JACC: CASE REPORTS VOL. 3, NO. 6, 2021

MINI-FOCUS ISSUE: IMAGING

INTERMEDIATE

CASE REPORT: CLINICAL CASE

Multimodal Imaging to Understand Left Ventricular Systolic Dysfunction in a Patient With Sepsis-Related Myocardial Calcification

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ABSTRACT

A 52-year-old man with acute peritonitis developed severely decreased left ventricular (LV) ejection fraction. Multimodal imaging allowed the diagnosis of sepsis-related myocardial calcification. Moreover, 2-dimensional speckle tracking echocardiography allowed a better understanding of LV dysfunction and confirmed the hypothesis that regional LV dysfunction is in accordance with the localization of calcifications. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:966–70) © 2021 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 52-year-old man with acute peritonitis following surgery of the prostate was admitted to the intensive care unit with septic shock. On admission, the patient presented with fever at 39°C with sweats and chills, tachycardia at 160 beats/min, and initially not measurable blood pressure. Antibiotic therapy, high-dose catecholamine therapy for blood pressure support, mechanical ventilation, and continuous renal replacement therapy were administered for 1 month. At 10 days, the patient presented hemodynamic impairment, a sinus rhythm and dynamic change of ST-segment on electrocardiogram, and an elevated cardiac-specific troponin (188 μg/L, N <0.045 μg/L). Transthoracic echocardiography (TTE) showed a severely decreased left ventricular ejection fraction (LVEF) at 30%. Coronary angiography indicated the presence of nonobstructive coronary artery disease.

LEARNING OBJECTIVES

- To be able to make a differential diagnosis of troponin-positive nonobstructive coronary arteries with multimodality imaging.
- To select optimal imaging modalities for a patient with sepsis-related myocardial calcification to improve diagnosis and treatment.

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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 Author Center.

Manuscript received February 12, 2021; revised manuscript received April 26, 2021, accepted April 29, 2021.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2021.04.037

PAST MEDICAL HISTORY

Before presentation, the patient reported good health and he had no relevant previous medical history.

DIFFERENTIAL DIAGNOSES

The differential diagnosis was myocardial infarction with nonobstructive coronary arteries secondary to plaque rupture, embolic phenomenon to coronary vessels, or nonischemic causes such as sepsis, pulmonary embolism, and pericarditis/myocarditis.

INVESTIGATION

The review of coronary angiography indicated the presence of nonobstructive coronary artery disease in the setting of an atypical myocardial signal (**Figure 1,** Video 1). Computed tomography (CT) showed diffuse left ventricular (LV) wall calcifications circumferentially that were not observed on the previous CT scan performed a week earlier (**Figures 2 and 3**), which suggested sepsis-related myocardial calcification. Cardiac magnetic resonance showed calcification with low signal on T2 without enhancement. Late gadolinium enhancement was, however, observed in the scarred myocardium surrounding calcifications. Late gadolinium enhancement was distributed circumferentially but variably among the 3 layers of the myocardium (endocardial, mid-wall, and epicardial), from the apex to the base, surrounding only the entire left ventricle (**Figure 4**).

MANAGEMENT

The acute phase has been resolved by modification of catecholamine therapy (decrease of norepinephrine and introduce dobutamine). In chronic phase, after the intensive care unit period, the 2-dimensional (2D) echocardiogram performed showed a 45% global LVEF and demonstrated the association of calcific deposits with cardiac dysfunction. To investigate the mechanistic basis of dysfunction in this patient, LV deformation was assessed offline using speckle tracking echocardiography (STE). The longitudinal and circumferential shortening and radial thickening were measured to analyze the endocardial, epicardial, and mid-wall myofibers functions, respectively. Epicardial dysfunction was noticed at the LV apex with relatively normal mid-wall and endocardial function, causing abnormal circumferential mechanics and relatively normal longitudinal and radial mechanics. At the mid-LV level, there was marked mid-wall and epicardial dysfunction, with relatively normal endocardial function, causing abnormal radial and circumferential mechanics and relatively normal longitudinal mechanics. At the basal level, there was relatively normal epicardial, mid-wall, and endocardial function. The apex-to-base gradient was also lost by longitudinal strain (**Figure 5**). So, disease processes predominantly affected specific myofibers depending on the LV level being considered, which affected the regional LV deformation accordingly. The concordance between the presence of extensive calcifications and an anormal strain according to LV level was good with a kappa coefficient at 0.72. In view of this evidence, it was decided to introduce an optimal treatment for heart failure combining maximum doses of angiotensin-converting enzyme inhibitors, beta-blockers, and spironolactone to control cardiac remodeling and

