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MINI-FOCUS ISSUE: INTERVENTIONS

INTERMEDIATE

CASE REPORT: CLINICAL CASE

A Unique Case of STEMI STEALing the Flow

Asra Khalid Butt, MD,[a] Nureddin Almaddah, MD,[b] Qasim M. Mirza, MD,[c] Uzoma N. Ibebuogu, MD[b]

ABSTRACT

We report a case of a 55-year-old woman with previous coronary artery bypass grafting. She presented with acute ST-segment elevation myocardial infarction secondary to occlusion of the proximal left subclavian artery causing reduced flow in the left internal mammary artery to left anterior descending artery graft. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2020;2:2419–23) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 55-year-old African-American woman with hypertension, hyperlipidemia, coronary artery disease (CAD), and a history of a 2-vessel coronary artery bypass graft (CABG) presented to the emergency department with reports of chest pain for 1 day.

The patient described the pain as crushing, substernal, and nonradiating. She reported shortness of breath that started with the chest pain. She denied any cough, hemoptysis, dizziness, syncope, palpitations, or edema. She had smoked occasionally in the past but quit when she underwent CABG 20 years ago. She rarely consumed alcohol and never used any illicit drugs. She had a family history of hypertension, hyperlipidemia, and CAD. On examination, the patient was afebrile, with a heart rate of 110 beats/min, respiratory rate of 18 breaths/min, and oxygen saturation of 95% on room air. The systolic blood pressure in the left arm was 69 mm Hg and 112 mm Hg in the right arm. She appeared anxious and clammy. She had regular tachycardia with no murmurs, rubs, or gallops. Her lungs had fine rales at the bases. Jugular venous pulse was elevated, and there was no pedal edema. The left arm was noted to be cooler and with a diminished pulse compared with the right arm.

LEARNING OBJECTIVES

- To highlight the importance of understanding the vascular anatomy in the setting of prior CABG.
- To recognize CSSS as one of the causes of myocardial infarction in a patient with a previous CABG with a LIMA to LAD graft.

MEDICAL HISTORY

The patient had a medical history of hypertension, hyperlipidemia, CAD, and ischemic cardiomyopathy. She had undergone a 2-vessel CABG 20 years ago with bypass grafting of the left internal mammary artery to left anterior descending artery (LIMA to LAD) and saphenous venous graft to right coronary artery for symptomatic CAD.

From the [a]Department of Internal Medicine, University of Tennessee School of Medicine, Memphis, Tennessee, USA; [b]Section of Cardiology, University of Tennessee School of Medicine, Memphis, Tennessee, USA; and the [c]Section of Pulmonary & Critical Care Medicine, University of Tennessee School of Medicine, Memphis, Tennessee, USA.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass grafting

CAD = coronary artery disease

CSSS = coronary subclavian steal syndrome

CT = computed tomography

ECG = electrocardiogram

LAD = left anterior descending artery

LIMA = left internal mammary artery

A coronary angiogram had been performed 2 years before the current presentation for atypical chest pain. It showed chronically occluded native coronary arteries with an occluded saphenous venous graft to right coronary artery and a reduced left ventricular ejection fraction of 40%. The LIMA to LAD graft was widely patent, and coronary perfusion was dependent on this graft. She had been prescribed dual antiplatelet therapy but was noncompliant.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis included coronary subclavian steal syndrome (CSSS), aortic dissection, and acute myocardial infarction.

INVESTIGATIONS

The initial electrocardiogram (ECG) (Figure 1) showed no new changes. Troponin levels were minimally elevated at 0.7 ng/ml. She was started on intravenous heparin for non-ST-segment elevation myocardial infarction. A computed tomography (CT) scan of the chest with contrast showed thrombosis of the proximal left subclavian artery propagating into the arch of the aorta (Figures 2 to 4). There was no contrast seen in the LIMA to LAD graft. Shortly after, the patient developed increased chest pain and acute respiratory distress requiring intubation and mechanical ventilation. A repeat ECG (Figure 5) showed dynamic changes with ST-segment elevation in leads V_4 to V_6. A chest radiograph revealed acute pulmonary edema. Repeat measurement of troponin levels showed they had increased to 3.4 ng/ml. Cardiology and Vascular Surgery were consulted.

MANAGEMENT (MEDICAL/INTERVENTIONS)

Options for percutaneous coronary intervention were limited because native coronary vessels were chronically occluded. An endovascular approach to the left subclavian artery was deemed to be unsafe as there was an increased risk of the clot breaking up within the aortic arch and embolizing distally. A joint decision was therefore made by Cardiology and Vascular Surgery to proceed with an emergent left common carotid artery to left subclavian artery bypass. The patient was taken to surgery within 1 h of the diagnosis of acute ST-segment elevation myocardial infarction.

