Aneurysm of the cervical internal carotid artery following chiropractic manipulation

Sir: Aneurysms of the extracranial portion of the carotid artery are uncommon. Atherosclerosis and trauma are now considered to be the most common causes.1 2 Traumatic aneurysms of the carotid artery in the neck may be due to either open or closed injury.3 4 A case of internal carotid artery aneurysm at the origin following neck manipulation is reported and is to our knowledge the first reported account of such a case.

A 40 year old male was admitted for a swelling of the left side of the neck, hoarseness of voice and mild drooping of the left eyelid with loss of sweating on the left side of face of one month’s duration. He developed these symptoms within 1–2 hours of neck manipulation by a barber for the relief of pain in the neck. The neck manipulation included sudden jerky rotary movements to either side as well as extension and flexion. On examination he was normotensive, all peripheral pulses were felt equally, there was a pulsatile globular swelling (3 cm × 2 cm) on the left side of the neck below the jaw. It was noncompressable and no bruit could be heard. There was Horner’s syndrome on left side and hoarseness of voice, without vocal cord paralysis.

Syphilitic serology was negative. Lipid profile was normal. Carotid angiogram showed a lobulated aneurysm at the origin of the left internal carotid artery (fig). The right carotid was normal. The left carotid bifurcation was explored 5 days after angiography. A thin walled highly friable 3 cm × 2 cm aneurysm of the distal carotid and proximal internal carotid was found. A clot could be felt within the sac of the aneurysm. A common carotid to internal carotid shunt was inserted before clamps were placed below and above the aneurysm. On opening

References

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the aneurysm no intimal tear could be seen as it was very friable. The aneurysmal sac was excised. Arterial continuity was restored by using a graft. Pulsations were good and no significant bleeding occurred from the graft or the anastomosis. The post operative course was uneventful and he returned to work. His hoarseness of voice improved but not the Horner’s syndrome.

The usual presentation of these lesions is a symptomless pulsatile cervical mass; neurological symptoms may be the presenting features. Rapid expansion can result in dysphagia and respiratory obstruction. The hoarseness of voice in this case was probably due to compression of the larynx by the aneurysm as he had no vocal cord palsy. Horner’s syndrome was due to cervical sympathetic chain involvement.

In India it is a common practice among the rural population to have manipulation of neck with oil massage for relief of neck pain, by a barber. The manipulation consists of sudden jerky and forcible flexion, extensions and rotatory movements. Hyperextension and rotation movements of neck can pull the internal carotid artery tightly against the lateral mass of either the C1 or C2 vertebra as the carotid is firmly rooted in the carotid canal at the base of the skull. Subsequent stretching or compression of the artery against the bony prominence can result in intimal tears and subsequent pseudoaneurysm formation. This mechanism was probably responsible for aneurysm formation in this case. Cases of cervical manipulation presenting with signs and symptoms of an acute intercervical ophthalmoplegia and brainstem dysfunction have been reported.

**Unusual idiosyncratic reactions to carbamazepine**

SIR: Two patients developed unusual idiosyncratic reactions to carbamazepine. H.T., aged 59 years, had a past history of alcohol abuse and chronic pancreatitis. His medication included atenolol 100 mg/day, isosorbide dinitrate 10 mg/t.d.s. and Gaviscon (a combination of alginic acid, magnesium trisilicate, aluminium hydroxide and sodium bicarbonate). He developed blackouts associated with vomiting and was prescribed carbamazepine 200 mg/b.d. After receiving carbamazepine for 2 days he slept for 16 hours and on recovery exhibited bizarre, uncontrollable movements of both hands and arms, throwing any objects he grasped. The movements lasted 12 hours and no further carbamazepine was given.

J.T., aged 15 years, had episodes of loss of consciousness which were later shown to be psychologically determined. On valproate he developed hypersomnia and was changed to carbamazepine 200 t.d.s.. He developed a rash and the dose was reduced to 100 mg/t.d.s. The rash cleared and he remained well for 5 months before complaining of tiredness and dizziness. A month later he started to walk slowly with small, shuffling, stiff limbed steps. His whole body was rigid and on examination he had repeated attacks of shivering involving most of his body. He recovered completely within a week of stopping his medication.

The shivering dystonia exhibited by J.T. is similar to the dose dependent dystonic movements induced by carbamazepine. Ephemerol hemiballismic movements have not been reported with carbamazepine. Theoretically, interaction with atenolol or isosorbide could have raised the serum concentration of the carbamazepine; alternatively interaction with metal-containing drugs as described by Okada et al may have altered the pharmacodynamics of carbamazepine and explained the unusual nature of the dyskinetic movements.

EMR CRITCHLEY, M PHILLIPS
Royal Preston Hospital, Preston, PR2 4HT, Lancs, UK

**References**


3.specifically, 12 patients were...