A 56-year-old man was admitted on our intensive care unit with septic shock after 10 days of diarrhea, shortly after he had visited India. He had no relevant previous medical history. Blood and fecal cultures grew various pathogens, including Shigella flexneri. ECG showed diffuse ST elevation with PR depression (Figure 1), and laboratory results revealed significant troponin (peak level of 5.72 ng/mL) and creatine phosphokinase-MB (peak level of 117 ng/mL) release, most likely caused by a perimyocarditis in the setting of Shigella sepsis. Further diagnostic evaluation revealed hairy cell leukemia. After initial improvement of his clinical condition and treatment with rituximab and prednisolone, he was readmitted to the intensive care unit with respiratory failure and new-onset left ventricular dysfunction as was observed with echocardiography. Compared with the prior tracing, his ECG now showed microvoltages in the extremity leads, this time with normal ST segments (Figure 2). Computed tomographic imaging revealed extensive myocardial calcification of the left ventricle that was not seen on previous computed tomographic images (Figures 3 and 4). Comparable images of a pronounced dense myocardium with normal aspect of the endocardial layer were observed with transesophageal echocardiography (Figure 5). Serum levels of calcium were repeatedly not elevated during the admittance.

Localized myocardial calcification is commonly observed after myocardial infarction. Diffuse calcification, however, is a rare phenomenon. Interestingly, similar cases of extensive left ventricular calcification after a period of severe sepsis have been described previously. Two different pathophysiological mechanisms resulting in calcium deposition in cardiac myocytes can be distinguished. First, significant disturbances in calcium metabolism can lead to metastatic calcification, as is occasionally observed in patients with chronic renal failure. Furthermore, calcium may accumulate in necrotized cardiac myocytes, which is known as dystrophic calcification. The latter mechanism is believed to underlie myocardial calcification in the setting of myocardial infarc-

![Figure 1. ECG at day 1 of admission showing diffuse concave ST elevation with PR depression (arrow).](http://circheartfailure.ahajournals.org/doi/fig/10.1161/CIRCHEARTFAILURE.111.962183)
tion and severe sepsis. At present, no therapies, other than symptomatic treatment of the restrictive cardiomyopathy due to myocardial calcification, are available.

**Disclosures**

None.

**References**


**Key Words:** calcium ▪ heart failure ▪ imaging ▪ myocardial calcification ▪ sepsis

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**Figure 2.** ECG at day 31 of admission showing micro voltages in the extremity leads with normal ST segments.

**Figure 3.** Computed tomography scan at day 1 of admission showing normal appearing myocardium. LA indicates left atrium; RA, right atrium; LV, left ventricle; and RV, right ventricle.

**Figure 4.** Computed tomography scan at day 31 of admission showing extensive calcification of the left ventricular myocardium. LA indicates left atrium; RA, right atrium; LV, left ventricle; and RV, right ventricle.
Figure 5. Transesophageal echocardiogram (4-chamber view) showing pronounced echodensity of the myocardium of the midseptal, apical, and lateral walls, with a normal endocardial layer. LA indicates left atrium; LV, left ventricle; and RV, right ventricle.
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