosis of such patients in literature.

Methods: Patients with acute posterior circulation ischemic stroke and bilateral vertebral artery occlusion (including contra-lateral hypoplastic vertebral artery without contribution to the basilar artery system) were identified prospectively from two academic centers. Data including clinical presentation, medical management, angiographic findings, recurrent events and outcome were collected and reported.

Results: A total of 4 patients presenting with acute ischemic events in the posterior circulation were identified to have bilateral vertebral artery occlusion at our center. One additional patient had a vertebral artery occlusion and a contra-lateral hypoplastic vertebral artery. In the functional evaluation of the blood flow with catheter angiography, the basilar artery was filling from the anterior circulation, with no antegrade flow from bilateral vertebral arteries injection in all 5 patients. Patients were treated with anti-platelets (n=4) or started on anti-coagulation after failing anti-platelet therapy (n=2). All patients had recurrent ischemic stroke with new ischemic lesions proven by diffusion weighted images on MRI within 2 to 70 days after the initial event.

Conclusion: Patients with acute posterior circulation ischemic stroke and bilateral vertebral artery occlusion are at high risk of having early recurrent ischemic events. Reestablishment of the antegrade vertebro-basilar blood flow through endovascular re-canalization might be an option to decrease stroke recurrence in selected patients with acute posterior circulation stroke and bilateral vertebral artery occlusion.

Keywords: vertebral artery occlusion, bilateral, ischemic stroke, prognosis

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Introduction

Symptomatic bilateral vertebral artery occlusion (BVAO) is a serious condition that can carry a high potential for further ischemic stroke. The prognosis of this disease reported in literature is unclear.[1,2] Most of the published cases were identified days after symptoms onset and the prognosis of acute BVAO is not well known.[1,2] It is likely that in previous studies some cases who acutely deteriorated were never identified and/or under-reported.. We report 5 cases of symptomatic bilateral vertebral artery occlusion identified within the initial 24 hours of presentation, and report their management and clinical outcome.

Methods

Between December 2006 and December 2008, all patients with posterior circulation acute ischemic events with either BVAO or occlusion of the dominant vertebral artery, with contra-lateral hypoplastic vertebral artery (not contributing to the basilar artery blood flow) were identified from two hospitals. Data including age, sex, race, vascular risk factors (hypertension, diabetes mellitus, coronary artery disease, hyperlipidemia, history of previous stroke/ TIA, cigarette smoking and drug abuse), clinical presentation, time of primary event and the imaging studies was collected. Medical and occasional interventional treatment (including anti-thrombotic, thrombolytic therapy), as well as angiographic findings, and recurrent events and outcome were collected and reported.

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Table 1: Table showing patient characteristics and outcomes.

Age/ Sex	Qualifying event	Time from symptom onset to diagnosis (hours)	NIHSS		Initial medical therapy	Lesion Location	Neurological deterioration during hospital stay	Procedure/ Vertebral recanalization	Outcome mRS at discharge	Event	Time from symptom onset till recurrent event
			Initial	Discharge							
24/M	Stroke	20	4	42	Heparin	Bilateral Extracranial (V-1)	Yes	Yes / Failed	6	Death	2 days
30/M	Stroke	120	6	7	Aspirin, plavix	Bilateral Intracranial (V3-V4 junction)	Yes	No	4	Stroke	12 days
52/M	Stroke	24	5	3	Aspirin, Plavix	Extracranial (V-2) Hypoplastic right V-1	No	No??? was any	1	TIA	70 days
54/M	Stroke	48	2	42	Aspirin, Plavix, Heparin	Bilateral Intracranial (V-4)	Yes	No	6	Stroke/ Death	13 days
64/F	TIA	24	2	1	Aspirin, Plavix, Clopidogril	Bilateral Intracranial (V-4)	No	Yes / successful	1	Nil	Nil

Patients with recurrent ischemic events in the posterior circulation despite best medical treatment were considered for endovascular re-canalization of the occluded vessel. For endovascular treatment an introducer sheath was placed in the femoral artery for percutaneous access. Heparin was intravenously administered as a bolus dose to achieve an activated coagulation time (ACT) in the range of 250 to 350 seconds. A guide catheter was placed in the cervical vertebral artery. If the lesion was considered to be atherosclerotic, angioplasty was performed before stent placement. In cases of presumed dissection, endovascular reconstruction of the arterial lumen was achieved through stent deployment without angioplasty. The selected balloon catheter or stent delivery device was advanced over a 0.014 inch micro guide wire and navigated to the site of stenosis. Balloons and stents were selected carefully to provide sub-maximal inflation to avoid vessel dissections and rupture. The goal of the procedures was to reduce the stenosis to approximately 30% or less. Post-procedure, a combination of aspirin (325 mg daily) and clopidogrel (75 mg daily) was used for at least a month followed by aspirin indefinitely.

