Case Report

Acute pulmonary embolism with loss of consciousness as the first manifestation: a case report

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Background: The clinical manifestations of pulmonary embolism are varied, and atypical pulmonary embolism can easily be missed in some patients, resulting in serious clinical consequences and injuries.

Case Description: This report describes a rare case of acute pulmonary embolism with loss of consciousness as the first manifestation. A 50-year-old male was admitted with loss of consciousness and difficulty breathing. Acute coronary syndromes and neurological disorders such as seizures were excluded by clinical history and electrocardiogram dynamic changes. Multiple clues such as coagulation function and myocardial enzymes are highly suggestive of pulmonary embolism, after the completion of computed tomography pulmonary angiogram (CTPA) diagnosis, the severity of the acute pulmonary embolism was evaluated, after which the patient was given low-molecular-weight heparin sequentially overlapping with oral warfarin as the anticoagulation treatment. Following this, the life signs of the patient were stable, and there were no special complaints; thus, this patient was discharged smoothly. As of this writing, the patient is still being followed up clinically with no recurrent embolism or deterioration occurred.

Conclusions: This case is of guiding significance for the early detection and rapid diagnosis and treatment of such patients with pulmonary embolism. It is necessary to acquire the vital signs, including those related to heart rate, electrocardiography, respiration, and blood oxygen saturation in the first clinical contact for patients with syncope as soon as possible. Patients with problems related to the above-mentioned basic vital signs should be highly suspected of cardiopulmonary diseases, and CTPA should be performed as soon as possible after the evaluation of the clinical possibility of pulmonary embolism and D-dimer screening. Moreover, the critical degree of pulmonary embolism should be evaluated, and then reperfusion or anticoagulation treatment should be performed appropriately. This should be followed by etiology screening. To avoid recurrence or aggravation of pulmonary embolism, the cause of the disease should be determined and treated.

Keywords: Loss of consciousness; pulmonary embolism; clinical possibility; etiology screening; case report

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Introduction

Pulmonary embolism is a common thromboembolic disease in clinic. Its typical clinical manifestations include dyspnea (more than 50%), pleural pain (39%), cough (23%), retrosternal pain (15%), fever (10%), hemoptysis (8%), syncope (less than 5%), unilateral limb swelling (24%), and unilateral limb pain (6%) (1). Acute pulmonary embolism is a clinical and pathophysiological syndrome of

pulmonary circulation obstruction caused by endogenous or exogenous emboli blocking the main artery or branches of the pulmonary artery. It is a critical and severe disease with a high mortality rate. However, due to the lack of sufficient attention to the prevention and treatment of the disease for a long time, doctors often miss the diagnosis. The vast majority of these patients have disease causes, such as lower limb or pelvic vein thrombosis, long-term bedridden or inactive, chronic cardiopulmonary disease, surgery, trauma, malignant tumors, pregnancy and oral contraceptives. Treatment methods include anticoagulation, thrombolysis and interventional therapy (2).

Syncope can be the only first symptom of pulmonary embolism, but fewer than 1% of patients with pulmonary embolism have syncope or loss of consciousness as the main symptom (2). Pulmonary embolism is not a routine screening item in the differential diagnosis of syncope in our region. The patient described in this report was a rare case of clearly diagnosed acute pulmonary embolism with loss of consciousness as the first manifestation. If such patients are not detected in time and continue to undergo a series of screening and examination on the cause of syncope in the outpatient clinic according to the general syncope patients, it will lead to a series of serious consequences such as deterioration of the condition of such patients and even death. We present the following article in accordance with the CARE reporting checklist (available at https://atm.amegroups.com/article/view/10.21037/atm-23-656/rc).

