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# CASE REPORT

# Vertebral artery dissection following a posterior cervical foraminotomy

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# Abstract

Vertebral artery dissection following a posterior cervical foraminotomy with rhizolysis of the subaxial spine has not been described before. A 46-year-old lady underwent the procedure for a left C6 radiculopathy with a focal disc herniation with no intraoperative complications. Seven hours post-operatively, she developed a right homonymous hemianopia, thalamic dysphasia, gait and memory impairment. Imaging demonstrated an occlusion to the left vertebral artery from C7/T1 to C4 with a dissection flap noted at the inferior margin. This was further complicated with thrombosis of the dissected artery and subsequent emboli causing acute posterior circulation infarctions. Given that the dissection occurred at the level of the surgery, an indirect surgical cause is likely. We hypothesize that the vibration transmitted via bone from the high-speed drill led to arterial injury and dissection.

# INTRODUCTION

Vertebral artery injury is a rare but potentially catastrophic complication of cervical spinal surgery with a reported incidence of 0.07% [1]. Those injuries that have been reported have most commonly involved an anterior approach or following lateral mass screw placement to the cervical spine. Of the 1.8% that have occurred following a posterior approach, all have involved the axial cervical spine. To date, there have been no reports of injury to the subaxial vertebral artery following a routine foraminotomy of the subaxial cervical spine [1–3].

We describe a case of a patient who developed a delayed left-sided vertebral artery dissection following an uncomplicated left C5/6 foraminotomy.

# CASE REPORT

A 46-year-old female presented with a 5-month history of progressive left-sided C6 radiculopathy that had not responded to

medical management. Clinical examination confirmed paraesthesia in the left C6 dermatome and mild weakness of the left biceps muscle. Neurological examination was otherwise unremarkable. A magnetic resonance imaging scan revealed a left-sided C5/6 foraminal stenosis secondary to a focal disc herniation.

An uncomplicated left-sided C5/6 foraminotomy and rhizolysis was performed. The initial decompression was performed using a standard high-speed drill with a matchstick attachment and manual irrigation. Following surgical exposure, it was noted that the C6 nerve root was somewhat swollen. There was no soft disc encountered. A successful decompression was achieved and at the time there was no evidence of arterial injury. Throughout the procedure she had remained stable in the Mayfield head clamp with the neck slightly flexed. There were no episodes of excessive flexion, extension or lateral rotation at any stage of the procedure.

She awoke from the anaesthetic neurologically intact; however, 7 hours later she developed an acute onset of right homonymous hemianopia, expressive dysphasia and became

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drowsy. An initial computed tomography (CT) head was unremarkable. We elected to not thrombolyse her due to her surgery but commenced aspirin. Following further neurology review by a stroke consultant on the following morning, we noted that she demonstrated a right homonymous hemianopia without macular sparing, bilateral gaze-evoked and rebound nystagmus, thalamic dysphasia, multifactorial gait difficulty and memory impairment.

A CT arteriogram demonstrated an occlusion to the left vertebral artery distal to C7/T1 with reconstitution at the inferior endplate of C5. This was followed by an area of attenuation and a return to normal calibre at C4. A small dissection flap was noted at the inferior margin of the opacified artery. In addition to this, there was an occlusion of the left distal P3 segment of the posterior cerebral artery. She had acute infarcts involving the left mesial occipital lobe, left posterolateral thalamus, posterior limb of the left internal capsule and left hippocampal tail. In addition, she has bilateral superior cerebellar infarcts with the right side affected more so than the left.

She was referred to stroke rehabilitation where she underwent occupational therapy support and visual field testing. She was subsequently discharged. At the 3-month review, her visual fields demonstrated some improvement. She also had resolution of her paraesthesia and weakness.

### DISCUSSION

As far as we are aware this is the first reported case of delayed vertebral artery dissection following a posterior foraminotomy and rhizolysis [1–3]. The precise pathophysiology of this complication is difficult to determine given that there was no evidence of injury to the vertebral artery at the time of surgery and there had been no abnormal movements of the neck.

Spontaneous dissection of the vertebral artery has been reported in the setting of genetic changes such as low levels of alpha-1 antitrypsin, connective tissue diseases, various genetic polymorphisms, gene mutations, hyperhomocystienemia, the presence of migraines and vessel abnormalities [4]. Environmental factors have also reportedly contributed and these include infections and oral contraceptive use [4].

However, our patient had none of these risk factors and given that the dissection occurred precisely at the surgical site it is difficult to attribute the injury to anything other than either direct or indirect surgical trauma. The absence of any significant haemorrhage at the time of surgery would seem to make indirect trauma more likely and a possible mechanism may be damage induced by use of the high-speed drill.

This may occur due to thermonecrosis if the area is not cooled appropriately [5] or due to vibrations transmitted through the bone.

Animal studies have demonstrated that repetitive vibration can induce arterial constriction increasing shear stress [6] and subsequent thrombosis [7]. Arterial injury increased with increasing frequency of vibration [8]. There is also an increase in oxidative activity [9] and an increased sensitivity to alpha-2Cadrenoreceptor mediated vasoconstriction [6]. It has also been demonstrated that vibration between 60 and 800 Hz, in the arteries of rat tails, increased discontinuities in the internal elastic membrane with patches of missing tunica intima [10].

In this particular case, it may be that vibrational shear stress contributed to vessel wall injury and subsequent thrombosis [7]; however, this remains to be established. The use of a high-speed surgical drill could have transmitted the vibration through the bone and subsequently caused damage to the tunica intima of the vessel causing a dissection.

### FUNDING

None.

### CONFLICT OF INTEREST STATEMENT

None declared.

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