



A Case Report of Endovascular Therapy of Acute Basilar Artery Occlusion: Beyond the Restricted Time Window

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Abstract— Approximately 60% of patients with acute basilar artery occlusion (BAO) have stuttering and progressive symptoms beyond 24 hours. Recent randomized trials have demonstrated benefit of endovascular therapy of large vessel occlusion in anterior circulation within 6 hours of symptom onset. Time window for endovascular therapy of BAO remains unknown. Case series and multicenter registry have shown potential benefit of recanalization within 24 hours of symptom onset. We describe a case of acute BAO after 2 weeks of recurrent stroke and its successful endovascular therapy. A 25-year-old female developed recurrent cerebellar and brainstem infarcts from bilateral vertebral artery dissections following chiropractic neck manipulation. Two weeks after her initial stroke, she became locked-in from acute BAO despite combined warfarin and aspirin therapy. After emergent reversal of warfarin effect, she underwent endovascular therapy without complications. She had complete functional recovery at 6-month follow-up. Our case report and literature review suggest that endovascular therapy of BAO can be safe and beneficial in patients with recurrent stroke beyond 24 hours.

Keywords — Basilar artery occlusion, stroke, endovascular therapy

I. INTRODUCTION

Acute basilar artery occlusion (BAO) is associated with poor outcome. With conservative therapy, the chance of recovery is next to zero in patients presenting with coma or tetraplegia.¹ Recent randomized endovascular trials have excluded posterior circulation stroke.^{2,3} The time window for endovascular therapy of BAO remains unclear. Case series and multicenter registry have shown that endovascular therapy within 24 hours of symptom onset was associated with favorable outcome in 34%

to 54% of patients.³⁻⁷ However, more than 60% of patients with proven BAO have progressive stroke beyond 24 hours.⁸ Such patients would be excluded from endovascular therapy. Given progressive course and grave outcome with conservative therapy, it is rational to consider individualized time window for endovascular therapy of BAO.

Here, we present a case of acute BAO and its successful endovascular therapy after 2 weeks of recurrent stroke.

II. CASE PRESENTATION

A 25-year-old right-handed female presented with neck pain, headache, and vertigo after chiropractic neck manipulation. MRI of the brain showed a left cerebellar infarct (Figure, A). CTA revealed irregularities of bilateral vertebral arteries (VA) at C1-C2 segment (Figure, B). She was started on Aspirin 81 mg and Lipitor 80 mg daily for stroke prevention. A day later, she developed dysarthria and decreased hearing in both ears. Repeat MRI showed new infarcts in bilateral cerebellar hemispheres (Figure, C & D). She was switched from aspirin to IV heparin and transferred to our stroke center for higher level of care. Cerebral angiography confirmed bilateral VA dissections without other vascular anomaly. Hypercoagulable state and cardiac workup were unremarkable. Her exam stabilized with only mild hearing loss and dysarthria. She was discharged home on warfarin.

Two weeks later, she presented with transient episodes of headache, tinnitus, horizontal diplopia, vertigo, and left hemiparesis. Vertigo and left hemiparesis resolved after arrival in ED but diplopia persisted. MRI brain showed new infarcts in bilateral middle cerebellar peduncles, right pons and rostral medulla (Figure, E). Since she was symptomatic with therapeutic INR (2.4), Aspirin 81 mg daily was added to warfarin therapy. On the following day, she became locked-in with dense quadriplegia and inability to speak. She was only able to blink to simple commands. Stat CTA demonstrated complete proximal BAO (Figure, F).

III. ENDOVASCULAR THERAPY AND OUTCOME

After informed consent from patient's family, the patient was given prothrombin complex concentrates (Profilnine SD) 25

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units/kg and Phytonadione 10 mg intravenously for reversal of warfarin-related coagulopathy (INR 2.84). Heparin drip was started with a target aPTT of 45-60 seconds to minimize periprocedural thrombosis. Under sterile condition, the right common femoral artery was accessed with a 19-gauge needle. A 6 French 10 cm vascular sheath was placed into the right common femoral artery over a guidewire utilizing Seldinger technique. Under fluoroscopic guidance a 5 French angled taper glide diagnostic catheter was navigated into the aortic arch followed by diagnostic angiogram. For thrombectomy

from the occluded basilar artery, the guiding catheter, a 6 French neuron 95 CM, was prepared with Cook Vert diagnostic catheter with glide guidewire. The system was carefully navigated into the left subclavian artery. Under real-time roadmap images, the system was carefully navigated into the left vertebral artery. Successfully guiding catheter was positioned in the high cervical segment (V2 segment). At this point pre-procedure angiogram of the left vertebral artery cerebral view was performed. 50 microgram of Nitroglycerin

