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REVIEW ARTICLE

Unraveling the complexity: A comprehensive guide to subclavian steal syndrome

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A B S T R A C T

Subclavian steal syndrome is a diagnosis that is thought to be relatively uncommon even though its occurrence is not well understood. Some individuals may experience crippling symptoms of arm ischemia and vertebral-basilar insufficiency due to subclavian steal, which is more frequent than the accompanying illness. Patients with uneven arm blood pressure or unilaterally faint pulses should be evaluated for subclavian steal. Although it is not always necessary to check the blood pressure and pulse on both sides, they become significant when these measurements differ and are accompanied by vertebral-basilar or arm ischemia symptoms. Although subclavian steal does not significantly increase the risk of stroke, it is nevertheless essential to recognize and treat asymmetric blood pressure and weak pulses as these may be signs of subclavian steal syndrome. This page thoroughly analyses the aetiology of subclavian steal syndrome, signs, symptoms, diagnosis, and available treatments.

Keywords: *Vertebral-basilar, Subclavian steal syndrome, Stenosis, Blood flow.*

Introduction

There is widespread agreement that subclavian steal syndrome is a relatively unusual diagnosis, while the incidence of the condition is not well established in contemporary literature.[1] With an incidence rate of about 2.5%, subclavian steal syndrome (SSS) is a relatively uncommon disorder. Internal mammary artery (IMA) grafting in coronary artery bypass surgery might occasionally result in the unusual complication known as coronary subclavian steal syndrome (CSSS).[2] Myocardial ischemia caused by decreased blood flow, or flow reversal in the IMA graft, is the medical term for this condition. Most often, this is the result of proximal subclavian artery stenosis, which is hemodynamically severe. Unstable angina, myocardial Infarction, and even abrupt cardiac arrest are all possible clinical presentations. If one is not actively looking for it, CSSS is a problematic entity to diagnose. The clinical diagnosis is frequently challenging, and the disorder's frequency is commonly overestimated.[3] Clinicians have developed diagnostic and therapeutic standards that discriminate between asymptomatic and symptomatic subclavian steal syndrome. [4] The usefulness of subclavian steal syndrome in treating cerebrovascular illness was examined in the 1959 Joint Study of Extracranial Arterial Occlusion. As evidenced by arteriography, 17% of the 6,534 individuals in the study had subclavian or innominate artery occlusions or severe stenosis. Out of these, 168 people were diagnosed as having "true" subclavian steal syndrome.[5, 6]

Early in the 19th century, the idea that vertebral circulation could make up for a blocked subclavian artery was discovered. Harrison first acknowledged the significance of the vertebral-vertebral circulation in subclavian artery obstruction in 1829.[7] Smyth initially noted the reversal of blood flow in the vertebral vasculature in 1866 while treating a traumatic aneurysm with a subclavian artery ligation. In a patient who had no symptoms in 1960, Contorni reported a subclavian steal that had been

angiographically demonstrated. In the case of left subclavian artery obstruction, he documented collateral circulation in which the right vertebral artery was fully anastomosed with the left vertebral artery, causing a reversal of blood flow towards the left subclavian artery and total revascularisation.[8]

Reviech et al. reported cases resembling those Contorni described in 1961. Patients in these cases had vertebro-vertebral collateral circulation and had neurological symptoms such as headaches, scotomas, aphasia, and transient limb paralysis. The origin of these symptoms was determined to be cerebral ischemia brought on by a drop in pressure distal to the stenosis, which led to a decreased pressure gradient in the vertebral artery and a reversal of cerebral blood flow. The name "subclavian steal syndrome" was created by Fisher to describe this disease pattern in an editorial that was published the same year.[9, 10] Steal syndrome has been documented in medical literature since 1960. Blood flows in the opposite direction due to a diseased process, and if vessels supplying the intracranial structures are affected, various neurological symptoms may appear.[11]

According to the Joint Study of Extracranial Arterial Occlusion, Subclavian steal syndrome is more common in men than women, with an average age of 61 and 59 years. The subclavian and innominate vessels rarely have atherosclerotic occlusive disease, which is the cause of this. Additionally, the majority of patients with asymptomatic lesions don't also have concomitant carotid lesions. Additionally, the subclavian and innominate vessels are more slowly affected by atherosclerotic disease than other sites.[12]

ANATOMY:

The internal carotid and vertebral arteries make up the vascular supply of the brain. 80% of the blood flows to the brain through the internal carotid arteries, which divide into the middle and anterior cerebral arteries. [13] The vertebral arteries supply 20% of the blood flow, which unite to form the basilar artery and branch into the posterior cerebral arteries. The circle of Willis, a vascular ring that encircles the diencephalon, is made up of these arteries. When one of the significant blood veins becomes blocked, the circle of Willis assures blood flow between the vertebral-basilar and internal carotid systems.[14] Subclavian steal occurs when one of the subclavian or innominate arteries has stenosis or is blocked, resulting in a higher pressure gradient in the affected vessel and a decreased pressure gradient in the corresponding vertebral artery. The vertebral artery's blood flow is reversed as a result. The posterior cerebral circulation receives less blood due to this reversed flow, frequently called "stealing" (Figure 1). [15]

