Table 24-h urinary excretion of calcium (Ca^{++}) and uric acid prior to (pre Rx), and during therapy (post Rx) with spironolactone at 2 months and again at 12 years in a patient with primary hyperaldosteronism and renal calculi. Figures are given as mean $\pm$ SEM of four values at each time point

	Calcium (mM/day)	Uric acid (mM/day)
pre Rx	104.5 $\pm$ 3.7	5.10 $\pm$ 0.13
post Rx (2 months)	55.5 $\pm$ 2.0	3.18 $\pm$ 0.10
post Rx (12 years)	54.30 $\pm$ 2.31	3.25 $\pm$ 0.08
normal value	62.5–75.0	3.87–4.16

Summary points

- renal calculi may occur in primary hyperaldosteronism
- raised urinary excretion of calcium and uric acid may occur in primary hyperaldosteronism
- spironolactone therapy reduces urinary excretion of calcium and uric acid, and helps prevent recurrent renal calculi in primary hyperaldosteronism

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Giant cell carcinoma of the lung

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Summary

A 50-year-old non-smoking, hypertensive female, presenting with superior vena caval compression, was found to have giant cell carcinoma of the lung. She received intensive combination chemotherapy. However she died in the following 36 hours, as a consequence of refractory hypotension.

Keywords: giant cell carcinoma, superior vena caval obstruction

Giant cell carcinoma is a rare, distinctive lethal variant of large cell cancer of the lung.¹ The tumour is quite extensive at diagnosis and survival for more than one year is exceptional.²

Case report

A 50-year-old, non-smoking, hypertensive woman was admitted to our hospital on 19 October 1994, with a non-productive cough and breathlessness of one month duration. Clinical examination revealed features suggestive of superior vena caval compression, with partial collapse of the right upper lobe of lung. Her blood pressure was 150/100 mmHg (on calcium channel blockers); the rest of her cardiovascular and other systemic examination was non-contributory. Fundus oculi were normal. Her investigations showed a normal haemogram and biochemical parameters. Electrocardiogram (ECG) was normal. Chest X-ray revealed a mass shadow in the right upper and middle zone with mediastinal invasion.

Abdominal ultrasonography was normal. Computed tomography (CT) scan of the chest showed a homogenous mass of soft-tissue density in the anterior and middle mediastinum, impressing on the arch of aorta and causing displacement and occlusion of the superior vena cava. There was compression of the right upper lobe bronchus with extension into the chest wall and pleural effusion on the right side (figure). A clinical diagnosis of bronchogenic carcinoma (T4N2MO-IIIB) was made. She was put on decompressive treatment and a Trucut biopsy was done, which subsequently revealed features suggestive of giant cell carcinoma of the lung. She was put on combination chemotherapy consisting of intravenous cyclophosphamide 750 mg/m², adriamycin 50 mg/m² and cisplatin 100 mg/m² on day 1; the

Figure CT scan of the chest showing a homogenous mass of soft density in the anterior and middle mediastinum with impression on arch of aorta, displacement and occlusion of superior vena cava with pleural effusion

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treatment was to be repeated every 21 days). On the day following the chemotherapy the patient's performance status deteriorated, with rapidly progressive hypotension unresponsive to fluid challenge and inotropics. Her chest X-ray showed no new findings, however, serial ECGs showed a persistent sinus tachycardia, with elevated cardiac enzymes. Chemotherapy was abandoned and the patient died after another 6 hours, probably due to anthracycline-induced pump failure.

Discussion

Giant cell carcinoma is a highly malignant form of large cell cancer of the lung. The tumour is essentially undifferentiated, with bulky necrotic masses, having no distinct or distinguishing architectural pattern. Histologically, the main element of the tumour is a polygonal, spindle or strap giant cell with one or several nuclei and prominent nucleoli. The cytoplasm is uniformly eosinophilic or finely vacuolated, and prominent acidophilic nuclear inclusions are frequently seen. A complement of smaller undifferentiated cells is present with little desmoplastic response. The tumours grow in a characteristic non-cohesive manner. Typical and atypical mitoses are abundant. A characteristic finding is the presence of collections of polymorphonuclear leukocytes within the giant cells, apparently phagocytosing cytoplasmic contents, a phenomenon known as emperipolesis. Electron microscopic analysis of giant cell carcinoma has suggested some exocrine features. Little is known about the immunohistochemical characteristics of these

Giant cell carcinoma: features
• highly malignant form of large cell cancer of lung   • peripheral in location   • histology: polygonal, spindle, or strap giant cells with one or several nucleoli, eosinophilic cytoplasm, abundant mitoses   • emperipolesis

cancers, however, their epithelial nature is confirmed by their expression of cytokeratin and, in a few instances, by demonstration of phenotypic exocrine features with the 44-3A6 monoclonal antibody.³

The differential diagnosis of giant cell carcinoma of the lung includes pleomorphic rhabdomyosarcoma, malignant fibrous histiocytoma, and metastatic adrenocortical cancer.⁴ In view of the characteristic light and electron microscopic features and clinical behaviour, giant cell carcinoma of the lung is classified as a specific tumour type.⁵

Clinically, the tumour follows a highly malignant course that parallels that of small cell lung cancer, with development of widespread lymphangitic and haematogenous metastasis. These tumours are generally peripheral in location. The clinical course is aggressive and most tumours are inoperable at the time of diagnosis, as noticed in the present patient. The survival is less than one year, in spite of any therapy. Longer survival times have been reported, but only in less aggressive forms combined with adenocarcinoma.⁶

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