



Basilar Artery Occlusion with “Seizures” as a Presenting Symptom: Three Cases Treated Using Mechanical Thrombectomy

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Key words

- Acute ischemic stroke
- Basilar artery occlusion
- Mechanical thrombectomy
- Seizures

Abbreviations and Acronyms

BA: Basilar artery
BAO: Basilar artery occlusion
CT: Computed tomography
GCS: Glasgow Coma Scale
MRI: Magnetic resonance imaging
MT: Mechanical thrombectomy
NIHSS: National Institute of Health Stroke Scale
PCA: Posterior cerebral artery
rt-PA: Recombinant tissue plasminogen activator
TICI: Thrombolysis in cerebral infarction
TPA: Thalamoperforating artery
VA: Vertebral artery

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INTRODUCTION

Basilar artery occlusion (BAO) is a rare, devastating disease that is found in approximately 1.1% of patients with cerebral infarctions.¹ A poor prognosis is reportedly seen in >80% of patients.^{2,3} However, it is a treatable form of acute ischemic stroke. Early diagnosis allows for subsequent reperfusion therapies such as intravenous thrombolysis and mechanical thrombectomy (MT), which may improve outcomes. Diagnosing BAO can be extremely challenging, particularly in patients presenting with convulsive seizures or convulsive-like involuntary movements,⁴⁻⁶ since it can be confused with epilepsy. Here, we report 3 cases of patients with BAO who presented initially

■ **BACKGROUND:** Basilar artery occlusion (BAO) is a rare, potentially fatal cause of ischemic stroke. It is often challenging to diagnose, especially when the presenting symptom is “seizures”. We present 3 cases of patients with BAO presenting with seizures.

■ **CASE DESCRIPTION:** The first patient was a 53-year-old man with clonic convulsions. On angiography, BAO was detected and mechanical thrombectomy (MT) was performed. The modified Rankin Scale score at 3 months after treatment was 1. The second patient was a 64-year-old man with generalized convulsions. He was diagnosed with BAO and vertebral artery dissection and was treated with MT, percutaneous transluminal angioplasty, and stenting. The modified Rankin Scale score at 3 months after treatment was 3. The third patient was a 77-year-old man with tonic convulsions. He was diagnosed with BAO and treated with MT. However, he did not survive.

■ **CONCLUSIONS:** BAO is devastating; however, it is a treatable disease. Our report suggests that BAO should be suspected in patients presenting with initial convulsive seizures.

with “seizures.” All patients were treated using MT.

