—Case Reports—

Formation of a Large Fusiform Aneurysm near a Medullary Infarction Caused by Dissection of the Posterior Inferior Cerebellar Artery

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Infarction of the posterior inferior cerebellar artery (PICA) can lead to ischemic stroke in the lateral medullary oblongata. PICA dissection can also elicit an ischemic event in this region, but its detection on radiological images is difficult because of the small diameter of the vessel. We report a case of Wallenberg syndrome due to PICA dissection in a 48-year-old man, which was difficult to diagnose on first admission. He reported sudden onset of sensory disturbance on the right side of his face, ataxic gait, and headache. Brain magnetic resonance imaging (MRI) revealed a fresh cerebral infarct in the right lateral medulla oblongata. Magnetic resonance angiography (MRA) performed at the time of his admission showed no cerebral vessel abnormalities. An MRI study 18 months after the event revealed a fusiform aneurysm on the lateral medullary segment of the PICA, which was extremely close to the cerebral infarct. We concluded that the infarct was due to PICA dissection because of the sudden onset of symptoms and because the infarcted territory of the occluded penetrating branch of the dissecting aneurysm was consistent with Wallenberg syndrome. The aneurysm was trapped and an occipital artery-PICA bypass was placed. At the latest follow-up, 1 year after the procedure, he had no neurological symptoms. Imaging findings at the time of his first admission indicated that the PICA was intact. However, 18 months later, MRI revealed enlargement of an aneurysm at the site of the dissection. A cerebral infarct with headache may indicate PICA dissection. (J Nippon Med Sch 2024; 91: 129–133)

Key words: posterior inferior cerebellar artery, arterial dissection, cerebral infarction, large aneurysm, occipital artery-posterior inferior cerebellar artery bypass

Introduction

Ischemic stroke at the lateral medulla oblongata results in Wallenberg syndrome. In most cases the infarct site is on the posterior inferior cerebellar artery (PICA) or vertebral artery (VA)[1]. Kim[2] reported that among 130 patients, most vascular events leading to infarcts were atherothrombotic and that among other vessels, PICA occlusion eliciting ischemic stroke was related to cardiogenic embolism. According to Kobayashi et al.[3], PICA dissection can also result in ischemic events in this region. However, it may be difficult to identify ischemic PICA dissection on radiological images because the diameter of the vessel is small[4,5].

We describe a man with ischemic stroke at the lateral medulla oblongata that was initially thought to be atherothrombotic because PICA dissection was obscured on imaging scans obtained at symptom onset. However, PICA dissection resulted in chronic progression, namely, formation of a large fusiform aneurysm during the 18-month clinical course.

Case Report

A 48-year-old man with hypertension and diabetes mellitus reported sudden onset of sensory disturbance on the right side of his face, ataxic gait, and right occipital headache. He provided prior written informed consent for in-