Case presentation

The patient was 50-year-old male, who 2 days before admission, had difficulty breathing when standing at home after bending over and working, which was accompanied by shortness of breath and amaurosis. He subsequently fell to the ground unconsciousness, but experience no fall injuries. After being unconscious on the ground for 10 seconds, the patient regained consciousness and had experienced no convulsion when falling, no rolling up of the eyes, and no incontinence. There was no dizziness, headache, chest tightness, chest pain, nausea or vomiting, palpitation, or sweating before the onset of loss of consciousness. After waking up, the patient continued to have dyspnea, general fatigue, intermittent shortness of breath, and chest pain. After that, the patient developed intermittent dry cough but no fever or expectoration, no dizziness or headache, no palpitation or back pain, and no residual neurological symptoms and was able to walk. He complained that when he went up the stage, he had increased shortness of breath and amaurosis but no chest pain or fainting. Moreover, the patient had no history of chest distress, chest pain, or asthma. He could lie down quietly and sleep at night. For further diagnosis and treatment, he visited to the cardiology department of Beijing Anzhen Hospital. From the onset of the disease, the patient had a clear mind, good spirits, normal stool and urine, and no significant change in weight. He had a history of gout for about 3 years. Before admission, he had experienced gout attacks and swollen and painful feet. The patient had oral administration of Celebrex and sodium bicarbonate 1 month before admission which reduced the activity of both his lower limbs. He had a history of varicose veins of the lower limbs for 20 years and a history of fatty liver for 10 years. The patient underwent radiofrequency ablation for paroxysmal atrial fibrillation 3 years prior in our hospital, and there was no recurrence during postoperative follow-up. His father had a history of high blood pressure, diabetes, and pulmonary embolism, while his mother had coronary heart disease. Physical examination results on admission were the following: temperature, 36.5 ℃; pulse, 82 times/minute; respiration, 16 times/minute; and blood pressure, 131/74 mmHg. The superficial lymph nodes of the whole body were not palpable or swollen. The breath sounds in both lungs were clear, no dry or wet rales were heard, the heart boundary was normal, the heart rhythm was regular, and no pathological murmur could be heard in the auscultation area of each valve. P2 showed no hyperactivity, the abdomen was flat and soft with no tenderness, there was no rebound pain or muscle tension, and bowel sounds were normal. The patient had mild edema in both the lower limbs but was

Highlight box

Key findings

- Patients admitted with the primary clinical presentation of loss of consciousness alone may have pulmonary embolism without the typical signs.

What is known and what is new?

- The clinical manifestations of pulmonary embolism are varied.
- Loss of consciousness may also be an atypical clinical manifestation of pulmonary embolism.

What is the implication, and what should change now?

- For patients admitted to hospital due to loss of consciousness, attention should be paid first to the exclusion of life-threatening diseases, as the clinical manifestations may be obscure and easily missed.

