Case Report

Traumatic vertebral artery dissection and cerebral infarction following head and neck injury with a lucid interval

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Cases: Two patients with cerebral infarction following head and neck injury who showed a lucid interval are presented.

Outcome: A 70-year-old male showed infarctions in the cerebellum bilaterally and the right hypothalamus on the sixth day after an injury with no fracture of the cervical spine, and bilateral dissection of the vertebral arteries was diagnosed. A 74-year-old male showed infarctions in the territory of the right posterior cerebral artery and posterior inferior cerebellar artery 2 days after injury with fractures of the cervical spine (C2 and C3) and was diagnosed as having artery-to-artery embolism based on dissection of the right vertebral artery.

Conclusion: Head and neck injury is a very common presentation in the emergency department. Three-dimensional computed tomography angiography is an effective screening imaging method for vertebral artery dissection that should be carried out on arrival in every patient with fracture of the cervical spine, and even considered in doubtful cases with no fracture.

Key words: 3D-CTA, cerebral infarction, dissection, trauma, vertebral artery

INTRODUCTION

A VERTEBRAL ARTERY (VA) injury, such as VA dissection (VAD), after minor head and neck injury (HNI) is considered relatively common. Although HNI is often seen in daily practice, the diagnosis of cerebral ischemia following HNI is difficult because of the presence of lucid intervals. Two elderly patients who showed late onset cerebral infarction due to traumatic VAD following HNI after falls are presented.

CASE REPORTS

Case 1

A 70-YEAR-OLD MAN DEVELOPED neck pain after falling from a tree, but he did not immediately visit the hospital. On the sixth day after injury, he developed nausea, dizziness, and right-sided deafness and was urgently hospitalized. On admission, he had a right facial palsy, dysarthria, dysphagia, and cerebellar ataxia (right side dominant). Computed tomography (CT) showed a low-density lesion in the right cerebellar hemisphere (Fig. 1A), and diffusion-weighted magnetic resonance imaging showed high-intensity lesions in bilateral cerebellar hemispheres and the right hypothalamus, indicating fresh infarctions (Fig. 1B). Computed tomography showed no skull or cervical spine (CS) fractures. Magnetic resonance angiography (MRA) and 3-D CT angiography (3D-CTA) showed occlusion of the right vertebral artery (VA) and poor filling of the left VA at the proximal portion (Fig. 1C–E). Digital subtraction angiography (DSA) showed proximal occlusion of bilateral VAs and the existence of collateral flow from the left VA leading to the basilar artery (Fig. 1F–H). These findings were considered to indicate bilateral traumatic VAD. The infarction was caused by this pathology. Medical treatment was started with argatroban hydrate, cilostazol, and edaravone. Two months later, the symptoms were improved, and the patient could walk with crutches.

Case 2

A 74-year-old man who suffered CS (C2 and C3) fractures due to a fall from a tree was admitted to our hospital (Fig. 2A,B). On admission, he was alert and had no neurological deficits. He complained of neck pain, controlled by medication. Two days after admission, he suddenly

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developed nausea, fecal incontinence, left hemiparesis, and dysarthria. He was referred to our department, and emergency CT showed hemorrhagic infarctions in the right cerebellar hemisphere, occipital lobe, and thalamus (Fig. 2C,D). Diffusion-weighted magnetic resonance imaging suggested acute cerebral infarction in the territories of the right posterior cerebral artery and posterior inferior cerebellar artery (Fig. 2E,F). The 3D-CTA suggested an irregular arterial wall and stenosis at the level consistent with the C2 fracture (Fig. 2G). Digital subtraction angiography confirmed traumatic VAD (Fig. 2H). Occlusion of the right P2 segment of the posterior cerebral artery was observed that appeared to have been caused by artery-to-artery embolism from the VAD. Because of the presence of hemorrhagic infarction, anticoagulation and antiplatelet therapies were initially avoided, and conservative treatment with edaravone and glycerol was started. After confirming that the hemorrhage had not worsened, antiplatelet therapy was started on the fifth day after symptom onset. The symptoms improved gradually, and the patient was transferred to the rehabilitation hospital 2 months later.