CASE PRESENTATION

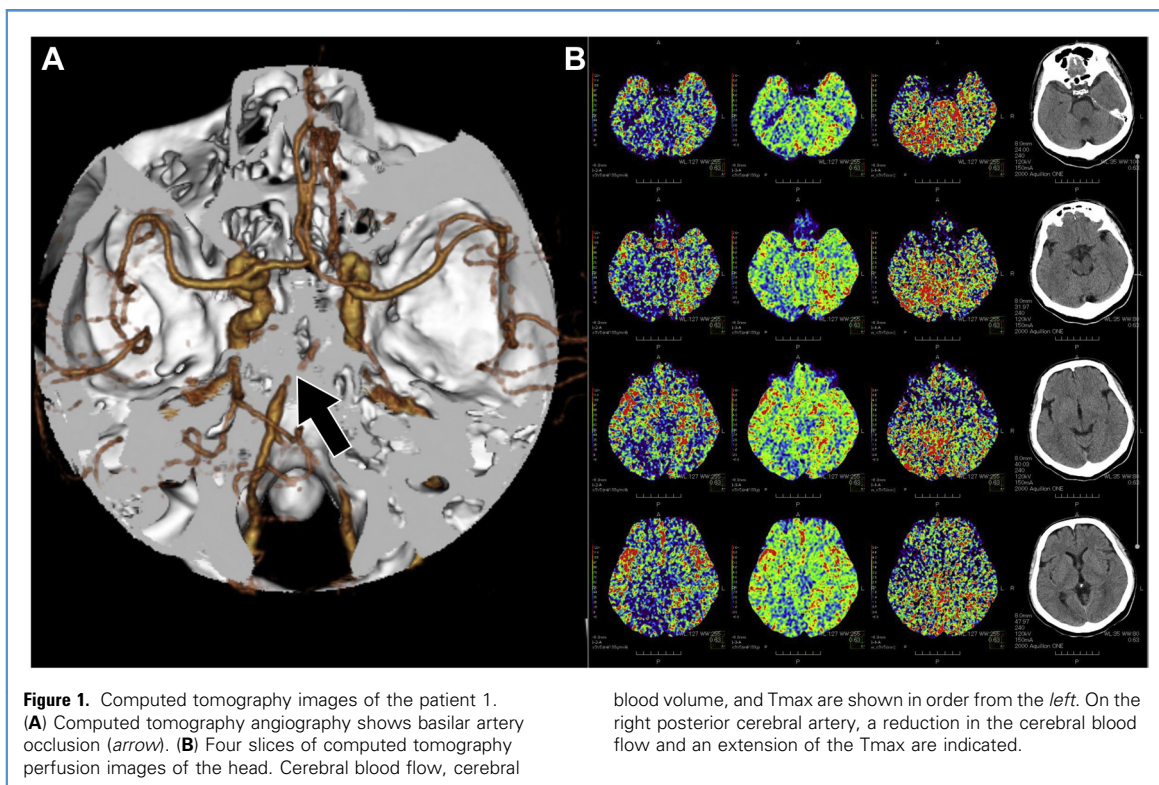
Patient 1

A 53-year-old man was admitted to the emergency department of our hospital because of convulsions that lasted for about 1 minute, followed by a disturbed level of consciousness. The patient was last seen well the night before at bedtime, which was estimated to be 9 hours earlier. On admission, he had clonic convulsions on the left side of his body, impaired consciousness of Glasgow Coma Scale (GCS) of 6, and tetraparesis. A National Institute of Health Stroke Scale (NIHSS) score of 34 was recorded. No medical history of epilepsy, stroke, migraine, atrial fibrillation, or diabetes mellitus was noted. In addition, there was no notable family history.

Computed tomography (CT) revealed no intracranial hemorrhage or early ischemic signs. CT angiography demonstrated a BAO

(Figure 1A), and CT perfusion images indicated cerebral ischemia of the right cerebellum and right occipital lobe (Figure 1B). Since it was estimated that more than 4.5 hours had passed since the stroke onset, the patient was not administered an intravenous injection of recombinant tissue plasminogen activator (rt-PA). MT was performed with the patient under sedation using diazepam and pentazocine for local anesthesia. An angiogram of the left vertebral artery (VA) revealed a BAO with an embolus in its mid-portion, which was occluded just distal to the bilateral anterior inferior cerebellar artery (PCA) was occluded at its origin. The left posterior cerebral artery (PCA) was occluded at its origin. The right thalamoperforating artery (TPA) could not be visualized due to the occlusion of the right PCA with a thrombus at P1. The embolus at the basilar artery (BA) was aspirated with a direct aspiration first-pass technique using a Penumbra 5MAX ACE (Penumbra Inc., Alameda, California, USA).

After the procedure, the left P1 was partially recanalized and the left TPA was



visualized on angiogram (**Figure 2B**). A 4×20 -mm Trevo (Stryker Neurovascular, Fremont, California, USA) was deployed to the left PCA (**Figure 2C**), and a large amount of thrombus was retrieved after 2.5 minutes of stent deployment in 2 passes. Thereafter, a small amount of thrombus was retrieved using a 4×20 mm Trevo in the right PCA. Complete perfusion (grade 3) was achieved according to the thrombolysis in cerebral infarction (TICI) scale in a total of 4 passes (**Figure 2D**). The total time from the onset of convulsions to recanalization was 253 minutes.

Immediately after treatment, the patient's consciousness was improved (GCS 13). On the following day, his NIHSS score improved to 4. He had ataxia in the right upper limb and mild vertical gaze palsy. Postoperative magnetic resonance imaging (MRI) revealed acute infarctions in the bilateral cerebellum (**Figure 3A**), right occipital lobe, left midbrain (**Figure 3B**), and bilateral thalamus (**Figure 3C**) and well-visualized arteries of the posterior circulation (**Figure 3D**). On ophthalmologic examination, a mild defect in the upper left visual field was confirmed. The patient's

ataxia and gaze palsy fully recovered. He was discharged with an NIHSS score of 0. At the 90-day follow-up, his modified Rankin Scale score was 1.

Patient 2

A 64-year-old man was admitted to our hospital about 45 minutes after developing generalized convulsions while working. On his way to the hospital, tonic convulsions in both upper limbs were reported, and his GCS was 6. On admission, his convulsions had ceased, and his GCS was 12. The patient had impaired consciousness, tetraparesis, and aphasia. An NIHSS score of 28 was recorded. There was no medical history of epilepsy or any other significant illness and no family history was noted.

CT angiography revealed an occlusion in the right VA and at the tip of the BA (**Figure 4A**). A CT perfusion image indicated ischemia of the right cerebellum and right occipital lobe (**Figure 4B**). Rt-PA was not administered due to suspicions of VA dissection. An angiogram of the right VA revealed a severe stenosis in the V4 portion. Thus, we performed a percutaneous transluminal

angioplasty. A 2×20 -mm Jackal RX (Kaneka Medix Corporation, Osaka, Japan) was used for transluminal dilatation for 1 minute to reach a pressure of 4 atm (**Figure 5A**). Further dilatation was performed using a 2.5×20 -mm Jackal RX for 1 minute to reach a pressure of 4 atm. Afterward, the BA was clearly visualized (**Figure 5B**). A direct aspiration first-pass technique was performed using a 4 MAX (Penumbra Inc.) for the occlusion at the tip of the BA. A complete recanalization (TICI 3) was achieved in 1 pass (**Figure 5C**). The total time from the onset of convulsions to recanalization was 260 minutes.

