Sudden onset vomiting and vertigo following chiropractic neck manipulation

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A previously healthy 38-year-old woman presented to her chiropractor with a 2-week history of right neck pain with radiation to the right occipital area. The pain was exacerbated by neck extension and rapid lateral movements of the neck. It was temporarily relieved by paracetamol. There had been no history of neck trauma. The chiropractor noted that cervical rotation to the left and neck extension reproduced her right sub-occipital pain. The chiropractor then proceeded to neck manipulation which involved sudden lateral flexion 'adjustments'. Immediately after this procedure the patient began to feel vaguely unwell with an increase in the right neck pain. After 5 minutes the patient was helped into a sitting position and started to experience true vertigo shortly followed by profuse vomiting. When the vomiting had not subsided after several minutes the patient was transferred to the Accident and Emergency department.

On arrival, the patient complained of severe vertigo and right neck pain. On initial examination the patient looked unwell. Neurological examination revealed ophthalmoscopic nystagmus to the right and a right Horner's syndrome. The patient did not look Marfanoid, there was no sign of ligament laxity or rheumatological disease. Initial investigation included normal routine haematology, biochemistry and short coagulation screen. Magnetic resonance imaging (MRI) of the head was performed 24 hours after admission (figure 1). This investigation was followed by formal angiography (figure 2).

Questions

1. What do the hyperintense lesions seen on the axial MRI image represent (figure 1)?
2. What is shown in figure 2?
3. What is the diagnosis?
4. How would you treat this patient?
Answers

QUESTION 1
The MRI demonstrates a T2-weighted axial image through the cerebellar hemispheres. The multiple hyperintense lesions (black arrow) represent cerebellar infarction.

QUESTION 2
Formal cerebral angiography was performed and the left vertebral artery was demonstrated to be narrowed and 'bead-like', the radiographic appearances consistent with vertebral artery dissection. There was no radiographic appearance of fibromuscular dysplasia. A 30% filling defect was noted in the basilar artery consistent with basilar artery thrombosis. An attempt was made to cannulate the vertebral artery but the patient became unwell and developed profuse vomiting. The procedure was therefore abandoned.

QUESTION 3
The diagnosis was traumatic dissection of the extracranial vertebral artery(ies) with subsequent brainstem and cerebellar ischaemia secondary to therapeutic neck manipulation.

QUESTION 4
Intravenous heparin was used as treatment in this case, followed by 6 months anticoagulation with warfarin. Although heparin is of unproven value in most acute ischaemic strokes there is some evidence to support its use in this particular situation.

Discussion
Manipulation of the head and neck, a mechano-therapeutic procedure, is becoming increasingly practised. Cerebral and brainstem ischaemia following rotational head movements is well described in the medical literature.1,2 Chiropractic neck manipulations often involve sudden rotational and hyperextensive head movements, which represent a risk of traumatic dissection of the extra cranial vertebral arteries.3 Chiropractors recognise that there is an association of stroke with neck manipulation and have estimated the incidence at one in three million neck manipulations.4 We believe this that this is an underestimate of the actual incidence and that the majority go unreported. A recent report of vertebral artery dissection estimates that 12% of dissections are preceded by therapeutic neck manipulation.5

The question arises whether one can predict who is at risk from stroke during neck manipulation, and whether sudden rotational movements of the neck, as opposed to gentle neck traction, causes increased risk of dissection to the vertebral arteries. The vertebral artery originates from the subclavian artery in the majority of cases. It courses within the transverse foramina to the level of second cervical vertebra before entering the transverse processes of the axis and atlas. It is at this point that the vessel is at particular risk from the shearing forces associated with vertebral artery dissection.

It would seem unwise to manipulate the neck of patients with an increased risk of atlantoaxial subluxation (eg, rheumatoid arthritis) or those with vertebral ligament laxity (Ehlers-Danlos syndrome, Marfan’s disease). Those with cervical spondylosis should probably avoid sudden rotational ‘thrusts’. But beyond this it is impossible to predict who is at risk before neck manipulation takes place.

We believe stroke is an under-recognised complication of neck manipulation. In order to reduce the morbidity and mortality from this procedure we recommend the avoidance of sudden hyperextensive and rotational manoeuvres or ‘thrusts’. Patients who complain of unpleasant symptoms during neck manipulation should always be taken seriously and these symptoms should contraindicate further neck manipulation, with appropriate referral.

Final diagnosis
Traumatic extracranial vertebral artery dissection with subsequent brainstem and cerebellar ischaemia secondary to therapeutic neck manipulation.

Keywords: neck manipulation; stroke; vertebral artery

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