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Pathological Circumvention: A rare case of Subclavian Steal Syndrome causing posterior cerebral vascular ischemia, thence sparking off repeated Transient Ischemic Attack and Stroke

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Abstract

Background: Subclavian steal syndrome (SSS) is an uncommon but clinically significant vascular disorder caused by hemodynamic compromise in the vertebrobasilar circulation due to proximal sub-clavian artery stenosis. Its presentation with acute or subacute ischemic stroke remains rare, posing diagnostic and management challenges.

Case Presentation: An 80-year-old woman with hypertension and diabetes presented with three days of left leg weakness and slurred speech. Examination revealed left lower extremity weakness (3/5) and dysarthria. CT and MRI confirmed an acute/subacute ischemic lesion in the left basal ganglia. CTA demonstrated severe left subclavian stenosis and US Carotid Doppler Neck with near-complete reversal of vertebral artery flow, diagnostic of SSS, along with intracranial atherosclerotic disease. She was managed with dual antiplatelet therapy, high-dose statin, permissive hypertension, insulin adjustment, and early rehabilitation. Cardiovascular and thoracic surgery recommended subclavian angioplasty and stenting along with aggressive lifestyle modification.

Discussion: This case emphasizes the importance of comprehensive vascular imaging in elderly stroke patients, where atypical mechanisms such as SSS may contribute to ischemia. Management includes aggressive secondary prevention with dual antiplatelet therapy, statin use, strict blood pressure and glycemic control, and rehabilitation. In selected patients with hemodynamically significant stenosis and recurrent symptoms, subclavian revascularization is recommended to reduce future stroke risk.

Conclusion: Subclavian steal syndrome should be considered in stroke patients with diffuse atherosclerosis. Early recognition, multidisciplinary care, and timely revascularization are key to improving outcomes.

1. Background

Subclavian steal syndrome (SSS) is a vascular phenomenon where there is backward detour of the blood from the vertebral artery to the arms due to subclavian artery stenosis. Nevertheless, SSS does not usually produces any symptoms due to competent collateral circulation within the circle of Willis. Henceforth, this can be encountered as an incidental finding on routine workup without any clinical significance. SSS becomes pathologic when subclavian artery stenosis occurs in the backdrop of less than potent collateral circulation, thus triggering cerebral ischemia, Transient Ischemic Attack (TIA) and Stroke. SSS is more common in males as compared to females[1]. The age of presentation is older age groups, which is 59-61 years[1]. The increased occurrence of SSS in older age groups is interconnected with higher prevalence of risk factors for atherosclerosis, which are more common in these

populations[1]. The incidence and prevalence of SSS in the populations is around 2.5 and 5.4% respectively[2, 3]. Left subclavian artery stenosis is more prevalent than the right subclavian, although the reason for this preponderance is not exactly known. Here, we present a rare case of SSS in an 80-year-old women with risk factors who presented with TIA and stroke. Further workup revealed 70% stenosis in the subclavian artery with classical steal phenomenon.

2. Case Presentation

An 80-year-old female with PMH of hypertension (HTN) and Diabetes Mellitus (DM) presented to the Emergency Department (ED) with chief complaints of left leg weakness and slurred speech which progressively worsened over the preceding three days. She denied any preceding trauma, heart disease or similar episodes. Her medication list included Plavix, for a prior "TIA". Initial neurological examination in the ED revealed left lower extremity weakness (3/5 motor strength) and compromised, & slurred speech. Appropriate workup for an acute cerebrovascular event including a CT scan of the brain showed subtle low-density changes in the left basal ganglia, suggestive of subacute ischemia, without evidence of acute hemorrhage or acute ischemic changes. Given the 3-day duration of symptoms, acute stroke interventions such as intravenous tenecteplase or endovascular embolectomy were not feasible. Therefore, the patient was admitted to the inpatient service for further evaluation and management of a possible ischemic stroke.

