Cerebral Infarction following Traumatic Internal Carotid Artery Dissection without Significant Neck Injury: A casereport

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A 37-year-old female had an occlusion of right middle cerebral artery (MCA) due to traumatic internal carotid artery (ICA) dissection without significant neck injury after a traffic accident. Although this phenomenon has been documented in recent studies, it remains a relatively rare event. She presented with a left shoulder abrasion injury and mild neck pain initially, although there was no direct head injury or loss of consciousness after the traffic accident. After 12 hours, she had developed confusion, weakness over left limbs and slurred speech. A hyperdense area on the right frontotemporoparietal lobes was found on magnetic resonance imaging (diffusion-weighted imaging). The infarct of the right M1 segment of the MCA was confirmed with magnetic resonance imaging and magnetic resonance angiography. An atherosclerotic plaque and dissection in her right ICA were also found after Doppler examination. Craniectomy was performed on the next day after the accident due to the severe brain edema. After six months of rehabilitation, her functional status progressively improved. This case report reminds clinicians that delayed cerebral infarction could occur following an accident of cervical stretching with ICA injury, especially in patients with carotid atherosclerosis, and a Doppler examination of carotid artery is a good screening tool to detect the dissection. (Tw J Phys Med Rehabil 2011; 39(4): 233 - 238)

Key Words: neck injury, carotid artery dissection, atherosclerosis, cerebral infarction

INTRODUCTION

Traumatic occlusion in the middle cerebral artery (MCA) is a rare cause of cerebral infarction. This article reports a 37-year-old female who initially presented with neck and left shoulder pain after a traffic accident, twelve hours later, left hemiplegia has developed progressively. An atherosclerotic plaque and a dissection in her right internal carotid artery (ICA) were noted from Doppler examination. Right MCA occlusion caused by carotid artery injury was highly suspected. Injury information,
review of diagnosis and pathogenesis are discussed here.

**CASE REPORT**

A 37-year-old healthy female riding a motorcycle at slow speed fell over on her left side due to sudden loss of driving control (no impact with another vehicle was noted), and her left shoulder heavily struck the road. She had worn a helmet and denied any direct collision on her head or neck. There was no initial loss of consciousness, only a 2x2cm abrasion injury over her left shoulder. She could even walk to the roadside and wait for an ambulance. At the emergency department, Glasgow Coma Score (GCS) of 15 was noticed. She just complained of left shoulder and right neck pain initially. No neurological defect and muscle weakness were observed. X-ray image showed a fracture over the greater tubercle of left humerus (Figure 1). However, 12 hours after the accident, sudden onset of consciousness change with left limbs weakness developed. Emergent brain-computed tomography (CT) image showed no significant lesion. Unfortunately, left limbs weakness progressively developed and she even briefly lost consciousness. An infarction of right MCA territory was noted on brain magnetic resonance imaging (MRI). The MRA definitely showed right M1 segment of MCA occlusion and irregular surface in right internal carotid arterial wall (Figure 2). A CT scan was also performed after 24 hours and revealed infarction involving right MCA territory with severe brain edema (Figure 3). A Doppler examination of the extracranial carotid system revealed a dissection above bifurcation (Figure 4), atherosclerotic plaque and reduced blood flow velocities in right ICA. These findings of atherosclerosis were also compatible to magnetic resonance angiography (MRA) that showed irregular arterial wall. However, she denied the established risk factors of atherosclerosis, such as diabetes, hypertension, smoking and hypertriglyceridemia. Most of the blood laboratory examinations for blood coagulation were tested, but no abnormal results were found. Transthoracic echocardiogram revealed no circulating embolic particles or intracardiac shunt. Antiplatelet agent (aspirin) was used in the right MCA infarction. Emergent decompressive craniectomy was done for severe brain edema. No surgical therapy was done for ICA dissection. After controlling the increased intracranial pressure, she was transferred to the rehabilitation department after three weeks.

During rehabilitation, the major problems found were left limbs hemiplegia, impaired transfer ability and sitting balance, however, no constructional apraxia and hemineglect were observed. The purpose of rehabilitation training is to prevent complications arising from infarction, such as joint contracture, muscle wasting, and decreased fitness. Furthermore the training can help improve activities of daily living (ADL) and quality of life. After 6 months of rehabilitation, she could ambulate with a quadricane and ADL increased.

