

# Cerebral embolism: Distal subclavian disease as a rare etiology

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### Abstract

Subclavian artery occlusive disease is usually secondary to persistent compression caused by thoracic outlet syndrome (TOS) and rarely due to focal atherosclerosis. Emboli from diseased vessel can flow retrograde to the vertebral or carotid arteries to produce ischemic infarct with or without neurological deficit. We are reporting two cases of distal subclavian artery disease presenting with cerebral embolization, an unusual manifestation. Such surgically correctable lesions producing cerebral emboli and stroke needs consideration while evaluating patients with unusual presentation to prevent further occurrence of stroke.

### Introduction

A transient ischemic stroke (TIA) is defined as temporary disruption of blood flow to the brain with resultant neurological dysfunction, which subsequently resolves within 24 hours of onset.<sup>1</sup> TIAs are usually caused by thromboembolism from the heart or neck vessels. Rarely, retrograde cerebral embolism via the right carotid and vertebral arteries may complicate distal subclavian disease (DSD).<sup>2</sup> We are reporting two cases of distal subclavian artery disease who presented with TIAs. The objective of reporting these cases is to create awareness of distal subclavian disease as a cause of cerebral emboli in patients with ischaemic strokes without carotid or vertebral artery atherosclerotic lesions.

### Case Summary

#### Case 1

An 18 years old right handed boy presented to emergency room (ER) with a history of sudden severe vertigo and left sided body weakness two hours prior to presentation. Weakness lasted for approximately 10 minutes. There was no history of loss of consciousness, trauma or seizure disorder.

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On further questioning, he admitted to right upper limb claudication for the past 6 months. On examination, he was conscious. There were no neurological deficits. Pulses were absent in the right upper limb, however the arm did not have signs of ischaemia. Biochemical and haematological indices were normal. Magnetic Resonance Imaging (MRI) of the brain with angiography revealed right basal ganglia infarct with occlusion of right middle cerebral artery. A chest x-ray showed right cervical rib (Figure 1 A); hence a provisional diagnosis of vascular TOS was made. Digital subtraction Angiography (DSA) showed an occluded right subclavian artery at the thoracic outlet, distal to vertebral artery. Thrombus was extending into axillary and proximal half of the brachial

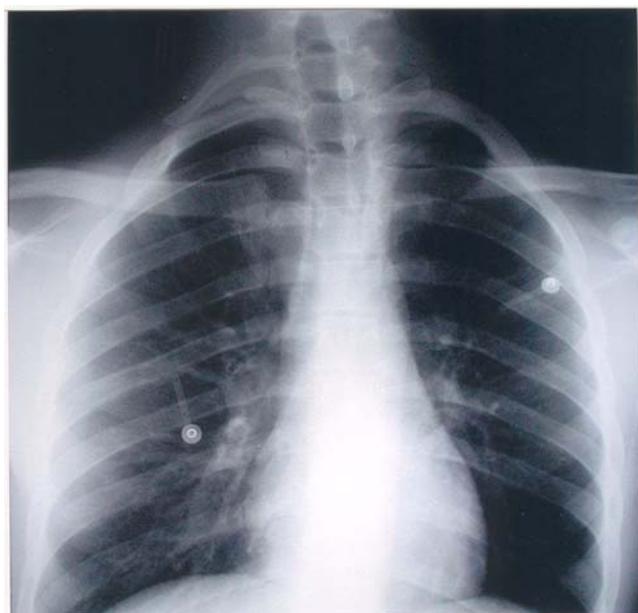


Figure 1 A. Chest X-Ray showing right cervical rib.