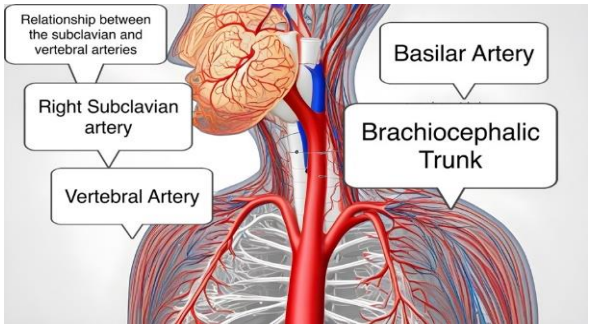

Figure 1: Relationship between subclavian and vertebral arteries[16]

PATHOPHYSIOLOGY:

The proximal subclavian artery can become stenotic or blocked, leading to retrograde flow in the ipsilateral vertebral artery and the subclavian steal phenomenon. The same phenomenon is called subclavian steal syndrome but with additional cerebral ischemia symptoms.[17] Reduced perfusion to the arm and hand on the afflicted side may follow, but symptoms depend on how well the intracranial collateral circulation functions. Patients with sufficient collateral circulation frequently exhibit no symptoms, whereas symptomatic patients may have abnormalities in other regions of the cerebral circulation. Interestingly, those with the left vertebral artery from the aortic arch are not susceptible to the left-sided subclavian steal syndrome.[12]

Blood flow from the brain to the arm is diverted in subclavian steal syndrome, which causes vertebrobasilar insufficiency symptoms (Figure 2). These symptoms are frequently brought on by strenuous arm movements or rapid head movements in the direction of the affected side. The syndrome can be brought on by arteriovenous distal arm shunt disorders or, less frequently, subclavian artery stenosis.[13] Three subclavian steal syndrome severity levels are Grade I: Reduced antegrade vertebral flow (pre-subclavian steal). Alternating flow with the antegrade flow

during the diastolic phase and retrograde flow during the systolic phase. Grade II (intermittent/partial). Retrograde vertebral flow that is persistent and progressive.[14, 15]

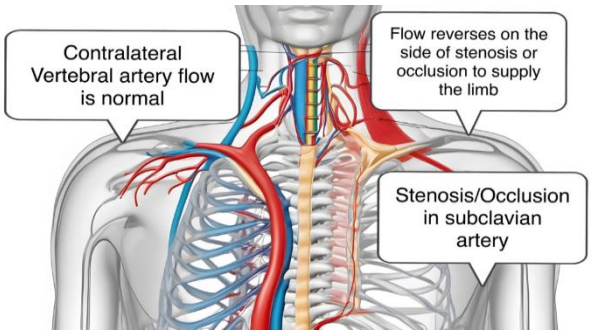

Figure 2: Subclavian Steal Syndrome [16]

CLINICAL PRESENTATION:

The subclavian steal syndrome is hemodynamically linked to proximal subclavian artery stenosis, blockage, and altered vertebral artery blood flow patterns. Retrograde blood flow in a vertebral artery typically doesn't result in symptoms. Except during strenuous arm work, proximal subclavian stenosis or occlusion rarely results in signs of arm ischemia. If arm exercise surpasses the capacity of collateral vessels, the brain may experience symptoms of cerebral ischemia. [17] Symptoms of posterior-circulation cerebral ischemia can include dysarthria, vertigo, syncope, and dizziness. Due to proximal subclavian stenosis or blockage, true subclavian steal syndrome necessitates retrograde flow in a vertebral artery. Blood pressures and pulses in the upper extremities can be used to identify significant subclavian artery lesions. Following surgery to graft the left internal mammary artery, recurrent angina may be a symptom of severe proximal left subclavian stenosis. Consider vertebral artery occlusive disease if posterior circulation symptoms appear with normal blood pressure in the affected arm.[18, 19]

DIAGNOSTICS:

A comprehensive physical examination, including a blood pressure reading and an evaluation of the radial pulse on the affected side, is crucial to determining the severity of subclavian steal syndrome. Additional information can be obtained by measuring the blood pressure in both arms and listening for carotid and supraclavicular bruits.[21] Regular laboratory tests, such as fasting lipid profiles and blood glucose levels, should be requested to evaluate atherosclerosis risk factors. Further evaluation may consider imaging tests like four-vessel cerebral arteriography, computed tomography angiography, duplex ultrasonography, and duplex ultrasonography. Electrocardiography and a thorough history taking are also crucial to recognize neurologic symptoms and arm ischemia. Diagnosed and risk factors for subclavian steal syndrome are evaluated in these tests. The most conclusive diagnosis is made by direct subclavian angiography (DSA). The routine use of DSA before CABG is debatable.[22]

