Subclavian Steal Phenomenon With Right Hemiparesis A Case Report

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ABSTRACT

Subclavian Steal Syndrome results from stenosis of the subclavian artery proximal to the origin of the vertebral artery. This rare disease, commoner in males results in reversal of the normal direction of blood flow in the vertebral artery from the cerebral circulation.

We report a case of a 42 year old female who presented with right sided hemiparesis and history of giddiness, precipitated by activity involving right upper limb especially with overhead abduction. Radial and Brachial pulsations in right upper limb were absent. Echocardiogram, MR brain angio, Aortogram and right upper limb arterial Colour Doppler confirmed a right subclavian steal. This rare condition even rarer in females had an unusual right upper limb involvement instead of the usual left upper limb involvement. Though she had initially presented as a Cerebrovascular accident she was finally found to have a subclavian steal.

Key Words: Subclavian steal syndrome, Right Hemiparesis, Atherosclerosis

Introduction

Atherosclerotic disease of the innominate artery is an infrequent condition comprising less than 2% of extracranial cerebrovascular insufficiencies.[1] Vertebrobasilar insufficiency symptoms occur when compensatory flow to the subclavian artery from the vertebral artery diverts blood toward the arm and away from intra-cranial structures.[2] Clinical presentation ranges from asymptomatic stenosis to severe vascular compromise precipitating subclavian steal syndrome manifesting with transient ischaemic cerebral and arm symptoms. Male-to-female ratio is 2:1 and average age is above 55 years.[3] Clinical marker is a difference in blood pressure between the two arms exceeding 20 mm Hg. Doppler ultrasound confirms the diagnosis, although MRI, MR Angiography, CT Brain and Aortography are complementary. Patients with disabling symptoms require interventions.

Case Report

A 47 year old female, a known case of hypertension and newly diagnosed diabetes mellitus came with complaints of sudden onset of weakness in right upper and lower limbs since 7 days associated with tingling and numbness. Patient also had a history of giddiness, aggravated by strenuous activity involving right upper limb

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particularly movements above the head. The giddiness was relieved by rest and lowering the upper limb. Complaints were of 2 months duration which subsided with the onset of weakness. She had no history of recent trauma, loss of consciousness, seizures, febrile illness, bladder and bowel involvement or any features suggestive of cranial nerve involvement.

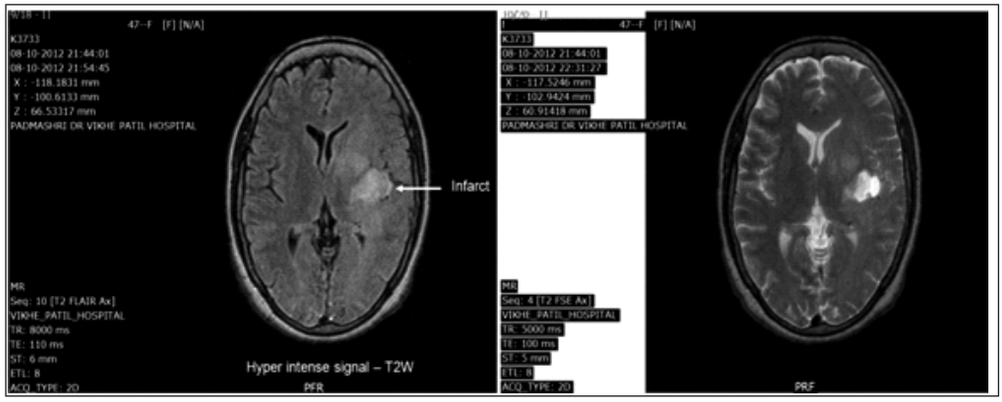

Fig 1 and 2. T2 FSE Flair Sections and T2 FSE Axial Section showing focal hyper intense signal at left gangliocapsular region (External capsule).

The General physical examination was unremarkable except absent radial and brachial pulsations in right upper limb. All other peripheral pulsations were normal. BP was not recordable on the right arm. The left arm BP was 110/74 mm of Hg. On neurological examination her higher functions and speech were normal. Cranial nerves were normal. Tone was normal. Power in right upper limb and lower limb was grade II/V. Reflexes and sensations were normal. Both plantars were flexors. Other systemic findings were normal. Patient had no history of pain over any part of the hand, side of the neck, pectoral region, axillary area, and the upper back with no history of any discoloration of the hands, weakness of the hand and arm muscles. Clinically there were no recent symptoms of malaise, fever, night sweats, weight loss, arthralgia, rash and fatigue suggestive of inflammatory arthritis. There was also no history of Raynaud's phenomenon. No history or finding of rheumatic or other heart diseases was present and carotid bruit was absent.

