

study in which indicators of insulin sensitivity were associated with suicide risk.¹ The explanation for the reverse J shaped association seen when diabetes was included as a fifth category alongside insulin resistance may be due to patients with a clinical diagnosis of diabetes developing depression as a result of this diagnosis.

We based our assessment of depression on current use of medication and self reports of past diagnoses and current mood rather than clinical assessment with international diagnostic criteria. However, the consistency of our findings across the three different assessments supports a causal association, and any measurement error in our assessment of depression would tend to dilute the results. Insulin resistance is positively associated with diabetes and cardiovascular disease, and we do not believe that our results should be used to discourage appropriate interventions to prevent and treat insulin resistance. Further, these are novel findings and need to be replicated in other studies. However, if our findings are confirmed there may be an indication for assessing depressive symptoms among individuals receiving treatments that affect insulin resistance, since depressive symptoms are often disabling and could affect compliance with treatment and quality of life.

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Drug points

Pulmonary embolism possibly associated with olanzapine treatment

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Antipsychotic drugs have been associated with an increased risk of venous thromboembolism.¹ We report for the first time the case of a patient who developed a pulmonary embolism after starting treatment with olanzapine.

A 28 year old man was admitted to hospital due to a psychotic disorder. Treatment with olanzapine (10 mg/day) was started, and the dose was gradually increased to 30 mg/day. He also received levomepromazine (50 mg/day), oxazepam (10 mg/day), and flunitrazepam (1 mg/day). After 10 weeks, the patient complained of respiratory pain and he had two episodes of haemoptysis. Clinical examination showed no auscultatory findings, no dyspnoea, no tachypnoea, no fever, and normal blood pressure and heart rate. Blood analysis showed raised concentrations of C reactive protein (113 mg/l (normal range <10 mg/l)), fibrinogen (6 g/l (2-4 g/l)), and D-dimer (0.89 mg/l (<0.50 mg/l)). Spiral computed tomography showed a pulmonary embolism in the left lower lobe. Standard anticoagulant treatment was started, and the patient recovered. Olanzapine was discontinued, and his medication changed to quetiapine.

Recent reports suggest an association between clozapine and venous thromboembolic events.²⁻⁵ However, thromboembolic complications have not previously been described in patients taking olanzapine. The sedating effects as well as the weight gain associated with this antipsychotic treatment can lead to a more sedentary lifestyle,

thus creating predisposing conditions for venous thrombosis. In this case, the patient was overweight (body mass index 28.5), but his weight had not substantially changed since starting to take olanzapine. He was otherwise healthy, and his level of physical activity was normal. Tests for possible coagulation disorders—including tests for antiphospholipid antibodies (immunoglobulin lupus anticoagulants and anticardiolipin antibodies), mutation of the methylenetetrahydrofolate reductase C677T thermolabile variant, prothrombin G20210A mutation, activated protein C resistance, protein C, protein S, antithrombin III, and homocysteine—did not show any underlying risk factors. This leaves the question of the medication's possible direct causal effect.

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