In-hospital and peri-procedural monitoring

As per the hospital protocol, each patient underwent evaluation by a vascular neurologist before, immediately after, and 24 hours after the re-vascularization procedure. If general anesthesia was used, patients were evaluated serially for level of arousal until a comprehensive neurological examination could be performed. The patients were observed in the neuro-intensive care unit for 24 hours post-procedure. Subjects were examined hourly by the nursing staff. As soon as any episode of neurological deterioration was recognized, the treating physicians were notified for further evaluation and management. Imaging studies (CT [computerized tomography], MRI [magnetic resonance images], MRA [magnetic resonance angiography] and DSA [digital subtraction angiography]) were analyzed serially during admission till last follow up available. Neurological examination was recorded as NIHSS by a neurologist. Clinical charts were reviewed by a neurologist to determine mRS (modified Rankin scale).

Results

Among the patients admitted with acute ischemic stroke between the period of December 2006 till December 2008, BVAO was identified in 5 patients. Patients with BVAO ranged in age from 24 to 64 years and four were male. Table 1 presents the summary of clinical characteristics of the patients included in this study. Duration from symptom onset to diagnosis ranged from 20 to 120 hours. Four patients had clinical stroke and one had transient ischemic event as initial presentation. The underlying etiology was likely atherosclerotic in 4 patients. One patient had bilateral vertebral artery occlusion from trauma. Three patients had neurological deterioration during hospitalization. One other patient was discharged on anti-thrombotic treatment and readmitted after 2 months with recurrent ischemic events. Two patients underwent re-canalization procedures.

Patient 1

A 24 year old male was brought in after a road traffic accident. He was moving all extremities but required intubation for drowsiness. Initial NIHSS at admission was 20. Cervical fracture along with skull base and facial bone fracture was seen on the first CT scan in ER. CT angiogram suggested traumatic BVAO. Patient was admitted to intensive care unit. Anti-thrombotics could not be administered secondary to multiple injuries. Patient had a drop in blood pressure which required resuscitation for about 1 hour. After that patient was noted to have neurological deterioration with clear posterior circulation ischemic symptoms due to basilar insufficiency, a catheter based angiography was performed

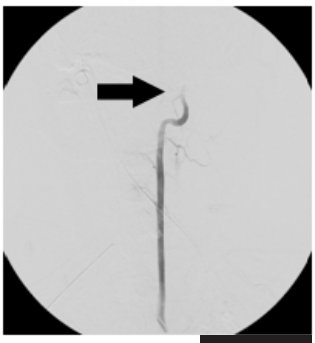

Figure 1a

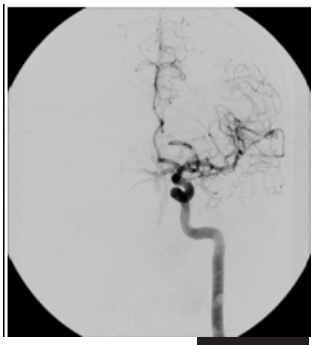

Figure 1b

Cerebral angiograms:

Figure 1a: Bilateral selective injection showing occlusion of the V3 segment of left and right VA.

Figure 1b: Selective injection of left ICA showing patency of the left posterior communicating artery with retrograde filling of the basilar artery and terminal branches.

emergently which confirmed the findings of extra-cranial BVAO at the level of C-1. Basilar artery filled from left internal carotid injection through a patent left posterior communicating artery. Successful reconstitution of the left vertebral artery was obtained after deployment of 3 self expanding stents. However, there was failure of re-canalization possibly secondary to thrombus. Patient did suffer from a massive cerebellar and brain stem infarction complicated with posterior fossa intra-cranial hypertension, superior trans-tentorial herniation and inferior cerebellar tonsilar herniation followed by brain death.

