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Integrative Case Study

Suggestive ECG Findings in A Patient with Syncope and Acute Dyspnea

Hari Vivekanantham*

Department of Cardiology, Bern University Hospital, Freiburgstrasse, Switzerland

***Corresponding Author:** Hari Vivekanantham, Department of Cardiology, Bern University Hospital, Freiburgstrasse, Switzerland.

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Case presentation

A 51 -year-old man is brought to the emergency department by the paramedics after having a syncope about 2 hours earlier. The patient came out of his car, suddenly felt lightheaded with loss of strength in his lower limbs, and then passed out. He complains of oppressive left perithoracic pain, which increases on deep breathing, and has been noticing dyspnea, malaise and excessive sudation for the past 3 days. He had had a 7 -hour bus trip about 3 weeks ago and has been having right calf pain for the following 10 days. The past medical history is irrelevant.

On physical examination, the patient is normotensive (BP 126/86 mmHg), tachycardic (pulse 135 bpm), tachypneic (21 breaths per minute), hypoxic (peripheral oxygen saturation 87% under room air) and febrile (tympanic membrane temperature 38.6°C). Heart and lung auscultation, as well as the rest of the physical examination are unremarkable. The following 12-Lead ECG is obtained (Figure 1).

Figure 1: ECG at presentation.

Solution

The ECG shows a sinus tachycardia with a heart rate of approximately 140 bpm. QRS complexes are narrow and axis is normal (QRS axis 80°). PR and QT intervals are in normal range (PR interval 160 ms; corrected QT interval according to Bazett's formula 463 ms). There is poor anterior R wave progression (descending black arrow), defined as the R waves being < 3 mm in V3. Additionally, there is late R to S wave transition, with R wave > S wave only in V6. There is mild ST segment depression in V5 and V6. The T wave appears flat in lead II and aVF, and is inverted in lead III (red arrows). A significant Q wave is present in lead III (red arrowheads). A deep S wave in lead I (black arrowheads), with a RS complex, can be noted.

Figure 2: (with solution): ECG at presentation.

This ECG shows a S1Q3T3 pattern with a deep S wave in lead I, significant Q wave in lead III and inverted T wave in lead III. This is an electrocardiographic evidence of an acute core pulmonale with pressure and volume overload of the right ventricle (RV) which is observed in roughly 10% of cases of acute pulmonary embolism (PE) [1]. Additional electrocardiographic features of RV strain and ischemia include poor anterior R wave progression and T wave inversion in leads V1 through V4 which is termed the "pseudoinfarction" pattern. The latter cannot be noted in our patient's ECG, although low amplitude T waves are present in those leads.

Numerous electrocardiographic manifestations have been described in acute pulmonary embolism including supraventricular arrhythmias, QRS axis/morphology changes, conduction blocks and/or repolarization abnormalities [2]. None of these changes have proven to be sensitive or specific for acute PE. Nonetheless, the history of the patient (i.e. prolonged travelling with ensuing calf pain) and actual clinical presentation (i.e. syncope and sub-acute dyspnea) all suggest an acute PE. The blood work revealed an elevated D-dimer (6940 µg/L) as well as high-sensitivity Troponin I (1208 ng/L). Of note, as the clinical pre-test probability for a PE is high, a dosage of D-dimer would not have been mandatory. Chest CT angiogram confirmed a central, segmental and sub-segmental bilateral PE associated with pleuritis and signs of RV strain.

Using the Pulmonary Embolism Severity Index (PESI) for risk stratification, the patient was classified as PESI Class III i.e. moderate-risk. In the absence of hemodynamic instability or hypotension, and presence of positive cardiac biomarkers as well as radiologic signs of RV dysfunction, he was further classified in the intermediate-high-risk PE category according to the ESC guidelines [3]. The patient had no contra-indication for systemic thrombolysis but refused to undergo such a treatment, should a hemodynamic deterioration occur. Therefore, as hemodynamic instability was clinically evaluated to be imminent, an ultrasound-assisted catheter-directed thrombolysis was performed.

Bibliography

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