Case Report

An unusual presentation of dysarthria in a young patient, a stroke mimic

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Abstract

Internal carotid artery dissection commonly affects younger patients. We present a case of a previously fit and well 43-year-old gentleman who presented with a sudden onset of slurring of speech, with right-sided tongue deviation and fasciculation on examination. Signs and symptoms began following participation in a home workout class. Magnetic resonance angiography revealed right-sided extracrainal internal carotid artery dissection leading to right-sided unilateral twelfth cranial nerve palsy.

Keywords

hypoglossal nerve palsy, palsy, stroke, Acute Medicine

Key Teaching Points

- To consider internal carotid artery dissection in patients with isolated twelfth cranial nerve palsy and to highlight the importance of full neurological examination in young potential stroke patients
- Internal carotid artery dissection can cause lower motor neuron as well as upper motor neuron pathology and signs, depending on its size and location
- Not all 'FAST positive' patients are stroke, some are stroke 'mimics'

Case

We present a case of a previously fit and well 43-year-old gentleman who was admitted with sudden onset slurring of speech and right-sided tongue deviation. It started during participation in a home workout class, where he developed sudden onset dysarthria. This persisted for several hours before he sent his General Practitioner (GP) an electronic letter (eConsult) reporting a "strange feeling of swelling" of the right side of his tongue with difficulty sounding out words, which came on quickly. The GP arranged for an immediate assessment and identified deviation of the tongue to the right side, which was an isolated neurological finding. Being FAST positive[1] with speech abnormality an acute admission of the patient into hospital was arranged querying a stroke and aspirin 300 mg as a stat dose was given. He was then admitted via the acute medical unit and quickly directed onto the hyperacute stroke unit where he was reviewed by the stroke team.

This gentleman had no prior history of: headache, trauma, visual or other neurological disturbance. There was no history of loss of consciousness or collapse. He did not suffer with any medical conditions and had no significant family history of note. This gentleman described himself as an ex-smoker who enjoyed a few glasses of wine on weekends. He did not use any regular medications and did not partake in the use of recreational drugs. Formal neurological examination of his limbs; tone, power, co-ordination, reflexes were all unremarkable and cranial nerve examination I to XI was also unremarkable. He did not demonstrate expressive or receptive dysphasia, there was only isolated dysphonia.

Further detailed examination of cranial nerve XII revealed a right-sided tongue deviation with fasciculation noted on the right, lateral aspect. This suggested lower motor neuron pathology (Figure 1). Examination of his cardiovascular system demonstrated a regular normal pulse (both arms) with no murmur, while his carotid pulsations were initially felt to be unremarkable with no audible bruit. Further general systemic examination did not reveal any other pathology.

Observations taken at the point of presentation to his GP surgery were as follows: blood pressure 123/79mmHg, pulse 70bpm, saturation 98% in air, a respiratory rate of 14 breaths per minute and temperature of 36.4°C. Following hospital admission an ECG was carried out which demonstrated normal sinus rhythm. Standard Stroke blood tests were requested, including full blood count, coagulation screen, urea and electrolytes, liver function tests, bone profile, thyroid function, lipids, inflammatory

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Table 1. Blood results on admission to hospital

Initial Results	Value	Hospital Normal Range
Sodium	145 mmol/L	(133 – 147)
Serum Potassium	3.9 mmol/L	(3.5 – 5.3)
Urea	3.4 mmol/L	(2.5 – 7.8)
Creatinine	83 umol/L	(60 – 110)
eGFR/1.73m*2	99 ml/min	(60 –)
CRP	1 mg/L	(0-5)
ESR	2 mm/hr	(0-12)
Coagulation Screen	Normal	
Subsequent Results:	**Value**	**Normal Range**
Cholesterol	4.2 mmol/L	(0.0 – 5.0)
Triglycerides	1.9 mmol/L	(-)
Gamma GT	34 u/L	(0 - 75)
Serum Anti-cardiolipin Level	Negative	-
ANA, ANCA screen	Negative	-
HbA1c	30 mmol/mol	(20 - 41)
SARS-CoV-2 RNA PRC	Not detected	

markers and a vasculitic screen including ANA and ANCA. All results were unremarkable (Table 1).

This gentleman had an urgent CT head scan which showed no acute abnormality. Given his clinical findings, a more detailed scan in the form of magnetic resonance angiography of the head and neck was requested (Figure 2). This showed extracranial dissection of the right internal carotid artery. This gentleman was then diagnosed with internal carotid artery dissection with resultant twelfth cranial nerve palsy. He was provided with aspirin for several months while under outpatient review.

