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Successful treatment of a massive pulmonary embolism using alteplase in a patient taking antipsychotic drugs

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An increased risk of pulmonary embolism (PE) has been reported in female patients taking antipsychotic drugs. Furthermore, patients who take antipsychotic drugs are likely to present with malaise; therefore, the onset of PE is easily overlooked and often discovered by necropsy following PE-related sudden death. (1)

A 73-year-old woman was admitted to the emergency room because of severe shortness of breath and a rapid heartbeat, which had started 2 h earlier. Her medical history included severe depression that had left her bedridden for roughly 3 years. During that period, she frequently tired easily; however, her neighbors believed this to be a result of her depression and, therefore, were not concerned. On her arrival to the emergency room, the clinical examination confirmed hypotension (91/40 mmHg), tachycardia (109 bpm), and a peripheral oxygen saturation in room air of 80%. Examination of the cervix and chest revealed jugular venous distension. She had bilateral pitting edema of the lower extremities. On auscultation of the lungs, bilateral coarse crackles were audible.

Key words: Female, antipsychotic drugs, massive pulmonary embolism, depression, alteplase.

An arterial blood gas test performed with the patient breathing room air showed evidence of type 1 respiratory failure (pH 7.49, pO2 52.7 mmHg, pCO2 21.0 mmHg). A scan obtained using portable, simple thoracic radiography revealed patchy shadowing (**Figure 1**). At this point, we suspected a massive pulmonary embolism, and a computed tomography (CT) pulmonary angiogram revealed extensive bilateral pulmonary embolisms (**Figure 2**). The patient was immediately thrombolysed with alteplase, which commenced 3 h after symptom onset. The dose was calculated for her body weight of 60 kg, resulting in 4 mg given as an intravenous bolus, followed by an intravenous infusion of 36 mg administered over a 1-h period. Eight hours later, following the thrombolytic treatment, the patient improved and was hemodynamically stable (blood pressure 130/68 mmHg, heart rate 90 bpm) with a peripheral oxygen saturation in room air of 93%. Therefore, the patient was successfully thrombolysed without any complications (**Figures 3a** and **3b**).

Several mechanisms have been proposed for the thrombotic complications associated with the platelet aggregation induced by antipsychotic drugs including the presence of anticardiolipin antibodies and type 2A serotonin receptors. (2,3) However, anticardiolipin antibodies were not detected in this patient, and she had not taken medication related to type 2A serotonin receptors. We were able to diagnose the pulmonary infarction by recognizing the presence of abnormal vital signs that we could not explain. Therefore, the proper evaluation of vital signs is important even in patients with considerable malaise.

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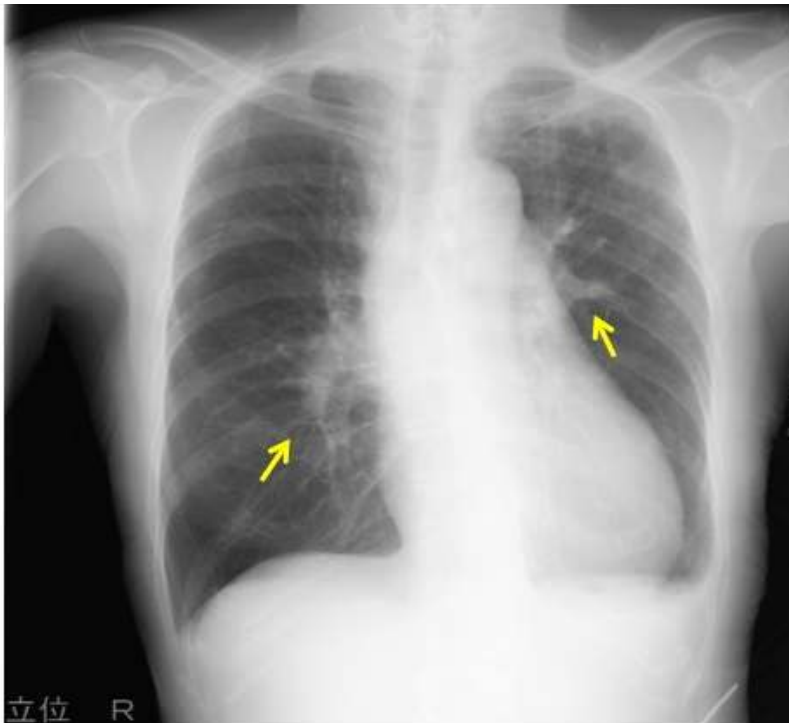
Figure 1. Simple thoracic radiography revealed patchy shadowing

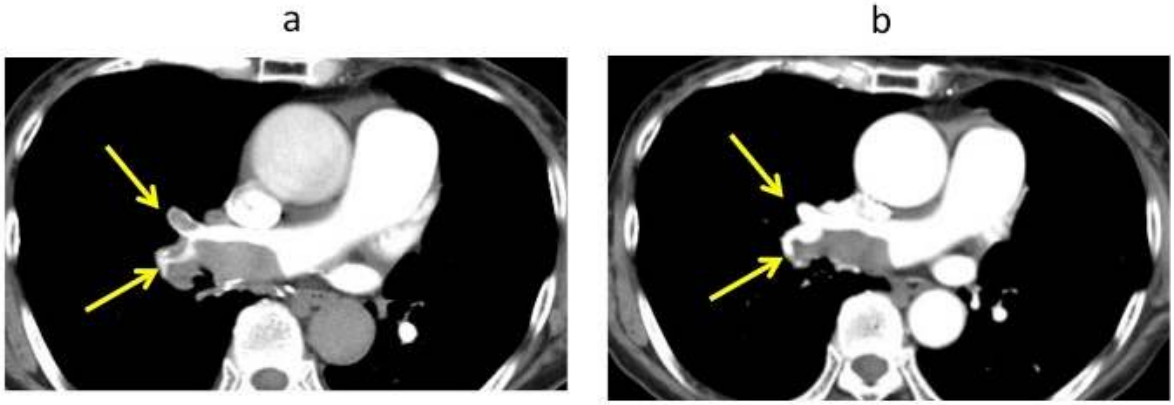


Legend: Frontal chest radiography revealed a dilated pulmonary artery with abrupt tapering of the occluded vessels distally or a “knuckle sign” (arrows).

Figure 2. A computed tomography pulmonary angiogram revealed extensive bilateral pulmonary embolisms

Legend: Contrast-enhanced computed tomography (CT) scan of a patient with a massive pulmonary embolism. As an incidental finding, the examination revealed multiple emboli in the right pulmonary artery (A4, 5, 6) and left pulmonary artery (A1+2, 4,6) (arrows).

Figure 3. A computed tomography pulmonary angiogram before and after treatment with alteplase



Legend: Contrast-enhanced computed tomography (CT) scan of a patient with a massive pulmonary embolism before (a) and after (b) 8 h of thrombolytic treatment with alteplase. Recanalization of the right pulmonary arteries following the intravenous infusion of alteplase was observed (arrows).

References

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