

Delayed Hemorrhagic Manifestation of Blunt Carotid Artery Injury

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Blunt carotid artery injury is uncommon, yet not rare. However, it is often underdiagnosed because of inconsistent early symptoms or masking by the presence of coexisting brain and spinal injuries. The delay between the accident and the onset of cerebral ischemic symptoms is variable and has been reported to range from minutes to ten years. However, to our knowledge, there has been no report on a case presented with delayed intracerebral hemorrhage 25 months after blunt carotid artery injury. We report on a case with discussion of supporting evidence and possible mechanisms.

KEY WORDS : Blunt trauma · Carotid injury · Delayed intracerebral hemorrhage.

Introduction

Blunt internal carotid injury is generally considered to be an underdiagnosed and potentially avoidable cause of delayed deterioration among blunt trauma patients^{2,8}). In published series, the diagnosis of blunt carotid artery injury has usually been made following a sudden onset of unexpected neurological deficits. The delayed deficits were usually ischemic in origin, resulting from occlusion of the carotid artery or a thromboembolic phenomenon these ischemic symptoms may develop months or years after the initial trauma^{4,7,10}). Other modes of presentation include subarachnoid hemorrhage or visual loss secondary to the formation of an intracranial aneurysm^{7,11}). However, to our knowledge, there have been no reports of a case presented with delayed intracerebral hemorrhage 25 months after blunt carotid artery injury. We report on the case with discussion of supporting evidences and possible mechanisms.

Case Presentation

A 45-year-old man visited the emergency room ten minutes after his head and right shoulder had been compressed under a hydraulic machine. His consciousness was intact, but there was epistaxis and ear bleeding on both sides. There were

swelling and scratch wounds around the right periorbital area, zygoma, mandible, and the right shoulder. Upon neurologic examination, the right pupil was enlarged to 5mm and had lost light reflex, in addition to ptosis and ophthalmoplegia. His right eye could only perceive the presence of the light. There were no motor or sensory deficits. Radiologic evaluation including skull X-rays and brain, orbit, and facial bone computed tomography(CT) scans, revealed complex fractures of the anterior skull base, right lateral orbital wall, zygomatic arch, both temporal bases, and mandibular body (along with pneumocephalus) (Fig. 1). There was no evidence of cervical spine injury, in spite of the floating shoulder on the right side with fractures of the scapular neck and clavicle. He was treated with mannitol, antibiotics, and high-dose steroids. Cerebrospinal fluid leakage and pneumocephalus resolved, but the visual acuity of the right eye didn't improve. He had received an operation for the right shoulder fractures and was discharged one month after admission.

He followed up regularly in the outpatient department. He

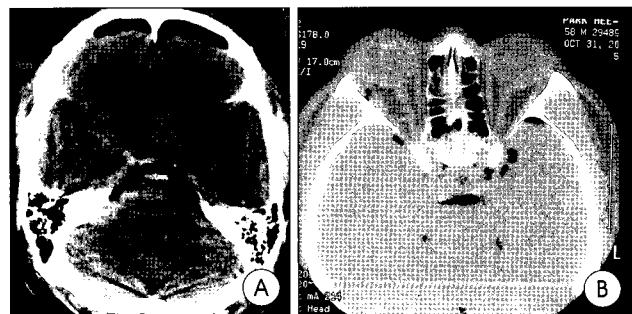


Fig. 1. Precontrast computed tomography scan (A) and bone setting view (B) taken at the emergency room show a large amount of pneumocephalus and multiple fractures at the cranial base.

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showed stable course without episodes of sensorimotor deficits or other neurologic deficits other than the initial deficits of visual loss and ophthalmoplegia in the right eye. About 20 months after the injury, he complained intermittent frontal headaches and excessive nasal discharge. Glucose was not detected in the nasal discharge. CT scans which was taken to rule out CSF rhinorrhea revealed no abnormalities (Fig. 2A). Twenty-five months after the injury, he experienced sudden severe headache and vomiting, and visited the emergency room. His consciousness was intact, but he showed mild left hemiparesis. Brain CT scans revealed a huge intracerebral hemorrhage extending from the right temporal pole to the high parietal lobe accompanied by an intraventricular clot (Fig. 2B). Because he was not hypertensive and had no systemic factors for intracranial hemorrhage, we performed transfemoral cerebral angiography. The angiography revealed complete occlusion of the cervical internal carotid artery (ICA) 1cm distal to the carotid bifurcation and the development of collateral vessels from the external carotid system (Fig. 2C). The left ICA (Fig. 2D) and the vertebrobasilar system were normal. Collateral blood flow through the anterior communicating artery was not sufficient due to stenosis of the proximal right A1 segment. Collateral blood flow through the posterior communicating artery was not noticeable. The patient became drowsy on the next day. An emergent hematoma evacuation was performed with concurrent encephaloduroarteriosynangiosis using the posterior branch of the right superficial temporal artery. He recovered well and was discharged one month after admission without hemiparesis.

