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CASE FOR DIAGNOSIS

A patient with right upper quadrant abdominal pain, hypotension and dyspnoea

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Case history

A 67-yr-old female was transferred to the National Taiwan University Hospital, Tapai, Taiwan on 2 May 2001 after several days of abdominal pain, dyspnoea and hypotension. She initially complained of an intermittent cramping pain over the right upper quadrant (RUQ) of her abdomen 4 days prior to admission. Sudden onset of dyspnoea and altered mental status were noticed 2 days later. She was then sent to the emergency room of a local hospital where a body temperature of 37.5°C and blood pressure reading of 80/30 mmHg were noted. Abdominal sonography showed multiple gallstones and swelling of the gallbladder wall with the "triple-layer" sign. Her haemodynamics were stabilised by fluid challenge and empirical antibiotic therapy. She was then transferred to the National Taiwan University Hospital for further treatment.

The patient was hypertensive for several years while under regular medical control. She had also received hormone replacement therapy for treatment of osteoporosis (1 tablet·day^{-1} of an unknown drug for 2 yrs). Her medical history was otherwise unexceptional.

On examination, she was drowsy. Her body temperature was 37.5°C, blood pressure 160/92 mmHg, pulse rate 120 beats·min^{-1} and respiratory rate 24 breaths·min^{-1}. The sclerae were not icteric. The pupils were isocoric with prompt light reflex. The neck was supple, without jugular vein distension or lymphadenopathy. The chest expanded symmetrically and breathing sounds were clear. No heart murmurs were detected. There was tenderness and muscle guarding at the RUQ abdomen with equivocal Murphy′s sign. The liver and spleen were not palpable. The bowel sounds were hypoactive. No lower leg oedema was noticed. Neither skin rash nor petechiae were found. The initial laboratory studies revealed: a white blood cell count of 16.61×10^9 cells·L^{-1}, with 86.2% neutrophil, 5.6% monocytes and 8.1% lymphocytes; a red blood cell count of 4.19×10^{12} cells·L^{-1}; haemoglobin 7.87 mmol·L^{-1}; and 104×10^9 platelets·L^{-1}. The prothrombin time and activated partial thromboplastin time (PTT) were within normal limits. The asparate aminotransferase was 57 U·L^{-1}, total bilirubin 21.55 μg·dL^{-1}, the alkaline phosphatase 253 U·L^{-1}, the amylase 123 U·L^{-1}, blood urea nitrogen 2.0 mmol·L^{-1}, and creatinine 67.2 μmol·L^{-1}. The arterial blood gas analysis when breathing through a mask at an inspired oxygen fraction of 35% showed the following pH 7.5; carbon dioxide tension in arterial blood (P_{CO_2}) 4.78 kPa (36 mmHg); oxygen tension in arterial blood (P_{O_2}) 17.1 kPa (128 mmHg); and HCO_3^- 25.0 mmol·L^{-1}. The electrocardiography revealed sinus tachycardia only.

The patient underwent a laparoscopic cholecystectomy on 3 May 2001. The operation findings and pathology report were compatible with the diagnosis of chronic cholecystitis. Unfortunately, she developed haemoptysis, persistent RUQ abdominal pain and fever immediately after the cholecystectomy. Chest examination showed decreased breathing sounds over the lower right lung field with some basal crackles.

Imaging studies, including the initial chest radiograph at the local hospital (fig. 1), the chest radiographs immediately after cholecystectomy (fig. 2) and 8 weeks after the cholecystectomy (fig. 3), and the computer tomography (CT) scans of the chest 7 days after the cholecystectomy (figs. 4a and b) are shown.

