Sarcoidosis presenting as recurrent left laryngeal nerve palsy

F A El-Kassimi, M Ashour, R Vijayaraghavan

Abstract
A patient with sarcoidosis presented with hoarseness caused by mediastinal lymph nodes compressing the left recurrent laryngeal nerve. The response to corticosteroids was dramatic and complete.

Hoarseness secondary to left recurrent laryngeal nerve palsy in a patient with mediastinal lymphadenopathy is usually due to malignancy. Tuberculosis and pneumonia occasionally produce this complication, however, and sarcoidosis has been implicated in two reports.

Case report
A non-smoking woman of 45 was admitted with a three week history of progressive hoarseness and occasional bouts of cough and choking during swallowing. She was otherwise symptomless and her weight was steady. Examination showed nothing abnormal except for the hoarseness. Laryngoscopy showed paralysis of the left vocal cords with no intrinsic lesion of the larynx. The palate moved normally and the gag reflex was present bilaterally.

The full blood count was normal and her erythrocyte sedimentation rate was 10 mm in the first hour. Chest radiography (fig 1) showed bilateral interstitial infiltrate in the lower and middle zones of the lung, with widening of the mediastinum and probable hilar lymphadenopathy. Computed tomography of the lung confirmed the presence of interstitial infiltrate in the lung as well as paratracheal, carinal, and hilar lymphadenopathy (fig 2). The Mantoux test (10 units tuberculin) produced a negative response on two occasions. Results of liver function tests and the serum calcium concentration were normal. Arterial blood gases sampled at rest were normal, as were spirometric values and total lung capacity; carbon monoxide transfer factor was substantially reduced, however, at 39% predicted (transfer coefficient 40% predicted). A gallium scan showed increased uptake in hilar nodes, mediastinum, lungs, and parotid glands.

A paratracheal lymph node biopsy specimen, obtained by mediastinoscopy, showed non-casating granuloma with no acid fast bacilli or fungi, and no mycobacteria or fungi were grown in culture. A diagnosis of sarcoidosis was made and prednisolone 40 mg daily was started. Ten days later the hoarseness and nerve palsy had cleared and chest radiography showed total resolution of both interstitial and nodal lesions.

The patient has remained symptomless with a normal chest radiograph for six months after presentation.

Discussion
Hoarseness in association with mediastinal lymphadenopathy is usually due to malignant disease. As this case shows, sarcoidosis should also be considered, particularly in patients who are generally well or who have concomitant interstitial lung disease. Hoarseness in sarcoidosis may be caused by three
Constrictive pericarditis caused by primary liposarcoma

R J Lionarons, J van Baarlen, J F Hitchcock

Abstract
A 30 year old man presented with symptoms of constrictive pericarditis. Echoangiography and computed tomography showed a mass extending from the pericardium to surround the heart and penetrating the left ventricular apex. An unresectable pleomorphic liposarcoma arising from the pericardium was found at thoracotomy.

Primary liposarcoma of the pericardium has not been reported previously. We describe constrictive pericarditis caused by primary liposarcoma in a 30 year old man.

Case report
A 30 year old white man presented with a two month history of progressive dyspnoea on exertion, left sided chest pain that radiated to the left shoulder, general malaise, weight loss, anorexia, night sweats, fever, and pronounced peripheral oedema. He was transferred to us from a small regional hospital with a diagnosis of suspected right sided endocarditis. On examination the patient was ill with severe dyspnoea at rest. His blood pressure was 120/85 mm Hg with pulsus paradoxus of about 30 mm Hg. The heart rate was regular at 116 beats/min and the temperature was 37.8°C. Venous pressure was visible at the angle of the jaw and his abdomen was swollen and tender with considerable hepatomegaly and ascites. Severe pitting oedema of the legs extended to the sacrum. There were broadened precordial pulsations, an apex beat palpated outside the midclavicular line, and very faint heart sounds, with a grade 2/6 systolic murmur along the right sternal border and a pericardial friction rub near the apex.

A chest radiograph showed enlargement of the heart, with irregular margins extending to the left thoracic wall, and a pleural effusion. An electrocardiogram was of low voltage and showed non-specific T wave changes. Echoangiography showed a huge, irregular tumour mass surrounding the greater part of both ventricles and infiltrating the apex of the left ventricle. There was considerable limitation of diastolic filling. Computed tomography showed no extension outside the pericardium. An anterolateral thoracotomy was performed for diagnostic purposes and to decompress the myocardium. The heart was encircled by a large, richly vascular, mucoid, multilobular, green-yellow, glistening mass, encapsulated by the pericardium and attached to the apex of the left ventricle.

Figure 1  Transverse section of the heart at the level of both ventricles showing the tumour extending from the parietal to the visceral pericardium. At the lower right side the tumour has infiltrated the left ventricular wall.