# Auricular chondritis in a cat

**A four-year-old male neutered domestic shorthaired cat developed bilateral thickening of the pinnae, with slight curling, intense erythema and pain. No ear canal disease was present. The cat was negative for feline immunodeficiency virus, feline leukaemia virus and feline coronavirus. Biopsy of the ear lesion revealed auricular chondritis. In humans, histologically similar lesions may involve the pinnae, nose, trachea, joints, eyes and heart, and the disease is termed relapsing polychondritis. The cat reported had a history of corneal damage, resulting in corneal vascularisation and opacity, eyelid distortion, necessitating an entropion operation, and radiological evidence of mild cardiac enlargement. The ear disease responded rapidly to treatment with prednisolone and, apart from slight thickening and curling of the pinnae, the cat remained normal and pain-free. After two years, the prednisolone was withdrawn, and there was no recurrence of the condition in a follow-up period of 14 months.**

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## INTRODUCTION

Relapsing polychondritis is very rare in the cat, and is diagnosed by characteristic pinnal lesions (Scott 1987, Bunge and others 1992, Gauguere and others 1992), involving thickening, distortion, pain and intense erythema. To the authors' knowledge, only three cases have been reported in the literature. The present report describes a cat with a lesion histologically similar to the previously reported cases. Ocular disease was also present, and mild cardiomegaly was found on routine radiography. The latter signs were also described in the case by Bunge and others (1992), but not in the other two cases.

## CASE HISTORY

A four-year-old male neutered domestic shorthaired cat was presented with thickening and intense erythema of the inner surfaces of both pinnae; the ear flap edges were thickened, slightly curled, distorted and painful (Fig 1). The condition had developed on both pinnae simultaneously, and the cat twitched and flicked the ear flaps frequently. The outer (haired) surfaces were unaffected, and no external ear canal disease was evident.

The cat had exhibited an altered demeanour for about three months before presentation, manifested as hiding away, cowering, resentment of handling by the owner, and aggression towards the other cat in the household. Increased skin scaling was evident on the lumbodorsal area, and was non-pruritic, with no parasites found

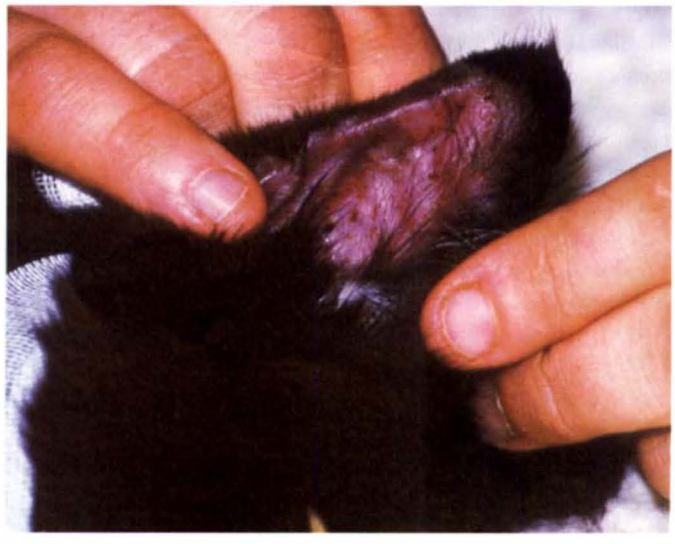

**FIG 1. Thickening, distortion and intense erythema of the right pinna (inner surface only) in the cat**

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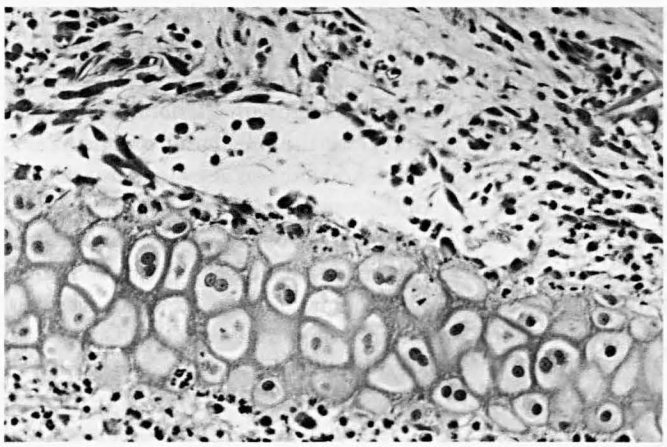

