Published online: 2019-11-22

Osteochondrodysplasia in a 9-Year-Old Scottish Fold Cat

Gulcan Turan[1] Zeynep Bozkan[1]

[1] Department of Surgery, Faculty of Veterinary Medicine, University of Adnan Menderes, Aydin, Turkey

Address for correspondence Zeynep Bozkan, DVM, PhD, Department of Surgery, Faculty of Veterinary Medicine, University of Adnan Menderes, Aydin 09016, Turkey (e-mail: zbozkan@adu.edu.tr).

VCOT Open 2019;2:e60–e63.

Abstract

Objective The aim of this study was to share clinical and radiological findings of extremely advanced osteochondrodysplasia.

A 9-year-old Scottish Fold cat was presented with the complaint of difficulty in urination, defecation, respiration, standing and walking. Physical findings such as kyphosis, hyperflexion of joints and thick tail and, radiological findings including periosteal bone proliferation in distal extremities, ankylosing changes in spine and tail and, calcification of triceps brachii insertion were consistent with the most advanced osteochondrodysplasia ever documented. Antibiotic medications, anti-inflammatory drugs, vitamin C and enema were administered considering difficulty of urination, defecation and respiration. Then, glycosaminoglycans and prednisolone were administered for osteochondrodysplasia and increase in the movement of the cat was observed in a few days.

Conclusion By sharing clinical and radiological findings of severe osteochondrodysplasia in advanced age, the importance of informing breeders and owners about uncontrolled breeding of these cats was emphasized.

Keywords
- cat
- osteochondrodysplasia
- folded ear
- Scottish Fold

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.

The cat also had marked kyphosis, different degrees of hyperflexion in the shoulder, elbow, hip and stifle joints (►**Fig. 1**). Contracture of the quadriceps muscle and triceps brachii tendon was very prominent and the joints were very stiff and sensitive when palpated. The cat could only walk by advancing the hind limbs forward together like a rabbit. The tail was very thick, stiff and immobile. The owner indicated that the cat had skeletal deformations for years and received no treatment for this condition. Therefore, their primary concern was urination, defecation and respiration difficulty.

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 $\times 10^{-3}$/mL, reference

received
March 1, 2019
accepted after revision
September 25, 2019

DOI https://doi.org/10.1055/s-0039-1700850.
ISSN 2625-2325.

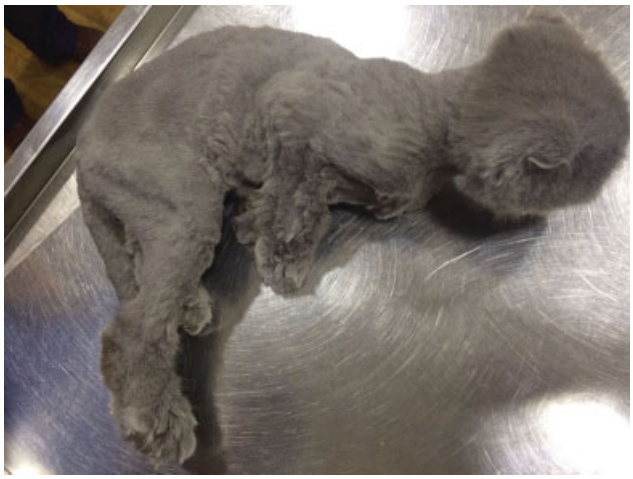

Fig. 1 Clinical appearance of the cat; marked kyphosis, different degrees of hyperflexion in shoulder, elbow, hip and stifle joints.

interval 0.8–6.1 mg/dL), thrombocytopenia (78×10^{-3}/mL, reference interval $175–500 \times 10^{-3}$/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, cephalexin (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.[2] 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.[3–5] 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.[6]

Radiographic changes of osteochondrodysplasia are evident in distal extremities and spine.[1] 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.[6] To the authors' knowledge, this cat had the most severe abnormalities to be documented hitherto, in terms of both radiological and clinical findings.

