considered as a possible infectious etiology in AIDS patients who develop ARDS. Additional information needs to be collected to determine the overall impact of intensive care unit interventions, maintenance amphotericin B therapy, and AZT on the course of disseminated cryptococcosis in AIDS patients.

REFERENCES

Castleman's Disease*

An Uncommon Computed Tomographic Feature

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A case of localized intrathoracic Castleman tumor demonstrated, on CT scan, calcifications in a circumferential distribution. The prevalence of calcifications in Castleman's disease and the differential diagnosis of the above unusual CT findings are discussed.

Castleman's disease is characterized by localized lymph node enlargement, most commonly located in the

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Figure 1. Frontal chest x-ray film demonstrating right mediastinal mass lesion.

Figure 2. CT scan at the level of the main pulmonary arteries shows right mediastinal and hilar mass containing calcified densities in a peripheral distribution (arrows).
A chest roentgenogram demonstrated a 5 × 7 cm right mediastinal mass (Fig 1). A CT of the lesion displayed two contiguous round masses in the central mediastinum, anterior and lateral to the mainstem bronchus and not encroaching upon it (Fig 2). The larger of the two masses, which underwent postcontrast enhancement, contained calcific densities in a peripheral distribution. Digital subtraction angiography (DSA), performed to exclude any vascular structure involvement, demonstrated normal pulmonary arteries.

The patient underwent thoracotomy and a red-brownish polycyclic mass in the right hilum, right of the SVC and anterior to the pulmonary vessels was noted and excised. The mass weighed 44 g, measured 4 × 5 × 3 cm, and was covered by a thin capsule. Calcifications were noted on bisecting the mass.

The histopathologic features of the lesion were typical of Castleman’s disease of the hyaline-vascular type with follicular hyperplasia and proliferation of hyalinated capillaries.

Recovery of the patient was uneventful except for mild postoperative fever that persisted for a few days. The patient has been symptom-free during the six months since the operation.

**DISCUSSION**

Around 70 percent of mass lesions proven histologically to be cases of Castleman’s disease are within the chest. Such a location lends itself readily to the simplest of roentgenographic techniques, namely: the chest radiograph.

Despite this fact, there is a paucity of reports on the roentgenographic features of intrathoracic Castleman’s disease.

The first series of patients with the localized form of Castleman’s disease was reported by Abell (who included some relevant chest radiographs). Keller et al. presented their large series comprised of 81 patients and remarked that the roentgenographic appearance of the intrathoracic lesions was that of a rounded mediastinal or hilar mass. Usually, a single rounded well-outlined mass is identified with histologic examination showing it to be of the hyaline-vascular type. The mass can be up to 16 cm in size with an average of about 6 cm and demonstrates no necrotic or cystic changes.  

In the case of the plasma cell type, multiple discrete lymph nodes in an aggregate form make up the roentgenographically observed mass. These reports were followed by a few sporadic reports, some adding the angiographic findings, while Phelan and Fiore et al. presented CT demonstration of the lesion. The mass lesions are extremely hypervascular on angiography, stain homogeneously in the capillary phase, and show postcontrast enhancement on CT.

Calcifications of the lesion noted on preoperative x-ray films were reported in a small number of cases (three of 81 in the series by Keller et al.). When calcifications were visible, they were described as being central and lumpy, or loculated, or irregular. In our case, a unique pattern of calcification was demonstrated on CT scan. The calcifications were of a circumferential distribution with one satellite calcific density (Fig 2).

The major pathologic entities to be considered in the differential diagnosis of a central mediastinal mass with calcifications are as follow: aneurysm of the aorta or other major artery; lymph node enlargement (due to TB, sarcoidosis, or siliconosis and other pneumoconioses); old hematomata; mediastinal abscess; bronchogenic cyst; and ectopic thyroid tumor. The postcontrast enhancement of the lesion indicating its vascular nature is noteworthy, since it may aid in limiting the extent of the differential diagnosis, as reported by Onik and Goodman.

To the best of our knowledge, a pattern of peripheral calcifications within a Castleman tumor has not yet been reported, and its importance lies in the fact that it could be mistaken for a calcified blood vessel, a bronchogenic cyst, or an old hematoma (due to past trauma).

The definitive diagnosis, of angiofollicular lymph node hyperplasia, was made on histopathologic examination of the excised mass.

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**Pulmonary Veno-occlusive Disease in a Patient with Unilateral Absence of Right Pulmonary Artery**

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A 25-year-old patient had unilateral absence of the right pulmonary artery (UARPA) and severe left pulmonary artery hypertension. After death from congestive right heart failure, autopsy revealed histologic signs of pulmonary veno-occlusive disease (PVOD) and pulmonary hypertension (PH). An accessory arterial vessel that was thrombocytically occluded was found connecting the ascending aorta and the right pulmonary hilum. There was also histologic evidence of arterial thrombi within the right lung arterial vascular bed. The PH in UARPA usually occurs very early during the course of disease. From histologic findings and medical history, it is likely that in this case, late-onset elevation of pulmonary pressures was triggered by the occurrence of PVOD. This is the first case of UARPA and PVOD—a congenital unilateral arterial malformation in the presence of bilateral involvement in a possibly acquired venous obliterative disease.

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