Numbness, tingling and bubbles

Introduction

Subclavian steal syndrome is a circulatory disorder usually caused by atherosclerosis and is accompanied by ischaemic symptoms in the vertebrobasilar region and the associated limb. In more than 80% of cases the vascular lesion is on the left. A stenosis or occlusion in the subclavian artery causing the inter-arm pressure gradient to be more than 20 mmHg can result in retrograde blood flow in the ipsilateral vertebral artery to supply the arm with lower blood pressure. This causes a vascular 'steal' by depriving the posterior brain circulation of its blood supply, resulting in vertebrobasilar symptoms.

Ischaemia in a limb often manifests as arm weakness, claudication, paraesthesia or coldness in the arm. New imaging techniques have drastically improved the diagnosis of subclavian steal syndrome and understanding of its prevalence.

Discussion

Subclavian steal syndrome generally occurs in patients >55 years of age and has a 2:1 male:female ratio. Incidence in the general population is reported to be about 3–4% but flow reversal within the vertebral artery is present in only a minority (2–5%) of these cases because of the abundant collateral blood supply in the head, neck and shoulder (Perler and Becker, 1988); only about 5% of these patients suffer symptoms. This emphasizes that flow reversal within the vertebral artery alone does not always result in neurological symptoms.

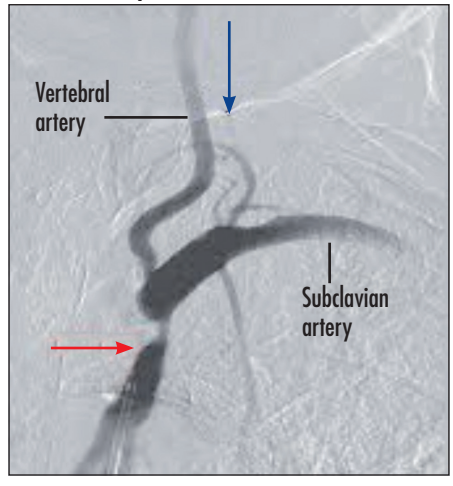
When symptomatic, subclavian steal syndrome can manifest with a variety of vertebrobasilar insufficiency symptoms such as headache, downbeat nystagmus, blurred vision, diplopia, impairment of consciousness, dysarthria, sensorineural deafness and facial paraesthesiae. Neurological symptoms such as vertigo are considered to be of central origin as the vertebrobasilar arterial system feeds both the peripheral and central auditory and vestibular systems (Rosengart and Hedges, 1993).

The most important collateral circulation to the posterior fossa is through the circle of Willis, principally via the posterior communicating artery; when this communication is absent or inadequate, possibly as a result of concurrent extracranial carotid stenosis, then vertebrobasilar symptoms manifest.

Patients in whom central nervous symptoms predominate should have carotid stenoses addressed before any attempt at surgical revascularization of the subclavian system. Neurological symptoms abate after re-establishment of normal carotid anterograde flow, even in patients with significant subclavian lesions (Burihan and Soma, 2011). It is possible that the progression of carotid stenosis in this patient over years precipitated the subclavian steal symptoms.

If a patient has undergone coronary revascularization with a left internal mammary artery graft, new-onset angina may herald proximal left subclavian stenosis as the left internal mammary artery arises from the inferior aspect of the proximal subclavian artery.

Figure 1. Digital subtraction image showing narrowing (red arrow) of the left subclavian artery and retrograde filling (blue arrow) of the post stenotic subclavian artery from the left vertebral artery.



Case Report

A 76-year-old woman presented with subacute onset of left arm numbness, tingling and a sensation of 'bubbles' running under her left shoulder. The left radial pulse was feeble and the systolic blood pressure in the left arm was 20–30 mmHg lower than in the right. Incidentally, a subclavian bruit was also heard on the left side.

Her past medical history was of a thrombotic cerebrovascular accident, hypertension, high cholesterol and atrial fibrillation. Asymptomatic left carotid stenosis was diagnosed on carotid duplex a few years previously but she was managed conservatively. Relevant medications included warfarin, bisoprolol, enalapril and simvastatin. Initial haematological and biochemical investigations showed normal cholesterol and glucose levels and an international normalized ratio of 2.2. A computed tomogram of the head was negative for any acute ischaemic change and the case was initially managed as a sensory lacunar stroke.

A computed tomography aortogram was done next day to rule out an aortic dissection because of the persistent discrepancy in upper limb blood pressures. This showed bilateral carotid stenosis (right: 50–60%, left: 70–80%), a tight subclavian stenosis on the left and multiple collateral arterial vessels around the left scapula (*Figure 1*). A two dimensional echocardiogram did not report any structural cardiac abnormalities.

A subsequent carotid and vertebral Doppler showed retrograde flow in the left vertebral artery confirming a vascular steal phenomenon; the distal subclavian artery showed parvus tardus and monophasic waveforms.

The sensation of bubbles under her left shoulder was probably caused by the turbulent blood flow in the collaterals around the scapula and the intercostal arteries that opened in an attempt to bypass the subclavian obstruction and supply blood to the distal arm (similar to the 'Suzman sign' reported in coarctation of the aorta).

A final diagnosis was made of a stroke mimic resulting from a vertebrobasilar insufficiency caused by a vertebral subclavian steal as a result of underlying subclavian artery stenosis. No medication changes were made and her warfarin was continued. Vascular surgery declined any intervention as the symptoms were deemed moderate and only involved the limb.

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Upper extremity exercise increases blood flow to the arm by reducing arterial resistance and can precipitate lateralizing symptoms of vertebrobasilar insufficiency among persons without sufficient collateral flow. This can help evaluate the 'steal' phenomenon during Doppler evaluation of the neck (Hennerici and Klemm, 1988).

