

Pulmonary Adenocarcinoma Complicated with Pulmonary Infarction Presented as Intrapulmonary Metastases: A Report of a Case

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Pulmonary adenocarcinoma complicated with a pulmonary infarction presenting as an intrapulmonary metastasis is relatively rare. We present a case of pulmonary infarction manifesting as intrapulmonary metastases of lung cancer. A previously healthy 59-year-old woman was admitted to our hospital for evaluation of abnormal shadows in the right lower lung field. Laboratory tests showed no abnormalities except for a slight elevation of carcinoembryonic antigens (CEAs). Computed tomography (CT) of the chest revealed a hilar mass lesion with parenchymal lesions in the periphery of the right lower lobe, highly suspected to be a pulmonary adenocarcinoma with intrapulmonary metastases. A diagnosis of pulmonary adenocarcinoma was confirmed by a transbronchial brushing examination. A right middle and lower bilobectomy with mediastinal lymph node dissection was due to hilar lymphadenopathy and a lower lobe invasion of the main tumor. Histopathological findings of the resected specimens revealed poorly differentiated adenocarcinoma of the lung with N1 (#11i and 12 l) disease and multiple pulmonary infarctions with coagulation necrosis and recanalization. Our case suggests that pulmonary infarction associated with lung cancer should be considered as one important cause of peripheral pulmonary nodules. (Ann Thorac Cardiovasc Surg 2006; 12: 189–93)

Key words: pulmonary adenocarcinoma, pulmonary infarction, peripheral pulmonary nodule, intrapulmonary metastasis

Introduction

Pulmonary adenocarcinoma complicated with a pulmonary infarction presenting as an intrapulmonary metastasis is relatively rare. A pulmonary infarction may mimic resolving or stable pulmonary metastasis in patients with lung cancer. A survey of English reports revealed that

approximately 0.16-5% of surgical cases of lung cancer are complicated by pulmonary infarctions. We present a case of pulmonary infarction manifesting as intrapulmonary metastases of lung cancer.

Case Report

A previously healthy 59-year-old woman was admitted to our hospital on May 16, 2002 for evaluation of the multiple abnormal radiographic shadows in the right lower lung field (Fig. 1). She had not developed any right chest pain, breathlessness on exertion, dry cough, malaise, or weight loss. Physical examination revealed no abnormal findings except for a little reduction in breath sounds around a small area of the right lower lung field. There were no palpable supraclavicular, axillar or inguinal nodes. The forced expiratory volume in 1 second (FEV1.0) was

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Fig. 1. Chest x-ray showed multiple abnormal shadows in the right lower lung field.

1.97 L, and the vital capacity (VC) was 2.62 L.

Laboratory tests, which included alkaline phosphatase (ALP), creatine kinase (CK), lactate dehydrogenase (LDH), and tumor markers of sialyl Lex-i antigen (SLX), and CYFRA21-1, showed no elevation and no inflammation except for a slight elevation of carcinoembryonic antigens (CEAs). Sputa were negative for tuberculosis and fungi, and cytological studies were unremarkable. Although a fiberoptic bronchoscopy revealed no endobronchial abnormalities, a diagnosis of pulmonary adenocarcinoma was confirmed by the examination of transbronchial brushing.

A computed tomography (CT) of the chest revealed a hilar mass lesion (3.1 cm in diameter) with parenchymal lesions in the periphery of the ventrobasal right lower lobe with some thickening of the overlying pleura, which was highly suspected to be a pulmonary adenocarcinoma with intrapulmonary metastases (Fig. 2). No associated mediastinal lymphadenopathy except of the hilum node (#11i) was evident. No distant metastases were observed with bone scintigraphy, abdominal or chest CT, or brain magnetic resonance imaging (MRI). The clinical diagnosis

