CASE REPORT

Localised right upper-lobe pulmonary oedema caused by extension of giant cell carcinoma to the mitral valve

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ABSTRACT. Giant cell carcinoma of the lung is a very rare primary malignant tumour and localised right upper-lobe pulmonary oedema is also unusual. We report a case of giant cell carcinoma, which invaded the left atrium through the left pulmonary vein and caused localised right upper-lobe pulmonary oedema.

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Giant cell carcinoma of the lung is a very rare primary malignancy associated with highly aggressive clinical behaviour and poor prognosis. To our knowledge very few CT images of patients with pulmonary giant cell carcinoma have been reported. Furthermore, localised right upper-lobe pulmonary oedema is also unusual. The case presented here exhibited clinical, radiographic and pathophysiological features of right upper-lobe pulmonary oedema. We report a case of localised right upper-lobe pulmonary oedema caused by extension of giant cell carcinoma to the mitral valve.

Case report

A 54-year-old man presented with an increasing cough and haemoptysis for 2 weeks. His past medical history included prior myocardial infarction and he underwent coronary artery bypass graft surgery 7 years ago. He had no history of pleuropulmonary disease. He was an alcoholic with a daily consumption of alcohol of more than 80 g during the past 10 years and had smoked for more than 10 pack-years (1 pack-year is an average of 1 pack of cigarettes smoked per day for a year).

A chest radiograph showed complete opacification of the left hemithorax and elevation of the left diaphragm (not shown). Contrast chest thin-section CT images showed an ill-defined huge mass with extensive necrosis in the left lower lobe and multiple mediastinal and hilar lymph node enlargement (not shown). The left main bronchus was obstructed by a tumour with resultant left lung atelectasis. Moreover, the tumour

had invaded the left atrium through the left pulmonary vein. Histopathological examination of a transbronchial biopsy showed non-small cell carcinoma (not shown).

Although chemotherapy was administered to the patient, the tumour grew rapidly. A chest radiograph, which was obtained 1 month after the initial chest radiograph, revealed airspace shadowing confined to the right upper lung field (Figure 1). Contrast-enhanced thin-section CT images showed that the masses in the left lung, the left main bronchus and the left inferior pulmonary vein had increased in size (not shown). In addition, the tumour in the left atrium had also grown and the mitral valve dilated with invasion into the left ventricle (Figure 2). The CT images showed localised consolidation with air bronchogram in the right upper lobe, which was thought to be localised pulmonary oedema caused by mitral valve regurgitation owing to tumour progression to the left ventricle (Figure 3). In addition, metastatic right pulmonary small nodules and bilateral adrenal masses were observed on CT images (not shown).

One week later the patient died of massive haemoptysis. The autopsy revealed that the left huge mass had invaded the left pulmonary vein, the left atrium and the left ventricle, and caused mitral valve regurgitation. Pathological findings showed that the tumour consisted of widespread necrosis and a solid component had formed from cells with large and bizarre nuclei and was therefore diagnosed as pulmonary giant cell carcinoma (Figure 4). The extent of consolidation of the right upper lobe corresponded to the extent of localised pulmonary oedema without infection or infiltration of the carcinoma (Figure 5). Multiple metastatic foci were found in the mediastinal and hilar lymph nodes, duodenum, jejunum, spleen, bilateral kidneys and both adrenal glands.

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Figure 1. Chest radiograph (1 month after the initial chest radiograph) shows that the huge mass has increased in size and the presence of airspace shadowing in the upper field of the right lung (arrow).

Discussion

Giant cell carcinoma is a very rare primary pulmonary malignancy. This type of tumour produces a huge mass, increases rapidly, develops distant metastasis at a high rate and shows a poor prognosis. Based on the recent World Health Organization classification (2004) giant cell carcinoma is classified as pleomorphic carcinoma. Of all lung cancers, only 0.1% to 0.4% are pleomorphic carcinomas [1]. Pulmonary giant cell carcinoma is described as a carcinoma in which anaplastic and

Figure 2. A transverse contrast thin-section CT image at the level of the mitral valve shows extension of the tumour into the left ventricle through the mitral valve from the left atrium (arrowheads). Mucoid impactions of the left lower bronchus were present (arrows).

Figure 3. Transverse thin-section CT image of right upper lobe shows localised consolidation with peripheral sparing (arrows).

pleomorphic giant cells predominate, ranging from multinucleated forms to large mononuclear forms. When polygonal cells or adenocarcinoma cells become part of the tumour it is classified as pleomorphic carcinoma, therefore, giant cell carcinoma is very rare.

To our knowledge only three CT images of patients with pulmonary giant cell carcinoma have been reported [2–4]. Kim et al [5] reported the CT findings of 30 patients with pleomorphic carcinoma. The CT features included a subpleural location and a large area of low attenuation suggestive of extensive tumour necrosis on contrast CT; these findings are similar to our case. A poorly defined margin with surrounding areas of ground-glass attenuation was noted. In this patient, metastases of multiple organs, including the intestine, were found, which is similar to a previous report [6].

