CASE REPORT

A rare presentation of pulmonary thromboembolism as seizure

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ABSTRACT

Acute pulmonary thromboembolism is a critical and sometimes fatal event that is difficult for clinicians to diagnose because of its various initial manifestations. Here, we report a previously healthy 50-year-old man without any history of seizures who presented to the emergency ward with the new-onset seizure. Neurology consult was performed, but found no focal neurological deficits. The diagnosis of massive pulmonary embolism was confirmed by echocardiography and pulmonary CT angiography. Alteplase and heparin with therapeutic dosage were started for the patient. After initiating treatment, patient's dyspnea, arterial O2 saturation, and general condition were significantly improved. Echocardiography was repeated and showed a smaller right ventricle size and lower pulmonary artery pressure than the first echocardiography.

KEYWORDS: pulmonary thromboembolism; seizure; massive pulmonary embolism; thrombosis

INTRODUCTION

Acute pulmonary thromboembolism (PTE) is a common and often fatal venous thromboembolic disease complication. It is the third leading cause of death from cardiovascular disease, accounting for more than 250,000 hospital admissions in the United States hospitals each year [1-3]. Diagnosis of acute PTE is often challenging because clinical manifestations can mimic many diseases and lead to a delay in diagnosis and treatment [4]. The new onset of an unexplained seizure in adults without a history of seizures and structural damage to the brain requires careful examination to find the etiology; interestingly, some separate reports have described seizure as the first manifestation of acute PTE [5]. Herein, we reported a rare case of a healthy man without any previous history of seizure who had experienced massive PTE manifested by a new-onset seizure.

CASE PRESENTATION

A 50-year-old man without any past medical history of seizure, presented with generalized tonic-clonic seizure to our emergency ward. Three days before this occasion, he had shortness of breath while swimming, which led him to stop swimming. Then, his symptoms resolved with rest. On the morning of presenting to the hospital, while he was climbing the stairs at home, he experienced shortness of breath again, even continued after sitting on a chair. According to his family, he had tonic-clonic movements similar to a seizure for about 40 seconds and a post-ictal period of about 15 minutes.

The patient used to work as a framer 10 years ago. As a result, he had had chronic mercury exposure and further toxicity, which caused him mild lung dysfunction but the patient's pulmonary function class has been good since then. The patient was able to perform his daily routine activities and had no complaints. He didn't have any drug history and was a non-smoker with no alcohol and substance abuse history.

When admitted to the emergency room, the patient was hemodynamically stable, with a blood pressure of 110/70 mmHg, pulse rate of 90 beats per minute, respiratory rate of 23 breaths per minute and an arterial oxygen saturation of 94% in room air. He had a Glasgow coma scale of 15/15 with normal routine neurological examination and the power in all of the limbs was 5/5. Pupils were mid-sized, bilaterally equal and reactive to the light. He had bilateral lower extremities edema.

A neurology consult was performed and levetiracetam was started for the patient. In the laboratory tests, the patient had three high serum cardiac troponin I levels but the electrocardiogram showed no ST-segment changes. Instead, right bundle branch block (RBBB), negative T wave in V1-3 due to RBBB, S1Q3T3 aspect, and QR aspect in V1 were present (Figure 1). Details of the laboratory data are presented in Table 1. The patient was transferred to the critical care unit where due to recurrent dyspnea, worsening tachycardia and decreasing arterial oxygen saturation he

