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Case Report

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Missed acute pulmonary embolism and sudden death: A case report

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ABSTRACT

Rationale: Pulmonary embolism is a severe cardiovascular disease. Acute pulmonary embolism is an extremely common and potentially the serious pattern of venous thromboembolic disease. Unfortunately, missed diagnosis of pulmonary embolism is lethal and common because of its non-specific symptoms and signs.

Patient concerns: A 42-year-old male patient presented with acute chest pain that was treated as gastroesophageal reflux disease.

Diagnosis: Suspected acute pulmonary embolism.

Interventions: O_2 inhalation, urgent electrocardiography, and cardiopulmonary resuscitation.

Outcomes: Deterioration and sudden cardiac death.

Lessons: Physicians should pay much attention to the symptoms and signs of pulmonary embolism to reduce the rate of missed diagnosis.

1. Introduction

Pulmonary embolism (PE) is an important manifestation of venous thromboembolism (VTE), which is the third most frequent fatal cardiovascular disease[1,2]. Indeed, the annual incidence of PE is 100-200 per 100 000 inhabitants[2]. Nearly, 33.4% of patients with VTE presented with PE, and 66.6% manifested with deep vein thrombosis (DVT)[1]. Approximately, 500 000-600 000 cases of acute PE (APE) were reported yearly in the US[3]. Nearly, 150 000-200 000 of APE patients represented either primary or secondary cause of death[3]. The mortality rate of treated APE ranged from 3% to 8%, but the rate elevated to around 30% in untreated cases[3]. The lower limb DVT was reported in nearly 70% of sustained APE, with the remaining 30%, has already become detached embolism. On contrariwise, APE occurred in approximately 50% of proximal lower limb DVT[4]. APE is a prevalent and potentially-lethal disease with high morbidity and mortality[5]. APE may be classified into three categories: massive, sub-massive, and non-massive[6]. Massive APE is defined as hemodynamic instability or systolic hypotension, lower than 90 mmHg. The cases of massive APE are the most serious class that indicates prompt thrombolytics. Sub-massive APE refers to the presence of right ventricular dysfunction (RVD) without hemodynamic instability[5]. Though RVD can be firstly diagnosed with echocardiography, the sub-massive APE carries higher morbidity and mortality than those without RVD[5]. Otherwise, the APE not under the definitions of massive or sub-massive embolism is usually named as non-massive type[5]. Confirmed APE is defined as a probability of PE high enough to indicate the need for PE-specific treatment[2]. Excluded APE is a low likelihood of APE enough to withholding the specific treatment[2]. The risk factors of APE are as followings; postoperative

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period, old age, trauma, prolonged immobilization, post-infarction period, hyperhomocysteinemia, heart failure, obesity, gestation, oral contraceptive pills or hormone replacement therapy, polycythemia, and malignancy[2,4,7]. Absence of risk factors may happen in sporadic cases of APE[2,4,7]. Chronicity and disability are the possible and preventable outcomes of APE[2]. Indeed, approximately 10% of symptomatic APE causes sudden cardiac death within 60 min after the onset[5]. APE may be entirely asymptomatic and may be diagnosed coincidentally during investigations or autopsy[2]. All of the manifestations of APE are suggestive but they are non-specific or not-sensitive to diagnosis[2,8]. It is important for physician to be oriented with the following percentage of clinical symptoms and signs of suspected pulmonary embolism according to the literature reports: dyspnea (65%), pleuritic chest pain (47%), substernal chest pain (33%), pre-syncope or syncope (13%), haemoptysis (9%), fever with temperature higher than 38.5 ℃ (28%), cough (17%), signs of unilateral DVT (33%), totally asymptomatic (16%), sudden death (25%), sinus tachycardia with heart rate more than 100/min (26%), tachypnea with respiratory rate more than 20/min (75%), hypotension with shock (20%), hypoxaemia on arterial blood gases (75%), electrocardiographic right ventricular hypertrophy (50%) and radiological; atelectasis (49%), pleural effusion (51%), raised hemidiaphragm (36%), peripheral opacification with infarct (33%), and lung oligemia (36%)[2,5,7,9,10].

