A Case of Pulmonary Embolism after Abdominal Angiography

HISAFUMI KINOSHITA, MASAO HARA, YUICHIRO MARUYAMA, HIDEKI MATSUO, RYUICHI KAWAHARA, KAZUNORI NISHIMURA, TAKAHITO KODAMA, MASAHARU ODO, KAZUO SHIROUZU AND SHIGEAKI Aoyagi

Department of Surgery, Kurume University School of Medicine, Kurume 830-0011, Japan

Summary: The patient was a 72-year-old woman who had been diagnosed with cholecystolithiasis and had undergone laparoscopic cholecystectomy. Since the postoperative pathologic diagnosis was a gallbladder cancer with a depth of wall penetration of subserosa, she was admitted to Kurume University Hospital for a second-look operation. After admission, abdominal angiography was performed with a right femoral arterial puncture. After the release of inguinal compression with a belt, chest pain and difficulty in breathing appeared. Despite her normal blood pressure, arterial blood gas analysis showed a PO2 of 74.7 mmHg and a PCO2 of 41.5 mmHg, representing a slight decrease in PO2. Chest X-rays showed an increased cardiothoracic ratio and decreased lucency in the left upper lung field. The electrocardiogram revealed atrial premature contraction. Cardiac ultrasound did not show expansion of the right heart and blood vessels or abnormal structures in the main pulmonary artery. Since lung perfusion scintigraphy revealed perfusion defects in the left upper to middle and right upper lung fields, acute pulmonary embolism was diagnosed, and oxygen inhalation, thrombolytic, and anticoagulant therapy were instituted immediately. The symptoms improved the following day, but 240,000 u/day of urokinase was administered for 5 days, and 1,500 u/day of heparin for 10 days. On lung perfusion scintigrams 6 days later, the defects had disappeared. Moreover, no definite abnormal shadows were noted on chest X-rays. Radical surgery for gallbladder cancer was performed 3 weeks later. Considering the possible development of pulmonary embolism, we felt the need for careful management if the patient is released from bed rest after abdominal angiography.

Key words pulmonary embolism, abdominal angiography, lung perfusion scintigraphy

INTRODUCTION

The incidence of pulmonary embolism in Europe and the United States is high, while the condition has been thought to be relatively rare in Japan. However, in recent years, it has been on the rise in Japan with the westernization of dietary habits. Here we report a patient in whom pulmonary embolism developed after abdominal angiography and whose life was saved by thrombolytic and anticoagulant therapies. We also review the relevant literature.

CASE REPORT

The patient was a 72-year-old woman who had been diagnosed with cholecystolithiasis one month previously and had undergone laparoscopic cholecystectomy. Since the postoperative pathologic diagnosis was a gallbladder cancer with a depth of wall penetration of subserosa, she was admitted to Kurume University Hospital for a second-look operation. The patient was 145 cm tall and weighed 65.6 kg on admission. The palpebral conjunctivae were not anemic, and the bulbar conjunctivae were not icteric.
Superficial lymph nodes were not palpable. A surgical wound due to laparoscopic cholecystectomy was present on the abdomen, but no mass was palpable. Blood biochemical tests did not show any abnormalities or an increase in tumor markers. As a preoperative examination for radical surgery for gallbladder cancer, abdominal angiography was performed with a right femoral arterial puncture. An angiographic sheath was placed in the artery for 27 min, and the puncture site was compressed for 10 min after withdrawal of the sheath. The patient was transferred to the ward, where the compression was released 3 hrs after the end of the angiography. She lay quietly for 2 hrs after the release of pressure. On standing up after the pressure was released, she felt chest pain and difficulty in breathing. Her blood pressure was normal, but arterial blood gas analysis showed a PO$_2$ of 74.7 mmHg and a PCO$_2$ of 41.5 mmHg, representing a slight decrease in PO$_2$. Blood biochemical tests did not show any abnormalities. Chest X-rays showed an increased cardiothoracic ratio (CTR) and decreased lucency in the left upper lung field (Figs 1a, b). The electrocardiogram revealed arterial premature contraction. Cardiac ultrasound did not show expansion of the right heart and blood vessels or abnormal structures in the main pulmonary artery (main PA) (Fig. 2). Lung perfusion scintigraphy revealed perfusion defects in the left upper to middle and right upper lung fields (Fig. 3a). Acute pulmonary embolism was diagnosed, and oxygen inhalation, thrombolytic and anticoagulant therapies were instituted immediately. The symptoms improved the following day, but 240,000 u/day of urokinase was administered for 5 days, and 1,500 u/day of heparin for 10 days. On lung perfusion scintigrams 6 days later, the defects had disappeared (Fig. 3b). Moreover, no definite abnormal shadows were noted on chest X-rays (Fig. 1c). Radical surgery for gal-
PULMONARY EMBOLISM

Fig. 3. Lung perfusion scintigraphy revealed perfusion defects in the left upper to middle and right upper lung fields (a). On lung perfusion scintigrams 6 days after angiography, the defects had disappeared (b).

bladder cancer was performed 3 weeks later. The patient's course has been favorable since she was discharged.

