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Unexpected Stealing From the Heart

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Conflict of interest: None declared

Patient: Female, 74
Final Diagnosis: Coronary subclavian steal syndrome
Symptoms: —
Medication: —
Clinical Procedure: Balloon angioplasty
Specialty: Cardiology

Objective: Challenging differential diagnosis

Background: Coronary subclavian steal syndrome (CSSS) is defined as retrograde flow in the internal mammary artery graft, after coronary artery bypass surgery, resulting in anterior wall myocardial ischemia. If undiagnosed, it may lead to significant infarction. Its incidence has been under-reported.

Case Report: A 74-year-old woman presented with sudden onset of typical angina, which was associated with dyspnea, nausea, and vomiting. Her medical history was significant for coronary artery disease, with prior coronary artery bypass surgery (CABG), chronic obstructive pulmonary disease (COPD), hypertension, diabetes type 2, dyslipidemia, hypothyroidism, and depression. Physical examination demonstrated a weak left radial pulse, and a differential blood pressure reading in her arms. She developed massive hematochezia that resulted in electrocardiogram changes: ST segment depression in the inferior and lateral leads with isolated ST segment elevation of aVR. Cardiac catheterization demonstrated retrograde flow through the left internal mammary artery (LIMA), resulting in CSSS.

Conclusions: This case highlights the diagnostic challenge of CSSS as the underlying etiology for ischemia in a patient with remote coronary artery bypass surgery (CABG). It underscores the importance of thorough physical examination in patients who are at high risk for coronary atherosclerosis and remote complications after CABG. It also highlights optimal strategies for management of CSSS with percutaneous transluminal angioplasty.

MeSH Keywords: Angioplasty, Balloon, Coronary • Coronary Artery Disease • Coronary-Subclavian Steal Syndrome

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/895498v

Background

Coronary Subclavian Steal Syndrome involves retrograde flow through a patent internal mammary graft leading to myocardial ischemia. This is secondary to occlusion of the proximal left subclavian artery. High-risk patients present remotely after coronary artery bypass grafting, with vague symptoms such as left arm pain, variations of syncope, and dizziness, but may present with angina and active ischemia if there is coronary flow compromise.

Case Report

A 74-year-old woman presented to the Emergency Department with a 4-day history of typical chest pain, associated with shortness of breath, anorexia, nausea, and vomiting. Her past medical history was significant for coronary artery disease, with 2-vessel CABG (left main stem, and circumflex) 4 years prior to presentation, chronic obstructive pulmonary disease (40 pack-year smoking history), hypertension, diabetes mellitus type 2, dyslipidemia, hypothyroidism, and depression. She denied alcohol or illicit drug use. Physical examination demonstrated a weak left radial pulse, a blood pressure reading in her right arm of 148/70mmHg and left arm of 98/5 3mmHg, heart rate 86/min, and respiratory rate 20/min. She was afebrile, with an oxygen saturation of 90% on 2 L/min by nasal cannula. She appeared anxious, in mild respiratory distress, with nasal turbinate hyperemia. Cardiac examination revealed a regular rate and rhythm, and there were no carotid bruits, gallops, rubs, or murmurs. Respiratory examination revealed diminished breath sounds and dry, end-inspiratory bibasilar crackles, with minimally audible wheezing. She had a cool, dusky left upper arm, with faint pulsation in left radial pulse. Distal pulses were symmetrical and synchronous. Neurological examination was intact. Laboratory data revealed: complete blood count, hemoglobin (Hgb) 12.6 g/dl, hematocrit (Hct) 38, normal metabolic panel except serum urea 32 mg/dl, creatinine 2.3 mg/dl, anion gap 12, CPK 614 U/liter, myocardial fraction 7.5%, and basic natriuretic peptide (BNP) 182 pg/ml. D-dimer was 605 ng/ml. Serial cardiac markers were normal. Influenza A and B were negative. Sputum gram stain and culture were negative. Electrocardiogram (ECG) showed sinus rhythm, with diffuse ST segment depression in inferolateral leads, which were more prominent than in the previous ECG, and a 1-mm segment elevation in aVR, similar to previous EKGs. Chest radiograph revealed slight pleural thickening on the left upper lung, and interstitial lung prominence, with no radiographic evidence of pneumonia or pulmonary edema. There was no widening of the mediastinum. Echocardiography revealed normal left ventricular ejection fraction of 60–65%, without valvular abnormality. She was treated for non-ST elevation myocardial infarction, COPD exacerbation, and acute kidney injury.