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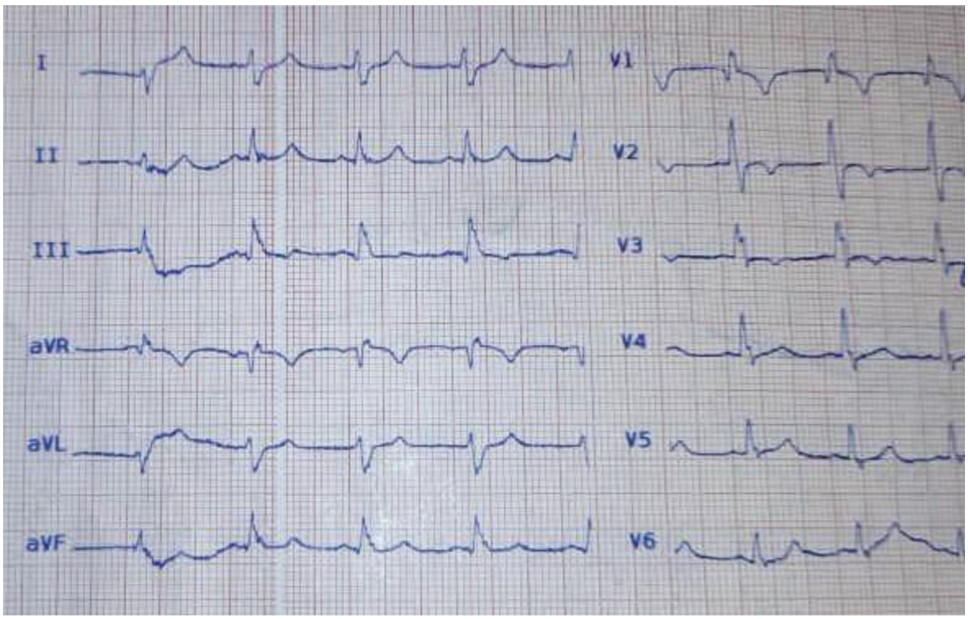

Fig. 1. Initial electrocardiography demonstrating no ST segment change, right bundle branch block (RBBB), negative T wave in V1-3 due to RBB, S1Q3T3 aspect, and QR aspect in V1.

Table 1. Patient's laboratory data.

	2022/1/21	2022/1/22	2022/1/23	2022/1/24	2022/1/25	2022/1/26	2022/1/27
WBC (x10^3 per mm^3)	12.7	15.6		6.7	7.1	7.7	8
Lymphocyte (%)	30	19.2		18	29	29.4	34.6
RBC (x10^6 per mm^3)	5.8	5.99		4.9	5.3	5.54	5.63
Hb (g/dl)	13.4	12.9		11.4	12	12.3	12.2
Platelet count (x10^3 per mm^3)	170	141		112	149	149	197
PT (sec)	13.3	17.3	22	16.2			
PTT (sec)	23.5	67.1	29.7	27.7			
INR	1.20	1.56	2.17	1.49			
VBG							
PH	7.34	7.37	7.34	7.44	7.39	7.44	
PCO2 (mmol/l)	37.8	27.8	34.8	34.3	47.7	35.5	
HCO3 (mmol/l)	20.3	15.8	18.4	23	28.2	24	
BE	-4.7	-7.9	-6.5	-0.7		0.3	
BUN (mg/dl)	37.5	72.7		79.4	45	38.5	32.1
Creatinine (mg/dl)	1.28	3.66		1.70	1.11	1.07	1.05
Na	141	138	141	141	143		142
K	3.7	6.4	4.1	4.1	3.9		3.6
Troponin I (normal range $<$0.12)	0.21	1.42	1.96				
CpK	78	140	167				
CKMB	19	28					

Abbreviations: BUN, blood urea nitrogen; CKMB, creatine kinase MB; Cpk, creatine phosphokinase; Hb, hemoglobin; INR, international normalized ratio; K, potassium; Na, sodium; PT, Prothrombin time; PTT, Partial thromboplastin time; RBC, red blood cell; VBG, venous blood gas; WBC, white blood cell.

underwent transthoracic echocardiography. Severe right ventricular (RV) enlargement was present, which was consistent with PTE. Pulmonary CT angiography was performed and the diagnosis of massive PTE was confirmed (Figure 2). After performing pulmonary CT angiography, the patient developed hypotension and was subsequently treated with 100 milligrams alteplase intravenous infusion in 2 hours, and unfractionated heparin with bolus dose of 80 IU followed by 18 IU/kg which is a therapeutic dose for pulmonary embolism based on AHA guidelines [6]. After receiving alteplase, patient's dyspnea, arterial O2 saturation, and general condition were significantly improved. Echocardiography was repeated 24 hours later, which showed a smaller RV size and lower pulmonary artery pressure than the first echocardiography.