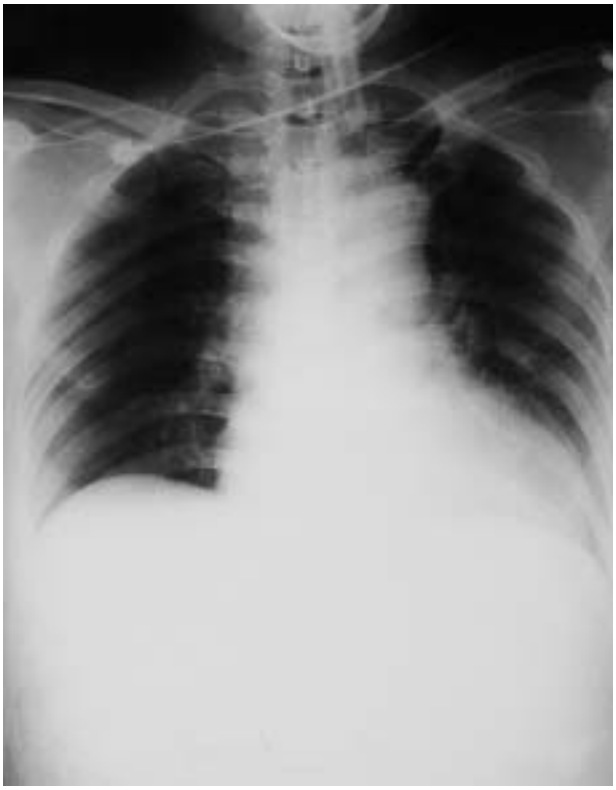

Fig. 1. – Initial chest radiography of a 67-yr-old female after visiting a local hospital because of right upper quadrant abdominal pain, dysponea, and hypotension.

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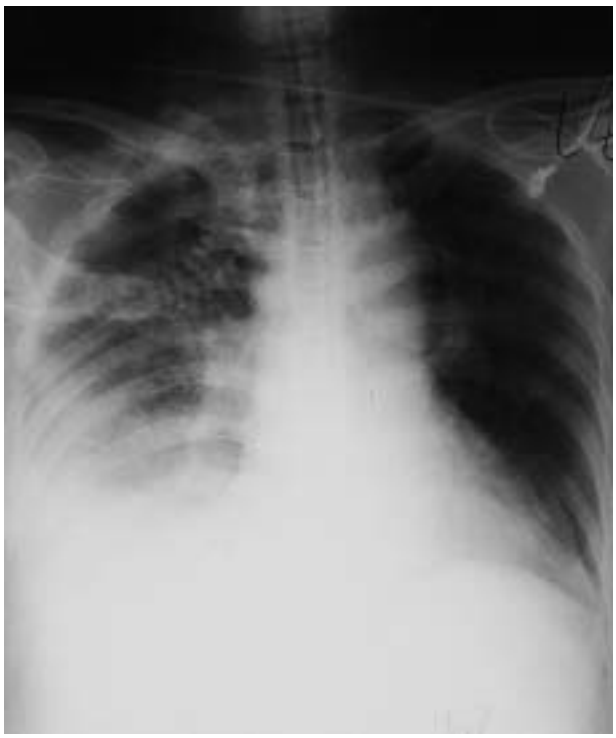

Fig. 2. – Portable bedside chest X-ray film immediately after cholecystectomy, while fever, right chest pain and haemoptysis developed.

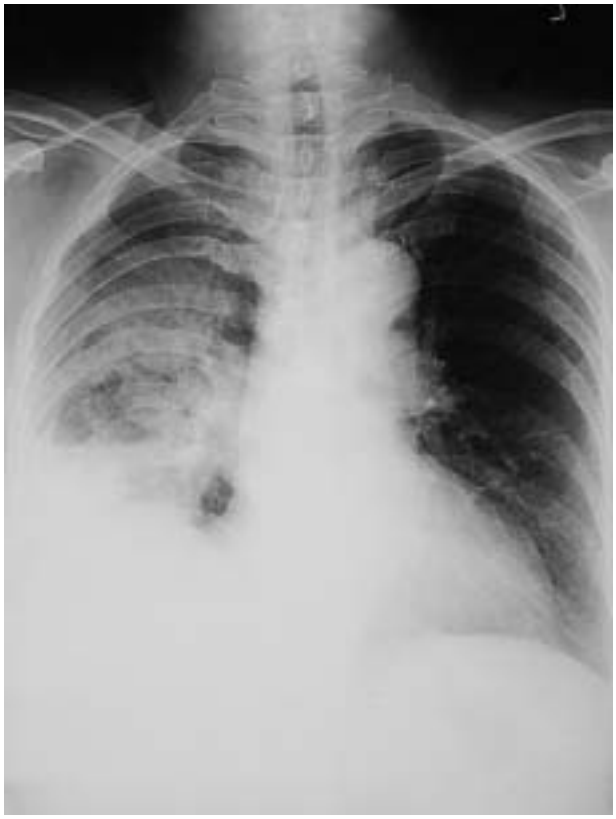

Fig. 3. – Follow-up chest radiography 8 weeks after cholecystectomy.

