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Spontaneous regression of a pleural thickening with the histological appearance of an inflammatory pseudotumour

Henning Rasmussen[1], Niels Graudal[1], Thomas Horn[3], Dorthe Francis[2], and Kaj Viskum[1]
Departements of [1]Internal Medicine P and of [2]Pathology, Bispebjerg Hospital, and [3]Department of Pathology, Hvidovre Hospital, University of Copenhagen, Denmark

Summary. The inflammatory nature of a tumour-like lesion not formerly observed in the parietal pleura was confirmed histologically using immunohistochemical analysis and clinically by spontaneous regression. A study of the literature revealed that the histological picture of the lesion was consistent with that of the rarely described inflammatory pseudotumour.

Key words: Granulomatous inflammation – Inflammatory pseudotumour – Immunohistochemistry – Light microscopy – Pleura – Retroperitoneal fibrosis

Introduction

The term "inflammatory pseudotumour" (IPT) was introduced by Titus et al. in 1962 and is generally accepted as the designation of a pathological process which simulates a tumour, but which histologically is of an inflammatory nature, although often showing neoplasia-like changes such as cellular pleomorphism and mitoses. About 200 cases of IPT have been described in the literature. The majority have been confined to the lungs (Berardi et al. 1983) and the liver (Anthony and Telesinghe 1986). Three cases have been found in the visceral pleura (Brown and Johnson 1951). The present report presents the first described IPT of the parietal pleura which simulated a mesothelioma radiographically.

Case history

A 75-year-old formerly healthy woman was admitted to hospital in may 1985 because of right sided pleural effusion. Cytological specimens from the yellowish and serous pleural liquid showed a mixed population of normal appearing lymphocytes, granulocytes, eosinophils and mesothelial cells. After pleurocenthesis a pleural thickening simulating a mesothelioma was apparent on the x-ray and a tru-cut biopsy was performed (see below). Operation was considered but a conservative management was chosen. The lesion regressed spontaneously, and after four months only minimal thickening was left.

Investigation on admission showed a high erythrocyte sedimentation rate (120 mm/h (0–20)), raised acute phase reactants and low hemoglobin (5.4 mmol/1 (7–10)). After resolution of the pleural thickening these data were unchanged. In the search of a malignant disease a tumour in the left kidney was found by urography and computerized tomography. The kidney was removed and histological examination showed a well differentiated clear cell adenocarcinoma. Postoperatively the biochemical variables were normal.

In September 1988, three years after the disappearance of the pleural lesion, an x-ray of the lungs was unchanged confirming that the primary lesion had vanished.

Offprint requests to: N. Graudal, Wiedeweltsgade 56, 2100 Copenhagen, Denmark

Results

Light microscopical examination of the pleural lesion showed a tumour-like lesion composed of large cells with bulky slightly indented nuclei containing prominent nucleoli and with an abundant eosinophilic cytoplasm (Fig. 1). Scattered mitoses including few abnormals strengthened the impression of a neoplastic lesion. However, few lymphocytes, plasma cells and eosinophils and the background of fibroblasts and thin walled vessels in a coarse reticulin framework gave the appearance of an inflammatory process.

Immunohistochemical analysis by the indirect immunoperoxidase technique on deparaffinized 5 μm sections was performed using polyclonal antibodies directed towards EMA (epithelial membrane antigen), keratins, alfa-1-antitrypsin, chymotrypsin, lysozyme, fibronectin and factor VIII [DAKO (Denmark)].

Staining for keratins and EMA were negative.