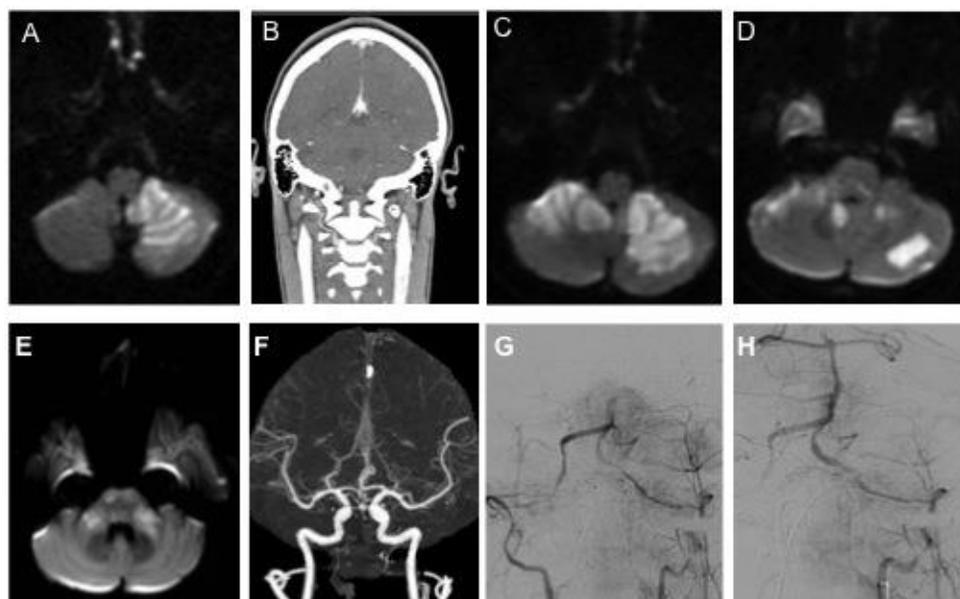


Figure. BAO from bilateral VA dissection 2 weeks after recurrent stroke and endovascular recanalization

Initial MRI DWI showed L cerebellar infarct (A). CTA revealed irregularities of bilateral VAs at C₁-C₂ segment, suggestive of dissection (B). One day later, new infarcts in bilateral cerebellum (C & D). 2 weeks later, recurrent infarcts in middle cerebellar peduncles and right lower pons (E). On the following day, CTA and conventional angiogram showed proximal BAO (F & G). Successful BA recanalization following thrombectomy (H).

was infused into the left vertebral artery. Penumbra 3 Max aspiration system was carefully navigated into the left vertebral artery intra-dural segment. There was some resistance to advance the catheter secondary to a small caliber of the artery and the dissection, However, we were able to successfully pass through the dissection site and able to reach the basilar artery. 3 Max was connected to the patient pump and several passes of aspiration were performed. Thrombectomy was successfully performed using the Penumbra device with TIC1 score 2b without complications (Figure, G & H). The diagnostic catheter was advanced into left VA via aortic arch and left subclavian artery for a post procedure angiogram. The puncture site was then treated with StarClose after removing the vascular sheath.

Following the procedure, the patient was extubated next

morning. On exam, she was alert and oriented with moderate dysarthria, horizontal diplopia, dysmetria, and mild left hemiparesis. IV heparin was discontinued after loading with 300 mg clopidogrel. In addition to clopidogrel 75 mg and aspirin 325 mg daily, she was instructed to wear a soft cervical collar for 3 months to minimize trauma to the dissected arteries. She improved gradually and was discharged home from acute rehabilitation unit 4 weeks after thrombectomy. At 3-month follow-up, she had only mild residual diplopia and dysmetria on the left. Aspirin was decreased to 81 mg daily and clopidogrel was continued for a total of 6 months. Repeat CTA showed distal left VA occlusion without abnormality in right VA and BA. MRI showed no new infarct. She had complete functional recovery at 6 months.



IV. DISCUSSION

Our case is unique in that the patient had acute BAO with locked-in syndrome after 2 weeks of recurrent stroke from bilateral VA dissections, despite maximal medical therapy with both warfarin and Aspirin. Per conventional wisdom and current practice guidelines,² she would be excluded from endovascular therapy and her chance of functional recovery would be close to zero.¹

Previous reports by us and other investigators have suggested feasibility and safety of endovascular therapy of BAO beyond 24 hours of symptom onset.⁹⁻¹⁴ The prolonged time window for intervention of BAO could be attributed to relatively low risk of symptomatic intracerebral hemorrhage, better collaterals, and higher ischemic tolerance in the posterior than in the anterior circulation.