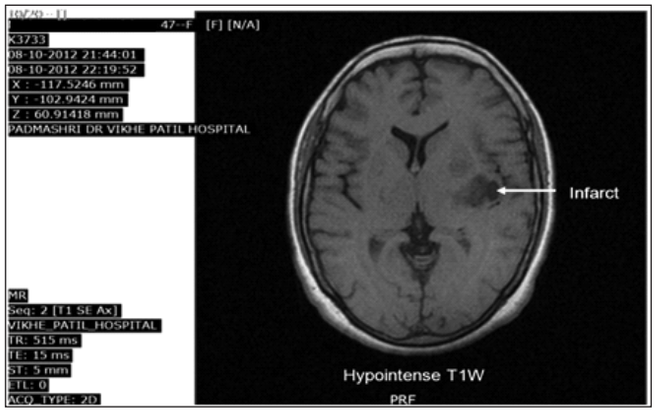

Fig 3. T1 weighted image showing focal hypo intense signal at left gangliocapsular region (External capsule).

With onset of right hemiparesis the symptoms subsided which later recurred on recovery and resumption of right upper limb. Adson's sign was negative. Patient's Haemogarm and ESR were normal. Liver and Renal Function Tests / Lipid Profile / Serum Homocysteine levels were normal. ECG was normal. Collagen disorder serology, HBsAg, anti HCV, HIV were negative. Non contrast CT Brain revealed a resolving ischaemic infarct with early haemorrhagic transformation located in left Ganglio Capsular region extending upto Corona Radiata. Multiplanar MRI was suggestive of evolving (late subacute) infarct located in Internal Capsule (Figs 1-4).

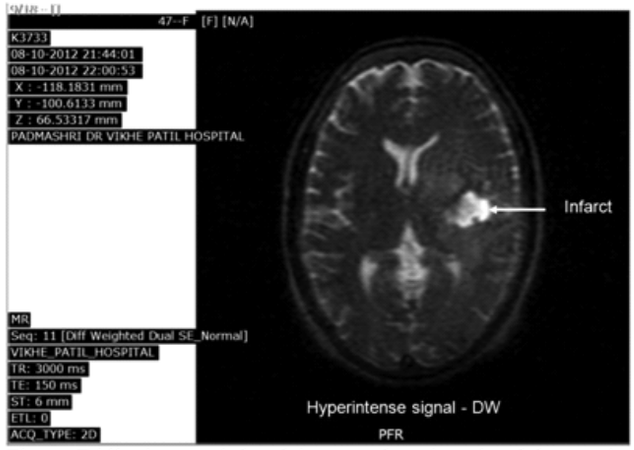

Fig 4. Diffusion weighted image showing focal hyper intense signal in the corresponding area suggestive of restricted diffusion.

2D echo and Colour Doppler showed mild concentric LVH, no RWMA and no intra cardiac clots and vegetations. Origin of common carotid artery showed pressure gradient of 66 mm Hg and mean gradient of 36 mm Hg. Right Carotid and upper limb arterial Doppler showed dampened velocities along with monophasic waveform in right upper limb arterial system associated with right vertebral steal syndrome suggestive of lesion proximal to origin of right subclavian artery (Fig.5).

A 64 slice CECT of Arch of Aorta and its branches (Aortography) was done which showed concentric mixed calcific plaque with focal severe narrowing at the origin of the right subclavian artery (Figs 6,7).

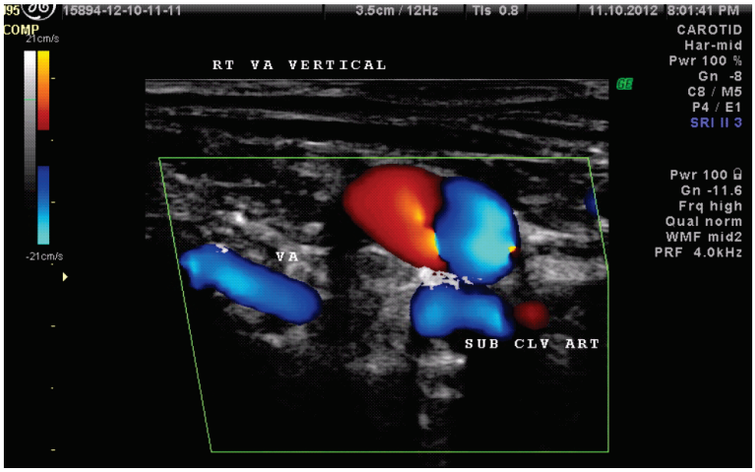

Fig 5. The above colour Doppler image showing reversal of flow in the vertebral artery. Note the colour difference between CCA & vertebral artery indicating different directions.