**DISCUSSION**

Although VAD after minor HNI has previously been considered rare, the possibility of encountering this condition in the emergency department has been increasing. Traumatic VAD has been frequently underdiagnosed or misdiagnosed, mainly because many VAD patients remain asymptomatic if the VA is damaged only unilaterally.
Only 12%–20% of unilateral VAD patients initially presented with symptoms and signs of vertebrobasilar ischemia. Even acute occlusion of the dominant-side VA does not always result in neurological deficits. Undiagnosed VAD has been known to cause acute deterioration of previously conscious patients with CS injury. Delayed onset of symptoms with a variable lucid interval, ranging from several days to 3 months, has been reported.

In general, vascular injury after blunt cervical trauma results either from shearing forces secondary to rotational injuries or from direct trauma to the vessel wall from bony prominences or fragments. Distraction/extension, distraction/flexion, and lateral flexion injuries are the underlying mechanisms of traumatic VAD. The mechanism of traumatic VAD is considered to be based on the fact that the intima is the most vulnerable anatomic structure in the vascular wall, and disruption involving the media, coupled with formation and accumulation of thrombus, results in stenosis or complete occlusion which may progress over time causing a lucid interval, as shown in Case 1. The intraluminal blood clot located distal to the dissection is washed out by the bloodstream, causing artery-to-artery embolism, which causes a lucid interval, as shown in Case 2. Although specific CS fractures (location at upper CS, or involvement of the transverse foramen) are predictive of VAD, normal radiographs following minor trauma do not always exclude...

Fig. 2. (a,b) Computed tomography (CT) scan of 74-year-old man who fell from a tree shows the body fracture of C2 and spinous process fracture at C3. (c,d) CT shows hemorrhagic infarction in the territory of the right (R) posterior cerebral artery. (e,f) Diffusion-weighted magnetic resonance imaging suggests fresh infarction. (g) The source image of 3D-CT angiography suggests an irregular arterial wall with stenosis at the level of the C2 fracture (arrows). (h) Right vertebral angiography confirms the findings of the right vertebral artery dissection (arrows).
serious pathology, as in Case 1. Vertebral artery injury without CS fractures, which results from low-energy trauma, frequently involved the V3 or V4 segment and usually presented as VAD, which was the supposed pathology in Case 1. Because this is a dynamic process, there may be substantial change in the injury over time, which leads to the lucid interval after the trauma.

The diagnosis of VAD begins with a suggestive history and clinical signs. It is confirmed traditionally by angiography, or more recently by MRA or 3D-CTA. Presently, the VA in the transverse foramen can be imaged reliably with the use of multi-detector row helical CT, and several authors have advocated that CTA is an effective screening imaging method for VAD, especially in the emergency situation.

The importance of screening guidelines is becoming more evident. As seemingly minor fracture patterns have also been shown to induce VAD, all patients with CS fracture should be examined by 3D-CTA on arrival, or alternatively by MRA in cases with renal failure or allergy to contrast medium.

The important point is how to detect the patients at risk of VAD without CS fracture and how to screen the patient in an asymptomatic phase.

Neurological findings incompatible with brain imaging and/or neck soft tissue injury (neck pain/hematoma/swelling) are the major suspicious factors for VAD without fracture. Such patients should undergo 3D-CTA or MRA in addition to the routine image analysis on arrival. In doubtful cases with only neck and occipital pain, appearing out of proportion to the magnitude of the trauma, 3D-CTA or MRA should be recommended to be carried out within 1 week of the trauma at the outpatient clinic, because a significant number of VADs evolve within 7 to 10 days. As the symptoms and signs may be masked or confused, and some patients may neglect to mention neck trauma on initial arrival, all patients with HNI with suspicious VAD should be informed of the risk for stroke and followed up carefully. Traumatic VAD may be diagnosed in the asymptomatic phase through appropriate screening, enabling early treatment to prevent stroke.

CONFLICT OF INTEREST

NONE.

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