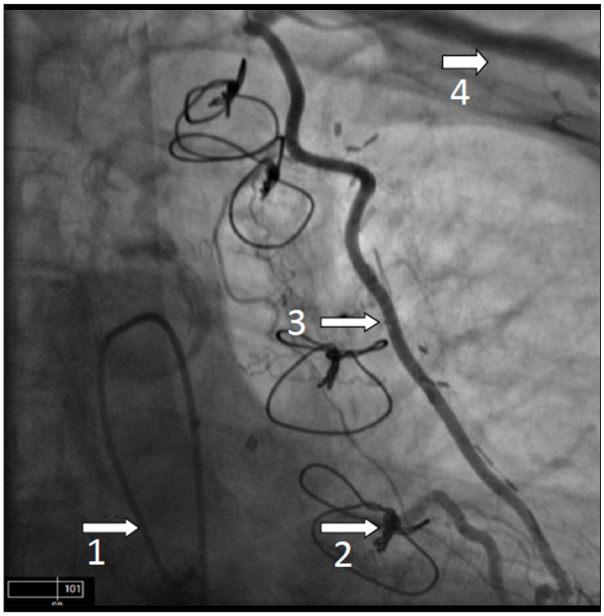

Figure 1. Left coronary just following end-of-contrast injection showing retrograde flow through LIMA graft and fully opacified distal left subclavian. 1. Five French left Judkins diagnostic catheter, 2. End of contrast column, 3. LIMA graft showing complete opacification, 4. Distal left subclavian artery showing opacification. Reproduced with permission from Dr. James Omeara III. Cardiac catheterization report reproduced with permission from North Florida Regional Medical Center, Catheterization Laboratory, Gainesville, Florida.

She received heparin infusion, aspirin 81 mg daily, clopidogrel 75 mg once daily, simvastatin 40 mg once daily, and metoprolol 25 mg twice daily. She received the remainder of acute coronary syndrome guideline management.

She received ceftriaxone 1 gram intravenously twice daily and azithromycin 500 mg intravenously daily, with scheduled albuterol/ipratropium nebulizations and prednisone 20 mg daily for COPD exacerbation, with presumptive atypical pneumonia. During hospitalization, she continued to wheeze and BNP increased to 1000 pg/ml. Her hospitalization was complicated by massive hematochezia. She became hypotensive and required aggressive fluid resuscitation and transfusion with 5 units of packed red blood cells. Her hemoglobin fell from 12.6 g/dl to 8.6 g/dl during her bleeding episode. Heparin and clopidogrel were discontinued. Serial cardiac markers trended upward to 0.23 ng/ml, with ECG demonstrating more prominent ST segment elevation in aVR and V1, and contiguous ST segment depression in precordial, inferior, and lateral wall leads. This progressed to new-onset atrial fibrillation, with rapid ventricular response of 120–140 beats/minute. Upper and lower endoscopy studies were unremarkable for obvious etiology of bleeding. She was appropriately volume-resuscitated, but remained hemodynamically unstable. Her atrial fibrillation converted to