Intraoperatively, a 6-mm standard wall thickness polytetrafluoroethylene graft was used to create a bypass between the left common carotid artery and the left subclavian artery. The completion arteriogram showed a widely open bypass with antegrade flow within the left vertebral artery and the left internal mammary graft that filled the left anterior descending coronary artery.

FIGURE 1 Initial Electrocardiogram on Presentation Showed Normal Sinus Rhythm With No ST-Segment Changes

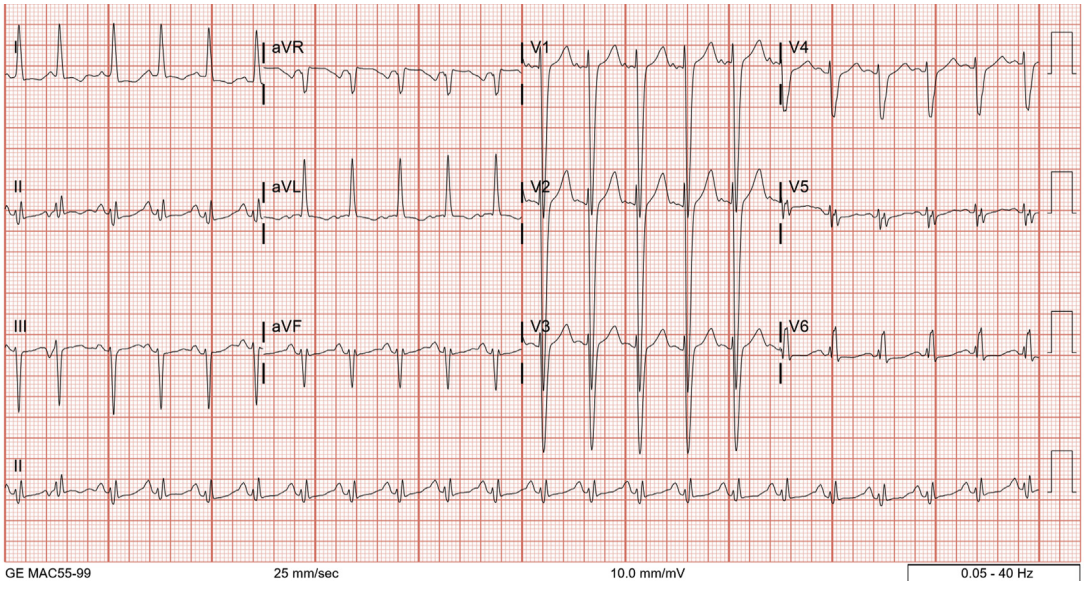
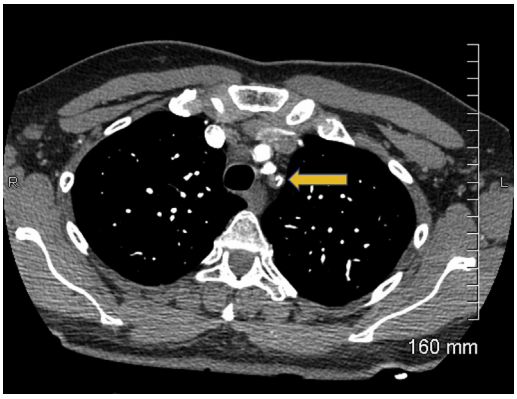


FIGURE 2 Transverse Section of Computed Tomography of Chest With Contrast



Transverse section of computed tomography of chest with contrast showing left subclavian artery thrombosis **(marked by an arrow)**.

DISCUSSION

Subclavian artery occlusion or stenosis proximal to the origin of the vertebral artery can lead to a myriad of clinical presentations, including an asymptomatic incidental finding, claudication of the upper extremity, subclavian steal syndrome with vertebrobasilar insufficiency presenting as vertigo, diplopia, and ataxia secondary to decreased or reversed flow in ipsilateral vertebral artery, or CSSS in patients with a previous CABG with a LIMA to LAD graft. Features of CSSS include angina or myocardial infarction resulting from reduced flow in the LIMA, a branch of the second part of the subclavian artery (1).

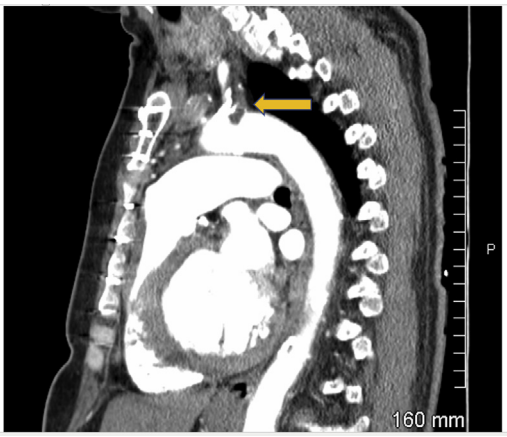
Both non-ST-segment and ST-segment elevation myocardial infarction have been described with CSSS, although the latter is much rarer (2–5). The initial diagnosis of CSSS is usually by noninvasive imaging with duplex ultrasound, CT angiography, or magnetic resonance angiography. Invasive testing with angiography confirms the diagnosis.