During the follow-up period, he did not complain the headache. A follow-up arteriogram which was taken seven months after the operation showed mild hypertrophy of the superficial temporal artery and faint neovascularization (Fig. 3A, B). The left ICA (Fig. 3C) and the vertebrobasilar system showed normal features without progression of stenosis, and poor collateral blood flow through the anterior and posterior communicating arteries was the same as before. It is currently 21 months after the bleeding and he is doing well without headache or further neurologic deficits.

Discussion

Causes of carotid occlusion

The patient's head was compressed, resulting in complex skull

base fractures and a mandibular fracture. This indicated that his mandible was compressed against the upper cervical vertebrae. Compression of the carotid artery between the mandible and the upper cervical vertebrae (which may result

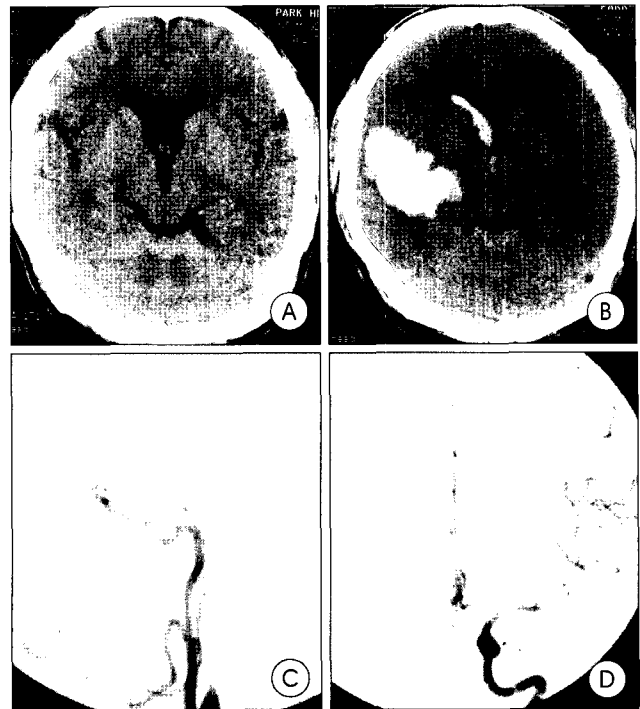


Fig. 2. A : Precontrast computed tomography(CT) scan taken 20 months after the injury shows no abnormalities. B : Precontrast CT scan taken 25 months after the injury when the patient complained sudden headache shows a huge intracerebral hemorrhage extending from the right temporal pole to the high parietal lobe, accompanied by an intraventricular clot. C : The right common carotid arteriogram taken on the same day as the CT scan (B) shows complete occlusion of the right internal carotid artery just distal to the bifurcation. Collateral blood flow from the internal maxillary artery to the ophthalmic artery is well-noted in the more delayed phase. D : The left internal carotid arteriogram shows normal features without stenosis. Collateral blood flow through the anterior communicating artery is limited due to the stenosis of the proximal A1 segment.

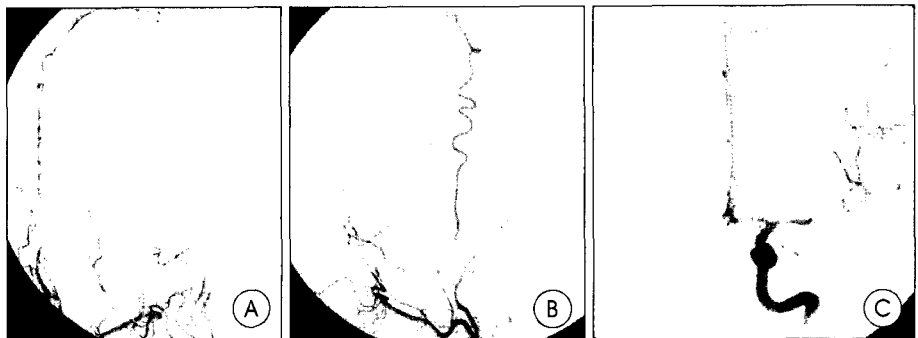


Fig. 3. Arteriograms taken seven months after the operation. Right external carotid arteriograms with anterior-posterior (A) and lateral (B) views show mild hypertrophy of the superficial temporal artery and faint neovascularization. The left internal carotid arteriogram (C) shows normal features without progression of stenosis while poor collateral blood flow through the anterior communicating artery is the same as before.

in intimal disruption or mural hematoma) is one of the representative mechanisms of blunt cerebrovascular injury¹. Angiography revealed an occlusion of the carotid artery just distal to the carotid bifurcation, the most common site of traumatic carotid artery injury⁶. Furthermore, other components of the vascular system, such as the left ICA and the vertebral system, showed normal features and no changes in the follow-up evaluation. This suggested that the possibility of an idiopathic cerebrovascular occlusive disease (such as moyamoya disease) was less likely. He had no hypertension or atherosclerosis. Aforementioned evidences suggest that the blunt carotid artery injury which occurred at the time of the compressive trauma of the head was probably the cause of the progressive carotid artery occlusion.