ABBREVIATIONS AND ACRONYMS

2D = 2-dimensional

CT = computed tomography

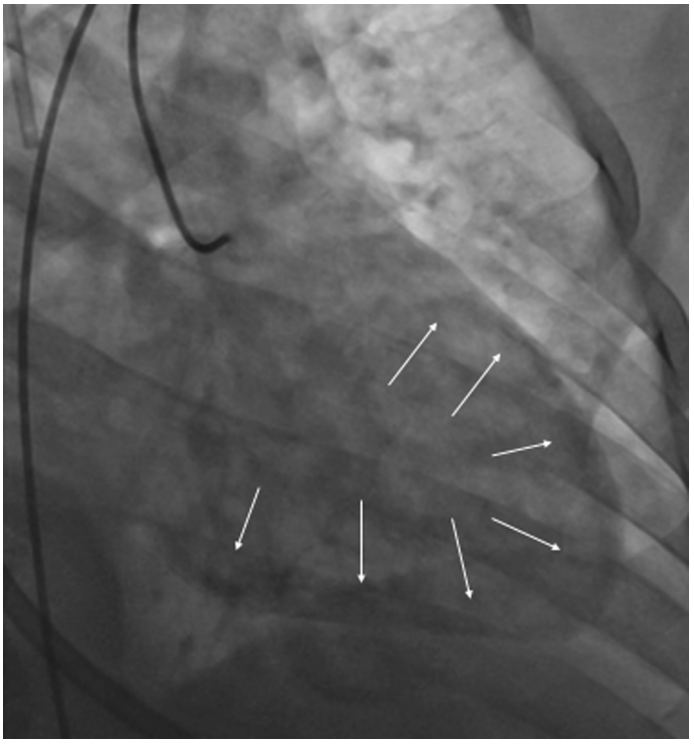
LV = left ventricular

LVEF = left ventricular ejection fraction

STE = speckle-tracking echocardiography

TTE = transthoracic echocardiography

FIGURE 1 Invasive Coronary Angiogram



Coronary angiography indicated the presence of an atypical myocardial signal independent of contrast injection **(arrows)**.

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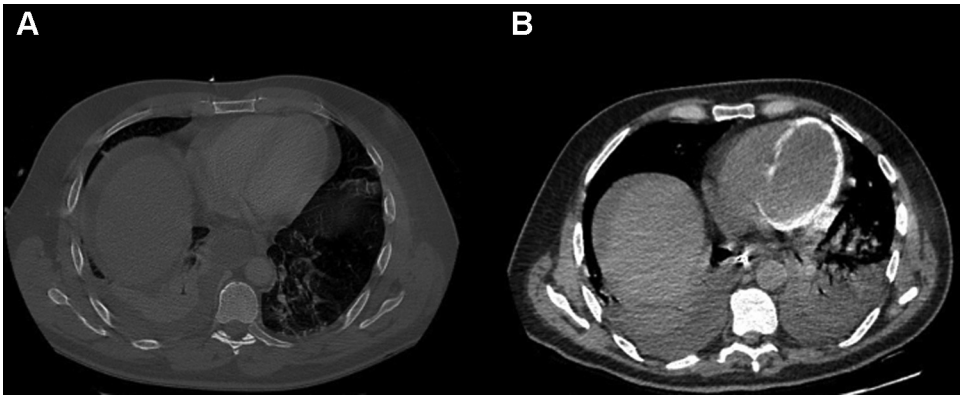
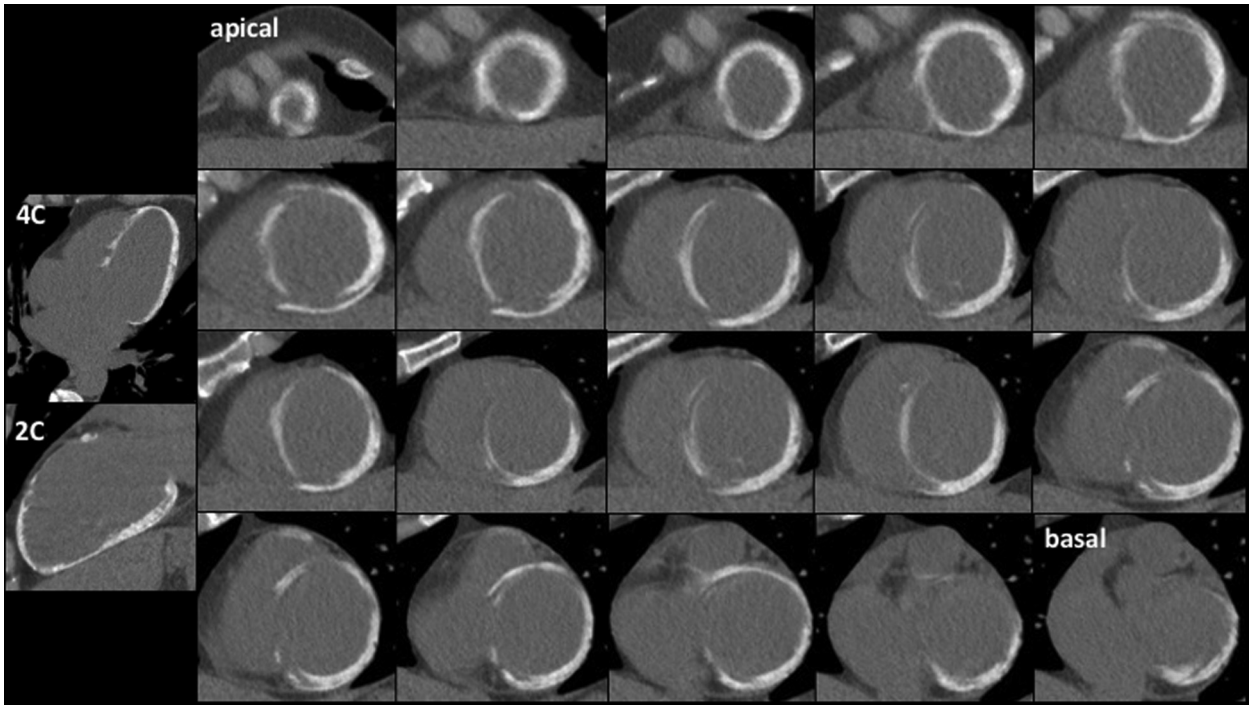
FIGURE 2 Computed Tomography



