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Letters to the Editor

☐ UNRAVELING THE RIDDLE OF ISCHEMIC FINDINGS IN PULMONARY EMBOLISM

☐ To the Editor:

The article by Dasa et al. describes a misleading case of acute pulmonary embolism (PE) showing ST-elevation (STE) in the anterior leads on the electrocardiogram (ECG) in the setting of a cardiac arrest (1).

Although ECG is usually the first procedure performed in the initial assessment of patients with chest pain or dyspnea, ischemic findings can often lead to diagnostic pitfalls in distinguishing between pulmonary embolism and acute coronary syndrome, thus representing a diagnostic challenge in clinical practice.

During massive acute PE with cardiogenic shock, hypotension, hypoxemia, and right ventricular (RV) strain can lead to left ventricular (LV) subendocardial ischemia and RV transmural ischemia. Accordingly, ECG leads that reflect mainly the LV electrical activity (such as I, II, aVL, and V_4–V_6) will record ST-depression (STD), while leads reflecting the border area between the two ventricles (aVR, III, and V_1) will record a net effect from both the RV and LV electrical activity. Specifically, lead aVR directly reflects myocardial ischemia from the right upper cardiac region, including the RV outflow tract and the basal portion of the interventricular septum. Nevertheless, it is also electrically opposed to the left-sided leads, thus reflecting the reciprocal LV subendocardial ischemia. On the other hand, leads III and V_1 face the RV inferior and anterior region, respectively (in case of RV enlargement, also leads V_2–V_3/V_4 will be adjacent to the RV anterior wall) (2).

On the basis of the ratio between RV transmural ischemia and LV subendocardial ischemia, Zhan et al. have previously described three different ECG ischemic patterns in acute PE: 1) dominant LV subendocardial ischemia (presenting STE in lead aVR and STD in leads I and V_4-V_6), which can also occur in the left main coronary occlusion or multivessel coronary artery disease; 2) dominant RV transmural ischemia (showing STE in leads V_1–V_3/V_4); and 3) concomitant LV subendocardial and RV transmural ischemia (characterized by STE in leads III or V_1/V_2, with STD in leads V_4/V_5–V_6) (3).

Furthermore, in acute PE, as well as acute coronary syndrome and Takotsubo cardiomyopathy, the presence of negative T-waves (NTW) in the precordial leads following STE has been speculated to represent an evolutionary "post-ischemic" change as a consequence of transmural myocardial ischemia. The presence of myocardial edema after a prolonged period of impaired overall myocardial perfusion may adduce a plausible explanation for such ECG manifestation, which gradually normalizes when myocardial edema disappears and the patient's hemodynamic status improves (2,4). Another potential mechanism linked with the development of NTW in PE involves the plasmatic surge of several chemical mediators, such as catecholamine and histamine. Specifically, the effect of histamine-induced myocardial ischemia acts directly at the cellular level by uncoupling mitochondrial oxidative phosphorylation, and indirectly by its vasoconstrictive effects on pulmonary circulation (5).

In conclusion, several ECG manifestations of myocardial ischemia may occur in the setting of acute PE with hemodynamic instability. Prompt recognition of such ECG findings, together with a comprehensive echocardiographic assessment, enables a timely differential diagnosis and therapeutic approach.

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☐ RESPONSE TO LETTER TO THE EDITOR

☐ The Reply:

We thank Dr. Scagliola et al. for their insightful comments regarding the electrocardiogram (ECG) patterns observed during acute pulmonary embolism (PE). Our case demonstrates an acute massive PE with cardiac arrest, with the initial ECG demonstrating ST elevation in leads V1 through V4 (1). The ST-elevation myocardial infarction team was activated, but careful attention to the clinical presentation and the critical use of bedside ultrasound were instrumental in making the correct diagnosis and delivering life-saving therapy. We agree with Dr. Scagliola et al. that the ECG findings in acute PE can have significant overlap with acute coronary syndromes. Emergency, pulmonary, and cardiovascular physicians must be familiar with this overlap and be well trained in the use of ultrasound. The ultrasound images from our case demonstrated severe right ventricular dilatation and dysfunction, helping us to make the correct diagnosis.

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