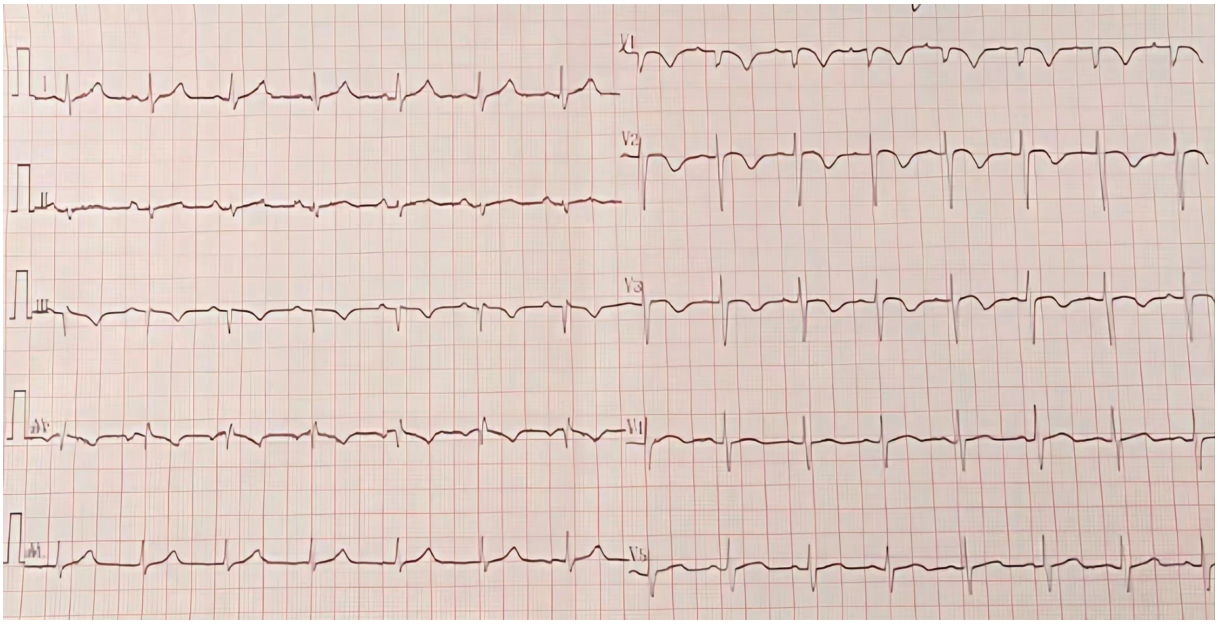

Figure 1 The patient's electrocardiogram on admission.

negative for gastrocnemius gripping pain. Other physical examinations showed no obvious abnormalities.

The complete results of laboratory examination after admission are described below. (I) The results of routine laboratory examinations were the following: blood gas analysis (without oxygen inhalation) indicated PH 7.442, CO_2 partial pressure 28.2 mmHg↓, O_2 partial pressure 95.4 mmHg, and oxygen saturation 98.1%; no abnormalities were found in routine blood or urine tests. Liver and kidney function results were the following: alanine aminotransferase, 8 U/L↓; aspartate aminotransferase, 16 U/L; α-hydroxybutyrate dehydrogenase; 205 U/L↑; albumin, 38.0 g/L↓; uric acid, 598 μmol/L↑; triglyceride, 1.84 mmol/L↑; creatinine, 75 μmol/L; estimated glomerular filtration rate (eGFR) 102 mL/min ×1.73 m^2; and blood electrolytes, normal. B-type natriuretic peptide (BNP) levels were 136 pg/mL↑. Myocardial injury marker results were as follows: troponin I (TNI), 0.053 ng/mL↑; tumor markers (–); and thyroid functions (–). (II) Results for coagulation markers were the following: fibrin degradation product, 49.3 ug/mL↑; and D-dimer, 5,366 ng/mL↑. (III) Screening results for pulmonary embolism etiology and thrombophilia were the following: lupus anticoagulant factor test, 1.33↑; β-2 determination of glycoprotein I, 34.16 RU/mL↑; autoantibody spectrum, antinuclear antibody negative; immune index: complement 3:1.540 G/L↑; no abnormality in immunoglobulin determination; protein C activity, 148.0%↑; protein S activity, negative; blood M protein, negative; antithrombin III activity measurement, (–); blood homocysteine (–); anticardiolipin antibody, 5.7 U/mL; anti-nucleosome antibody, 0.67 RU/mL; and anti-double-stranded DNA antibody, 7.3 IU/mL.

Other auxiliary examination results after admission were conducted. (I) Electrocardiogram (ECG) results were the following: sinus rhythm, typical SIQIIITIII manifestation on electrocardiogram, V1–V3 lead T wave inversion (in *Figure 1*), and no dynamic changes in the recheck. (II) Chest radiography revealed heavier bilateral lung markings and possibly a small amount of pleural effusion on the left side. (III) Echocardiogram indicated a slightly increased pulmonary artery systolic pressure (41 mmHg). The echocardiogram of this patient did not show the characteristic features of pulmonary embolism, and only mild to moderate tricuspid regurgitation was observed. (IV) Colored Doppler ultrasound of lower limb veins revealed no vein thrombosis in the left superficial femoral vein, popliteal vein, posterior tibial vein, or lower leg soleus muscle, but there was bilateral great saphenous vein inflow segment reflux (severe). (V) Computed tomography pulmonary angiogram (CTPA) revealed the following (see *Figure 2*): multiple pulmonary embolisms at the bifurcation of left and right pulmonary arteries and at each segment

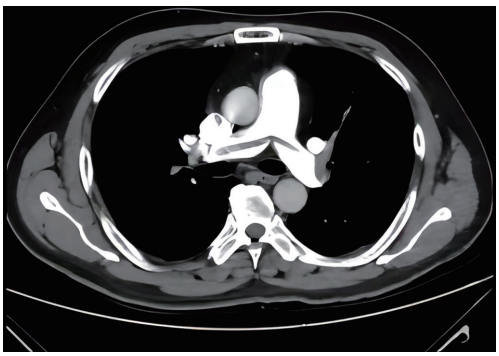

Figure 2 CTPA image of the patient. CTPA, computed tomography pulmonary angiogram. CTPA, computer tomography pulmonary angiography.

