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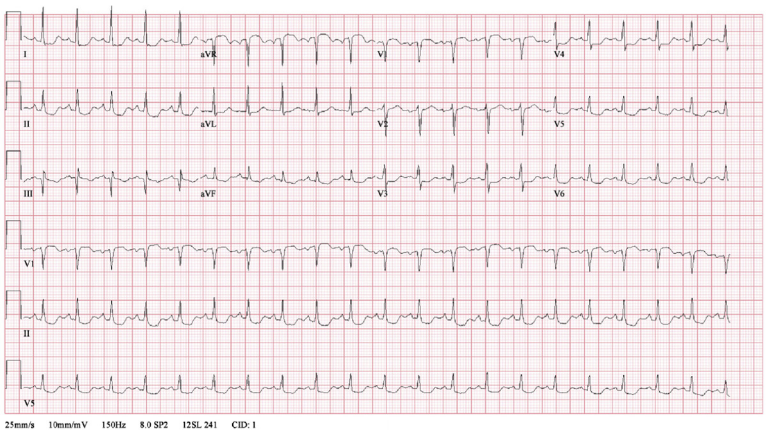

Figure 1. Initial ECG on presentation.

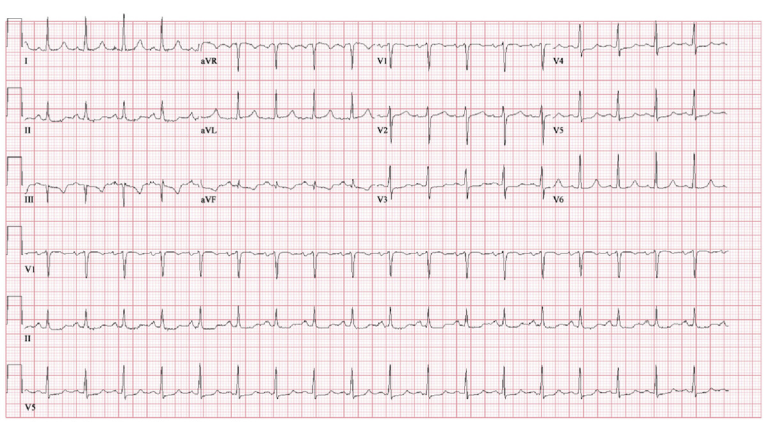

Figure 2. Repeated ECG after administration of thrombolytics (2 hours, 47 minutes after the initial ECG).

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A 63-year-old woman with a history of diabetes mellitus and hypertension was brought to the emergency department by emergency medical services for dyspnea on awakening several hours earlier. In the field, the patient had a reported blood pressure of 84/60 mm Hg and oxygen saturation of 85% on room air. On arrival, her pulse rate was 124 beats/min, blood pressure 79/50 mm Hg, respiratory rate 31 breaths/min, oxygen saturation 100% on a nonrebreather mask, and temperature 98.2°F (36.8°C). She appeared to be in moderate respiratory distress despite clear lungs to auscultation bilaterally. A 12-lead ECG was obtained (Figure 1).

For the diagnosis and teaching points, see page 407.
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ECG OF THE MONTH

(continued from p. 406)

DIAGNOSIS:

Interpretation

The ECG revealed sinus tachycardia with a rate of 124 beats/min, with 2-mm of ST-segment elevation in aVR (aVR-STE) and multilead ST-segment depressions in precordial leads V3 through V6, and limb leads I, II, and aVL.

CLINICAL COURSE

The patient's presentation prompted an immediate bedside echocardiogram, which revealed elements of right-sided heart strain, including flattening of the intraventricular septum, and an enlarged right ventricle. Massive acute pulmonary embolism was suspected, and the patient was taken for emergency computed tomography (CT), which confirmed the diagnosis (Figure E1, available online at http://www.annemergmed.com).

The patient consented to thrombolysis and was given a 15-mg bolus dose of tissue plasminogen activator, followed by an infusion of 85 mg over 90 minutes. Within 2 hours, the patient's blood pressure improved to 151/82 mm Hg and pulse rate to 109 beats/min. The patient's oxygen requirement was weaned to 2 L/min via nasal cannula, with improvement in work of breathing.

A repeat ECG was obtained and displayed interval resolution of aVR-STE and resolving multilead ST-segment depression (Figure 2). Heparin was initiated, and the patient was admitted to the medical ICU. The following day, she was downgraded to floor status, and 12 days later she was discharged. Her course was complicated by a right knee hemarthrosis, poorly controlled diabetes, and a labile international normalized ratio after she was bridged to warfarin.

DISCUSSION

Historically, the purpose of lead aVR was to obtain specific information from the right upper side of the heart, such as the outflow tract of the right ventricle and the basal part of the septum. In practice, however, lead aVR was largely ignored and thought to yield only reciprocal information from the left lateral side.[1] More recently, aVR-STE with coexistent multilead ST-segment depression has been identified as a strong predictor of left main coronary artery or 3-vessel disease.[2]

However, occlusive coronary artery disease is not the only cause of this ECG pattern. Frequently, this pattern results from nonocclusive causes such as baseline left ventricular hypertrophy or conditions that create a supply-demand mismatch such as acute blood loss, sepsis, respiratory failure, tachydysrhythmias, and aortic stenosis.[3] In one series of 133 patients showing this ECG pattern, only 28% had acute coronary syndromes, whereas 45% had hypertensive heart disease.[4] Although the mechanism is not clear, it is postulated that acute pulmonary embolism can lead to profound right and left ventricular ischemia, causing aVR-STE, which can lead to a misdiagnosis of primary ischemic disease.[5] In one retrospective analysis of 396 patients with acute pulmonary embolism, aVR-STE was present in 34% of patients.[6] This case presents a patient whose ECG showed aVR-STE as a result of a massive acute pulmonary embolism, which resolved after administration of thrombolytics.

In the setting of acute pulmonary embolism, several features of this ECG portend a worse prognosis and warrant discussion. First, STE in V1 is a predictor of cardiogenic shock[5] and is associated with a greater than 2-fold increase in mortality (12.9% versus 5.1%; P=.009).[6] Second, aVR-STE is associated with a more severe clinical course, including the development of cardiogenic shock.[5,6] Third, ST-segment depression in V4 to V6 is also associated with the development of cardiogenic shock.[4] With the presence of all 3 of these ECG features, one could predict a poor outcome for this patient if the diagnosis of acute pulmonary embolism had not been recognized and treated early. Other features (not present on this ECG) associated with worse prognoses include S1Q3T3 sign, STE in lead III, right bundle branch block, and a qR sign in lead V1.[5]

In conclusion, ECGs with aVR-STE and coexistent multilead ST-segment depression should prompt the emergency physician to consider not only primary ischemic pathology but also conditions that create a supply-demand mismatch, such as acute pulmonary embolism. Lack of recognition of the other causes of aVR-STE may lead to misdiagnosis, significant delays, or improper care.

PEARLS

Acute pulmonary embolism is a potential cause of aVR-STE with coexistent multilead ST-segment depression.

This ECG pattern should be interpreted in the context of the patient's clinical presentation because many conditions can cause it.

In the setting of acute pulmonary embolism, aVR-STE, STE in V1, and ST-segment depression in V4 to V6 are features that portend a poorer prognosis.

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