Discussion

Dissection of the cervical neck vessels is a major cause for young adult patients to present to hospital with stroke.[2] The median age of presentation is mid-40s with the incidence slightly higher in males vs females.[2] A tear in the intimal layer allows blood to flow into the tunica media, propagating the tear along

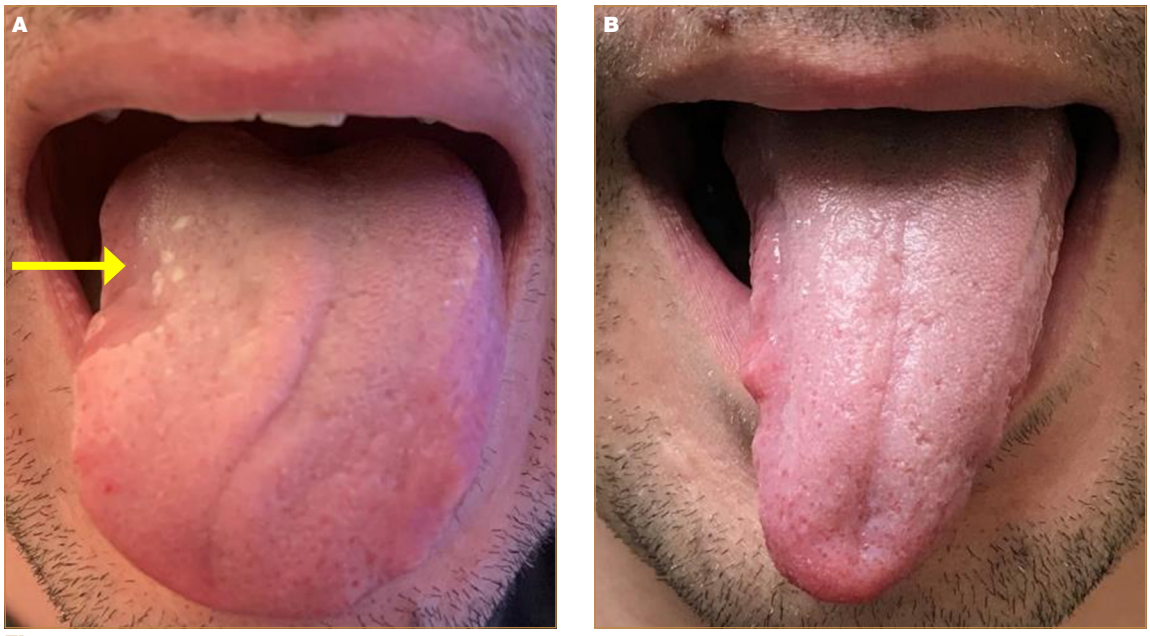

Figure 1. (A) 43-year-old gentleman with right-sided tongue deviation and muscle wasting (yellow arrow). (B) Extent of deviation at two months post internal carotid artery dissection. (informed consent was obtained from the individual on the photo)

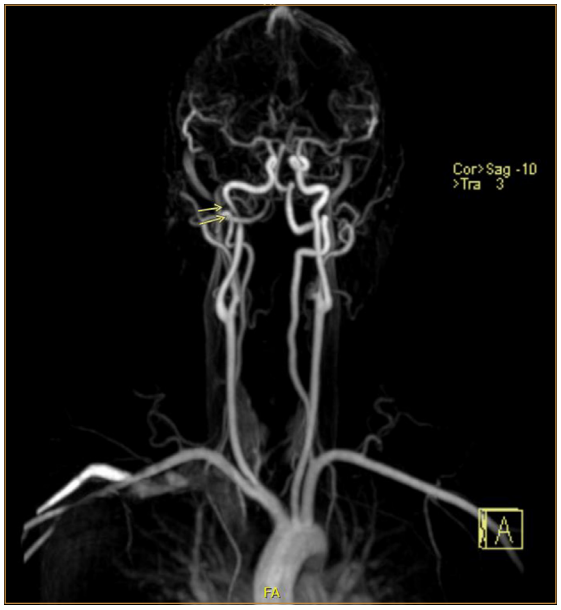

Figure 2. Magnetic resonance angiography showing narrowing of right internal carotid artery (arrow), characteristic of ICA dissection.

the length of the vessel creating a false lumen. The concern is that the resultant intramural haematoma formation will then lead to critical stenosis or occlusion of the vessel with thrombus formation at the site of dissection.[3] The subsequent reduction in perfusion pressure commonly results in a low flow stroke. Or a stroke can occur if thrombus breaks free from the damaged intima to migrate rostrally to occlude an end territory cerebral vessel, or vessels if thrombus breaks off as an embolic shower.