Upon admission, the patient was initiated on a management plan including dual antiplatelet therapy (aspirin and clopidogrel) and high-dose statin therapy. Her home antihypertensive medications (amlodipine, losartan, hydrochlorothiazide) were continued with a target of permissive blood pressure initially, not exceeding 180 mmHg systolic, followed by a transition to a long-term goal of less than 130/80 mmHg. Blood glucose levels were closely monitored, and insulin glargine and insulin lispro were adjusted to optimize glycemic control. DVT prophylaxis with Lovenox was initiated. The patient was initially kept NPO until a swallowing evaluation could be performed by Speech Therapy. Rehabilitation was commenced early with Physical and Occupational Therapy consults placed for mobilization. Neurological assessments and vital signs were monitored regularly.

Further diagnostic workup included a brain MRI, which confirmed an acute or subacute ischemic lesion in the left basal ganglia with restricted diffusion, corresponding to the area of low density seen on the initial CT scan. TTE revealed a preserved ejection fraction of 65% and left ventricular hypertrophy but no valvular abnormalities. An EKG demonstrated sinus rhythm. A CTA of the head and neck was performed, uncovering extensive atherosclerotic disease. This included severe stenosis (>70%) at the origin of the left subclavian artery, resulting in subclavian steal syndrome confirmed by duplex ultrasound showing near-complete reversal of flow in the left vertebral artery. There was also a 50% stenosis in the left cervical internal carotid artery and significant intracranial atherosclerotic disease, including severe focal stenosis in a proximal left M2 branch, at the origin of the left A1 segment, and in the left P1 segment, along with multifocal moderate stenosis in the bilateral A2/A3 segments.

Given the complex cerebrovascular findings, interventional cardiology was consulted. Initially, there was consideration for medical management only if collateral circulation was deemed sufficient and vertebral artery flow in antegrade manner. However, the subsequent duplex ultrasound confirming near-complete reversal of flow in the left vertebral artery, combined with the recent stroke and severe intracranial stenosis, led the interventional cardiologist to recommend pursuing subclavian revascularization. Discussions regarding the risks, benefits, and alternatives of subclavian angioplasty & stenting are ongoing with the patient and her family.

3. Discussion

We present a textbook example of subclavian steal syndrome in an 80-year-old women with classic risk factors for atherosclerosis presenting with recurrent TIAs and stroke. Upon workup, she had a 70% and 50% stenosis of left subclavian and internal carotid arteries respectively, thus providing the deep-rooted basis for her basal ganglia stroke. High grade subclavian artery stenosis (>70% stenosis), SSS and cerebral ischemia warrants cost-effective percutaneous endovascular angioplasty with or without stenting, which offers 60% risk reduction as compared to conservative medical management[4].

Here briefly, we deep dive into the physiology of cerebral circulation and its transgression that ultimately forms the concealed basis for stirring up SSS. Subclavian artery originates from the aortic arch and later gives rise to vertebral artery [5]. Vertebral artery on

both sides conjoin to form basilar artery, which later bifurcates into two posterior cerebral arteries[5]. The posterior circulation of brain which predominantly includes posterior cerebral artery, basilar artery and vertebral artery supplies posterior cortex, midbrain and brain stem[5]. Physiologically, the blood moves in an anterograde manner from subclavian artery, to vertebral artery, and then into basilar artery and posterior cerebral artery, all of which entwine with the Circle of Willis[5]. Subclavian steal syndrome (SSS) occurs when there is a stenosis of subclavian artery, thus triggering retrograde flow of blood from the posterior cerebral circulation to the arm. Hemodynamically, the pressure gradient in the vertebral artery drops, thus stirring up the retroflux of cerebral blood circulation[6]. Pertinently, there is a swindling of blood from the contralateral vertebral or carotid arteries to the basilar and ipsilateral vertebral arteries, thus spurring diversion of blood to the ipsilateral arm and resultant cerebral ischemia [6]. This pathological and aberrant detour forms the underlying pathological basis of SSS.