of the pulmonary artery, high density shadow at the dorsal side of the lower lobes of both lungs. Before discharge, CTPA reexamination showed that the multiple pulmonary embolism at the bifurcation of the left and right pulmonary arteries and at each segment of the pulmonary artery had basically disappeared, while a slight embolism might have remained at the residual left upper lobe and lower lingual segment of the pulmonary artery. (VI) Abdominal ultrasound showed no obvious abnormalities in liver, spleen, or kidney.

After admission, the relevant examinations were completed, the diagnosis of pulmonary embolism was confirmed. Since the patient's vital signs were stable upon admission, no recurrent syncope episodes were observed, and no significant right ventricular dysfunction was observed on echocardiography, we chose conventional anticoagulant therapy over thrombolytic therapy. Immediate anticoagulation was administered with 6,000 IU q12h (based on the patient's body weight (body weight ×100 U), this patient weighed about 60 kg) via a subcutaneous injection of enoxaparin sodium. After this, the patient did not complain of dyspnea, chest pain, or shortness of breath and did not experience a loss of consciousness. The current examination results of this patient could not confirm the diagnosis of thrombophilia, so the treatment plan after discharge was to review CTPA and other items after anticoagulation for 3–6 months to determine the discontinuation of medication and follow-up. We decided to use warfarin instead of the new oral anticoagulant for health insurance and financial reasons. Subsequently, administration for oral warfarin and heparin were overlapped for anticoagulation. The international normalized ratio (INR) was 1.22 on admission.

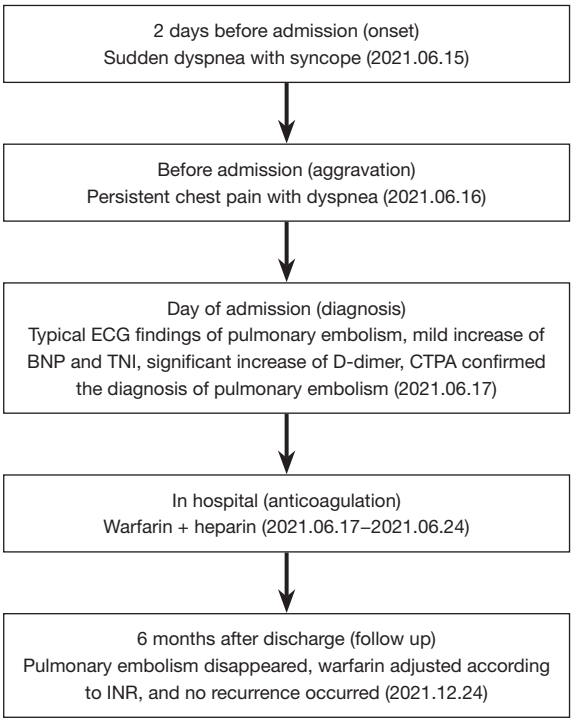

Figure 3 The progression, change, and outcome of the patient. ECG, electrocardiograph; BNP, brain natriuretic peptide; TNI, troponin I; CTPA, computer tomography pulmonary angiography; INR, international normalized ratio.

After warfarin was added, the INR was 2.38 and heparin was stopped. After discharge, the CTPA reexamination showed that the multiple pulmonary embolisms at the left and right pulmonary artery bifurcation and at each segment of the pulmonary artery had basically disappeared. Therefore, the patient was arranged to leave the hospital, and the patient was ordered to have regular outpatient follow-up, monitor the INR, and be alert to bleeding. The patient was additionally instructed to adjust the dose of warfarin as appropriate. As of this writing, this patient is still in the process of clinical follow-up (see *Figure 3* for a summary of the case).

The latest European guidelines classify syncope as reflex syncope, postural hypotension and cardiogenic syncope. Due to the clinical manifestations of the patient's characteristic dyspnea and a series of subsequent auxiliary examination results that clearly indicated embolism, the syncope was considered to be caused by acute pulmonary embolism and no further screening was conducted for other syncope causes.

