Successful Endovascular Recanalization of Chronic Basilar Artery Occlusion

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Abstract—Acute basilar artery (BA) occlusion may cause devastating stroke or locked-in syndrome. The rarity of acute BA occlusion, however, often results in delayed diagnosis and inadequate treatment. The feasibility and benefit of revascularization in chronic stage is unknown. We report a case of successful endovascular recanalization of chronic BA occlusion. A 20 years old man presented with dysarthria and quadriparesis. He had acute BA occlusion at age 7 and was in a locked-in state for almost 3 years. The patient then experienced gradual improvement over the years but remained severely disabled. His family searched for revascularization therapy relentlessly in the last 13 years. After informed consent, the patient underwent endovascular recanalization of the chronically occluded BA without complication. He had small but appreciable improvement in coordination following the procedure. CT angiography at 3 month follow-up showed patent BA. Endovascular recanalization of chronic BA occlusion appears feasible and potentially safe. Further study is warranted.

Keywords—Basilar artery occlusion, endovascular recanalization, stenting, and stroke.

I. INTRODUCTION

Basilar artery (BA) supplies brain stem, cerebellum, thalami, occipital lobes, and medial temporal lobes. Acute BA occlusion causes ischemic damage to the basis pontine and results in devastating locked-in syndrome (LIS) which is characterized by quadriplegia, anarthria, and preserved consciousness.1-3 The diagnosis of acute BA occlusion is often delayed.3-6 The prognostic of LIS from acute BA occlusion is very poor. In adults, mortality was reported as high as 75%.3 In children, mortality was reported as low as 23%, partly due to lower rate of withdrawal of life support.6 Survivors of LIS may experience slow, gradual but incomplete improvement.3,6 Although thrombolysis and/or endovascular therapy has proven to be effective for acute BA occlusion,2,7 there has been no report on revascularization of a remote BA occlusion.

II. CASE REPORT

A 20 years old man presented for an endovascular procedure. He had a confirmed diagnosis of locked-in syndrome from BA occlusion at age 7. Six weeks after a mild trauma from playing soccer, he developed severe headache, nausea, speech difficulty, unsteady gait, and screaming spells. A CT scan was reported normal. He was treated with phenytoin for possible seizure activities at a local hospital. In the next few days, he became locked-in. MRI and SPECT showed a lesion in the pons. Four months later, repeat MRI with MRA showed old pontine infarct and mid BA occlusion. The patient was in the locked-in state for 3 years followed by a slow and gradual improvement over the last 10 years.

The patient’s parents had relentlessly searched for therapy for the patient, particularly after learning case reports of successful recanalization of subacute or chronic BA occlusion.8,9 At the time of evaluation at our medical center, the patient was awake and oriented to person, place and time with severe dysarthria. His pupils were equal and reactive to lights and the extraocular movements were full. His motor strength at left side was 3/5 (Medical Research Council) proximally and 1/5 distally in both upper and lower extremities while the right side were 4/5 proximally and 2/5 distally. He had significant contractures in both hands and feet. He was able to make a series of static slow movements at the right side. His sensation was intact to light touch.

After informed consent, the patient was loaded with 300 mg clopidogrel and 325 mg aspirin. Diagnostic digital subtraction angiography (DSA) showed mid BA occlusion (Fig 1A) and reconstitution of bilateral posterior cerebral arteries (PCA) and superior cerebellar arteries (SCA) via right posterior communicating artery. Roadmap fluoroscopy with synchronous right vertebral artery (VA) and internal carotid artery (ICA) injections demonstrated BA occlusion from origin of anterior inferior cerebellar arteries (AICA) to SCA ostia (Fig 1B). The occluded BA segment was then traversed by the use of a microcatheter over a guidewire from the basilar apex via the right ICA. A microcatheter exchange allowed navigation of a Gateway Balloon (Boston Scientific, Fremont, California) to the site of occlusion. The balloon was subsequently exchanged for the Wingspan Stent System (Boston Scientific, Natick, MA). The balloon and stent were sized based on the proximal BA diameter and the length of the occluded segment. A single
stent (4.5 x 20 mm) was deployed to reopen the distal BA (Fig 1C). Intravenous heparin was administered during the procedure to maintain an activated clotting time between 2 and 2.5 times normal. Systolic blood pressure was kept at 90-120 mmHg during and after the procedure to prevent hyperperfusion syndrome. The patient had no new neurologic deficit after the procedure. He complained headache a few hours post procedure, which was controlled with pain medications. Follow-up brain MRI showed no new infarct or hemorrhage (Fig 1D and 1E). He was discharged on aspirin 325 mg and clopidogrel 75 mg daily.