After treatment, the patient's level of consciousness improved (GCS 14). However, left paresis, left homonymous hemianopsia, left ataxia, dysarthria, and dysphagia persisted. His NIHSS score was 12. Postoperative MRI revealed infarctions in the bilateral cerebellar hemispheres, right side of the pons (**Figure 6A**), and right occipital lobe (**Figure 6B**). About 1 month after treatment, the stenosis in the right VA progressed, and the patient required retreatment with percutaneous transluminal angioplasty, which was

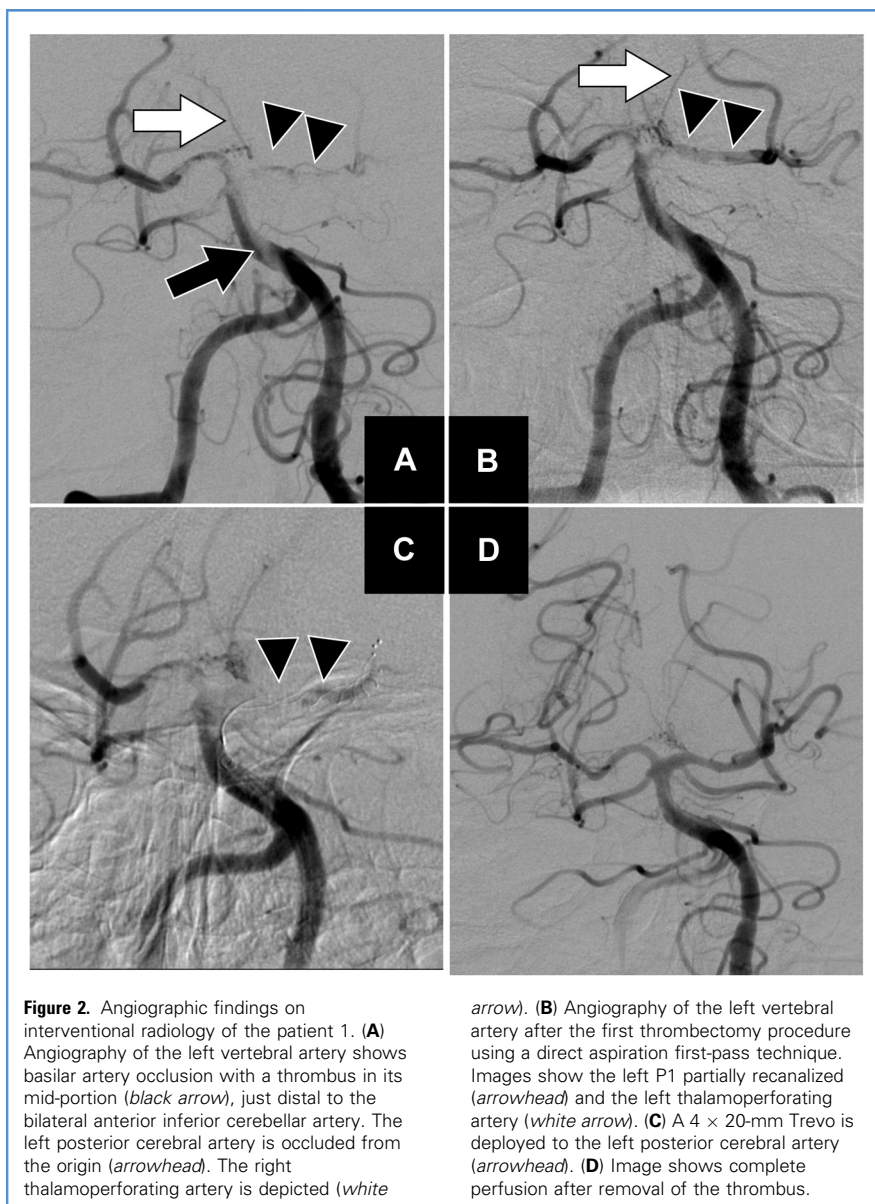


Figure 2. Angiographic findings on interventional radiology of the patient 1. (A) Angiography of the left vertebral artery shows basilar artery occlusion with a thrombus in its mid-portion (*black arrow*), just distal to the bilateral anterior inferior cerebellar artery. The left posterior cerebral artery is occluded from the origin (*arrowhead*). The right thalamoperforating artery is depicted (*white*

arrow). (B) Angiography of the left vertebral artery after the first thrombectomy procedure using a direct aspiration first-pass technique. Images show the left P1 partially recanalized (*arrowhead*) and the left thalamoperforating artery (*white arrow*). (C) A 4 × 20-mm Trevo is deployed to the left posterior cerebral artery (*arrowhead*). (D) Image shows complete perfusion after removal of the thrombus.

performed with the insertion of an indwelling Wingspan (Stryker Neurovascular; **Figure 5D**). After rehabilitation therapy, the patient was discharged. At the 90-day follow-up, his modified Rankin Scale score was 3.