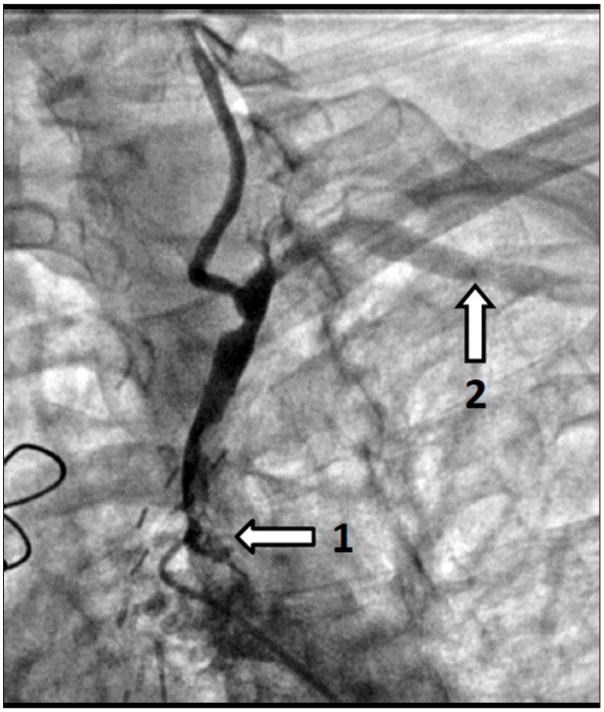

Figure 2. Left subclavian pre-intervention. 1. Subtotal occlusion of proximal left subclavian, 2. Poorly opacified distal left subclavian due to non-contrasted blood from the LIMA graft. Reproduced with permission from Dr. James Omeara III, Cardiac catheterization report reproduced with permission from North Florida Regional Medical Center, Catheterization Laboratory, Gainesville, Florida.

sinus rhythm after amiodarone infusion (refractory to diltiazem and metoprolol). Cardiology was consulted with a view to proceed with cardiac catheterization. Her massive gastrointestinal bleeding precluded anticoagulation. Coronary angiography revealed: left ventricular ejection fraction at 70%, the left anterior descending (LAD) artery had 70% ostial stenosis, and a patent LIMA graft was noted in the mid-LAD that filled in a retrograde fashion up to the left subclavian artery during injection of contrast medium into the left coronary artery (Figure 1). The circumflex artery was anatomically non-dominant, with 70% ostial stenosis. The right coronary artery was anatomically dominant, without significant disease.

Left subclavian angiography revealed a 95% occluded proximal left subclavian artery, with retrograde filling via the anterior wall and LAD (Figure 2). The body of the internal mammary artery graft to the LAD was free of significant disease. Vein graft angiography to the ramus intermedius artery revealed a well constructed vein graft without significant disease. Following this finding, percutaneous transluminal angioplasty was performed; a 6×40-mm balloon yielded a suboptimal result; therefore, a 7×30-mm expandable stent in the left subclavian artery was deployed at 12 atmospheres. A 7×20-mm post-dilatation

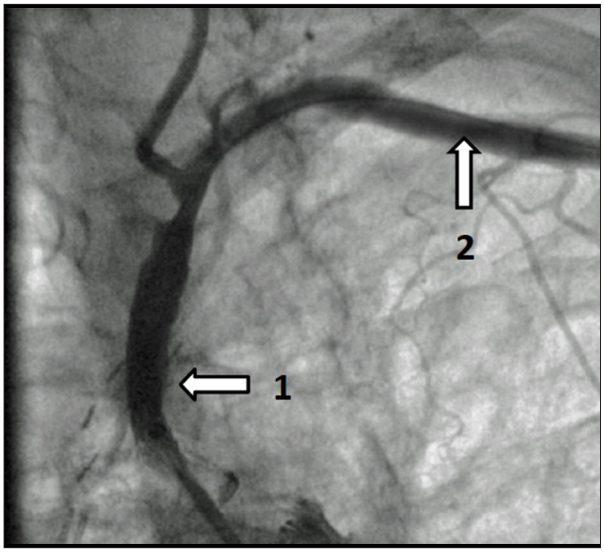

Figure 3. Left subclavian following successful intervention. 1. Balloon expandable stent, 2. Fully opacified distal left subclavian and early opacification of LIMA indicating return to normal flow pattern. Reproduced with permission from Dr. James Omeara III. Cardiac catheterization report reproduced with permission from North Florida Regional Medical Center, Catheterization Laboratory, Gainesville, Florida.

