# An interesting case of headache and dysphagia

## Introduction

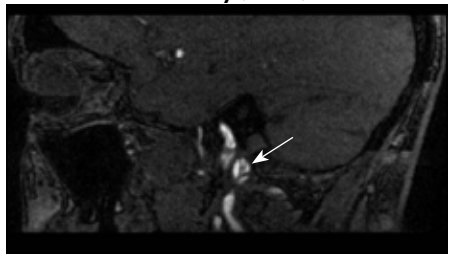
A 45-year-old woman presented with headache, followed by dysphagia and dysphonia. Examination revealed IX, X and XII cranial nerve weakness. Magnetic resonance angiography of neck vessels confirmed a left internal carotid artery dissection at the skull base, with haematoma in the aneurysmal left internal carotid artery. This dilated area of the internal carotid artery was thought to be pressing on the lower cranial nerves as they exited the base of the skull. Symptoms improved with nasogastric feeding, speech therapy and antiplatelet agents. At follow up she had complete resolution of dysphagia, but hypophonia persisted. Magnetic resonance angiography revealed improvement in the appearances of the aneurysmal left internal carotid artery. This is an uncommon presentation of carotid artery dissection and management is debatable.

## Discussion

Lower cranial nerve palsy associated with internal carotid artery dissection is uncommon but has been reported. The mechanism could be through lower cranial nerve nuclei involvement in the brainstem (although other neuronal tracts should also be affected) or pressure effects as the nerves exit the skull base. Another plausible mechanism could be local ischaemia from dissection or compression causing cranial nerve infarction or injury.

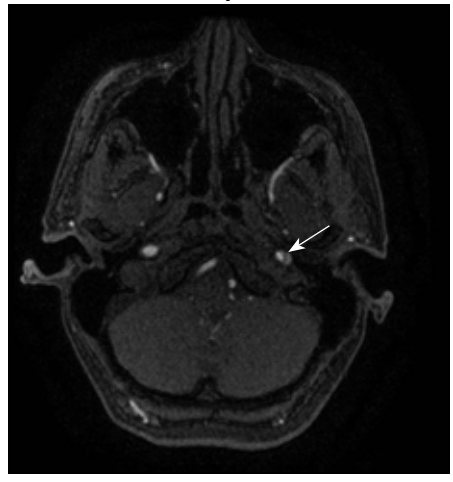
In this patient, the neurological deficit was initially thought to be caused by involvement of the brainstem nuclei. Cranial nerves IX and X originate from the solitary and ambiguous nuclei. The hypoglossal nucleus lies caudally to these. For all three of these nuclei to be affected simultaneously the lesion has to be large and would involve further brainstem nerves with extensive signs (Caplan et al. 2012). Hence, a lesion along the pathway of the lower cranial nerves was considered. Magnetic resonance angiography confirmed left internal carotid artery dissection with haematoma (*Figures 1–3*) which was thought to exert pressure on the affected lower cranial nerves as they exited the skull base.

***Figure 1. Contrast-enhanced magnetic resonance angiogram (sagittal section) showing dissection of left internal carotid artery (arrow).***



This patient did not have evidence of Horner's syndrome on repeated examinations, probably as a result of sparing of the sympathetic fibres running along the internal carotid artery. Management of carotid

***Figure 2. Contrast-enhanced magnetic resonance angiogram (axial section) showing dissection of left internal carotid artery (arrow).***



### Case Report

A 45-year-old, normally fit and well woman, was admitted through the medical intake. She gave a 6-day history of sore throat and gradual onset of left-sided headache. She felt nauseous with episodes of regurgitation of food, and felt that her tongue was not moving properly. She saw her GP pre-admission who suspected a neuralgic migraine or viral illness and prescribed aciclovir. On probing, she admitted to slurred speech and worsening dysphagia to solids and liquids to the point that she was now drooling. She reported no other neurological symptoms. She had recently joined a gym; otherwise there was no history of head or neck trauma.

On examination she had normal visual fields and eye movements. Facial sensation was normal with no facial asymmetry and no hearing loss. Oral examination revealed decreased palatal movement to the left with absent gag reflex and tongue deviation on protrusion to the left. She had nasal speech and failed a swallow assessment. There were no features of Horner's syndrome and optic discs were normal on fundoscopy. She had normal peripheral nervous system and cerebellar examination. The patient did not have Marfanoid morphology. The findings suggested an IX, X and XII cranial nerve palsy.

Initially a brainstem lesion was suspected. Computed tomography of the brain was normal. Magnetic resonance of the brain and magnetic resonance angiography showed normal intracranial appearances, but revealed narrowing of the left internal carotid artery at the first spinal vertebral level displaying arterial dissection with haematoma in the arterial wall (*Figures 1–3*).