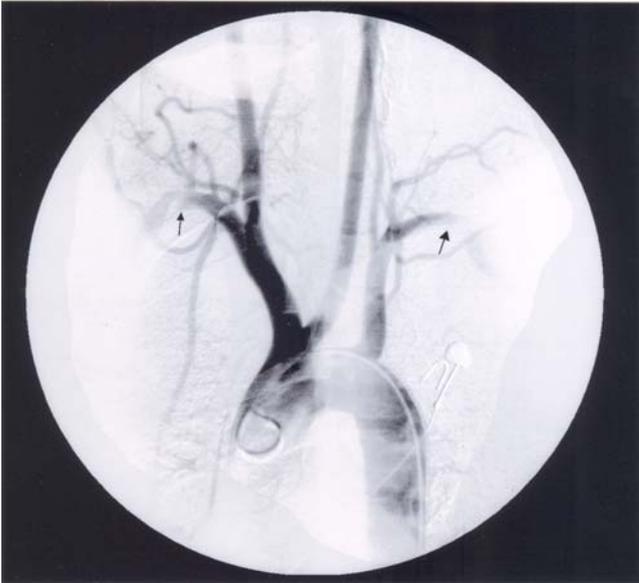


Figure 1 B. Arch Aortogram with four vessel runoff showing occlusion of right subclavian artery at thoracic outlet (thin head arrow) and 50% stenosis of left subclavian artery (thick head arrow).

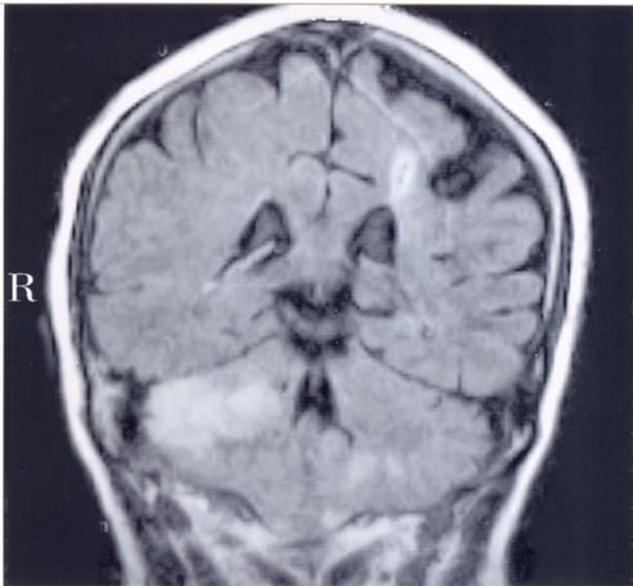


Figure 2 A. MRI Brain showing bilateral cerebellar infarct.

extending into axillary and proximal half of the brachial artery. The left subclavian artery showed 50% stenosis, exaggerated by hyper abduction of left shoulder (Figure 1 B). Carotids, vertebral and intracranial vessels on both sides were normal. Hence a diagnosis of retrograde cerebral embolism was made.

He underwent right subclavian exposure through standard supraclavicular approach. The cervical rib was excised, brachial artery exposed in the mid arm and a reverse great saphenous vein was used as conduit between subclavian and brachial artery bypassing the diseased segment. Three months later, he underwent exploration for left TOS and



Figure 2 B. Angiogram showing localized stenosis of left distal subclavian artery as shown by arrow.

anterior scalenectomy was performed. He had an unremarkable recovery. Now he has been asymptomatic for the past three years.

## Case 2

A 52 year old right handed woman with no known co morbid conditions was admitted under the care of a neurologist with a provisional diagnosis of posterior circulation TIA. She gave a history of severe vertigo and drowsiness for 1 day. She had had severe left upper limb pain a week ago, which resolved with analgesia within 12 hours. On examination, she was haemodynamically stable with absent pulses in left upper extremity. Initial laboratory investigations were within normal limits. MRI of brain showed multiple acute infarcts involving both cerebellar hemispheres (Figure 2 A).

A Chest x-ray did not reveal cervical rib. Duplex ultrasound examination of carotid arteries was normal. The finding of absence pulses on left side prompted an evaluation of the major neck vessels for possible cause of posterior circulation TIA. Arch aortogram showed severe stenosis of left subclavian artery distal to origin of internal mammary artery with normal vessels distally (Figure 2 B). Both common carotids, vertebral and intracranial vessels were normal. A provisional diagnosis of distal subclavian disease with subsequent retrograde embolism via vertebral artery was made. She underwent left subclavian exposure through supraclavicular incision. Localized stenosis of subclavian artery was found. This area was resected and an interposition 6 mm PTFE graft was placed in the segment. Histopathology revealed atheromatous plaque with thrombus. She was started on warfarin.

