Basilar artery occlusion successfully treated with delayed intravascular intervention and mild hypothermia

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To the Editor: Acute basilar artery occlusion (BAO) is a catastrophic variant of stroke, which accounts for about 1% of all strokes.[1] Though rare, BAOs are associated with high morbidity and mortality rates.[2] Although treatable, the rate of death or disability associated with BAO is still as high as 80%.[2] Early recanalization either with thrombolysis or intravascular thrombectomy was shown to significantly improve the outcome of patients with BAO.

Animal stroke models have shown that post-ischemic induction of hypothermia significantly reduces the final infarct volume. Moderate hypothermia suppresses the post-ischemic generation of oxygen free radicals and inflammatory responses known to play a role in "reperfusion injury." Therapeutic hypothermia was suggested to be the most potent neuroprotective strategy, which simultaneously activates numerous pathways by several mechanisms during ischemic and reperfusion window.[3] Based on the robust and consistent laboratory data, clinical studies on therapeutic hypothermia for patients with acute ischemic stroke have been designed. Recent studies have demonstrated that therapeutic hypothermia is safe and achievable in patients with acute ischemic stroke. Definite efficacy trials of therapeutic hypothermia combined with artery recanalization in acute ischemic stroke are ongoing.

Herein, we presented a case of a BAO that was successfully treated by intravascular intervention and therapeutic hypothermia.

A 49-year-old Chinese male patient was transferred to the Emergency Department (ED) of Nanfang Hospital, Southern Medical University, in deep coma. He had complained of sudden onset of dizziness, vertigo, numbness of bilateral arms, and legs and slight weakness of both his upper and lower extremities before 16 h. He was admitted to a local hospital and cranial computed tomography (CT) revealed no abnormalities. Acute stroke of the posterior circulation was suspected and anti-platelet therapy was initiated. The neurological status of the patient deteriorated with decreased level of consciousness 9 h before his arrival in our ED. He was quickly transferred to our institute.

Initial ED assessment revealed that the patient was comatose with Glasgow coma scale (GCS) of 4 (E1V1M2). His vital signs were stable with temperature of 37°C, blood pressure of 130/75 mmHg, heart rate of 86 beats/min and respiratory rate of 18 breaths/min.

Prompt cranial CT revealed hypointensity of the right cerebellum [Figure 1A]. Hence, BAO was highly suspected. With the informed consent of the family members of the patient, the patient was orally intubated and transferred to our interventional group for urgent vascular evaluation.

Initial angiography via the left vertebral artery revealed upper and middle occlusions of the basilar artery along with the occlusions of bilateral superior cerebellum arteries and right posterior cerebral artery [Figure 1B]. Left posterior cerebral artery was visible via opening of the left posterior communication artery [Figure 1C]. Urgent thrombectomy was initiated. A Solitaire-FR 6 mm × 30 mm stent (Irvine, CA, USA) was deployed from the right P2 segment to the mid-basilar trunk and slowly withdrawn during simultaneously guiding catheter aspiration. Two passes were made with the pulled thrombosis of 3 mm × 10 mm [Figure 1D]. Recanalization was achieved 20 h from onset of the disease with thrombolysis in cerebral infarction (TICI) grade 3 [Figure 1E]. During the procedure, 5 mg tirofiban was administrated to prevent thrombosis.

Following recanalization, the patient was transferred to the neuro-intensive care unit. He was still in coma with GCS score of 4 (E1VTM2). Mechanical ventilation was