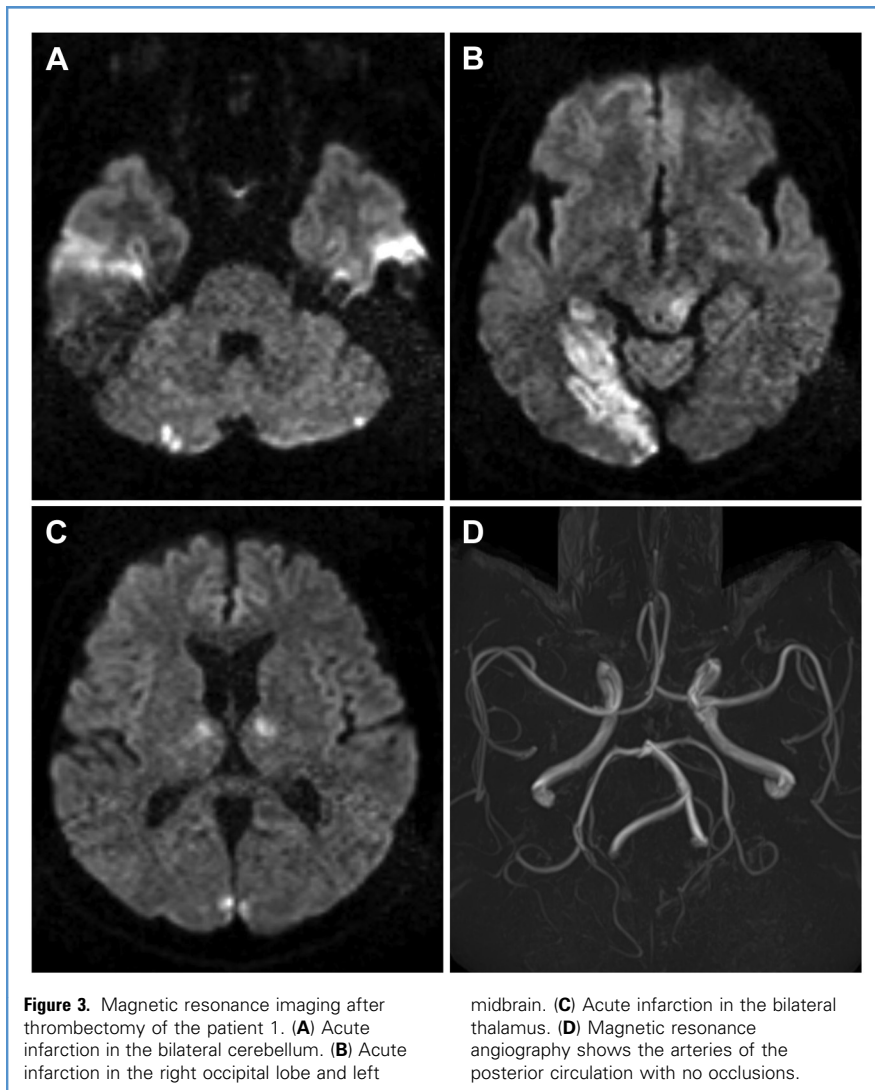
Patient 3

A 77-year-old man was admitted to a nearby hospital because of tonic convulsions with upward rotation of the eyes. On arrival, he had a disturbed level of consciousness with a GCS of 10. MRI indicated a BAO. The patient was transferred

to our hospital 4 hours after the onset of symptoms. The trachea was intubated by a previous doctor to protect the airway from aspiration due to frequent vomiting. After evaluating the patient, we determined that he had an impaired consciousness (GCS 9T), right homonymous hemianopsia, and tetraparesis. His NIHSS score was 15. The patient had a history of atrial fibrillation, for which he received rivaroxaban. He had no history of diabetes mellitus, and no family history was noted.

MRI performed at the previous hospital revealed an occlusion in the left PCA at the

tip of the BA with infarct lesions confined to the left occipital lobe and left lateral posterior choroidal artery (**Figure 7A**). MT was performed without the administration of an rt-PA. A right vertebral angiogram revealed a BAO with an embolus at the tip of the BA (**Figure 7B**). A 3 × 20-mm Trevo was deployed in the left PCA (**Figure 7C**). The embolus was retrieved in 1 pass and the right PCA and left P1 were clearly visualized afterward (**Figure 7D**). Effective reperfusion (TICI ≥2B) was achieved. However, remnants of the embolus were noted distal to the left P2. The total time



from the onset of convulsions to recanalization was 376 minutes. After treatment, no improvement in his level of consciousness was noted. In addition, subarachnoid hemorrhage occurred 4 hours postoperatively and the patient died on the following day.