Indications for treatment of subclavian artery occlusion include upper arm ischemia, digital embolization, vertebrobasilar insufficiency from subclavian steal syndrome, or myocardial ischemia from CSSS.

Treatment of CSSS involves revascularization of the subclavian artery to restore LIMA flow and can be achieved by percutaneous or surgical interventions (carotid-subclavian bypass, subclavian-carotid transposition, or axillo-axillary bypass) (6).

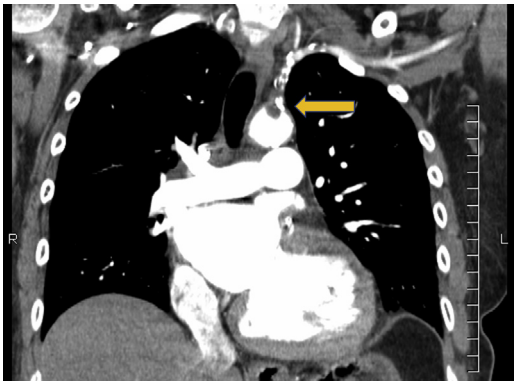
Review of the literature comparing nonsurgical endovascular intervention with surgical bypass of subclavian artery occlusion shows that technical success and short-term patency are similar for both.

FIGURE 3 Sagittal Section of Computed Tomography of Chest With Contrast



Sagittal section of computed tomography of chest with contrast showing left subclavian artery thrombosis with extension into the aortic arch **(marked by an arrow)**.

FIGURE 4 Coronal Section of Computed Tomography of Chest With Contrast



Coronal section of computed tomography of chest with contrast showing left subclavian artery thrombosis with extension into the aortic arch **(marked by an arrow)**.

FIGURE 5 Subsequent Electrocardiogram Showing New ST-Segment Elevation in Leads V_4 to V_6 Consistent With ST-Segment Elevation Myocardial Infarction

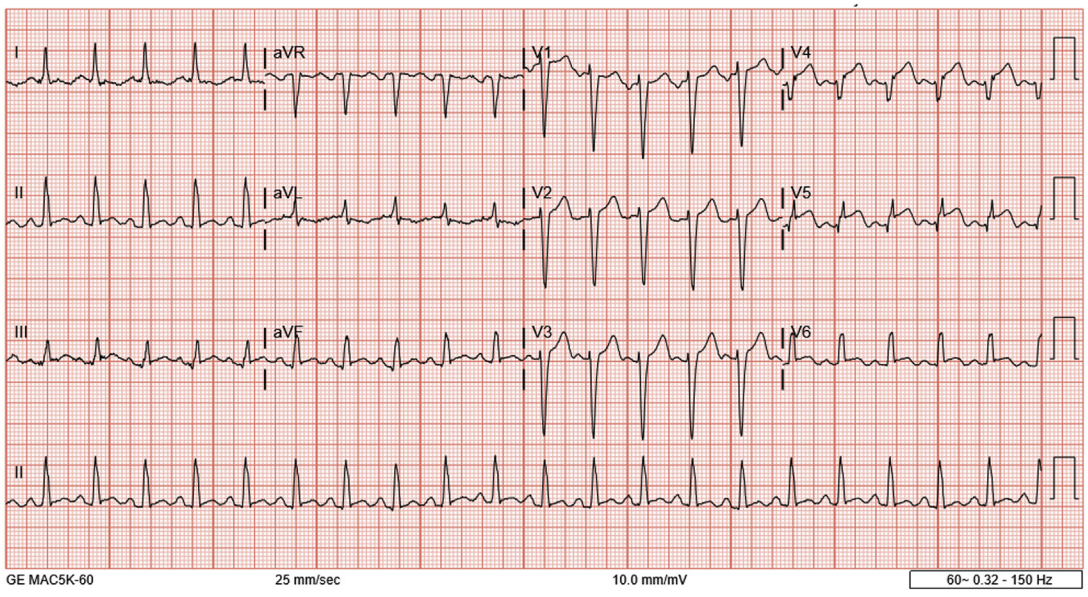
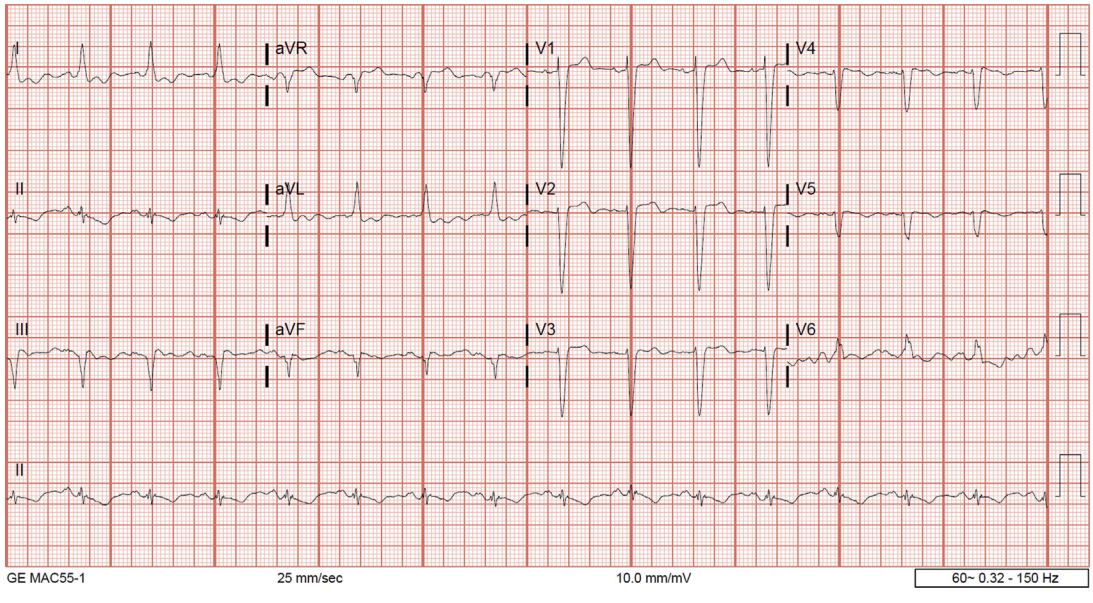