Mechanisms of presentation

The hallmarks of blunt cerebrovascular injury are delayed presentation and delayed diagnosis¹. Delayed focal cerebral ischemic symptoms are the most common presenting symptoms^{4,7,10}. Very rarely, subarachnoid hemorrhage or visual loss secondary to a supraclinoid aneurysm formation is reported^{7,11}. The time between the injury and the development of vessel occlusion and thrombosis is variable. The vast majority of neurological symptoms occurred from the vascular injury present more than ten hours after injury, with 23% to 50% becoming symptomatic more than one day after injury¹. Fabian et al., reported that the mean time before diagnosis was 58 hours². In another report, the delay between the accident and the onset of cerebral ischemic symptoms ranged from 30 minutes to ten years. In more than two-thirds of patients, transient ischemic attacks or strokes developed within hours or days after the accident⁴. Regarding the delayed presentations of blunt carotid dissection, the symptoms developed two weeks to six months after the accident. The presenting symptoms were cerebral ischemia or visual loss secondary to an intracranial dissecting aneurysm⁷.

To our knowledge, there is no report regarding a delayed intracerebral hemorrhagic manifestation of blunt carotid artery dissection. Angiography of the patient at the time of hemorrhage showed complete occlusion of the ICA just distal to the carotid bifurcation. Collateral circulation through the anterior communicating artery was observed, but not effective in rescuing the right hemisphere because of the severe stenosis of the proximal A1 segment. There was no significant collateral blood flow through the posterior communicating artery on the vertebral arteriogram. Nevertheless, he showed no cerebral ischemic symptoms during the two-year period. Considering

the development of extracranial to intracranial collateral blood flow through the communication between the internal maxillary artery and the ophthalmic artery, the occlusion of the ICA might have progressed gradually. These processes seem to have prevented the development of cerebral ischemic symptoms, but fully dilated collateral vessels may put the patient at risk for cerebral hemorrhage, just like in cases of moyamoya disease.

Considerations on management

Early diagnosis and successful management of traumatic carotid artery dissections require a high index of clinical suspicion. Besides disturbance of consciousness, hemiparesis, or dysphasia that do not correspond to CT findings, the risk factors of blunt carotid artery injury should be suspected. These include a clear history of severe hyperextension or direct cervical trauma, a basilar cranial fracture extending through either foramen lacerum, a mandibular fracture, a cervical hematoma, unexplained anisocoria or Horner's syndrome, and diabetes insipidus without impending brain death^{2,5,8,12}. Among all blunt trauma patients, the incidence of blunt carotid artery injury is ranged from 0.08 to 0.33%. In a prospective study regarding the incidence of unsuspected blunt carotid artery injury in a large consecutive series of blunt trauma patients, the detected incidence of blunt carotid artery injury among all patients undergoing aortic evaluation was 3.5%. Incidence among the patients undergoing aortic evaluation that had not been suspected of having blunt carotid artery injury was 2.5%, which is similar to 3.5%. They thus concluded that blunt carotid artery injuries often remain asymptomatic and no specific risk factor for blunt carotid artery injury beyond the severity of trauma were identified, suggesting the importance of initial screening for this group of severely injured patients⁸. Though the gold standard for the diagnosis of carotid artery injury is conventional angiography, the effectiveness of Doppler sonography⁹, CT angiography, and MR angiography¹³ is reported despite the inherent shortcomings.

The mainstay in the initial management of blunt carotid artery is anticoagulation. There are many reports about the beneficial results of anticoagulation following the diagnosis of blunt carotid artery injury before it becomes symptomatic^{2,5,8}. In our case, the occlusion of the carotid artery seems to have progressed slowly because he had remained asymptomatic with collateral vessels for two and a half years. If it had been diagnosed early with screening measures and anticoagulation therapy had been done, more favorable results could have been obtained. In our case, the treatment principle of moyamoya

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disease can be applied. Although recent publications favor direct revascularization rather than indirect for adult hemorrhagic moyamoya disease, we think that indirect revascularization performed concurrently with hematoma evacuation still deserves further research. However, this is outside the scope of this report.

Conclusion

We would like to suggest two meanings of this report. One is to report a case of blunt carotid artery injury with a very rare course of natural history that is, very delayed hemorrhagic presentation. The other is to suggest the importance of early diagnosis by some kind of screening protocol and initiation of specific treatment.

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