Multiple Pulmonary Infarctions Associated with Lung Cancer

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We present a case of right lung adenosquamous cell carcinoma that had obstructed the main pulmonary artery and superior pulmonary vein, causing multiple pulmonary infarctions in the right upper and middle lobes. Multiple peripheral pulmonary nodules showed clinical features that are characteristic of pulmonary infarction: rapid appearance and gradual reduction in size, pleural-based parenchymal density with a truncated apex and a round nodular shadow with a blurred margin and a centrally directed linear shadow. The nodules were more intense than the primary tumor in both T1- and T2-weighted magnetic resonance imaging (MRI). We conclude that pulmonary infarction can look like a nodule when lung cancer invades both the pulmonary artery and vein and that such cases can be distinguished from pulmonary metastasis by MRI, computed tomography and a series of radiological examinations.

Key words: lung cancer – pulmonary infarction – magnetic resonance imaging – computed tomography – pulmonary metastasis

INTRODUCTION

Pulmonary infarction due to obstruction of the pulmonary artery and vein is sometimes associated with central lung cancer (1–4). Differentiating pulmonary infarction from metastasis is critical to tumor staging and the indications for surgery. Several reports have demonstrated the usefulness of computed tomography (CT) and magnetic resonance imaging (MRI) in diagnosing pulmonary infarction (5–7). We present a case of central lung cancer that caused obstruction of the right main pulmonary artery and superior pulmonary vein and was associated with multiple peripheral pulmonary nodules. The nodules were preoperatively diagnosed as pulmonary infarctions rather than pulmonary metastatic lesions based on CT and MRI findings and a series of chest roentgenograms.

CASE REPORT

A 66-year-old male complained of a dry cough and dyspnea on exertion in March 1998, but a chest roentgenogram showed no abnormal findings. On May 20, 1998, he complained of hemoptysis and a chest roentgenogram showed right pleural effusion and parenchymal lesions in the upper and middle lobes. He was admitted to our hospital on June 9, 1998.

A chest roentgenogram taken on admission showed that the upper lobe lesion had decreased in size, whereas the middle lobe lesion was enlarged (Fig. 1). Laboratory test findings revealed no inflammation. Bronchoscopy showed stenosis of the right middle lobe bronchus and a biopsy specimen showed squamous cell carcinoma. Chest CT and MRI showed that a tumor at the lung hilum 5 cm in diameter had invaded the main pulmonary artery, superior pulmonary vein and left atrium (Fig. 2). A lung perfusion scan using ⁹⁹ᵐTc macroaggregated serum albumin showed no perfusion to the right lung (Fig. 3). The lesions in the upper lobe were multiple round nodules, 10–20 mm in diameter, with blurred margin on CT. They were located in the subpleural zone and also had centrally directed...
linear shadows (Fig. 4). The lesion in the middle lobe was 4 cm in diameter and showed broadly pleural-based parenchymal density with a truncated apex and convex border. No abnormal shadow was seen in the right lower lobe or in the left lung. MRI showed that the pulmonary lesions in both the upper and middle lobes were hyperintense compared with the primary tumor on both T1- and T2-weighted images (Fig. 5). These findings suggested that the upper and middle lobe lesions were probably pulmonary infarctions, caused by obstruction of both the main pulmonary artery and superior pulmonary vein by tumor invasion rather than metastatic lesions. Other examinations showed no distant metastasis.

On June 22, 1998, intrapericardial right pneumonectomy with mediastinal lymph node dissection was conducted. The tumor had invaded the main pulmonary artery, superior pulmonary vein and left atrium. The main pulmonary artery was cut within the pericardium and the left atrium was partially resected. The postoperative course was satisfactory and the patient was discharged 17 days after surgery.

Histopathological examination showed adenosquamous cell carcinoma originating in the middle lobe bronchus, obstructing the main pulmonary artery and superior pulmonary vein and extending to the left atrium. All of the peripheral pulmonary lesions in both the upper and middle lobes showed coagulation necrosis with slight hemorrhage, which were diagnosed as pulmonary infarctions. No tumor thrombus was seen. The patient died of tumor recurrence in the mediastinum, bone, kidney and intestine 11 months after surgery.

DISCUSSION

Pulmonary infarction is rarely caused by obstruction of a pulmonary artery and/or vein by lung cancer rather than by...
tumor thrombus (1–4). The radiological characteristics of pulmonary infarction associated with lung cancer have been reported (1–5) to be (i) pulmonary shadows at the periphery of the same lobe containing the primary tumor, (ii) peripheral pulmonary shadow appearing rapidly and/or gradually decreasing in size, (iii) smaller infarctions 10–25 mm in diameter in the subpleural zone or away from the pleural that are round or polygonal in shape, blurred at the margin and that demonstrate a centrally directed linear shadow and (iv) larger infarctions showing a broadly pleural-based parenchymal density with a truncated apex, convex border, centrally directed linear shadow and central low attenuation.

While no reports of MRI findings for pulmonary infarction associated with lung cancer have been published, to our knowledge, Kessler et al. (7) reported their MRI findings for pulmonary infarction due to thromboembolism: (i) isointense lesions on T1-weighted and hyperintense lesions on T2-weighted images in the early acute phase due to alveolar hemorrhage and (ii) hyperintense lesions on T1- and T2-weighted images 24 h to 7 days after infarction due to alveolar hemorrhage and methemoglobin formation. The histological findings of the lesions in our case showed coagulation necrosis with slight hemorrhage, which was considered to have occurred some time after infarction. From the clinical course, the pulmonary infarctions had occurred more than 1 month before operation. Although we did not measure methemoglobin in the lesions, the hyperintensity on T1- and T2-weighted images would be caused by methemoglobin formation rather than a hemorrhage.

In our case, peripheral pulmonary lesions in both the upper and middle lobes demonstrated features characteristic of pulmonary infarction: (i) they appeared rapidly, within 2 months, and the upper lobe lesions decreased in size; (ii) the right main pulmonary artery and superior pulmonary vein were obstructed by the primary tumor; (iii) multiple nodules in the upper lobe were round, blurred at their margins and had centrally directed linear shadows; (iv) the lesion in the middle lobe was pleural-based with a convex border; and (v) MRI showed that these lesions were more intense than the primary tumor in T1- and T2-weighted images.

Pulmonary infarction does not occur in all cases of central pulmonary artery occlusion because of the presence of a more distal broncho-pulmonary artery anastomosis (8). Increased pulmonary vein pressure, as seen in heart failure, shock and neoplasm, is an important factor in pulmonary infarction (8–10). In the present case, multiple pulmonary infarctions were seen in the upper and middle lobes but there were none in the lower lobe, the pulmonary vein of which was not obstructed by the tumor. We therefore conclude that pulmonary infarction can occur when lung cancer invades both the pulmonary artery and vein and that such cases can be distinguished from cases of pulmonary metastasis by the CT and MRI findings and a series of radiological findings.

References