International Heart and Vascular Disease Journal
Volume 3, Number 6, June 2015

Journal of the Cardioprogress Foundation

CLINICAL CASE

Acute pulmonary embolism complicated with coronary slow flow in a morbidly obese patient: a case report

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Summary
Pulmonary embolism (PE) is frequently misdiagnosed as acute coronary syndrome (ACS) due to common symptoms and electrocardiographic (ECG) findings. Coronary slow flow contributes to ECG changes observed in acute pulmonary embolism. In morbidly obese patients, the efficacy of a fixed dose of novel oral anticoagulants (NOACs), which are commonly used for treatment, needs further investigation.

Keywords
Pulmonary embolism, slow flow, obesity

Introduction
Pulmonary embolism (PE) is still one of the leading causes of hospitalization, morbidity and mortality [1]. Numerous electrocardiographic (ECG) findings have been defined for PE diagnosis and ECG changes that raise suspicion of acute coronary syndrome (ACS) can cause delays in PE treatment. Although the reasons of ECG changes are not well determined, right ventricular strain and paradoxical embolism have been implicated [2,3]. Coronary slow flow presented with ECG changes triggered by or incidentally accompanied with pulmonary embolism has not been reported so far. Here we report a clinical case on a morbidly obese patient who presented with deep T wave inver-

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sion in V1–6 leads and was diagnosed with acute PE and coronary slow flow, and we also aim to discuss the safety and efficacy of anticoagulant therapy with novel oral anticoagulants (NOACs) in morbidly obese patients.

Case report

A 39-year-old male patient was admitted to emergency department with chest pain and palpitation symptoms. The patient had no history of coronary artery disease. His body mass index (BMI) was 42.6 kg/m^2 and body surface area was 2.5 m^2. He had uncontrolled hypertension, hyperlipidaemia, and was an active-smoker. He was admitted to another medical centre with chest pain and could not complete an exercise stress test due to fatigue. Physical examination revealed blood pressure of 100/60 mmHg and heart rate of 97 beats per minute. His creatinine level was 0.99 mg/dL, white blood cell count 10.9 K/mm^3, haemoglobin 15.5 g/dL, platelets 212 K/mm^3 and troponin I 0.028 ng/mL. His ECG showed deep T wave inversion in V1–6 leads at admission (Figure 1). Ejection fraction of 55% was recorded and mild mitral regurgitation was noted. Because of impaired and limited imaging quality during ECG examination, it was not possible to evaluate the right chambers of the heart. A preliminary diagnosis of ACS was made and the patient was admitted into catheterisation laboratory for coronary angiography. The angiography demonstrated significant coronary slow flow and aneurysms across the left anterior descending and right coronary arteries. The patient was transferred to the intensive coronary care unit for medical therapy and follow-up. His chest pain decreased significantly and during arrangements for his discharge he began to suffer shortness of breath and D–Dimer levels were found elevated. A prompt diagnosis of PE was considered and computerised tomography (CT) pulmonary angiography was performed. CT images revealed PE in segmental and subsegmental branches of the bilateral pulmonary artery (Figure 2). The pulmonary embolism severity index (PESI) of our patient was calculated as Class IV whereas his simplified PESI score was 4 [4]. Fibrinolytic therapy was administered via intravenous route and no bleeding complication was observed. Following fibrinolytic therapy, shortness of breath was abolished and sinus tachycardia resolved along with increased oxygen saturation levels. T wave inversion in V4–6 leads diminished on ECG (Figure 1). Lower extremity venous Doppler ultrasound demonstrated acute vein thrombosis in the right popliteal

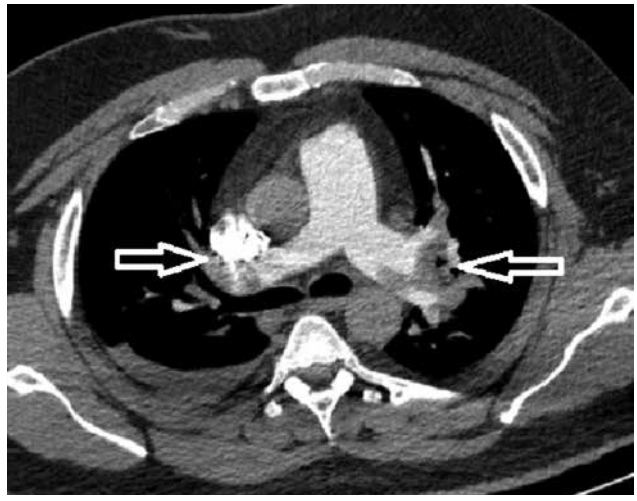

Figure 2. CT images revealed pulmonary emboli in right and left pulmonary arteries (red arrows)

