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MINI-FOCUS ISSUE: CORONARIES

CASE REPORT: CLINICAL CASE

Acute Myocardial Infarction With Cardiogenic Shock Due to Pericardial Constriction and Multivessel Coronary Obstruction



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ABSTRACT

We present a rare case of cardiogenic shock and multivessel coronary compression due to focal pericardial inflammation and constriction. The patient was treated in the acute phase with coronary stenting and temporary mechanical support. Multimodality imaging was essential in elucidating the diagnosis. (Level of Difficulty: Beginner.) (J Am Coll Cardiol Case Rep 2020;2:1708-12) © 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/).

62-year-old woman awoke from sleep with acute-onset chest pressure. In the emergency department, her blood pressure was 83/ 63 mm Hg, and an electrocardiogram (ECG) showed ST-segment elevation consistent with an inferoposterolateral myocardial infarction (Figure 1).

LEARNING OBJECTIVES

- To learn about the entity of coronary compression due to focal pericardial constriction and fibrous bands.
- To learn the role of multimodality imaging in diagnostic dilemmas.
- To understand a potential late complication of pericarditis treated with partial pericardiectomy.

PAST MEDICAL HISTORY

There was history of rheumatoid arthritis and an episode of pericarditis 4 years prior, with pericardial tamponade requiring a pericardial window. There were no coronary artery disease risk factors.

DIFFERENTIAL DIAGNOSIS

Because of diffuse ST-segment elevations on the ECG and shock, the patient was brought emergently to the cardiac catheterization lab.

INVESTIGATIONS

Coronary angiography revealed acute total and subtotal occlusions of the left anterior descending artery, first diagonal, and 3 obtuse circumflex branches

Manuscript received November 7, 2019; revised manuscript received April 25, 2020, accepted May 6, 2020.

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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 *JACC: Case Reports* author instructions page.

(Central Illustration). The coronary occlusions were in the mid to distal portions of each vessel, linearly at nearly identical distances from the left main. There was no evidence of thrombus or of intracoronary plaque. The 5 narrowed coronary artery segments resembled spasm, yet there was no response to intracoronary nitroglycerin. There was a dynamic component to the occlusions with the disappearance of several coronary artery segments only during systole (Video 1).

MANAGEMENT

Because of cardiogenic shock, a transvalvular left ventricular support device (Impella CP, Abiomed, Danvers, Massachusetts) was placed, and the patient was intubated. She underwent stenting of the LAD and obtuse marginal (OM) 2 and balloon angioplasty of OM1. Stented segments remained patent, but the ballooned vessel continued to exhibit dynamic narrowing after angioplasty, as did the untreated OM3 and diagonal branches.

The patient was transferred to the cardiac care unit, where she was extubated and weaned from mechanical support after 1 day. Twenty-four hours later, she complained of recurrent chest discomfort, which was associated with new ST-segment elevations. She was taken back for cardiac catheterization and underwent stenting of OM1 (Figure 2), upon which her symptoms improved and STsegments normalized. Left ventriculography demonstrated mild to moderate left ventricular dysfunction with apical and anterolateral akinesis. Peak troponin I level was 8.9 ng/ml.

After stabilization, echocardiogram showed asymmetric pericardial thickening and a wall motion abnormality in the posterolateral wall (Figure 3). Because of concern that there was extrinsic pericardial scarring

affecting coronary flow, coronary computed tomography angiography was performed, which demonstrated pericardial thickening and coronary stents that were bent at acute angles as if externally compressed (**Figure 4**). Cardiac magnetic resonance imaging (MRI) demonstrated delayed gadolinium enhancement of the anterior and lateral pericardium as well as enhancement and edema on T2 short tau inversion recovery sequences (**Figure 5**), which suggests active pericarditis.

DISCUSSION

We identified 2 prior case reports of multivessel coronary obstruction occurring in the setting of pericardial calcification. Bhagia et al. (1) described a man with presumed prior tuberculous pericarditis who later developed unremitting angina from severe LAD obstruction caused by focal calcified

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ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

LAD = left anterior descending artery

MRI = magnetic resonance imaging

OM = obtuse marginal





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pericardial constriction. Angina was relieved with partial pericardiectomy. Similarly, Gaur et al. (2) described a man with chronic rheumatoid arthritis who developed LAD obstruction secondary to focal calcific pericardial band and was treated with percutaneous coronary intervention. In both of

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The second cardiac catheterization performed for recurrent chest pain: stent placed to OM1 during this procedure. Narrowing can be seen in the stents to OM1, OM2, and the LAD. D = diagonal; LAD = left anterior descending artery; OM = obtuse marginal.

these cases, pericardial calcification was identified by chest x-ray or computed tomography scan.

Hsi et al. (3) described a case of multivessel coronary constriction due to a focal pericardial band in the absence of calcification. A woman with prior pericardial window for pericardial effusion due to chest wall trauma from basketball developed cardiac arrest and multivessel myocardial infarction. The obstructions all occurred in a linear pattern as if constricted externally by a fibrous band. All vessels demonstrated



Asymmetric pericardial thickening seen on echocardiogram. Large pleural effusion is demonstrated.



Thrombolysis In Myocardial Infarction flow grade 3, so the patient underwent multimodality imaging before coronary stenting to determine the etiology of the occlusions.

We describe, to our knowledge, the first case of cardiogenic shock and multivessel infarction requiring emergent percutaneous coronary intervention and advanced mechanical support due to presumed noncalcified pericardial thickening and tethering with secondary coronary compression. We suspect that the initial trigger may have been recurrent but asymptomatic pericardial inflammation, as demonstrated on subsequent MRI, along with preexisting extensive pericardial scarring and adherence to the epicardium. We speculate that this inflammation formed a pericardial band that was capable of generating enough force to compress multiple coronary arteries and stents. Because of intermittent coronary compression and immediate intervention, the infarction size was small, with relatively low troponin levels and absence of subendocardial enhancement on MRI. Interestingly, in the case of focal pericardial constriction described by Bhagia et al. (1), coronary compression was also



(A) The arrows point to delayed gadolinium enhancement of both the parietal and visceral pericardium. (B) The late gadolinium sequence with fat suppression, which confirms that the enhancement is not due to pericardial fat. (C) The arrow points to pericardial enhancement on the T2 short tau inversion recovery sequence. The findings are consistent with active pericardial inflammation and possible scarring.

intermittent and occurred only during systole, as in our case.

FOLLOW-UP

The patient remained chest pain free after stenting and was discharged home with colchicine, dual antiplatelet therapy, angiotensin-converting enzyme inhibitor, and beta blocker. She remained chest pain free at the 2-month follow-up. Her rheumatologist has added a nonsteroidal anti-inflammatory medication to treat the pericarditis. Follow-up echocardiography showed normalization of left ventricular systolic function.

CONCLUSIONS

Focal pericardial constriction and coronary compression is a rare cause of acute multivessel myocardial infarction and cardiogenic shock. Multimodality imaging is helpful in the diagnosis of this entity.

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KEY WORDS 3-dimensional imaging, acute coronary syndrome, cardiac assist devices,

computed tomography, echocardiography, MR sequences, percutaneous coronary intervention

APPENDIX For supplemental videos, please see the online version of this paper.