All procedures performed in this study were in

accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

This patient was a middle-aged male who experienced a sudden loss of consciousness as the first symptom of pulmonary embolism, which was followed by continuous dyspnea, shortness of breath, and chest pain. After admission, ECG, echocardiography, D-dimer, blood gas analysis, BNP, TNI and other relevant examinations suggested the possibility of pulmonary thromboembolism. The patient had high blood pressure and normal oxygen saturation when he was admitted. After the diagnosis of pulmonary embolism was confirmed with CTPA, low-molecular-weight heparin overlapped with warfarin anticoagulantion therapy was administered to the patient. After anticoagulantion therapy, the patient did not complain of any dyspnea, shortness of breath, chest pain, or other discomfort, and thus warfarin was taken via oral administration, INR was monitored, and the patient was discharged according to the doctor's advice.

In this case, the patient may have been bedridden for a short time due to recent gout attacks, and venous thrombosis was formed after lower limb immobility, which eventually led to the occurrence of pulmonary embolism. At the same time, the first clinical manifestation of pulmonary embolism in this patient was the transient disorder of cerebral blood supply resulting in loss of consciousness due to the sudden restricted pulmonary gas exchange.

Therefore, this is a rare case of acute pulmonary embolism with sudden loss of consciousness as the first symptom. The possibility of pulmonary embolism should be considered in patients with syncope and dyspnea. Examination should be improved in time to assist in the diagnosis, anticoagulation and reperfusion therapy should be given in time.

The pathophysiological mechanism of pulmonary embolism causing syncope

Acute pulmonary embolism can lead to increased pulmonary circulation resistance, increased pulmonary artery pressure, and decreased pulmonary vascular bed area. When the pulmonary vascular bed area is reduced by 30% to 40%, the mean pulmonary artery pressure can reach more than 30 mmHg; when the pulmonary vascular bed area is reduced by 40% to 50%, the mean pulmonary artery pressure can reach 40 mmHg; when the pulmonary vascular bed area is reduced by 50% to 70%, persistent pulmonary hypertension can occur; when the pulmonary vascular bed area is reduced by more than 85%, sudden death can occur (2). As pulmonary vascular resistance increases, the pressure and volume of the right ventricle increase, the right ventricle dilates, and wall tension increases to maintain blood flow in the blocked pulmonary vascular bed, while systemic vascular constriction stabilizes systemic blood pressure. However, the degree of this immediate compensation is limited, and right cardiac insufficiency eventually occurs, resulting in reduced left cardiac return blood volume and thus reduced cardiac output. The cerebral cortex is unable to meet the demand for blood supply, and hypoxemia caused by the imbalance of the ventilation: blood flow ratio in the clogged capillary bed also affects the oxygen demand of the cerebral cortex. The normal functioning of the higher nervous system mainly in the cerebral cortex depends on the continuous provision of a sufficiently large blood supply and oxygen demand. As the demand for adequate blood and oxygen supply cannot be met during acute pulmonary embolism, amaurosis, fainting, and loss of consciousness may result, which explains the patient's clinical symptoms (2). However, our center has also admitted patients who were only clinically suspected of pulmonary embolism, but in whom timely CTPA examination and anticoagulation-related treatment were not implemented. The patients' ischemic and anoxic state could not be corrected accordingly for a long period of time. During hospitalization, these patients might present various nonspecific clinical manifestations, such as continuous disturbance of consciousness and even shallow coma, compared with syncope alone. These patients could have acute chest pain with corresponding ST segment elevation in the thoracic leads, which is similar to the symptoms of acute myocardial infarction or variable angina pectoris.

Differential diagnosis of syncope or loss of consciousness in clinic

The diagnosis and differential diagnosis of syncope or loss of consciousness is an area of intense research focus. In the modern era, clinicians cardiology departments are required to quickly and accurately identify the causes of a