**FIG 2. Photomicrograph of pinnal biopsy showing inflammation and fibroplasia around the cartilage. Haematoxylin and eosin × 428**

**FIG 3. Photomicrograph of pinnal biopsy showing linear loss of metachromasia (arrows) from the cartilage matrix. Intense metachromasia is present throughout most of the cartilage. Toluidine blue × 68**

on coat brushings and scrapings. This was presumed to be due to reduced self-grooming. No other skin disease was found, and there was no previous history of skin or ear disease.

Nine months previously, the cat had been seen by another veterinary surgeon for corneal vascularisation of the right eye. The condition was fluorescein-negative, and was diagnosed as an old wound; probably a resolving corneal ulcer. One month later, the cat had conjunctivitis in both eyes, in addition to blepharospasm and slight entropion of the lower lid of the right eye. Steroid/antibiotic drops were administered for these conditions. Two months later, the entropion had worsened, and a surgical correction was performed. Both eyes continued to show intermittent conjunctivitis, at times fairly severe, for several months. Corneal opacity, extensive vascularisation and a weakly positive uptake of fluorescein stain were noted during this time, but no further investigations were performed.

At the time of presentation for the ear problem, the ocular lesions had largely resolved, leaving a slight corneal opacity in the right eye. Both eyes were now fluorescein-negative, and a Schirmer tear test showed a slight reduction in tear production in the right eye (13 mm; normal range 15 to 25 mm), with normal tear production in the left eye (18 mm).

The cat was anaesthetised to obtain biopsy samples; the left pinna was biopsied, with a V-section taken from the edge, and a circular punch biopsy from the centre (including cartilage). Blood samples were taken for haematological and serum biochemical analyses. Results were unremarkable, except for hypoalbuminaemia (21 g/litre; normal range 26 to 40 g/litre) of undetermined cause. The blood sample was negative for feline leukaemia virus (FeLV) (ELISA), feline immunodeficiency virus (FIV) (ELISA) and feline coronavirus (immunofluorescence test). An antinuclear antibody test was also negative. A direct Coombs' test was negative at 30°C, but positive at a dilution of 1:2 at 4°C.

Thoracic radiographs showed mild to moderate globular cardiomegaly on both lateral and dorsoventral views. The vertebral heart score was approximately 8·5 (normal range 6·9 to 8·1). The heart rate was 128 beats/minute and there was no radiological or clinical evidence of congestive heart failure. It was felt that the heart changes were suggestive of cardiomyopathy, either dilated, hypertrophic or intermediate. However, since no echocardiography or ECG was performed, a diagnosis of cardiomyopathy could not be confirmed. The cat was euthyroid at this time (total thyroxine 34 nmol/litre; normal range 19 to 65 nmol/litre).

Biopsy of the pinna showed a curling distortion of the tip of the auricular cartilage, with extensive intense fibroplasia, capillary proliferation and infiltration by neutrophilic leucocytes (Fig 2). Inflammatory cells extended into eroded superficial cartilage, where there was extensive linear loss of matrix metachromasia (Fig 3). There was intense mastocytosis of auricular dermis, but no histological evidence of its cause. Focal areas of dermal mixed lymphocyte, plasma cell and mast cell accumulation were noted.

The cat was given 10 mg of oral prednisolone daily (3 mg/kg bodyweight daily; Prednicare; Animalcare) and within 10 days showed a marked improvement. The ear flaps were much less painful and erythematous, and the cat was playing with the other cat in the household and showed improved demeanour. The dorsal scaling had resolved. There was no alteration to the right corneal opacity, and no pulmonary or cardiac abnormalities were detected on auscultation.