The primary arteries effected in the SSS include subclavian arteries, vertebral arteries and coronary arteries [3, 7]. Atherosclerosis is the one of the primary and most important risk factor for the transpiration of SSS[3, 7]. On the grounds of this, hypertension, diabetes mellitus, smoking, alcohol hyperlipidemia, and old age can be presumed to be causative factors for inception of this clinical entity in most instances [7]. Rare and other plausible risk factors that are speculated to foment SSS include atherosclerosis, Takayasu arteritis, subclavian artery compression at the thoracic outlet, vertebral artery compression, cervical rib, surgical repair of coarctation of the aorta, and congenital anomalies of right aortic arch [3, 7].

According to Ossiro, S et al, SSS can classified three types based upon the severity of the disease process [7]. Grade I or pre-SSS is an attenuated anterograde flow[7]. Grade II or intermittent SSS where there is an anterograde flow in diastolic phase while retrograde flow in the systolic cycle[7]. Grade III advanced SSS where there is continuous retrograde vertebral flow[7]. Hemodynamic changes in the vertebral artery in SSS can be classified into reduced forward flow (grade I), backward flow during arm exertion (grade II) and consistent backward flow (grade III)[8]. Four types of SSS that can be present in patient include vertebro-vertebral, carotid-basilar, carotid-subclavian and external carotid-vertebral[9].

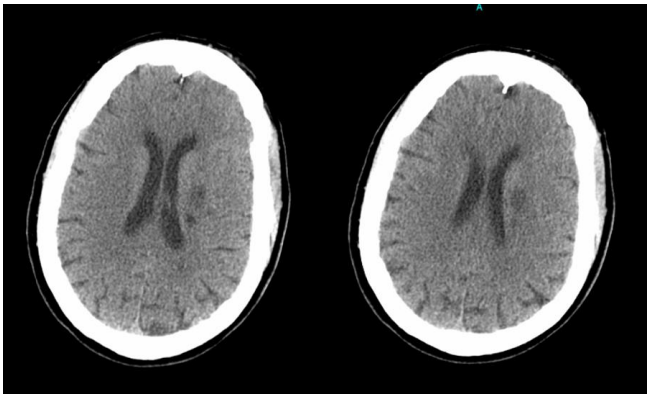

Figure 1: CT scan brain revealing Atrophy and Low density change left basal ganglia suggesting subacute ischemia

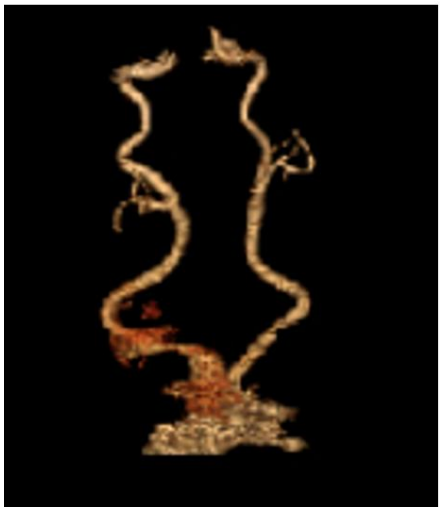

Figure 2: CT angiography (CTA) uncovering the following findings. a) No large vessel occlusion. b) 50% stenosis at the origin of the left cervical internal carotid artery. c) Severe, 70% plus stenosis at the origin of the left subclavian artery. D) Intracranial atherosclerotic disease including moderate bilateral supraclinoid ICA stenosis, severe stenosis in a proximal left M2 branch, severe stenosis in the left A1 segment anterior cerebral artery, multifocal moderate stenosis in the bilateral anterior cerebral arteries, and severe focal stenosis in the left P1 segment posterior cerebral artery.