![Figure 1. Left humeral head fracture (arrows).](image1)

![Figure 2. MRA showing an infarction in M1 segment of right MCA (white arrow). ICA: internal carotid artery with irregular wall (black arrow).](image2)
DISCUSSION

Several hypotheses were recently proposed concerning the mechanism of traumatic cerebral infarction: (1) direct blunt neck injury can cause ICA dissection and atheromatous plaque rupture;\(^1\)\(^2\)\(^7\) (2) excessive neck rotation, bending, and flexion/extension can cause carotid artery traction injury resulting in intimal damage and developing a thrombosis within the carotid artery;\(^8\)\(^9\) and (3) other mechanisms of traumatic cerebral infarction such as cerebral artery vasospasm, spontaneous stenosis, or emboli from either skull and facial fracture may be involved.\(^{10-14}\)

ICA dissection is a more common cause of cerebral ischemia following a direct blunt neck injury. Previously, the reported incidence of blunt carotid artery injury is 0.08–0.4 percent; for patients who were more severely injured and at risk for occult carotid artery injury, the incidence increased to 3.2 percent.\(^{3,4}\)

Occlusion of the MCA following neck stretch is a rare cause of cerebral infarction; the mechanism could be due to rapid deceleration with resultant hyperextension and rotation of neck causing the artery traction injury.\(^{8,9}\)

We highly suspected the right ICA dissection of this reported case was caused by a strong stretching on the right neck during her left shoulder hitting the road with such force that could even cause fracture of the left humerus. Although the patient in this report denied direct injury to the head or neck, excessive stretching of the neck could not be excluded as an initiating pathogenic factor. Some victims usually have a minor neck injury history but they have forgotten about it.\(^{1,2}\) The MCA occlusion may have similar pathology as noted in acute rotational injury of the vertebral artery at the C1-C2 region, leading to intramural dissection and thrombosis.\(^8\)

Any motion of the brain may lead to disruption of the arteries between the mobile extra-cerebral segment and the fixed intra-cerebral portion. This may lead to mechanical disruption of distal blood supply or intimal trauma and subsequent thrombosis or spasm.\(^{5,6}\) Atherosclerosis is also a relative risk due to the atherosclerotic plaques making the intima more fragile to injury. Thus, any excessive neck motion could cause the ICA injury, especially in vessel with atherosclerotic changes. Although the carotid atherosclerosis was found in this case, many victims had no apparent risk factors. In this case, genetic factors might also have a role in the pathophysiology of ICA dissection, mainly as part of a multifactorial predisposition. Brandt et al have postulated that patients with ICA dissection can have a constitutional and genetic weakness of vessel wall and that factors such as minor trauma can act as triggers.\(^{15}\)

Patients who have delayed neurological manifesta-
tions should be considered as the pathogenesis of vasospasm following a head injury. However, vasospasm mostly starts around day 2 and reaches maximum around day 4,[14] besides, vasospasm is significantly more frequent after traumatic subarachnoid hemorrhage (SAH). [9,17] In this case, neurological deficit developed within 12 hours after the injury and no hemorrhage was found in the first CT image, moreover, the Doppler examination showed the exact location of the dissection on right ICA. Thus, the relative delay of symptoms in this case could be explained on the basis of thrombus generation rather than vasospasm.

Diagnosis of carotid artery injury following a neck stretch is difficult, owing to the remarkable absence of visible trauma to the neck and the delayed clinical symptoms.[16] Because traumatic ICA injury was infrequent but has high morbid-mortality, early diagnosis is very important. The diagnostic tools recently used in carotid artery dissection are angiography, Doppler examination, computed tomography angiography, and MRA.[6,9,10,13,14] Although cerebral angiography remains the gold standard for diagnosing the ICA dissection,[4,12] angiography carries a recognized mortality and morbidity. Therefore, the noninvasive examinations should be considered firstly when the physicians just initially suspect an ICA dissection after a head or neck injury. The role of Doppler examination in dissection of the carotid artery seems to be a useful screening test before performing angiography.[17]

