Missed diagnoses of Blunt Cerebrovascular Injuries result in Ischemic stroke: two cases report

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Case Report

Keywords: Blunt cerebrovascular injury, Traumatic internal carotid artery dissection, Acute ischemic stroke, carotid artery injuries.

Posted Date: January 3rd, 2023

DOI: https://doi.org/10.21203/rs.3.rs-2374033/v1

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Abstract

Here we report two patients with a car accident injury who suffered a cerebral infarction following a missed diagnosis of blunt cerebrovascular injury. Difficulty in diagnosis of these cases because of no neurological signs or symptoms on initial assessment is a lesson for physicians from emergency room. Our conclusion upon dealing with the situation is that traumatic blunt cerebrovascular injury has potentially devastating consequences. There is often a delay between the trauma and the onset of symptoms of neurological deficit. Therefore, clinicians should be cautious in this situation. Early detection and intervention are needed to prevent the development of ischemic stroke secondary to blunt cerebrovascular injury in combination with cerebral infarction, thereby improving the prognosis of patients.

Introduction

Ischemic stroke is an important health issue because of the high cost it imposes on patients, families and society. Stroke survivors may experience years of cognitive or physical disability, as well as poorer health-related quality of life[1]. Ischemic stroke is a serious complication of blunt traumatic cerebrovascular injury [2]. Symptoms of blunt cerebrovascular injury such as headache, neck pain and dizziness are not highly specific. Therefore, missed diagnoses can lead to serious neurological damage and sequelae. Meanwhile, failing to diagnose these injuries meaningfully increases the morbidity and mortality. In order to focus on reducing the occurrence of ischemic stroke after trauma and improving the rate of early diagnosis, we conducted this case report in our trauma cohort to characterize patients who were diagnosed with acute ischemic stroke with some delay after hospitalization.

Case Presentation

Case 1

A previously healthy 55-year-old male was referred to our hospital 20 minutes after a collision with a car on a bicycle, presenting with headache and drowsiness. On admission, the patient had a Glasgow Coma Scale score of 15, a scalp tear, normal limb muscle strength and no focal neurological deficits. There was no head or neck pain. The initial computed tomography (CT) scan of the head was normal (Fig. 1A). Eleven hours after the traffic accident the patient's left limb was hemiplegic with muscle strength of grade 1. Repeat CT of the head showed changes consistent with a right middle cerebral artery infarction (Fig. 1B). Enhanced CTA of the head and neck showed traumatic entrapment of the right internal carotid artery (Fig. 1C) as well as complete occlusion of the right middle cerebral artery lumen (Fig. 1D). The patient immediately underwent a total cerebral angiogram, which revealed a right internal carotid artery dissection (Fig. 2A and B). We first performed a right internal carotid artery stenting (Fig. 2C and D) followed by a middle cerebral artery thrombectomy (Fig. 2E and F). Postoperative antiplatelet therapy with aspirin and clopidogrel was administered. He was admitted to the ICU for close observation of his vital signs and neurological status. On the third postoperative day, the patient developed decreased
consciousness and a dilated right pupil, and a repeat cranial CT indicated a large cerebral infarction (Fig. 3A). The patient underwent emergency decompressive craniectomy. A repeat cranial CT indicated cerebral hemorrhage in the infarcted area (Fig. 3B). After 15 days in the intensive care unit and 17 days in the neurosurgery department, he was transferred to a rehabilitation facility for further rehabilitation, but still had severe left hemiplegia. MRI prior to transfer out of hospital showed ischemic infarction in the right frontotemporal lobe (Fig. 3C) with good flow in the right internal carotid and middle cerebral arteries (Fig. 3D).

