

Case Report

A case of internal carotid artery dissection with delayed cerebral infarction caused by penetrating trauma

Hong-Wei Xu¹, Guo-Fang Yang²

¹Department of Radiology, Shaoxing Second Hospital, Shaoxing 312000, P.R. China; ²Department of Neurosurgery, Shaoxing Second Hospital, Shaoxing 312000, P.R. China

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Abstract: Traumatic internal carotid artery dissection (TICAD) is a rare but significant condition that could be easily un- or misdiagnosed. It could result in severe neurologic deficit and even lead to death. In this study, we present a TICAD case of a 24-year-old man who suffered from a trivial penetrating injury in his left side of neck and presented as delayed severe cerebral infarction. Physical examination on admission showed that he was in good general condition. Neck spiral computed tomography (CT) showed a little air within the channel of a stab wound, but without arterial injury. Twenty-four hours later, the patient couldn't speak and his right upper and lower limbs become weakness and CT scanning illustrated high density in the left middle cerebral artery, suggesting the existence of thrombosis. Nuclear Magnetic Resonance imaging demonstrated absent flow of the left internal carotid artery (ICA) and left middle cerebral artery. The patient received only conservative treatment including absolute bed rest, sustained supine position with the head-of-bed elevation of 30-45°, continuous administration of low flow oxygen, enteral nutritional supplementation via a nasogastric tube, and other symptomatic treatment. No endovascular therapy was attempted. After the 30-hospital day, he showed progressive improvement. As for this rare case, imaging examinations were of great importance in making the accurate diagnosis, which, was help to early diagnosis and timely management that potentially reduced the risks of ischemic stroke and death.

Keywords: Penetrating neck injury, internal carotid artery, dissection, case report

Introduction

Internal carotid artery dissection (ICAD) is one of the major causes of cerebral infarction in young and middle-aged adults [1]. It is categorized as either spontaneous or traumatic [2, 3]. Traumatic internal carotid artery dissection (TICAD) can be caused by blunt or penetrating injury [3]. In penetrating head and neck trauma, injury to the carotid vessels accounts for 5-10% of arterial injuries, with an overall incidence of approximately 0.45% [4]. Except for violent crime, military conflict, motor vehicle accidents, and accidental penetrating injuries are most often due to falls on sharp objects such as sticks and glass. However, these stab injuries usually result in lesser degrees of trauma than other wounds. The actual frequency of accidental stab neck trauma remains unknown, and the development of TICAD caused by it has not been reported. We herein describe a 24-year-

old patient of TICAD following minor penetrating injury and presenting with delayed severe ischemic cerebrovascular accident.

Case report

A 24-year-old male was brought into the emergency room after sustaining a stab wound on the left side of his neck. Approximately 1 hour before admission, he suddenly slipped and fell and therefore his left side neck was punctured by a steel bar projected from the ground. After initial hemostatic therapy and wound debridement in local community health center, he was immediately transferred to our hospital. This study was approved by the ethics committee of Shaoxing Second Hospital, and informed consent was obtained from the patient.

Physical examination on admission revealed an alert and young man with the general condition of T 37.1°C, P 20 time/min, R 62 time/min, and

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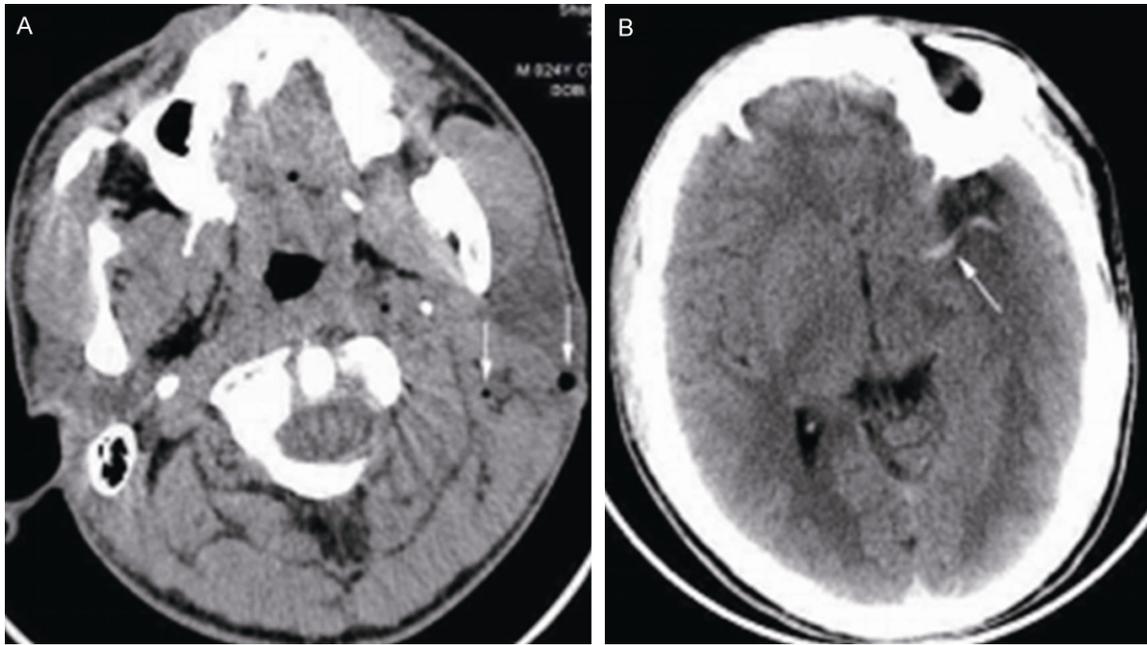


