

# Pulmonary Cavitation and Hydropneumothorax Associated With Pulmonary Embolism

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## CLINICAL SUMMARY

A 70-year-old woman, with no addictions presented with sudden onset of breathlessness and dry cough to a local hospital. A chest radiograph (postero-anterior view; Figure 1) showed a right-sided hydropneumothorax for which an intercostal chest drain (ICD) was inserted and the patient was referred to us for further assessment. She had a history of right-sided femur neck fracture following a trivial trauma about a month ago and was operated 15 days ago with a right bipolar hemiarthroplasty. There was no complaint of chest pain or fever and there was no remarkable past history. On general examination, the patient was afebrile with a pulse of 117 per minute, blood pressure of 100/70 mmHg, a respiratory rate of 34 per minute with usage of accessory muscles of respiration and a pulse oximetry saturation of 90 percent. Jugular venous pulse was not raised. The right lower limb revealed swelling. On systemic examination with the ICD *in situ*, breath sounds were decreased in intensity over the right hemithorax and

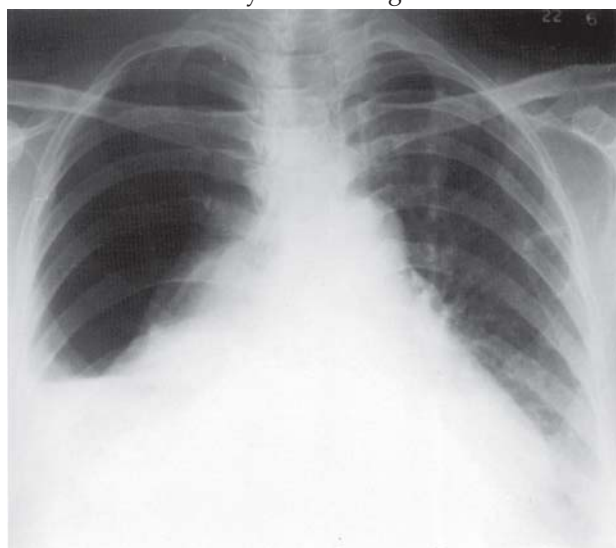


Figure 1. Chest radiograph (postero-anterior view) showing a right-sided hydropneumothorax.

sucussion splash was heard. The pulmonary component of the second heart loud sound was loud and a left parasternal heave was palpated.

## INVESTIGATIONS

Haemogram showed a hemoglobin of 7.2 g/dL with a white blood cell (WBC) count 12600/mL. Serum biochemistry was in normal range. A repeat chest radiograph and computed tomography (CT) of the thorax showed a right-sided lower lobe cavity with a hydropneumothorax and the ICD *in situ* (Figure 2). Her Wells diagnostic score was 9 and the revised Geneva score was 12, suggestive of high clinical probability of pulmonary embolism. The electrocardiogram (ECG) showed a QR pattern in  $V_1$ , T wave inversion in lead  $V_1$ - $V_4$  and an incomplete right bundle branch block (RBBB). Computed tomography pulmonary angiography (CTPA) (Figure 3) showed a

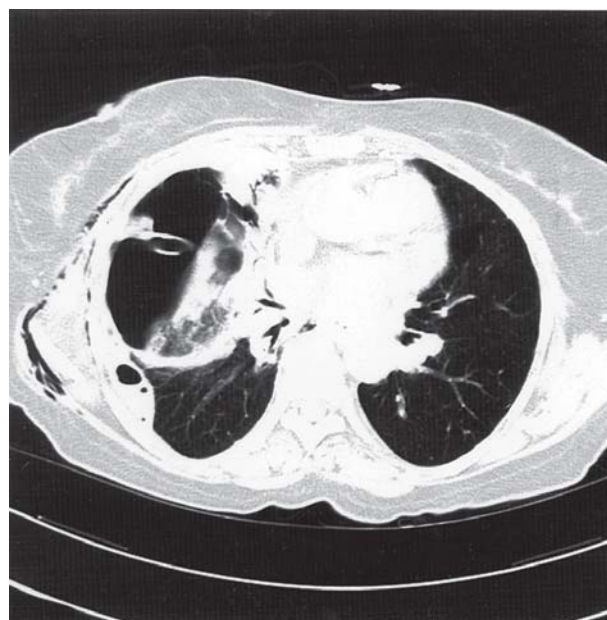


Figure 2. Computed tomography of thorax (lung window) showing a cavity in the right lower zone with a right-sided hydropneumothorax and a chest tube *in situ*.