During diagnosis, clinical stability, and the pre-test probability will determine the diagnostic approach[2,8]. APE is suspected by the signs of dyspnea, chest pain, pre-syncope, syncope, and hemoptysis[2]. Hypotension and cardiogenic shock are rare symptoms that can indicate PE[2,7]. Chest pain is a frequent symptom of APE and is usually caused by pleural irritation due to distal emboli resulting in pulmonary infarction. For patients with central PE, chest pain may have typical angina[2] or have acute and severe dyspnea; while for patients with small peripheral PE, these symptoms are often mild and transient. However, deterioration of dyspnea may be the only sign indicative of APE especially in patients with preexistent HF or respiratory disease[2]. Clinical probability should be assessed in all suspected cases of APE[2]. Different diagnoses should always be considered until APE exclusion[11]. Testing of d-dimer should be used after the evaluation of clinical probability[11], but the testing should not be done in the patients with a higher clinical probability of APE. A negative d-dimer test strongly excludes APE in the cases of low or intermediate clinical probability[11]. Computed tomography pulmonary angiogram (CTPA) is a choice of radiological lung modality for non-massive APE, and negative CTPA indicates no further investigation or treatment for APE[11]. CTPA or echocardiography will strongly confirm the massive APE[11]. The electrocardiography (ECG) is often abnormal in APE, and only 33% of patients have normal ECG[12]. Lack of specificity and sensitivity of ECG signs pose a challenge to the diagnosis of APE[12]. The most common ECG findings in APE patients are sinus tachycardia, right bundle branch block, T-wave inversion/ST-segment deviations (V1-3), low QRS voltage, McGinn-White sign, and right axis deviation[12]. Anticoagulation is the mainstay of VTE treatment[2]. Surprisingly, the low-molecular-weigh heparin is a choice of therapy for inpatient cases[1]. However, transient risk factors may affect the final decision on the duration of anticoagulants after the first attack of APE[2]. The current guidelines encourage thrombolytic drugs in the cases with hypotension or shock secondary to APE[9]. Unstable APE with minimal bleeding will be alleviated by thrombolytics[1] that is the therapeutic choice for massive APE[11].

2. Case report

A 42-year-old married, Egyptian male accoutant presented with severe acute chest pain. Profuse sweating, dizziness on sitting, and increased breathing were the associated symptoms. An informed consent has been signed by the patient's family. The patient gave a recent history of burning sensation in his chest one week ago, and he denied the history of cardiovascular diseases, smoking, drugs or any special habits. He was managed by internist physician as gastroesophageal reflux disease. Omeprazole capsule (20 mg, twice daily), ranitidine tablet (150 mg, twice daily) were prescribed for one week. The general condition became more deteriorated and the chest pain gradually worse. There was no any signs of improvement rather than deterioration. Chest pain had become compressible. The patient was brought by his family for consultation from the cardiologist (author) at 2 o'clock after midnight at the clinic outpatient. The cardiologist urgently called the ambulance for hospital referral. O_2 inhalation was given with generator (100%, by nasal cannula, 5 L/min) before the ambulance arrived. Urgent ECG was done. Unfortunately, the cardiac arrest had happened. Cardiopulmonary resuscitation according to the current guidelines was proceeded by the cardiologist at his clinic outpatient but regrettably with no response. Upon general physical examination, the patient undergone tachypnea, central cyanosis, dyspnea, severe sweaty, and had cold extremities, with a regular heart rate of 135 bpm, blood pressure of 70/40 mmHg, respiratory rate of 60 bpm, the temperature of 36.1 ℃, pulse oximeter of O_2 saturation of 63% (normally; 95%-100%) and tachycardia on heart auscultation. No more relevant clinical data were noted during the clinical examination. The case was initially managed as a suspected APE with shock. Massive APE was the most probable diagnosis. Several ECG tracings were taken that showed sinus tachycardia, S1Q3T3 pattern, incomplete right bundle branch block, P-pulmonal, and ST-segment depressions in V1-3 leads (Figure 1). The only measured random blood sugar was 213 mg/dL (normally ≤200 mg/dL). No more workup was done.