Figure 1. Changes revealed by computerized tomographic (CT) scanning. A. Axial plain CT image at the level of inferior mandible illustrated a little air within the channel of a stab wound (the white arrows). B. Cranial plain CT image showed high density in the left middle cerebral artery, suggesting the existence of thrombus (the white arrow).

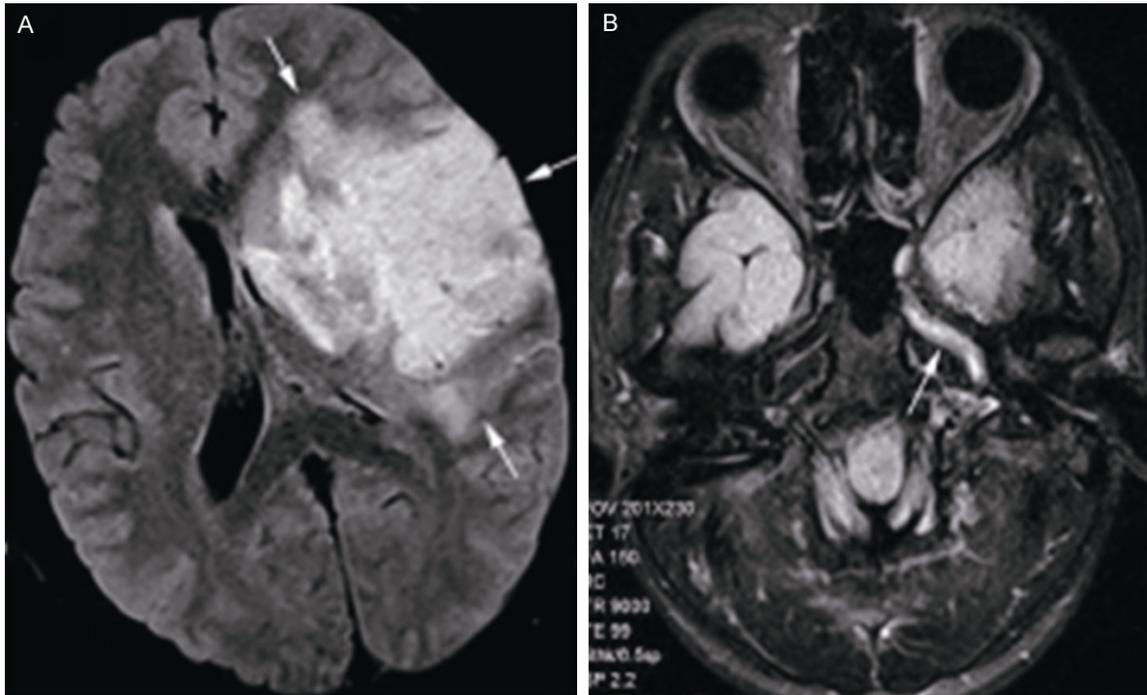


Figure 2. Nuclear magnetic resonance imaging (MRI) results of the patient. A. Axial diffusion-weighted MR image showed an extensive acute infarction of the left middle cerebral artery territory (the white arrows). B. Axial T_1 weighted MRI demonstrated a high signal covering petrous segment of internal carotid artery (the white arrow).