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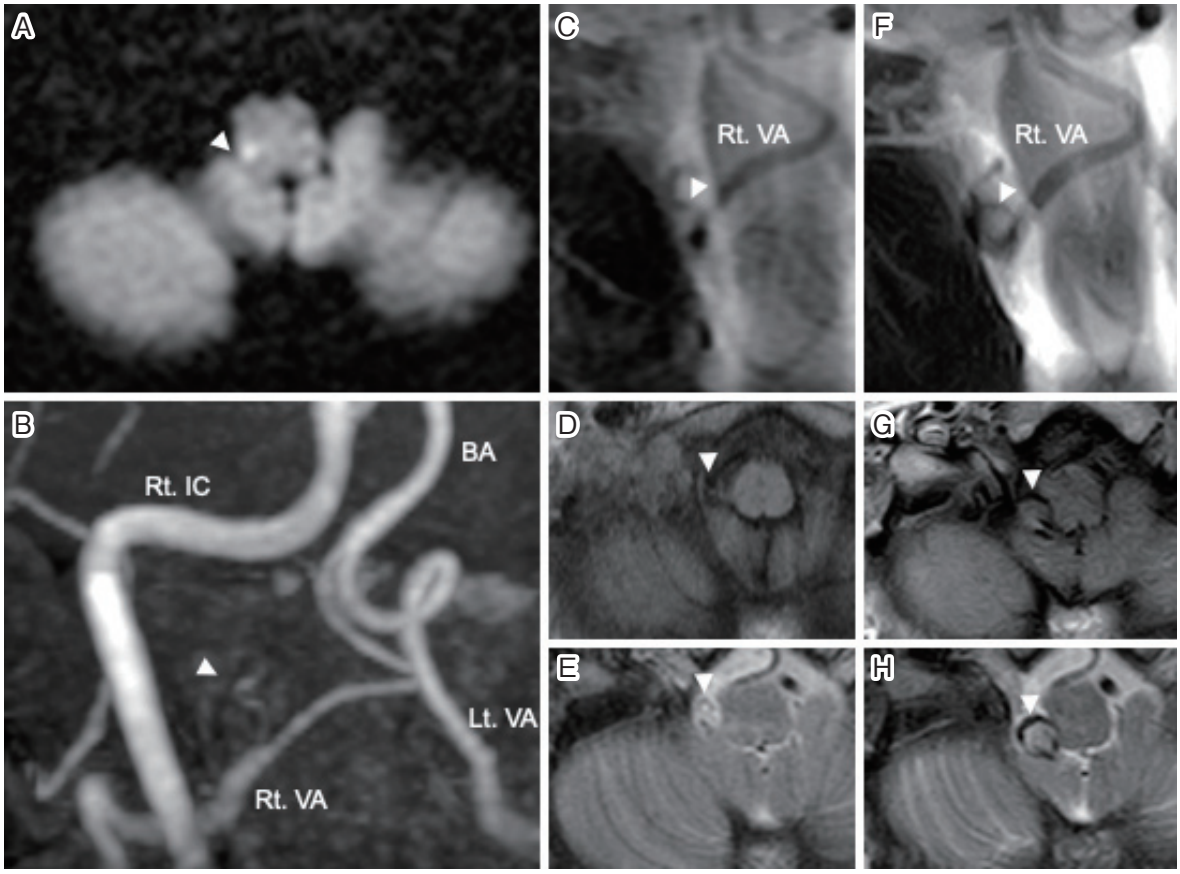

Fig. 1

A-E. Images at the time of admission and 6 days later (T1-weighted images)

A. Diffusion-weighted image obtained at the time of admission. Note the spotty high-intensity area in the right lateral medulla oblongata. (arrowhead)

B. 3D-TOF MRA. No abnormal findings. The arrowheads indicate the proximal dissection site.

C. Basi-parallel anatomical scan. No abnormal findings. The arrowhead indicates the dilation site.

D. T1-weighted image. No abnormal findings. The arrowhead indicates the dilation site.

E. T2-weighted image. No abnormal findings. The arrowhead indicates the dilation site.

F-H. Images acquired 18 months after admission.

F. Basi-parallel anatomical scan. The arrowhead indicates the dilated dissecting aneurysm.

G. T1-weighted image. The arrowhead indicates the dilated dissecting aneurysm.

H. T2-weighted image. The arrowhead indicates the dilated dissecting aneurysm.

clusion in this report.

He was admitted to our hospital with symptoms of Wallenberg syndrome. Diffusion-weighted brain MRI (**Fig. 1A**) revealed a fresh cerebral infarct at the right lateral medulla oblongata; 3D-time-of-flight (TOF) MRA (**Fig. 1B**), basi-parallel anatomical scanning (**Fig. 1C**), and T2-weighted imaging (**Fig. 1D**) showed no dissection of the VA or PICA. Our provisional diagnosis had been atherothrombotic cerebral infarction, and for 2 weeks he was placed under in-hospital observation with antiplatelet therapy. At discharge he continued to manifest sensory disturbance on the right side of his face (modified Rankin Scale, mRS = 1) and he was followed as an outpatient. MRI scans at 2 and 4 months after the attack revealed no new cerebral infarcts. However, a routine follow-up brain MRI scan performed 18 months after the ictus showed a dissecting aneurysm on the lateral medullary segment of the PICA, which was extremely close to the cerebral infarct (**Fig. 1A, 2**). Re-examination of magnetic resonance angiography (MRA) and MRI findings obtained 2 months after his hospital admission revealed a small dissecting aneurysm. The medulla oblongata harboring the first cerebral infarct was supplied by the PICA. On the basis of his post-hospitalization clinical course we ultimately diagnosed medullary infarction due to PICA dissection and recorded the development of a gradually growing fusiform aneurysm on the lateral medullary segment of the PICA. His earlier sudden-onset

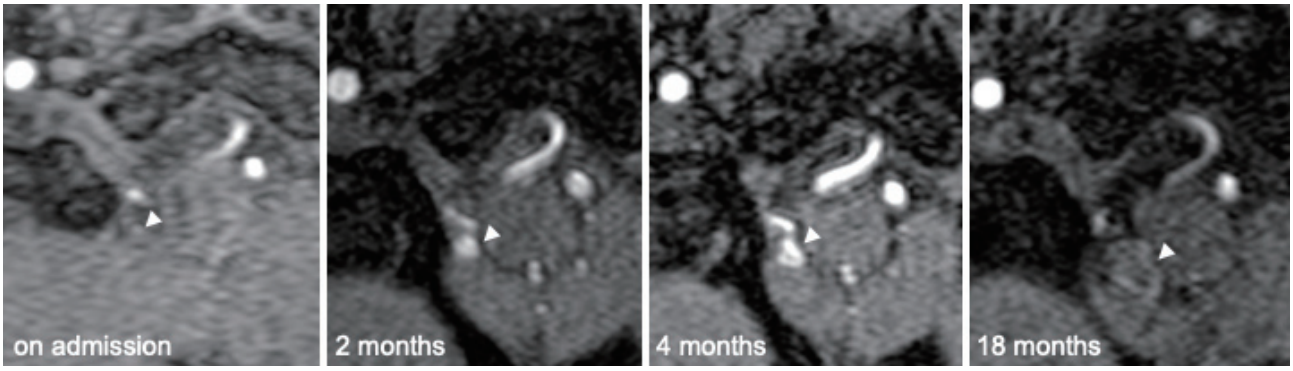

Fig. 2 3D-TOF MRA (axial view) performed 2, 4, and 18 months after first admission. The images confirm gradual enlargement of the aneurysm (arrowheads).

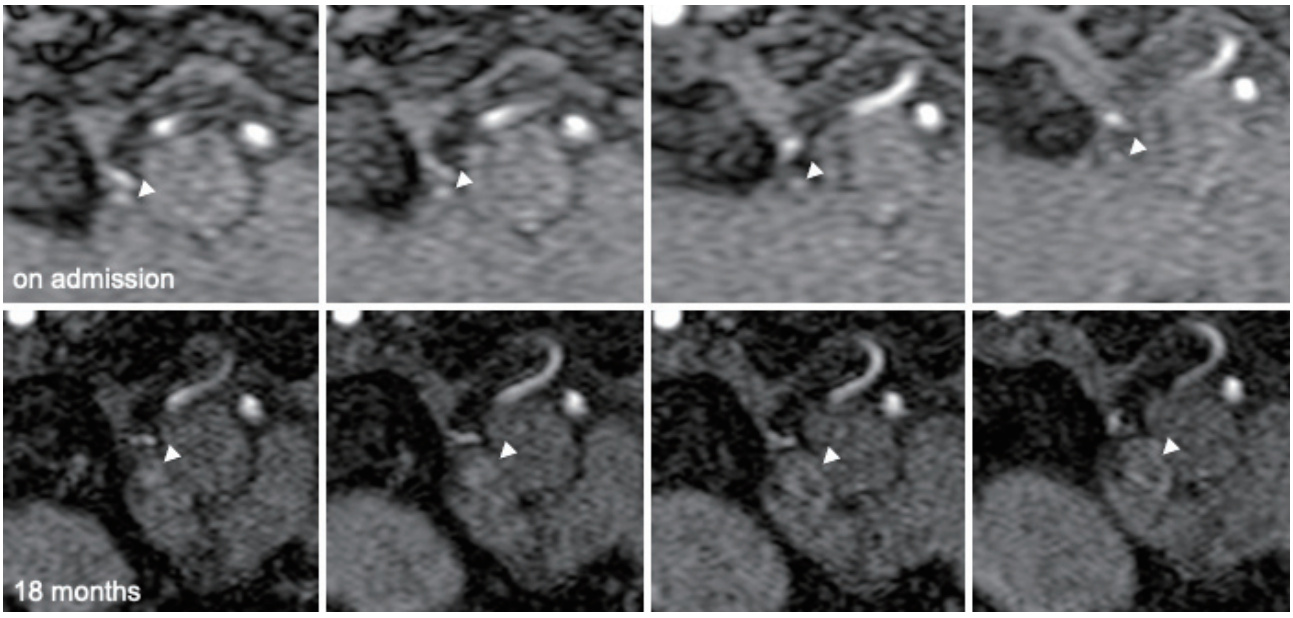

Fig. 3 Comparison of MRA-TOF axial images obtained at the time of first admission and 18 months after attack. Arrowheads indicate the dilated dissecting aneurysm.

headache was consistent with this diagnosis.

Although he did not develop new symptoms or recurrent cerebral infarction during the follow-up period, the aneurysm grew gradually. Thus, we planned surgical treatment of the dissecting aneurysm to prevent rupture. 3D-TOF MRA scans obtained 2, 4, and 18 months after his first admission showed a 10 × 17-mm fusiform aneurysm at the lateral medullary segment with extension to the caudal loop of the PICA (**Fig. 3**). The pearl-and-string sign was present (**Fig. 4**). The aneurysm was trapped and an occipital artery (OA)-PICA bypass was placed (**Fig. 4B, C**). Although a postoperative MRI scan showed no new cerebral infarct, truncal ataxia and sensory disturbance in the left part of his body were observed. We repaired liquorrhea and he was transferred to a rehabilitation hospital 44 days later. Three months after the second operation we placed a V-P shunt for hydrocephalus. His neurological deficits gradually improved and at the latest follow-up examination, 1 year after his OA-PICA bypass, he had no neurological symptoms.

Discussion

We report a patient with Wallenberg syndrome attributable to PICA dissection that was obscured on imaging studies performed at onset. During the follow-up period he manifested chronic progression, with formation of a large fusiform aneurysm close to the dissection site. Mizutani[6] reported that over time approximately 4% of unruptured dissecting aneurysms, particularly those involving VA dissection, become enlarged. It is not known how often PICA dissecting aneurysms enlarge.

