Osteochondrodysplasia in a 9-Year-Old Scottish Fold Cat

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Introduction

Folded ear, which is characteristic specification of the Scottish Fold cats, arises from an autosomal dominant gene mutation that causes a deterioration of osteochondral ossification. Also, this mutation may cause osteochondrodysplasia that is a hereditary disease affecting joint and bone structure, leading to progressive skeletal deformations at the distal extremities, lumbar vertebral joints and tail.¹

There are some management methods for osteochondrodysplasia, but there is no definitive treatment because it is of heritable origin. The clinical signs and pain can be reduced but the condition is always progressive.¹ Therefore, breeding of this breed should be controlled and breeders and owners should be more widely informed about the prognosis.

History and Clinical Findings

A 9-year-old, female, Scottish Fold cat was presented to the Adnan Menderes University Veterinary Faculty Small Animal Clinic with a history of difficulty in urination, defecation and respiration of 5 days duration. The condition of the cat was stated to be better and worse from time to time.

Clinically, no specific reason other than secondary problems arising from ankylosing changes in vertebral column and joints which may cause urination, defecation and respiration difficulties was found. Urinary dipstick, complete blood count and serum biochemical profile were evaluated in the laboratory examination. Leucocytosis (33.4 mg/dL, reference interval 6–19 mg/dL), lymphocytosis (20,278 × 10⁹/mL, reference
interval 0.8–6.1 mg/dL), thrombocytopenia (78 × 10^3/mL, reference interval 175–500 × 10^3/mL) and elevated aspartate aminotransferase (55 U/L, reference interval 1–37 U/L) were determined. Laboratory findings caused us to suspect an infection, but the other parameters were in the reference range.

**Radiological Findings**

Radiographs were taken in ventrodorsal and lateral recumbency and superposition of the limbs was avoided as much as possible (COMED EVA HF 525, 500 mA X-ray, Korea and Konica Minolta Regius Sigma 2 CR, Japan). However, proper positioning of the extremities was impossible because of the muscle contractures. Forcing the limbs into extension of abduction caused signs of severe pain.

Radiologically, severe periosteal bone proliferations were seen on the phalangeal and carpal joints (Fig. 2). Huge exostoses and severe bone proliferations around the tarsal joints were noticed (Fig. 3). Ankylosing changes in the lumbar and caudal vertebrae characterized by severe ossified bridging were seen. The bone proliferations on the tail base were twice the width of the caudal vertebrae (Figs. 2 and 3). There was marked calcification together with contraction at the triceps brachii insertion tendon (Fig. 4).

**Management**

Considering the difficulty of urination, defecation and respiration, cepalexin (20 mg/kg intramuscularly, q12h), metronidazole (10 mg/kg intravenously, q12h), carprofen (2.2 mg/kg, subcutaneously q48h, two times) and vitamin C (20 mg/kg, per os [PO], q24h) as well as enema were administered. A week later, the cat’s laboratory findings were in the reference range, and glycosaminoglycan chondroitin sulphate (0.5–1 mg/kg, PO, 24h) and methylprednisolone (2 mg/kg, intramuscularly q12h for 3 days, later 1 mg/kg PO q12h) were administered and a slight increase in the movement of the cat was observed in a few days.

**Discussion**

Previously it was assumed osteochondrodysplasia affects only homozygous cats in terms of folded ear (Fd) gene. Subsequent research has revealed that it may affect heterozygous cats (Fd/fd) and lesions can be seen at various levels. Skeletal deformations such as progressive lameness, stiff and stilted gait may have later onset in heterozygous cats. Onset age and severity of clinical signs are variable among affected heterozygous cats. In recent years, studies have begun on the identification of the gene that causes this disease and a naturally occurring TRPV4 mutation has been found responsible. Radiographic changes of osteochondrodysplasia are evident in distal extremities and spine. Because of defective endochondral ossification, abnormal shape of the metatarsal and metacarpal bones, osteoarthritis and progressive periarticular new bone formation in distal limb and vertebral joints are seen. To the authors’ knowledge, this cat had the most severe abnormalities to be documented hitherto, in terms of both radiological and clinical findings.
Therapies for osteochondrodysplasia mainly aim to reduce the clinical signs and accompanying pain, thereby raising the living standards of the patient. Because the disease is genetic, any therapy is unlikely to prevent it from progressing. Some cats may show improvement in lameness and pain by using non-steroidal anti-inflammatory drugs, pentosan polysulphates or glycosaminoglycans. Also, new studies demonstrated good outcomes with radiation therapy and samarium-153-1,4,7,10-tetraazacyclododecane-1,4,7,10-tetramethylene-phosphonic acid therapy. In our case in addition to glycosaminoglycans, carprofen was considered to be more suitable therapy in the first place because of leucocytosis. Later, when treatment was continued with corticosteroid, it was observed that the cat was better. Also vitamin C was added, because its contribution to collagen synthesis may contribute to reduce the cartilage degeneration.

In some cases, it was reported that decrease in lameness was provided by surgical interventions such as ostectomies and pantarsal arthrodeses, removal of exostoses or by radiation therapy. Surgical intervention options were never considered for this case, because all the osteophytic lesions, especially those between the vertebrae, were so severe, and improvement would not be provided in the cat’s general condition by surgery.

In this report, clinical and radiological findings of this disease which occurred in advanced age and severe level were shared to emphasize the importance of considering this untreatable heritable osteochondrodysplasia, in the breeding of Scottish Fold cats.

**Note**
Part of this case report was presented as a poster presentation at the 11th Small Animal Veterinary Association Continuous Training Congress, 4–5 November 2016, Istanbul, Turkey.
Authors’ Contributions
Both the authors contributed to conception of study, study design, acquisition of data and data analysis and interpretation. Both the authors drafted, revised and approved the submitted manuscript.

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Conflict of Interest
None declared.

References