DISCUSSION

We report 3 cases of patients with BAO who presented with seizures and were treated with MT. At our institution, of 40 patients with BAO who were treated with MT between January 2012 to December 2017, 3 presented with convulsions at the stroke onset. This demonstrates the rarity of such cases. Several cases were reported on the treatment of BAO patients with seizures

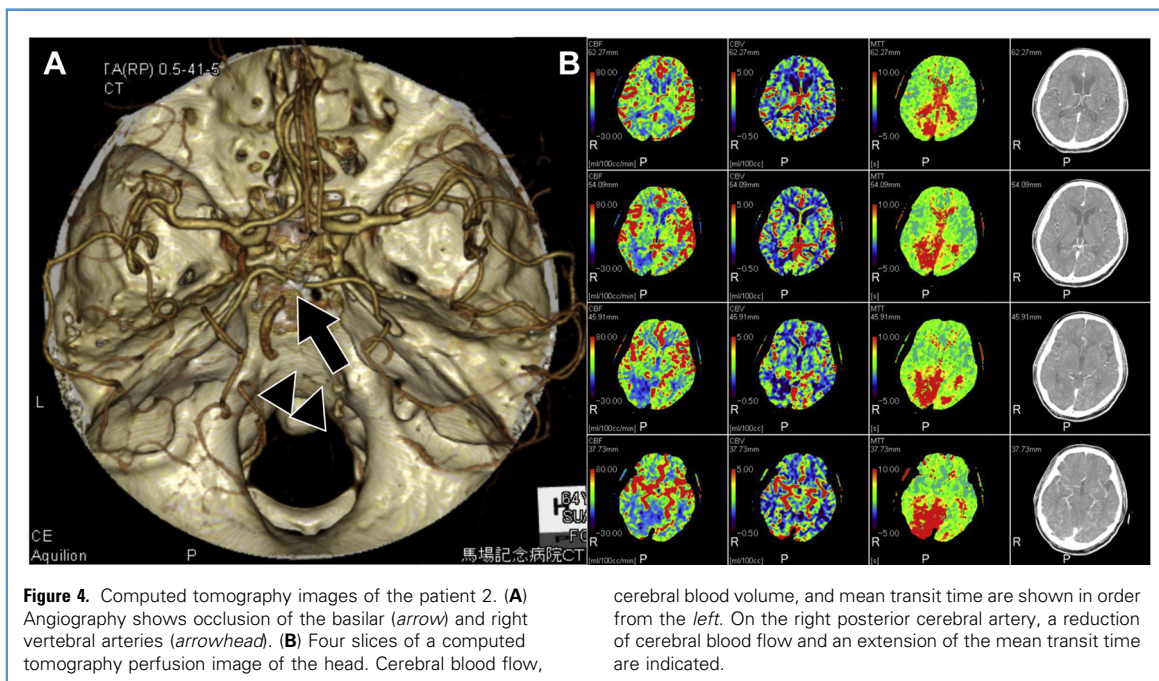
using intravenous heparin^{7,8} and rt-PA.⁹ However, few reports¹⁰ exist on their treatment with MT similar to the present cases.

Although it has been long known that convulsions and involuntary convulsive-like movements can present at the onset of BAO,^{5,6} they are relatively infrequent and are only seen in about 4.7% of patients with BAO.⁴

“Convulsions” after BAO reportedly originate from the brain stem, in particular the tegmentum and pons.⁵ This is supported by the occurrence of clonic movements in response to brain stem stimulation.¹¹ Although the pathophysiological mechanism is not clear, it is believed to be related to decerebrate posturing. In patients 2 and 3,

generalized convulsions without laterality were observed, which were believed to be caused by a similar mechanism. In case 2, infarcts at the pons were noted postoperatively, whereas in case 3, a severe brain stem injury was suspected that may have necessitated tracheal intubation. These suggest that “convulsions” may originate from the brain stem.

In contrast, early seizures are known for their occurrence at the early onset of ischemic strokes. They are believed to be caused by a cellular biochemical dysfunction leading to electrically excitable tissues.^{12,13} Early seizures reportedly occur in about 2% of ischemic strokes,^{14,15} particularly within 24 hours from the onset of the ischemic stroke. Large cohort studies reported that seizures sometimes originated



from the posterior circulation infarctions and lacunar infarctions¹⁴ and convulsions after BAO may include early seizures.

Patient 1 presented with clonic convulsions affecting the left side of his body. The laterality was clear, and it was easy to distinguish from decerebrate postures. CT perfusion images indicated ischemia of the right occipital lobe, suggesting a right occipital lobe seizure focus. Perforators from P1 to P2, which feed the brain stem and the thalamus, were well visualized on the angiogram of the right VA before we performed MT, particularly the right TPA, which is an important perforator from the right P1, whereas the left side was not clearly visualized. This indicated that ischemia may have developed on the left side of the brain stem. Furthermore, infarctions were noted on the left side of the tegmentum after MT. However, it was difficult to explain the convulsions on the left side. It is well-known that cortical involvement is the best-characterized risk factor for early seizure after ischemic stroke, which was supported by studies with widely differing designs.^{16,17}

On the multivariate analysis in the Seizures After Stroke Study (SASS), cortical location of infarction was one of the

significant risk factors for seizures after ischemic stroke (hazard ratio, 2.09; 95% confidence interval 1.19–3.68; $P < 0.01$).¹⁶ Although there was no report that seizures in BAO may occur due to cortical involvement of the occipital lobe, considering cortical involvement in the occurrence of early seizures, it may be reasonable to assume that the occipital lobe may be involved in the induction of convulsion in patients with BAO.