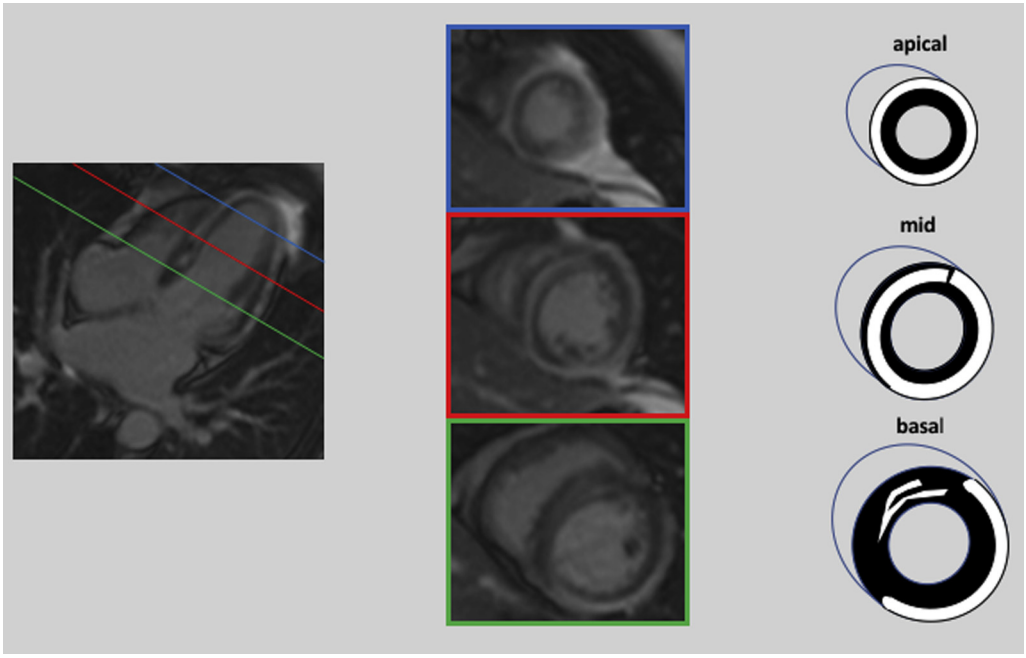
Computed tomography (CT) showed left ventricular wall calcifications **(arrow) (B)** that were not observed on the previous CT scan performed a week earlier **(A)**.

FIGURE 3 Cardiac Computed Tomography



Computed tomography (CT) showed diffuse, circumferential left ventricular wall calcifications on short axis view **(arrows)**. 4C = 4-chamber view; 2C = 2-chamber view.

FIGURE 4 Cardiac Magnetic Resonance Imaging



Late gadolinium enhancement was distributed circumferentially but variably among the 3 layers of the myocardium (endocardial, mid-wall, and epicardial), from the apex to the base of the left ventricle.

improve hemodynamics as in post-myocardial infarction heart failure.

FOLLOW-UP

At 1 year of follow-up, the patient was asymptomatic with an optimal treatment of heart failure. However, TTE parameters were similar with 45% of LVEF, a similar amount of calcification, and abnormal LV deformation.