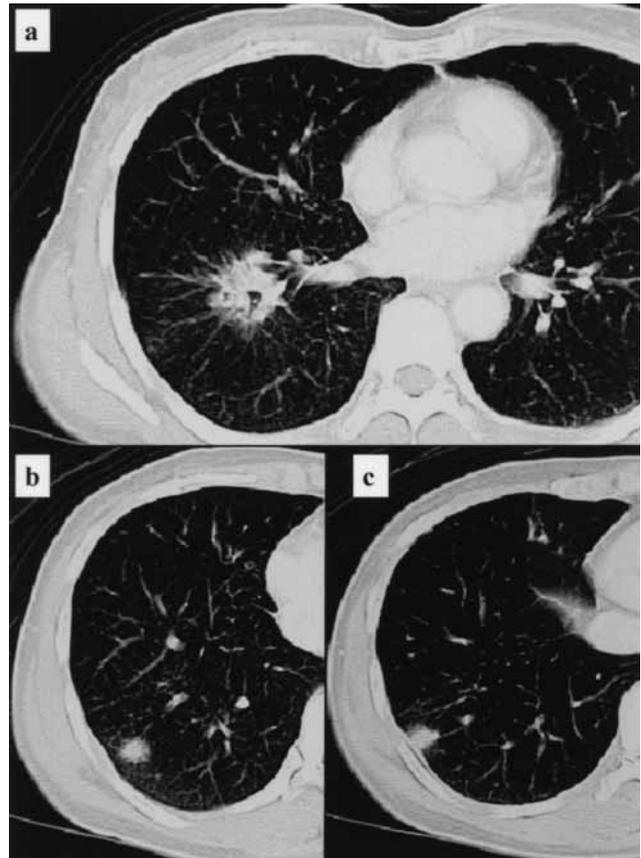


Fig. 2. CT of the chest revealed a hilar mass lesion (a) with parenchymal lesions (b, c) in the periphery of the ventrobasal right lower lobe.

was primary lung cancer of stage IIIB disease (T4N1M0) originating in the right lower lobe with intrapulmonary metastases at the periphery of the same lobe containing the primary tumor.

Surgical operation was performed through a right postero-lateral thoracotomy by using a double-lumen endotracheal tube for contralateral ventilation and ipsilateral lung collapse on May 21, 2002. Before thoracotomy the trocar was introduced into the pleural cavity through the seventh intercostal space in the mid-axillary line for evaluation of an absence of dissemination or pleural effusion. The central surface of the peripheral mass was gray-white (Fig. 3). Right middle and lower bilobectomy with mediastinal lymph node dissection was due to hilar lymphadenopathy (#11i) and suspicion of having a middle lobe invasion of the main tumor. Frozen section examination revealed primary lung cancer with multiple pulmonary infarctions with no evidence of metastases.

Permanent histopathological findings of the resected

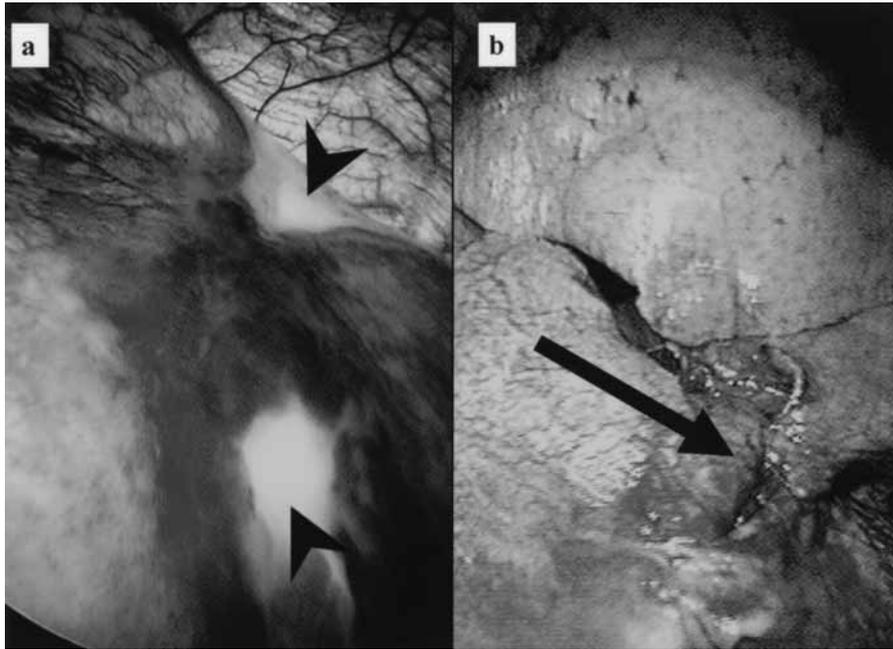


Fig. 3. The central surface of the peripheral mass was gray-white (a, arrowheads). Right middle and lower bilobectomy was needed by suspicion of having a middle lobe invasion of the main tumor (b, arrow).