BAO is a rare disease that accounts for 1.1% of cerebral infarctions.¹ However, it can be fatal with a poor prognosis in more than 80% of patients.^{2,3} Because of its rarity, there are no reports from large randomized controlled trials; however, several studies reported the efficacy of MT for the treatment of patients with BAO.^{18,19} Furthermore, it was reported that early recanalization of the BA with MT can improve the prognosis of patients.²⁰ This was evident in patients 1 and 2, in whom early recanalization was performed. Both were able to function independently and were discharged. In contrast, the prognosis of patient 3 was poor, which may have been related to delayed recanalization. Thus, a delayed diagnosis and treatment of BAO may directly impact the prognosis of patients.

The diagnosis of BAO can be challenging because of the heterogeneity of its symptoms, even to neurologists, particularly in cases presenting with "seizures," which can be confused with epileptic seizures and status epilepticus. Although paralysis and a disturbed level of consciousness are important clinical features of BA, it is possible to misinterpret paralysis as a post ictal state or misdiagnose an impaired level of consciousness as status epilepticus in cases of epilepsy.⁷⁻¹⁰ With effective treatments available for BAO, there should be a high level of suspicion of cerebrovascular diseases including BAO in patients presenting with convulsive seizures for an early diagnosis and treatment. This report highlights the importance of identifying patients with BAO who present with seizures.

CONCLUSIONS

We reported 3 cases of patients with BAO presenting with "seizures." Although BAO is a rare, devastating disease that is difficult to diagnose, it is treatable. We recommend raising the suspicion index of cerebrovascular diseases including BAO in patients presenting with convulsive seizures.

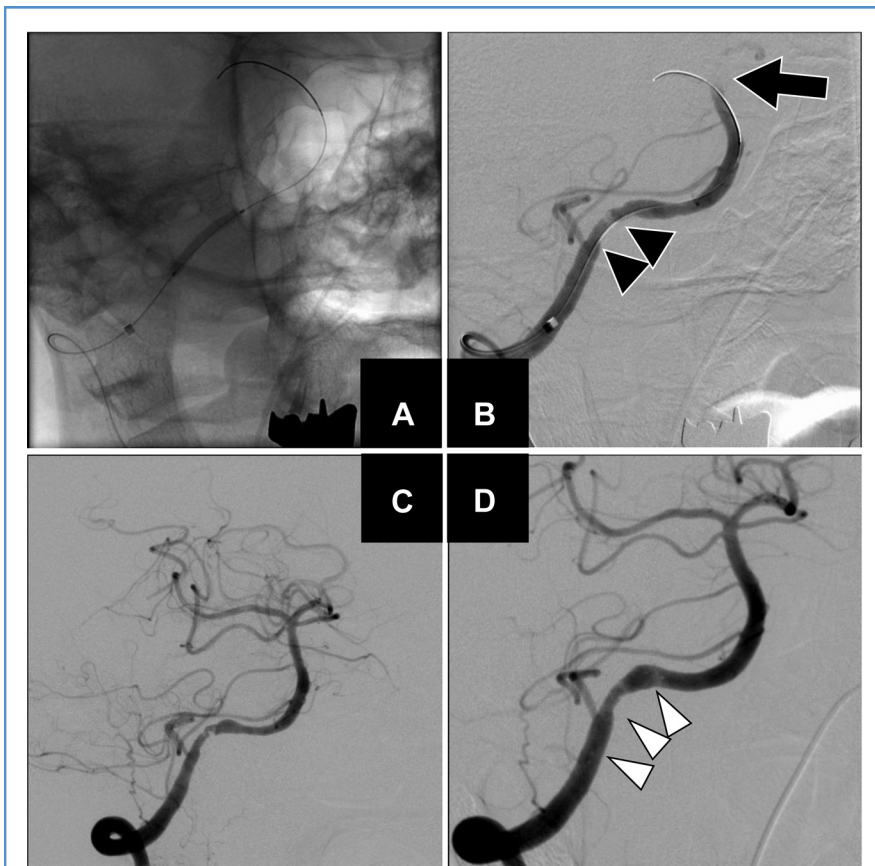


Figure 5. Angiographic findings on interventional radiology of the patient 2. **(A)** A percutaneous transluminal angioplasty is performed with a balloon. **(B)** After the procedure, the basilar artery with thrombus (*arrow*) is depicted clearly. The stenosis of the right vertebral artery persists (*arrowhead*).

(C) After a direct aspiration first-pass technique, complete recanalization is depicted using an angiogram. **(D)** Image at 1 month after treatment showing an indwelling stent performed for treatment of the stenosis of the right vertebral artery (*white arrowhead*).