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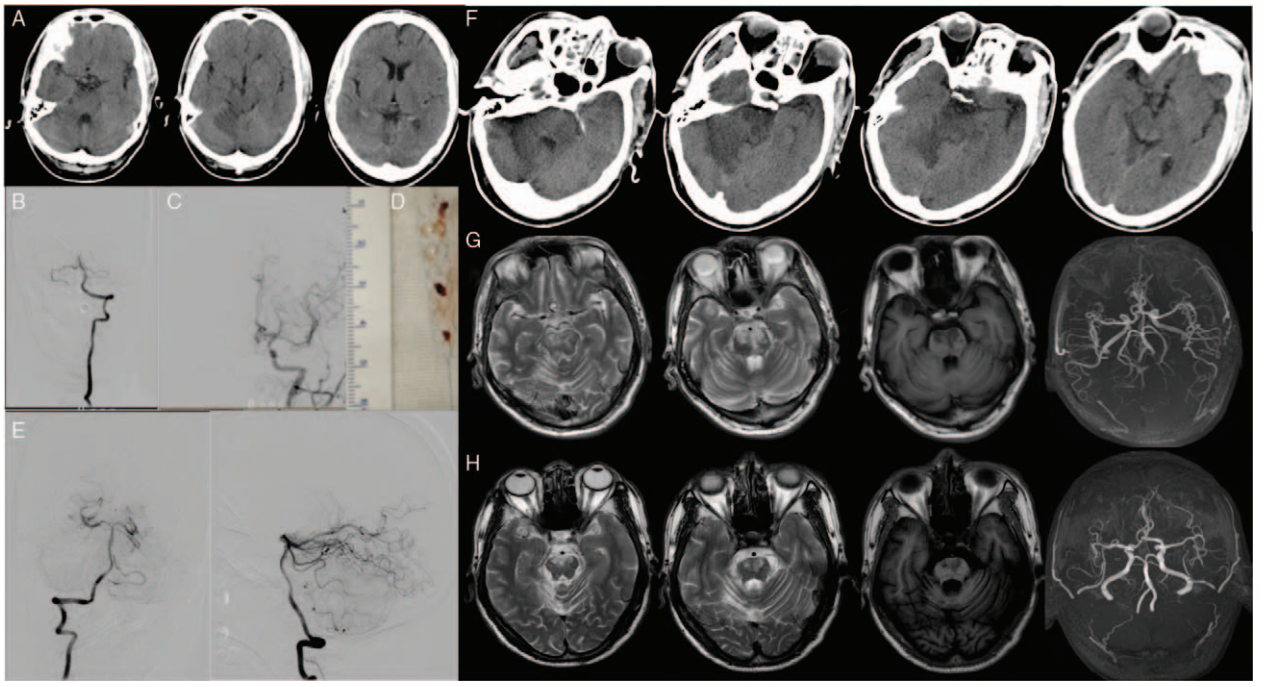

Figure 1: Cranial CT and DSA images of the patient. CT revealed hypointensity of the right cerebellum (A). Initial angiography via the left vertebral artery revealed upper and middle occlusions of the basilar artery (B). Angiography via the left internal carotid artery revealed that left posterior cerebral artery was visible via opening of the left posterior communication artery (C). (D) Thrombosis of 3 mm × 10 mm pulled by Solitaire-FR 6 mm × 30 mm stent. Successful recanalization was achieved after the procedure (E). On day 4 post-procedure, CT confirmed hypointensity of the pons and right cerebellum (F). Two weeks post-procedure, MRI revealed infarcts of the bilateral midbrain, pons, and right cerebellum. MRA confirmed the presence of basilar artery (G). At the 6-month follow up, MRI revealed old infarcts of the bilateral midbrain, pons and right cerebellum. MRA was normal (H). CT: Computed tomography; DSA: Digital subscription angiography; MRI: Magnetic resonance imaging; MRA: Magnetic resonance angiography.

provided to support his respiration. To avoid the reperfusion injury of the brainstem, the patient was given intravascular mild hypothermia with target temperature of 33°C (COOLGARD3000, Alsius, San Jose, USA). Intravascular hypothermia is based on the technology of intravascular heat exchange cooling. The principle of the technology is to use an interventional method to insert a temperature control catheter into deep human veins (such as subclavian vein to superior vena cava, or femoral vein to inferior vena cava), directly cooling and rewarming the blood. The system consists of four parts, including an *in vitro* cooling device, a closed catheter, an Icy catheter with heat exchange in deep veins and a catheter with a central body temperature probe. The target temperature of 33°C was achieved within 2 h and lasted for 12 h. Then, the patient was gradually rewarmed to 36.5°C at the rate of 1°C/12 h (0.1°C/h for 10 h and keep the target temperature for 2 h). Midazolam was used for sedation and vecuronium for neuromuscular blockade.

On day 4 after recanalization, the patient had fever with peak temperature of 38.6°C. Chest X-ray confirmed the diagnosis of pneumonia involving bilateral lower lobes. Sputum culture revealed *Klebsiella peumoniae* infection. Antibiotics were given according to the susceptibility results. The patient was extubated on day 11 post the recanalization. The neurologic status of the patient improved gradually. Fourth day post the procedure, the patient's NIH Stroke Scale (NIHSS) score was 26. A post-procedure CT confirmed the hypointensity of the pons and right cerebellum [Figure 1F]. Further evaluations of the risk factors for the patient revealed heavy smoking, but no hypertension, diabetes, hyperlipidemia, atrial fibrillation, cardiac source of embolism, vasculitis, and thrombophilia.

