

中文題目：碎裂 QRS 波於病患因肺栓塞合併右心室功能障礙而導致暈厥時提供新的診斷訊息：案例報告

英文題目：Fragmented QRS in a Patient with Syncope – a Novel Hint of Pulmonary Embolism with Right Ventricular Dysfunction :A Case Report

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Introduction

Syncope manifests as initial presentation in about 10% in patients with pulmonary embolism (PE), which is mainly associated with right ventricular dysfunction (RVD). Electrocardiography (ECG) is often the first test performed in the diagnostic flow chart for PE. Several ECG changes, such as T-wave inversion in leads V1-V3, S1Q3T3, sinus tachycardia, and right bundle branch block, were reported as the major findings of PE with RVD; however, these abnormalities did not reach the satisfactory sensitivity yet. Fragmented QRS (fQRS), which has additional spikes within the QRS and denotes conduction disturbance, has been demonstrated to be a marker of acute left ventricular dysfunction. Nevertheless, its relationship with acute RVD has not been reported before. Herein we reported the dynamic change of fQRS in a patient with syncope due to PE with RVD.

Case Report

This 59-year-old man presented to our emergent department (ED) at night with an episode of syncope during climbing upstairs several hours ago. He had no hypertension nor diabetes mellitus before but suffered from exertional chest tightness and dyspnea for a period of time. On arrival, the pulse rate was 65 beats per minute (BPM), blood pressure 124/66 mmHg, and respiratory rate 19 breaths per minute. Grade 2/6 systolic murmur was heard at the left lower sternal border; no respiratory crackles were heard at bilateral lung fields. The room air oxygen saturation was 94%. Arterial blood gas analysis revealed hypoxemia (PO₂ = 65 mmHg). ECG showed normal sinus rhythm with T wave inversion (TWI) from V1 to V4 and fQRS in leads II and aVF. Chest X-ray revealed normal heart size and mild focal oligemia in the right middle lung field. Blood tests showed mildly elevated values of cardiac enzymes (CPK, 294 IU/L; CK-MB 6.4 ng/mL; Troponin I, 0.425 ng/mL) and D-dimer (3,563 mg/L). Acute coronary syndrome (ACS) and PE were initially diagnosed and the patient was treated in coronary care unit (CCU) with antithrombotic therapy, including aspirin, clopidogrel, and low-molecular-weight heparin (fondaparinux). In the next morning, echocardiography showed dilatation of the right heart, D-shaped left ventricle in diastole, and moderate TR. Chest computed tomography (CT) revealed

evident thrombi at the right pulmonary trunk and left upper/lower (mainly lower) pulmonary arteries. To rule out ACS, the patient underwent coronary angiography, which showed normal coronary arteries. Then, pulmonary angiogram showed radiolucent defects in bilateral pulmonary arteries, mainly at right pulmonary trunk and left lower pulmonary artery. Ultrasound-assisted catheter-directed thrombolysis by EkoSonic Endovascular System (EKOS, EKOS corporation, U.S.A.) was planned. EKOS catheters were inserted to the right and left lower pulmonary arteries, respectively. Urokinase infusion was then administered with dose adjustment according to the levels of fibrinogen and D-dimer at CCU for 5 days. During the hospitalization, on the next day after urokinase treatment, the patient's oxygenation status was improved and then he was transferred to the ordinary ward. However, the follow-up ECG still showed more prominent TWI in leads V3 and V4 and new fQRS was found in lead III. The doppler scan of the lower limb did not reveal evidence of deep vein thrombosis. Two days later, echocardiographic follow-up showed decrease of right heart size and disappearance of D-shaped left ventricle. He was then discharged and regularly followed up at the out-patient department (OPD) with use of rivaroxaban 15 mg bid. One week after discharge, ECG showed only shallow TWI in precordial leads but still obvious fQRS in inferior leads. Follow-up CT showed residual thrombi. One month after discharge, ECG showed nearly normalization of TWI but persistent fQRS in inferior leads. Three months later, ECG showed sinus bradycardia (54 BPM) with nearly disappearance of fQRS in leads II and III. During the follow-up, the chest tightness and exertional dyspnea were much improved.

Conclusion

To our knowledge, we first reported a dynamic change of fQRS in inferior leads in a patient with syncope due to PE. This finding later provided a potential link to acute RVD. Therefore, we suggest that systemic evaluation for PE in syncopal patients at the ED or OPD with fQRS in inferior leads would be considered.

Keyword: Fragmented QRS, Syncope, Pulmonary Embolism, Right Ventricular Dysfunction