Patient 2

A 30 year old male with a previous history of drug abuse, smoking, uncontrolled hypertension and hyperlipidemia presented to the emergency room eight-day history of dys-arthria, gait instability, nausea, vomiting, and left-sided weakness. Patient was taking aspirin 81 mg daily. His blood pressure on admission was 180/100 mmHg. Admission NIHSS was 8 secondary to facial palsy, ataxia and hemi-paresis. Brain MRI demonstrated acute right medullary infarction and multiple scattered subacute infarcts in both cerebellar hemispheres. MRA of the neck demonstrated small caliber left vertebral artery with poor flow in the basilar artery. Catheter based angiography was performed and complete occlusion of bilateral vertebral artery at the V3-V4 junction was noted. Left VA was hypoplastic. Right VA was occluded just distal to the PICA origin. The basilar artery, PCAs, SCAs, AICAs and left PICA filled retrograde from the right internal carotid injection. Patient was kept on anti-thrombotic medication. One week later patient had a recurrent ischemic event with worsening dys-arthria and ataxia. MRI of the brain demonstrated new areas of diffusion restriction in the cerebellum. A repeat catheter based angiogram was unchanged. At discharge, he had gait ataxia and left sided weakness. NIHSS was 7 and mRS was 4. He was discharged on aspirin and plavix. He remained event free and at 6 months the mRS was 3.

Patient 3

A 52 year old male with history of hypertension, hyperlipidemia and smoking presented with sudden onset of dizziness, gait imbalance and left sided hemi-paresis. His admission NIHSS was 5. Brain MRI demonstrated a left cerebellar infarct. MRA of cervical vessels was suggestive of occlusion of left VA at its origin and right VA was terminating in PICA. Catheter based angiography demonstrated retrograde filling of the basilar artery from the injection in the left ICA through a prominent posterior communicating artery. The distal left VA was reconstituted through left ascending cervical artery. The patient was placed on dual anti-platelet therapy (aspirin and clopidrogel). At the time of discharge his NIHSS was 2 and mRS was 1. Six-months follow up revealed no recurrent event.

Patient 4

A 54 year old African American male with past history of Ischemic stroke, uncontrolled hypertension, diabetes type II, smoking, alcohol abuse and coronary artery disease status post

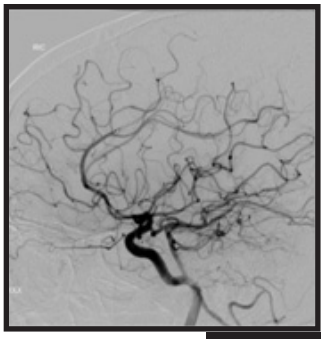

Figure 2a

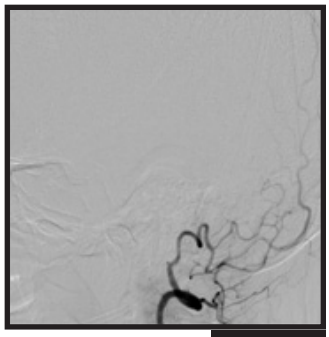

Figure 2b

Cerebral angiograms:

Figure 2a: Lateral view of the cranium from selective injection in the right proximal ICA demonstrates robust filling of the post circulation including basilar artery from the distal tip to the origin of the Left PICA.

Figure 2b: Selective injection in the right VA showing occlusion of the intra-cranial right VA immediately after the origin of the right PICA.

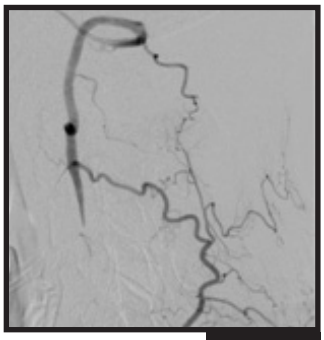

Figure 3a

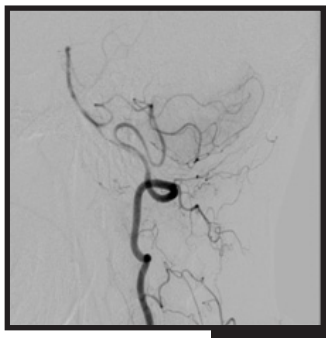

Figure 3b

Cerebral angiograms:

Figure 3a: Cerebral Angiogram showing lateral view of injection of the left subclavian artery showing occlusion of the proximal left vertebral art with distal reconstruction of the left distal V2 & V3 segment via ascending cervical collaterals.

Figure 3b: Cerebral angiogram lateral view of selective injection in the right VA showing hypoplastic right VA that functionally ends up in right PICA with faint filling of the distal basilar and SCA.