Two weeks post-procedure, the magnetic resonance imaging (MRI) of the brain confirmed the presence of the infarcts of the bilateral midbrain, pons and right cerebellum. Magnetic resonance angiography (MRA) confirmed the presence of basilar artery [Figure 1G]. The patient was transferred to a local hospital for rehabilitation with NIHSS score of 13 and modified Rankin Scale (mRS) score of 5.

The patient was advised to regularly take anti-platelet medicines and quit smoking. During the follow-up, the patient gradually improved with mRS score of 3 at the third month and mRS score of 2 at the sixth month. The MRI evaluation revealed old infarcts of the bilateral midbrain, pons, and right cerebellum [Figure 1H].

BAO is rare, accounting for about 1% of all strokes.[1] The estimated annual incidence of BAO is approximately one patient per 100,000, or a maximum of a few individuals per 100,000.[4] Patients are usually elderly, although younger people and even children can also have BAO. Though rare, BAO is devastating for the patient and the family.[2] The clinical presentation of BAO varies from mild transient symptoms to stroke. Vertigo and headaches are the most common prodromal, followed by the hallmarks of BAO, including decreased consciousness, quadriparesis, pupillary and oculomotor abnormalities, dysarthria, and dysphagia.[4] Clinicians should recognize the prodromal and closely monitor the patient. If available,

multimodal CT or MRI should be promptly performed to confirm the diagnosis of BAO and appropriate treatment for the patient should be given without delay in the diagnosis. Our patient lived in a remote rural area with limited medical resources. The diagnosis of BAO was delayed though suspected. Close cooperation between comprehensive stroke center and local hospital should be encouraged to help the patients.

Early recanalization of BAOs was shown to improve the outcomes. This was achieved using intravenous or intra-arterial thrombolytic agents, thromboaspiration, and/or thrombectomy,[5] but the best treatment approach remains unknown. A recent systematic review and meta-analysis revealed that stent retriever thrombectomy achieved a high rate of recanalization (TICI 2b-3, 80.0%) and functional independence (mRS≤2, 42.8%) while being relatively safe for patients with BAO.[5]

Optimal time window of thrombectomy for BAO remains unknown. Recanalization of BAOs beyond standard treatment window is reported.[5] For selected patients with favorable prognostic factors such as young age, small core infarct and collateral circulation, recanalization beyond the treatment window may be beneficial. Since our patient is young and previously healthy, with opening of the left posterior communication artery, urgent thrombectomy was conducted.

Experimental stroke animals have proven the efficacy of hypothermia.[6] Therapeutic hypothermia coupled with reperfusion strategies may yield synergistic benefits for patients with stroke.[3] In a recent study involving patients with anterior circulation treated with successful recanalization, the hypothermia group ($n=39$) had less cerebral edema ($P=0.001$), hemorrhagic transformation $P=0.016$), and better outcome ($P=0.017$) as compared to the normothermia group ($n=36$). Mortality, hemicraniectomy rate, and medical complications were not statistically different. After adjustment for potential confounders, therapeutic hypothermia (odds ratio, 3.0; 95% confidence interval, 1.0–8.9; $P=0.047$) was an independent predictor for favorable outcome.[7] This patient was recanalized 20 h after the symptom onset, and the reperfusion injury may be quite obvious. Based on these studies, we conducted intravascular hypothermia for the patient. The patient achieved a favorable outcome 6 months after the procedure.

We described a case of a 49-year-old Chinese male patient with BAO who benefitted from delayed thrombectomy and mild hypothermia. Therapeutic mild hypothermia combined with successful recanalization may improve the prognosis of patients. Since this is a case report, the conclusions should be interpreted carefully and more research was warranted. Large randomized clinical trials are required to elucidate the efficacy of thrombectomy and mild hypothermia for treating BAO.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s)/patient's guardians has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the article. The patients/patient's guardians understand that their names and initials will not be published and due efforts will be made to conceal the identity of the patient, although anonymity cannot be guaranteed.

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Conflicts of interest

None.

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