DISCUSSION

Acute pulmonary embolism occurs in 10 to 15% of the European and US populations [1,2], while it is thought to occur less frequently in Japan. However, in recent years, the condition has been on the rise in Japan with the westernization of life styles, the increase in cardiovascular disease, and advances in diagnostic techniques, and it is predicted that the condition will occur in ever-increasing numbers. Over 90% of pulmonary embolisms are thought to be caused by deep vein thrombosis [3]: thrombi formed in deep veins become dislodged from the site of formation to embolize to the pulmonary arteries, causing chest pain, dyspnea, and other symptoms. Deep vein thrombosis is thought to result from (1) stasis, (2) vascular endothelial damage, and (3) hypercoagulability. Stasis is caused by prolonged sitting or prolonged bed rest following surgery or childbirth. Vascular endothelial damage is due to tumor invasion or trauma. Hypercoagulability is produced by pregnancy, splenectomy, or malignant tumor. In this patient, acute pulmonary embolism occurred after celiac arteriography and common hepatic arteriography by percutaneous aortic catheterization (the Seldinger technique), and the direct cause was bed rest following angiography, probably producing thrombi in deep veins. The reported complications of the Seldinger technique include arterial thrombosis, injection of contrast medium into the arterial wall or beneath the intima, arterial spasms, bleeding from the puncture site or hematoma formation, severance of a catheter or guidewire, and contrast medium-induced shock. The prognosis of acute pulmonary embolism is extremely poor. Bell et al. [4] reported that the mortality rate was 33%, of which 10% died within 1 hour of onset, 2% died after diagnosis and treatment, and 21% remained undiagnosed until death. Thus, it has been indicated that the prognosis depends on early diagnosis and treatment. The clinical symptoms include chest pain, dyspnea of sudden onset, cough, tachycardia, and, in severe cases, shock. However, the diagnosis based on clinical symptoms alone is difficult, and requires examinations such as blood biochemical tests, arterial blood gas analysis, chest X-ray, electrocardiography, and cardiac ultrasound. Lung perfusion scintigraphy and pulmonary angiography are useful to establish the diagnosis [4,5]. Blood biochemical test findings of elevated LDH and bilirubin levels and a normal GOT level are not diagnostically useful as has been pointed out. Arterial blood gas analysis generally reveals hypox-
emia and hypocarbia. It is difficult to diagnose acute pulmonary embolism from chest roentgenograms, but increased lucency in lung fields, the expansion of pulmonary arterial shadows at the hilus, and an increased cardiac shadow are findings suspicious for the condition. Electrocardiograms show inverted T waves in V1-3 and right axis deviation; however, small emboli produce little change, which is thought rather important in excluding acute myocardial infarction. Lung perfusion scintigraphy is very useful for the diagnosis. This technique is less invasive and superior in follow-up studies, and visualizes lesions as solitary or multiple perfusion defects. In order to differentiate between pulmonary embolism and disturbed pulmonary blood flow due to ventilatory impairment as in chronic obstructive pulmonary disease, the concomitant use of lung perfusion scintigraphy is needed. In pulmonary embolism, blood flow disturbance is marked in the absence of ventilatory impairment, and this discrepancy produces a ventilation-perfusion mismatch [5]. Pulmonary arteriography is the most effective examination for diagnosing the condition and deciding on the course of treatment. Angiographic demonstration of a filling defect and a cutoff sign establishes the diagnosis. Recently, a less invasive digital subtraction angiography has been performed [6]. In this patient, arterial blood gas analysis showed a decrease in PO2 in the absence of chest X-ray or electrocardiographic features arousing suspicions about this condition. The lung perfusion scintigraphic visualization of perfusion defects in both lung fields established the diagnosis of pulmonary embolism.

The conservative treatment of this condition includes hemolytic therapy chiefly with the thrombolytic agent urokinase and anticoagulant therapy with heparin or warfarin, both of which prevent additional thrombus formation. In addition, recent studies have reported the efficacy of the administration of a tissue plasminogen activator having a high affinity for fibrin and specifically degrading only thrombi, or of prostaglandin E1, dilating pulmonary blood vessels [7]. In severely ill patients or those who fail to respond to conservative therapy, surgical treatment such as embolectomy with a pump-oxygenator or a balloon catheter is performed [8,9]. In this patient, pulmonary embolism was suspected at a relatively early stage, and thrombolytic and anticoagulant therapies with urokinase and heparin promptly corrected blood gasses and diminished the pulmonary emboli; thus, the symptoms were relieved by conservative treatment alone.

It has been reported that antithrombotic measures, such as lower extremity massage, application of an elastic bandage, and low-dose heparin therapy, are effective in preventing thrombus formation. As antithrombotic measures, we use elastic stockings, which have a low risk of hemorrhage and are non-invasive and simple, and perform the intermittent air compression technique. It is essential that examinations be conducted if the condition is suspected, and thrombolytic or antithrombotic therapy be instituted if suspicions increase.

REFERENCES