Table 1: Subclavian Steal's Related Symptoms [20]

Vertebrobasilar	Arm Ischemia
Visual disturbances	Muscle fatigue
Vertigo	Claudication
Ataxia	Ischemic rest pain
Syncope	Ulcers
Dysphagia	Digital necrosis
Dysarthria	Atheroembolization
Sensory deficits of the face	
Motor and sensory deficits of the extremities	

A 20 mm Hg difference in systolic blood pressure between the contralateral arms raises the possibility of subclavian steal syndrome and calls for more testing. The afflicted side may have a decreased or nonexistent radial artery pulse. Although the existence of a supraclavicular bruit aids in the diagnosis, its absence does not always rule out the existence of the illness, and its presence does not always signify a major lesion. Nevertheless, the subclavian steal syndrome should be considered in light of these clinical findings.[23] The primary diagnostic procedure for assessing the carotid, vertebral, and subclavian arteries is ultrasonography, particularly duplex US. It can identify occlusive lesions in the carotid arteries and detect retrograde blood flow in the vertebral artery. The neck arteries are routinely examined with a Doppler US to diagnose subclavian steal syndrome. The vertebral artery exhibits early changes, including reduced velocity and biphasic flow, frequently exacerbated by arm activity or blood pressure cuff inflation. It can be challenging to evaluate the proximal subclavian artery; however, the distal subclavian artery often displays particular waveform

patterns. It's essential to comprehend these alterations to diagnose and treat subclavian steal syndrome.[20]

Subclavian artery stenosis or blockage is frequently found during the endovascular intervention. A delayed filling of the ipsilateral vertebral artery indicates insufficient blood supply to the brain and retrograde flow. [21] Other cerebral vascular lesions can also be found with this evaluation. It's crucial to look for severe lesions in the ipsilateral carotid artery. An arch aortogram may be necessary if the brachial artery pressure drops significantly (>20%) from the contralateral side. It has a high sensitivity and specificity and can detect additional abnormalities, so computed tomography angiography (CTA) is the first-line test. A four-vessel cerebral arteriography pinpoints the underlying anatomy of the issue and directs alternative solutions. A different option is magnetic resonance angiography (MRA). However, this method is more likely to exaggerate blockage and produce false-positive results.[17]

TREATMENT:

Because of the underlying atherosclerotic condition, subclavian artery stenosis is linked to higher morbidity and mortality rates. As a cardiovascular risk indicator, it increases the risk of cerebrovascular ischemic events.[7-11] Except in circumstances where a coronary artery bypass graft with an ipsilateral internal mammary artery graft is intended, incidental subclavian stenosis without symptoms does typically not require revascularisation therapy. Treatment focuses on secondary prevention strategies such as blood pressure control, dyslipidemia treatment, smoking cessation, diabetes glycemic control, and lifestyle modifications. Endovascular or surgical revascularisation is the preferred course of treatment, and percutaneous balloon angioplasty with stent support is recommended as the first-line procedure. Despite having a high percentage of success, the carotid-subclavian bypass has been less popular in recent years due to the minimally invasive nature of endovascular techniques.[18]

Treatments for subclavian artery stenosis that are successful and have good long-term results include balloon angioplasty and stenting. Surgical revascularisation, such as bypass surgeries, may be an option for lengthier or farther-reaching occlusions. Axillo-axillary bypass, on the other hand, is usually only used in high-risk patients because of its inferior patency rates.[9] Treatment options for patients with high perioperative risk and unfavourable anatomy include antiplatelet treatment and general cardiovascular preventive measures. Patients experiencing symptoms of subclavian artery stenosis should only be provided treatment, while those without symptoms typically do not need it. Antiplatelet therapy should be used permanently to treat subclavian steal syndrome brought on by atherosclerotic occlusions. Surgery or interventional treatment is advised if retrograde vertebral artery blood flow is the cause of ischemic symptoms. The efficiency of stenting in comparison to angioplasty alone is currently unknown.[10]

Less invasive bypass techniques have primarily replaced DeBakey's 1962 transthoracic approach to subclavian artery endarterectomy. Endarterectomy involves opening the artery, removing the plaque, unhealthy intima, and internal elastic lamina, and removing the occlusive lesions. The aortic arch must be partially occluded to guarantee thorough eradication of lesions. An anteromedial thoracotomy on the left side and a transverse incision on the right side are used to provide surgical exposure while avoiding a thoracotomy.[11-14]

