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Relapsing polychondritis presenting with stridor from bilateral vocal cord palsy

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Abstract

A case of bilateral vocal cord palsy caused by relapsing polychondritis is presented. The diagnosis was not suspected preoperatively and was made solely on histopathology. A case is made for histological examination of cartilage removed during routine tracheostomy.

Introduction

Relapsing polychondritis is a rare condition of unknown aetiology but thought to be an autoimmune disease linked with collagen vascular group of diseases (van den Broek, 1987).

Case report

A 52-year-old man was referred to the ENT Department at Seacroft Hospital Leeds, with increasing stridor. He was a known asthmatic and had been treated for what appeared to be an exacerbation of his asthma. However he soon developed inspiratory stridor which prompted this referral. On indirect laryngoscopy, there was swelling of the aryepiglottic folds and bilateral vocal cord paralysis with a small slit opening between the cords. The nose and ears were normal. An urgent tracheostomy was done. The window was fashioned at the third ring of what looked to be normal trachea. In line with our practice the removed cartilage was sent for histological examination. The patient made an uneventful recovery from this procedure. It was not possible to do a direct laryngoscopy at this time.

Histology (Figs. 1 & 2) showed inflammation of the tracheal wall with the major infiltrate centred on the tracheal cartilage. The entire perichondrium was clothed in granulation tissue rich in plasma cells and histiocytes, with erosion into and degeneration of the cartilage itself. The occasional histiocytic granuloma was also present. These appearances were consistent with the diagnosis of relapsing polychondritis. His dose of prednisolone was increased from 10 mg to 60 mg per day and this was continued for a month.

Further investigations including an antibody screen, Rh factor screening test, electrophoresis, liver function tests and plasma viscosity save for a slightly decreased IgG level, were normal.

Discussion

Relapsing polychondritis presents with recurrent inflammation of the cartilages of the head and neck particularly of the auricle, nose, larynx and trachea. Twenty-five per cent of patients have a preceding or coexistent rheumatic or autoimmune disease (Valenzuela *et al.*, 1980). Laryngeal and tracheal lesions produce swelling of the mucosa especially around the epiglottis and the aryepiglottic folds. Loss of cartilage may lead to segmental narrowing through collapse and fibrosis in the later stages or the illness may suddenly become fulminant and severe with bronchitis and tracheal collapse producing death by airway obstruction (Dolan *et al.*, 1966).

Antibodies to cartilage and to type II collagen have been demonstrated (Foidart *et al.*, 1978) and the observation that antibodies to the latter occur mainly in patients with active disease suggests an important role for antibody in this disease. The diagnosis is confirmed on histology. Corticosteroids are the mainstay of treatment and a maintenance dose is usually required.

In our patient relapsing polychondritis was not suspected and the diagnosis was made entirely on histology. There is therefore a case for submitting cartilage removed during tracheostomy for histological examination.

The use of immunosuppressive agents in the treatment of relapsing polychondritis has been reported (Ruhlen *et al.*, 1981), however in the absence of a definite aetiology such treatment remains controversial.

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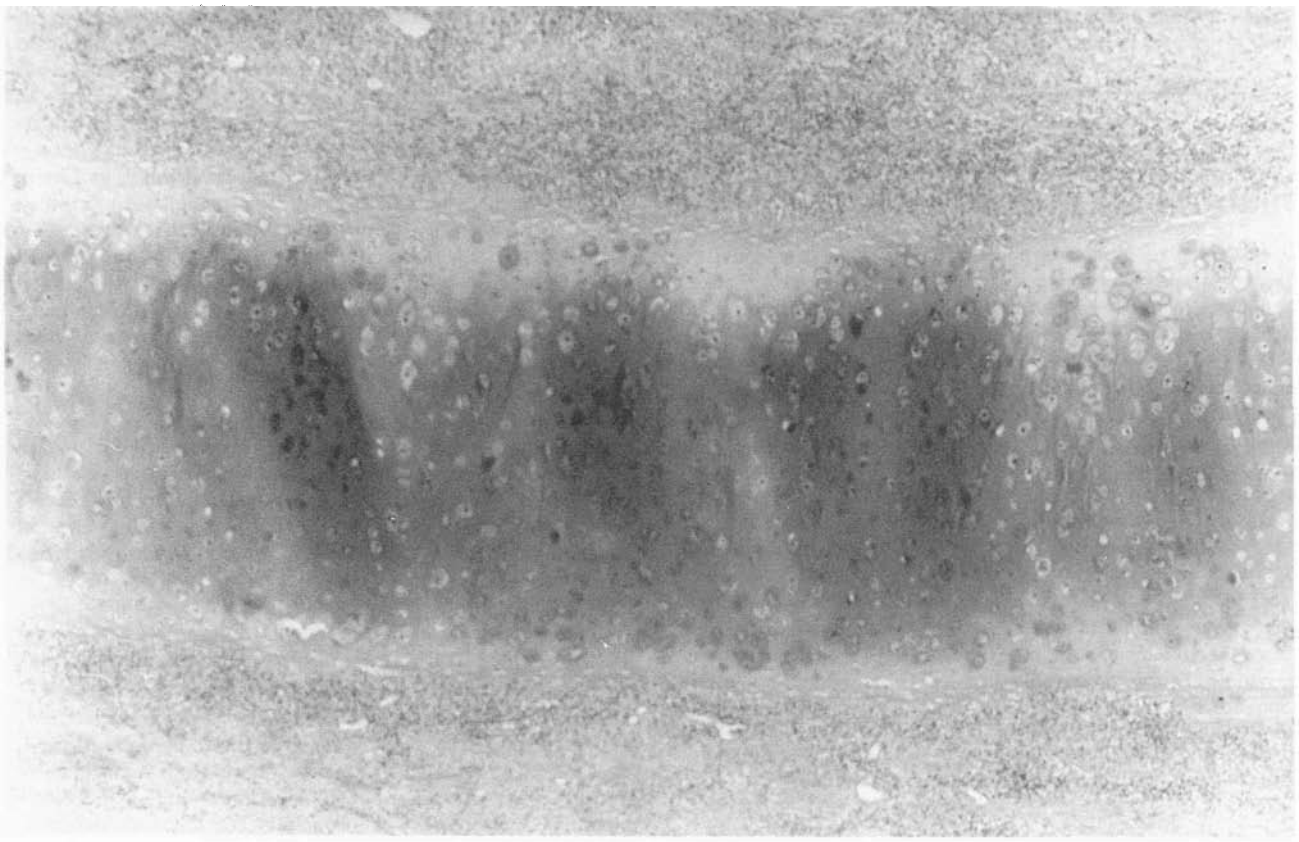

