—Case Reports—

Internal Carotid Artery Stenosis Due to Atherosclerotic Plaque Damage after Whiplash Injury

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Abstract

Blunt traumatic occlusion or stenosis of the internal carotid artery is a rare complication of whiplash injury and may not be recognized until the onset of neurological symptoms. The clinical course can vary considerably, with regard to both the symptoms and the interval between injury and manifestation. A dissecting aneurysm and intimal tear are usually observed after blunt internal carotid artery injury. Atherosclerotic plaque is often observed in the cervical internal carotid artery and carotid bifurcation, but involvement of plaque has been confirmed in relatively few cases of blunt traumatic internal carotid injury. We describe a 58-year-old man who developed cerebral embolism due to intraplaque thrombus after a minor whiplash injury and was successfully treated with carotid endarterectomy. Because thrombosis occurred within the atherosclerotic plaque, we named the mechanism of this case "plaque injury".

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Key words: atherosclerosis, carotid artery, whiplash injury, cerebral infarction, carotid endarterectomy

Introduction

Traumatic occlusion or stenosis of the internal carotid artery (ICA) is a rare complication of blunt cervical trauma[1–3]. Blunt ICA injury is usually caused by motor vehicle accidents and may not be recognized until it causes neurological deficits[4,5]. The clinical course may vary considerably with respect to both the type of symptoms and the interval between injury and the onset of symptoms[5]. We report an unusual case of a 58-year-old man who had a cerebral embolism due to intraplaque thrombus after minor whiplash injury and was successfully treated by carotid endarterectomy.

Case Report

A 58-year-old male taxi driver was involved in a rear-end collision in June 2004. It was a minor traffic accident, and the car's rear bumper was not dented. He did not lose consciousness and drove the car home himself. Five hours after the accident, he complained of nausea, neck pain, and aphasia. He then walked to a local orthopedic clinic, and whiplash injury was diagnosed. After 35 days, he

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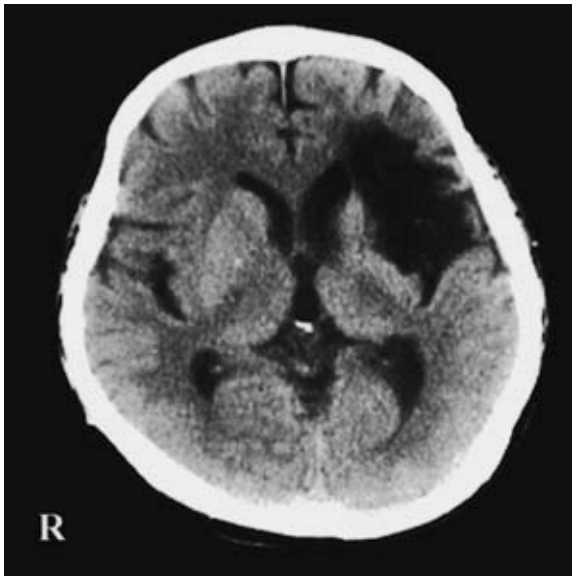

Fig. 1 Brain CT scan of the patient from the initial examination shows cerebral infarction on the left frontal lobe, left basal ganglia, and left insula.

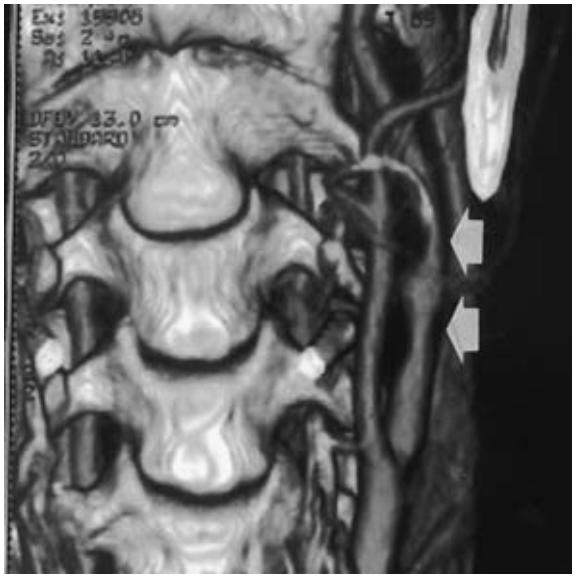

Fig. 3 Three-dimmentional CT angiography shows an irregular stenosis of the left ICA. **White arrows** indicate left internal carotid artery.