Since subclavian endarterectomy has a mortality rate of 0.5%, extrathoracic carotid-subclavian bypass with a prosthetic conduit has taken its place. Utilizing Dacron or PTFE prosthetic grafts, the surgery calls for a transverse neck incision. An alternative treatment that avoids requiring prosthetic material but necessitates more extensive dissection is transposing the subclavian artery to a new origin on the common carotid artery.[12] When the ipsilateral carotid is unsuitable, an axillary-axillary or axillofemoral bypass can be done. A sequential retro oesophagal carotid-carotid and carotid-subclavian bypass can be performed in severe innominate artery disease cases. Arch aortography is done before surgery to evaluate the health of the distal subclavian and proximal common carotid arteries. An incision is made above the collarbone, the left thoracic duct is avoided, and a prosthetic graft with end-to-side anastomoses to the carotid and subclavian arteries creates a bypass. Postoperatively, brachial blood pressure is compared, and patients are evaluated for neurological impairments.[3-7]

Endovascular therapy is the most popular method for treating lesions of the proximal subclavian artery.

Comparison to open bypass or transposition reveals that it may produce results that are at least as excellent. Technical success rates are high (86–100%), and access site haemorrhage and plaque emboli are more common than nerve damage in terms of consequences. The majority of endovascular operations are performable as outpatients. Retrospective studies have shown that stenting is preferable to balloon angioplasty alone. Perfusion is improved, and subclavian steal syndrome is treated with endovascular recanalization and stenting. Archaortography is done before the procedure, and there are different ways to implant the guide wire. Proper location is crucial to prevent compression of adjacent vessels when using balloon-expandable stents, which are frequently employed. After the surgery, the patients are watched for a few hours while their neurologic condition and blood pressure are assessed.[21-23]

COMPLICATIONS:

Complications from surgical treatment can be cerebral (brain ischemia symptoms) or local (damage to nearby structures). Local problems are rare, although thrombosis or embolism can lead to cerebral issues. The use of a shunt is often not required since cerebral ischemia during common carotid closure is uncommon. The incidence of postoperative stroke ranges from 1.5% to 2.1%.[11] Complications from endovascular therapy can develop at the access site or target vessel. Hemostasis or access site haemorrhage are uncommon. Though less than 4% of instances include them, target vascular thrombosis, dissection, or distal embolization can occur. Transient ischemic episodes have been described in a small number of trials comparing endovascular treatment in patients with subclavian steno-occlusive disease in certain patients during the operation. Long-term recurrent interventions were needed for some patients. Primary 4-year stent patency rates varied, and patients with lengthier stents had a higher risk of problems.[5-8]

LONG TERM MONITORING:

Patients should follow up with doctors three to six months after treatment, then once a year after that. At every appointment, blood pressure in both arms should be measured. Duplex scans should be carried out to evaluate the reconstruction after six months and a year. Usually, patients with subclavian stents are given aspirin and clopidogrel for 6 to 12 months, then a single antiplatelet medication. Aspirin blocks prostaglandins' production, whereas clopidogrel explicitly blocks the binding of adenosine diphosphate (ADP) to platelet receptors. For patients who cannot tolerate or do not react to aspirin therapy, ticlopidine hydrochloride is an option.[9]

SUMMARY:

Subclavian steal syndrome is a very uncommon disorder marked by the reversal of blood flow in the vertebral artery due to stenosis or blockage of the proximal subclavian artery. It may result in vertebral-basilar insufficiency and signs of arm ischemia. Although the prevalence of subclavian steal syndrome is not well known, it is usually acknowledged as a rather unusual diagnosis. Upper-extremity blood pressure readings, pulse rates, and bilateral arm blood pressure measurements can all be used to identify SSS.

Conservative care and revascularisation therapies, including endovascular therapy or surgical bypass techniques, are available treatments for subclavian steal syndrome. The first-line choice is endovascular therapy, such as percutaneous balloon angioplasty with stent support, which has shown promising results in retrospective investigations. In some circumstances, less frequent surgical techniques like carotid-subclavian bypass or subclavian transposition may be explored. Regular follow-up visits, blood pressure tests, and duplex scans to evaluate the reconstruction are also part of long-term monitoring. Antiplatelet therapy may be used to treat patients with subclavian stents. Treatment-related complications are relatively uncommon but can occur and include thrombosis, hematomas, and bleeding at the access site.

In conclusion, the vertebral artery's blood flow is reversed in subclavian steal syndrome, a relatively uncommon disorder. It may result in vertebral-basilar insufficiency and arm ischemia symptoms. Clinical assessment, blood pressure readings, and imaging tests contribute to the diagnosis. Conservative management and revascularisation treatments are available as treatment options. Long-term monitoring is crucial in evaluating therapy effectiveness and identifying potential side effects.

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