Lower extremities ultrasonography with color Doppler was performed and showed obstruction in left superficial femoral, left external iliac vein, and right popliteal vein due to thrombosis. Thrombosis in multiple locations and massive pulmonary embolism raised our concern regarding the underlying etiology of thromboembolism such as malignancies and hypercoagulable states. A consult with a hematologist/oncologist was scheduled for the patient who yielded no positive results after performing full

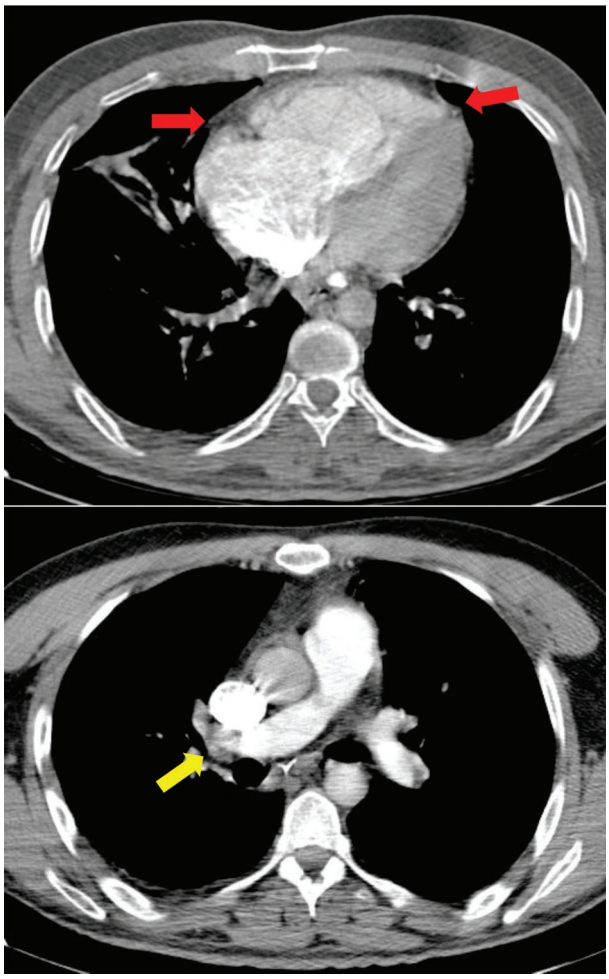

Fig. 2. Continued to next column.

