Asymptomatic ST-Segment–Elevation ECG in Patient With Kidney Failure

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ECG CHALLENGE

A 32-year-old man with a medical history of chronic kidney disease on chronic hemodialysis and nontransfusion-dependent thalassemia was referred to the cardiology unit for evaluation before kidney transplantation. He denied any symptoms of chest pain, palpitation, or syncope. His family history was unremarkable for cardiac disease. His functional status was class I. His physical examination revealed a soft systolic ejection murmur at the left upper parasternal border. His chest radiograph demonstrated cardiomegaly without pulmonary edema. A routine 12-lead ECG was performed as shown in Figure 1.

Please turn the page to read the diagnosis.

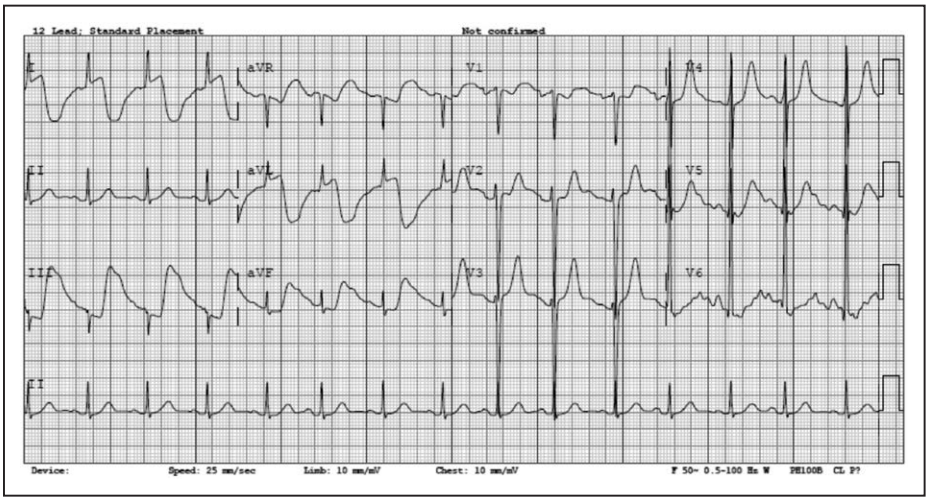

Figure 1. An initial 12-lead ECG.

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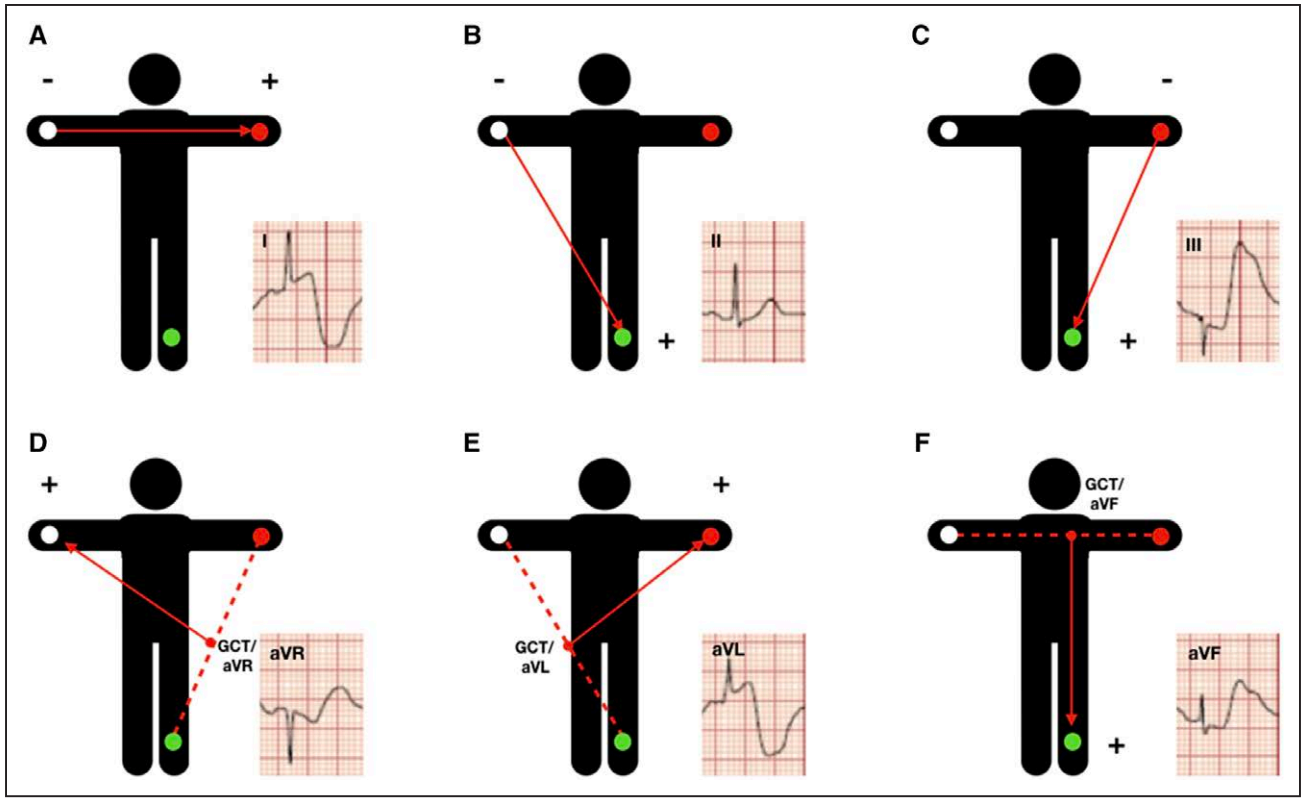