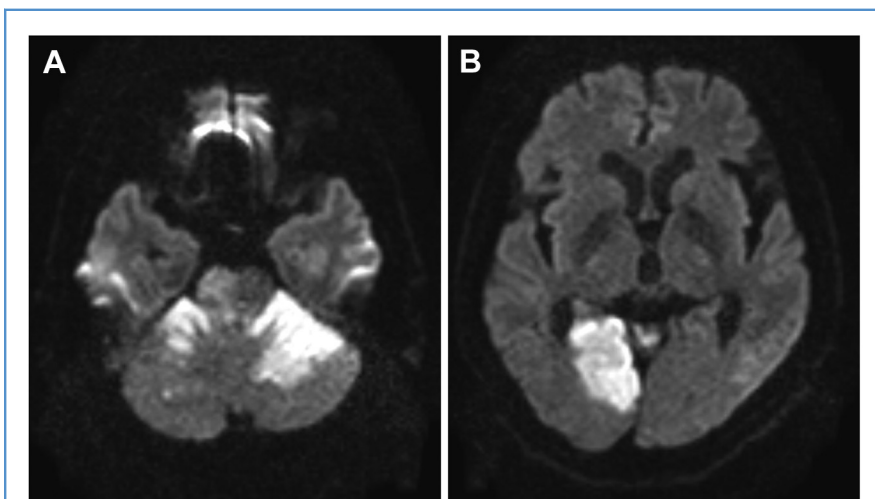
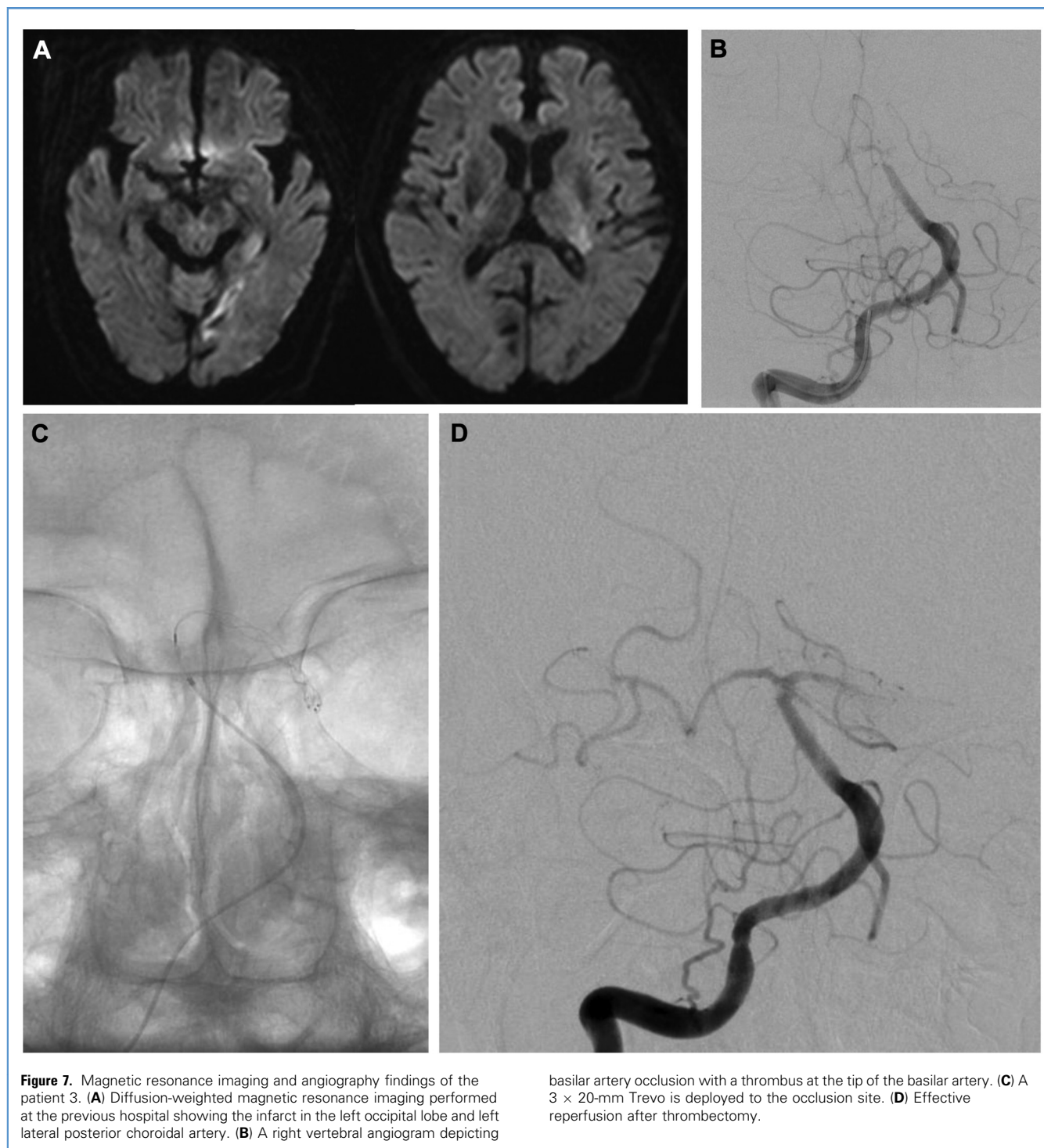


Figure 6. Diffusion-weighted magnetic resonance imaging after thrombectomy of the patient 2. **(A)** Acute infarction in the bilateral

cerebellum. **(B)** Acute infarction in the right pons and right occipital lobe.