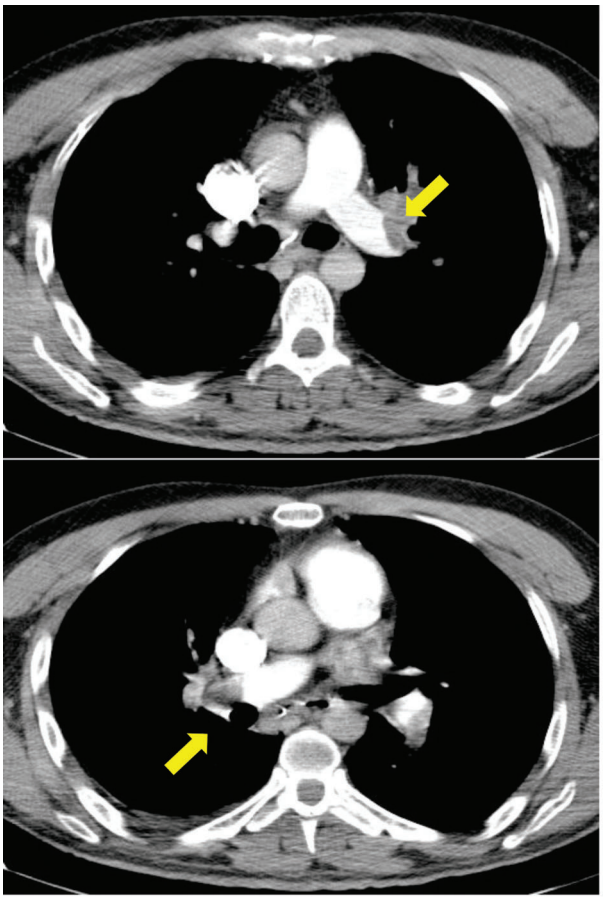

Fig. 2. Pulmonary CT angiography showing thrombosis in pulmonary arteries (yellow arrows) and RV dilation (red arrows).

work-up for malignancies and thrombophilia. The patient was discharged after 7 days in good general condition with Apixaban 5 milligrams twice a day.

DISCUSSION

In this case, we presented a rare report in which the patient came to the hospital with a new-onset seizure which neurological causes were excluded. He was hospitalized due to high cardiac troponin levels and subsequently was diagnosed with massive PTE. In 1995, a previously healthy 37 year-old man was presented with sudden syncope and generalized seizures. The patient developed dyspnea during hospitalization and despite receiving intravenous tPA, he died 6 hours after the onset of symptoms. The autopsy confirmed the presence of massive PTE [6]. In another report, a 50-year-old man was reported with sudden syncope and seizure. Although he denied shortness of breath, the D-dimer test was positive and the perfusion-ventilation scan was positive for bilateral PTE. Echocardiography also confirmed an increase in right ventricular pressure, and he was eventually diagnosed with acute PTE [7]. Respiratory acidosis, hypoxemia, and cerebral hypoperfusion due to decreased cardiac output are suggested to be the pathophysiology behind this clinical presentation. Therefore, PTE should be considered in the differential diagnosis of new-onset seizures and other unexplained seizures [8].

Seizure is classified as an atypical presentation of PTE which occurs in less than 1% of PTE cases [9]. PTE has different etiologies such as venous stasis as in immobilization, surgery, trauma, hypercoagulable states as in pregnancy, oral contraceptive consumption, estrogen replacement therapy, malignancy, and hereditary deficiency of factors and anti-phospholipid syndrome. Mortality in PTE patients who present with seizure have been estimated as approximately 55%, mostly due to the delay in diagnosis [9].

Another recent case report describes a 76-year-old man who presented to the emergency department after two tonic-clonic seizures at home. However, he had a history of a seizure four years ago but was not prescribed any medication. In the emergency room, he had resting tachycardia and 92% arterial oxygen saturation in room air. Electrocardiography and D-dimer were examined and both were abnormal. Computed tomography scans were positive for multiple pulmonary emboli. The patient was given the appropriate dose of enoxaparin and was hospitalized [10].

Assessment of the severity of PTE at the first presentation is an important prognostic factor which is mostly based on hemodynamic instability. Patients with high-risk or massive PTE present with hemodynamic instability. These patients have a mortality rate of approximately 30% within one month [11]. Therefore, the diagnosis and management of high-risk PTE patients are pivotal. Treatment of the patients

with high-risk PTE includes systemic thrombolytic therapy and anticoagulation with unfractionated heparin if not contraindicated. In patient with low- or intermediate-risk of PTE, further risk stratification is assessed by an overall consideration of the clinical, laboratory, and imaging indicators of PTE severity combined with patients' comorbidities. Pulmonary embolism severity index (PESI) is the most validated, widely-used score for the risk stratification of hemodynamically-stable PTE patients [12,13].

In conclusion, in this case, we described a previously healthy 50-year-old man with a new-onset seizure whose diagnosis of pulmonary embolism was confirmed by echocardiography and pulmonary CT angiography. Our findings, and similar findings in other reports, emphasize that physicians should be more aware that pulmonary embolism is essential in the differential diagnosis of unexplained new-onset seizure.

Conflict of Interests

The authors have no conflict of interests to declare.

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Ethical Statement

A written informed consent was obtained from the patient. All of the authors declare that confidentiality of the patient was respected.

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Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author.

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