CABG, presented to the ED with unsteady gait, light headedness, and vertigo for the past 2 days. NIHSS at admission was 7 (limb ataxia, facial palsy, left sided weakness and gaze limitation). An emergent CT Scan showed a wedge shaped hypo-density in the infero-medial posterior left cerebellar hemisphere. Aspirin and Clopidogrel were started on day 1. Overnight the patient deteriorated neurologically with worsening left sided weakness, tongue deviation towards the right, dysarthria & worsening dysphagia. CT angiogram showed bilateral intra-cranial vertebral arteries occlusion. MRI of the brain demonstrated multiple acute to early subacute infarcts scattered in the posterior circulation including brainstem, cerebellar hemispheres, bilateral occipital lobes and right thalamus. The left cerebellar hemisphere was causing mass effect without brainstem compression and 3% hypertonic saline was started. IV heparin was started after MRI results but later stopped secondary to epistaxis. Catheter based angiography confirmed intra-cranial BVAO. There was reconstitution of the proximal basilar artery from the meningopial branches of the right PICA and right VA as well as the anterior spinal artery. There was also reconstitution of the distal basilar artery through bilateral small posterior communicating arteries.

He was remained ataxic, with profound dysphagia, severe dysarthria and moderate cognitive deficit. Subsequently he re-infarcted and went into coma and died. CT of the head revealed new infarcts in the cerebellum, midbrain and pons with surrounding edema and mass effect.

Patient 5

A 64 year old female with history of hypertension and hyperlipidemia was admitted for a transient episode of headache, dizziness, nausea, loss of balance and dysmetria. Her initial NIHSS was 2. An initial MRI demonstrated small scattered infarcts in the cerebellum and pons. . Catheter based angiography demonstrated BVAO distal to the origin of PICA. A slow retrograde flow of the basilar artery from the posterior communicating artery was noted. The micro-catheterization of the left VA demonstrated a poor flow. After initiation of dual anti-platelet regimen, (aspirin and Clopidogrel) balloon angioplasty and stent placement of the left VA was performed successful re-canalization of the left VA was achieved with no residual stenosis and establishment of antegrade flow in the basilar artery. At the time of discharge NIHSS was 1 and mRS was 1. Patient remained event free and at 12 months NIHSS was 1 and mRs 1 .

Discussion

This report emphasizes the poor prognosis of patients with acute ischemic stroke in the posterior circulation associated with bilateral vertebral artery occlusion. . In our case series, only one of 5 patients remained event free after the initial presentation. All other four patients had recurrent events within 3 months of initial presentation and two of these four patients died. .

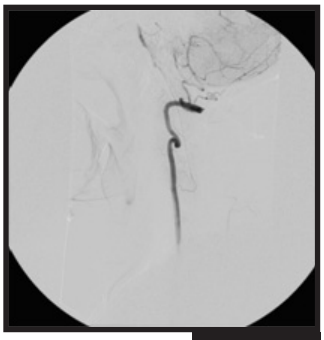

Figure 4a

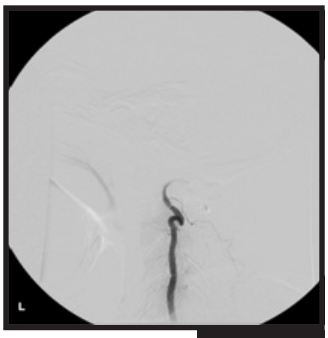

Figure 4b

Cerebral angiograms:

Figure 4a: Cerebral angiogram selective injection in the right VA showing a hypoplastic right vert that ends up in PICA.

Figure 4b: Cerebral angiogram selective injection in the left VA showing occlusion of the left VA at the distal V-3 segment.

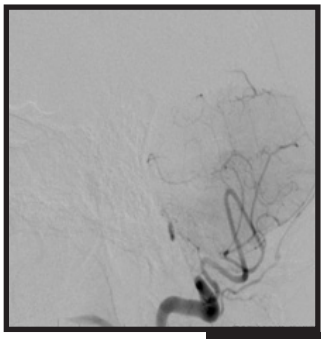

Figure 5a

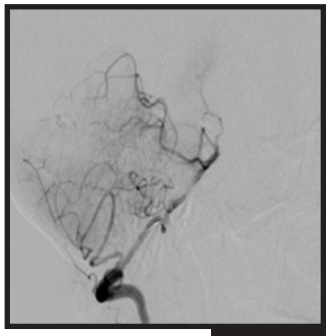

Figure 5b

Cerebral angiograms:

Figure 5a: Lateral view of selective injection of the Lt VA showing severe stenosis (near occlusion) of the proximal intra-cranial left VA after the origin of the Left PICA.

Figure 5b: Lateral view of selective injection of the Lt VA showing complete re-canalization of the left intra-cranial VA with good distal flow throughout the basilar artery, bilateral AICA & SCAs after successful intra-cranial angioplasty and stent placement.

Possible etiologies for BVAO include atherosclerosis,[1-4] giant cell arteritis,[5] trauma[6,7] and spontaneous dissection. Atherosclerotic stenosis of the extra and intra-cranial segments of the VA, are not uncommon cause of posterior circulation ischemic stroke, however, clinical presentation with bilateral vertebral artery occlusion is uncommon. In the New England Medical Center posterior circulation stroke registry[8] complete occlusion of vertebral artery was identified in 37(9%) of 407 patients with posterior circulation ischemic events and 12 (2.7%) had BVAO.

