

# Vertebral Artery Dissection Due To The Use Of A Hand Held Electric Massager

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## Citation

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## Abstract

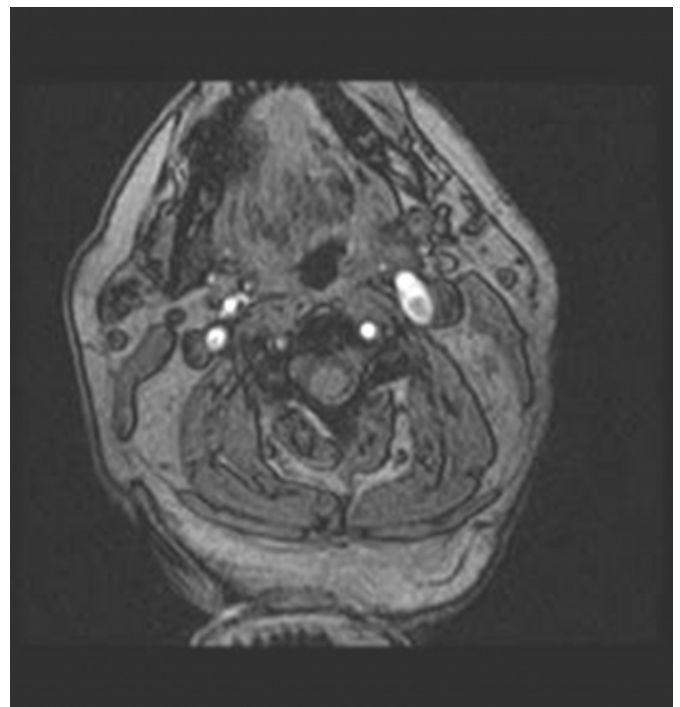
Vertebral artery dissection (VAD) is now an increasingly recognized cause of stroke and is being diagnosed more frequently owing to the widespread use of magnetic resonance imaging for the diagnosis of stroke especially in the young. We report here a case of vertebral artery dissection (VAD) with resultant lateral medullary infarction due to the use of a hand held electric massager. Treatment with heparin can help prevent recurrent embolic events and should be given in the absence of subarachnoid hemorrhage or other contra-indications.

## CASE REPORT

A-65-year old Caucasian male presented to our hospital with sudden onset slurring of speech and unsteadiness of gait. There was no complaint of headache or neck pain. Neurological examination was suggestive of a brain stem syndrome. The patient's speech was dysarthric; he had a Horner's syndrome on the right with right pronator drift and appendicular ataxia. Minimum loss of pain sensation to pinprick and temperature sensation was reported on the left side of the body. The patient reported the onset of the above symptoms while he was hyper-extending his neck as he massaged his back with a hand-held electric massager. Magnetic resonance imaging of the brain and magnetic resonance angiography revealed dissection of the V3 segment of the vertebral artery with intracranial extension and a lateral medullary infarct (Fig 1, 2). There was no evidence of a pseudoaneurysm or subarachnoid hemorrhage. Patient was started on intravenous heparin. His symptoms resolved over 3 days while on heparin and he was discharged home on Warfarin sodium.

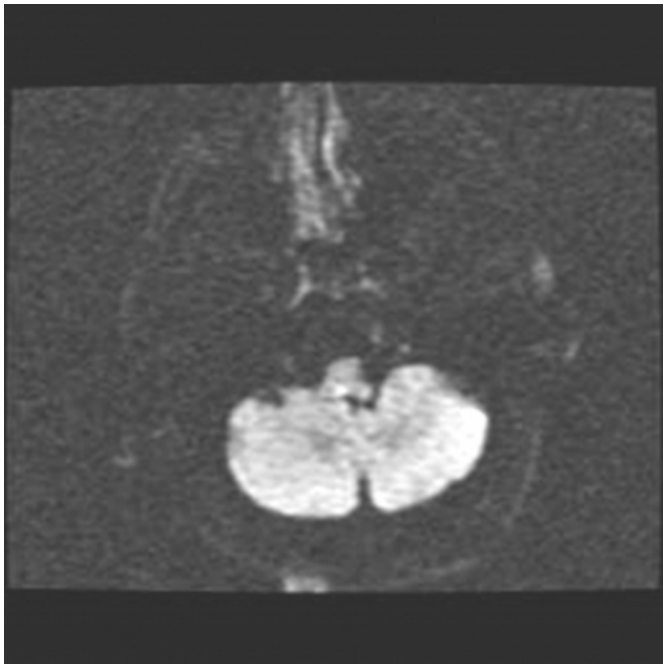
## Figure 1

Figure 1



**Figure 2**

Figure 2



### DISCUSSION

Spontaneous dissection of the carotid or vertebral arteries accounts for approximately 2 percent of all ischemic strokes, but is responsible for 10 to 25 percent of cases in young and middle aged patients<sup>1,3,4,6,9,10</sup>. While the etiology of these dissections is incompletely understood, trivial traumatic insults may play an important role in susceptible individuals.<sup>7</sup> The complete triad of neck pain, headache, and ipsilateral Horner's syndrome is uncommon, often only one or two of these features is present. MRI/MRA has now replaced conventional angiography in the diagnosis of cervicocerebral arterial dissections. Anticoagulants or antiplatelets are the mainstay of treatment for extracranial VAD, but there is a paucity of controlled studies demonstrating effectiveness of this therapy. The prognosis of extracranial VAD is generally much better than that of the intracranial VAD.