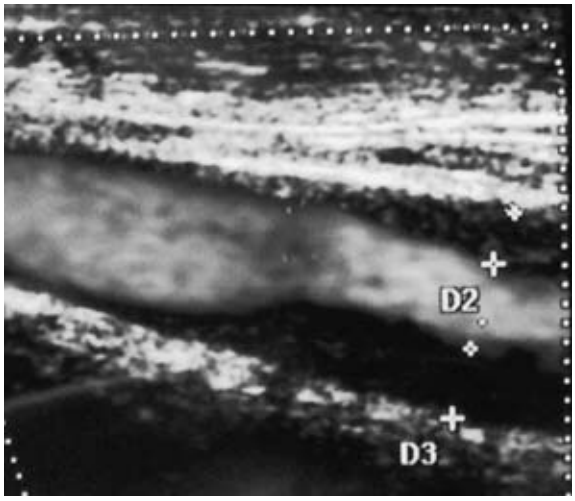

Fig. 2 Doppler ultrasonography shows stenosis of the left internal carotid artery and a hypoechoic atherosclerotic plaque. Right side: rostral, left side: caudal.

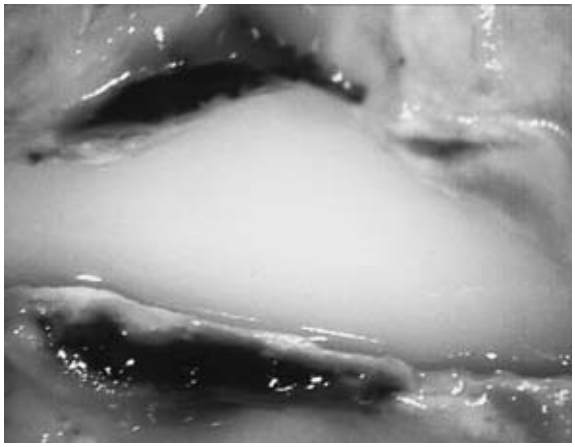

Fig. 4 The photograph shows a thrombus within the removed atherosclerotic plaque after carotid endarterectomy. Right side: rostral, left side: caudal.

visited our department with aphasia. His consciousness was clear. Neurologic examination revealed motor-dominant aphasia. An X-ray film of the cervical spine was normal.

Computerized tomography (CT) of the brain demonstrated infarction in the left frontal lobe, left basal ganglia, and left insula (**Fig. 1**). Doppler ultrasound showed a hypoechoic atherosclerotic plaque in the left ICA (**Fig. 2**). Three-dimensional CT angiography revealed irregular stenosis of the left ICA from just above the bulb extending 5 cm distally (**Fig. 3**). We diagnosed cerebral embolism due to blunt trauma to the cervical ICA after whiplash injury.

First, the patient was given anticoagulant therapy (warfarin at 4～8 mg/day). Fifty-two days after the injury, he experienced transient right hemiparesis for 30 minutes. Serial three-dimensional CT angiography revealed that the ICA stenosis was unchanged. Revascularization surgery was considered because of the new neurological deficit

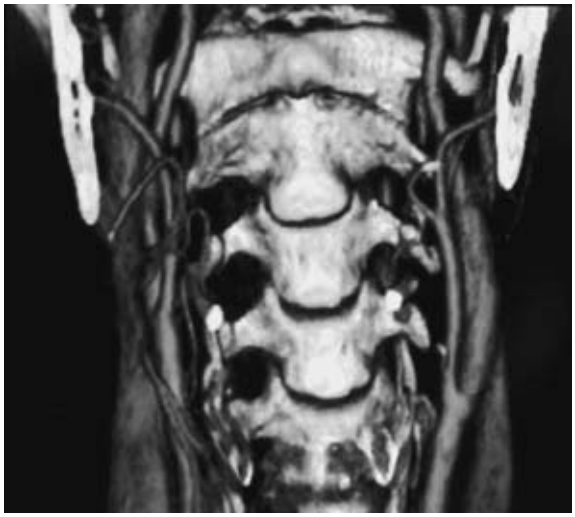

Fig. 5 Three-dimmensional CT angiography after carotid endarterectomy shows a widely patent ICA and decreased stenosis.

despite anticoagulant therapy. Seventy-five days after injury, carotid endarterectomy was performed under general anesthesia. Routine endarterectomy was performed, and the arteriotomy was repaired with a 6-0 prolene suture and patch angioplasty. A thrombus was found in the resected atherosclerotic plaque **(Fig. 4)**. Three-dimensional CT angiography performed 2 weeks after the operation revealed a widely patent ICA and decreased stenosis **(Fig. 5)**. The patient remained neurologically unchanged and was discharged 20 days after surgery.

