Low velocity traumatic dissection of the internal carotid artery presenting as a stroke after a slash injury

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ABSTRACT
Low velocity traumatic dissection of the carotid artery is an infrequent but serious complication of blunt cranio-cervical injury. A 25-year-old man was hit by a blunt sickle over his chest and left side of his neck, and sustained open wounds over zone II of the neck. At that time, he had no neurological deficits and the wounds were sutured primarily. On the sixth day, while he was undergoing a change of dressing for the wounds, he suddenly became unconscious, and on recovery hours later, he was aphasic. Computed tomography showed a left posterior fronto-temporal ischaemic infarct in the territory of the middle cerebral artery. Colour Doppler ultrasonography showed an intimal tear and thrombus in the left common carotid artery. We discuss the possible mechanism of neurological deficits and also emphasise that in any patient with blunt injury to the neck, new neurological deficits should prompt us to suspect carotid artery injuries.

Keywords: carotid artery dissection, embolic stroke, neck injury complication, neurological deficit, stroke

INTRODUCTION
Traumatic dissection of the carotid artery is an infrequent but serious complication of blunt cranio-cervical injury.[1] The effects of blunt trauma are very tissue specific. The degree of injury is intimately related to the elasticity and cohesiveness of the affected organ or anatomical region, which determines its ability to withstand stretching or compression. Using a more liberal criteria that include routine catheter angiography, some authors have found a 1.1% rate of carotid injuries.[2] Partial or complete vessel occlusion is reported to be the most common carotid artery injury following blunt trauma (33%).[2] These lesions (vessel narrowing and marginal irregularities) can be seen in traumatic stenosis, and can be due to intimal flaps or small haematoma of the vessel wall that may lead to stroke in 15% of patients with a mortality rate of up to 22%.[3-5] The present case emphasises that in a patient with neck injury, a new onset of neurological deficit should arouse the suspicion of carotid artery dissection and should be evaluated accordingly.

CASE REPORT
A 25-year-old man was hit by a blunt sickle over his chest and the left side of his neck by his wife (Fig. 1). At that time, he had no neurological deficit and the wounds were sutured primarily at a primary health centre. On the sixth day, while he was undergoing a change of dressing for the neck wounds, he suddenly became unconscious. When he regained consciousness six hours later, he was not able to speak and had facial asymmetry. He presented to our casualty department. On examination, he was conscious, and had motor aphasia with normal comprehension. There was right upper motor neuron type of facial palsy. The rest of his neurological examination was normal.

Fig. 1 Photograph shows multiple sutured lacerations over the anterior chest wall and left side of the neck (note one that is crossing the carotid artery).
Computed tomography (CT) showed an ischaemic infarction involving the left posterior frontal and part of the temporal lobe in the territory of the left middle cerebral artery (Fig. 2). With these clinical features and in view of his neck injury, the possibility of carotid artery injury and embolic stroke precipitated by trivial manipulation of the artery during dressing was considered. Further evaluation with colour Doppler ultrasonography (US) supported our suspicion and showed an intimal tear and thrombus in the left common carotid artery (Fig. 3). Magnetic resonance (MR) imaging and angiography could not be done as these facilities were unavailable. The patient was treated conservatively with anti-platelet agents and physiotherapy for six weeks and was doing well on follow-up.

**DISCUSSION**

The neck may be divided into three zones using anatomical landmarks (Table I). Each zone has a group of vital structures that can be injured and may determine the kind of trauma management required. Partial or complete vessel occlusion is reported to be the most common carotid artery injury in blunt trauma (33%). Vessel narrowing and marginal irregularities can be seen in traumatic stenosis due to intimal flaps or small haematoma of the vessel wall, as in the present case. In carotid dissection, the neurological damage is mainly attributable to thromboembolism. There is controversy regarding the need for diagnostic screening and management. Diagnosis is with carotid colour Doppler US, CT angiography of the neck and conventional angiography. Duplex US (DUS) is an excellent diagnostic modality with cost-saving, patient-friendly characteristics and a low rate of morbidity. It should be instituted as the primary diagnostic procedure of choice for penetrating neck trauma.

Colour Doppler US is as accurate as angiography in screening clinically-stable patients with zone II or III injuries and no signs of active bleeding, as in the present case. Furthermore, management decisions can be based on the specific type of injury, pathological condition of the arterial wall, and haemodynamic factors identified by DUS.
Clinically-occult arterial injuries can be followed-up by repeat DUS to define the natural history of these injuries. However, the usefulness of colour Doppler US is limited in patients with subcutaneous air or large haematomas. In addition, it is operator-dependent and may be more time-consuming than desirable in the acute care setting. Nowadays, the diagnosis of carotid artery dissection is essentially established with cervical MR imaging and MR angiography, but it may be limited for depiction of concurrent osseous injuries. Overall, blunt trauma to the neck has a low rate of associated vascular injuries (0.7%), and this has justified the tendency toward a more conservative approach and the use of noninvasive techniques in these patients.

Current treatment includes supportive therapy and antiplatelet agent, either alone or with anticoagulation. Anticoagulants or antiplatelets may prevent arterial thrombosis in extracranial internal carotid artery dissection, but these benefits may be offset by increased bleeding. Dissections of the carotid artery have a benign long-term prognosis with low stroke rates that are not related to the persistence of severe carotid stenosis or occlusion. Surgical or endovascular therapy of stenosis or occlusion related to dissection should only be taken into consideration in the very rare patients with stroke recurrence in spite of an optimal medical treatment. Cervical aneurysms caused by carotid artery dissection have an excellent long-term outcome with a low stroke risk, and no vessel rupture has been reported. Thus, surgical or endovascular therapy should be restricted to the very rare cases developing ischaemic symptoms in the vascular territory supplied by the dissected aneurysm in spite of antithrombotic therapy. Our patient sustained a carotid artery injury by a blunt sickle and trivial manipulation during dressing resulted in dislodgement of the thrombus and manifested as stroke. In any patient of blunt injury of the neck, neurological deficits should prompt us to suspect carotid artery injuries.

### REFERENCES


### Table I. Zones of the neck using anatomical landmarks.

<table>
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<tr>
<th>Zone</th>
<th>Landmarks</th>
<th>Contents</th>
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<tr>
<td>I</td>
<td>Horizontal area between the clavicle/suprasternal notch and the cricoid cartilage encompassing the thoracic outlet structures</td>
<td>proximal common carotid artery, vertebral and subclavian arteries, trachea, oesophageal, thoric duct, and thymus</td>
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<tr>
<td>II</td>
<td>Area between the cricoid cartilage and the angle of the mandible</td>
<td>internal and external carotid arteries, jugular veins, pharynx, larynx, oesophageal, recurrent laryngeal nerve, trachea, thyroid and parathyroids, spinal cord</td>
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<tr>
<td>III</td>
<td>Area between the mandible and the base of the skull</td>
<td>distal extracranial carotid and vertebral arteries, uppermost segments of the jugular vein</td>
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