Unexpected outcome (positive or negative) including adverse drug reactions

Cerebral infarction following a carotid Doppler ultrasound: a chance association?

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Summary

The authors present a case report of a 64-year-old lady with multiple risk factors for atherosclerosis, who suffered an ischaemic stroke immediately following carotid Doppler ultrasound despite therapeutic warfarin and antiplatelet therapy. This event was only 16 days after the patient suffered a first ischaemic stroke during diagnostic coronary angiography following a syncopal episode accompanied with a raised troponin-T. The temporal relationship between the carotid Doppler ultrasound and second ischaemic stroke raised the question whether it was possible that the investigation had caused the stroke, a complication not previously recognised.

BACKGROUND

National Institute for Health and Clinical Excellence guidelines state those with suspected non-disabling stroke or transient ischaemic attack who are candidates for surgery should have imaging, usually carotid Doppler ultrasound, within 1 week of onset of symptoms.¹ We report a patient who suffered recurrent ischaemic strokes, one of which raises the question as to whether there could be at least a theoretical causal relationship between stroke and carotid Doppler ultrasound.

This case raises an interesting question for discussion and provides a useful focus for reflective practice with regard to the use of carotid sinus massage and carotid Doppler ultrasound and the (albeit extremely small) potential to do harm with these procedures. Documenting this case in the literature will allow for sensible discourse should it be repeated elsewhere.

CASE PRESENTATION

A 64-year-old lady with a background of hypertension and tablet-controlled diabetes presented to the Emergency Department after a syncopal episode. Examination revealed rapid atrial fibrillation (AF). She received carotid sinus massage, was rate controlled with digoxin, anticoagulated with enoxaparin and admitted for monitoring. Subsequent troponin-T was elevated at 0.032 ug/l.

Diagnostic coronary angiography was complicated by anterolateral ST elevation and development of a left hemiplegia. National Institutes of Health Stroke Scale (NIHSS) score was 28. Immediate CT-head scan was normal.

With ongoing ST elevation, thrombolysis was administered.

CT-scan 20 h post-thrombolysis confirmed infarction in the right basal ganglia. Aspirin (300 mg) and clopidogrel (75 mg) were commenced. She improved over the next 2 weeks to an NIHSS score of 2; remaining in AF, with impaired left ventricular function but no mural thrombus on echocardiogram.

INVESTIGATIONS

Inpatient anticoagulation with warfarin was commenced 15 days poststroke. With an international normalised ratio of 2.0 and antiplatelet therapy consisting of clopidogrel, having stopped aspirin the previous morning, she underwent carotid Doppler ultrasound at 11.14 hours, day 16 poststroke. This revealed 40%–59% (peak systolic velocity (PSV) of 138 cm/s) left internal carotid artery (ICA) stenosis and 88%–99% (PSV of 292 cm/s) right ICA stenosis with normal vertebral flow. No stenotic plaque or thrombus was visualised. The velocities were recorded and checked. During the procedure, the patient felt uncomfortable and immediately following the procedure she likened her 'strange feeling' to a state of alcohol intoxicity with an odd feeling in her head. Minutes later, she reported trying to stand but was unable to due to weakness in her legs. At 15.10 hours she called for the assistance of a doctor. Clinical examination revealed 0/5 power in her left upper limb, no sensory deficit and no other new neurological findings. NIHSS score was 7. CT head demonstrated a newly thrombosed right middle cerebral artery.

OUTCOME AND FOLLOW-UP

Subsequent CT carotid angiogram with 3D reconstruction demonstrated tortuous vessels without significant stenosis either side. Repeat carotid Doppler ultrasound was declined. There was no functional recovery of the arm by day 30 poststroke.

DISCUSSION

Stroke has been documented following carotid sinus massage (CSM), which can dislodge atheromatous emboli from the carotid artery.² With regard to the aetiology of stroke described in this report, the question was raised as to whether it was possible that the Doppler examination may have caused embolic infarction in our patient. CSM and carotid Doppler ultrasound both apply pressure to the

internal carotid arteries in a similar way and therefore the investigation may have the theoretical potential to cause stroke. However, subsequent CT angiogram weakens this hypothesis, unless a partially occluding thrombus had been completely dislodged even though thrombus was not seen on the initial Doppler. CT angiogram can be effective in determining plaque morphology,³ which is becoming of greater concern in determining potential vulnerability than degree of stenosis.⁴

It is reasonable to suggest that the temporality between this stroke and the carotid Doppler is purely coincidental. There was a 4-h interval between procedure and clinical diagnosis. Exact time of onset is not clear due to either an evolving infarct or poor correlation of patient symptoms with clinical findings.

This case highlights a potential mechanism for carotid Doppler ultrasound to cause stroke. This case provides no proof to the above but nonetheless offers a point from which to speculate. Given the frequency with which carotid Doppler ultrasound is performed in individuals at high risk of stroke, it is likely this temporal relationship has been seen before although not previously reported in the literature. We suspect that this association has been under-reported, perhaps due to a publication or reporting bias.

Learning points

- Carotid Doppler ultrasound has at least the theoretical potential to cause stroke.
- Carotid Doppler ultrasound is routinely performed in individuals at high risk of stroke.
- Reporting or publication bias may explain why this has not been reported before.

Competing interests None.

Patient consent Obtained.

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Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Robertson A, Shinton R, Clow L, Tudway D. Cerebral infarction following a carotid Doppler ultrasound: a chance association? . *BMJ Case Reports* 2012;10.1136/bcr.09.2011.4749, Published XXX

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