**Case 2**

A 56-year-old male, without basic diseases, presented to the emergency department of our hospital with a one-hour history of multiple traumas following motor vehicle crashes. On admission, the patient had a Glasgow Coma Scale score of 15. There is a 6cm deep laceration in the right calf, visible muscle and bone. There is a 3cm*4cm lesion on the left knee joint. There's a 1cm*2cm laceration to the lower right abdomen. Bruising and swelling on the right side of her face, and abrasions all over the body. Computed tomography (CT) of the chest showed Rib fracture (3rd and 5th rib on left side). CT of abdomen and the spine showed no significant abnormalities. Cranial CT suggested a right occipital lobe cerebral contusion with a small hematoma and a small amount of hemorrhage in the subarachnoid space (Fig. 4A). CT examination of the extremities suggested dislocation of left shoulder with injury of glenoid cavity. His cardiac, pulmonary, and abdominal examinations are without significant findings. In the second day, we had examined the Computed tomography of brain again. The result was the same with the first day (Fig. 4B). For the next three days, the patient continued to show restlessness, but no abnormal muscle strength was observed. The symptom is unexplained by brain contusion. In the fourth day, the enhanced CTA of Carotid artery showed the left internal carotid artery was occluded and non-calcified plaque in the left common carotid artery with moderate stenosis. Hypoperfusion of the left cerebral hemisphere and massive cerebral infarction on the left side of the brain was showed in the enhanced Cranial CT (Fig. 4C). One day later, the Glasgow Coma Scale score of this patient was decreased to 7. The patient underwent emergency decompressive craniectomy. After 11 days in the intensive care unit and 13 days in the neurosurgery department, he was transferred to a rehabilitation facility for further rehabilitation with Glasgow Coma Scale score 12 (Fig. 4D). Three months later, the patient returned to the hospital for head CT examination (Fig. 4E).

**Discussion**

The prevalence of blunt carotid artery injury is as high as 0.50% in all patients with blunt trauma.[3–8] Motor vehicle accidents are the most common cause of blunt carotid artery trauma, with studies showing a prevalence of 0.67% in victims of motor vehicle accidents.[9] Our study describes the two trauma patients were diagnosed with acute ischemic stroke delayed after hospitalization. All these two patients were suffered acute ischemic stroke after motor vehicle accidents, but the reason of cerebral infarction
may be different in the two patients. The reason of the first patient is traumatic internal carotid artery dissection and the reason of the second patient may be plaque shedding due to blunt injury.

Crissey described four possible mechanisms that can lead to a blunt carotid injury, including direct blow to the neck, neck hyperextension associated with rotation, blunt intra–oral trauma and basilar skull fracture involving the carotid canal.[10] Although direct blow to the neck are the most common cause at 50%, it is hyperextension with rotation that is the direct cause of carotid artery injury in most motor vehicle accidents. The compression of the internal carotid artery by the mandible and upper cervical vertebrae during hyperextension with rotation in a traffic accident may have contributed to the development of carotid artery dissection (CAD) and plaque shedding.[11, 12]

Studies have shown that factors significantly associated with carotid artery injuries include closed head injury, basilar skull fracture, facial fracture, spinal (especially cervical spine) fracture and thoracic injury, with closed head injury being the most common single associated injury.[13] It has also been suggested that combined injuries to the head, face and cervical spine and combined head and chest injuries significantly increase the risk of carotid artery injury.[3, 14] Unlike previously reported carotid artery injuries, our patient's carotid artery injury was not combined with other injuries.

Although headache and neck pain are the most common clinical manifestations of traumatic carotid injury, the diagnosis of blunt cerebrovascular injuries is frequently delayed due to the absence of initial signs and symptoms.[15] Besides, two studies have shown that over 40% of patients develop signs and symptoms sometimes after the initial normal neurological examination.[4, 16] Koleitat et al. reported that a delayed diagnosis greater than 48 hours significantly worsens outcomes after blunt cerebrovascular injuries.[17] Biffi et al. recommended aggressive screening to reduce delayed diagnosis and missed diagnoses in patients with severe cervical hyperextension/rotation or hyperflexion compatible trauma mechanisms, evidence of cerebral infarction on CT, or unexplained neurological deficits, TIA or Horner's syndrome.[18]

CT angiography (CTA) and MRI/Magnetic Resonance Angiography (MRA) are currently the preferred imaging tests for suspected blunt cerebrovascular injuries. CTA allows rapid diagnosis of blunt cerebrovascular injuries and is particularly useful for early detection in the emergency room for severely injured patients, but some patients are unable to undergo CTA due to allergic reactions to the contrast medium. MRI/MRA can provide a comprehensive assessment of blunt cerebrovascular injuries, but is usually slower than CTA and some trauma patients are unable to cooperate with the completion of an MRI/MRA. Ultrasound is a non-invasive, bedside, real-time diagnostic tool that is an effective method for diagnosing blunt cerebrovascular injuries. Although ultrasound relies on operator experience and is poorly visualised for intracranial vascular ultrasound, it is useful for the progression or recanalisation of blunt cerebrovascular injuries. Although our patient was asymptomatic, if we had screened this patient with ultrasound, we might have detected his blunt cerebrovascular injuries much earlier.