Figure 2. Demonstration that only lead II is not affected by the left arm electrode artifact.
A, Lead I is constructed by comparing of the right arm (as a negative electrode) and the left arm (as a positive electrode). **B,** Lead II is constructed by comparing the right arm (as a negative electrode) and the left leg (as a positive electrode). **C,** Lead III is constructed by comparing the left arm (as a negative electrode) and the left leg (as a positive electrode). **D,** Lead aVR is constructed by comparing the average potential between the left arm and the left leg (as a negative electrode; GCT/aVR) with the right arm (as a positive electrode). **E,** Lead aVL is constructed by comparing the average potential between the right arm and the left leg (as a negative electrode; GCT/aVL) with the left arm (as a positive electrode). **F,** Lead aVF is constructed by comparing the average potential between the right arm and the left arm (as a negative electrode; GCT/aVF) with the left leg (as a positive electrode). GCT indicates Goldberger central terminal.

RESPONSE TO ECG CHALLENGE

The 12-lead ECG shows sinus rhythm with ST-segment–elevation in leads I and aVL and ST-segment–depression in leads III and aVF followed by the large bizarre T waves. The differential diagnosis for ST-segment–elevation includes conditions such as acute myocardial infarction, early repolarization, left ventricular hypertrophy, left bundle-branch block, acute pericarditis, hyperkalemia, Brugada syndrome, pulmonary embolism, and cardioversion.[1] Infrequently, an artifact should be considered to be the cause of ST-segment–elevation, especially in asymptomatic patients.

An important difference between true ST-segment–elevation attributable to myocardial infarction and an artifact is that the baseline elevation in an artifact may begin before or after the onset of QRS complex, as was observed in this case. The large bizarre T waves also support the diagnosis. When the electrodes are not properly adhered to a patient's skin, an artifact will occur, usually accompanied by a wandering baseline. In addition, in this case, the ST-segment–elevation was not organized in any specific distribution, as it would be in true ischemia. This ECG revealed that the artifact was synchronous with the cardiac cycle, suggest-

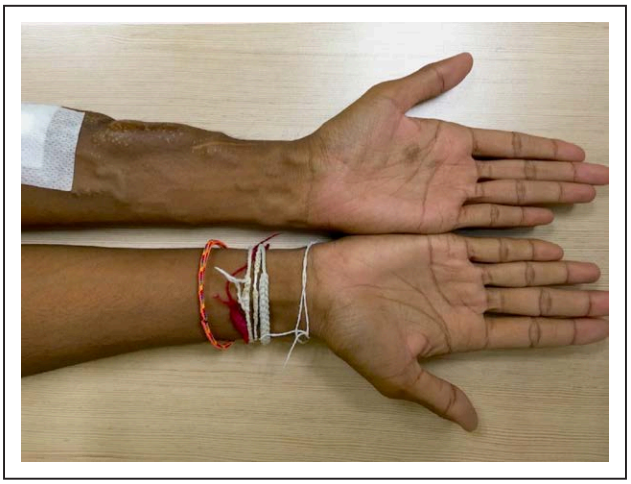

Figure 3. The patient's forearm showed an arteriovenous fistula on the left side.