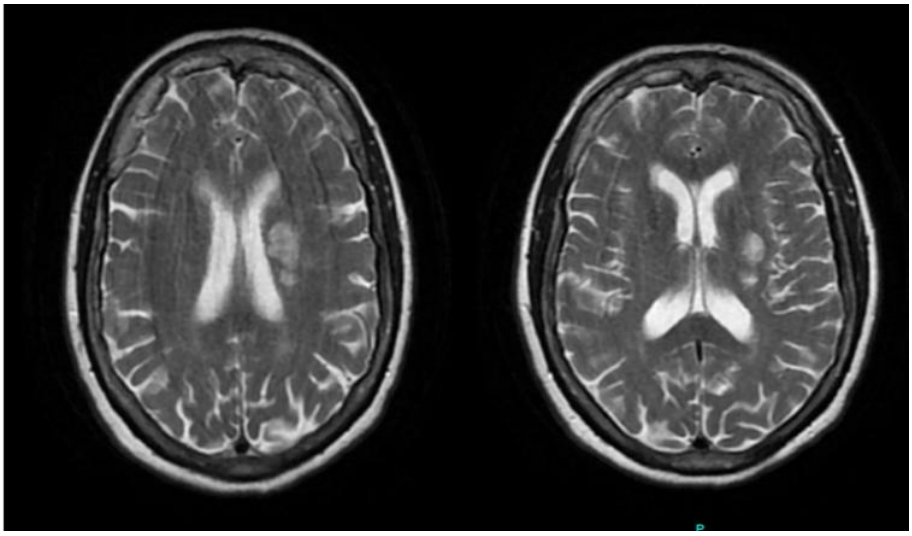

Figure 3: MRI brain showing focal area of acute or subacute ischemia left basal ganglia

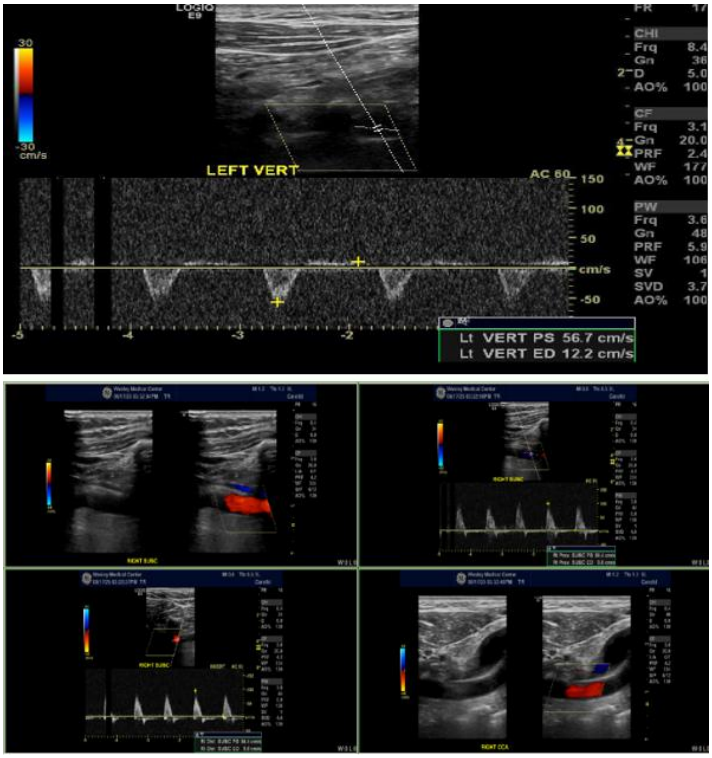

Figure 4: USG color Doppler - Since left subclavian stenosis is present, blood flow is coming from the right side and going to the left vertebral artery. The downward deflection in vertetral artery indicates the backward rerouting of blood into the arm vessels instead of supplying the brain. As this stealing of blood occurs in the context of less than adequate collateral ciruculation in the posterior brain, cerebral ischemia can be the ultimate result. This anamalous circumventing of blood can be instigating the transient ischemic attack and stroke in this patin

Habitually, this abnormal circumnavigation of blood does not cause symptoms, thus patients are usually asymptomatic. Primarily, the presence of subclavian stenosis in association with suboptimal collaterals in the posterior circulation precipitates the

