

Endovascular Intervention in Basilar Artery Entrapment within the Longitudinal Clivus Fracture: A Case Report

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Abstract

Although vascular complications after head trauma is well recognized, basilar artery entrapment within the longitudinal clivus fracture is rare. A 69-year-old man presented with progressive disturbance of consciousness and right hemiplegia after trauma. Computed tomography scan showed a right-sided acute subdural hematoma and multiple skull fractures, including a longitudinal clivus fracture. Magnetic resonance imaging revealed basilar artery occlusion and a small infarction at the ventral part of the pons. On the assumption of acute arterial occlusion caused by thrombus, endovascular thrombectomy was attempted, but resulted in perforation. After the procedure, basilar artery entrapment within the longitudinal clivus fracture turned out to be the cause of the occlusion. The present case suggests that basilar artery entrapment within the longitudinal clivus fracture is a possible cause of neurological deficits after trauma. In this subset, endovascular intervention without a correct diagnosis of this phenomenon is high risk.

Key words: basilar artery entrapment within the longitudinal clivus fracture, endovascular intervention

Introduction

Basilar artery entrapment within the longitudinal clivus fracture is a rare vascular complication after trauma. In this condition, basilar artery is sandwiched by the fractured clivus bones, resulting in arterial occlusion or stenosis. Despite the poor prognosis due to posterior circulation infarction,¹⁾ the diagnosis is difficult in most cases and appropriate treatment has not been established.^{2,3)}

Here, we report a case of basilar artery entrapment within a longitudinal clivus fracture, for which we attempted endovascular intervention based on misdiagnosis.

Case Report

A 69-year-old man was transferred to our hospital due to a fall from stairs. On admission, the patient

presented with Glasgow Coma Scale (GCS) score of 13 and slight right hemiparesis. A computed tomography (CT) scan showed a right-sided acute subdural hematoma and frontal lobe contusion (Fig. 1A). The sphenoid sinus and the ethmoid sinus were filled with blood. On the bone window, multiple skull fractures were detected: a longitudinal clivus fracture extending to the sphenoid sinus, a right frontal bone fracture involving the skull base, a right temporal bone fracture, and a right sphenoid bone fracture at the lesser wing (Fig. 1B).

After 13 hours of conservative treatment, his consciousness showed a sharp decline to a GCS score of 5, and his right hemiparesis got worse to complete hemiplegia. Brain MR imaging was performed immediately. Basilar artery was occluded at the proximal segment, and acute infarction on the left side of the ventral pons was detected (Fig. 1C). On the assumption of acute basilar artery occlusion caused by thrombus, endovascular intervention was performed to save the ischemic penumbra in the posterior circulation.

Right vertebral angiography showed basilar artery occlusion at the middle segment (Fig. 2A). Perfusion

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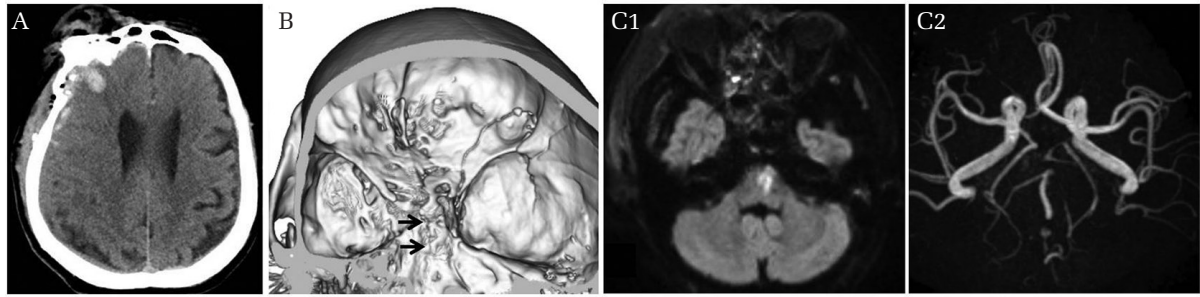


Fig. 1 (A) Computed tomography scan showing the right-sided acute subdural hematoma and the right frontal lobe contusion. (B) Three-dimensional reconstruction of the bone window of the computed tomography scan showing multiple skull base fractures, particularly a longitudinal clivus (*arrow*) fracture. (C) Magnetic resonance images. (1) Diffusion-weighted image showing the acute infarction in the left caudal pons. (2) Magnetic resonance angiogram showing the occlusion of the basilar artery.