Figure 4. Photomicrograph of autopsy specimen from the tumour of the left lung shows the tumour was formed from cells with large and bizarre nuclei ($\times 400$, haematoxylin and eosin staining).

Figure 5. Photomicrograph of an autopsy specimen from the right upper lobe shows that the alveolar spaces were filled with effusion ($\times 200$, haematoxylin and eosin staining).

Although uncommon, asymmetric pulmonary oedema has been reported in several different clinical situations. Ipsilateral pulmonary oedema occurs on the same side as a focal insult to one lung without evidence of oedema in the other lung. The main causes of this type of oedema include bronchial obstruction, veno-occlusive disease, unilateral aspiration, pulmonary contusions, mitral regurgitation and several others [7]. The second classification, contralateral pulmonary oedema, is oedema that occurs in a normal lung only as a result of an abnormality in the opposite lung. This occurs in people with congenital absence of a pulmonary artery, Swyer-James syndrome, pulmonary embolism, and lobectomy, among other conditions [8].

Pulmonary oedema in association with mitral valve regurgitation is usually cardiogenic pulmonary oedema. It is occasionally isolated or predominantly right upper-lobe pulmonary oedema. Schnyder et al [9] reported a prevalence of 9% for predominantly right upper-lobe pulmonary oedema in cases of severe mitral valve regurgitation. In 1982, Miyatake et al [10] showed that the direction of the regurgitant jet caused by flail mitral valve leaflets was dependent on which leaflet was involved. Roach et al [11] later reported that the regurgitant flow in a patient with a flail posterior valve leaflet was directed specifically towards the right pulmonary vein. Thus, the hydrostatic pressure increases locally in the right upper lobe. On the other hand, some reports have described an eccentric mitral regurgitation

jet caused by papillary muscle dysfunction towards the left pulmonary vein, which caused the predominantly left upper-lobe pulmonary oedema [12].

The case presented here exhibited clinical, radiographic and pathophysiological features of right upper-lobe pulmonary oedema. The giant cell carcinoma of the left lung extended into the mitral valve through the left atrium and caused mitral regurgitation and jet flow towards the right pulmonary vein.

References

1. Chang YL, Lee YC, Shih JY, Wu CT. Pulmonary pleomorphic (spindle) cell carcinoma: peculiar clinicopathologic manifestations different from ordinary non-small cell carcinoma. *Lung Cancer* 2001;34:91–7.
2. Aziz SA, Ahmad M, Shah A, Bhat GM, Lone A, Ahmad K, et al. Giant cell carcinoma of the lung. *Postgrad Med J* 1995;71:562–63.
3. Forst T, Beyer J, Cordes U, Pfützner A, Küstner E, Moll R, et al. Gynaecomastia in a patient with a hCG producing giant cell carcinoma of the lung. *Exp Clin Endocrinol Diabetes* 1995;103:28–32.
4. Shih WJ, Magoun S, Dillon M. Giant cell carcinoma of the lung exhibiting a large, well-circumscribed photon deficiency in ventilation/perfusion scintigrams. *Radiat Med* 1992;10:26–9.
5. Kim TS, Han J, Lee KS, Jeong YJ, Kwak SH, Byun HS, et al. CT findings of surgically resected pleomorphic carcinoma of the lung in 30 patients. *AJR Am J Roentgenol* 2005;185:120–25.
6. Ginsberg SS, Buzaid AC, Stern H, Carter D. Giant cell carcinoma of the lung. *Cancer* 1992;70:606–10.
7. Calenoff L, Kruglik GD, Woodruff A. Unilateral pulmonary edema. *Radiology* 1978;126:19–24.
8. Gluecker T, Capasso P, Schnyder P, Gudinchet F, Schaller MD, Revelly JP, et al. Clinical and radiologic features of pulmonary edema. *Radiographics* 1999;19:1507–31.
9. Schnyder PA, Sarraj AM, Duvoisin BE, Kapenberger L, Landry MJ. Pulmonary edema associated with mitral regurgitation: prevalence of predominant involvement of the right upper lobe. *AJR Am J Roentgenol* 1993;161:33–6.
10. Miyatake K, Mimura Y, Sakakibara H, Kinoshita N, Okamoto M, Nagata S, et al. Localisation and direction of mitral regurgitant flow in mitral orifice studied with combined use of ultrasonic pulsed Doppler technique and two dimensional echocardiography. *Br Heart J* 1982;48:449–58.
11. Roach JM, Stajduhar KC, Torrington KG. Right upper lobe pulmonary edema caused by acute mitral regurgitation. Diagnosis by transesophageal echocardiography. *Chest* 1993;103:1286–88.
12. Rice J, Roth SL, Rossoff LJ. An unusual case of left upper lobe pulmonary edema. *Chest* 1998;114:328–30.