We present a case of a 40-year-old man who was brought to our hospital after a sudden cardiac arrest. He had a history of human immunodeficiency virus infection and was receiving treatment with antiretroviral drugs with good compliance, showing an undetectable virus load in a test done 1 month earlier. The patient did not feel well in the morning and went to a walk-in clinic. While waiting to be assessed at the primary care center, the patient suddenly lost consciousness. Resuscitation protocol was started, and the first rhythm on the arrival of emergency services was a ventricular fibrillation. After 3 shocks, sinusoidal rhythm was restored. Although the patient breathed spontaneously, he was hemodynamically unstable so he was sedated and orally intubated.

At admission in our hospital, an ECG was obtained (Figure 1).

Please turn the page to read the diagnosis.

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**Figure 1.** Twelve-lead ECG performed on patient admission to the emergency department.
RESPONSE TO ECG CHALLENGE

The ECG revealed a de Winter’s ECG pattern, the equivalent of an anterior ST-segment elevation myocardial infarction. Consequently, an emergency coronary angiography was carried out where an acute proximal occlusion of the left anterior descending artery was observed (Figure 2).

De Winter’s ECG pattern is considered as an ST-segment elevation myocardial infarction equivalent that is present in ≈2% of all patients with an acute anterior myocardial infarction. Instead of the signature ST-segment elevation, in this ECG pattern, the ST-segment shows a 1- to 3-mm upsloping ST-segment depression at the J point in leads V1 to V6 that continue into tall, positive, and symmetrical T waves. The QRS complexes are usually not widened or are only slightly widened, and in some cases a loss of precordial R-wave progression occurs. In most patients, a 1- to 2-mm ST-segment elevation in the lead aVR is found. This pattern was thoroughly described by de Winter et al2,3 in 2008, and since then several cases have been reported. In most cases, the proximal segment of the left anterior descending artery was occluded.

The pathophysiological mechanisms of this ECG pattern are still a subject of discussion today. Some authors defend that a collateral blood supply might protect the myocardium from transmural ischemia and prevent the ST-segment elevation. However, in the article of Verouden et al, 3 in a cohort of 1890 patients with anterior ST-segment elevation myocardial infarction who were referred for primary coronary angiography and presented an occlusion of the proximal left anterior descending artery, only a few patients who also presented de Winter’s pattern had Rentrop Grade 3 collateral blood supply. These authors postulated that the area of transmural ischemia was so large that no injury currents were generated toward the precordial leads but only directed upward to an aVR lead. In the same manuscript, the authors propose additional potential explanations for this observed pattern. Thus, theoretically an anatomic variant of the Purkinje fibers with endocardial conduction delay could result in the described pattern. Another alternative explanation might be that the absence of ST-segment elevation may be related to the lack of activation of sarcolemmal ATP-sensitive potassium channels by ischemic ATP deple- tion, as has been shown in sarcolemmal ATP-sensitive potassium knockout animal models of acute ischemia.2,3 In 2009, Gorgels1 explained this pattern by changes in the subendocardial but not in the epicardial action potential, suggesting subendocardial ischemia as the underlying mechanism.

In any case, because of the impact in a patient’s prognosis, rapid recognition of this ECG pattern is crucial, especially in patients after sudden cardiac arrest. Ambulance staff, emergency physicians, cardiologists, and other caregivers involved in ST-segment elevation myocardial infarction networks should familiarize themselves with this atypical ECG pattern to ensure immediate reperfusion therapy.

DISCLOSURES
None.

FOOTNOTES
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