patients' loss of consciousness in order to quickly ascertain critical factors of this syncope. The causes of syncope can be divided into several categories: (I) hypotensive syncope; (II) hypoglycemia, in which, before the onset of hypoglycemic syncope, people usually experience hunger, sweating, and prolonged disturbance of consciousness, which can be significantly improved with eating; (III) hypoxia, which is more common in syncope caused by pulmonary embolism, such as the syncope in this case; (IV) cerebrovascular diseases, including transient ischemic attack of the vertebrobasilar artery system or severe stenosis of the corresponding vessels, subclavian steal syndrome, and rare cerebrovascular diseases (e.g., moyamoya disease); (V) abnormal electrical activity in the cerebral cortex, including seizures; and (VI) hystericus insultus, which includes states of anxiety and hysterical syncope. Among these, syncope caused by hypotension is still the most common (3-5). Syncope caused by hypotension can be subdivided into 5 types: (I) poor ventricular filling, including dehydration, bleeding, severe pulmonary hypertension, pericardial tamponade, and atrial myxoma; (II) poor ventricular emptying, including aortic coarctation, hypertrophic obstructive cardiomyopathy, and severe systolic heart failure; (III) abnormal heart rhythm, including various types of tachycardia and bradycardia, vasovagal syncope (cardiac inhibitory), and carotid sinus syncope (cardiac inhibitory); (IV) reduced peripheral resistance, including vasovagal syncope (vascular inhibitory type), postural hypotension, carotid sinus allergy (vascular inhibitory type), and drug-related hypotension (nitrates, alpha-blockers, tricyclic antidepressants, etc.); and (V) syncope with other specific causes, including cough-related syncope, urination-related syncope, etc. (6). Our center has a complete examination procedure for patients with loss of consciousness whose condition is stable. In addition to routine hospital-related examinations, routine blood tests (including myocardial injury markers, BNP, coagulation analysis and D-dimer, routine biochemical indicators and blood gas analysis, and long-term dynamic ECG monitoring for 24–72 hours), echocardiography, blood pressure detection in different positions, and ultrasonography of the carotid artery, subclavicular artery, and peripheral artery should be completed. The upright tilt test, head magnetic resonance imaging (MRI) and transcranial Doppler examination, and the further monitoring of the intracranial artery via magnetic resonance (MR) angiography and electroencephalogram are evaluated according to the consultation opinions of neurology department, and then a further examination and treatment plan are made according to the patient's clinical data and the results of the corresponding examinations described above (7).

Screening of patients at risk for pulmonary embolism

The most common clinical manifestation in patients with pulmonary embolism is dyspnea, while the most common sign is increased respiratory rate (>20 beats/min). Dyspnea, syncope, cyanosis, and hypotension tend to indicate a large pulmonary embolism near the main pulmonary artery, whereas pleural pain, cough, and hemoptysis tend to indicate a small pulmonary embolism near the pleura. Therefore, routine D-dimer screening should be performed for a patient with loss of consciousness, and the possibility of pulmonary embolism should be vigilantly monitored for patients with dyspnea. For patients suspected of pulmonary embolism clinically, the following should be considered for clinical possibility assessment: (I) previous history of pulmonary embolism or lower limb venous thrombosis; (II) a heart rate increase of more than 20% compared with the normal base value; (III) history of surgery, fracture, or breaking within the last 1 month; (IV) hemoptysis; (V) tumor activity stage; (VI) age over 65 years; (VII) unilateral lower limb swelling and pain; and (VIII) gastrocnemius muscle grip pain, deep vein tenderness of lower limb, and unilateral swelling of lower limb. If 3 or more of the above 8 criteria are met, the possibility of pulmonary embolism is high. At this time, CTPA should be able to confirm or exclude the diagnosis of pulmonary embolism regardless of the results of D-dimer. If only 0–2 of the 8 criteria meet, D-dimer screening should be further improved; if D-dimer is elevated, further CTPA examination can be considered for definite diagnosis; if D-dimer is not high, pulmonary embolism can be basically excluded (7).

