External Compression of Epicardial Coronary Arteries with Partial Calcific Pericarditis

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ABSTRACT
Calcific pericarditis (CP) is a rare disease which results from long-standing pericardial inflammation. Pericardial calcification may completely or partially encase the ventricles, resulting in impaired diastolic filling. We present a case of a 53-year-old male who was incidentally found to have annular CP resulting in external compression of a large territory diagonal branch (D1) reaching the apex with likely chronically occluded left anterior descending artery with collateral circulation from the right coronary artery with hemodynamic compromise on coronary angiography. This was emergently treated with a drug-eluting stent with improved D1 flow and entailed the importance of percutaneous coronary intervention as a viable option in cases of CP resulting in acute hemodynamic compromise.

Key words: Acute coronary syndrome, calcific pericarditis, percutaneous coronary intervention

INTRODUCTION
Calcific pericarditis (CP) presents as a long-term sequela of pericardial inflammation or local injury. This causes a normal pliable fibroelastic pericardium to thicken with rigid secondary to scarring and, dense fibrosis which impedes ventricular diastolic filling.¹ While constrictive physiology usually results from encasement of both ventricles, partial CP, presenting as bands of calcium, may only result in localized compressive symptoms or partial constriction.²

CASE REPORT
A 53-year-old male presented to an outpatient office for an epidural injection for chronic back pain. Presenting vital signs showed a blood pressure of 106/62 mmHg and tachycardia with a pulse rate of 135 bpm. He reported a week history of dyspnea on moderate to severe exertion but denied any history of palpitations, syncope, near syncope, or chest pain.

Past medical history revealed significant hypertension and diabetes mellitus which controlled with diet. He was a former roofer and denied smoking, alcohol or illicit drug use.

Electrocardiogram in office showed new onset atrial flutter with variable atrioventricular block. He was sent to emergency room (ER) where he was started on diltiazem and heparin infusions. Soon after his arrival in ER, he became diaphoretic, hypoxic, complained of substernal chest pain, and sustained a cardiac arrest with pulseless electrical activity. Subsequent electrocardiogram after successful resuscitation showed atrial flutter with evidence of anterior ischemia.

Blood cell counts and thyroid-stimulating hormone levels were within normal limits while basic metabolic panel was significant for creatinine of 1.4 mg/dl which subsequently rose to 3.4 mg/dl on postresuscitation.

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Troponin-I levels which were initially negative rose up to 1.17 ng/dl. Chest radiograph showed cardiomegaly with mild congestion and pleural opacity on the right chest wall [Figure 1]. Echocardiogram revealed moderately depressed left ventricular ejection fraction with anterior wall hypokinesis and pericardial thickening [Figure 2].

With ongoing hemodynamic instability, evidence of ischemia and elevation in cardiac enzymes, he was taken for emergency catheterization.

Coronary angiography revealed subtotal occlusion of mid-left anterior descending (LAD), first diagonal, and obtuse marginal arteries secondary to external compression by a calcific band, which was also confirmed on intravascular ultrasound [Figure 3a-c and Supplemental Video 1]. The right coronary artery had mild luminal irregularities giving of collaterals to distal LAD [Figure 3d and Supplemental Video 2]. Intra-aortic balloon pump (IABP) was placed for hemodynamic support with successful placement of a drug-eluting stent to improve flow in the first diagonal branch which was thought to be the culprit vessel considering the presence of collaterals to distal LAD from the right coronary artery [Figure 3e-f and Supplemental Video 3].

A proper evaluation for constrictive physiology was deferred due to patient’s hemodynamic instability requiring IABP. Findings of localized CP were later confirmed by computed tomography scan [Figure 4a and b] with hemodynamic improvement.

An extensive infectious workup for etiology of CP was performed along with serum antinuclear antibody and rheumatoid factor which were all negative. He was eventually planned to return to catheterization laboratory for evaluation of constrictive physiology. However, he developed hypercarbic respiratory failure with pulseless electrical activity. Recurrent percutaneous coronary intervention (PCI) and was unable to be revived.

Autopsy was performed which revealed cause of death to be massive right pulmonary hemorrhagic infarct [Figure 5a]. Extensive adhesions were found between pleura and chest wall. Severe calcific, complex atherosclerosis of proximal to mid-left anterior and proximal to mid-left circumflex arteries was seen with a patent and intact recently placed mid-LAD artery stent [Figure 5b]. Fibrocalcific constrictive pericarditis with a constrictive annular band in atroventricular groove was noted with dense fibrosis and calcification [Figure 5c], but no ongoing inflammation and no explanation for the healed pericarditis on microscopy. Despite the occupational history, no mesothelioma or asbestos-related lung disease was identified.

**DISCUSSION**

CP is a rare disease, which results from long-standing pericardial inflammation. Common etiologies include...
tuberculosis in developing countries while postsurgical constriction, radiation, neoplasms, and systemic diseases are more common culprits in the western world. However, majority of the cases remain idiopathic; and as in our case, histologic examination of calcified pericardium rarely provides specific diagnoses.

Pericardial calcification varies in its presentation encasing some but not all chambers of the heart with variable degrees of compression. In some instances, nonuniform indentations, such as in our case, may result in external compression of epicardial coronary arteries which have been reportedly treated with pericardiectomy.[3,4] Two cases of annular CP successfully treated with angioplasty with good outcomes have been reported.[5,6] In both instances, patients had a history of rheumatoid arthritis with pleuritic and thoracic surgeries.

In our case, the etiology of annular CP remained unclear; it was postulated that chest compressions during cardiopulmonary resuscitation may have played a role in the compression of epicardial coronary vessels in the presence of the external calcific band.

Clinically significant cases of CP require eventual pericardiectomy; however, PCI can be utilized in cases...
Figure 5: Autopsy gross specimens of lungs and heart. (a) Lung gross specimen showing pulmonary thromboemboli with massive right pulmonary hemorrhagic infarct and pleural hemorrhage which was thought to be the cause of death. (b) Dissected specimen of left anterior groove with patent and intact recently placed first diagonal artery stent (arrow). (c) Gross specimen of heart with hypertrophied left ventricle and fibrocalcific constrictive annular band in atrioventricular groove (arrow) with dense fibrosis and calcification

with fixed external compression of epicardial coronary circulation with acute hemodynamic compromise.

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Conflicts of interest
There are no conflicts of interest.

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