Unusual cause of neurological symptoms in a young man

Katharine E Elliott • Noel N Thin • Janice C Tsui • Meryl Davis

Department of Vascular Surgery, Royal Free Hospital, London, UK

Correspondence to: Katharine E Elliott. Email: kemelliott@doctors.org.uk

DECLARATIONS

Competing interests
None declared

Funding
None

Ethical approval
Written informed consent to publication has been obtained from the patient

Guarantor
MD

Contributorship
All authors contributed equally

Acknowledgements
This case report was presented at the International Trauma Conference 2009 in Manchester

Reviewer
Michael Dialynas

TIAs secondary to carotid stenoses are unusual in young patients with no obvious cardiovascular risk factors, and may be missed.

Case history

A 30-year-old, left-handed man presented to the emergency department on Boxing Day with a 20-min history of slurred speech and weakness affecting his right arm. This was preceded by a generalized headache earlier that day. Over the past three months he had experienced neurological symptoms on three other occasions. The first episode was bilateral distortion of vision, lasting 10 s. His GP suggested he see an optician, who diagnosed worsening myopia, and a neurologist, who discharged him with the reassurance that his symptoms were not based on organic pathology. The second episode occurred one week prior to admission and consisted of sudden right arm numbness with difficulty in movement of the arm, slurred speech and visual distortion, lasting 5 min.

A third episode occurred on Christmas Eve, whereby the patient noticed numbness of the left side of his head and slurred speech, lasting 20 min. His wife, a witness to this event, described her husband as inventing words. The following day, Christmas Day, a fourth episode occurred, where he experienced numbness of the right side of the face, photophobia and difficulty opening the right eye for which he presented to his local hospital's emergency department. He was once again discharged home without diagnosis.

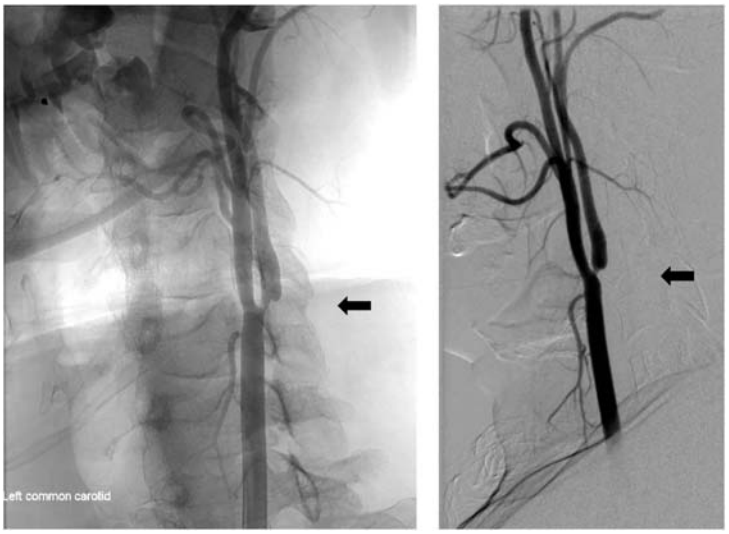
The patient was otherwise fit and well. His medical history included one admission to hospital for concussion, aged 19 years, following a road traffic accident. He did not take any regular medications and was allergic to penicillin. There was no family history of cerebrovascular disease, ischaemic heart disease or peripheral arterial disease. His father was a type 2 diabetic. The patient had never smoked, did not drink alcohol, was a vegetarian and exercised regularly at the gym.

On Boxing Day he presented to the emergency department. He was apyrexial with a regular heart rate of 82 beats per min, a blood pressure of 110/70 mmHg and a Glasgow Coma Score of 15/15. A full neurological examination revealed slurring of speech and resolving right-sided upper limb weakness. Electrocardiogram revealed normal sinus rhythm; blood tests including full blood count, urea and electrolytes, auto-antibodies, erythrocyte sedimentation rate and lipid profile were normal. The patient was admitted into hospital where he had a CT scan of his head which showed no abnormalities.

He then underwent carotid duplex and magnetic resonance angiography and on initial review of the abnormalities of the left internal carotid artery shown, a diagnosis of carotid dissection was suspected. He was commenced on low molecular weight heparin and warfarin therapy and discharged once his INR had reached therapeutic levels. He was referred for further investigations at the National Neurological Institute at Queen Square where he had a cerebral artery angiogram. Digital subtraction angiography showed an isolated tight eccentric stenosis of the left internal carotid artery of greater than 90% at its origin, as opposed to the initial diagnosis of dissection (Figure 1). The patient's warfarin therapy was stopped and he was referred for urgent carotid endarterectomy to reduce his risk of further embolic events.

A standard left carotid endarterectomy with patch closure was performed under general anaesthesia. Postoperatively, the patient had an uncomplicated recovery.

Figure 1
Digital subtraction angiography showing tight stenosis of the internal carotid artery near its origin



The histology of the operative specimen confirmed that it was an eccentrically located intimal fibrous plaque which disrupted the internal elastic lamina. The plaque's composition featured predominantly fibroblasts, smooth muscle cells with inflammatory cells including lymphocytes and histiocytes. He was discharged from hospital on the second postoperative day on clopidogrel 75 mg od, simvastatin 40 mg od and analgesia for postoperative wound pain. He has remained asymptomatic since his operation. Further assessments of his cardiovascular risks including upper and lower limb duplex assessments, homocysteine levels and fasting lipoprotein profile, i.e. apolipoprotein (Apo) A, Apo B, Apo E, lipoprotein(a) were all within the reference range.