After revascularization procedure, he experienced appreciable improvement. He reported easier speech initiation and postural control. Within 2-3 weeks, he was able to turn from one side of his body to the other on the bed within 2 minutes as compared to 15 minutes before the procedure. At 5 month follow-up, he had appreciable improvement in his Motomed bicycle performance. His average readings were 17-33 W compared to pretreatment baseline at 9-25 W. There was no significant improvement in muscle strength.

Follow-up CT angiography at 3 month showed a patent BA (Fig 1F). Clopidogrel was discontinued after 3 month’s therapy. He was instructed to take aspirin 325 mg daily for life.

III DISCUSSIONS

LIS from acute BA occlusion portends poor prognosis.\(^3\)\(^,\)\(^4\)\(^,\)\(^5\) However, there have been case reports of LIS-patients with substantial clinical improvement.\(^6\)\(^,\)\(^7\) The mechanisms for delayed improvement remain poorly understood. Development of collateral circulation, neuronal regeneration and remyelination may play a role. If development of collateral circulation contributes to functional recovery, revascularization of a chronically occluded BA may be beneficial for functional recovery or increased pace of recovery.

Despite locked-in for almost 3 years, our patient eventually showed slow and gradual improvement. It is unknown whether the patient would do much better if he had revascularization earlier. It is also unclear whether re-open of the chronically occluded BA at such a late stage would improve perfusion in posterior circulation and pace of recovery.

Our case study has significant implications. First, it demonstrates that endovascular recanalization of chronic BA occlusion is feasible and potentially safe even 13 years after occlusion. Second, despite delayed revascularization, the patient still showed small but appreciable improvement in movement and coordination.

Of note, revascularization of chronic BA occlusion can be risky.\(^10\)\(^,\)\(^11\) In a recent case series of 9 patients, the periprocedural mortality was as high as 22%.\(^10\) However, due to dismal natural history and prolonged suffering from symptomatic BA occlusion, any potential therapy warrants further study.

References

6. Bruno MA, Schnakers C, Damas F, Pellás F, Lutte I, Bernheim J, Majerus S, Moonen G, Goldman S, Laureys S. Locked-in syndrome in children: report of five cases, the periprocedural mortality was as high as 22%.\(^10\) However, due to dismal natural history and prolonged suffering from symptomatic BA occlusion, any potential therapy warrants further study.

Fig. 1. DSA with right VA injection showed mid-BA occlusion (A). Synchronous right VA and ICA injection demonstrated BA occlusion between AICA origin and SCA ostia (B). The occluded BA was traversed and stented (C). Post-stenting MRI followed showed old pontine infarct without bleed or new ischemic changes (F). CT angiography at 3 month follow-up showed patent BA without in-stent restenosis.

Questions

1. The common vascular etiology of locked-in syndrome is

A. Internal carotid artery occlusion
B. Acute basilar artery occlusion
C. Middle cerebral artery occlusion
D. Cardiac arrest

2. The treatment of choice for acute stroke from basilar artery occlusion includes

A. IV tPA within 3-4.5 hours of symptom onset
B. IA tPA
C. Endovascular recanalization
D. All of above.

3. Locked-in syndrome is caused by ischemic damage to which of the following:

A. Mid-brain
B. Left frontal lobe
C. Basis pontine
D. Left temporal lobe.

4. The effective treatment of chronic basilar artery occlusion is:

A. IA t-PA
B. EC-IC bypass
C. CEA
D. Unknown

Correct answers: