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Case Report

A Case of Intracranial Vertebral Artery Dissection Undetected by CT, MRI, and MRA at the Onset of Headache That Caused Subarachnoid Hemorrhage Seven Days Later

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Objective: We report a patient with normal imaging findings at the onset of preceding headache who developed subarachnoid hemorrhage (SAH) due to intracranial vertebral artery dissection 7 days later.

Case Presentation: A 51-year-old woman with a history of chronic headache visited our emergency outpatient department with a complaint of mild to moderate right nuchal pain. CT, MRA, and MRI (diffusion-weighted image, T2-weighted image, FLAIR, MR cisternography, and basi-parallel anatomical scanning) were normal. Seven days later, she was admitted to our hospital with sudden disturbance of consciousness. CT revealed SAH and CTA demonstrated dilatation of the right vertebral artery (VA). The dilated lesion with an intimal flap on the right VA proximal to the posterior inferior cerebellar artery was confirmed on DSA. The dilated lesion and the proximal VA were occluded endovascularly using coils. The condition of the patient improved gradually, and she was transferred to the rehabilitation hospital on day 45 with a modified Rankin Scale score of 2.

Conclusion: The clinical course of the presented case, although rare, should be kept in mind in daily clinical practice.

Keywords vertebral artery dissection, endovascular therapy, vessel wall imaging, natural history, diagnosis

Introduction

Although subarachnoid hemorrhage (SAH) due to vertebral artery dissection (VAD) is often preceded by headache, there are few reports of detailed imaging examinations before rupture of VAD. We report a case of VAD that occurred and ruptured 7 days after the onset of headache without abnormal CT, MRI, or MRA findings. This case is highly informative from the viewpoint of the prevention of rupture and highlights the importance of evaluating the course until the rupture of VAD.

Case Presentation

The patient was a 51-year-old woman with chronic headache associated with menstrual pain and shoulder stiffness. She complained of pinpoint pain in the right upper nuchal region and visited the emergency department of our hospital at 20:00. At the initial examination, there were no significant neurological findings, meningeal irritation signs, or abnormal findings on head CT (**Fig. 1A**), MRA (**Fig. 1B**–**1D**), FLAIR (**Fig. 1E**), MR cisternography (**Fig. 1F**), basi-parallel anatomical scanning (BPAS; **Fig. 1G**), diffusion-weighted imaging, or T2-weighted imaging, and she was allowed to go home. According to the family, pain persisted, but there was no particular change in her condition.

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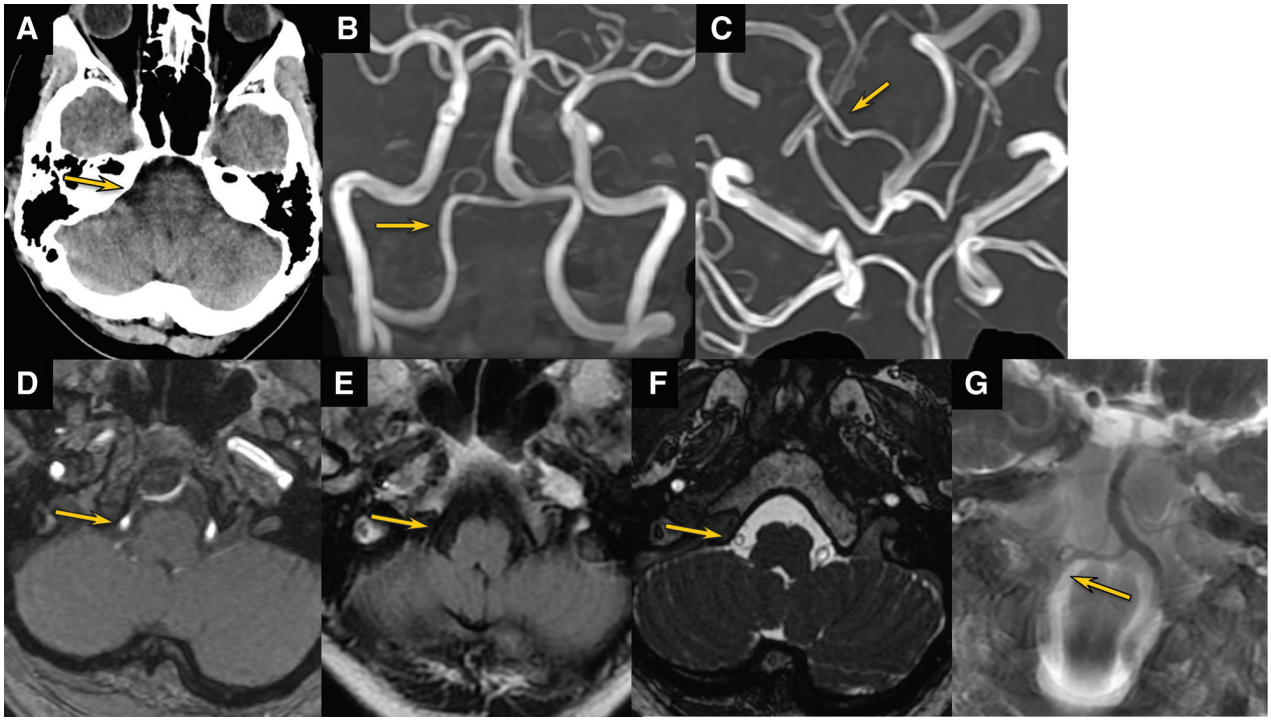

Fig. 1 (**A**–**G**) No abnormality was noted by CT (**A**), MRA (**B**–**D**), FLAIR imaging (**E**), MR cisternography (**F**), or BPAS (**G**). The arrows indicate the future site of VAD. BPAS: basi-parallel anatomical scanning; VAD: vertebral artery dissection

Seven days later, after she complained of sudden headache and nausea, she developed disturbance of consciousness while in the restroom and was emergently transported. On arrival, the Japan Coma Scale was III-300, Glasgow Coma Scale was E1V1M1 (3), CT demonstrated diffuse SAH (**Fig. 2A**), and 3D-CTA revealed previously unnoticed dilatation in the right vertebral artery (VA) proximal to the posterior inferior cerebellar artery (**Fig. 2B**, arrow). A diagnosis of rupture of VAD, Hunt and Hess grade 5, World Federation of Neurological Surgeons (WFNS) grade 5, and Fisher group 3 was made.

On the same day, emergency endovascular treatment was performed. Under general anesthesia and systemic heparinization, a 4-F guiding sheath (Fubuki Dilator Kit; Asahi Intecc, Aichi, Japan) was inserted into the right VA via the right femoral artery and an angiographic catheter was placed in the left VA. On preoperative DSA, the dilated area of the right VA had an intimal flap on the dorsal side and was 7.8 mm in transverse dimension and 8.2 mm long (**Fig. 2C**). Excelsior SL10 90° and 45° microcatheters (Stryker, Kalamazoo, MI, USA) were guided to the dilated area using a Traxcess 14 (Terumo, Tokyo, Japan), Target 360 soft coils 8 mm × 20 cm (Stryker) were placed through the SL10 90°, Target 360 soft coils 7 mm × 15 cm (Stryker) were added through the SL10 45°, and the dilated area was occluded with a total of 14 coils. Thereafter, the VA proximal to the lesion was occluded (parent artery occlusion) with a total of 10 coils (**Fig. 2D**) and complete occlusion was confirmed postoperatively by DSA (**Fig. 2E**).

After surgery, ventricular drainage was temporarily required, but the postoperative course was otherwise uneventful. Due to impaired attention and short-term memory, the patient was transferred to another hospital for recuperation and rehabilitation on the 45th hospital day. She was discharged 3.5 months after onset and is presently followed up on an outpatient basis (modified Rankin Scale 2).

Discussion

In this case, no abnormality was noted on imaging examinations at the onset of nuchal pain, and SAH occurred after 7 days when VAD was newly observed. This is considered to be a rare case in which detailed imaging evaluation was carried out before the rupture of VAD. Moreover, although detailed examination by CT, MRI, and MRA was performed, VAD was unable to be detected by the protocol employed before rupture at the onset of headache, and anticipation of the subsequent hemorrhage was difficult. This point is considered to be highly significant for daily practice and from a social viewpoint.