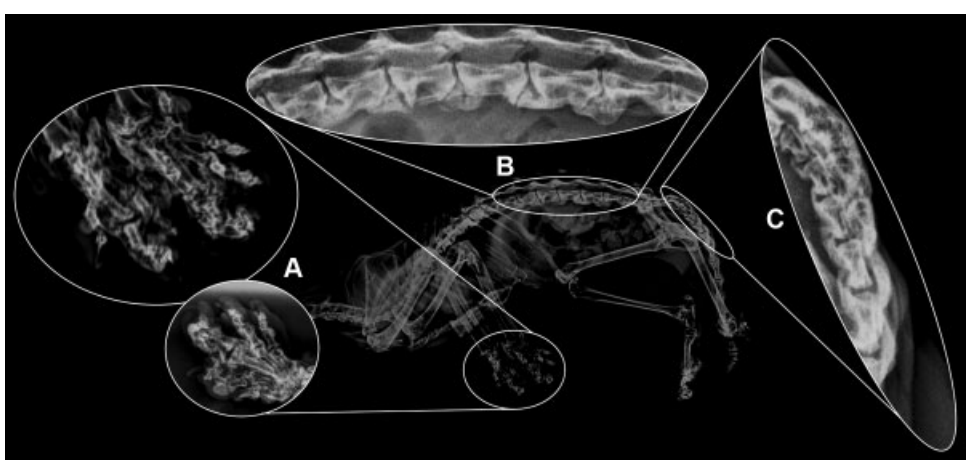

Fig. 2 Right lateral radiograph of the cat showing severe periosteal bone proliferations seen on the phalangeal and carpal joints (**A**), ankylosing changes in the lumbar (**B**) and the caudal (**C**) vertebrae characterized by severe ossified bridging.

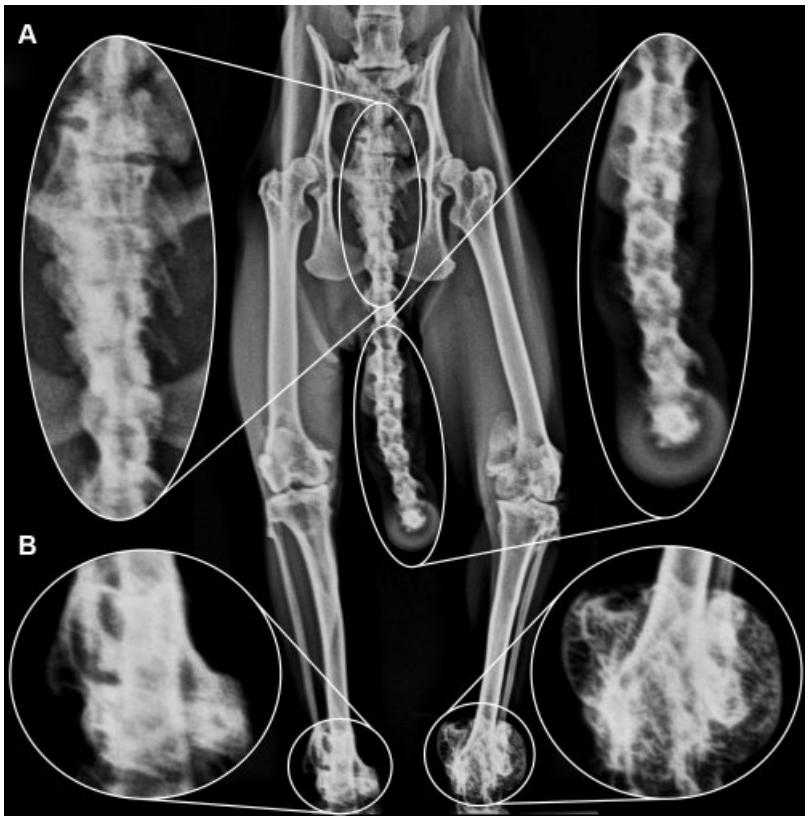

Fig. 3 Ventrodorsal radiograph of the hip and caudal vertebrae (**A**) together with craniocaudal projection of exostoses around the tarsal joints (**B**).