The Doppler examination is a simple non-invasive test, carrying little risk and is a reliable method of screening patients considering to have carotid disease.[17] Some reports have shown that combining ultrasound with transcranial Doppler examination increases sensitivity to 95% in traumatic ICA dissection.[17,18] However, the limitations of Doppler scanning are that it does not detect subtle dissections or adequately image the carotid vasculature at the skull base and above.[19] Further alternative examination should be arranged, such as the MRA. The use of MRA has value in the screening of the head and neck for patients who have suspected carotid artery injury or intracranial artery occlusion, and can accurately diagnose both intracranial and extracranial carotid injuries,[4,12] and provide sensitivity from 80% to 95% in detecting traumatic ICA dissection.[18]

The potential predisposing factor in these cases is interesting, due to the incidence of ICA dissection following neck injury is only in a small range, not all of the cases. Some underlying vascular risk factors may exist in the case with ICA dissection, such as weakness of the vessel wall and vessel with atherosclerotic changes. In our case, atherosclerotic plaque was found in ICA, it making the intima more fragile to injury. Taking into consideration, we suspect atherosclerosis to be related to her arterial damage.

Carotid injuries could be treated with medical or surgical therapies. Medical therapies include observation, anticoagulation, and antiplatelet treatments. Antiplatelet regiments include various doses of aspirin, while anticoagulation includes both heparin and warfarin. Surgical therapies include thrombectomy, arterial ligation, carotid artery stenting, revascularization and radiologic embolization. The strategy and time for such therapeutic trials have been discussed by Beletsky et al.[20]

To compare the present case of patients with ischemic stroke, the outcome of rehabilitation training, like physical performance, functional ability and quality of life, was similar. However, the improvement of communication and neglect can not be concluded in present case, due to the present case presented no aphasia and hemineglect initially.

In conclusion, excessive neck stretching probably injured the ICA, especially in patients with atherosclerosis, resulting in the subsequent cerebral artery occlusion by a thrombus. This case report also reminds physicians to carefully evaluate the carotid artery, particularly in cases presenting a history of neck stretch. Early diagnosis and treatment are necessary for improving the prognosis of this disease entity for the avoidance of thrombosis and embolism. The Doppler examination and MRA are highly sensitive screening methods to detect clinically suspected ICA dissection, and are suggested to be arranged before performing angiography.

REFERENCES


無明顯頸部挫傷之創傷性內頸動脈剝離致腦梗塞：病例報告

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頸部扭傷造成頸動脈剝離之案例十分罕見，它可能導致遠端血管堵塞，引起腦梗塞及神經功能障礙。本篇報告一位 37 歲女性病人，因車禍後造成左肩及頸部疼痛，當時意識清楚，送院後發現左側肩部輕微擦傷，影像學檢查發現左側肱骨頭骨折，其他理學與神經學檢查均無異常。然而在十二小時後，個案意識狀況突然改變，同時出現左側肢體無力之情形，當時腦部電腦斷層影像沒有發現異樣。受傷二十四小時後，個案再度昏迷，磁振造影檢查顯示右中腦動脈區域有訊號增強之病變(high density in diffusion-weighted imaging)，磁振血管攝影則顯示右中腦動脈堵塞之情形。頸部超音波檢查發現右內頸動脈血流變慢及粥狀硬化斑(atherosclerotic plaque)，並於右內頸動脈近端處出現假腔。經六個月復健後，個案雖然可以持杖行走，但左手肌力仍然不足，部分日常生活需他人協助。本病例報告提醒臨床醫師，造成意外性腦梗塞的原因並非只有頭部挫傷，過度頸部伸展也能導致頸動脈剝離，特別是個案本身患有頸動脈粥狀硬化。因此早期使用頸部超音波及磁振血管攝影之檢查，可提高正確之診斷率。（台灣復健醫誌 2011；39(4)：233 - 238）

關鍵詞：頸部挫傷(neck injury)，頸動脈剝離(carotid artery dissection)，粥狀動脈硬化(atherosclerosis)，腦梗塞(cerebral infarction)