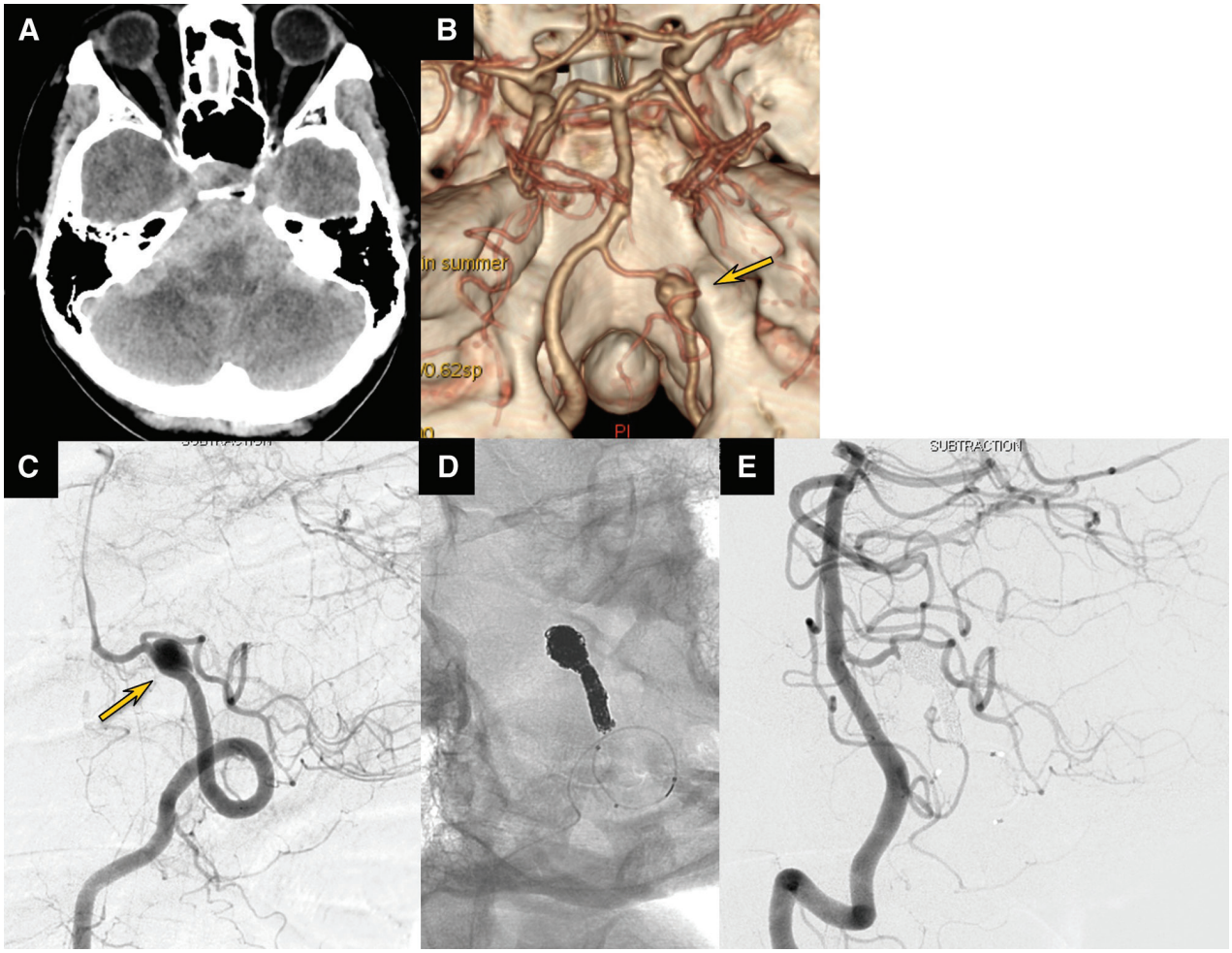

Fig. 2 Images at the time of rupture of VAD 7 days after the onset of headache. (**A** and **B**) CT showed SAH (**A**) and CTA showed formerly undetected dilatation (arrow) in the right VA (**B**). (**C**) Preoperative DSA revealed dilatation (arrow) accompanied by an intimal flap in the right VA. (**D**) The dilated area and the proximal VA were occluded using coils. (**E**) Complete occlusion of the lesion was confirmed by postoperative DSA. SAH: subarachnoid hemorrhage; VA: vertebral artery; VAD: vertebral artery dissection

In general, rupture of VAD is preceded by headache in 77.8% of patients.[1] On the other hand, the natural course of the so-called non-stroke-type VAD, which may present with only headache at onset, involves no hemorrhage or ischemia and is diagnosed as VAD by imaging examinations, being favorable. The rate of hemorrhage was 0.4% (1 of 230 cases) in a nationwide survey in Japan,[2] 1.0% (1 of 98 cases) in the report by Mizutani et al.,[1] and 0.9% (1 of 113 cases) in the report by Kobayashi et al.,[3] being low. However, of these 3 patients who exhibited rupture, 2 underwent antithrombotic therapy before rupture.[2,3] In the above nationwide survey,[2] the initial imaging findings in patients with VAD presenting with headache at onset were fusiform dilatation in 41%, pearl and string sign in 29%, string in 14%, occlusion in 7%, multiple findings in 5%, and others in 4%, but there was no mention of patients with no initial findings, as in the present case.