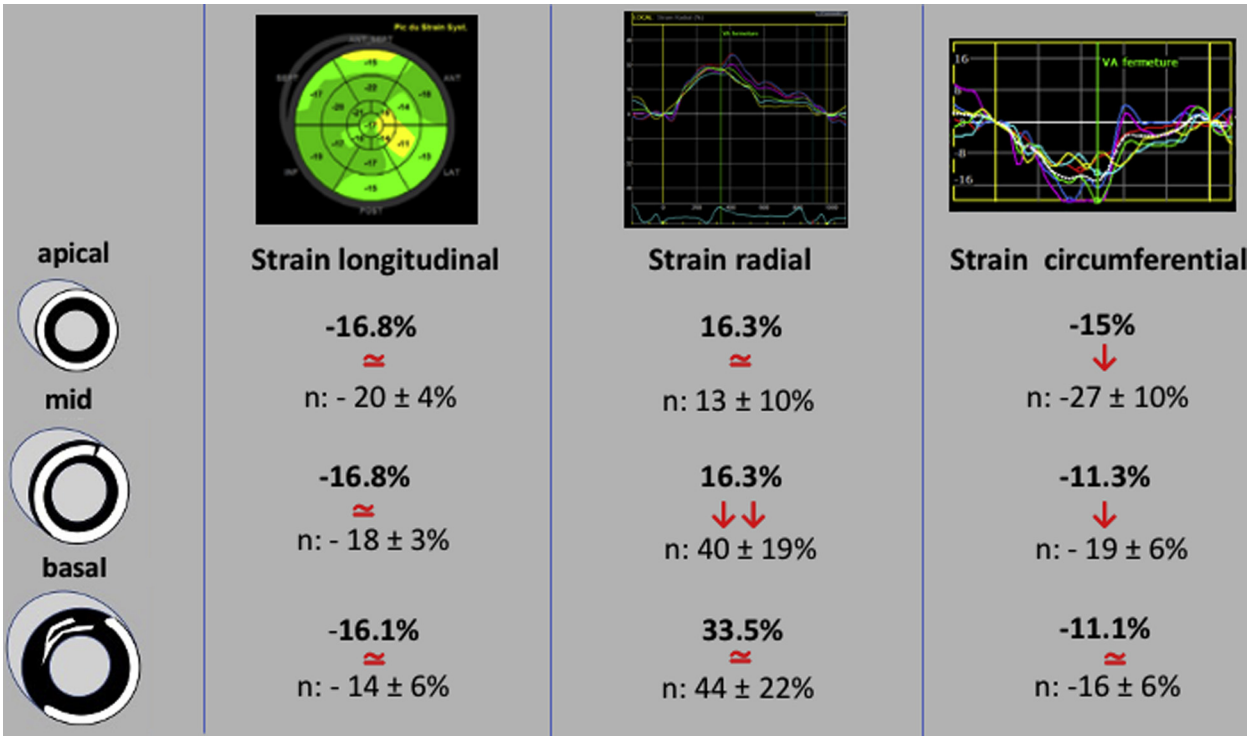
DISCUSSION

The exact mechanism of sepsis-related myocardial calcification is unclear. The cardiac dysfunction is initially due to proinflammatory mediators. The microcirculation then seems to be involved in the mechanism of cell injury, very likely due to flow disturbances caused by septic shock and the dystrophic calcification occurring in myocytes showing myocytolysis (1). This diagnosis should be considered in patients with established sepsis with new-onset diffuse myocardial calcifications and with no arguments for metastatic myocardial calcification (2). The combination of the imaging pattern and clinical history may help clinicians. Several cases of extensive LV calcification after a period of severe sepsis have been described previously (3) but none of them has used STE. In the present case, 2D-STE allowed a better characterization of LV dysfunction. This case confirms the hypothesis that patients with sepsis-related myocardial calcification have impaired regional LV function in accordance with the localization of calcifications (4).

CONCLUSIONS

Sepsis-related myocardial calcification is a rare case of sepsis complication. It is important to use multimodality imaging to perform correct diagnosis and evaluation of this disease. Indeed, evolution can be worse than myocarditis, which can appear similar on cardiac magnetic resonance imaging, because myocardial calcifications are long-term myocardial damage that leads to LV systolic dysfunction and heart failure. 2D-STE should be done systematically in sepsis-related myocardial calcification because it is more accurate for analyzing subtle systolic dysfunction. Imaging of calcification using CT and a simple measurement of ejection fraction in TTE are not sufficient.

FIGURE 5 Speckle-Tracking Echocardiography



Left ventricular (LV) deformation was assessed offline using speckle tracking echocardiography. Longitudinal and circumferential shortening as well as radial thickening were measured to analyze the endocardial, mid-wall, and epicardial myofiber functions, respectively. Disease processes predominantly affected specific myofibers depending on the LV level being considered, which affected the regional LV deformation accordingly.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

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

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KEY WORDS multimodal cardiac imaging, myocardial calcification

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