BP 130/70 mmHg. The wound, 0.5 cm in length and 1.0 cm in depth, was on his left neck,

above the level of the angle of the mandible, and posterior to the ear. There was no active

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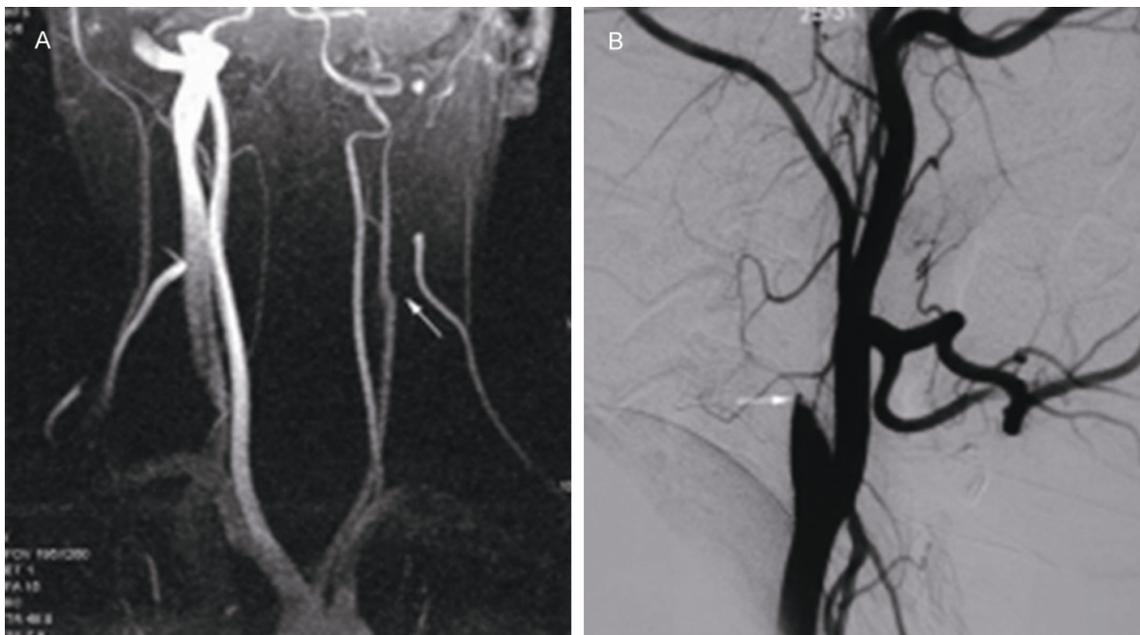


Figure 3. Occlusion unraveled by nuclear magnetic resonance imaging (MRI) and angiography. A. MR angiogram showed occlusion of the left internal carotid artery (the white arrow). B. Angiography confirmed the complete occlusion of the left internal carotid artery with a characteristic “flame-shaped” appearance (the white arrow).

bleeding, palpable thrill, audible bruit, or subcutaneous air. Neurological examination was normal and neck spiral computed tomography (CT) showed a little air within the channel of a stab wound (**Figure 1A**), without arterial injury. Admission laboratory studies, plain radiographs of the neck (antero-posterior, lateral), and bilateral carotid artery Doppler ultrasonography (US) results were normal.

The patient was suggested to stay in our institution for observation despite lack of any symptoms and other signs. Twenty-four hours later, he suddenly couldn't speak and his right upper and lower limbs become weakness. Neurologic examination revealed decreased consciousness, aphasia, and right hemiplegia with grade 0/5 power in lower and upper limbs. Glasgow coma scale (GCS) was 10 (E4, V1, M5) and he was immediately taken for a cranial plain CT scan, which showed high density in the left middle cerebral artery, suggesting the existence of thrombosis (**Figure 1B**). Additionally, a large left middle cerebral artery infarction with a mass effect compressing the lateral ventricle was found. Nuclear Magnetic Resonance imaging (MRI) was performed several hours later and demonstrated a fresh cerebellar infarct of the left middle cerebral artery territory (**Figure 2A**).

Axial T_1 weighted MRI displayed a hyper signal covering petrous segment of internal carotid artery (**Figure 2B**), indicating that there was mural haematoma and thrombosis. Cranio-cervical magnetic resonance angiography (MRA) also revealed absent flow of the left internal carotid artery (ICA) (**Figure 3A**) and left middle cerebral artery. At 48 hours after the trauma, cerebral digital subtraction angiography (DSA) was carried out and confirmed occlusion of the proximal left internal carotid artery with a flame-shaped configuration to the occlusion (**Figure 3B**). Finally, he was diagnosed as left TICAD.

After diagnosis, the patient received only conservative treatment including absolute bed rest, sustained supine position with the head-of-bed elevation of 30-45°, continuous administration of low flow oxygen, enteral nutritional supplementation via a nasogastric tube, and other symptomatic treatment. No endovascular therapy was attempted. Antiplatelet and anticoagulation therapy was not given because he developed a hemorrhagic infarction verified by repeat MR scan. By the 30-hospital day, the patient showed progressive improvement, with a GCS of 13 (E4, V3, M6). Neurologic examination revealed a mild motor aphasia and mild

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right hemiplegia with grade 4/5 power in lower limbs and grade 3/5 in upper limbs. Follow-up MRI documented the recovery of blood flow and the presence of a significant stenosis on the left internal carotid artery. Finally, the patient discharged from hospital to receive rehabilitation therapy.

