Letters to the Editor

Huge localised fibrous tumour of the retroperitoneum

Sir,

Localised fibrous tumours of serous membrane, which usually arise in the pleura, are rare neoplasms.¹ The tumours have been termed benign local fibromas, localised fibrous mesotheliomas, and submesothelial fibromas, because there is still controversy as to their cellular origin. Occasionally, cases have been reported in other sites such as the lung, the mediastinum and paranasal sinuses,² which led to some confusion and difficulty in diagnosis. We present a rare case of localised fibrous tumour of the retroperitoneum, which was probably derived from submesothelial mesenchymal cells.

An 81-year-old man was admitted to our hospital because of abdominal fullness. On physical examination, he appeared healthy and no abdominal mass was palpable. Laboratory investigations revealed no abnormalities and serum levels of tumour markers were within normal ranges. Computed tomographic scan of the abdomen, however, showed a giant contrast-enhanced mass behind the urinary bladder, and urinary cystoscopy demonstrated an extraluminal compression in the posterior wall of the bladder. Exploratory operation revealed a well-circumscribed huge tumour in the retroperitoneum, with an intact layer of mesothelium overlying the tumour. Histologically, the tumour was composed of uniform spindle cells separated by thick bands of collagen fibres with some pleomorphism and mitosis (figure).

Figure Histology of the tumour (hematoxylin-eosin, $\times 50$).

The patient was diagnosed as suffering from localised, benign, fibrous tumour of the retroperitoneum. Immunohistochemical analysis of the tumour showed positive staining for vimentin but not for keratin, suggesting that it probably originated from submesothelial mesenchymal cells.^{2,3}

In conclusion, we should always remember localised fibrous tumours of serous membranes when we consider the differential diagnosis of asymptomatic huge tumours, even if the tumour arises in unusual sites, such as the retroperitoneum.

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Pituitary mass and inflammatory pseudotumours of lung and retroperitoneum

Sir,

Alvarez *et al*, in this journal, described the unusual association of two inflammatory pseudotumours: retroperitoneal fibrosis and lymphocytic hypophysitis.¹ Both lesions had a histological appearance of lymphocytic inflammation with densely textured fibrosis, and, presumably, a similar autoimmune pathogenesis. We report the case of a white man who successively exhibited an inflammatory pseudotumour of the lung, idiopathic retroperitoneal fibrosis, and a pituitary mass.

At age 47 years, a routine chest X-ray examination revealed a 4 $\times$ 6 cm round lesion in the right lung. Fine needle aspiration cytology yielded a few inflammatory cells. Subsequently, the pulmonary lump was resected. The histologic features were consistent with an inflammatory pseudotumour composed of a predominantly lymphocytic infiltrate, myofibroblasts and a densely textured connective tissue.^{2,3} Two years later, the patient complained of flank pain caused by encasement of the left ureter by a retroperitoneal mass extending from the renal pelvis down to the aortic bifurcation.

The mass was resected *en bloc* together with the left ureter and kidney. The histologic appearance of the retroperitoneal specimen was similar to the pulmonary mass excised two years earlier. Flow cytometric DNA analysis exhibited a DNA diploid pattern indicating normal DNA content.⁴ The post-operative course was complicated by headache, visual disturbance, and hyponatraemia, leading to the diagnosis of a pituitary tumour measuring 32 mm on computed tomography. Pituitary function tests were all within normal values: free thyroxine was 1.1 ng/dl, free testosterone 12.5 pg/ml, prolactin 18 ng/ml, basal and adrenocorticotropin-stimulated cortisol levels were 6.6 and 20.1 μ g/dl, respectively. Results of blood cell count, kidney and liver function tests, antinuclear, anticentromere, antitopoisomerase antibodies, and complement fractions were within normal range. Nailfold capillaroscopy showed normal capillary loops.

In view of the patient's apparent susceptibility for the development of inflammatory masses, we assumed that the pituitary lesion could be of inflammatory nature. At surgery, a non-functioning pituitary adenoma was found and partially resected. No signs of inflammation were noted in the resected specimen. Pituitary irradiation with 4500 rad was then given. Four years later there is no evidence of residual tumour on repeated magnetic resonance imaging. The patient went into complete pituitary insufficiency and receives replacement therapy with thyroxine, cortisone and testosterone.

This patient history teaches two lessons: first, in a patient with an inflammatory pseudotumour a subsequent mass may be another pseudotumour, with similar histologic features. Second, tissue sampling from a new lesion is mandatory, even if inflammatory pseudotumours have been previously demonstrated in more than one site.

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