CASE PRESENTATION

A 51-year-old man with hypertension develops acute onset of dysarthria and left hemiparesis. On arrival at another hospital, the hemiparesis resolves. Telestroke evaluation shows his National Institutes of Health Stroke Scale (NIHSS) score is 1.

CTA reveals a right vertebrobasilar artery aneurysm associated with an intraluminal nonocclusive thrombus. The aneurysm exerts mass effect on the adjacent pons, pontomesencephalic junction, as well as rostral midbrain (Figure 1).

The patient is transferred to our hospital with a stable neurologic examination remarkable for dysarthria and an NIHSS score of 1. The next day, 48 hours after symptom onset, the patient becomes stuporous with decreased withdrawal on the left arm and leg to noxious stimulation.

What is your next step?

- MRI/MRA
- CTA
- CT perfusion imaging
- Proceed directly to the angiographic/interventional suite

Dr. Froehler: The initial imaging is consistent with a fusiform aneurysm, or dolichoectasia, of the basilar artery. Given the initial symptoms, there was likely some thrombosis along the wall that caused perforator occlusion and brainstem ischemia. Fortunately, his symptoms improved dramatically, although not completely, and he remains at risk for recurrent thrombosis and infarction. I favor aggressive treatment with antithrombotics in such cases, which could take the form of antiplatelets, anticoagulation, or some combination thereof.

It is unclear what the initial treatment strategy was prior to this sudden decline. The most likely cause is recurrent thrombosis and ischemia, although it is important to realize that fusiform aneurysms can also rupture and result in sudden neurologic decline. I would favor emergent CT/CTA to evaluate for subarachnoid hemorrhage as well as reassessment of clot within the fusiform aneurysm.

Dr. Jadhav: In a patient presenting with presumed ischemic symptoms in the setting of a right vertebrobasilar artery aneurysm and nonocclusive thrombus, clinical worsening with focality is most concerning for progressive vessel occlusion and further aneurysmal thrombosis. Additional concerns would include occlusion of a small vessel perforator, progressive aneurysmal growth with mass...
effect, or aneurysmal rupture. To distinguish these possibilities, it is essential to perform neuroimaging to exclude the presence of hemorrhage or herniation (parenchymal imaging) as well as vessel imaging. An MRI would readily identify mass effect, but the sensitivity for acute blood would be higher with CT of the head. Additionally, CT of the head is a faster study and much more readily available on an emergent basis at most centers. CTA of the head/neck would confirm vessel patency. CT perfusion would likely not be of any additional diagnostic value in this scenario.

Dr. Narayanan: My next step would be MRI/MRA. The patient was last known well 48 hours ago, and the time of deterioration is not clearly indicated. MRI is the gold standard to ascertain the location and extent of ischemic injury in the posterior circulation and should be performed without delay to assess the patient’s new radiographic baseline. Because sudden clinical deterioration in a patient with a partially thrombosed fusiform basilar artery aneurysm raises more of a concern for ischemic rather than a hemorrhagic process, a rapid MRI of the head without gadolinium focusing on diffusion-weighted imaging, apparent diffusion coefficient, and gradient echo or susceptibility-weighted imaging sequences saves time. I would also request an MRA of the head (and consider with gadolinium) to determine collateral flow through the circle of Willis or alternative access to the distal aspect of the aneurysm. Vascular imaging would be helpful to determine in-situ thrombosis (more likely etiology), presence of distal embolism, and possibly delineate the extent of clot, which could inform device selection during thrombectomy.

CASE CONTINUED

Two hours after his most recent neurologic deterioration, the patient undergoes emergent cerebral angiography. Selective right vertebral artery digital subtraction angiography reveals a complete mid-basilar occlusion, dominant right vertebral artery, and flow defect outlining the previously seen thrombus at the proximal aspect of the fusiform aneurysm (Figure 2).

What would your technical approach to thrombectomy consist of?

Dr. Jadhav: Acute occlusion of the basilar artery with coma is a neurologic emergency with an associated poor prognosis. Given that the right vertebral artery is dominant, this is likely the most favorable access point to attempt thrombectomy. There are several technical considerations pertinent to this scenario. Access can be achieved via a traditional right femoral artery approach, or given the proximity of the right vertebral artery, a right transradial approach may also be favorable. Via either approach, I would advance a Neuron Max guide catheter (Penumbra, Inc.) in the distal right vertebral artery (segment 2). A coaxial system consisting of a large aspiration catheter (Ace 68, Penumbra, Inc.), microcatheter (Velocity, Penumbra, Inc.), and microwire (0.014-inch Synchro2, Stryker) would then be advanced across to the right V4 segment. From this position, I would advance the microwire and microcatheter to select the left P1 segment. After removing the wire, I would deploy a 6- X 40-mm Solitaire stent retriever (Medtronic) from the left P1 segment into the basilar artery. I would then remove the microcatheter with the stent retriever deployed and perform an angiographic run through the Ace catheter prior to removal of the stent retriever, with manual aspiration applied to the Ace catheter.

