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CASE REPORT

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Pulmonary thromboembolism associated with quetiapine: a case report

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ABSTRACT

Venous Thromboembolism (VTE), which includes pulmonary embolism and deep-vein thrombosis is also a potentially fatal adverse drug reaction and little attention has been focused on this topic. Atypical antipsychotics are associated with an increased risk of pulmonary embolism. In this case we want to show pulmonary thromboembolism associated with quetiapine. A 36-year-old man with bipolar disorder, presented to the Emergency Department complaining of epileptic seizure, general weakness, mild fever, and dizziness. Pulmonary thromboembolism was considered as the result of clinical evaluation. There were no risk factors such as age, smoking, trauma, immobilization, surgery, heart disease, and genetic risk factors to explain pulmonary embolism. In this case we see that the pulmonary embolism was associated with quetiapine. We should be more careful about pulmonary thromboembolism. Physicians and individuals must be aware of this potentially fatal, though treatable, adverse drug reaction when starting treatment, especially in patients who have other risk factors for VTE.

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Pulmonary thromboembolism; quetiapine; adverse effect; mortality; antipsychotics; bipolar disorder

Introduction

Venous Thromboembolism (VTE), which includes pulmonary embolism and deep-vein thrombosis is also a potentially fatal adverse drug reaction and little attention has been focused on this topic. Several studies have identified age, immobilization, obesity, smoking status, allergy, autoimmune disease, heart failure, lower leg fracture, surgery, diabetes, pregnancy, antipsychotics, physical restraint, and cancer as acquired risk factors for VTE [1].

It has been reported that the risk of VTE is increased in people using antipsychotic in epidemiological cohort and case control studies [2–4]. Specially it is known that low-potency antipsychotic drugs were more associated with VTE develops than high potency antipsychotics [5]. Clozapine which is an atypical antipsychotic is associated with an increased risk of pulmonary embolism [6–10]. Also the association between other seconder generation antipsychotics especially with olanzapine and risperidone was reported by case reports and limited studies. Pulmonary thromboembolism is often misdiagnosed as sudden cardiac death. In this case we want to show pulmonary thromboembolism associated with quetiapine.

Case

A 36-year-old man presented to the Emergency Department (ED) complaining of epileptic seizure, general weakness, mild fever, and dizziness. There was no chest pain, leg oedema or swelling, orthopnea, paroxysmal nocturnal dyspnea, or sweats. That was his first epileptic seizure. Results of cerebrospinal fluid study, brain computed tomography (CT) scan, and chest X-ray study were unremarkable. Also EEG and brain MR was normal. The patient has a history of bipolar affective disorder for six years and taking 1200 mg/day lithium. Cause of his depressive episode the psychiatrist added quetiapine 200 mg/day a week before the recourse to ED. The physical examination revealed a lethargic man with apathetic but oriented appearance. The vital signs recorded were blood pressure 120/70 mmHg, heart rate 132 beats/min, respiratory rate 21 breaths/min, and body temperature 37.9°C Oxygen saturation in room air was 70%. The lungs were clear bilaterally. The haemogram and blood chemistry values were normal, only D-dimer was 1773 µg/L. The helical chest CT scan revealed; bilateral superior and inferior lober branches trunk thrombosis. There were no risk factors such as age, smoking, trauma, immobilization, surgery, heart disease, and genetic risk factors such as protein C, S to explain pulmonary embolism. Antitrombin III deficiency, disfibrinogenemy, Factor V Leiden thrombophilia, PT20210 and MTHFR gen mutation, lupus anticoagulant, and Homocysteine level was normal and there was no family history of hypercoagulable state. In this case we see that the pulmonary embolism was associated with quetiapine. The quetiapine treatment of the patient, whose emergency unit was followed clinically,

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was terminated. On the third day of admission, his complaints disappeared and his vital findings were stable. Patient was discharged by recommending the chest diseases and psychiatric outpatient clinic control.

Informed consent of the patient was obtained.

Discussion

VTE has been associated with risk factors such as smoking, trauma, immobilization, surgery, pregnancy, use of combined oral contraceptives, malignant disorders, and certain cardiac and haemostatic disorders, including factor V Leiden mutation. In our case there was no family history of hypercoagulable state, nor any past surgical or chronic systemic medical history. He did not have any risk factors for pulmonary embolism. We strongly suspected that quetiapine might have contributed to his pulmonary thromboembolism on the basis of published reports [3–7]. The biological mechanism explaining the relation between antipsychotic drugs and VTE is unknown. Many biological mechanisms have been proposed to explain this relationship until this time. Previous studies in the literature have shown that antipsychotics increase platelet aggregation, especially due to the effects on 5-hydroxy tryptamine [11]. A second possible explanation is about anticardiolipin antibodies. Anticardiolipin antibodies are associated with increased risk of venous or arterial thrombosis and it has been observed that anticardiolipin antibodies are increased in patients using chlorpromazine. At the same time, no relationship has been found between VTE and antipsychotic drug use in those in whom anticardiolipin antibodies were detected [12]. Patients treated with low-potency antipsychotic drugs have the side effect sedation much more often. A third hypothesis is that venous stasis can be aggravated by sedation and this can increase the risk of thrombosis. Also it is thought that putting on weight, high body mass index (BMI), sedative life style developing with the use of these drugs could be the risk factors. Pulmonary thromboembolism is often misdiagnosed as sudden cardiac death. Only in necropsy in psychiatric patients 10 of 27 cases of idiopathic, fatal pulmonary embolism were diagnosed [13]. There is an association between sudden cardiac death and antipsychotic drug use which has been described by the spontaneous reports [14,15]. However, evidence to explain the causal relationship between antipsychotic drugs and VTE is still insufficient.

Conclusion

In recent years atypical antipsychotics are widely used in many different psychiatric disorders. In many cases, the thrombosis process begins shortly after antipsychotic therapy begins. Most of the patients continue to use these drugs after the past of their first VTE, because physicians probably do not associate VTE with antipsychotic drug use.

With this case report, it may be emphasized that clinicians should keep in mind the risk of pulmonary thromboembolism in patients who are receiving antipsychotic treatment and especially those who have other risk factors for VTE.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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