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thrombus in the right pulmonary artery extending into the lower lobe branches. On right lower limb venous Doppler, a thrombus was found in the right common femoral vein.

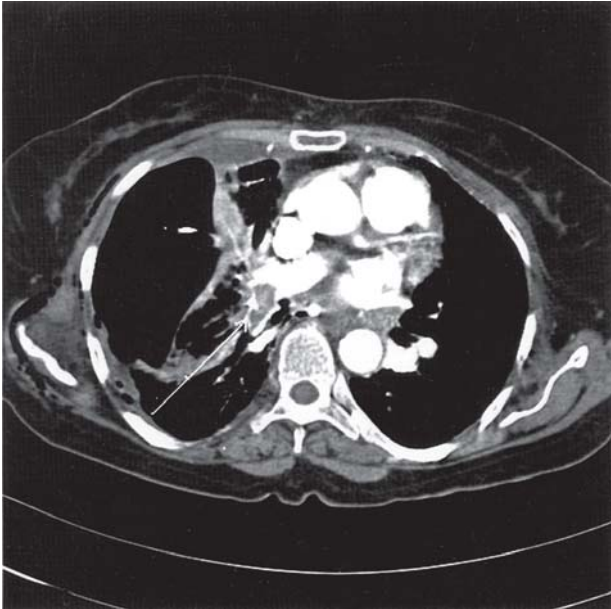


Figure 3. Computed tomographic pulmonary angiography showing thrombus (arrow) in the right pulmonary artery.

Two-dimensional echocardiography showed dilated right atrium and ventricle, right ventricular dysfunction, pulmonary artery systolic pressure estimated by TR jet of 55 mmHg, a dilated pulmonary artery with evidence of thrombus in the right pulmonary artery and a mild to moderate tricuspid regurgitation. Anticoagulation therapy was started. The chest tube was removed after the lung expanded. The patient was stable, thereafter and discharged with an advise to continue anticoagulation therapy for six months with international normalised ratio monitoring. Chest radiograph and CTPA after six months of the therapy showed resolution of the filling defect, regression of the pleural effusion and a cavity with a small residual lesion in the right lower lobe.

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### DIAGNOSIS

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*Pulmonary cavitation and hydropneumothorax associated with pulmonary embolism.*

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### DISCUSSION

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In the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II) study, 94% of patients with pulmonary embolism had one or more predisposing factors.<sup>1</sup> Patients undergoing major, orthopaedic surgery of the lower extremity (total hip replacement, total knee replacement, hip fracture

surgery), are at a very high risk of developing deep vein thrombosis.<sup>2</sup> Cavitation of lung<sup>3,4</sup> and pneumothorax<sup>5</sup> are two uncommon complications of pulmonary infarction, that may occur separately or may rarely coexist.<sup>6,7</sup> Cavitating pulmonary infarcts are either due to a super-added infection of the infarct, or less commonly, due to an aseptic liquefactive necrosis.<sup>3,4,6,8</sup> Levin *et al*<sup>9</sup> found sterile cavitation in 23 (42%) of 550 cases of bland pulmonary infarct. Wilson *et al*<sup>10</sup> found sterile cavitation more, common in infarcts larger than 4cm×4cm, predominantly involving upper and mid lung zone though 20% of their cases did involve the lower lobe. Redline *et al*<sup>4</sup> described barotrauma due to positive pressure ventilation as a cause for cavitation in patients with a bland pulmonary infarct.

Our patient had a right lower lobe cavity and a right-sided hydropneumothorax. As discussed above the cavity possibly resulted from an aseptic necrosis of the pulmonary infarct, as the patient did not have fever and the slight increase in leukocyte count may have been result of pulmonary embolism itself. The incidence of pneumothorax or hydropneumothorax secondary to pulmonary infarction is not known. However, a pneumothorax may result from a rupture of an infarct or cavity near the visceral pleura. The pleural fluid component can also be explained. Pleural effusion due to pulmonary embolism is common, occurring in 20% to 50% cases.<sup>11</sup> This results from an increased permeability of capillaries of lung, either due to inflammatory mediators released by platelet rich emboli or ischaemia of capillaries distal to the site of embolism.<sup>12</sup> Thus, pulmonary embolism should be considered as a differential diagnosis in a case of cavitory pulmonary lesion with or without pneumothorax or hydropneumothorax in the appropriate clinical context.

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