In VAD, intimal formation and normalization of the vascular lumen begin within 15 days at the shortest, and unruptured VAD is often in the healing process at the time of diagnosis,[1] but it may continue to enlarge in some patients. Kai et al.[4] reported that 96 of 100 patients with unruptured VAD, after exclusion of 4 who presented with the mass effect and underwent surgery in the acute phase, were followed up by MRI and MRA 2 weeks and 1, 3, 6, 12, and 24 months. They observed enlargement during the course in 5 and treated them, but as no rupture was observed, they considered that the risk in the natural course of unruptured VAD is not high but that it should be occluded if enlargement is noted.[4] According to the evaluation of 56 cases of unruptured VAD by Shibahara et al.,[5] 4 exhibited enlargement or new aneurysm formation and 3 required treatment, and they were all treated within 1 month. However, there was time for follow-up by

imaging examinations and surgical intervention unlike in the present case, in which hemorrhage developed in the acute phase, and whether they can be regarded as similar cases is questionable.

As special cases, there have been a few reports of the appearance and bleeding of new contralateral VAD (contralateral de novo VAD) during treatment for initially unilateral VAD. In the report of a case of contralateral de novo VAD that developed SAH on the 9th hospital day during conservative treatment for VAD due to unilateral infarction of the medulla oblongata,[6] the following similarities to the present case were observed: 1) the contralateral VA was normal on MRA on the 1st and 4th hospital days, 2) the site of de novo VAD enlarged and ruptured in a short period, 3) the patient suddenly developed disturbance of consciousness at the time of rupture with no preceding clinical symptoms, and 4) no antithrombotic therapy was administered before rupture. The authors considered the possibility that small dissection of the vascular wall undetected by imaging studies enlarged later and resulted in rupture, and referred to the possibility of diagnosis by MRI BPAS or vessel wall imaging. There was also a case in which contralateral de novo VAD developed after occlusion of the parent artery for unilateral VAD,[7] suggesting the involvement of hemodynamic changes.

In the present case, small dissection undetectable by imaging examinations may have developed in the wall of VA, causing nuchal pain, and it may have developed into a detectable aneurysmal lesion during the following week and ruptured. It is reasonable to consider headache as the initial symptom to be related to the occurrence of VAD based on its site, but there were no meningeal irritation signs, and no hemorrhagic or ischemic lesions or abnormality of the vascular lumen or contour were noted by imaging examinations. Although the possibility of change in the vascular wall at this point cannot be excluded, it was unable to be detected by imaging examinations. Silent VAD, which has only changes in the vascular wall without changes in the vascular lumen or contour, was observed by autopsy in 43% of patients who developed SAH due to VAD, and this small rupture of the internal elastic lamina covered by thickened intima was present at sites distant from the ruptured VAD such as the opposite side.[8] For the diagnosis of VAD, MRI and MRA, which provide information about both the vascular lumen (e.g., luminal irregularity and intimal flap) and vascular wall (e.g., intramural hematoma), are considered useful.[9] There have been reports concerning sequences, including 3D-T1 weighted,[10] 3D-black blood T1 weighted,[9] fat-suppressed T1 weighted,[11] T2 weighted,[4] fat-suppressed T2 weighted,[11] proton-density weighted,[4,10] susceptibility weighted,[9] and BPAS,[6] and these modalities may be considered when VAD is suspected. The information obtained by CTA or DSA is limited primarily to the vascular lumen. In addition, CTA requires a contrast agent and DSA is invasive. Therefore, these modalities may lack versatility. The establishment of a protocol that enables evaluation of early changes of VAD and is applicable to daily clinical practice is awaited.

In the present case, the timing of when dilatation occurred between the onset of headache and hemorrhage is unclear. There was no symptomatic clue of pathological change. The present case suggests the presence of ruptured VAD that exhibits no change in the vascular lumen or contour at the initial occurrence of headache but later develops marked morphological change and hemorrhage. However, due to its rarity, it is not considered practical from a medical economic viewpoint to uniformly perform frequent detailed imaging studies for similar patients with headache or nuchal pain. Further accumulation of knowledge is required.

Conclusion

We presented a case of VAD that demonstrated no abnormality on CT, MRI, or MRA at the onset of headache in which SAH developed after 7 days. Although such a pathological condition is rare, it is considered clinically important to remember the possibility of similar cases.

Disclosure Statement

The authors declare no conflicts of interest.

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