The vertebral arteries are branches of the subclavian artery and anatomically are divided into 4 segments. Segment I runs from its takeoff at the first branch of the subclavian artery to the transverse foramina of cervical vertebra C5 or C6. Segment II runs entirely within the transverse foramina of C5/C6 to C2. Segment III begins at the transverse foramen of C2, runs posterolaterally to loop around the posterior arch of C1, and passes subsequently between the atlas and the occiput. It is a tortuous segment, and most frequent site of dissection. Segment IV is the intracranial

segment. It begins as the vertebral artery pierces the dura at the foramen magnum and continues until the junction of the pons and medulla, where the vertebral arteries merge to form the basilar artery. Before joining to form the basilar artery, however, each vertebral artery branches to form the left and right posterior inferior cerebellar arteries (PICA). The first major branches of the basilar artery are the anterior inferior cerebellar arteries (AICA), followed by several small arteries that supply the pons, and finally the superior cerebellar arteries. The basilar artery ends in the posterior cerebral arteries that complete the Circle of Willis posteriorly. Thus the vertebrobasilar circulation feeds the brain stem (medulla, pons, and midbrain), cerebellum, occipital lobes, posterior temporal lobes, and thalamus.

Dissections often involve loops and redundant portions of the extracranial vertebral artery such as the tortuous segment III. They begin with a tear in the media, which leads to bleeding within the arterial wall. Intramural blood then dissects longitudinally, spreading proximally or distally. The dissection can tear through the intima, compress the lumen, dissect into the media (revealing intimal flaps on conventional angiography), or cause an aneurysmal outpouching of the arterial wall (pseudo aneurysm. Each may lead to partial or total lumen occlusion.

Patients usually present with a constellation of brainstem signs, specifically lateral medullary (Wallenberg) syndrome. They signs most often include the following:

- Ipsilateral facial dysesthesia (pain and numbness) - Most common symptom
- Dysarthria or hoarseness (cranial nerves IX and X)
- Contralateral limb or trunk numbness (spinothalamic tract)
- Ipsilateral loss of taste (nucleus of tractus solitarius)
- Hiccups
- Vertigo
- Nausea and vomiting
- Diplopia or oscillopsia
- Dysphagia (CN IX and X)
- Disequilibrium

Cervical spinal cord ischemia is an uncommon, but potential complication, of VAD. Furthermore, there may be other presentations, such as thalamic, cerebral, or cerebellar signs. Headache and neck pain, though common, may be absent and may be confused as musculo-skeletal pain.

Before the widespread use of modern imaging techniques in the 1970s, cervicocranial vessel dissection was generally a post-mortem diagnosis. Modern diagnostic techniques are now available, and include neurovascular ultrasound as a screening technique, helical CT, CT angiography and magnetic resonance techniques (MRI/MRA). Conventional angiography remains the gold standard for diagnosing dissections. However, invasiveness of the procedure and associated complication rates has led to MR angiography replacing conventional angiography as the initial investigation of choice. The resolution does not quite approach that of conventional angiography, but the procedure is non-invasive, can be repeated to document recanalization and allows the physician to look at the surrounding soft tissue, elaborating the intramural hematoma in relation to the size of the vessel lumen <sup>2, 11, 12</sup>.

Vertebral artery dissection may be associated with preceding trivial neck trauma. Neck manipulation is the most common setting of VAD, including chiropractic manipulation, though VAD has been documented following yoga and vigorous aerobic exercise <sup>8, 13</sup>.

The outcome of VAD is usually benign, with most patients making a complete recovery. Anticoagulation has been accepted as the treatment of choice but its use in the setting of intracranial extension of VAD remains controversial. The mechanism of ischemia after dissection probably results from distal microemboli. Medical treatment is aimed at reducing clot propagation and distal embolization and includes the use of antiplatelet drugs and anticoagulation <sup>5</sup>. Early anticoagulation with heparin is usually the standard of care though there are no controlled studies to validate its use. Anticoagulation is usually continued with warfarin until the vessel has recanalised, as determined by repeated Doppler

ultrasound studies. Recanalisation may occur at any time and anticoagulation with warfarin is usually continued for 6 months following which warfarin may be replaced by aspirin.

Our case highlights the above mentioned features and reinforces the fact that dissection of the major arteries in the neck is an important and probably underdiagnosed cause of stroke and craniocervical head pain.

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