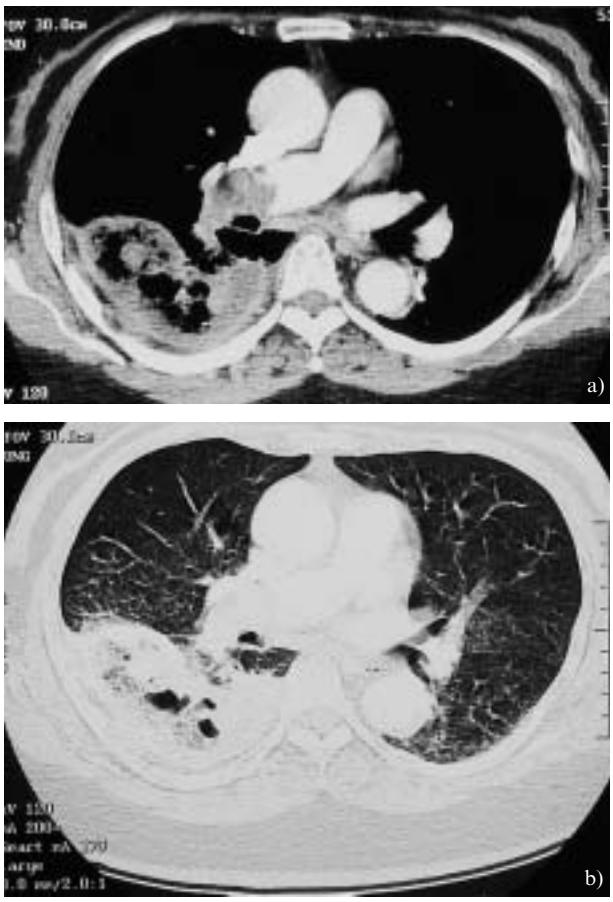

Fig. 4. – Computed tomography scans of the chest 7 days after cholecystectomy. a) The mediastinal window at the level of the aortopulmonary window and b) the parenchymal window at the level of the lower lung fields.

BEFORE TURNING THE PAGE, INTERPRET THE LABORATORY AND IMAGING STUDIES AND SUGGEST A DIAGNOSIS.

Interpretation of the chest radiograph and computed tomography scan

The initial chest radiograph (fig. 1) is clear, but the image immediately after the cholecystectomy (fig. 2) shows alveolar consolidation over the right lower lung and blunting of the right cardiophrenic angle. The endotracheal tube was also visible in the right lung field. The CT scans of the chest (figs. 4a and b) reveal a large filling defect in the right main pulmonary artery and consolidation of the lower right lung with cavitation. The follow-up chest radiograph (fig. 3) revealed prominent cavitation of the previous lung consolidation with air-fluid level.

The pulmonary angiography showed a large filling defect in the right pulmonary artery, without visualisation of the branches of the right pulmonary artery to the right lower lobe. The duplex study of deep veins of the lower limbs was negative, and the echocardiography did not show right ventricular (RV) thrombi. The serum levels of protein C, protein S and antiphospholipid antibodies were within normal limits. The autoimmune profiles were also normal. The sputum culture grew *Pseudomonas aeruginosa*.

Diagnosis: "Cavitary pulmonary infarct"

High doses of intravenous ceftazidine and heparin were administered immediately after the findings of the CT scans were obtained on the 7th day after the cholecystectomy, and the activated PTT was maintained at 1.5–2.0 folds of the control. However, the follow-up chest radiograph 8 weeks later showed only partial resolution at the consolidation of the lower right lung with cavity formation, and the CT scans did not reveal shrinkage of the thrombi in the right pulmonary artery. Both the perfusion scan and the Doppler sonography failed to show reperfusion of the infarcted lung. The patient therefore underwent a lobectomy of the right lower lobe and an embolectomy on 5 July 2001. Culture of the removed lung tissues still yielded *P. aeruginosa*, which was sensitive to the antibiotics used. The postoperational course was smooth and the patient was discharged on the 10th hospital day after the operation, with continued maintenance therapy of anticoagulation.

Discussion

The most striking and unexpected CT scan finding of the case reported here is the huge filling defect in the right main pulmonary artery. An attempt to take biopsy of the lesion during angiography was not successful due to technical difficulties. Besides pulmonary embolism, other unusual aetiologies that should be listed in the differential diagnosis include primary or metastatic angiosarcoma or leimyosarcome, and lung cancer invading the main pulmonary artery. Right ventricular outflow tract tumour mimicking a thrombus in the main pulmonary artery has also been reported [1].