Caplan et al[1] reported a grave prognosis of a total of 9 patients in which both VA were occluded. Eight of them had recurrent strokes and all of them dying within 6 months after presentation. However, all of these patients had preexisting comorbidities including hypertension, DM- type II, Prior Ischemic strokes and TIAs. Later, Bogousslavsky et al[2] noted a better prognosis in chronic bilateral vertebral occlusion. Out of 17 patients, none had died at discharge, and during a mean follow up of 6 years only 5 had died, 2 had strokes and three suffered from TIAs. It is important to note that among 14 out of the total 17 patients in the above mentioned study,[2] the basilar artery was either supplied by collaterals or anastomotic channels from either occipital artery, posterior communicating artery, PICA or thyro-cervical trunk. It was not mentioned whether the remaining 3 patients had either complete occlusions of bilateral vertebrals with no supply to basilar artery or they too had collateral supply to the basilar artery. However in our study there was angiographic evidence of complete bilateral vertebral occlusion with no collateral supply to the basilar artery except retrograde filling of basilar from posterior communicating arteries. Hence all cases were acute in presentation. A better prognosis in the case series reported by Bogousslavsky et al can be because of more cases of chronic BVAO with chronic compensation from collateral vessels. Also the initial presenting symptoms of patients in the above study,[2] had over all less grave symptoms at the time of presentation than our study population. From a total of 17 patients in the study, only 8 had ischemic strokes, 7 presented with TIA and two were asymptomatic. In our study, 4 out of 5 patients presented with stroke and only one with TIA.

It is important to note that in these two previous case series, the data was collected over a long time period (12 years in Bogousslavsky et al[2] and 13 years in Caplan et al[1]) and the patients were selected based on their presenting symptoms and angiographic findings. We extracted these ABVO patients from a prospectively maintained database over a span of 2 years. The signs and symptoms of our patients were typical of posterior circulation syndrome including diplopia, gait imbalance, drop attacks, vertigo, and different combination of contra-lateral sensory and motor deficit. In BVAO, the PICA supply is usually compromised leading to cerebellar infarction and poor collateral supply to brain stem.[1] The usual site of vascular occlusion in patients with cerebellar infarction is the VA proximal to the PICA branch.[9] We had similar findings in 3 of our non traumatic acute BVAO patients and in one patient there was an acute occlusion of left vertebral artery at its origin and a hypoplastic right VA terminating in to PICA.

Treatment of intra-cranial stenosis has been mainly limited to anti-coagulation or anti-platelet agents. The risk of recurrent ischemic event in the vessel of distribution is 23% over first year and there is no clinical advantage of using warfarin over aspirin.[10] In contrast to these patients, our study suggests that patients with compromise of bilateral vertebral artery flow have much worse prognosis with medical management alone. The likely reason for poor prognosis is inadequate perfusion of basilar artery through posterior communicating arteries.

Surgical bypass between occipital artery and PICA was attempted in the late 70s and early 80s with some success but later this surgical bypass procedure lost its importance after the negative results of EC/IC Bypasss trial, published in 1985.[11] In the 90s, endovascular treatment was reserved for seriously ill, symptomatic patients with complication rates ranging from 12 to 33%.[12-14] With recent advances in technique, endovascular intervention combining percutaneous trans-luminal angioplasty (PTA) followed by stent placement has become a promising option for intra or extra-cranial vertebral artery stenosis.[15,16] A growing number of case series describe a technical success rates of 94–100% for primary deployment of stents in lesions with 50% or greater stenosis.[17-25]

We tried endovascular treatment for the traumatic patient considering his young age, no co-morbid factors and based on previous studies supporting the use of this treatment in injuries resulting in acute unilateral vertebral occlusion.[7,26-28] In this

patient, successful reconstitution of the left vertebral artery was achieved after deployment of 3 consecutive stents. However, there was failure of re-canalization possibly secondary to acute in-stent thrombosis.

Our case series is small that obviously limit the significance of our findings. We consider that the possibility of performing randomized trials is unrealistic due to the lack of other adequate alternative therapies, the low incidence of this disease, and that the management of these patients will remain controversial until there are more studies to evaluate the use of acute intervention in these patients. In conclusion, we believe that patients presenting with posterior circulation symptoms from acute compromise of vertebral antegrade flow have grave prognosis and endovascular re-canalizaiton should be considered as an early option.

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