Dr. Narayanan: If there is no hemorrhagic conversion on MRI, I begin all ischemic stroke interventions with a 5,000 unit intravenous bolus of heparin after femoral artery puncture (if alteplase was not given) to achieve a therapeutic activated clotting time. Particularly in the posterior circulation, this sometimes results in thrombolysis by the time the thrombectomy catheters are in place, especially if the underlying pathophysiology is dissection. I strongly favor balloon guide catheters (BGCs) in all anterior circulation and most posterior circulation thrombectomies, provided that the BGC can be tracked across the tortuous extracranial segment to a stable position and the V2 segment is sufficiently large in caliber to accommodate balloon inflation. I use a 9-F short sheath and 9-F Cello BGC (Fuji Systems Corporation), tracked over a 125-cm Tempo vertebral catheter (Cordis, a Cardinal Health company) and a 0.035-inch, 180-cm Glidewire Advantage guidewire (Terumo Interventional Systems). I find the combination of a 125-cm VTK catheter and Glidewire Advantage guidewire helpful to handle brachiocephalic, right subclavian,
right vertebral artery tortuosity. Because one is dealing with a closed system of thrombus and proximal vessel occlusion (once the balloon is inflated in the BGC), akin to a supraclinoid internal carotid artery occlusion proximal to the posterior communicating artery origin, it may be possible to avoid intracranial access by using a proximal aspiration thrombectomy technique: inflating the BGC in the V2 segment and reversing flow through Max aspiration tubing (Penumbra, Inc.). In the meantime, I would prepare for intracranial access in case this initial approach fails by tracking an Ace 68 reperfusion catheter into the basilar artery over a Velocity microcatheter and Fathom-16 microguide wire (Boston Scientific Corporation). The latter two would ideally be positioned in one of the P1-2 segments in the event that anchoring with a stent retriever is required to help advance the Ace 68 catheter.

**Dr. Froehler:** Given the extremely dilated diameter of this vessel, it is impossible to perform conventional mechanical thrombectomy. Some have tried intra-arterial thrombolitics, although this is anecdotally associated with an extremely high risk of hemorrhagic complication and I would strongly advise against it. Rather, a modified mechanical thrombectomy approach should be utilized. I would begin with a goal of partial recanalization, as the vessel is too large to expect complete removal of all clot. A large stent retriever (6-mm diameter) could be tried, but in particularly large fusiform vessels, one could also consider side-by-side stent retrievers. This would necessitate two microcatheters in the basilar artery, with parallel deployment to effectively create a 12-mm-diameter thrombectomy system.

**CASE CONTINUED**

A Neuron Max guide catheter is advanced over a 5-F catheter to select the right vertebral artery. An Ace 68 reperfusion catheter is advanced over a 3MAX microcatheter (Penumbra, Inc.) and Synchro 14 microwire (Stryker). Mechanical thrombectomy of the intracranial vessel occlusion is performed using aspiration alone through the Ace 68 for the first pass. The aspiration catheter is advanced under fluoroscopic guidance into the thrombus to maximize clot engagement. No significant recanalization is achieved. A second pass consists of the same Ace 68 catheter but also tracking a Phenom 27 catheter (Medtronic) over a Synchro wire to select the left P1 segment. After removal of the wire, a 6- X 40-mm Solitaire stent retriever is advanced within the microcatheter and deployed from the left P1 segment to the mid basilar artery. Curiously, the stent retriever completely unravels within the fusiform basilar artery just prior to withdrawal into the Ace 68 catheter under aspiration.

**Dr. Jadhav:** Given the persistent large flow defect, I would advance the aspiration catheter to the face of

**What would you do next?**
- Continue with thrombectomy
- Intra-arterial alteplase
- Stent placement to jail the thrombus
- Observation

**Dr. Narayanan:** I would be hesitant to pursue additional stent retriever use in such a large, dysplastic vessel. I would consider additional thromboaspiration attempts with a 60-mL syringe or with vacuum aspiration tubing, ideally with the Ace 68 catheter tip directed into the thrombus. Depending on the ischemic stroke burden on preangiographic diffusion-weighted imaging, I might consider off-label use of a glycoprotein IIb/IIIa inhibitor to lyse refractory thrombus. This salvage technique has been useful in select patients. An eptifibatide 180 µg/kg intravenous bolus followed by a control angiogram 10 minutes later may demonstrate dramatic results in this challenging situation. Eptifibatide has a short half-life (~2.5 hours), which is preferable in a patient receiving multiple blood thinners, as this patient likely is.

**Dr. Froehler:** It is very curious that the stent retriever device unraveled. One wonders if this was a result of an intraluminal feature such as very hard clot or a fenestration versus a manufacturing defect. Partly because of the behavior of this device, I would not continue with thrombectomy. Intra-arterial thrombolitics are not indicated in these cases and create a significant risk for catastrophic hemorrhage. At this point, I would favor observation with repeat imaging and neurologic reassessment.

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the thrombus over the microcatheter/microwire system. After removal of the microcatheter/microwire, I would apply vacuum suction to the aspiration catheter to attempt further clot retrieval.

