'Organic Anxiety' in a Middle-aged Man Presenting with Dyspnoea: a Case Report

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Abstract

We report a case of pulmonary embolism in a patient who presented with repeated anxiety attacks and psychotic symptoms and was misdiagnosed as having withdrawal seizure or anxiety disorder not otherwise specified. This case highlighted the nonspecific clinical features of pulmonary embolism and the principles in making psychiatric diagnosis. Careful history taking, thorough physical examination, appropriate investigation, and a high index of suspicion led to the correct diagnosis. The principle of hierarchy of psychiatric diagnosis (ie, organic over non-organic) and the possibility of comorbidities should always apply.

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Introduction

Arrhythmia and asthma can be associated with both physical and psychic symptoms such as feelings of panic. Anxiety disorder is characterized by both apprehensive feelings and somatic symptoms, which may overlap with those of medical conditions. Autonomic arousal can be a common denominator for both medical and physical conditions. Its diagnosis can only be made by physical pathology, in addition to the classic symptomatology. In general, the diagnosis is easily missed in less common conditions such as multiple sclerosis or in those with episodic spells such as epilepsy. In fact, misdiagnosis in psychiatric patients is not uncommon. A hospital-based study reported that about 60% of patients with delirium were initially misclassified and referred for psychiatric evaluation for other reasons (eg, psychosis or anxiety), and about 70% of them had a past psychiatric diagnosis.[1] Often, physical and mental state examinations have not been performed completely in psychiatric patients presenting to emergency departments.[2] Thus, 'diagnostic overshadowing', a form of disparity in diagnosis, is a major cause of misdiagnosis in which physical symptoms are misattributed to psychiatric causes.[3] We report a case of pulmonary embolism in a patient who presented with repeated anxiety attacks and psychotic symptoms and was misdiagnosed as having withdrawal seizure or anxiety disorder not otherwise specified.

Case presentation

In April 2016, a 53-year-old married unemployed Chinese man was admitted to the psychiatric ward from the emergency department with a 1-week history of worsening anxiety and psychotic symptoms. He lived with his wife and was described as a non-anxious person. No family history of mental illness was reported. He was a non-smoker and non-drinker. He had diabetes, hypertension, hyperlipidaemia, and obesity and had undergone bariatric surgery at age 42 years. He had had chronic insomnia since his 40s and had been using over-the-counter sleeping pills.

In May 2015, he had been admitted to a general medical ward for loss of consciousness and twitching of limbs after running out of sleeping pills. He was diagnosed as having hypnotics dependence syndrome with withdrawal seizure. During his inpatient stay, he was noted to have shortness of breath, tremor, and palpitation as well as paranoid and referential ideas and auditory hallucinations. He was transferred to the psychiatric unit and was diagnosed as having acute and transient psychotic disorder. His symptoms resolved soon after taking risperidone and citalopram. In view of his stable condition, he was discharged and followed up regularly, with medication discontinued.

In September 2015, he had been admitted with similar anxiety and psychotic symptoms and given citalopram and risperidone for the same diagnosis. Again, the symptoms quickly resolved and he was discharged after 2 weeks of hospitalisation and remained stable with regular medications.

For the index admission in April 2016, he presented with decreased exercise tolerance, tremor, sweating, palpitation, and shortness of breath. He heard voices and

feared being monitored. The diagnosis was revised to anxiety disorder not otherwise specified. Benzodiazepine was added to citalopram and risperidone with little benefit. On day 3, he fainted in the toilet but regained consciousness promptly with no convulsions. On examination, he was not anxious but was breathless and sweating. His blood pressure was low (90/70 mm Hg), with tachycardia (130 bpm) and desaturation (SpO_2 88%) in room air. Air entry to both lungs was good. Heart sound was dual without murmurs. There was no lower limb oedema, and both calves were soft. Electrocardiogram showed sinus tachycardia and right bundle branch block, deep S wave at axis I, prominent Q wave at axis III, and T wave inversion over axis III via right lateral leads. He was transferred to the medical ward, and blood tests showed elevated troponin T (179 ng/ml), lactate dehydrogenase (377 U/L), and white blood cell count (12.2 $\times 10^9$/L). Venous blood gas data showed metabolic acidosis (pH 7.34 and base excess -8). Liver function tests and electrolyte levels were normal. Computed tomography of the brain and chest radiography were unremarkable. However, contrast computed tomography of the thorax showed massive bilateral pulmonary embolism with dilated right atrium and right ventricle. A thrombolytic agent was given, followed by heparin infusion. Citalopram and risperidone were withheld. On day 3 after transfer, he complained of left leg oedema with tenderness and increased warmth. Doppler ultrasonography confirmed a deep vein thrombosis in the common femoral vein of the left leg. Anti-coagulant (rivaroxaban) was initiated. However, his condition was complicated by ventricular standstill and necessitated implantation of a pacemaker. Low dose pregabalin was started upon inpatient psychiatric review to promote sleep and relieve anxiety. After 2 weeks of hospitalisation, his physical symptoms gradually subsided and the haemodynamics stabilised. He was discharged home and continued to have regular psychiatric follow-ups. There was no recurrence of anxiety or psychotic symptoms as at late 2017.

Discussion

This case illustrates the rare presentation of pulmonary embolism as symptoms mimicking an acute anxiety attack. In retrospect, the last episode was distinct from previous episodes of 'anxiety attacks'. There were a recent history of sweating and decreased exercise tolerance, absence of symptomatic improvement despite the use of previously effective psychotropic medications, and an episode of syncope without clear evidence of benzodiazepine withdrawal. 'Diagnostic overshadowing' might have biased the attending physician and psychiatrist to attribute physical symptoms to psychiatric causes. Although pulmonary embolism is difficult to diagnose given its nonspecific signs and symptoms, the subtle but salient difference in clinical presentation, unstable haemodynamics, and complicated medical background (diabetes, hypertension, hyperlipidaemia, and bariatric surgery for obesity) should have alerted the attending doctor of an organic cause.

The differential diagnoses of anxiety disorder not otherwise specified include recurrent acute and transient psychotic disorder and recurrent pulmonary embolism. Acute and transient psychotic disorder is marked by its polymorphic presentation and is likely a composite category, as it has a high recurrence rate (45.2%).[4] Recurrent pulmonary embolism (rather than a withdrawal seizure) may explain the previous episodes of seizure and syncope, although this would be an atypical presentation. The recurrent pulmonary embolism could be caused by unremitting sources of emboli from the legs. In fact, the risk of recurrent venous thromboembolism has been reported to be 5% to 7%.[5] However, it may be argued that the patient had a long history of hypnotic dependence, and that his symptoms had readily remitted in the first two admissions with psychotropic medications without the need for other medical interventions.

This case highlights the nonspecific clinical features of pulmonary embolism and the principles of making psychiatric diagnosis. Careful history taking, thorough physical examination, appropriate investigation, and a high index of suspicion led to the correct diagnosis. The principle of hierarchy of psychiatric diagnosis (ie, organic over non-organic) and the possibility of comorbidities should always apply.

Declaration

The authors have no conflicts of interest to disclose.

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