REVIEW ARTICLE

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Pulmonary embolism masked by symptoms of mental disorders

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Abstract

Purpose: The objective of this article is to draw attention to an unusual clinical manifestation of pulmonary embolism (PE) as symptoms suggesting an underlying mental disorder. This seems all the more important since PE is one of the most common causes of potentially preventable hospital deaths and delayed diagnosis of PE in patients consulted at emergency departments has been shown to be most common in elderly patients and the ones presenting with significantly altered mental status.

Views: PE is a life-threatening condition that requires prompt diagnosis and appropriate management. There is no symptom that can be found characteristic for PE. Clinical picture of this condition is varied and may resemble many other disorders. The article emphasises that PE may also manifest with psychopathological symptoms that mimic psychiatric emergencies. Three most common groups of psychopathological symptoms that can mask a developing PE, such as panic attacks, symptoms of psychosis, and catatonia, are discussed here based on the literature review.

Conclusions: Particular care must be exercised while evaluating such a patient because initial misdiagnosis results in the patient being referred to a mental health care unit instead of a unit treating somatic disorders. There are no diagnostic or treatment facilities in large psychiatric hospitals to assist such a patient. This, in turn, leads to a significant delay in management and, consequently, to major complications or death.

Key words: psychosis, pulmonary embolism, catatonia, emergency, panic attacks.

INTRODUCTION

Pulmonary embolism (PE) is one of the components of venous thromboembolism (VTE). It is a blockage or obstruction of a pulmonary artery or sections of its branches by an embolic material, being a thrombus, originating in majority of cases from deep veins of lower limbs. Acute PE is the most common life-threatening cardiovascular disorder which is associated with mortality of 30% when managed incorrectly [1]. PE is one of the most common causes of potentially preventable hospital deaths [2].

There are no symptoms specific to PE. The clinical picture is varied and may resemble many other disorders. Patients most often complain of dyspnoea. It may be sudden and severe or mild and transient in case of central and peripheral PE, respectively. Whenever dyspnoea is exacerbated in patients with chronic lung diseases or circulatory failure, PE should be taken into account as one of the causes. Embolism must also be considered in case of chest pain, being another common symptom. It usually results from irritation of the pleura by peripheral emboli, making the chest pain pleuritic. Patients describe it as sharp, stabbing, usually located on the sides of the chest, often unilaterally. It may exacerbate during inspiration, cough, or trunk movements. The pain may radiate to the intercostal region. Less often, in case of central PE, pain may be stenocardial. It is then described as a crushing, burning, or squeezing sensation, located retrosternally. It may worsen on exertion and radiate to the neck, jaw, shoulder, elbow, or epigastrium. Other possible symptoms may include but not be limited to cough (usually dry), syncope or presyncope and/or haemoptysis. Physical examination reveals tachypnoea and tachycardia in more than half of patients. Arterial blood oxygen saturation (SaO_2) is reduced in ≥ 60% of patients. One third

of PE cases present with symptoms of deep vein thrombosis (DVT) in lower limbs, such as swelling of the lower leg, pain on pressure in the calf, increased warmth in the limb, dilatation of superficial veins which persists when the limb is elevated, and low-grade fever/fever.

Given the above description, there is no symptom that may be found characteristic for PE. The following disorders are listed most often in the context of differential diagnosis with PE: acute coronary syndrome, aortic dissection, cardiac tamponade, ventricular septal rupture, cardiogenic shock, pneumonia and pleurisy, asthma, pneumothorax, and intercostal nerve neuralgia [1].

It is pointed out that PE may manifest also as psychopathological symptoms that mimic psychiatric emergencies. This often results in misdiagnosis and patients being referred to mental health units instead of units treating somatic disorders. This, in turn, leads to a significant delay in diagnosis and treatment, and – consequently – often to the patient's death. Delayed diagnosis of PE in patients consulted at emergency departments has been shown to be most common in older patients and the ones presenting with significantly altered mental status [3].

PSYCHOPATHOLOGICAL SYMPTOMS MASKING PULMONARY EMBOLISM

Panic attacks

Repeated panic attacks are regularly observed in patients with anxiety disorder presenting with anxiety attacks; they can be reported in patients with other mental disorders or be caused by a substance (such as a drug) or a general medical condition. A panic attack is an episode of intense fear with various somatic symptoms, with the most frequent being shortness of breath, tachycardia, and chest pain. These symptoms may also suggest PE, which can coexist with panic attacks. Statistical data on the prevalence of PE in patients who reported to a doctor because of panic attacks are not available. However, PE has been shown to be less frequently suspected, both in psychiatric emergency rooms and emergency departments, when anxiety disorder seems to be the most likely alternative diagnosis [4]. Some patients report panic attacks with prevailing respiratory symptoms. The threshold of response to stimuli that can induce a panic attack, such as hyperventilation, inhalation of 35% carbon dioxide, holding one's breath, sodium lactate, and caffeine, is in these patients lower as compared to patients with panic attacks without respiratory symptoms [5, 6]. Patients with recurrent PE have an increased dead space, resulting in impaired gas exchange. Hypoxia and hypercapnia develop secondary to hyperventilation which can induce panic attacks [7]. Several case reports with PE misdiagnosed as panic attacks are referred to below.