Formulation of the subsequent treatment plan after definite diagnosis

Risk stratification should be performed simultaneously for patients with suspected acute pulmonary embolism to guide the subsequent diagnosis and treatment measures. There are multiple different criteria and scores for the risk stratification of pulmonary embolism, and the clinical indicators observed by the corresponding criteria and scores are not the same. A comprehensive assessment of risk stratification as recommended by the earliest guidelines

for the diagnosis, treatment, and prevention of pulmonary thromboembolism was previously performed (8), with patients being classified into a high-risk group (with shock or hypotension), middle-high-risk group (stable hemodynamics but positive laboratory indicators and imaging), medium-low risk group (stable hemodynamics, with single positive laboratory indicators or imaging), and low-risk group (stable hemodynamics but no laboratory indicators or imaging abnormalities). There are also several other kinds of complicated scores for assessment of pulmonary embolism used at present (9,10). The scoring indicators are as follows: older age (over 65-year-old), male sex, tumor presence, chronic heart failure, chronic lung disease, pulse greater than 110 beats/min, systolic blood pressure less than 100 mmHg, respiratory rate greater than 30 beats/min, arterial oxygen saturation less than 90%, new characteristic ECG changes, hypothermia and changes in mental state, ultrasound changes in right ventricular shape, positive findings of deep venous thrombosis of lower extremities, and abnormal increase of cardiac TNI and BNP, etc. However, in clinical practice, no significant clinical benefit has been observed when thrombolytic therapy is actively applied to certain patients with high risk scores or grades compared with conventional anticoagulant therapy. Pulmonary embolism patients with syncope as the first symptom are not necessarily high-risk patients, nor do they necessarily need reperfusion therapy, but the presence of shock or persistent hypotension is suspected to be high-risk acute pulmonary embolism. Shock or persistent hypotension refers to systolic blood pressure <90 mmHg and/or a decrease to >40 mmHg for more than 15 minutes, except in cases of new arrhythmias, decreased blood volume, sepsis, etc. (11). These patients can be directly treated with reperfusion. In the absence of shock or persistent hypotension, conventional anticoagulant therapy can be used, and remedial reperfusion therapy can be used as appropriate in patients with elevated troponin combined with changes in right ventricular morphology and function (12,13).

Etiology screening of pulmonary embolism

In view of the etiology of pulmonary embolism in this patient, relevant risk factors of pulmonary embolism should be identified first, including major trauma, surgery, lower limb fracture, spinal cord injury, autoimmune disease, hereditary hypercoagulability, thromboembolism, inflammatory bowel disease, tumor, oral contraceptives and hormone replacement therapy, arteriovenous catheterization, stroke paralysis, chronic heart failure and respiratory failure, pregnancy, long-term bed immobilization, sedentary, aging, varicose veins, risk factors associated with atherosclerosis, history of use of chemotherapy drugs [e.g., erythropoietin (EPO) (hemopoietin) and some non-steroid anti-inflammatory drugs (NSAIDs)] (14-17). This patient had a clear history of varicose veins of the lower limbs and a family history of pulmonary embolism. One month before admission, he had taken NSAIDs orally and had relative immobilization due to gout attack and increased bed rest and thus belonged to the population prone to pulmonary embolism. In addition, the improvement of the screening related to thrombolysis after admission was demonstrated by the following results: lupus anticoagulant factor test, 1.33↑; and β2 glycoprotein I, 34.16 RU/mL↑. The possibility of antiphospholipid antibody syndrome was not excluded. The patient was asked to undergo review for the indicators related to the thrombolysis in the outpatient department of rheumatology and immunology 2 months later to confirm whether the diagnosis of antiphospholipid antibody syndrome could be established and to continue to follow up.

Conclusions

The possibility of pulmonary embolism should be considered particularly in patients with syncope complicated with dyspnea. Vital signs, heart rate, electrocardiogram, respiration, and blood oxygen saturation of patients with syncope should be determined immediately, and appropriate respiratory support and blood pressure support should be given to high-risk patients as soon as is needed. Patients with the above basic vital signs should be highly suspected of loss of consciousness caused by cardiopulmonary diseases. After finishing the clinical possibility assessment for pulmonary embolism and D-dimer screening are completed, CTPA should be performed according to the circumstances to help clarify or exclude the diagnosis of pulmonary embolism. Meanwhile, the severity of pulmonary embolism should be evaluated, and then reperfusion or anticoagulation therapy should be administered accordingly. Following this, etiological screening and medical history should be conducted to identify the risk factors related to pulmonary embolism, and corresponding treatments and disease guidance should be provided accordingly to avoid recurrence or aggravation of pulmonary embolism.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://atm.amegroups.com/article/view/10.21037/atm-23-656/rc

Peer Review File: Available at https://atm.amegroups.com/article/view/10.21037/atm-23-656/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-23-656/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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