FIG. 1
Low power photomicrograph showing inflammatory infiltrate in tracheal cartilage. (H&E ×60)

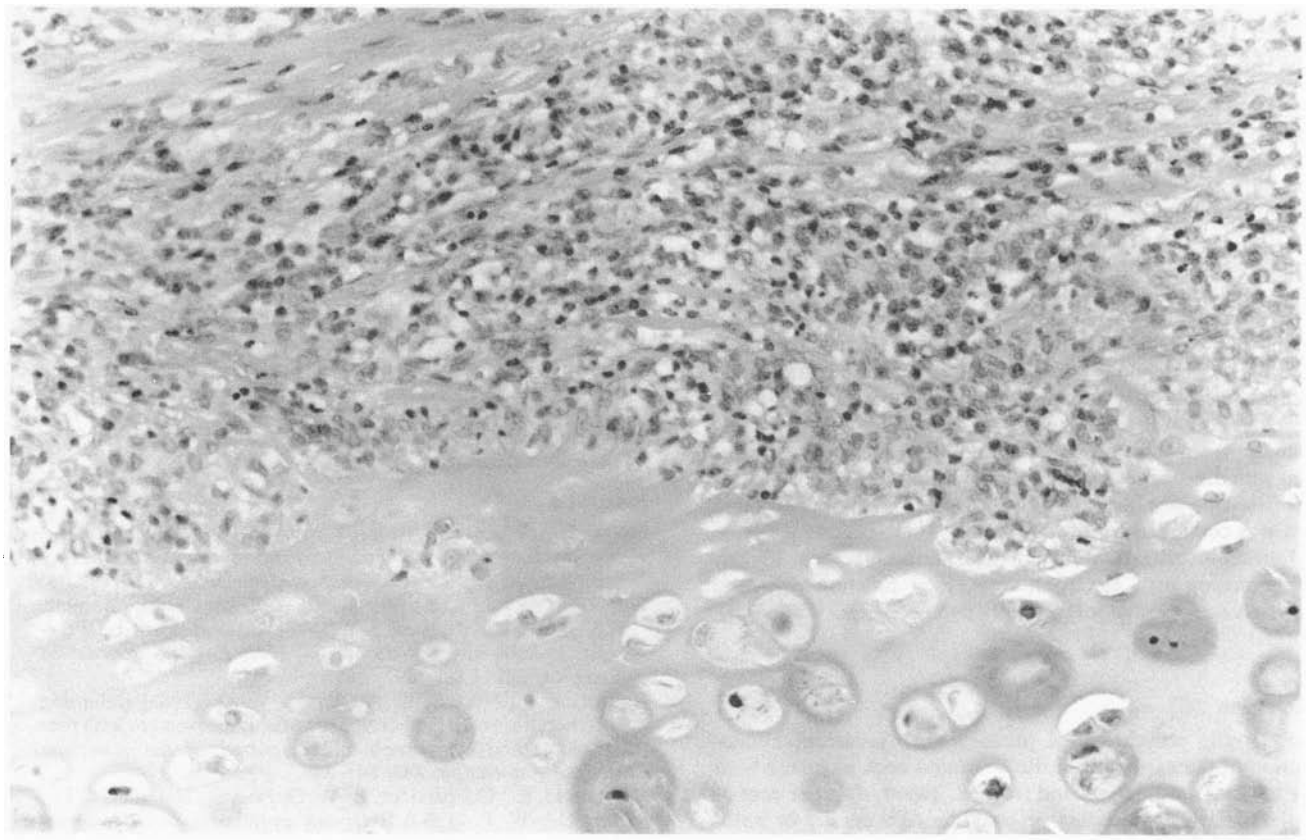

FIG. 2
High power photomicrograph showing plasma cells and histocytes with erosion and degeneration of the cartilage (H&E ×120)

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