The first case was a female patient, aged 21, hospitalised for diazepam abuse. She was healthy and had no obvious thromboembolic risk factors. She had been taking duloxetine 60 mg/day for 1.5 years because of depressive and anxiety symptoms that developed after she gave birth to her child. As far as abnormalities are concerned, physical examination upon admission revealed only tachycardia (105 bpm). The patient reported feeling intense anxiety and fatigue four times during a three-day hospitalisation. The examination showed tachycardia (up to 123 bpm) and hyperventilation (20-24 breaths per minute). SaO_2 was normal. The symptoms were interpreted as a panic attack. Diazepam 2.5 mg was successfully administered. The symptoms reappeared just before discharge and the patient collapsed. Sudden cardiac arrest (SCA) was diagnosed. She died despite resuscitation. Autopsy showed pulmonary embolism as the main cause of death. According to her family, the patient had never experienced hyperventilation and fatigue during panic attacks before [8].

The second case was a female patient, aged 48, who was referred to a psychiatric emergency room, complaining of repeated anxiety attacks for 5 weeks. The patient had been treated for postpartum depression 12 years before, had never been treated for an anxiety disorder, and had no panic attacks. Her symptoms developed after a surgery she had for a fracture of a right ankle joint, which was immobilised in a cast. She received enoxaparin 40 mg subcutaneously as a thromboprophylaxis. Anxiety attacks recurred on average twice a week, at night. The patient woke up feeling intense restlessness, anxiety, a sense of danger, dizziness, and increased sweating. She presented with these symptoms to her family doctor, who advised her to take opipramol. A week before reporting to the emergency room, the patient also consulted a psychiatrist, who confirmed panic attacks and recommended increasing the opipramol dose. She denied having any physical symptoms at the emergency room. She reported that her last panic attack had occurred 2 hours before and had been preceded by additional shortness of breath and skin pallor. The examining psychiatrist suspected PE and referred the patient to the accident and emergency department. She denied having dyspnoea and chest pain upon admission. Blood pressure was normal. ECG showed tachycardia (105 bpm) and prolonged QT interval, while laboratory tests revealed elevated D-dimer and troponin I. Contrast-enhanced CT scan was performed, showing extensive bilateral pulmonary congestion. She survived due to an intravenous anticoagulant treatment. The patient was examined 6 months later and reported that the anxiety attacks had not recurred since then [9].

The third case is a female patient, aged 15, diagnosed with stage 4 Hodgkin lymphoma. She had been taking hormonal contraception for the past six months due to heavy menstrual bleeding. She had a peripherally inserted central

catheter (PICC) for an extended period to maintain vascular access. After the PICC line insertion, the patient reported several brief, self-limiting episodes of breathing difficulties that resolved without intervention. They were believed to be associated with stress-induced panic attacks. Shortness of breath lasted for over 1 hour in one of these episodes, in addition to being accompanied by chest tightness and abdominal pain. There were no symptoms suggestive of DVT. The patient was referred to the hospital where laboratory tests revealed elevated D-dimers. A CT pulmonary angiogram was performed and revealed large-volume pulmonary emboli in the distal right pulmonary artery and extensive thromboembolism of the lower lobe of the left lung. After she had been diagnosed, anticoagulation treatment was initiated, a PICC line was removed, and hormonal contraception was discontinued. The patient survived. Her condition was stable at a follow-up 6 months later and cancer was in remission [10].

Symptoms of psychosis

Delusions and hallucinations may be caused by a primary mental disorder, systemic disorder, or a particular substance.

Contact with the patient during a psychotic episode is usually difficult. The patient may, for example, be agitated, showing disorganised behaviour, or may be distrustful for delusional reasons, which significantly impedes the history-taking. Two case reports with PE misdiagnosed as psychosis are referred to below.

The first case is a male patient, aged 68, with Parkinson's disease, who was admitted to a mental care unit due to aggressive behaviour and visual and auditory hallucinations. Similar symptoms had been reported in the patient 3 years before, when doses of his medications for Parkinson's disease had been increased. Since then, the symptoms did not recur, and medication doses were kept the same. Physical examination upon admission did not raise any concerns. Three days later, sudden dyspnoea and cardiac arrest were noted. Computed tomography (CT) of the chest was performed after successful resuscitation, revealing pulmonary embolism. The patient's condition became stable after a successful thrombolytic therapy and symptoms of psychosis completely resolved [11].