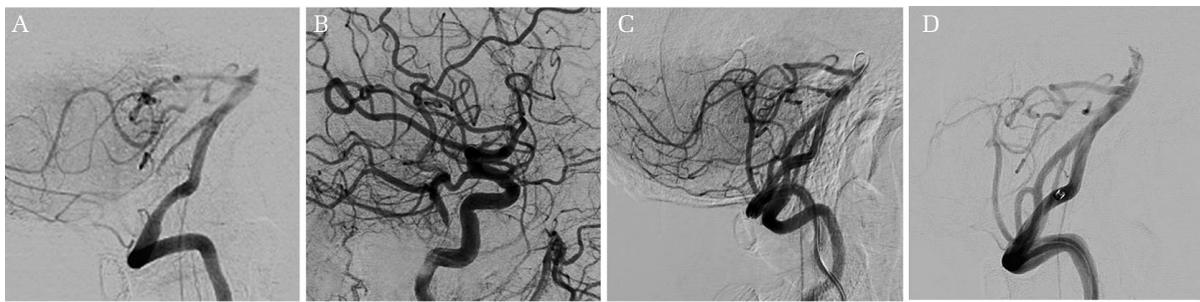


Fig. 2 Right vertebral angiogram demonstrating the occlusion of the basilar artery at the middle segment. (A) Right common carotid angiogram showed that the distal segment of the basilar artery was filled through the right posterior communicating artery. (B) Right vertebral angiogram after lesion crossing showing the deviation of the microguidewire from the basilar artery. (C) Right vertebral angiogram after the failure of lesion crossing showing the leakage of contrast agent from the basilar artery.

for distal basilar artery was preserved by collateral flows through posterior communicating artery (Fig. 2B). A direct-aspiration first-pass technique with a 5 French aspiration catheter (5Max Ace; Penumbra, Alameda, CA, USA) was attempted first, but could not catch the thrombus. Then, we tried to cross the occlusion site with a j-shaped microguidewire (Chikai 14; Asahi Intecc, Aichi), but the site was too hard to cross, resulting in perforation (Fig. 2C, 2D). The extravasation stopped spontaneously, and we finished the procedure at that point. A post-procedural CT angiogram revealed that the occluded part of basilar artery is trapped in the longitudinal clivus fracture (Fig. 3).

We did not initiate antiplatelet or anticoagulant therapy because of the intracranial hemorrhage and other traumatic complications. The patient's neurologic condition improved gradually to GCS score of 10, leaving severe right hemiparesis. About 6 months after this event, he is now under rehabilitation aiming for home life.

Discussion

Basilar artery entrapment within the longitudinal clivus fracture is a rare vascular complication after trauma. As subtypes, vertebral artery entrapment within the clivus and basilar artery herniation into the sphenoid sinus are known. Seventeen cases have been reported previously, and those outcomes are poor (Table 1).¹⁻¹⁷⁾ Of these 17 patients, 7 died, 1 remained in a vegetative state, 3 suffered from locked-in syndrome, and 5 had some paralysis.

The mechanism of basilar artery entrapment within the longitudinal clivus fracture is proposed as follows: (1) the trauma, which causes arrest of forward motion of the skull, produces a longitudinal clivus fracture and lacerates the dura; (2) inertia of the brainstem and cerebellum, which continue to move forward relative to the skull, thrust the basilar artery into the breach of the longitudinal clivus fracture; and (3) the longitudinal fracture then closes while inserting basilar artery

