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CASE REPORT

Tumour-like presentation of pulmonary sarcoidosis

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Tumour-like presentation of pulmonary sarcoidosis. M.J. Abramowicz, V. Ninane, M. Depierreux, P. de Francquen, J-C. Yernault.
ABSTRACT: We report a case of pulmonary sarcoidosis, which initially presented as a left apical infiltrate. The later course mimicked a pulmonary neoplasm, with left upper lobe atelectasis secondary to bronchial stenosis, resulting from both endobronchial sarcoidosis and extrinsic compression by enlarged lymph nodes. Extrinsic pressure from sarcoid nodes on the left main pulmonary artery and recurrent laryngeal nerve, also caused a reduction in pulmonary parenchymal perfusion and left vocal cord paresis.
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The usual modes of presentation of thoracic sarcoidosis are well-known and readily recognized. We report here an unusual case of thoracic sarcoidosis, the features of which mimicked those of a malignant tumour.

Case report

A 60 yr old male smoker was admitted with fever. There was a history of left pulmonary apical infiltrate, discovered at the age of 53 yrs, on a routine chest radiograph. Physical examination only revealed diminished lung sounds in the left upper field. Chest radiograph disclosed an enlarged left hilum with left upper lobe atelectasis (fig. 1, left panel). Fibreoptic bronchoscopy revealed a left vocal cord paresis and a complete stenosis of the left upper lobe bronchus. Non-caseating granulomas with giant and lymphoid cells were seen microscopically. A tuberculin skin test was negative. A computed tomographic (CT) scan of the thorax (fig. 1, right panel), and digital subtraction angiography of the pulmonary artery, disclosed multiple enlarged mediastinal lymph nodes and a left hilar mass, that compressed the left main pulmonary artery and upper lobe bronchus, with collapse of the left upper lobe and a marked reduction in left pulmonary parenchymal perfusion. Microscopic examination of lymph nodes obtained during mediastinoscopy confirmed epitheloid cell granulomatosis. An extensive search for infectious agents known to produce pulmonary granulomatosis was negative.

The diagnosis of sarcoidosis was suggested and a steroid therapy was instituted (methylprednisolone, initial dose 1 $mg \cdot kg^{-1}$ *q.d.*). Six weeks later, the patient

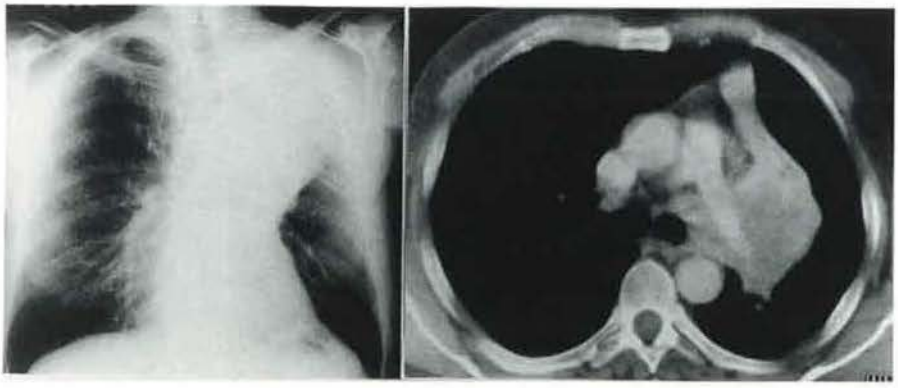

Fig. 1. – Left panel: chest radiograph revealing the left hilar mass with upper lobe collapse. Right panel: computed tomographic scan of the chest at the level of the carina showing the left hilar mass constricting the left pulmonary artery.

was looking healthy. A CT scan of the thorax showed a partial resolution of the hilar mass and of the left upper lobe collapse. The corticosteroid dose was, thus, gradually tapered 16 mg·day^{-1}. However, on subsequent evaluation three months later, high grade fever had resumed. A radiograph examination showed that the tumour had grown back to its initial dimension. Surgical resection of the left upper lobe was thus performed.

One week later, the patient died of a ruptured pulmonary artery. On anatomohistological examination, a white nodular mass was found at the apex of the left upper lobe and it showed non-caseating granulomas with fibrous tissue. The bronchostenotic area revealed narrowing due to mucosal thickening and external compression by several lymph nodes (fig. 2). These nodes also compressed the left pulmonary artery. Microscopic sarcoid process was present in bronchial mucosa as well as in lymph nodes and left lung parenchyma. No neoplasm was found and all microbiological samples were negative.

Fig. 2. – Resected left upper lobe exhibiting massive lymphadenopathy (arrowheads) with major compression of the left upper bronchus (arrow).

Discussion

We report a case of bronchogenic tumour, with mediastinal adenopathies and multiple organ compressions, where all the microscopic examinations failed to reveal a malignant disease but rather showed a granulomatous disorder.

Bronchocentric granulomatosis was unlikely because of massive involvement of hilar and mediastinal lymph nodes [1], and no microorganism was detected that could account for an infectious granulomatous disease. We thus believe that the diagnosis in the present case was sarcoidosis, which had presented several years earlier as a left pulmonary apical infiltrate [2, 3]. The later course mimicked a malignant tumour, with multiple compressions affecting the left pulmonary artery and upper bronchus and the recurrent laryngeal nerve.

Lobar atelectasis secondary to bronchial occlusion is rare in sarcoidosis and may be linked either to endobronchial sarcoid with narrowing of the lumen, to extrinsic bronchial compression by enlarged nodes [4] or, as in the present case (fig. 2), to a combination of both processes [5, 6]. Corticosteroid therapy of bronchial stenosis has yielded conflicting results: whereas, improvement is reported by some authors [4], others emphasize its poor prognosis.

Unlike bronchial occlusion, the left pulmonary artery narrowing seems to be due solely to extrensic compression by enlarged nodes, since no sarcoid process was found in the wall of this vessel. Although pulmonary artery occlusion in sarcoidosis has occasionally been reported in the literature [4], narrowing of a main pulmonary artery has, to our knowledge, never previously been described. The same compression probably accounts for the left recurrent laryngeal nerve palsy [7–9], as radiographic investigations clearly showed involvement of the aorto-pulmonary window by lymphadenopathies.

In conclusion, this observation further emphasizes that the clinical presentation and course of thoracic sarcoidosis can be misleading. This granulomatous disorder must be considered in patients presenting with clinical and radiological features that strongly suggest bronchial carcinoma, when no pathological evidence of neoplasm can be found.

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