Vertebral artery dissection presenting as neuralgic amyotrophy

S Berroir, M Sarazin, P Amarenco

Vertebral artery dissection usually presents with neck, occipital, or shoulder pain along with symptoms of ischaemic stroke in the posterior circulation. Isolated pain, asymptomatic cases, or misleading presentation mimicking migraine or myocardial infarction have been seldom reported. Peripheral upper limb deficit or isolated radicular neuralgia due to vertebral artery dissection have also been reported. However, a clinical presentation mimicking a Parsonage-Turner syndrome has not been reported.

A 40 year old man had a rapidly increasing, intense left scapular, and left cervical pain followed by tactile, temperature, and pain sensory loss over the left shoulder and the neck. On the third day, he had gradually increasing proximal weakness of the upper limb while pain progressively improved within a week. At that time, the clinical diagnosis was typical of a Parsonage-Turner syndrome and the patient was on non-steroidal anti-inflammatory treatment. On the 10th day, neurological examination showed nearly complete deficit and amyotrophy of biceps, brachialis, deltoid, and supra and infra spinatus. He had no left bicipital and styloradial tendon reflexes. He had hypaesthesia to pain, temperature, and pinprick in the left C4-C5 dermatoma. He had no neck contracture, pain, or limitation in neck movements and he recalled that 2 days before the symptom onset, he had had neck hyperextension during several hours of cleaning a chimney. A CT of the neck showed an enlarged left vertebral artery with narrowing of the lumen, surrounded by a thin crescent of contrast enhancement, mainly at the C5-C6 level. Ultrasound examination of the left vertebral artery showed widening of the vessel diameter in its intertransverse portion presumably due to dissecting haematoma (fig 1 A). x Ray angiography showed stenosis of the V2 segment of the extracranial vertebral artery suggestive of a dissection (fig 1 A).

Brain MRI showed an enlarged left vertebral artery obstructing the foramen and compressing the nerve roots (fig 1 B). Oral anticoagulant treatment was prescribed for 2 months. The patient fully recovered over 5 weeks. Follow up ultrasound examination showed normalisation of the artery.

The sequence of pain deficit amyotrophy in our patient mimicked a Parsonage-Turner syndrome. The clinical presentation with isolated painful C5 C6 nerve injury and subsequent severe motor deficit, without any clinical or radiological sign of posterior circulation stroke, was remarkable by the rapid amyotrophy and the intensity of sensory loss. The correct diagnosis of vertebral artery dissection was not suspected and would have been missed without CT. The mechanisms was likely a direct root compression by the enlarged vertebral artery at the intervertebral segment as shown in figure 1 B. The course of symptoms, radicular distribution, predominance of motor weakness, and fast recovery are consistent with radicular compression. Hetzel et al described three cases of upper limb radicular injury associated with ipsilateral vertebral artery dissection after chiropractic manipulation. Neck pain followed by severe C5-C6 motor root involvement with no evidence of ischaemic stroke were the presenting symptoms in two cases. Dubard et al described a 31 year old woman with a left C5 motor deficit due to a left vertebral artery dissection. De Bray et al reported three cases of vertebral artery dissection associated with radicular C5-C6 motor deficit. In one case, no sign of CNS involvement was found. Cervical root compressions have been shown in these cases. Prognosis of cervical radicular palsy associated with vertebral artery dissection seems to be excellent as all the reported patients fully recovered.

Other mechanisms of upper limb involvement in relation to vertebral artery disease have been reported such as watershed
infarct in the anterior spinal artery territory due to reduced flow in a dominant vertebral artery, traumatic pseudoaneurysm, ischaemia of the C5 root due to giant cell arteritis of the vertebral artery, and coiling of the vertebral artery at the C6 level.

In conclusion, severe neck pain followed by upper limb radicular deficit and severe amyotrophy, so called Parsonage-Turner syndrome, is an unusual clinical feature of vertebral artery dissection. History of neck injury, chiropractic manipulation, or unusual neck motion during the days before onset should prompt clinicians to perform neck ultrasound examination and CT or MRI to ensure the correct diagnosis and appropriate treatment.
surrounding soft tissue and excised. Histopathological examination confirmed the diagnosis of pseudoaneurysm.

Pseudoaneurysm of the STA is very uncommon and is usually associated with blunt trauma. These lesions present as a painless pulsating mass and sometimes their size may rapidly increase. These lesions may also be associated with headache, ear discomfort or very rarely facial nerve palsy. There have also been reports of pseudoaneurysms of the STA after bypass procedures involving STA and intracranial vessels. These may rupture with consequent subarachnoid or intracerebral hemorrhage.

Pseudoaneurysm of the STA as a complication of craniotomy has not been reported in the literature to the best of our knowledge. We think that inadvertent damage to a segment of the STA not involving the entire thickness of the vessel wall (possibly by electrocautery) in the process of fashioning the scalp flap, led to the formation of a pseudoaneurysm. The pulsatile nature of the lesion and its location rendered the pre-operative diagnosis and subsequent surgical treatment simple. In retrospect, we recommend that where there is suspicion of injury to the STA this vessel should be ligated or coagulated and then divided.

Authors’ affiliations
G Y F Lee, R T Daniel, S Halcrow, Department of Neurosurgery, Royal Adelaide Hospital, Level 5, Theatre Block, North Terrace Adelaide, SA 5000, South Australia

Correspondence to: Dr R T Daniel, Department of Neurosurgery, Royal Adelaide Hospital, Level 5, Theatre Block, North Terrace Adelaide, SA 5000, South Australia

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