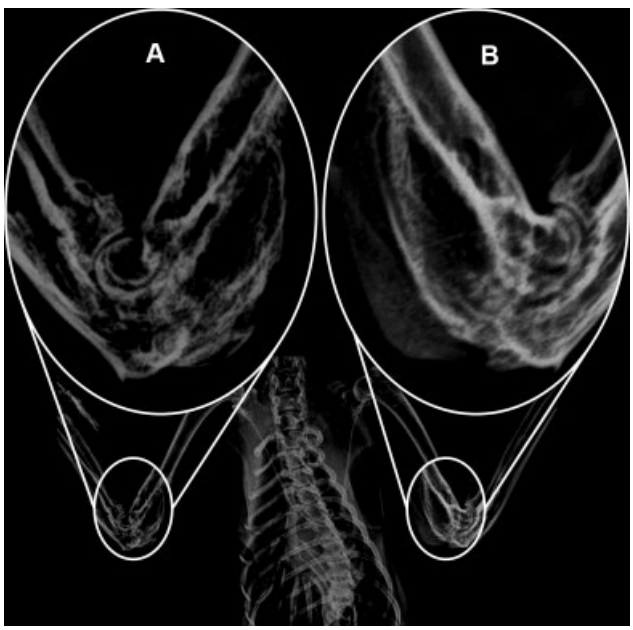

Fig. 4 Ventrodorsal radiograph of the thorax together with mediolateral projection of the right (**A**) and left (**B**) humerus showing calcification at the triceps brachii insertion tendon.

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.[1] Some cats may show improvement in lameness and pain by using non-steroidal anti-inflammatory drugs, pentosan polysulphates or glycosaminoglycans.[4,7,8] 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.[9] 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[10] 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.[1,3,11] 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.

Funding
None.

Conflict of Interest
None declared.

References

1 Hubler M, Volkert M, Kaser-Hotz B, Arnold S. Palliative irradiation of Scottish Fold osteochondrodysplasia. Vet Radiol Ultrasound 2004;45(06):582–585
2 Jackson OF. Congenital bone lesions in cats with fold-ears. Bull Fel Advis Bur 1975;14:2–4
3 Mathews KG, Koblik PD, Knoeckel MJ, Pool RR, Fyfe JC. Resolution of lameness associated with Scottish Fold osteodystrophy following bilateral ostectomies and pantarsal arthrodeses: a case report. J Am Anim Hosp Assoc 1995;31(04):280–288
4 Malik R, Allan GS, Howlett CR, et al. Osteochondrodysplasia in Scottish Fold cats. Aust Vet J 1999;77(02):85–92
5 Takanosu M, Takanosu T, Suzuki H, Suzuki K. Incomplete dominant osteochondrodysplasia in heterozygous Scottish Fold cats. J Small Anim Pract 2008;49(04):197–199
6 Gandolfi B, Alamri S, Darby WG, et al. A dominant TRPV4 variant underlies osteochondrodysplasia in Scottish Fold cats. Osteoarthritis Cartilage 2016;24(08):1441–1450
7 Chang J, Jung J, Oh S, et al. Osteochondrodysplasia in three Scottish Fold cats. J Vet Sci 2007;8(03):307–309
8 Aydin D, Altunatmaz K, Erdikmen DO, Ozer K, Durmus D, Avanus K. Hereditary osteochondrodysplasia in Scottish Fold Cats. Kafkas Univ Vet Fak Derg 2015;21:463–469
9 Selting KA, Lattimer JC, Hause W, Megan G. Osteochondrodysplasia in a Scottish Fold cat treated with radiation therapy and samarium-153-1,4,7,10-tetraazacyclododecane-1,4,7,10-tetramethylene-phosphonic acid. J Am Anim Hosp Assoc 2019;55(03):e55304
10 Murad S, Grove D, Lindberg KA, Reynolds G, Sivarajah A, Pinnell SR. Regulation of collagen synthesis by ascorbic acid. Proc Natl Acad Sci U S A 1981;78(05):2879–2882
11 Fujiwara-Igarashi A, Igarashi H, Hasegawa D, Fujita M. Efficacy and complications of palliative irradiation in three Scottish Fold cats with osteochondrodysplasia. J Vet Intern Med 2015;29(06):1643–1647