REFERENCES

1. Israeli-korn SD, Schwammenthal Y, Yonash-Kimchi T, Bakon M, Tsabari R, Orion D, et al. Ischemic stroke due to acute basilar artery occlusion: proportion and outcomes. *Isr Med Assoc J.* 2010;12:671-675.
2. Schonewille WJ, Algra A, Serena J, Molina CA, Kappelle LJ. Outcome in patients with basilar artery occlusion treated convention- ally. *J Neurol Neurosurg Psychiatry.* 2005;76:1238-1241.
3. Hacke W, Zeumer H, Ferbert A, Brückmann H, del Zoppo GJ. Intra-arterial thrombolytic therapy improves outcome in patients with acute verte-brobasilar occlusive disease. *Stroke.* 1988;19:1216-1222.
4. Ferbert A, Brückmann H, Drummen R. Clinical features of proven basilar artery occlusion. *Stroke.* 1990;21:1135-1142.

5. Ropper AH. 'Convulsions' in basilar artery occlusion. *Neurology*. 1988;38:1500-1501.
6. Halsey JH, Downie AW. Decerebrate rigidity with preservation of consciousness. *J Neurol Neurosurg Psychiatry*. 1966;29:350-355.
7. Matsuo K, Fujii C, Fuse I, Nakajima M, Takada M, Miyata K. Top of the basilar syndrome in a young adult initially presenting with a convulsive seizure. *Intern Med*. 2011;50:1425-1428.
8. Saposnik G, Caplan LR. Convulsive-like movements in brainstem stroke. *Arch Neurol*. 2001;58:654-657.
9. Wilson LK, Benavente OR, Woolfenden AR, Asdaghi N. Spontaneous limb movements and posturing secondary to acute basilar artery occlusion: a potentially devastating seizure mimic. *Pract Neurol*. 2014;14:42-44.
10. Gadot A, Halleli H. Basilar artery occlusion presenting as a tonic-clonic seizure. *Isr Med Assoc J*. 2011;13:314-315.
11. Kreindler A, Zuckermann E, Steriade M, Chimion D. Electro-clinical features of convulsions induced by stimulation of brain stem. *J Neurophysiol*. 1958;21:430-436.
12. Luhmann HJ. Ischemia and lesion induced imbalances in cortical function. *Prog Neurobiol*. 1996;48:131-166.
13. Heiss WD, Huber M, Fink GR, Herholz K, Pietrzyk U, Wagner R, et al. Progressive derangement of periinfarct viable tissue in ischemic stroke. *J Cereb Blood Flow Metab*. 1992;12:193-203.
14. Procaccianti G, Zaniboni A, Rondelli F, Crisci M, Sacquegna T. Seizures in acute stroke: incidence, risk factors and prognosis. *Neuroepidemiology*. 2012;39:45-50.
15. Burn J, Dennis M, Bamford J, Sandercock P, Wade D, Warlow C. Epileptic seizures after a first stroke: the Oxfordshire Community Stroke Project. *BMJ*. 1997;315:1582-1587.
16. Bladin CF, Alexandrov AV, Bellavance A, Bornstein N, Chambers B, Coté R, et al. Seizures after stroke: a prospective multicenter study. *Arch Neurol*. 2000;57:1617-1622.
17. Camilo O, Goldstein LB. Seizures and epilepsy after ischemic stroke. *Stroke*. 2004;35:1769-1775.
18. Gory B, Mazighi M, Blanc R, Labreuche J, Piotin M, Turjman F, et al. Endovascular Treatment in Ischemic Stroke (ETIS) Research Investigators. Mechanical thrombectomy in basilar artery occlusion: influence of reperfusion on clinical outcome and impact of the first-line strategy (ADAPT vs stent retriever). *J Neurosurg*. 2018;12:1-10.
19. Bouslama M, Haussen DC, Aghaebrahim A, Grossberg JA, Walker G, Rangaraju S. Predictors of good outcome after endovascular therapy for vertebrobasilar occlusion stroke. *Stroke*. 2017;48:3252-3257.
20. Uno J, Kameda K, Otsuji R, Ren N, Nagaoka S, Maeda K, et al. Mechanical thrombectomy for acute basilar artery occlusion in early therapeutic time window. *Cerebrovasc Dis*. 2017;44:217-224.

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