**CASE CONTINUED**

Not knowing the extent of posterior circulation infarction that may have occurred, we opt for observation. Repeat control angiographic runs after 30 minutes demonstrate the stability of the clot burden and persistence of anterograde filling of the distal posterior circulation. The patient is brought to the neurointensive care unit with consideration of surveillance catheter cerebral angiography and possible flow diversion embolization if his clinical status warrants it.

**How would you continue this patient’s management?**

- Modality and timing of surveillance imaging
- Use of anticoagulants
- Stent/flow diverter placement
- Observation

**Dr. Jadhav:** To guide further treatment planning, I would obtain an MRI to assess the stroke burden. If the stroke burden is small and there is no concern for craniectomy (e.g., suboccipital decompression for a large cerebellar infarct), I would place the patient on dual antiplatelet therapy (DAPT) and then plan for repeat angiography in 5 to 7 days.

**Dr. Froehler:** Repeat imaging after the procedure is essential to rule out hemorrhage as well as to assess infarct burden. MRI would be most appropriate, along with neurologic reassessment. If the patient’s condition improves, then I would start DAPT to encourage the remaining thrombus to shrink and support possible future vessel reconstruction with flow diversion.

**Dr. Narayan:** Anticoagulants. If significant thrombus burden persists, I prefer not to leave an implant, at least in the acute phase, which may serve as a nidus for rethrombosis even if the patient is on DAPT.

**CASE CONTINUED**

MRI of the brain shows small acute infarcts in the right lateral aspect of the pons and medial aspect of the right cerebellar hemisphere that is believed to be secondary embolization from the thrombosed aneurysm. The patient is started on a heparin drip. Over the next 4 days, his NIHSS score improves to 2. He remains intubated but nods appropriately, follows commands, has normal cranial nerves, and has no weakness other than right arm drift.

Because of his neurologic improvement and persisting concern for additional thromboembolic events, repeat catheter cerebral angiography is performed 5 days later, revealing a reduction of the size of the mid basilar artery intraluminal thrombus (Figure 4).

**What would you do at this point?**

- Thrombectomy
- Intra-arterial alteplase
- Stent placement to jail the thrombus
- Continue the heparin drip
- Observation

**Dr. Froehler:** The thrombus has shrunk with the use of heparin. I would favor antithrombotic treatment in the form of DAPT instead of heparin, as it is likely to be the chronic treatment choice. It is difficult to know what to do at this moment in this patient’s care. He has effectively had two ischemic events over the course of 2 days. One could argue that a trial of DAPT without endovascular treatment should be tried first because the thrombus has already gotten smaller and he has not had any further neurologic events. On the other hand, one more event could be fatal and vessel reconstruction could be performed now while the patient is doing relatively well. I have tried both approaches in similar patients and can only say that the choice is best made in the context of a thorough discussion with the patient and family.

If proceeding with arterial reconstruction using flow diversion, it is important to recognize that the diameter of the basilar artery is larger than the maximum diameter of flow diverter devices. It is necessary to ensure wall apposition at the proximal and distal ends of the construct. This can be achieved by beginning deployment in a posterior cerebral artery and building the construct back to the vertebral artery. However, I had a similar case where both posterior cerebral arteries were of internal carotid artery origin (fetal), and there was no acceptable distal landing zone. I used a “double-barrel” strategy with two Pipeline...
embolization devices (Medtronic) in parallel, similar to the stent retriever strategy I previously advocated. Although not ideal, this does enable vessel wall apposition in a dilated artery beyond 5 mm in diameter.

Dr. Narayanan: I would continue medical therapy for the first few weeks after the stroke to allow flexibility in decision-making and further healing of the ischemic injury. There is neurologic and angiographic improvement, so my preference is flow diversion in a subacute setting.

Dr. Jadhav: Given the patient’s neurologic improvement and small stroke burden, I would be concerned for vessel reocclusion related to rethrombosis of the fusiform basilar artery. Flow diversion would both maintain vessel patency and remodel the fusiform aneurysm. The choice of flow diverter would be guided by vessel size, but typically, I would favor placement of a Pipeline embolization device from the left P1 segment to the right V4 segment.

CASE CONCLUSION

Using a Neuron Max, Phenom Plus (Medtronic), and Phenom 27 over a Synchro wire, we select the dominant left P1 segment and proceed to deploy a series of three overlapping Pipeline embolization devices. Satisfactory wall opposition is achieved proximally and distally in the nonaneurysmal segments. There is no angiographic evidence of unintended iatrogenic branch vessel occlusion. The patient is discharged to acute inpatient rehabilitation 12 days after admission on DAPT.

At 1-year follow-up, the patient is alert and oriented to person, place, time, and situation. His dysarthria persists, but he has full strength in both arms and legs. His reflexes and muscle tone are increased in both legs and the left arm. He is ambulatory with a modified Rankin scale score of 2. The 1-year control catheter cerebral angiogram demonstrates significant improvement of the basilar artery fusiform dilatation (Figure 5). ■

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