Coronary Steal Syndrome — Definition and Symptoms

An incongruous reversal in the flow of blood in the form of coronary subclavian steal syndrome is a well-known complication of coronary artery bypass grafting (CABG). The condition can be discovered accidentally, during an ultrasonographic or angiographic examination performed for other reasons, or during a clinical examination, after a finding of reduced blood pressure or unilateral upper-limb pulse. Treatment mostly comprises open subclavian artery revascularization, normally via carotid-subclavian bypass or subclavian artery transposition, as well as other, less-invasive options such as endovascular intervention with recanalization, and angioplasty with stenting, if indicated.
Definition of Coronary Steal Syndrome

Coronary-subclavian steal syndrome is a **rare complication of CABG**. It usually occurs due to **stenosis of the left subclavian artery** which is proximal to the left internal mammary artery, compromising the myocardial blood flow.

It is mostly the result of long-standing subclavian stenosis that occurs due to the progression of the stenosis following CABG.

Epidemiology of Coronary Steal Syndrome

Coronary artery disease (CAD) and peripheral artery disease (PAD) prevalence, morbidity, and mortality rates are high in the developed world. The progression of atherosclerotic lesions is usually cited as the cause of CAD in patients who have a history of this condition and earlier coronary **revascularization** for angina pectoris.

Etiology of Coronary Steal Syndrome

Etiologic factors that can lead to stealing syndrome include **proximal subclavian artery occlusion** or **severe stenosis**. In the majority of cases, the disease results from atherosclerotic arterial disease, which is usually dominant on the left side. On the right side, disease or occlusion of the innominate artery can result in occlusion of the origin of the subclavian artery.

Risk Factors of Coronary Steal Syndrome

Coronary steal syndrome usually occurs when a patient is taking certain types of **vasodilator drugs** such as:

- Dipyridamole
- Adenosine
- Isoflurane
- Hydralazine
- Nitroprusside

**Risk factors** for developing atherosclerotic plaques are categorized as either modifiable or nonmodifiable. **Non-modifiable** risk factors include:

- Age
- Sex (more common in men)
- Family history of plaques

**Modifiable** risk factors include:

- Cigarette smoking
- Diabetes mellitus
- Hypertension
- Hyperhomocysteinemia
- Hypercholesterolemia

**Less common causes** include:

- Inflammatory arteriopathies such as **Takayasu arteritis** or **giant cell arteritis**
Pathophysiology of Coronary Steal Syndrome

Coronary-subclavian steal syndrome occurs via the **shunting of well-oxygenated blood from a critical area of low perfusion to an area of low perfusion**. It may be iatrogenic and can be in pharmacologic stress imaging by using dipyridamole to induce vasoconstriction. This leads to reduced blood flow to the subendocardium distal and to the site of the stenosed coronary artery.

In severe proximal subclavian artery stenosis or occlusion, **insufficient blood flow may be available to withstand the ipsilateral arm, resulting in low pressure in the distal subclavian artery**. Branches of the subclavian artery may begin to provide collateral reverse flow to the upper limb. Reversed blood flow in the vertebral artery assists as a collateral artery for the arm.

Through exercise, **distinctive and metabolite-induced vasodilation leads to a drop in peripheral resistance in the upper-limb vessels**. The gap between arterial inflow and metabolic demand may result in arm claudication. Additionally, when there is an increase in retrograde flow through the ipsilateral vertebral artery, it may “steal” blood away from the cerebral circulation.

Symptoms of Coronary Steal Syndrome

Coronary-subclavian steal syndrome is **characterized by retrograde flow of blood** from the left internal mammary artery to the left subclavian artery when a proximal left SA stenosis is present. It is **usually asymptomatic**. **When it becomes symptomatic, patients can experience:**

**When it becomes symptomatic, the patients mostly experience:**

- Upper-extremity symptoms such as pain in the arm, discomfort, or paraesthesia
- Neurological symptoms, which include dizziness, blurry vision, syncope.
- Relapse after initial improvement following CABG; it can be associated with the exercise of the left arm.
- Recurrent feeling of angina after CABG
- Examination may reveal a difference in the radial pulse volume and subclavian bruits.
- Vertigo — (most common presentation)

Physical examination may also reveal a difference in radial pulse volume and subclavian bruises.
Coronary-subclavian steal syndrome is usually suspected in patients who complain of recurrent angina pains after having undergone CABG with an internal thoracic artery graft. A bilateral preoperative blood pressure measurement should also be performed on these patients.

**Coronary steal syndrome can be diagnosed:**
- Detailed patient history
- Physical examination
- Intra-arm pressure difference reading
- Electrocardiogram
- Computed tomography angiogram
- Coronary angiography
- Stress testing with myocardial perfusion imaging

**Treatment of Coronary Steal Syndrome**

**Percutaneous Intervention**

Percutaneous intervention is considered the first line of treatment. It includes:

- Endovascular approach and percutaneous transluminal angiography
- Peripheral stenting

These treatment approaches have many advantages, including:

- Minimally invasive
- Shorter duration of stay in the hospital
- Lower chances of morbidity
- Avoidance of general anaesthesia
- Speedy recovery

**Surgical Treatment**

Surgical intervention may also be considered in some cases. Placement of a clip on
the anastomotic channel or on the bypass of the channel with a vein graft in order to induce occlusion may be performed. **ECA endarterectomy or angioplasty/stenting** is performed for external carotid artery stenosis/occlusion. Indications for the procedure this include:

- **Ipsilateral transient ischemic attacks** (either hemispheric or ocular) in patients with ipsilateral internal carotid artery occlusion and severe stenosis of the external carotid artery.
- **Ipsilateral internal carotid artery occlusion** and moderate stenosis of the external carotid artery, along with ulceration.
- **Ipsilateral transient ischemic attacks with a nonstenotic ipsilateral external carotid artery** and thrombus within the cul-de-sac of the occluded ipsilateral internal carotid artery.
- **Monocular amaurosis fugax** in patients with ipsilateral internal carotid artery occlusion and a microembolic source in the external carotid artery origin or the occluded carotid sinus.

When none of these criteria are present, however, there is no need for a patient to undergo this type of major surgical procedure.

**Revision using distal flow** is performed in cases of high-flow cardiac failure due to brachial arteriovenous fistula access. This method includes the closure of the anastomosis in the antecubital fossa and then the imposition of a graft between the forearm ulnar or radial artery. It effectively reduces blood flow by 50%.

**Proximalization of the arterial inflow** helps enhance access flow.

**Distal revascularisation with interval ligation** is a complex and time-consuming procedure. It is mostly possible when a suitable vein can be harvested.

**Prognosis of Coronary Steal Syndrome**

Long-term patency is significant, with nearly 90% of patients being free of **restenosis** after 4 years. Long-term patency is approximately 94%-97% at 20 months.

Endovascular approach with percutaneous transluminal angioplasty and stent placement in the subclavian artery has a very high success rate (80%-100%) with close to a 0% mortality rate. Most complications are due to distal embolization, which occurs in only 3%-6% of cases.

**References**


R. S. Bilku, S. S. Khogali, and M. Been, “**Subclavian artery stenosis as a cause for recurrent angina after LIMA graft stenting.**” Heart. 2003; 89(12): 1429. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1767990/


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