Intracerebral hemorrhage is the most feared complication of endovascular therapy.¹⁵ Abou-Chebl was the first to propose endovascular therapy with no time window in appropriately selected patients. In his single center studies, he demonstrated no difference in symptomatic intracerebral hemorrhage between early (3.4 ± 1.6 hours) and late (18.6 ± 16.0 hours) recanalization (8.8% vs 9.5%).¹² In a study of intravenous thrombolysis, patients with posterior circulation stroke were also found to have less symptomatic intracranial hemorrhage (0% versus 5%, $P=0.026$) and better outcome (66% versus 47%, $P<0.001$) than those with anterior circulation stroke.¹⁶ After multivariable adjustment, posterior circulation stroke was an independent predictor of lower symptomatic intracranial hemorrhage frequency (sICH) ($P=0.001$). Increased onset-to-treatment window did not influence the rate of sICH.¹⁸ Smaller infarct volumes may be a reason for lower sICH rates in posterior circulation stroke. In the BASIC study, symptomatic ICH was significantly more common in patients with pc-ASPECTS < 8 ,¹⁹ and the vast majority of patients with sICH died. Concomitant treatment with full-dose heparin may play a role. Kumar et al. performed a meta-analysis of recanalization of acute BAO. Of the 2056 patients with BAO from 45 studies, the ICH rate from endovascular therapy was 13%.²⁰ In the absence of extensive infarction, recanalization of BAO up to 48 hours was seldom futile and produced good outcomes in 50% of patients.

In addition to lower hemorrhagic complications, collateral flow through the posterior communicating or the cerebellar arteries may lead to a slower evolution of irreversible ischemia in the posterior circulation. Brainstem consists of mainly white matter and is therefore more resistant to ischemia than cortical brain tissue.

In the largest case series of delayed revascularization of BAO, the risk of periprocedural mortality was 22%.¹¹ Given the grave natural history of patients with locked-in syndrome from BAO,¹ the outcome of delayed endovascular therapy is still better than conservative therapy.

BAO is a heterogeneous stroke syndrome with severity ranging from minor stroke to coma and with time course from stuttering progression to sudden unresponsiveness. It is

therefore pivotal to have individualized time window for therapy according to evidence based medicine and benefit/risk ratio analysis. For patients presenting with acute symptoms, intravenous thrombolysis is recommended within 4.5 hours and endovascular therapy within 6 hours of symptom onset. For patients presenting within 6-24 hours of symptom onset, endovascular therapy should be considered for BAO. For patients with stuttering and progressive symptoms beyond 24 hours, endovascular therapy can be considered if there is no devastating infarct on imaging studies.

V. CONCLUSIONS

In summary, our case report and literature review suggest individualized time window for endovascular therapy of acute BAO. Given grave outcome of acute BAO with conservative therapy, the benefit of endovascular therapy of BAO beyond 24 hours of symptom onset may outweigh the risk of complications.

Footnotes

The University of California Irvine institutional review board determined that this case report did not meet the federal definition of research with human participants, and institutional review board approval was not required. The patient has kindly granted her consent for the publication of this case report.

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Questions (please choose one single answer):

1. Endovascular therapy is a proven therapy for which of the following strokes
 - A. Acute stroke from large vessel occlusion in the anterior circulation within 6 hours of symptom onset
 - B. Small vessel occlusion within 6 hours of symptom onset
 - C. Acute basilar artery occlusion
 - D. Lacunar infarct
2. The treatment of choice for acute stroke from basilar artery occlusion includes
 - A. IV tPA within 4.5 hours of symptom onset
 - B. SBP < 180/105
 - C. Endovascular therapy within 6 hours of symptom onset
 - D. All of above.
3. The prognosis of a patient with coma or tetraplegia from acute basilar artery occlusion is:
 - A. Excellent with conservative therapy
 - B. Excellent with iv Heparin
 - C. Excellent with iv tPA
 - D. Close to zero without recanalization
4. Based on the case report and literature review, patient with locked-in syndrome from acute basilar artery occlusion beyond 24 hours can be considered for
 - A. IV heparin
 - B. IV tPA
 - C. IA tPA
 - D. Endovascular therapy

Correct answers:

1. A. 2. D. 3. D. 4. D.