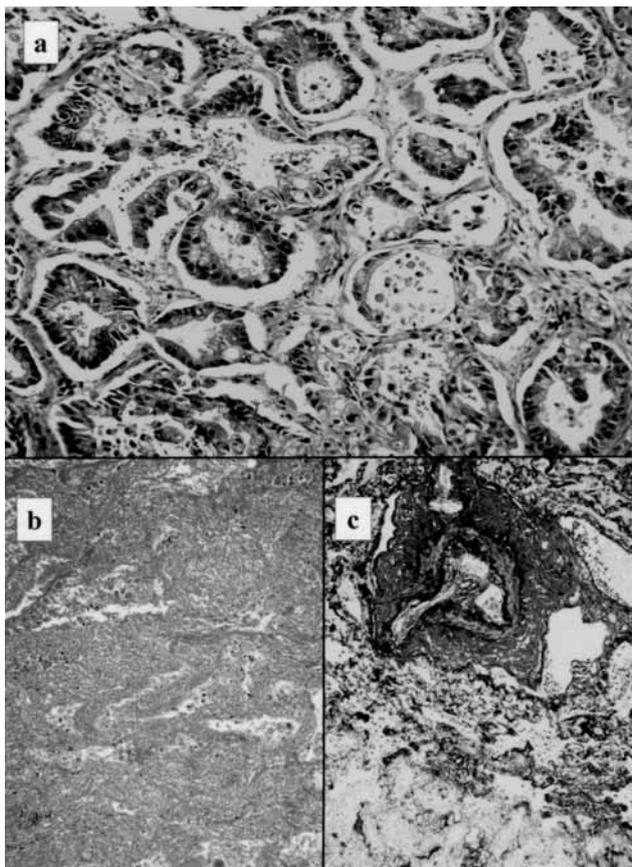


Fig. 4. Histopathological findings of the resected specimens revealed poorly differentiated adenocarcinoma of the lung (a) with multiple pulmonary infarctions with coagulation necrosis (b) and recanalization (c). (a, HE: $\times 50$; b, HE: $\times 10$; c, EVG: $\times 10$)

specimens revealed poorly differentiated adenocarcinoma of the lung with N1 (#11i and 12 l) disease and multiple pulmonary infarctions with coagulation necrosis and recanalization (Fig. 4). Blood vessel compression near the hilar tumor mass and enlarged lymphnodes were revealed. Occlusion of blood vessels was not observed.

The postoperative course was uneventful. The patient has been doing well for 41 months postoperatively.

Discussion

The incidence of pulmonary embolism/infarction is raising gradually in Japan. In addition, lung cancer is known as a risk factor of pulmonary embolism (PE). PE is a common disorder associated with considerable morbidity and mortality. Many cancers induce a hypercoagulable state and thrombotic pulmonary embolism is the second most common cause of death in patients with solid tumors.¹⁾ Postmortem studies have demonstrated that pulmonary arterial tumor embolism occurs in 8-20% of advanced cancers, being particularly associated with lung cancer. Hanbury et al.²⁾ described 10 cases of pulmonary infarctions associated with bronchogenic carcinoma, those were collected from a series of 100 pneumonectomy cases performed on bronchogenic carcinomas. Yoshida et al.³⁾ described 16 cases (3.1%) of pulmonary infarction, chosen from among 518 cases of lung cancer. Adenocarcinoma was found in 8 cases, squamous cell carcinoma in 6, and

adenosquamous carcinoma in 2. Chest radiograms and CT revealed infarction shadows in 8 of the 16 cases.

Scoggins et al.⁴⁾ reported 3 cases of false-positive cytological diagnosis of lung cancer in patients with pulmonary infarctions. On the other hand, lung cancer causes perfusion defects that are sometimes mistaken for pulmonary emboli on lung scans. This may result from several mechanisms: (1) compression of the blood supply by the tumor is the most obvious cause of pulmonary artery obstruction; (2) replacement of pulmonary arterial flow by bronchial arterial circulation through the tumor mass may cause the perfusion defects seen on lung scans; (3) abnormal perfusion may result from a region of obstructed ventilation; and (4) air trapping past a tumor may raise intra-alveolar pressures above pulmonary arterial pressures resulting in impeded blood flow.⁵⁾

Pulmonary infarction should be considered in patients with centrally invasive pulmonary carcinoma, when a small ill-defined opacity appears at the periphery of the same lobe over a short period.⁶⁾ Occlusion or invasion of the pulmonary artery in a hilar tumor is thought to be responsible for pulmonary infarction. Pulmonary infarction, however, is uncommon when emboli obstruct central arteries but frequent when distal pulmonary arteries are occluded. This is explained by the fact that collateral flow from bronchial artery circulation enters the pulmonary artery circulation distal to the site of central obstruction.⁷⁾ Another risk factor for pulmonary infarction is increased pulmonary vein pressure, as seen in heart failure, shock and neoplasm.⁸⁾ In our case, pulmonary arteries and veins were compressed by the hilar mass and enlarged lymph node. Occlusion of blood vessels was not observed.