In 2014, Matsumoto et al.[7] reported that most patients

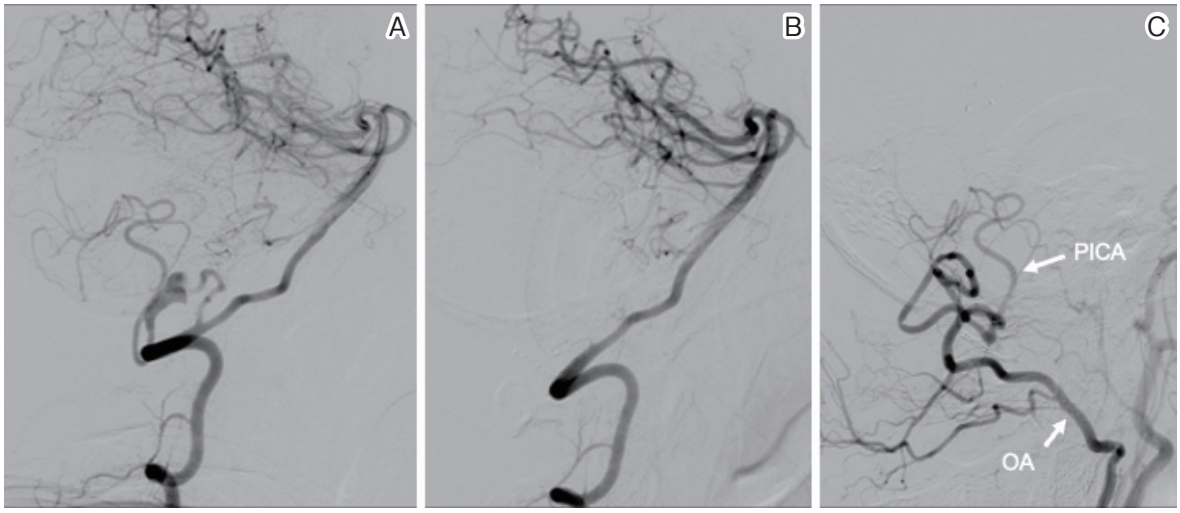

Fig. 4 A preoperative VA angiogram (lateral view) revealed a 10 × 17-mm aneurysm.
A. Postoperative angiogram
B. VA (lateral view). The aneurysm is trapped.
C. External carotid artery (lateral view). The occipital artery (OA) is anastomosed to the PICA distal to the aneurysm.

with PICA dissection presented with subarachnoid hemorrhage. A later study by Kobayashi et al.[3] showed that 6% of infarcts in PICA territory were due to PICA dissection and that in a similar number of patients they were attributable to VA dissection. They also reported that patients with PICA dissection tended to be younger than patients without PICA dissection and that their initial NIH stroke score was lower. Ischemic PICA dissection is almost always accompanied by occipital headache[4]. Our 48-year-old patient developed sudden-onset right occipital headache; the proximal PICA dissection that resulted in lateral medullary infarction and development of fusiform aneurysm was not detected on the initial TOF MRA or on (BPAS) images.

Because the PICA is tiny, it is difficult to diagnose ischemic PICA dissection[4,5]. Hosoya et al.[8] reported that a high-intensity signal on axial T1-weighted images assists in diagnosing VA dissection. Susceptibility-weighted and high-resolution MRI imaging were useful for identification of VA and PICA dissection[4]. However, we did not perform these studies. Our experience indicates that such studies should have been performed at the time of his first admission.

Patients with VA dissection must undergo follow-up radiological studies because the intramural lumen becomes fragile within 3 weeks after symptom onset. Although most unruptured VA dissections remain stable within a few months, some patients require long-term observation[9]. Sasaki et al.[10] reported a patient with a growing PICA aneurysm secondary to a cerebellar infarct. The aneurysm grew in the 2 weeks after symptom onset, and PICA dissecting aneurysm was diagnosed intraoperatively. To our knowledge, ours is the first case with a secondary enlarged PICA dissecting aneurysm detected 18 months after a cerebral infarct. During the preoperative observation period our patient developed no new neurological deficits despite the presence of an enlarging aneurysm near the medulla oblongata. Surgery avoided aneurysmal rupture and cranial nerve involvement.

Endovascular treatment has been used to address aneurysms of the posterior circulation, including VA-PICA aneurysms. Aneurysms at the trunk of the PICA can also be treated endovascularly; however, the procedure can be difficult and complex[11]. The aneurysm in our patient was large and fusiform. Although endovascular treatment was an option, it may have required parent artery occlusion. Therefore, we performed revascularization via an OA-PICA bypass and trapped the aneurysm.

Our experience suggests that in patients with ischemic stroke in the PICA territory who report occipital headache, PICA dissection may not be evident on radiological imaging. Susceptibility-weighted and high-resolution MRI scans can assist in the diagnosis of PICA dissection.

Conflict of Interest: None declared.

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