FIGURE 6 Electrocardiogram Performed 4 Days After Surgery Shows Resolving ST-Segment Elevation in Leads V_4 to V_6

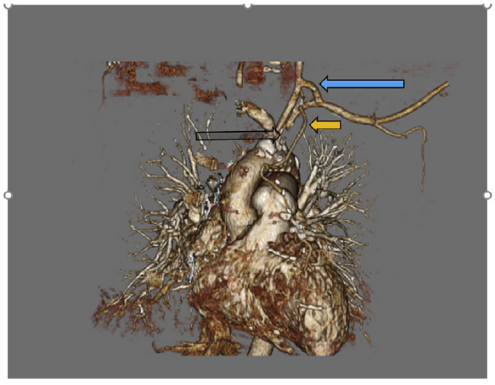


The incidence of complications such as stroke, restenosis, or death is lower with endovascular techniques; however, long-term outcomes are better with surgical bypass (6).

FOLLOW-UP

Clinically, the patient improved over the next few days. Blood flow in the left upper extremity was

FIGURE 7 3-Dimensional Reconstruction of Computed Tomography of Chest Performed 1 Month After Hospital Discharge



3-dimensional multiplanar reconstruction of computed tomography of chest performed 1 month after hospital discharge shows there is continued occlusion of the proximal left subclavian artery **(transparent arrow)**. The left internal mammary artery to left anterior descending artery graft is patent **(yellow arrow)**. Just distal to the origin of the left internal mammary artery graft, the left common carotid to left subclavian artery bypass graft is present **(blue arrow)**. The left subclavian artery distal to the occlusion is patent.

restored with equal pulses in both upper extremities. ST-segment elevation on the ECG resolved (Figure 6). An echocardiogram performed on the day after surgery showed a reduced left ventricular ejection fraction of 20%, which returned to baseline after 2 months. A subsequent CT scan of the chest (Figure 7) performed 1 month later showed patency of the left carotid artery to the left subclavian artery bypass graft, the LIMA to LAD graft, and the distal subclavian artery.

CONCLUSIONS

This case illustrates the importance of understanding the anatomy of the LIMA with its origin from the first part of the left subclavian artery. Occlusion of the proximal subclavian artery can cause reduced flow in a LIMA to LAD graft and result in myocardial ischemia. Diagnosis is often challenging, and prompt recognition can be lifesaving.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr. Asra K. Butt, University of Tennessee Health Sciences Center, 956 Court Avenue, Suite A312, Memphis, Tennessee 38163, USA. E-mail: akhalid2@uthsc.edu.

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KEY WORDS coronary artery bypass, myocardial infarction, thrombosis