## Discussion

Diseases in the heart or neck arteries are the usual cause of cerebral embolism.<sup>2</sup> Rarely cerebral embolism may be secondary to DSD. Isolated occlusive lesions of the subclavian artery are usually asymptomatic because of the rich arterial collateral supply of the head, neck, and shoulder. The mechanism for symptomatology may be haemodynamic or atheroembolic.<sup>3</sup>

Arterial complications of thoracic outlet compression have serious prognostic implications, although they are present in fewer than five percent of operations performed for TOS.<sup>4</sup> Distal subclavian occlusive disease whether due to TOS or atherosclerosis needs further workup for confirmation of diagnosis and planning treatment.<sup>5</sup> Depending on the salient features of the history and physical examination, further investigations are done for diagnosis of this entity. Imaging of brain (CT scan or MRI) should be done for patients thought to have had a stroke. Arch aortography with four-vessel views is necessary in all patients. Synchronous lesions in carotid, vertebrobasilar and intracranial vessels should be sought as this might adversely affect outcome of any intervention.<sup>3</sup>

Distal subclavian occlusive lesion usually caused by TOS requires surgical decompression, exclusion of diseased vessel and distal revascularization when needed<sup>6</sup>, as done in first case. In the second case; localized resection of the diseased segment with interposition graft was done.

Few case series<sup>2,4</sup> and case reports<sup>6</sup> have described retrograde cerebral embolism secondary to DSD. However, clinical practice guidelines for management of ischemic stroke in Pakistan<sup>7</sup> did not emphasize evaluation of the sub-

clavian vessel in the algorithm probably because of the rarity of this entity. Neurology Text does recommend evaluation by angiography in patients presenting with ischemic stroke in select group of patients.<sup>8</sup>

In summary, patients presenting with TIAs with synchronous positive finding on vascular examination require further work up to identify the underlying mechanism of neurological deficit. More importantly documentation of upper limb symptoms and absent pulses should raise suspicion of DSD as a cause of the cerebral ischemic event. Angiography with demonstration of lesions helps in planning intervention to prevent further episodes of TIAs.

## References

1. Elaine J Skalabrin, Gregory W Albers. Transient ischemic attack. In: Richard T Johnson eds. Current therapy in neurologic disease. 6th ed. Missouri, USA. Mosby, 2002, pp 195-99.
2. Prior AL, Wilson LA, Gosling RG, Yates AK, Ross-Russell RW. Retrograde cerebral embolism. *Lancet* 1979;8151:1044-7.
3. Kenneth J. Cherry Jr., M.D. Arteriosclerotic occlusive disease of brachiocephalic arteries. In Robert B Rutherford, eds. *Vascular Surgery*, 4th ed. Colorado, USA. W.B. Saunders, 1995, pp. 947-51.
4. Eduardo Kieffer, Carlo Ruotolo. Arterial complications of thoracic outlet compression. In: Robert B Rutherford, eds. *Vascular Surgery*, 4th ed. Colorado, USA. W.B. Saunders, 1995, pp 992-99.
5. Bearn P, Patel J, O'Flynn WR. Cervical ribs: a cause of distal and cerebral embolism. *Postgrad Med J* 1993; 69:65-8.
6. Nishibe T, Kuniyama T, Kudo FA, Adachi A, Shiiya N, Murashita T et al. Arterial thoracic outlet syndrome with embolic cerebral infarction. Report of a case. *Panminerva Med* 2000;42:295-7.
7. Shafiqat S. Clinical practice guidelines for the management of ischemic stroke in Pakistan. *J Pak Med Assoc* 2003; 53:589-93.
8. Chin-Sang Chung and Louis R. Caplan. Neurovascular disorders. In: Christopher G. Goetz. *Textbook of clinical neurology*, 2nd edition. Elsevier Science, USA 2003, pp. 991-1015.