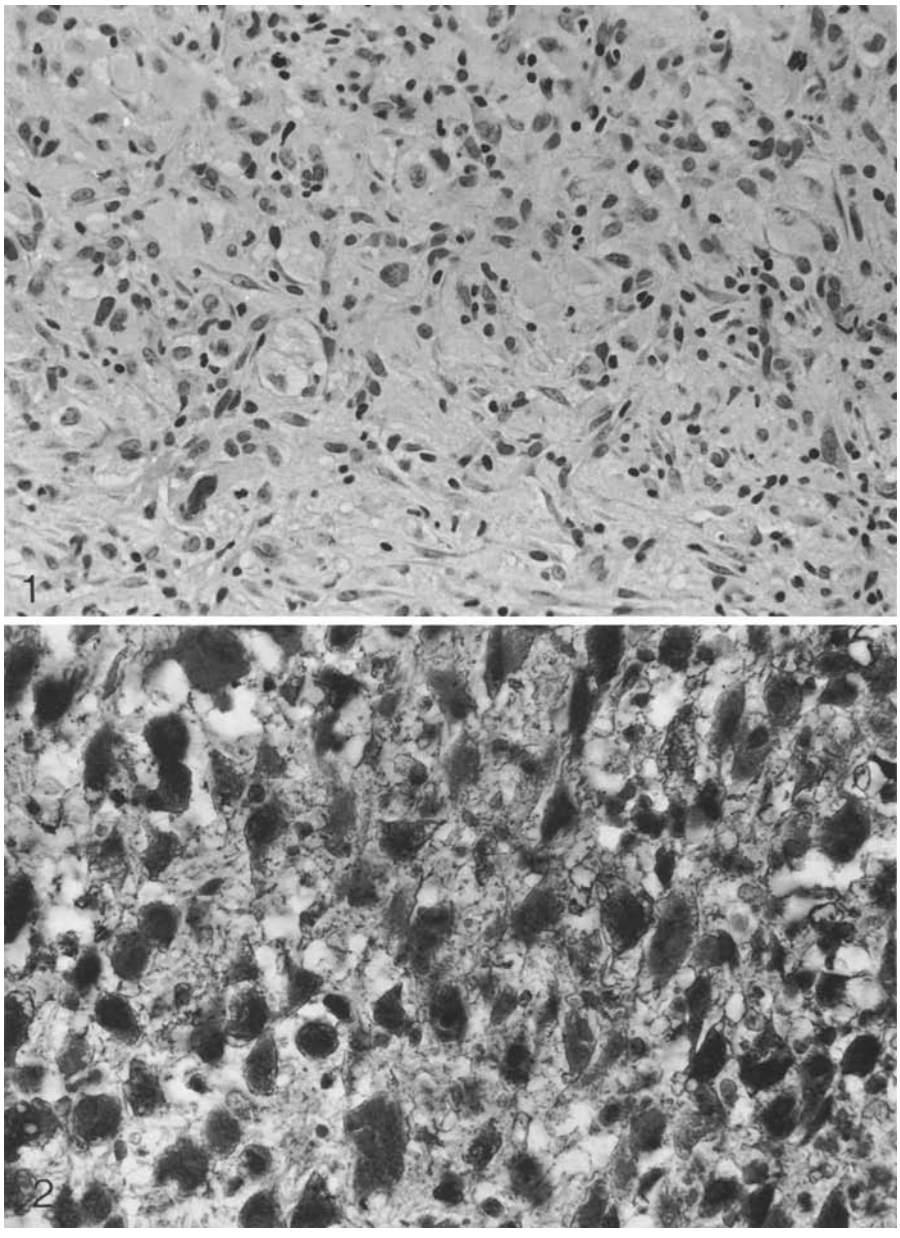

Fig. 1. Light microscopy, showing the mixed cellular composition with many large cells with bulky nuclei and prominent nucleoli. (H & E section, original magnification ×250)

Fig. 2. The large cells in the IPT were positive for chymotrypsin in the immunoperoxidase stain, verifying that the cells were histiocytes. (Original magnification ×400)

The large cells were strongly positive for chymotrypsin (Fig. 2), alpha-1-antitrypsin and lysozyme indicating that these cells were histiocytes. Abundant amounts of fibronectin appearing as a delicate fibrillar material were seen throughout the lesion and the significant vascularity was illustrated in the factor VIII stain.

Discussion

Clinically and radiographically the present case simulated a mesothelioma. Light-microscopically, it was not unequivocally inflammatory and the suspicion of a neoplasm was raised; operation was therefore considered. Immunohistochemical analyses and the spontaneous course finally revealed the inflammatory nature of the process. The possibility that it could be a metastasis from the kidney tumour with an inflammatory reaction seems to be unlikely since there were no signs of a metastasis after three years. This case shows the general characteristic features of an IPT as described in the literature. Pathognomonically IPT demonstrates a mixed inflammatory and mesenchymal infiltrate consisting of fibroblasts, plasma cells, histiocytes, foamy macrophages, mast cells and eosinophils (Berardi

et al. 1981). IPT has been published under various names according to the dominating cell type in the lesion such as plasma cell granuloma, plasma cell tumour, plasmacytoma (plasma cell preponderance), xanthoma, xanthogranuloma, xanthomatous tumour (foamy macrophage preponderance), histiocytoma, fibrous histiocytoma (histiocyte preponderance), mast cell granuloma and mast cell tumour (mast cell preponderance).

Less specific names have also been given to these tumours such as pseudotumour, postinflammatory tumour and solitary granuloma. The varying terminology reflects the varying histological picture and it is questionable if all these lesions are identical. However they are probably all a part of an immunological reaction and some of them may be different stages of development of the same lesion. Serial biopsies from the same lesion might elucidate the evolution of the lesion but as most IPTs are not diagnosed until they are removed in toto such biopsies have not been performed. IPTs that do not have a preponderance of one cell type cannot (histologically) be distinguished from chronic granulomatous inflammation. Normally, however, chronic granulomatous lesions do not simulate tumours and their causes are often known, in contrast to the IPT in which the pathogenesis remains obscure. A further differential diagnosis is fibrosis such as that seen in retroperitoneal fibrosis (RF) (Ormond syndrome). This has been described in the pleura (Partington 1961) and it has been found to coexist with retroperitoneal malignant disease and carcinoid tumours (Lepor and Walsh 1979). However, in a larger series of retroperitoneal fibrosis from one center the prevalence of cancer does not seem to be higher than in the background population (Baker et al. 1988) indicating that as a rule RF is not caused by malignant disease. In most cases of RF the disease is idiopathic, as in IPT (Lepor and Walsh 1979, Baker et al. 1988, Berardi et al. 1983). The inflammatory cell types of IPT are also found in active cases of RF (Mitchinson 1970) and as in chronic granulomatous inflammation, some of the described cases of IPT may be identical with RF. One complete and two partial spontaneous resolutions of intrapulmonary IPT have been described (Mandelbaum et al. 1981) and recurrence after operation is extremely rare (Berardi et al. 1983). Thus the prognosis is excellent, justifying a conservative attitude where the histological diagnosis is known. The coexistence of an IPT and a neoplasm has not been described before, but as the IPT in our patient disappeared spontaneously before the renal cancer was discovered and removed, their coexistence was most probably coincidental.

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