A Curious Case of Acute Myocardial Calcifications

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A 56-year-old woman was admitted to a regional hospital for increasing chest and abdominal pain after falling from the stairs. Initial screening showed a pulmonary contusion of the left lower lobe and signs of a myocardial contusion characterized by elevated troponins without ST-T abnormalities. She had acute renal failure without rhabdomyolysis and a highly elevated C-reactive protein (800 mg/L). As a result of increasing respiratory insufficiency, she was transferred to the intensive care unit for further treatment. Three days after hospital admission, she developed diffuse ST-segment elevations on ECG and elevated cardiac enzymes. Given the suspicion of coronary artery disease, she was transferred to our institution for an urgent coronary intervention. On arrival in our institution, laboratory findings revealed an elevated white blood cell count (15.3×10⁹/L), a highly elevated C-reactive protein (578 mg/L), severe renal failure with a glomerular filtration rate of 6 mL·min⁻¹·1.73 m⁻², hypocalcemia (1.55 mmol/L), elevated troponins (troponin I, 86.5 μg/L), elevated creatine kinase (5429 U/L), and elevated creatine kinase-MB (97.9 μg/L).

Despite the elevated cardiac enzymes, coronary angiography showed no coronary abnormalities. At this time, a tentative diagnosis was established of severe sepsis with concomitant septic cardiomyopathy. An initial contrast-enhanced computed tomography (CT) scan at hospital admission showed no morphological cardiac abnormalities, notably with a normal appearance of the left ventricular myocardium (Figure 1A). However, a follow-up CT scan 13 days after hospital admission revealed an ill-defined increased density of the left ventricular myocardium (Figure 1B). A follow-up CT scan 44 days after hospital admission showed extensive myocardial calcifications in the left ventricle (apex, septum, and anterolateral and inferior wall; Figures 1C and 2A–2C). Initial transesophageal echocardiography at hospital admission showed an estimated ejection fraction of 40% (Movie I in the online-only Data Supplement). Follow-up transthoracic echocardiography (5 months after hospital admission) showed a diffuse hypokinetic left ventricle with an estimated ejection fraction of only 20% (Movie II in the online-only Data Supplement). The right ventricular function, on the other hand, remained normal. This was illustrated by a normal tricuspid annular plane systolic excursion. No significant valvular disease was noted at baseline or at follow-up. On the basis of the combination of clinical, laboratory, and imaging findings, a final diagnosis of septic cardiomyopathy characterized by elevated cardiac enzymes without coronary abnormalities was established. This was further substantiated by the rapid appearance of myocardial calcifications, the concomitantly deteriorating cardiac function, and the absence of abnormal serum levels of calcium and phosphorus or any other relevant metabolic condition during hospital admission.

Discussion

Cardiac dysfunction is a well-recognized, common complication of severe sepsis and shock, acting as a major contributor to morbidity and mortality. Mortality in patients with cardiac dysfunction caused by sepsis is much higher compared with septic patients without cardiovascular involvement. In general, ≈15% of septic shock–related deaths are secondary to myocardial depression. A septic cardiomyopathy or sepsis-induced cardiomyopathy is characterized by ventricular dilatation and a reduced ejection fraction and contractility. Experimental models of sepsis show clear evidence of myocardial contractile disturbance. Furthermore, mice studies revealed that intracellular free calcium ion concentration markedly increases in response to the addition of serum from septic mice. Because calcium homeostasis is essential to a normal myocardium contraction/relaxation cycle, intracellular calcium overload is therefore considered a critical step in the pathophysiology of cardiac dysfunction in sepsis.

Diffuse myocardial calcifications are rare but have been reported. Historically, they have been classified as dystrophic or metastatic by Gore and Arons.1 Metastatic calcification is more common and results from underlying conditions with associated hypercalcemia such as renal failure (mostly in end-stage renal disease), hyperparathyroidism, and other toxic and septic factors. In the first 2 conditions, the calcifications are thought to be a result of abnormal serum levels of calcium and phosphorus. In dystrophic calcification, calcium deposition occurs in any necrotic area. The effect of diffuse myocardial calcifications on heart function is unpredictable. They can lead to disturbance of the cardiac conduction system, causing cardiac arrhythmias and sudden death.

Several authors have described the occurrence of diffuse myocardial calcifications secondary to septic cardiomyopathy. However, there is substantial variation in the chosen descriptive term. In this case report, we use the term sepsis-related...
myocardial calcification, as used by van Kruijsdijk et al. This diagnosis should be considered in patients with established sepsis with new-onset diffuse myocardial calcifications, without arguments for metastatic myocardial calcification (as seen in patients with an abnormal serum level of calcium and phosphorus).

A CT scan is the gold standard examination for the noninvasive detection of myocardial calcifications. In our case, it exquisitely revealed the extensive myocardial calcifications involving the left ventricular myocardium and the less pronounced calcifications in the right ventricular myocardium. Usually, the detection of these calcifications is unexpected. Imaging findings in our case suggest that, because initially the increased myocardial density may be minor, these myocardial calcifications may be missed on early contrast-enhanced CT scan, given the slight difference between these calcifications and the normal enhancing unaffected myocardium. A delayed-phase or nonenhanced CT scan can on such occasions better demonstrate the calcifications. Echocardiography, which is less sensitive in demonstrating the myocardial calcifications, is supplementary for functional information because diastolic dysfunction and hypokinesia may be present. It can depict the myocardial calcifications because they produce acoustic shadowing. However, echocardiography could be normal. Cardiac magnetic resonance imaging does not routinely contribute to the diagnosis.

Conclusions
Sepsis-related myocardial calcifications are a rare phenomenon in patients with septic cardiomyopathy but can lead to a significant morbidity and mortality. These dystrophic myocardial calcifications are typically extensive and involve the left ventricular wall or, less commonly, the right ventricular wall. Myocardial calcification is usually an incidental finding on a CT scan in patients with sepsis. Electrocardiography should be supplemented to assess the repercussions on cardiac function.

Disclosures
None.

References

Figure 1. A, Contrast-enhanced computed tomography (CT) scan (soft-tissue window, 4-chamber view) shows a morphologically normal-appearing left ventricular wall (5 days after hospital admission). B, On a follow-up CT scan (13 days after hospital admission), there is an ill-defined left ventricular wall with increased density (isodense compared with the intravascular compartments). C, Diffuse myocardial calcifications in the left ventricular wall (apex, septum, and anterolateral wall) on follow-up CT-scan (delayed phase) 44 days after hospital admission.

Figure 2. Contrast-enhanced computed tomography scan 44 days after hospital admission (delayed phase, 4-chamber view [A], long-axis 2-chamber view [B], and short-axis view [C]) depicts the extensive spread of myocardial calcifications (apex, septum, and anterolateral and inferior wall).
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