mushrooming of cerebral ischemic symptoms [10]. Furthermore, excess arm exertion can command overzealous diversion of blood from vertebral circulation, thus instigating ischemia symptoms in the arm, brain and occasionally in the heart. SSS is interconnected with ischemia of brain sectors supplied by opposite vertebral or carotid artery in 35-85% cases[10]. That being said, SSS is pathological state of affairs, when this anomalous detour of blood is associated with symptoms of cerebral ischemia, vertebra-basilar ischemia, arm ischemia and coronary ischemia [6]. Cerebrovascular hemispheric ischemia (10%) from SSS can manifest with varied symptomatology ranging from hemi-anesthesia, hemiparesis, headache, dysphagia, visual field disturbances and confusion [6, 11]. Alternatively, SSS takes shape in form of vertebra-basilar ischemia (51-84%), thus showcasing vertigo, dizziness, giddiness, lightheadedness, syncopal attacks, loss of consciousness, diplopia, dysarthria, ataxia, numbness and nausea [6, 11]. In remote cases, SSS might divulge itself as arm ischemia, thus bringing to light weakness claudication, numbness and paresthesias (10-92%)[6, 11]. Occasionally, acute coronary syndrome presenting after coronary artery bypass graft (CABG) can bare face the lurking presence of coronary SSS (4.7%-21%)[11]. Presumably during CABG surgery, internal mammary artery is anastomosed with coronary artery. With this, coronary circulation is principally dependent on subclavian artery blood flow. Resultantly, subclavian artery stenosis can open the door for backflow of blood through the coronary artery bypass graft towards arm, thus impelling the onset of angina, and myocardial infarction [12, 13].

Pertinent history and physical examination is very important in aiding with diagnosis of SSS. Due to differential blood flow to both arms, evidence of higher blood pressure difference >40mm hg between both arms is highly associated with grade III advanced SSS [14, 15]. Doppler ultrasonography, Computed Tomography Angiography (CTA) and magnetic resonance angiography (MRA) will be essential for diagnosis and grading of SSS[7, 16, 17].

Patient with SSS with no symptoms or minimal isolated symptoms should be managed with conservatively with aggressive management of hypertension, hyperlipidemia, diabetes and smoking & alcohol cessation[7]. Thence, aspirin, beta blocker, angiotensin converting inhibitor (ACE) and atorvastatin will reduce the risk of atherosclerosis burden and inception of SSS [18, 19]. In the contrary, presence of SSS along with arm claudication vertebra-basilar or cerebral ischemic symptoms and angina with left internal mammary bypass, surgical approaches should be considered [20].

Endovascular approaches include percutaneous transluminal angioplasty, carotid endarterectomy and stenting might be used for severe SSS [6, 7]. If endovascular approaches are not successful then, extra-luminal approaches include carotid-subclavian bypass, carotid-subclavian transposition and axillo-axillary bypass could be tried [6, 7, 11, 21]. Out of these, common carotid artery tend to have a high success rate (>80%) as a bypass conduit compared to contralateral subclavian or axillary artery (46%) [22]. Following percutaneous transcutaneous angioplasty and stenting, the primary patency rate at 5 years is around 84% [23]. Out the patients treated with percutaneous angioplasty, approximately 10% will develop restenosis, which can be successfully managed with re-angioplasty [18]. Retrograde intraluminal endovascular approach followed by angioplasty stenting resulted in complete revascularization in symptomatic SSS [20]. Few studies report long-term patency rates and nil to low mortality during follow up of 8 years following bypass surgeries (20, 21)[24, 25]. According to Potter, B. et al, SSS patients with cerebrovascular symptoms, management of concomitant carotid artery stenosis prioritized before surgical intervention of SSS is planned [18].

4. Summary

SSS is a vascular phenomenon, where stenosis of the subclavian artery triggers an anomalous circumventing of the blood from posterior brain circulation to the arms due to drop in pressure gradient in the vertebral artery. This rerouting of blood precipitates diversion of blood from the contralateral to ipsilateral vertebral-basilar system, thus paving the way for cerebral ischemic symptoms. It is classified into three types (I, II and III) based on the severity of blood diversion. In most instances, classic risk factors for vessel atherosclerosis can be identified. It is usually asymptomatic, but can unfold with cerebral ischemia, vertebra-basilar ischemia. Although asymptomatic SSS can be managed with aggressive medical management, presence of significant symptoms with grade III SSS warrants surgical management through endovascular or surgical bypass. Close follow up with aggressive cardiovascular risk modification is the key to prevent further episodes of SSS.

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