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Acute pericarditis confirmed by coronary computed tomography angiogram

<https://doi.org/10.1515/jom-2022-0029>

Received February 2, 2022; accepted July 14, 2022;

published online September 8, 2022

A 48-year-old man with a history of hypertension (for the past 10 years on metoprolol succinate and triamterene-hydrochlorothiazide), asthma (for the past 15 years on albuterol and fluticasone inhalers), and prior tobacco abuse (30 pack-year, quit in May 2010) presented in October 2021 with progressively worsening left-sided chest pain. The chest pain suddenly started approximately 24 h prior to arrival, he described it as dull and achy, and since the onset, the severity worsened from 5/10 to 9/10. Additionally, the chest pain severity increased with deep inspiration and when lying supine, but it was unchanged in the left lateral recumbent position. The patient admitted 2 days prior with a fever of 101 F, but he denied any other symptoms. Of note, he recently underwent a nuclear stress test 1 month prior to presentation, and it demonstrated an inferior fixed perfusion defect, which was attributed to diaphragmatic attenuation from obesity. Furthermore, the cardiopulmonary examination of the patient was unremarkable. No friction rub was auscultated while the patient was sitting straight during deep inspiration, leaning forward, lying supine, or in the left lateral recumbent position. On admission, an electrocardiogram showed minimal ST elevation with subtle PR depression in lead II (Figure 1). On laboratory investigations, he had mild leukocytosis of 13.6 10³/uL, and four sets of high-sensitivity troponin I were stable (<3, <3, 4, and 3 pg/mL). A chest x-ray showed no acute cardiopulmonary process, including the absence of infiltrates, effusions, or cardiomegaly. Additionally, a transthoracic echocardiogram illustrated a left ventricular ejection

fraction (LVEF) of 55%–60% at rest with no other structural abnormalities. Due to an equivocal diagnosis, a coronary computed tomography angiogram was ordered. This study demonstrated pericardial thickening and normal coronary arteries (Figure 2). With the suspected diagnosis of acute pericarditis, the patient was started on ibuprofen, colchicine, and omeprazole. As he symptomatically improved and electrocardiogram findings normalized, the patient was discharged on the above regimen with complete resolution of symptoms within a few days.

Acute pericarditis is defined as an inflammatory pericardial syndrome that can be diagnosed with at least two of the following five criteria: pericarditic chest pain, pericardial rubs, new widespread ST elevation, new PR depression, and pericardial effusion [1]. Other supporting evidence for the diagnosis of acute pericarditis is elevated inflammatory markers or pericardial thickening on imaging [1]. This inflammation causes fibrin strands and aggregates of inflammatory cells to be incorporated into a layer of granulation tissue and blood vessels. This new layer of connective tissues leads to focal and/or diffuse adhesions between the parietal and visceral pericardium, which can create tension on the pericardium and pericardial ligaments [2]. A recently developed noninvasive visceral osteopathic approach for the functional release of the pericardial ligaments has been applied to decrease the tension in the pericardial ligaments by releasing putative tension in the sternopericardial, pericardiophrenic, and fascial visceropericardial ligaments [3].

Research funding: None reported.

Author contributions: Both authors provided substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; both authors drafted the article or revised it critically for important intellectual content; both authors gave final approval of the version of the article to be published; and both authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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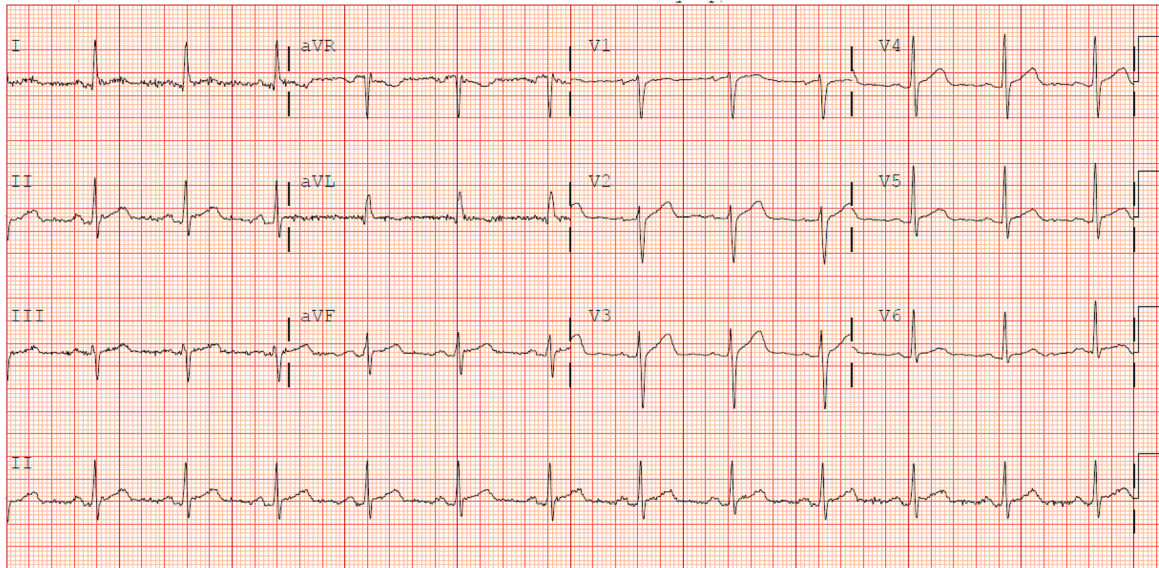


Figure 1: Admission 12-lead electrocardiogram demonstrated minimal ST elevation and subtle PR depression in lead II.

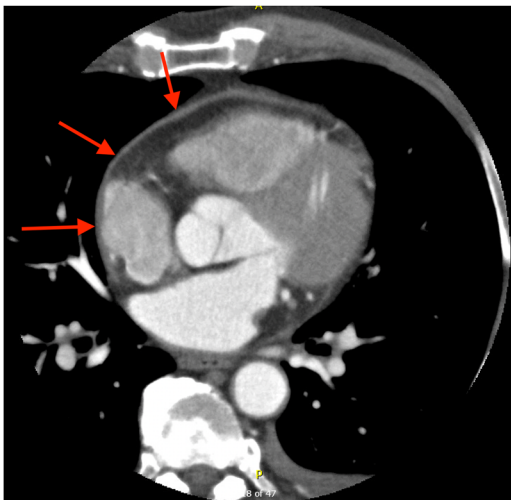


Figure 2: Coronary computed tomography angiogram illustrated pericardial thickening (arrows).

Competing interests: None reported.

Informed consent: The patient described in this report provided written informed consent.

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