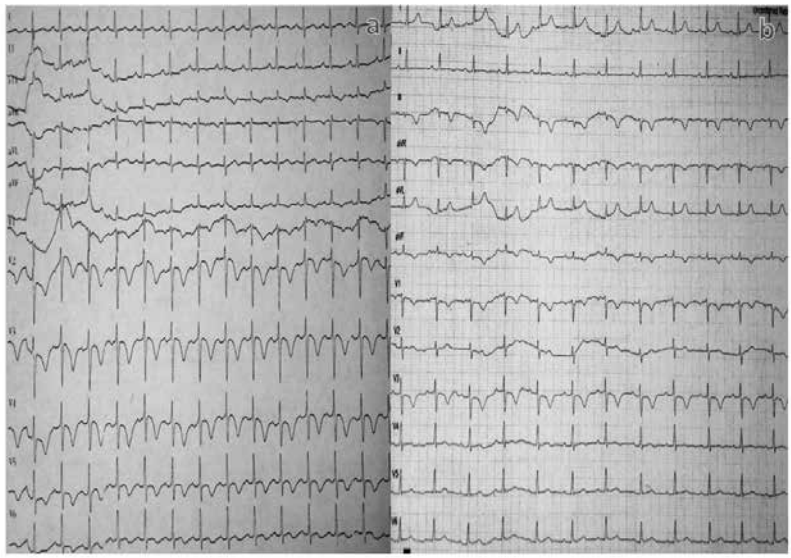

Figure 1. ECG revealed sinus rhythm, deep T wave inversion in V1–6 leads (a) and recovery of T wave inversion in V4–6 leads following treatment is shown (b)

vein. Warfarin therapy was initiated for long-term anticoagulation therapy.

Discussion

PE is a life-threatening disease and diagnosis is challenging when it presents with non-specific symptoms and non-specific clinical findings. Presence of chest pain along with increased cardiac biomarker levels often lead to misdiagnosis in particular ACS. Chest pain may occur as a result of pleuritic pain associated with pulmonary infarct and ischaemia secondary to increased right ventricular pressure [5]. Sinus tachycardia on ECG is recognized in most of the cases, though it is not a specific finding. Besides well-recognized S1Q3T3 pattern, inversion of T waves in V1–4 leads is indicative of right ventricular strain. First-degree atrioventricular block, complete or incomplete right bundle branch block, right axis deviation, and atrial arrhythmias including most frequent atrial fibrillation can be seen [2].

ST elevation in anterior or inferior leads has been reported in several PE cases, although the common ST-segment change is ST depression in V1–4 leads [6]. Our case demonstrates deep T wave inversion in all V1–6 leads. This indicates that involvement of heart tissue is not limited to the right heart. Through patent foramen ovale (PFO), paradoxical embolisms occurring during acute PE have been implicated in aetiology and may lead to coronary embolism and ST elevation [3]. After all, the causal relationship is still not clear. In our case, we assume that severe coronary slow flow in the left anterior descending and right coronary artery, hypotension in acute PE, and increased right-heart pressures may have caused compromised coronary flow during diastole. Moreover, reduced pulmonary flow, decreased preload and cardiac output may be the reasons for ECG changes. Nevertheless, absence of similar ECG findings in all acute PE cases may be due to aneurysmatic coronary arteries and coronary slow flow leading to deeper coronary ischaemia in our case.

Obesity and male gender are well-known predictors of coronary slow flow [7]. Understanding of the relationship between PE and coronary slow flow with further research is required for the enlightenment of aetiology.

Warfarin is a well-established anticoagulant agent in the secondary prevention of acute PE. However, NOACs are increasingly used for treatment of PE. They do not require dose adjustment for patient's body weight and are used in a fixed dose. We planned to use a NOAC for long-term treatment in accordance with guideline Class I recommendation [4] but the fixed dose in our morbidly obese patient made us doubtful about efficacy. It is widely accepted that creatinine clearance is increased in morbidly obese patients, and increased glomerular filtration rate may increase drug clearance. An acute cerebrovascular accident during dabigatran treatment in an overweight patient has been reported [8], however, there are studies declaring no difference in efficacy of treatment between patients with low and high BMI values [9,10]. The RE-LY, EINSTEIN-PE and AMPLIFY trials demonstrated that NOACs have a similar efficacy and safety with standard therapy but, it should be noted, the study participants with a body weight >100 kg only comprised 14.3–19.4 % of the total population studied. Further studies are required in order to understand the efficacy and safety profile of NOACs in morbidly obese patients [11,12,13].

Conclusion

Coronary slow flow may be the underlying mechanism responsible for these projections. Efficacy and safety of long-term anticoagulant therapy with NOACs in morbidly obese patients need further investigation.

Conflict of interest: None declared

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