Aggressive management of risk factors, such as hypertension, diabetes and tobacco use, is also essential for successful treatment of subclavian steal syndrome (Hennerici et al, 1988), percutaneous intervention is reserved for disabling symptoms. In one trial (Chimowitz and Lynn, 2005) warfarin was associated with significantly higher rates of adverse events and provided no benefit over aspirin. BJHM

Burihan E, Soma F, Iared W (2011) Angioplasty versus stenting for subclavian artery stenosis. *Cochrane Database Syst Rev* CD008461 (doi: 10.1002/14651858.CD008461.pub2)

Chimowitz MI, Lynn MJ, Howlett-Smith H et al (2005) Comparison of warfarin and aspirin for symptomatic intracranial arterial stenosis. *N Engl J Med* **352**: 1305–16

Hennerici M, Klemm C, Rautenberg W (1988) The subclavian steal phenomenon: a common vascular disorder with rare neurologic deficits. *Neurology* **38**: 669

Henry M, Henry I, Klonaris C, Hugel M (2004) Percutaneous transluminal angioplasty of the subclavian arteries. In: Henry M, Ohki T, Polydorou A, Strigaris K, Kiskinis D, eds. *Angioplasty and stenting of the carotid and supra-aortic trunks.* Springer, London: 655–6

Perler BA, Becker GJ (1998) *Vascular intervention: A clinical approach.* Thieme, London: 474–5

Potter BJ, Pinto DS (2014) Subclavian steal syndrome. *Circulation* **29**(22): 2320–3 (doi: 10.1161/CIRCULATIONAHA.113.006653)

Psillas G, Kekes G, Constantinidis J et al (2007) Subclavian steal syndrome: neurotological manifestations. *Acta Otorhinolaryngol Ital* **27**: 33–7

Rosengart A, Hedges TR 3rd, Teal PA et al (1993) Intermittent downbeat nystagmus due to vertebral artery compression. *Neurology* **43**: 216–18

LEARNING POINTS

- Subclavian steal syndrome is caused by retrograde blood flow from the vertebrobasilar artery circulation to supply the stenosed subclavian artery territory causing vertebrobasilar insufficiency symptoms.
- Subclavian stenosis identifies a population that will benefit from aggressive secondary prevention. Percutaneous angioplasty and stenting is the favoured approach if conservative management fails.
- Duplex ultrasonography of the neck vessels and transcranial Doppler is more sensitive than conventional angiography for detecting flow reversal.
- In patients with neurological symptoms, other factors must be considered, most commonly concurrent carotid stenosis and neurovascular instability.
- Patients with arm numbness, claudication and isolated neurological symptoms should be examined for subclavian steal syndrome and followed up in order to prevent other neurological deficits.

Facial emphysema following nasal fracture

A young man presented with slowly progressive facial swelling of 5 days' duration, following a physical altercation. Examination revealed bruising and crepitation over the swollen area (*Figure 1a*). His vision was not compromised.

Computed tomography demonstrated facial subcutaneous free air associated with nasal bone fracture (*Figure 1b*). The patient was managed with antibiotics, advised to avoid nose blowing and to sneeze with his mouth open. The facial swelling resolved spontaneously over the following 48 hours.

Air entrapment in the soft tissues following cervicofacial injury is generally a benign condition. However, severe complications may arise following such injury. Potential complications include respiratory obstruction, pneumocephalus secondary to airflow into the anterior cranial fossa with subsequent meningitis, ophthalmic compromise, pneumomediastinum as a result of air penetration of the parapharyngeal space and localized necrotizing fasciitis. Management consists of prophylactic antibiotics, observation, early fracture fixation where necessary and appropriate management of the complications. BJHM

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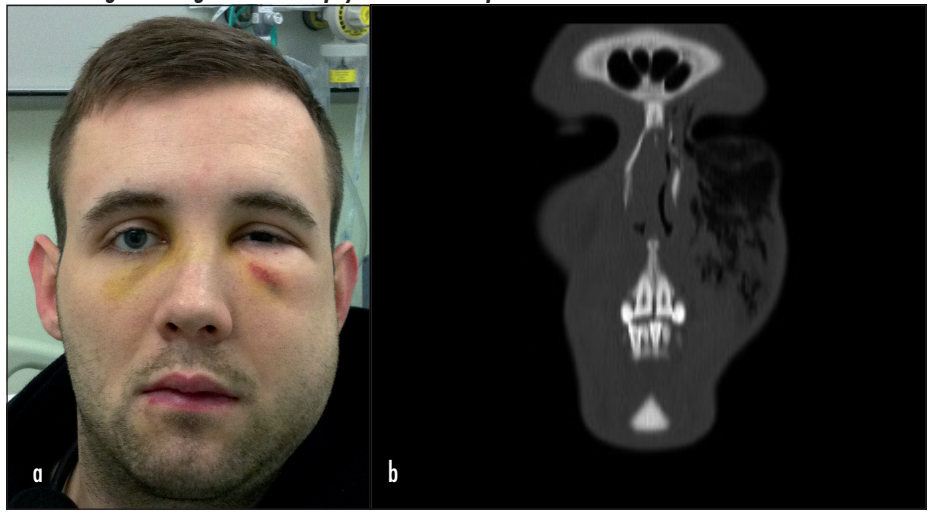
Figure 1. a. Facial image showing left maxillary and periorbital swelling. b. Computed tomographic coronal image showing left facial emphysema and disruption fracture of the left nasal bone.



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