OSTEOCHONDRODYSPLASIA IN SCOTTISH FOLD CATS CASE REPORT

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ABSTRACT

The Scottish Fold breed of cat has a genetic mutation that affects the development of cartilage. The most obvious manifestation of this is the typical appearance of the ears of Scottish Fold cat. This genetic defect also causes abnormalities of the cartilage of bones. Scottish Fold osteochondrodysplasia (SFOCD) is characterized by skeletal deformities such as short, thick, and inflexible tails and shortened splayed feet.

Two Scottish Fold cats aged between 8 months and 1 year were presented for signs of skeletal disease including lameness, pain and reluctance to jump. A diagnosis of osteochondrodysplasia was based on typical characteristic radiological findings. Clinical signs were ameliorated by administration of non-steroidal anti-inflammatory drugs and drugs, contain glycosaminoglycans.

Definitive treatment of the disease is impossible. Survival of the cats varies depending on the severity of clinical signs.

Key words: osteochondrodysplasia, Scottish Fold cat

Introduction

The breed “Scottish fold cat” is a domestic breed with an autosomal dominant gene mutation which affects the cartilage tissue in the whole body. That is the reason of the breed’s appearance – the folded ears. The osteochondrodysplasia in cats is a familial disease characterized with skeletal deformities like short, thickened and hard movable tail and short, buckled legs [6, 7].

The affected cats are showing signs of difficult mobility, lameness, lack of desire for climbing or jumping and the symptoms worsen over time. Sometimes there is presented vocalization when moving or jumping. The reason of this is the progressive developing of osteoarthritis due to defects in the maturing, dysfunction of the cartilage and abnormal endochondral ossification [2, 8].

Diagnosis is based on the characteristic anamnesis, the clinical signs and the x-ray examination. The x-ray signs include abnormalities in the size and shape of the tarsal, carpal, metatarsal, metacarpal bones, the phalanges and the sacrum. The articular spaces are decreased and with the progress of the disease around the distal joints are presented exostoses with diffuse osteopenia, much better expressed in the pelvic limbs [1]. For the moment it is not established a definitive method for treatment, but the non-steroidal anti-inflammatory drugs (NSAIDs) in combination with supplements containing glycosaminoglycans improve the condition [2, 5].

The purpose of this study was to summarize the clinical and x-ray signs and to evaluate the effect of the applied treatment.

Material and Methods:

Clinical case 1

It is about a male cat, 1 year and 3 months old, weighing 2.6 kg. It was brought to the clinic with anamnesis of difficult mobility, stiff gait, mixed lameness and periods of complete adynamia...
with a strong demonstration of pain. The gait was incorrect and when there was a spontaneous movement, the cat was moving squatted. The pelvic limbs occupied unnatural position and the animals were putting them slightly laterally (picture 1, 2, 3).

**Clinical case 2**

It is about a male cat, 8 months old, weighing 1.9 kg. with anamnesis of a periodic lameness, lack of desire for playing. Most of the times it prefers to hide and lay. The owners have noticed that the periods in which the cat is feeling good and is dynamic are followed periods of 3-5 days of adinamia, decreased appetite and signs of pain, especially after palpation in the area of the pelvic limbs (picture 4, 5).
Results:

The x-ray examination showed changes that characteristic of osteochondrodysplasia in both of the animals. The main radiologic findings were decreased articular spaces with a dim outline in the area of the metatarsal and metatarsophalangeal joints. In both of the animals the amendments were more severe in the right pelvic limb. In the first case was observed decreased bone density with unclear, blurred outline of the phalanges and the x-ray shadow of the tarsal bones was with reduced and unequal density – with zones of rarefication. In some places the bones looked like moth-eaten.

The cats were treated with firocoxib (Previcox®, Merial) 5 mg/kg p.o., for a period of 10 days, every month, and glucosamine HCl, Chondroitine sulfate, Harpagophytum procumbes (Flexadine®, Vetoquinol), one tablet dialy, for twenty days, each month.

After the administration of the therapeutic scheme the animals were significantly more dy-namic and playful. No signs of lameness or pain were presented.

Significantly better clinical response to the treatment was observed in the second patient, which was expressed in the lack of lameness and signs of pain.

Discussion:

It is found that the severity and the duration of expression of the clinical signs of osteochondrodysplasia in the Scottish-fold breed depend of the genetic expression of the defective gene. The severity of the clinical and x-ray signs is different for different animals and is most likely determined by the level of expression of the dominant gene carrier of the mutation (homo- or heterozygous expression). In homozygous as regards of this gene animals the disease develops progressively and is accompanied with severe defects in both epiphyseal and metaphyseal spaces. In parallel is developing osteoarthritis in early age [3, 4, 5, 6].

Osteochondrodysplasia in cats could be diagnosed relatively easily by using x-ray, the abnor-malities mainly affect the tarsal and carpal zones of the limbs. The x-ray signs are based on skeletal abnormalities and developing of polyarthropaties with a tendency for deforming and ankylosing osteoarthritis [8]. Leading radiological signs are reduced joint spaces with serrated and indistinct
outline, bone deformation, zones with unequal density and limited areas of rarefication of the bone substance.

Generally, osteochondrodysplasia occurs due to defective endochondral ossification, resulting in disproportionate dwarfism and morphological defects in the axial and appendicular skeletons (2, 8). It is seen in only Scottish Fold cats and fast-growing, large and giant breeds of dogs such as Alaskan malamute, Great Pyrenees, Labrador retriever, Norwegian elkhound, Samoyed, and Scottish deerhound. Prevention of the disease is to avoid cross-bred pure breed Scottish-fold cats so that the future generations are homozygous as regards to the defective gene [2, 5].

Treatment with glucosamine, chondroitin and NSAIDs is palliative, but shows very good clinical results and significantly improves the quality of life of affected cats.

References