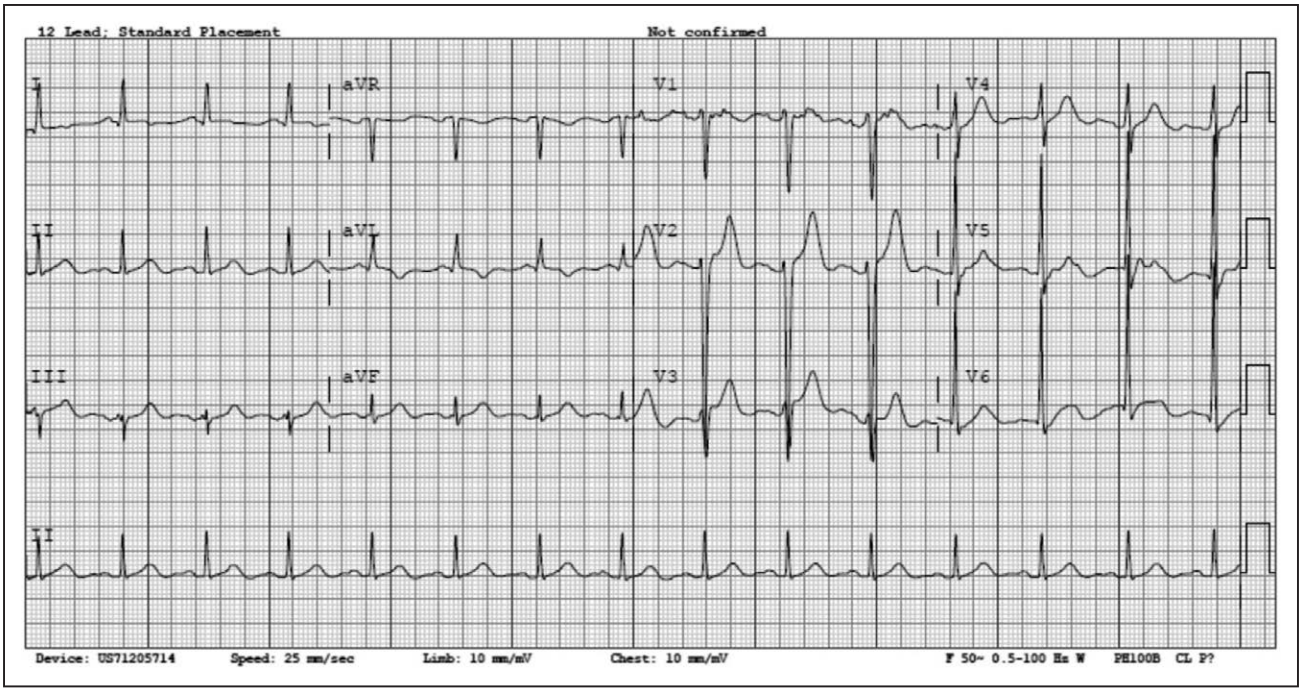

Figure 4. The next ECG after placing the left arm electrode distal to an arteriovenous fistula.

ing an arterial pulse-tapping artifact, a condition first described by Aslanger et al.[2,3] There is a clue to identify the problematic electrode from the ECG; the artifact can be observed in leads I, III, aVR, aVL, and aVF, whereas there is no artifact in lead II. According to the Einthoven triangle, leads I, II, and III are bipolar limb leads (Figure 2a through 2c). Lead I is composed by comparing electric differences between the right and left arms, lead II is composed by comparing the right arm and the left leg, and lead III is composed by comparing the left arm and the left leg. The augmented limb leads that use the Goldberger central terminal, an averaging of inputs from 2 of the 3 limb electrodes, as their negative pole were also abnormal (Figure 2d through 2f). For this reason, therefore, the left arm electrode is the culprit, either directly or indirectly via the Goldberger central terminal, because all leads are affected with the exception of lead II.

In addition, the artifact was seen on every cardiac cycle, having a fixed coupling interval between maximal amplitude of the QRS complex and the artifact in every affected lead. This finding suggests a cardiac cycle–related artifact. This patient was on hemodialysis treatment via the left arm arteriovenous fistula (Figure 3). This caused pulsatile movement of an attached ECG electrode. A subsequent ECG, in which the left arm electrode was placed distally to the arteriovenous fistula, was normal (Figure 4). The disappearance of ST-segment deviation and bizarre T waves in all leads supports the diagnosis of an arteriovenous fistula artifact.

In conclusion, we report a case of arterial pulse-tapping ECG artifact associated with a forearm arteriovenous fistula. The artifact can mimic a true ST-segment–elevation infarct. The ECG could potentially ascertain the origin of the artifact, and a thorough review of the patient's history and a detailed analysis of the initial and follow-up ECG are therefore crucial to achieve the correct diagnosis.

DISCLOSURES

None.

AFFILIATION

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FOOTNOTES

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