After three weeks, the prednisolone was reduced to 5 mg daily (1·5 mg/kg/day), and after six months to 2·5 mg on alternate

days. Eighteen months later, the prednisolone was withdrawn completely. There was no subsequent recurrence in the ear condition during the 14 months follow-up period.

## DISCUSSION

Relapsing polychondritis in humans is a rare condition in which auricular distortion and discomfort are major signs. Cartilage in other sites may also be affected, and in a proportion of cases this results in saddle nose deformity (collapse of the nasal septal cartilage), joint disease, laryngeal/tracheal disease (leading to collapse of the tracheal rings), ocular disease, cardiovascular disease and skin disease. Fever, hearing loss, vertigo and anaemia may also occur. Associated ocular diseases can include conjunctivitis, scleritis, keratitis and keratoconjunctivitis sicca. Cardiovascular changes include aortic insufficiency, pericarditis, arterial aneurysms and arteritis (White 1985, Michet and others 1986).

The disease in humans is believed to be a true autoimmune disorder in which autoantibodies (predominantly immunoglobulin [Ig]G) to native type II collagen are found in titres of up to 1:320 (McKee 1996). This contrasts with the situation in rheumatoid arthritis, in which autoantibodies to unfurled type I, II and III collagen are found, suggesting that antibody production in this condition is an epiphenomenon rather than the cause of disease (Foidart and others 1978, Ebringer and others 1981, Meyer and others 1981).

In some strains of rats, idiopathic auricular chondritis is common, and is characterised by histological changes similar to those described in the present case (that is, auricular inflammation, invasion of conchal cartilage by inflammatory cells and chondrolysis)(McEwen and Barsoum 1990).

In the cat, relapsing polychondritis is very rare. To the authors' knowledge, only three confirmed cases have been reported in the literature. The case described here is interesting because it is similar to that described by Bunge and others (1992); both cats had evidence of ocular and cardiac changes in addition to the pinnal disease, as can occur in human cases. The other changes seen in humans have not yet been reported in cats. Gauguere and Declercq (1999), calling the condition plasma cell chondritis, suggested a possible viral aetiology, since one reported case was FIV positive (Gauguere and others 1992) and one FeLV positive (Bunge and others 1992); the present case was negative for FIV, FeLV and feline coronavirus.

The significance of the positive cold Coombs' test result in the present case is uncertain. A titre of 1:2 is a low positive result; the cat was not anaemic, and did not have signs of cold agglutinin disease, such as punched-out necrosis of the ear-flap edges, or a vascular pattern of ulceration and necrosis. Also, the histopathological findings were not compatible with cold agglutinin disease. It is of interest that a weak positive Coombs' test result of 1:4 was found in the case reported by Bunge and others (1992).

Although the disease in cats has been designated relapsing polychondritis, the authors prefer the description of auricular chondritis in their patient, since they have no evidence of involvement of other cartilaginous tissues, nor does the benign clinical course in this case warrant the use of the qualifier 'relapsing'.

Clinical differentiation of auricular chondritis from aural haematoma and aural trauma is straightforward; haematoma and trauma are unlikely to be bilateral, nor to be as painful or intensely erythematous. Aural haematomas may exhibit cartilage degeneration and irregular erosion adjacent to the haematoma (Joyce and Day 1997) but do not show the severe, aggressive and extensive changes seen in the present case.

Other differential diagnoses include scarring from irritation associated with ear mite infestation, actinic scarring and distortion in cats with unpigmented ear-tips, discoid and systemic lupus erythematosus, and vascular diseases, such as frost-bite damage, and immune-mediated vasculitis.

Cases of floppy pinnae in cats (Pearson 1998, Rest 1998) may have the same aetiology as reported here. It is clear that such cases justify as full an investigation as possible, including cardiac and ocular assessment, and detailed histopathological examination, in order to assist with diagnosis and understanding of this condition.


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