

Angiofollicular lymphoid hyperplasia in a pulmonary fissure

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Since the original description of angiofollicular lymphoid hyperplasia or giant lymph node hyperplasia several reports have indicated the range of clinical presentation and of radiological and pathological appearances.¹

In most cases mediastinal nodes have been affected, although extrathoracic and multicentric² sites have been recognised. In the present case an unusual intrapulmonary mass was found to be the hyaline vascular variety of angiofollicular lymphoid hyperplasia occurring in a young caucasian woman.

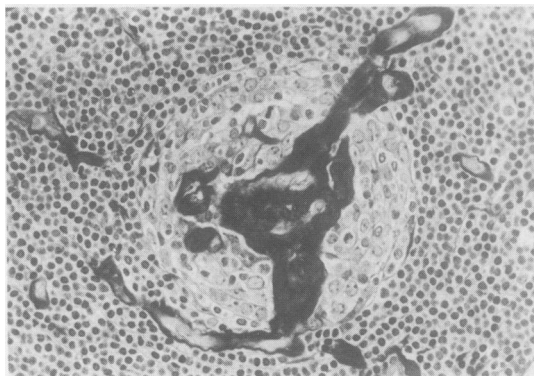
Case history

A female student aged 19 presented in the summer of 1983 with abdominal pain diagnosed as acute pancreatitis. A chest radiograph at that time showed a "coin" lesion in the right lung. The pain subsided and she was discharged. The patient remained symptom free and fit and was readmitted for investigation in March 1984. Physical examination showed nothing abnormal. Further radiographs showed no appreciable change in the size or character of the lesion and computed tomography showed no other lesion. Routine investigations, including lung function tests, yielded results within normal limits. Examination of material obtained by a needle aspiration biopsy suggested a differential diagnosis of pulmonary pseudolymphoma or a well differentiated non-Hodgkin's lymphoma. In view of this and the localised nature of the lesion a right thoracotomy was performed. Palpable enlarged lymph nodes were found in the subcarinal area and an ovoid mass was identified in the fissure between the middle and lower lobes. The nodes and the mass were subsequently resected. The postoperative recovery was uneventful and when she was seen seven weeks later the patient was found to be fit and healthy.

Pathological findings

The tan coloured mass measured $2.5 \times 4 \times 2$ cm and on being sectioned contained small nodules up to 0.1 cm in diameter. The subcarinal lymph nodes consisted of two lobulated masses of yellow tissue, which were of similar appearance.

Microscopy showed that the mass was partially encapsulated with a residue of a subcapsular sinus. The capsule contained many thin walled, dilated vascular spaces. Focal anthracotic pigment was present in subcapsular and interfollicular areas. The typical reactive type follicles varied in size from 218 to 872 μm (mean 412, SD 144 μm). Hyalin-



A typical follicle shows central hyalinised vessels containing collagen surrounded by a concentric cuff of lymphocytes. (Masson trichrome, $\times 285$.)

ised vessels were surrounded by epithelioid cells and a peripheral cuff of lymphocytes giving the appearance of an onion skin (figure) was often present at the centre of the follicles. In the interfollicular areas a mixture of lymphocytes, plasma cells, and immunoblasts was present.

Immunocytochemical analysis to show the major immunoglobulin classes and light chains showed patchy polyclonal staining within the follicles and in the interfollicular areas. A frozen section of the material was stained for pan T, T helper, and T suppressor cells, monoclonal OKT3, OKT4 and OKT8 respectively being used.³ The distribution of T cells was similar to that in a reactive tonsil.

Discussion

Angiofollicular lymphoid hyperplasia was first defined by Castleman¹ as an asymptomatic massive hyperplasia of the mediastinal lymph nodes. In our case the mass mimicked a thymoma both radiologically and on gross pathological examination.

Most patients have been in the age range 10-60 years, with a maximum incidence in the first and second decades, there being no definite sex predilection in most reports. We are not aware of any association of this lesion with acute pancreatitis, which we consider to be incidental in this case. A multicentric variety of the condition has been identified more recently, mainly affecting elderly patients.² Of over 340 cases reported up to 1983, this case represents only the fifth intrapulmonary lesion.⁴ Before the recognition of the clinical entity, these lesions may have been regarded as intrapulmonary thymoma.³

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On the basis of the histological variation between the two major forms of angiofollicular lymphoid hyperplasia Keller *et al*¹ characterised two types, hyaline vascular (91%) and plasma cell (9%). A transitional form in which both the hyaline vascular and plasma cell types are represented within the same lesion has been increasingly recognised and at present represents about 35% of the cases reported.⁴

The histogenesis of the disease is obscure. Various aetiologies have been suggested and these include an unusual reactive lymphoid response,^{1,6,7} a hamartomatous process,⁸ and true lymphoid neoplasia.⁹ Most authors support the reactive hypothesis. The two histological variants are thought to be different stages of the same reactive process. We are inclined to support this, because of the finding of B lymphocyte polyclonality and of a distribution of both T helper and T suppressor cells that is similar to that in a reactive tonsil.

We believe this is the first report of the use of T subset markers in this condition. Anthracotic pigment, not infrequently present in subpleural connective tissue, and bronchial associated lymphoid tissue were found in our case. We interpret this finding as suggesting that the lymphoid hyperplasia was subsequent to the pigment deposition.

The suggestion that the disease represents a true benign lymphoid neoplasm is difficult to accept in view of the demonstrable polyclonality of the inflammatory infiltrate, although there have been recognised cases of non-Hodgkin's lymphoma¹⁰ developing within lesions. We thus believe it is an inappropriate immune response to some unidentified agent that was probably inhaled.

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