The second case is a female patient, aged 64, who had not been previously treated for any somatic and mental disorders. She was brought for a consultation due to behavioural changes and persecutory delusions that had lasted for a week. The patient feared that other people wanted to harm her, accused neighbours of stealing food from her home, and suspected that her husband was feeding her piranhas. Physical examination and laboratory tests performed at the emergency department did not show any significant abnormalities. The patient was considered to have a mental disorder and was transferred to the mental care unit, at which she showed periodical psychomotor agitation and presented with attention disorders. She scored 22/30 in the mini-mental state examination (MMSE). Significant deficits in orientation, recall, and repetition functions were observed. Delirium was suspected and the patient was re-transferred to the accident and emergency department. She fainted shortly after. SCA with pulseless electrical activity was found. An echocardiogram was performed during resuscitation and revealed PE as a probable cause of SCA based on right ventricular dilatation. Tissue plasminogen activator was administered to the patient, resulting in haemodynamic stabilisation. This was followed by ultrasound of lower extremities, which showed thrombosis of the left posterior tibial vein, and CT, which revealed bilateral PE. The patient's condition significantly improved after a week, with only mild cognitive impairment reported. Three months later she scored 30/30 in the MMSE [12].

Catatonia

Psychopathological symptoms in patients with a history of mental disorder is most often interpreted as a relapse or exacerbation of mental disorder, resulting in further diagnosis being ceased.

A case report of a male patient, aged 75, diagnosed with schizophrenia and catatonia is an example of such management. Auditory hallucinations, stupor, mutism, and refusal to eat were interpreted as a recurrence of his disorder. Physical examination revealed dyspnoea, mild tachycardia (102 bpm), and low-grade fever. As the patient also suffered from chronic obstructive pulmonary disease (COPD), these abnormalities were thought to be associated with exacerbated COPD. The man had no typical thromboembolic risk factors.

Additional tests were performed due to deteriorating general condition, such as D-dimer levels, which were significantly elevated. Chest CT confirmed bilateral PE. Having retrospectively analysed the patient's medical history, it was concluded that his symptoms resulted from delirium caused by pulmonary embolism developing at the time. Despite delayed diagnosis, the patient was saved [13].

CONCLUSIONS

Psychopathological symptoms may prevail in the clinical picture of developing PE. Such an atypical manifestation of PE may occur both in patients who have been treated for mental disorders and those without such a history.

Panic attacks are one of the most common psychopathological symptoms that can mimic PE. Patients who experienced panic attacks in the past are usually able to describe an attack typical to themselves. As shown in the first case, it is always necessary to specify whether

current symptoms were present in the past or whether they are new. Panic attacks presenting with symptoms that are not specific to a given patient always require careful observation and diagnosis must be extended. It seems that particular care must be exercised in case of patients who have panic attacks with respiratory symptoms [7].

Taking into consideration the second patient, it is worth emphasising the fundamental importance of collecting a detailed history (also the occurrence of similar psychopathological symptoms, e.g., panic attacks in family members) and physical examination of the patient. These are the most important tools at psychiatric emergency rooms, at which diagnostic capabilities are usually limited. Panic attacks developing for the first time in life, following an event that is a thromboembolic risk factor (in this case it was an ankle fracture treated with surgery and immobilisation) should always raise suspicion of PE. If multiple thromboembolic risk factors are present as in the third case reported, differential diagnosis of a panic attack should always include PE.

Symptoms of psychosis may occur during somatoform delirium, the cause of which may include PE. Hypoxia is a probable factor causing delirium in patients with PE. The patient's condition usually suddenly deteriorates; however, symptoms are variable and include disorientation, attention disorders, generalised cognitive disorders, or impaired consciousness and contact with others [14, 15]. Due to concomitant hallucinations or delusions, patients are most often diagnosed with psychosis, in particular by physicians who are inexperienced in assessing mental status. It additionally seems that the reason for such errors is a belief that only staff at mental care units are capable of examining the mental status. One of the reasons may also be the staff's fear of coming in contact with patients showing symptoms of mental disorders [16], in particular psychosis. All these factors may result in potentially life-threatening somatic conditions being overlooked. Given the case reports referred to above, it should be emphasised that new behavioural changes in older patients always require care and monitoring of somatic status. PE should be taken into consideration in older patients with psychosis with sudden onset. Effective treatment of PE leads to complete resolution of psychosis. It is worth emphasising that in the case of older patients with symptoms of delirium, it is always necessary to exclude other somatic causes (e.g., hypoglycaemia, inflammation, electrolyte disturbances, headache, trauma, hormonal disorders, liver failure, and kidney failure).

Catatonia is the third condition discussed that may mask PE. In the past, catatonia was mainly associated with schizophrenia, but it is now known that it may occur in the course of other mental disorders, neurological conditions, or somatic disorders. In the above case report, recurring catatonia was interpreted as exacerbated schizophrenia. In daily clinical practice, it is always important to consider whether a somatic condition exacerbates or triggers psychopathological symptoms in patients with a diagnosed mental disorder.

Conflict of interest

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