Follow-up at 8 months after discharge showed significant functional recovery with grade 4/5 power in upper limbs. MRA examination demonstrated spontaneous improvement of the 90% stenosis to a residual of 30% stenosis.

Discussion

The patient demonstrated a rare case of TICAD presenting with delayed severe ischemic stroke following trivial penetrating neck trauma. TICAD occurred when the intimal wall of an artery is damaged as a result of direct trauma. As blood filled the layers of the arterial wall, intramural hematoma formed, which could lead to vessel severe stenosis or occlusion [1]. The main mechanism of the onset of focal brain ischemia is a distal embolization by loose thrombus as shown by our case. Hemodynamic insufficiency due to severe stenosis or occlusion is another cause [2, 3]. The actual incidence might be higher than we presently recognized in clinic, as many episodes might be asymptomatic or cause only minor transient symptoms in virtue of good collateral circulation via the circle of Willis [2].

Understanding the mechanisms of artery injury in the neck is of utmost important in the emergency setting because un- or misdiagnosed TICAD may have severe neurologic deficit and even leading to death [1]. The mortality after penetrating carotid trauma ranges from 5% to 20%, and more than 75% of these deaths relate to stroke [4]. Prompt and accurate diagnosis of TICAD is crucial, because timely and appropriate management could reduce the risk of ischemic stroke and death [5]. However, as shown in our patient, the signs and symptoms of TICAD may present a delayed fashion. The symptom-free period might vary from under an hour to months [5], while the majority might occur in less than 24 hours. This is a key factor that hampers early diagnosis.

A high degree of clinical vigilance on this rare condition is required for making the accurate

and early diagnosis. TICAD should be suspected in any individual with penetrating neck trauma and especially in young patients who present with neurologic symptoms of low-impact trauma. Choosing an appropriate imaging modality is the immediate concern because of its pivotal roles in the diagnosis of TICAD [6]. US, computed tomographic angiography (CTA), MRI, and DSA may be used for definite diagnosis [6]. DSA has long been taken as the gold standard in the diagnosis of TICAD. Angiographic findings are suggestive of dissection including an intimal flap or a double lumen, pseudoaneurysm, and luminal stenosis or occlusion. Stenosis of ICAD is typically irregularly starts about 2 to 3 cm distal to the carotid bulb, and extends for various lengths along the artery. Occlusions have a tapered and flame-like appearance as shown in our case. However, DSA is less commonly used today due to its invasive nature, potential complications, and non-analyzing the arterial wall. It has been increasingly replaced by the faster, noninvasive, less expensive, and more readily available vascular imaging methods, such as MRA and CTA [6]. MRA or CTA could demonstrate luminal narrowing, vessel irregularity, wall thickening/hematoma, pseudoaneurysm, and intimal flap. Source data of MRA and CTA together with additional cross-sectional images could provide direct visualization of the mural haematoma and information about the vessel lumen. US can provide dynamic and “real-time” information about blood flow, however it is less sensitive than CTA or MRA [6]. Non-contrast CT may miss uncomplicated cervical artery dissection, and hence leading to delays in the diagnosis of subsequent stroke. In our study, both plain helical neck CT and bilateral carotid artery US did not reveal any information about TICAD before the onset of ischaemic stroke, and the diagnosis was ultimately established through the imaging features of MRA and DSA.

Currently, many treatment options for TICAD have been pursued involving anticoagulation, antiplatelet treatment, thrombolysis, and revascularization treatment by surgical or endovascular techniques. However, the best therapeutic strategy remains unclear and there is no consensus on the proper management strategy. Unfortunately, we could not perform any of the therapeutic modality mentioned above due to a large middle cerebral artery hemorrhagic

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infarction. In our case, as only choice, conservative treatment which could lead to improvement of the neurological and overall prognosis was performed.

In conclusion, TICAD must be considered in patients with penetrating neck trauma, even the injury is trivial or concealed and the patient is asymptomatic. Negative imaging of the cervical arteries does not completely rule out the diagnosis of TICAD. It should be emphasized that the patients must remain in hospital for one or several days.

Disclosure of conflict of interest

None.

Address correspondence to: Hong-Wei Xu, Department of Neurosurgery, Shaoxing Second Hospital, No123, Yan'an Road, Shaoxing 312000, P.R. China. Tel: +86-0575-88055102; Fax: +86-0575-88055-102; E-mail: chinaxhw@163.com

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