balloon was then utilized to dilate the proximal half of the stent to 14 atmospheres. This produced an excellent angiographic result with 20% residual narrowing (Figure 3). There was no evidence of aortic dissection or distal embolization. The patient showed clinical improvement thereafter, denying angina, dyspnea, and syncope. At the follow-up visit with Cardiology, clopidogrel was resumed 30 days after her bleeding episode. She has subsequently stopped smoking, and remains symptom-free. Capsule endoscopy performed 1 month after her surgery demonstrated a remote, small intestinal bleeding site.

Discussion

This case highlights the diagnostic challenge of CSSS presenting as acute myocardial infarction in a patient after CABG. CSSS can present with an absent or diminished ipsilateral radial pulse, supraclavicular thrill or bruit, and a differential blood pressure reading greater than 20 mmHg between upper extremities [1]. In this case, the reversal of blood flow was potentially aggravated by massive gastrointestinal blood loss leading to acute myocardial infarction.

The use of the LIMA is associated with long-term patency but the proximal portion of the subclavian artery is a harbinger of atherosclerosis. This is secondary in part to the more prominent angle at its origin, where turbulent flow accelerates

atherosclerosis, accounting for 80% of cases [1]. The most frequent cause of the syndrome is atherosclerosis in the ipsilateral, proximal subclavian artery after revascularization in CABG. The incidence of CSSS is about 0.2% to 0.68% of patients referred for CABG surgery with LIMA, but its prevalence is rising as a result of the increased use of LIMA for CABG procedures. Other studies report an incidence of 0.44% as compared to that of subclavian steal syndrome at 5.23% [2,3]. Less frequent causes of left subclavian stenosis include Takayasu, giant cell, and radiation-induced arteritis. There is also a documented case secondary to an arteriovenous hemodialysis fistula in the left arm [2].

Diagnosis of CSSS is supported by noninvasive imaging modalities, such as echo-color Doppler sonography, computed tomographic angiography, and cardiac magnetic resonance angiography of the aortic arch or left subclavian artery. This is done prior to primary percutaneous coronary intervention to avoid potential diagnostic pitfalls [2,3].

Percutaneous transluminal angioplasty offers a less invasive strategy for management but in certain cases this is not possible due to chronic near-occlusion. Carotid-to-subclavian bypass with a polytetrafluoroethylene (PTFE) graft is an alternative strategy used in more difficult cases. CSSS occurs at between 2 and 31 years after CABG (average, 14 years). Since 1990, percutaneous transluminal angioplasty with stent implantation has been considered an effective treatment. However, in certain cases that preclude anticoagulation, such as recent massive GI bleed, other modalities such as balloon angioplasty may be considered. Ferrara reported a high incidence restenosis (40.7% over 5 years) in patients who undergo carotid-subclavian bypass [4]. Percutaneous transluminal angioplasty was the preferred method of treatment for CSSS in a literature review [5].

Conclusions

CSSS often presents a diagnostic challenge. Our patient denied left arm pain, dizziness, or syncope, but the finding of a cool left upper arm with faint pulsation and differential blood pressure reading should have triggered further investigation. Unfortunately, treating her NSTEMI led to complications of acute gastrointestinal bleeding and sudden volume depletion, which exacerbated a reversal in blood flow through the conduit of LIMA.

The spectrum of ischemia in CSSS can be provoked by volume depletion, resulting in a range of symptoms from unstable angina to acute STEMI. This may reflect either type 1 or type 2 myocardial infarction (MI). Acute MI in the setting of CSSS is most likely due to type 2 MI, as per the recent MI classification, and not to an ulcerated or complicated atherosclerotic plaque [6]. For clarification, type 2 MI is secondary to relative ischemia from increased oxygen demand or decreased oxygen supply such as in the case of anemia, tachy-arrhythmia, coronary artery spasm, coronary embolism, hypertension, and/or hypotension [7].

Acknowledgements

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