Pulmonary infarction follows an embolic event in ~10% of cases [2]. The reasons for this low incidence of pulmonary infarct are the dual blood supply systems, as well as oxygenation of the lung tissues *via* ventilation [3]. The predisposing factors for pulmonary infarct include congestive heart failure, pleural effusion, pulmonary infection, atelectasis, hypotension, positive-pressure ventilation, chronic lung disease, central venous catheterisation and an immunocompromised state [4].

Multiple complications have been associated with pulmonary infarct, including empyemae, pneumothorax, lung abscess, bronchopleural fistulae and lethal haemorrhage. Cavitary pulmonary infarct is a rare but frequently misdiagnosed disease entity. Large series of autopsies revealed cavitation in 4–5% of all pulmonary infarcts [4]. The greater the size of the infarct, the more likely its centre will be hypoxic and necrotic. Pulmonary infarcts larger than 4×4 cm in size have a greater tendency for cavitation [5]. The median time from the first detection of consolidation to cavity formation is 14 days [5]. Other diseases that should be listed in the differential diagnosis include aspergillosis, tuberculosis, norcardia, actinomycosis, malignancy and granulomatous vasculitis.

Cavitation after bland pulmonary infarcts may result from either aseptic necrosis of the infarcted lung or from secondary bacterial infection with subsequent abscess formation. It is infective almost as often as it is aseptic [6]. Two types of infected pulmonary infarct have been proposed based on the mode by which infection sets in [7]. One is called "primary" because the infection is from a septic embolus. The other is called "secondary" because the infection is bronchogenic in origin. Some authors suggest that the development of fever and/or purulent sputum following a pulmonary infarct is highly suspicious for secondary infection [2]. The spectrum of causative agents for infected pulmonary infarct is similar to that of nosocomial pneumonia [2].

Cavitary pulmonary infarct may sometimes be confused with pulmonary gangrene. Pulmonary gangrene is a rare complication of pneumonia, defined as the sloughing of a large amount of infected lung tissue with *in situ* thrombosis of large vessels. Typical radiological changes of pulmonary gangrene include an initial lobar pneumonia and then multiple small radiolucent densities that ultimately fuse into a large cavity.

Pulmonary gangrene is more often located in the upper than lower lobes [8, 9], but pulmonary infarct is predominantly located in the lower lobes [8, 9]. Pulmonary gangrene usually involves an entire segment or lobe because of extensive occlusion of the pulmonary and bronchial arteries. Pulmonary infarct following embolism is usually located peripherally, due to the supply of the proximal bronchi from bronchial circulation. The possibility of concomitant pulmonary embolism and pneumonia seems unlikely because the hypoperfusion in pneumonia decreases the lodge rate of an embolism [8].

Previous studies have reported a mortality rate as high as 41% and 73% for noninfected and infected cavitary pulmonary infarcts, respectively [4]. Anticoagulants and antibiotics are the mainstays of

therapy. Massive haemoptysis may persist even after discontinuation of anticoagulants [7]. Possible explanations for this phenomenon are an overdose of anticoagulants and reperfusion of necrotic lung tissue. Anticoagulant use in cavitary pulmonary infarction, therefore, must be very carefully monitored and caution should be exercised in monitoring clinical conditions and the status of coagulation. In this case, thrombolytic therapy was not performed because of stable haemodynamics and for fear of worsening the patient's persistent haemoptysis. Pulmonary embolectomy and percutaneous mechanical fragmentation are alternatives to thrombolytic therapy for patients at high risk of bleeding. Nevertheless, early surgical intervention has become controversial with the advance of antibiotics. Aggressive surgical therapy is indicated in uncontrolled sepsis, persistant haemorrhage and chronic organised pulmonary thromboemboli [4].

For patients with pulmonary embolism and subsequential infarct, reperfusion of the lung is important for the success of anticoagulant and thrombolytic therapies. Spiral computed tomography and magnetic resonance angiography could help track the resolution of the thromboemboli, but they are expensive and unavailable in many hospitals. Doppler sonography is a noninvasive and convenient tool for diagnosing pulmonary embolism and follow-up reperfusion of the lung [10]. Dynamic change in blood flow in consolidated areas provides information about the status of reperfusion. In this case, no visible blood flow was detected in the consolidated lung, even after large doses of heparin therapy for 8 weeks, indicating nonviability of the infarcted tissue and the need for surgical removal.

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