Discussion

Blunt trauma to the ICA occurs infrequently and accounts for only 3% to 10% of all reported carotid injuries[2]. The diagnosis of blunt ICA injury is often difficult at the time of initial presentation. Because it is unsuspected, there are few (if any) external signs, it is often masked by more obvious injuries, there is often a delay in the appearance of signs, and the results of initial head CT are often normal[2,5,6]. Early diagnosis can only be achieved by suspecting the injury and following up the patient with repeated neurological examination[2,5,6]. Thus, one must always keep in mind the causes and mechanisms of blunt carotid injury and intervene before cerebral infarction occurs[2,5,6].

Suggested mechanisms of ICA injury include: hyperextension of the neck with contralateral rotation of the head, a direct blow to the neck, blunt intraoral trauma, skull base fractures injuring the intrapetrous portion of the vessel, therapeutic and diagnostic carotid massage, and attempted strangulation[1–7]. Two areas are most frequently involved: (1) the cervical segment of the ICA, usually 1 to 3 cm above its bifurcation, and (2) the part of the vessel near the base of the skull and just distal to the origin of the posterior communicating artery[2,7,8]. The pathogenesis of traumatic occlusion or stenosis of the ICA is generally believed to be a tunica intima tear of the artery with subsequent elevation of a flap by the force of blood flow[2,7]. Yamada *et al.* have summarized the pathologic findings in 52 cases of blunt trauma to the ICA[9]. They have reported that a dissecting aneurysm and intimal tear lead to partial or complete thrombosis of the artery because of the resultant exposure of the underlying subintimal tissue to platelets. Propagation of the thrombus either proximally or distally could lead to total occlusion of the ICA[2,6,10]. Secondary to local thrombus formation, extension or embolization into the cerebral circulation can lead to severe neurologic sequelae or death[2,6,10]. The thrombus may also be the source of emboli with the potential to occlude small intracranial vessels and produce neurological deficits[2]. Atherosclerotic plaque predisposes a vessel to the development of intimal tears, but plaque has been confirmed in relatively few of the reported cases[1–10].

The pathogenesis of the injury in our case was traction on the carotid artery, followed by a tear in the plaque, platelet aggregation, and thrombus formation with a subsequent embolus causing cerebral infarction. Nontraumatic thrombus is sometimes observed in an atherosclerotic plaque. Therefore, the question arises in our case as to whether intraplaque thrombus occurred before or after the whiplash injury. In either case, the presence of intraplaque thrombus was a definite factor in the pathogenesis of our case. Because damage to the atherosclerotic plaque was important in the mechanism, we called it “plaque injury”.

The onset of neurologic symptoms in patients with ICA occlusion or stenosis due to blunt trauma

may be immediate, within 24 to 48 hours, or late (days or weeks after the injury)[2,5,11]. In our case, motor aphasia was noted 5 hours after the traffic accident.

Doppler ultrasound was positive in our case and may be a useful screening method. If the results of Doppler ultrasonography as negative, the patient can be followed up with serial neurological examination[2,11]. Angiography is the gold standard and should be done if a patient has any ipsilateral external signs of neck injury on physical examination associated with a change in the level of consciousness or lateralizing neurologic findings that are unexplained by CT[2,10].

Blunt cervical ICA injury has a mortality rate as high as 40% and a morbidity rate as high as 80%, and its treatment is still controversial[2,3,12,13]. The alternatives to angiography include observation, anticoagulation, ligation of the carotid artery with or without extracranial-intracranial bypass, and arterial reconstruction[2]. The primary goal of therapy is to prevent the development of neurological deficits. Patients with major fixed neurological deficits do not respond well to any treatment. The location of the arterial injury, type of vascular defect, neurological and cardiovascular status, and associated injuries are all important considerations when deciding on the treatment of these patients.

Although there are surgical techniques to improve access to the region, for one group of lesions the risks are probably too high to justify an operation[2,13]. These lesions include an intimal flap or dissection that may extend to the base of the skull. Patients with detectable lesions of the carotid artery that are surgically accessible should undergo vascular repair if there is evidence of transient cerebral ischemia or a completed mild neurological deficit[3,5,13]. In contrast, Li *et al.* have suggested anticoagulation as the primary treatment, while revascularization surgery might be considered for progressive neurologic deficits[2]. In fact, a clinical review of internal carotid injuries has shown no difference in outcome between patients treated operatively or with anticoagulation for small intimal tears. Our patient was treated successfully with carotid endarterectomy.

It is important for clinicians dealing with acute trauma patients to be mindful of this rare, but disabling, vascular complication that can follow even minor whiplash injury. To avoid prolonged morbidity, a high index of suspicion and early evaluation are recommended[1–13].

Conclusion

We have reported a case of cerebral embolism due to intra-plaque thrombosis after minor whiplash injury, which has not been reported previously.

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