The patient was given a nasogastric tube to achieve suitable nutrition and given aspirin 300 mg for 2 weeks followed by clopidogrel 75 mg for 6 months. She received speech and language therapy. Her swallow began to recover and she was discharged home once she was able to tolerate a soft diet. She had ongoing speech and swallow therapy in the community. A magnetic resonance of the brain and magnetic resonance angiography 8 weeks later showed no significant intracranial abnormalities but slight improvement in the left internal carotid artery haematoma. She had completely recovered her swallowing but still had some dysarthria and hypophonia. On close questioning on follow up, the patient mentioned that she had noticed disturbance of taste after regaining swallow. Examination confirmed persistent lower motor neuron weakness of cranial nerves X and XII. She did not develop further ischaemic events.

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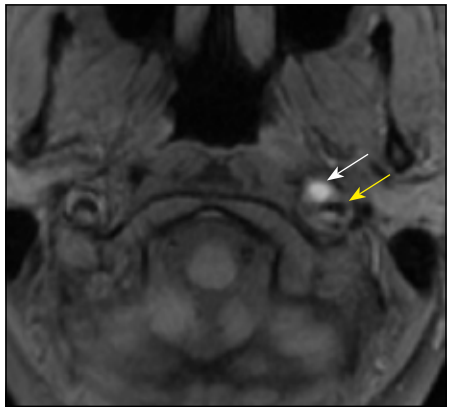

***Figure 3. Gradient echo magnetic resonance image (axial section) showing haematoma in the wall of dissected left internal carotid artery (white arrow). Yellow arrow shows narrowed lumen of the dissected artery.***

artery dissection focuses on preventing potential embolic ischaemic cerebral events from the dissection site. A Cochrane review has suggested that two thirds of embolic ischaemic strokes from a dissected internal carotid artery occur within a week, recommending early intervention. There is benefit in giving antiplatelet or anticoagulation therapy, but no clear advantage of one over the other. Surgical intervention increases death rates compared to antiplatelet therapy (Lyrer and Engelter, 2010). BJHM

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Caplan LR, Gonzalez RG, Buonanno FS (2012) Case records of the Massachusetts General Hospital. Case 18-2012. A 35-year-old man with neck pain, hoarseness, and dysphagia. *N Engl J Med* **366**: 2306–13

Lyrer P, Engelter S (2010) Antithrombotic drugs for carotid artery dissection. *Cochrane Database Syst Rev* (10): CD000255

## LEARNING POINTS

- In patients presenting with lower cranial nerve deficit and neck pain, carotid artery dissection should be considered.
- This case illustrates the importance of neuro-anatomical knowledge in making a diagnosis.
- Management of these patients is unclear; current guidance suggests use of either antiplatelet or anticoagulation therapy in the acute setting.



# A case of paraplegia post treatment for acute coronary syndrome

A 59-year-old woman presented to accident and emergency complaining of a 1-day history of central chest tightness. She had a normal cardiovascular examination. Her electrocardiogram revealed normal sinus rhythm and did not show any dynamic changes (*Figure 1*). Blood troponin levels were initially 28 ng/litre, but rose to 98 ng/litre 6 hours later (normal value <14 ng/litre). Antiplatelet agents (aspirin, clopidogrel and fondaparinux) were administered for a provisional diagnosis of acute coronary syndrome.

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Two days later she developed acute thoracic and lumbar back pain with loss of power and sensation in both lower limbs. She had a flaccid tone, power of 1/5 bilaterally and areflexia. Magnetic resonance imaging of the spinal cord revealed extensive spinal haemorrhage throughout the thoracic and lumbar spine (T11–L2) (*Figure 2*).

This case highlights the importance of correctly identifying patients with acute coronary syndrome and balancing the risks of treatment *vs* the risk of bleeding, to reduce major bleeding episodes. BJHM

***Figure 1. Electrocardiogram on admission.***

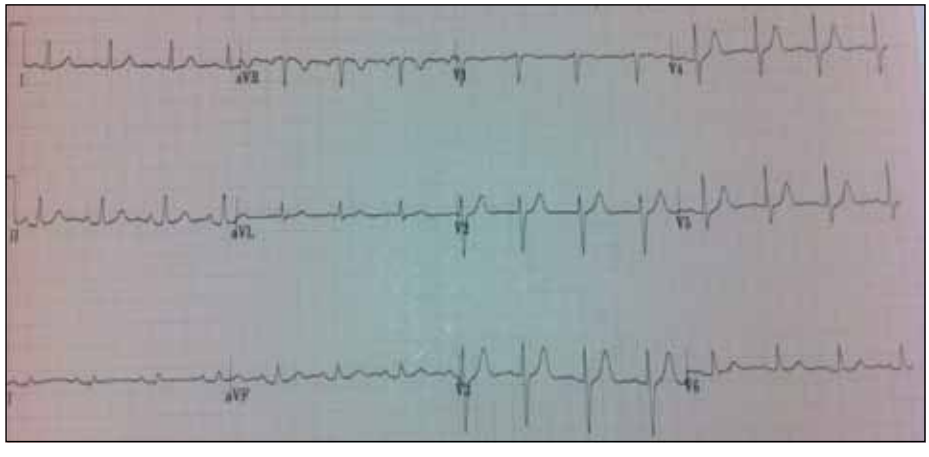

***Figure 2. Magnetic resonance imaging of the spine, showing spinal haemorrhage throughout the thoracic and lumbar spine T11–L2.***