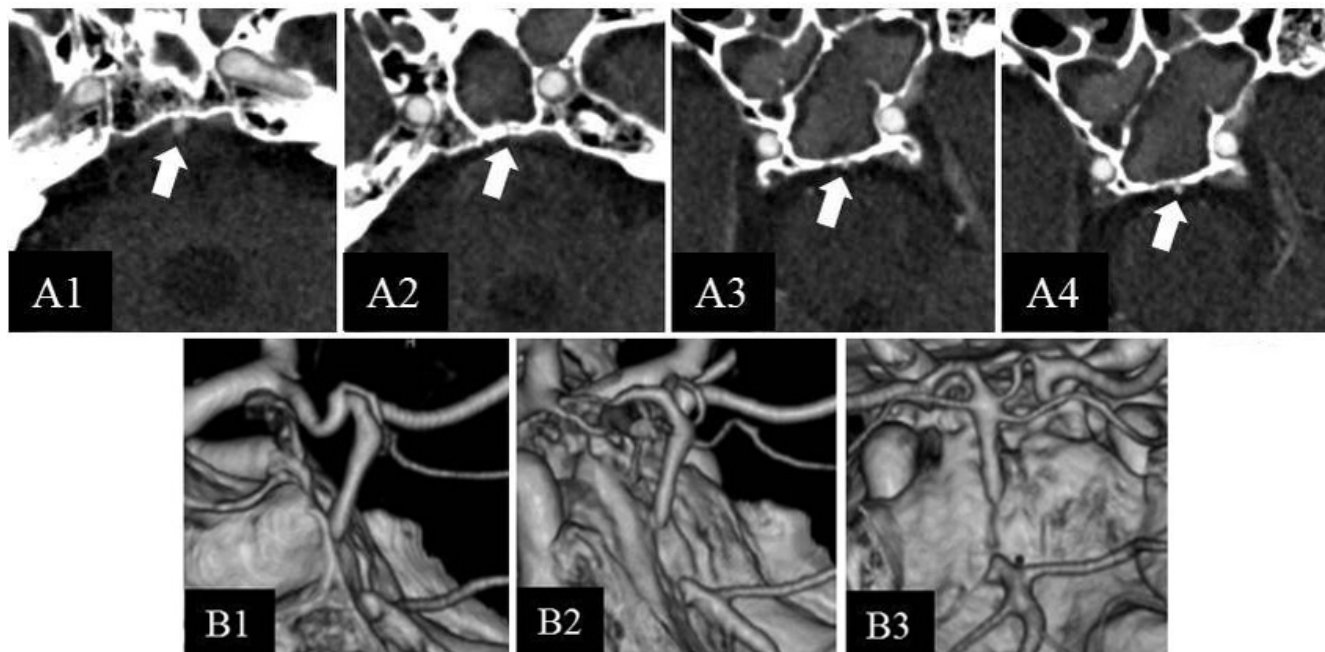


Fig. 3 (A) Axial images of computed tomographic angiography showing the basilar artery (*arrow*) disappearing superiorly and inferiorly into the line of longitudinal clivus fracture. (B) Three-dimensional reconstruction images of computed tomographic angiography: (1) lateral view, (2) oblique view, (3) anteroposterior view, showing the basilar artery entrapment within the longitudinal clivus fracture.

or perforators between bone fragments, resulting in arterial occlusion, stenosis, or dissection.⁶⁾ In addition to these direct mechanisms, subsequent thrombosis is another important factor. Injured intima and turbulent blood flow in stenosed or dissected artery lead to a formation of thrombus, which can narrow the arterial lumen even more and became a possible nidus for distal embolism. This subsequent event could account for some of the delayed deteriorations reported previously.^{1,3,5,11,15)}

Diagnosis can be made by CT angiography,¹⁾ which illustrates the exact image of basilar artery entrapment between clivus bone fragments. Without bone information and correct interpretation, the diagnosis is difficult because the clinical characteristics could mimic simple thromboembolic occlusion. This misdiagnosis can lead to high-risk endovascular intervention, as of the present case.

Effective treatment has not been established yet. Anti-thrombotic treatment is supposed to be effective for preventing aforementioned thrombotic progression. Four patients have received anti-coagulant or anti-platelet treatment.^{1,10,11,16)} Although none of these patients demonstrated deterioration after medication,

its effect is unclear in this small number. In addition, anti-thrombotic treatment in trauma patients is sometimes risky. Although there is no such report so far, endovascular treatment could be usable for some specific conditions.^{18–21)} Endovascular aspiration could be effective for patients presenting deterioration due to subsequent thrombosis,^{1,5)} and stent placement might be under consideration for patients with basilar artery stenosis or dissection.

