

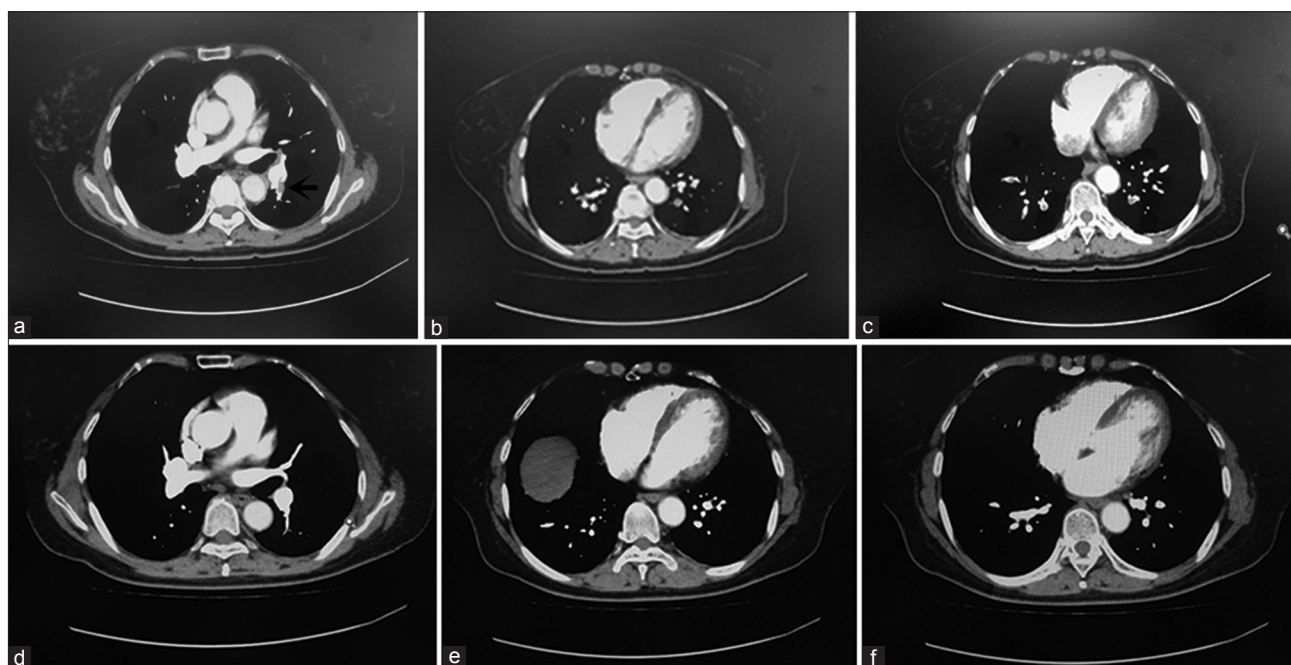
## Pulmonary Thromboembolism Associated with Olanzapine Treatment

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To the Editor: A 55-year-old female was admitted to the emergency department of our hospital complaining of dyspnea after catching a bus for about 5 m during last day. She had a past history of suffering from depression mood disorder recently and could not sleep well at night for which she was started on olanzapine tablets 10 mg/d, sertraline hydrochloride tablets 50 mg/d and oxazepam tablets 15 mg/d for 28 days by a physician of psychiatric hospital. She was otherwise without receiving any other medicine, including oral contraceptive, illicit drugs or alcohol and had no history of smoking, recent surgery, trauma, peripheral vascular disease, cancer or cardiovascular disease. What's more, she did not have a demonstrable lowered activity level or weight change recently. On admission, the vital signs recorded were: blood pressure 100/70 mmHg, heart rate 100 beats/min, respiratory rate 18 breaths/min, T 36.5°C, body mass index was 23 kg/m<sup>2</sup>.

Cardiovascular, respiratory, abdominal and neurological examinations were unremarkable. Arterial blood gas analysis showed hypoxemia and hypocapnia (PO<sub>2</sub> 49 mmHg, PCO<sub>2</sub> 27.5 mmHg, SO<sub>2</sub> 85.7%), plasma D-dimer was 11.79 mg/L. Blood routine revealed white blood cell count  $11.49 \times 10^9/L$  with 74.8% neutrophils and platelet count  $142 \times 10^9/L$ , hemoglobin 135 g/L. Cardiac enzyme showed cTnI was 0.066 ng/ml, a little higher than the normal range, but CK-MB as well as liver and renal function test were normal. Electrocardiogram showed T-wave inversion on III, V1-V3 leads. And computed tomographic pulmonary angiography (CTPA) demonstrated multiple pulmonary emboli on both lobes accompany with a relatively small amount of fluid on the right interlobar fissure [Figure 1a-1c]. Venous thrombosis between right calf muscle was discovered on bilateral Doppler ultrasound of the lower limbs. The ultrasonic cardiogram did not show



**Figure 1:** The selected computed tomographic pulmonary angiography images at presentation: (a) (2014/2/21): The image shows perfusion defect in the branch vessel of lower left pulmonary artery (arrow). (b) and (c) (2014/2/21): There are multiple perfusion defects in peripheral pulmonary arterioles on both lobes. (d-f) (2014/6/10): Noting abnormal detected.

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obvious abnormal. Based on the history and the above results, the diagnosis of pulmonary embolism (PE) was established. The patient was then admitted in the Respiratory Department and full work-up for coagulopathy were made, but did not reveal any abnormality. And at the same time the patient was asked stopping antipsychotic drugs immediately and received low molecular weight heparin ih at the dose of 12,000 IU daily, which was switched to warfarin sodium tablets after 3 days. Six days later, as the patient's condition improved, the blood analysis were rechecked which showed decreased concentrations of D-Dimer 2.56 mg/L and cTnI 0.035 ng/ml. Arterial blood gases were within normal limits (pH 7.44, PO<sub>2</sub> 86 mmHg, PCO<sub>2</sub> 40 mmHg). When her conditions recovered completely, she was discharged from our hospital after 12 days and then, she was asked to continue taking warfarin sodium tablets for at least 6 months with international ratio 2.0–3.0. As follow-up examination, repeated CTPA was made 4 months later and nothing abnormal was detected [Figure 1d-1f].

Several clinical and environmental factors have been associated with the development of venous thromboembolism (VTE). While, the absence of major congenital or acquired risk factors for PE in this case points to drug therapy on the most probable factor underlying the thromboembolic event in our patient.

In this case, the fact that the patient had also taken sertraline hydrochloride and oxazepam at that time that should also be

considered. While, unlike olanzapine, sertraline hydrochloride is a kind of selective serotonin reuptake inhibitors which are much more often implicated in bleeding rather than thrombotic events.<sup>[1]</sup> And as the treatment with oxazepam that belongs to the benzodiazepine, has not been reported to linked to VTE.<sup>[2]</sup> This evidence is not in support of a major contribution of other two kinds of drugs than olanzapine to PE episode in our case.

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