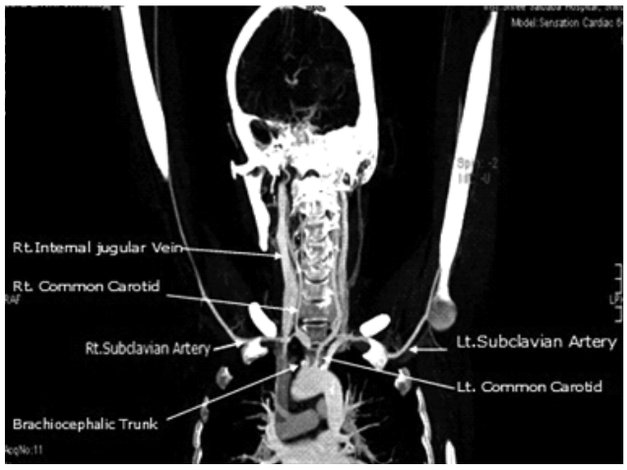

Fig 6. 64 Slice CECT of arch of aorta and its branches showing plaque at the origin of right subclavian artery.

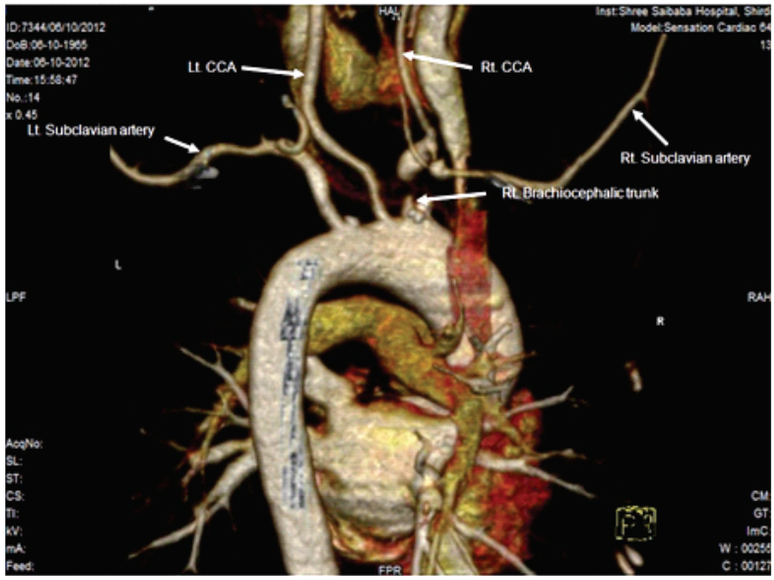

Fig 7. 3D Volume Rendered coronal sections of arch of aorta and its branches.

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Right Carotid and upper limb arterial Doppler showed dampened velocities along with monophasic waveform in right upper limb arterial system associated with right vertebral steal syndrome suggestive of lesion proximal to origin of right subclavian artery.

Discussion

Subclavian Steal Syndrome includes neurological symptoms due to cerebral ischaemia initiated by ipsilateral arm exercise and difference of blood pressure in the upper limbs (average, 45 mm Hg). Even if retrograde blood flow in the vertebral artery occurs in the presence of proximal subclavian artery stenosis or occlusion, subclavian steal syndrome by definition requires cerebral ischaemia initiated by ipsilateral arm exercise.[4,5] Retrograde blood flow in a vertebral artery is usually asymptomatic. Ischaemic upper limb symptoms occur after exercise involving elevation of limbs. Sometimes arm exercise exceeds the ability of collateral blood supply. Cerebral ischaemia may follow as blood is siphoned through the vertebrobasilar system.[6] Posterior circulation symptoms are common which include vertigo (> 50%), dysarthria and syncope (15%). Diplopia, transient visual loss, nystagmus occur secondary to vestibular dysfunction. This transient ischaemia progresses to infarction.[7-10]

Lifelong antiplatelet therapy is given to reduce risk of MI and stroke. Surgery is undertaken to restore antegrade blood flow in the vertebral artery and which includes endovascular catheter based therapy of the proximal subclavian artery which at present is the treatment of choice. Success rate of this Day Care procedure is above 85%. Primary stenting has superceded Balloon Angioplasty. Other treatment modalities include Endarterectomy [Less invasive extra thoracic bypass procedures have largely replaced it], Transposition [Subclavian artery is transposed to the common carotid artery by access from base of the neck with an end to side (subclavian to carotid) anastomosis thus not requiring a prosthetic graft], Extrathoracic carotid-subclavian bypass [A Dacron prosthetic graft is used making an end to side (subclavian to carotid) anastomosis].[11-15]

Our aim of this presentation is to highlight the uncommon subclavian steal, emphasise its clinical profile, the confirmation of diagnosis and its gratifying mode of treatment.

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