Discussion

Our case highlights the difficulty in managing a patient with an insidious history of non-specific neurological symptoms. Initially his vague spontaneously resolving symptoms were quickly dismissed as his age, and the absence of significant atherosclerotic risk factors, made the diagnosis of cerebrovascular ischaemia unlikely. However, his subsequent development of severe left hemispheric symptoms, lead to inpatient investigation of both non-traumatic and traumatic differential diagnoses of the hemispherical neurological symptoms.

Non-traumatic differential diagnoses that may have caused hemispherical symptoms include: intracranial bleeding, cerebral artery aneurysm, vasculitis or premature carotid atherosclerosis (PCA) affecting the left carotid artery. Intracranial bleeding and cerebral aneurysms were excluded by a CT scan and our patient's inflammatory markers and full vasculitic screen were normal.

Premature carotid atherosclerosis (PCA) is a virulent condition where premature development of atherosclerosis and subsequent carotid occlusive disease occur in young patients. Although uncommon, it accounts for 20–33% of cerebral infarctions in patients aged 15–49 years.[1] PCA is associated with smoking, hyperlipidaemia, hypertension, diabetes and coronary disease.[2] Although our patient is the right age for PCA, he is not diabetic, has no risk factors for atherosclerotic disease and is a non-smoking vegetarian with a normal body mass index, blood glucose and lipid/lipoprotein profile, making the diagnosis of PCA unlikely. In addition, both lower and upper limb arterial Doppler scans were normal.

In a young patient with a possible history of trauma, both intimal dissection and arterial thrombosis would be considered as potential diagnoses, as they account for the majority of traumatic carotid injury sequelae (62% and 20%, respectively).[3] In fact intimal dissection was the initial working diagnosis as suggested by the MR angiogram. However, cerebral angiography demonstrated focal stenosis of the left internal carotid artery consistent with atheromatous plaque disease. This posed the question of previous neck trauma which the patient described as minor, recalling being admitted to hospital for observation for a slight head and possible whiplash injury. We suspect that this traumatic episode, although 12 years prior to presentation, was the origin of a carotid artery injury which progressed to the arterial lesion detected.

Carotid artery injuries caused by blunt trauma represent up to 1.03% of all admitted trauma cases.[4] Some series have shown that permanent neurological deficits occur in up to 37% and mortality in 40% of these patients.[5] The initial injury may be caused by only minor head and neck trauma and often physical evidence of cervical

injury is absent.[5] Authors have suggested that carotid injury can be caused when seat belt shoulder straps are worn during road traffic collisions.[6] Forces during the collision hyper-extend the neck and may stretch the carotid artery over the upper C1–3 vertebrae, leading to an intimal disruption of the vessel.[7] The described mechanism of injury could be consistent with our patient's history of a road traffic collision while wearing a seatbelt.

As our patient did not present at the time of injury with neurological symptoms, no further investigations were warranted at that time. However, as evidence suggests that delayed diagnosis and treatment has a strong correlation with adverse prognosis,[8] we suggest that if blunt carotid injury is suspected after trauma, the goals of treatment are to prevent thrombosis, propagation of clot and intracranial embolization from the injured artery.[9]

We hypothesize that the pathogenesis in our case was traumatic injury to the carotid artery causing tunica intima disruption providing a focal thrombogenic area leading to accelerated atherosclerotic fibrous plaque formation. This consequently caused the significant eccentric narrowing of his internal carotid artery and emboli which manifest as cerebrovascular symptoms 12 years later.

Conclusions

This case demonstrates the difficulty in the diagnosis of a young patient with a focal carotid artery stenosis causing embolic complication. The most striking feature about this case is the absence of atherosclerotic risk factors in a young non-smoker with significant and symptomatic internal carotid stenosis. Given his history, pathology other than atherosclerosis was considered. Even then the suspected mechanism of blunt carotid injury may still have been easy to dismiss especially with a lengthy asymptomatic period of 12 years. His unusual presentation of neurological symptoms led to thorough investigation and diagnosis of post-traumatic accelerated atherosclerotic plaque formation. It is clear that this presentation of post-traumatic carotid stenosis is rare but we, as clinicians, must remain vigilant especially when transient symptoms of cerebral ischaemia present in the young.

References

1 Valentine JR, Myers SI, Hagino RT, Clagett P. Late outcome of patients with premature carotid atherosclerosis after carotid endarterectomy. *Stroke* 1996;**27**:1502–6

2 Martin GH, Allen RC, Noel BL, *et al.* Carotid endarterectomy in patients less than 50 years old. *J Vasc Surg* 1997;**26**:447–55

3 Fabian TC, Patton JH, Croce MA, *et al.* Blunt carotid injury. Importance of early diagnosis and anticoagulant therapy. *Ann Surg* 1996;**223**:513–25

4 Miller PR, Fabian TC, Croce MA, *et al.* Prospective screening for blunt cerebrovascular injuries. Analysis of diagnostic modalities and outcomes. *Ann Surg* 2002;**236**:386–95

5 Singh RR, Barry MC, Ireland A, Bouchier Hayes D. Current diagnosis and management of blunt internal carotid artery injury. *Eur J Vasc Endovasc Surg* 2004;**27**:577–84

6 Harrahill M. Suspected blunt carotid injury in a 54-year-old woman after a motor vehicle crash: a brief review. *J Emerg Nurs* 2003;**29**:582–3

7 Arthurs ZM, Starnes BW. Blunt carotid and vertebral artery injuries. *Injury Int J Care Injured* 2008;**39**:1232–41

8 Crissey MM, Bernstein EF. Delayed presentation of carotid intimal tear following blunt craniocervical trauma. *Surgery* 1974;**75**:543–9

9 Risgaard O, Sugrue M, D'Amours S, *et al.* Blunt cerebrovascular injury: an evaluation from a major trauma centre. *ANZ J Surg* 2007;**77**:686–9