Although there are several treatment options for cerebrovascular dissection, including observation with expectant management, antiplatelet agents, anticoagulation, thrombolysis, stenting and surgery.[19]
Patients with blunt cerebrovascular injuries are prone to thrombosis leading to stroke, so anticoagulation and antiplatelet therapy are the cornerstone treatment when contraindications are ruled out. In our cases, thrombolysis and therapeutic anticoagulation were not started as because this was a massive stroke with a high risk of haemorrhagic transformation; instead, he was initially started on antiplatelet agents.

In summary, car accident injuries may lead to traumatic blunt cerebrovascular injuries, especially in those who present with severe headaches and neck pain. Delayed clinical symptoms may occur in such a condition of traumatic blunt cerebrovascular injuries. Ultrasound, CTA and MRI are very important for the early diagnosis of blunt cerebrovascular injuries. Therefore, clinicians should be cautious in treating traumatic blunt cerebrovascular injuries patients with severe headaches and neck pain.

Declarations

**Ethical approval and consent**

The ethical approval and consent of this study are approved by Clinical Research Ethics Committee of the first affiliated hospital of Zhejiang university. All included patients gave their oral and written informed consent. This research involving human data have been performed in accordance with the Declaration of Helsinki.

**Consent for publication**

All included patients gave their oral and written informed consent.

**Availability of data and materials**

The data and materials of this study can obtained from the corresponding author.

**Competing interests**

All the authors do not have any competing interest to declare.

**Funding**

This study did not receive any funding in any form.

**Authors’ contributions**

Xiaofeng Yang conceived this study. Shaoyang Zhang designed the study. Weier Wang and Yadong Wang acquired and analyzed the data. Meng Jiang, Qiang Zhou and Mingjie Wu contributed analysis tools. Shaoyang Zhang wrote the paper. All authors read and approved the final manuscript.

**Acknowledgement**

Not applicable.
References


**Figures**

**Figure 1**

Brain CT on the first and second day of admission.
A: CT showed slight swelling of the right frontotemporal lobe on the first day of admission; B: repeat brain CT showed large hypodense lesion (red arrow) in the frontotemproparietal lobe. C: CTA showed right internal carotid artery dissection (red arrow); D: cerebral CT angiogram images showed a complete obliteration of blood flow in right middle cerebral artery (red arrow).

Figure 2
pre- and post-operative cerebral angiography. Preoperative frontal (A) and lateral (B) cerebral angiogram suggestive of right internal carotid artery dissection (red arrow); Ortho (C) and lateral (D) cerebral angiography showed disappearance of internal carotid artery dissection after stenting; Ortho-(E) and lateral (F) cerebral angiograms showed patency of middle cerebral artery blood flow after middle cerebral artery thrombectomy (red arrow).

Figure 3

Cranial CT before and after decompressive craniectomy.

A: brain CT showed large hypodense lesion in frontotemporoparietal region with a midline shift; B: head CT indicated cerebral haemorrhage in the area of infarction. C: MRI shows ischemic infarction of the right
frontotemporal lobe; D: MRA showed good blood flow in the right internal carotid artery and middle cerebral artery.

Figure 4

The CT of the patient in the first day showed a right occipital lobe cerebral contusion with a small hematoma and a small amount of hemorrhage in the subarachnoid space. B: There was little difference from the first day of examination. C: The enhanced CTA of Carotid artery showed the left internal carotid artery was occluded and non-calcified plaque in the left common carotid artery with moderate stenosis. Hypoperfusion of the left cerebral hemisphere and massive cerebral infarction on the left side of the brain was showed in the enhanced Cranial CT. D: CT scan of the head on the first postoperative day. E: CT scan of the head after three months.