It may be rare for lung cancer to be associated with pulmonary infarction showing a tumor mass. In some reports, pulmonary infarctions were resected as pulmonary tumors.⁹⁾ Although the most common clinical complaint in patients with pulmonary infarction is nonspecific chest wall pain or bloody sputum, our case had no complaints of pain or other signs and was not clinically suspected to have a pulmonary infarction. Therefore, the surgical operation was performed with a diagnosis of lung cancer with intrapulmonary metastases.

Pulmonary infarctions are demonstrated on chest x-rays as round or polygonal in shape, blurred in margin, and located at the periphery of the same lobe as the primary tumor. CT is more sensitive than conventional radiography in the detection of pulmonary thromboembolism and infarction. Balakrishnan et al.¹⁰⁾ concluded that their review showed that the CT appearance of pulmonary inf-

arction is distinctive enough to strongly suggest a diagnosis based on CT alone. The typical radiological characteristics of pulmonary infarction associated with lung cancer have been reviewed to be: (1) shadows located in the same lobe and periphery as the cancer; (2) ill-defined, 10-25 mm nodular shadows that are round or polygonal in shape, blurred at the margin and that demonstrate a centrally directed linear shadow; (3) peripheral pulmonary shadows appearing rapidly and/or gradually decreasing in size; and (4) larger infarctions showing a broadly pleural-based parenchymal density with a truncated apex, convex border, and central low attenuation.^{3,8,10)} Pulmonary infarctions may mimic resolving or stable intrapulmonary metastasis in patients with lung cancer.

Conclusion

Although it may be rare that lung cancer is associated with pulmonary infarction with evidence of pulmonary nodules, an awareness of the association between cancer and pulmonary thromboembolism is also valuable. Our case suggests that pulmonary infarction associated with lung cancer should be considered as one important cause of peripheral pulmonary nodules.

References

1. Edmondstone WM. Flitting radiographic shadows: an unusual presentation of cancer in the lungs. *Thorax* 1998; **53**: 906-8.
2. Hanbury WJ, Cureton RJ, Simon G. Pulmonary infarcts associated with bronchogenic carcinoma. *Thorax* 1954; **9**: 304-12.
3. Yoshida N, Sugita H, Nakajima Y, Nakano H, Kawabata Y. Relations between chest CT and pathologic findings in pulmonary infarction associated with lung cancer. *Nihon Kyobu Shikkan Gakkai Zasshi (Jpn J Thorac Dis)* 1995; **33**: 1064-72. (in Jpse.)
4. Scoggins WG, Smith RH, Frable WJ, O'Donohue WJ Jr. False-positive cytological diagnosis of lung carcinoma in patients with pulmonary infarcts. *Ann Thorac Surg* **24**; 1977: 474-80.
5. Marriott AE, Weisbrod G. Bronchogenic carcinoma associated with pulmonary infarction. *Radiology* 1982; **145**: 593-7.
6. Tsunoda HS, Saida Y, Doi M, et al. Pulmonary infarction associated with bronchogenic carcinoma. *Nippon Igaku Hoshasen Gakkai Zasshi (Nippon Acta Radiol)* 1989; **49**: 1112-21. (in Jpse.)
7. Dalen JE, Haffajee CI, Alpert JS 3rd, Howe JP, Ockene IS, Paraskos JA. Pulmonary embolism, pulmonary hemorrhage and pulmonary infarction. *N Engl J Med*

- 1977; **296**: 1431–5.
8. Nomori H, Horio H, Morinaga S, Suemasu K. Multiple pulmonary infarctions associated with lung cancer. *Jpn J Clin Oncol* 2000; **30**: 40–2.
 9. Whitesell PL, Peters SG. Pulmonary manifestations of extrathoracic malignant lesions. *Mayo Clin Proc* 1993; **68**: 483–91.
 10. Balakrishnan J, Meziane MA, Siegelman SS, Fishman EK. Pulmonary infarction: CT appearance with pathologic correlation. *J Comput Assist Tomogr* 1989; **13**: 941–5.