The present case suggests that basilar artery entrapment within the longitudinal clivus fracture could be a cause of neurological deficits after trauma. In this subset, endovascular intervention without a correct diagnosis is highly risky.

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Conflicts of Interest Disclosure

None of the authors have any conflicts of interest to disclose.

Table 1 Clinical data of the basilar artery entrapment within the longitudinal clivus fracture

Authors	Age/sex	Initial neurological examination			Significant change of neurological condition			Treatment over supportive care	BA condition	Infarction	Outcome
		Consciousness	Paralysis	Elapsed time	Consciousness	Paralysis	Paralysis				
Loop et al. (1964) ⁽⁴⁾	59/male	Unconscious	Quadripareisis		None		None	Occlusion	Midbrain, pons	Dead	
Lindenberg et al. (1966) ⁽⁵⁾	42/male	Lucid	None	A few hours	Unconscious	Hemiplegia	None	Thrombosed (vertebral artery)	Medulla	Dead	
Sights et al. (1968) ⁽⁶⁾	23/male	Unconscious	Hemiplegia		None		None	Occlusion	Pons	Dead	
Anthony et al. (1987) ⁽⁷⁾	78/male	Unconscious	Quadripareisis		None		None	Occlusion	Brain stem, posterior circulation	Dead	
Guha et al. (1989) ⁽⁸⁾	27/male	Unconscious	Quadripareisis		None		None	Occlusion	Midbrain, pons, medulla, cerebellum	Vegetable state	
Sato et al. (1990) ⁽⁹⁾	80/male	GCS score 5	Quadripareisis		None		None	Occlusion	None	Dead	
Taguchi et al. (2000) ⁽¹⁰⁾	52/male	Unconscious	Quadripareisis		None		Argatroban, ticlopidine	Occlusion	Pons	Quadripareisis	
Sato et al. (2001) ⁽²⁾	56/male	GCS score 5	Quadripareisis		None		None	Occlusion	Brain stem, posterior circulation	Dead	
Bala et al. (2004) ⁽¹¹⁾	46/male	GCS score 15	None	12 hrs	GCS score 15	Hemiparesis	Aspirin	Occlusion	Medulla, pons	Mild hemiparesis	
Kaakaji et al. (2004) ⁽¹²⁾	50/male	GCS score 6	Quadripareisis		None		None	Mild stenosis	Midbrain, pons, cerebellum	Locked in state	
Cho et al. (2008) ⁽³⁾	54/male	GCS score 8	Hemiparesis	Day 1	GCS score 5	Quadripareisis	None	Occlusion (vertebral artery)	Brain stem, posterior circulation	Normal cognitive function, quadripareisis	
Khanna et al. (2010) ⁽¹³⁾	55/male	GCS score 8	ND		None		None	Severe stenosis	Pons, cerebellum	Coma, hemiplegia	
Fang et al. (2012) ⁽¹⁴⁾	39/male	Difficult to evaluate with drinking	None		None		None	Severe stenosis	None	No symptom	

(Continued)

Table 1 (Continued)

Authors	Age/sex	Initial neurological examination			Significant change of neurological condition			Treatment over supportive care	BA condition	Infarction	Outcome
		Consciousness	Paralysis	Elapsed time	Consciousness	Paralysis	Paralysis				
Sen-Gupta et al. (2012) ¹⁵⁾	67/male	GCS score 11	ND	Just after admission	GCS score 3	ND	None	Occlusion	Brain stem	Locked in state	
García-García J et al. (2012) ¹⁶⁾	37/male	GCS score 15	Hemiparesis		None		Heparin	Occlusion	Pons	Mild hemiparesis	
Wang et al. (2017) ¹⁾	59/male	GCS score 15	None	Day 2	GCS score 5	ND	Aspirin	Stenosis (admission), occlusion (24 hrs)	Midbrain, pons, cerebellum, occipital lobe	Locked in state	
Kliesch et al. (2017) ¹⁷⁾	ND	GCS score 11	ND		None		None	Severe stenosis	None	Dead by brain contusion	
Present case	69/male	GCS score 13	Slight hemiparesis	13 hrs	GCS score 5	Hemiplegia	Endovascular intervention	Occlusion	Pons	GCS score 10, hemiplegia	

BA: basilar artery, GCS: glasgow coma scale, ND: not described.

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