Wolff–Parkinson–White Syndrome and Acute Pulmonary Embolism: A Case Report

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A 48-year-old woman admitted to the emergency departments due to sudden onset of dyspnea. The diagnosis of pulmonary embolism (PE) was made and thrombolytic therapy was started. Baseline ECG showed signs of right ventricular overload and after thrombolysis, short PR, wide QRS, and delta wave indicating Wolf-Parkinson-White (WPW) pattern were appeared. In this case report, we discuss ECG of patient with PE mimicking WPW.

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Wolff–Parkinson–White (WPW) syndrome is the classic example of preexcitation which include (1) a PR interval less than 120 msec in duration, (2) wide QRS complex, and (3) an initial slurring of the QRS complex called a delta wave or pre-excitation component. WPW is important in acute coronary syndromes because of mimicking the electrocardiographic (ECG) changes occurring in acute myocardial infarction. Herein, we report a patient with pulmonary embolism, where the ECG changes of WPW syndrome were masked during the acute phase of right ventricular pressure overload.

A 48-year-old woman presented to the emergency department with sudden onset of dyspnea. She was diagnosed with colon cancer 3 years earlier and treated with the FOLFOX 6 regimen (oxaliplatin, folinic acid, and 5-fluorouracil). Twelve-lead ECG revealed sinus tachycardia (142 beats per minute), left axis, incomplete right bundle branch block (RBBB), and T-wave inversions in the precordial leads (V₁ through V₆) and in leads I and aVL. The J point was depressed in V₃–V₆, I, and aVL. Slight ST elevation was present in leads III, aVR, and aVF (Fig. 1). Based on the symptoms, previous history of malignancy, and ECG changes,

acute pulmonary embolism (PE) was suspected. Transthoracic echocardiography showed normal systolic motions of the left ventricle (ejection fraction of 65%) with impaired right ventricular functions, i.e., right ventricle was dilated and systolic motion (Sm) on tissue Doppler was 8.9 cm/sec. Estimated pulmonary arterial systolic pressure was 45 mmHg. Pulmonary CT revealed bilateral massive PE more prominent in the right pulmonary artery (Fig. 2). Thrombolytic treatment with alteplase was initiated and the symptoms were significantly improved after the treatment. Because of sinus tachycardia, beta-blocker (bisoprolol 5 mg daily) was started. On the next day of hospitalization, the patient had ECG changes consistent with WPW syndrome (Fig. 3). The PR interval is short and the delta wave is positive in I, aVL, and V₂–V₆, while the delta wave is negative in lead III. These findings indicate a posteroseptal accessory pathway.

ECG changes in acute PE are important and include T-wave inversions in the precordial leads, signs of right ventricular overload (RBBB, S1Q3T3, etc.), atrial fibrillation, and sometimes ST-segment elevations.¹ The WPW syndrome with its classical

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Figure 1. Twelve-lead admission ECG showing incomplete RBBB with left axis. T waves are inverted in V₁ to V₄ leads and positive in leads V₄–V₆. Note the accentuated left axis deviation indicating left anterior fascicular block.

Figure 2. Pulmonary CT angiography showing massive and bilateral pulmonary embolism being more prominent in the right pulmonary artery.

preexcitation triad of PR-interval shortening, a delta-wave, and QRS-complex widening is a well-known imitator of myocardial injury.² There are some reports about the WPW syndrome mimicking acute myocardial infarction (MI).^{3,4} The WPW syndrome may also hamper identification of ischemic changes on the ECG. In the report by Astorri and Pattoneri,⁵ baseline ECG at rest demonstrated WPW with no evidence of prior MI and, while during an exercise test, 12-lead ECG showed signs of prior anterior wall MI with no evidence of WPW. The authors related this observation to increased sympathetic tone during physical activity, which may enhance conduction over the atrioventricular node (AVN) and unmask the ECG evidence of previous MI. On the other hand, in a case report by Kaya et al.,⁶ normal QRS complexes

Figure 3. ECG after beta-blocker initiation with shortened PR interval (110 msec) and delta-waves.

were present during hospital arrival, while a WPW pattern appeared during chest pain. The ECG during pain in their patient showed sinus rhythm 67 bpm. Hence, increased sympathetic tone, as suggested by the authors, was not necessarily the reason for the preexcitation. One could speculate that increased vagal tone was present, as the ischemic ECG changes were inferolateral.

In our case, the patient had acute PE presenting with enhanced sympathetic tone (tachycardia). After initiation of beta-blocker therapy, the ECG showed WPW syndrome; this change indicates inhibition of the AVN and conduction via the accessory pathway. To the best of our knowledge, this is the first case report in which WPW syndrome was masked in a patient with acute PE.

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