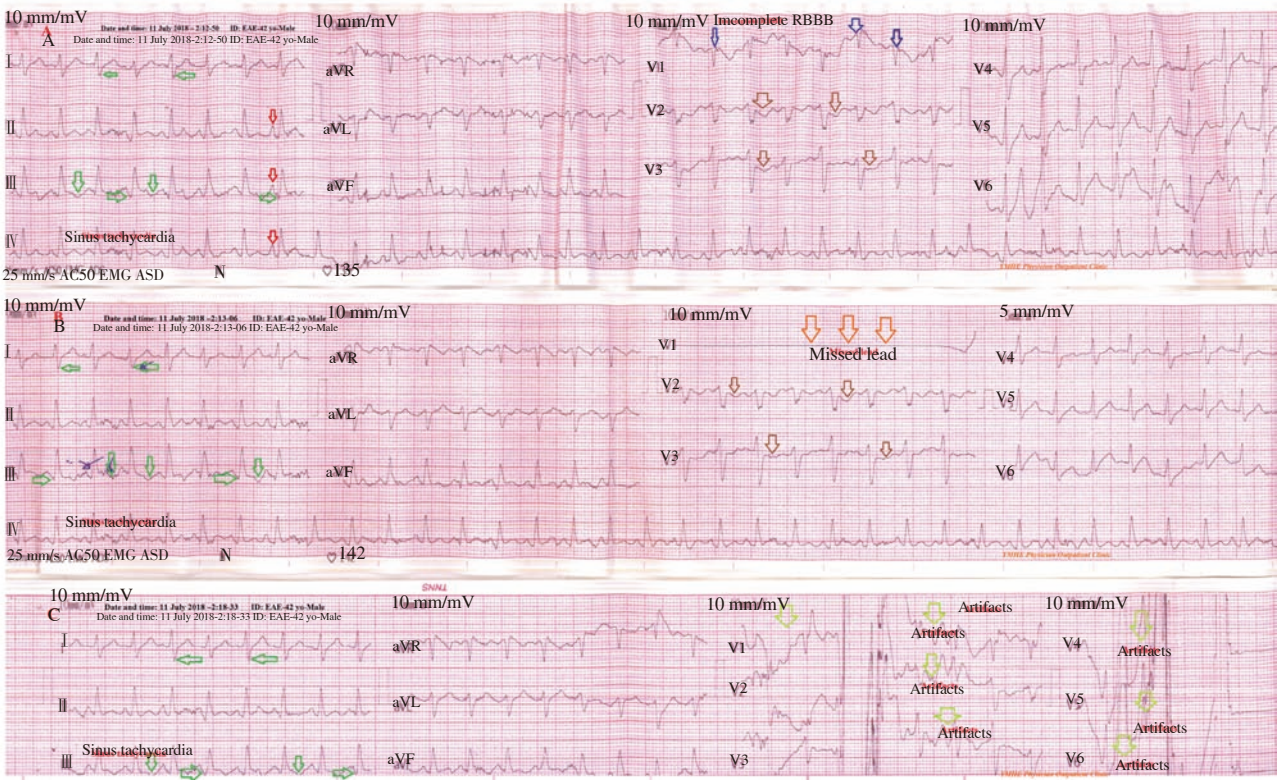

Figure 1. Serial ECG tracings. A-tracing showing sinus tachycardia (HR;135 bpm), an incomplete RBBB, S1,Q3,T3(green arrows), P-pulmonal(red arrows), and RV strain (ST-segment depressions in V2,V3 leads) (brown arrows). B-tracing showing like A with missed V1 lead due to profuse sweating. C-tracing showing like A with artifacts (V1-6) lead due to irritability (Lemon arrows).

3. Discussion

This APE case started with mild phase and progressed to massive embolism. Pleuritic chest pain was indicating mild APE or pulmonary infarction. But compressive anginal chest pain, hypotension, and central cyanosis were serious signals for massive APE. Sinus tachycardia may be presented with both fatal and non-fatal embolisms. McGinn-White sign or S1Q3T3 pattern is presented in about 8% of massive APE but with may normal variant. An incomplete right bundle branch block with anterior ST-segment depressions in V1, V3 leads, P-pulmonal are mostly signs of acute severe RVD.

The death in this case is due to ignorance in clinical diagnosis. The patient was not advised for urgent d-dimer assay or computed tomography pulmonary angiogram per se, and was not considered as APE at the beginning. Accurate diagnosis of APE would help save the life. Physicians should pay more attention on related symptoms and signs of APE.

It is important to make differential diagnosis among several diseases, such as acute coronary syndrome, aortic dissection, acute pericarditis, asthma, pneumonia, chronic obstructive pulmonary disease exacerbation, acute pulmonary edema, aortic dissection, pneumothorax, bronchogenic carcinoma, primary pulmonary hypertension, chest trauma with rib fracture, musculoskeletal chest pain, anxiety disorders, and hysteria. We can't compare the current case to other studies due to absent of similar condition. It is recommended to pay more attention to the identification of APE.

Conflict of interest statement

The author reports no conflict of interest.

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