Carotid artery dissection is classically described as presenting with neck pain, headache and partial Horner's syndrome followed by retinal or cerebral ischaemia.[3-4] Isolated twelfth cranial nerve mononeuropathy is a rarer presentation of carotid artery dissection. We therefore wanted to present a case for educational purposes.

The hypoglossal nerve is responsible for the unilateral innervation of the intrinsic and extrinsic muscles of the tongue with activation of the paired nerves causing protrusion of the tongue. Unilateral damage therefore causes weakness and wasting to the ipsilateral muscles of the tongue causing deviation towards the side of damage. Internal carotid artery dissection has been implicated as causing between 10-15% of strokes in young people with 5% of cases presenting as isolated hypoglossal nerve palsies.[5] They usually occur spontaneously, however, history taking often reveals preceding subclinical trauma such as sneezing, coughing, heavy lifting or neck manipulation.

There are currently two mechanisms cited by which internal carotid artery dissection causes twelfth cranial nerve palsy. Firstly, that of compression in which the formation of the intramural haematoma results in pseudoaneurysmal formation which compresses the nerve as it travels along its extracranial portion, between the internal carotid artery and the internal jugular vein.[6] Secondly, that of ischaemia in which the nerve is compromised as a result of damage to the vessels that form the fine direct branches of the blood supply to the nerve, the vasa nervorum.[5,7] Both computed tomography angiography and magnetic resonance imaging are adequate tools to image cervical artery dissections, with current guidance suggesting that either should be utilised.[8]

CT angiography is useful for its low cost, ease of access and patient tolerability. However, drawbacks for this method include delivery of harmful ionising radiation as well as presenting diagnostic challenges depending on user experience.[9,10] MRI conversely allows direct imaging of the intramural thrombus as well as high sensitivity and specificity for detection of cerebral ischaemia following dissection.[10] In our case, this gentleman had been exposed to the radiation from his first CT head and there were no contraindications to MRI use. Magnetic resonance angiography was therefore our method of choice. The characteristic narrowing of the internal carotid artery lumen is clearly demonstrated in Figure 2.

Internal carotid artery dissection is commonly treated conservatively with either antiplatelet therapy or anticoagulation therapy to prevent thrombus formation at the site of dissection. Surgery in the form of endovascular repair is usually reserved for those who have recurrent ischaemia despite conservative therapy.[11] At present, there is no definitive evidence to suggest optimal treatment for internal carotid artery dissection in young patients without recurrent ischaemia. It is known that anatomical vessel abnormalities following dissection tend to stabilise or resolve completely between three to six months with symptoms improving during this period.[12] Given this, patients are commonly asked to abstain from strenuous activities during this time as was the case with our patient. Treatment with antiplatelet or anticoagulation therapy according to the large prospective randomised controlled CADISS trial[13] has been shown to be equally efficacious. It should be noted that direct oral anticoagulants were not included within this trial due to lack of availability at recruitment.

The choice of treatment is therefore currently based on the clinical experience of the treating clinician, taking into account co-morbid conditions and patient preferences. Following discussion with the patient, the decision was made to take simple aspirin 75 mg as secondary prevention from further complication, though equally clopidogrel or anticoagulation could

have been offered. On outpatient follow-up at six months, the patient demonstrated no dysarthria and a complete recovery of his initial unilateral muscle wasting. He was discharged from further follow up with suitable reassurance. He was fortunate as some long-term complications of internal carotid artery dissection can be: ongoing neurological symptoms, retinal ischaemia or recurrent dissection – this is generally thought to be highest within the first two weeks after presentation although risk appears to be low.[14, 15]

Internal carotid artery dissection resulting in twelfth cranial nerve palsy is an uncommon presentation, of a common, though potentially severe and yet treatable condition in young patients. The risk of large stroke has been shown to be greatest within the first two weeks from symptom onset.[14] This case therefore highlights the importance of prompt diagnosis, investigation and treatment of young patients with presumed stroke, as well as the importance of full neurological examination in young potential stroke patients.

Declaration of competing interest

Nothing to declare

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