A generalized chondropathy of joint cartilage leading to deformity of the elbow joints in a litter of Newfoundland dogs

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ABSTRACT

A description of a litter of Newfoundland dogs of which six out of seven puppies suffered from, more or less, deformation of the elbow joints, is presented. The two male dogs were, because of the condition, destroyed at 20 weeks of age. The patho-anatomical examination revealed abnormalities of the joint cartilage of all the major joints of the extremities. The condition is described as a generalized fibroid, proliferative degeneration of the joint cartilage.

INTRODUCTION

Developmental abnormalities of the elbow joint other than ununited ancone process, osteochondritis dissecans of the humeral condyle, fragmentation of the coronoid process or lesions caused by disturbances of the growth plates, have occasionally been described (Fox, 1963, 1964; Flipo, 1965; Ljunggren et al., 1969, 1979; Campbell, 1969, 1979; Grøndalen, 1973; Stevens & Sande, 1974; Bingel & Ris, 1977; Carrig et al., 1977).

This report gives a presentation of the clinical, radiographical and patho-anatomical findings of a litter of Newfoundland dogs, suffering from a generalized chondropathy of the joint cartilage.

MATERIAL AND METHODS

The litter in question consisted of seven dogs, two males (cases 1 and 2) and five females (cases 3–7). Cases 1, 2, 6, 7, the dam and the sire were clinically and radiographically examined by the author. Case 3 was clinically and radiographically examined by a colleague. Cases 4 and 5 were radiographically examined by colleagues. All radiographs were sent to the author for interpretation.
### Table 1. The sex, age, clinical and radiographical findings of a litter of Newfoundland dogs and their parents

<table>
<thead>
<tr>
<th>Case number</th>
<th>Sex</th>
<th>At onset of symptoms</th>
<th>When examined</th>
<th>Clinical symptoms judged by the owners*</th>
<th>Radiographical findings of the elbow joints†</th>
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<tbody>
<tr>
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<td>M</td>
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<td>15, 20</td>
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<td>2</td>
<td>M</td>
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* a. Abnormal forelimb position, unwilling to move and lame. b. Lameness of one or both forelimbs. c. Occasionally lame, stiff. d. No abnormalities concerning the function of the limbs.


Both elbow joints of all dogs were radiographically examined. In addition the radius and ulna, the thoraco-lumbal columna, the hip and the stifle joints of cases 1 and 2 were radiographically examined.

Cases 1 and 2 were destroyed and autopsied at 20 weeks of age. In order to inspect subchondral bone and the epiphyseal plates, the long bones were partly divided. Thirty-four samples from the joint cartilage and subchondral bone tissue of all the major joints of the extremities as well as the costochondral junction were collected for histological examination. The specimens were decalcified ('Decalc', Bethlehem A-B Goteborg) and were sectioned and stained according to the haematoxylin-eosin (H&E) and van Gieson (vG) methods.

The material and the main clinical and radiographical findings are given in Table 1.

## Case Reports

The litter had been released by Caesarean section at 58 days of pregnancy (primary inertia). The dam had previously given birth to two litters (12 puppies). Of the previous offspring, two dogs suffered from severe hip dysplasia, another from osteochondritis dissecans of the humeral head. Otherwise none of the previous offspring had been reported to suffer from skeletal disease. The sire had not been mated before. Two of his litter mates had been destroyed due to skeletal abnormalities.
The nursing of the litter in question was limited, and the puppies were to a major extent artificially fed by bitch-milk substitutes. According to information given by the breeder, they were fed home-made food; rice, bread, egg and meat supplied with adequate amounts of minerals and vitamins from 3 weeks of age.

When the litter was 3 and 6 weeks of age, there were histories of diarrhoea and reduced weight gain. According to the breeder, the gait of some of the puppies was periodically stilted. Otherwise, no abnormalities were observed by the breeder. The puppies were sold and delivered at 9 weeks of age.

Cases 1, 2 and 3 were, independent of each other, admitted for veterinary examination because of abnormal foreleg position and unwillingness to move at 15, 20 and 18 weeks of age respectively.

**Clinical findings**

Cases 1 and 2. The forelegs appeared shorter than normal and the elbow joints were somewhat angled in the antero-posterior view (cubitus valgus). The paws were slightly rotated laterally. The dogs preferred to sit or to lie down. They were unwilling to move, but when forced, the steps were shorter than normal and the movements appeared stiff. Pain was elicited when bending and extending all the main joints of the extremities. The elbow joints were increased in diameter and the flexibility was decreased. The shape and the flexibility of the other main joints appeared normal. The dogs were, because of worsening of the clinical condition, destroyed at the age of 20 weeks.

Case 3. This dog was limping on its left foreleg. It moved more easily than cases 1 and 2, but otherwise the findings were identical to those described above.

Case 4. Stiffness and periodical lameness were observed by the owner at about 18 weeks of age.

Cases 5, 6 and 7. No abnormalities regarding the movements were observed by the owners. However, case 6 revealed a ‘stilted’ way of walking when examined by the author, and pain was revealed when bending and extending the elbow joints.

The dam. The dam had been periodically lame on both forelegs. Pain was elicited when palpatating and bending the elbow joints, and the flexibility was decreased. Otherwise, no abnormalities were observed.

The sire. Nothing abnormal was observed regarding the movements of the sire. No pain was elicited when examining the joints. The exterior conformation appeared normal.

**Radiographical findings**

Cases 1 and 2. The epiphyseal plates appeared normal. An abnormal angulation of the elbow joints was apparent in the antero-posterior view (Fig. 1). The coronoid process was underdeveloped, and little support was given to the medial part of the humeral condyle. The humeral condyle was dislocated medially and distally. The radial head gave the impression of being fragmented (Fig. 2). The anconeal process
was of normal size, ossified, but not fully fused to the ulna. At 20 weeks of age, the fusion was more complete in case 2 than in case 1.

Case 3. The radiographical findings of the elbow joints were similar to those described above, but fragmentation of the radial head was not seen.

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**Fig. 1.** Case 1. Radiograph of the elbow joint, antero-posterior view. The coronoid process is giving little support to the medial part of the humeral condyle which is dislocated medially and distally.

**Fig. 2.** Case 1. Radiograph of the elbow joint, lateral view. (a) Anteriorly inclined (underdeveloped) medial coronoid process. (b) Sign of fragmentation of the radial head. (c) The anconeal process, not fully fused to the ulna.
Fig. 3. Case 2. The right humeral head. Small protuberances of chondroid tissue are seen centrally on the joint surface.

Cases 4, 5 and 6. The coronoid process appeared somewhat anteriorly inclined, and a moderate angulation was seen in the antero-posterior view. Otherwise, no abnormalities were observed.

Case 7. The shape of the elbow joints was normal. A slight degree of osteophyte formation was seen in the lateral view, at the proximal edge of the anconeal process.

The dam. Severe degree of osteophyte formation was present in the periphery of the elbow joints. The shape of the bones appeared normal.

The sire. Nothing abnormal was found when interpreting the radiographs of the elbow joints.

Patho-anatomical findings

Gross examination. Cases 1 and 2 were destroyed and autopsied at 20 weeks of age. The principal changes were related to the superficial layer of the joint cartilage of the major joints of the extremities. No obvious abnormalities were observed regarding the shape of the bones except for the proximal radius and ulna. The epiphyseal plates and the joint capsules appeared normal. The lesions described were bilateral and close to similar in the two dogs.

The main changes may be described as follows: local areas of multiple small cartilage protuberances, most typically illustrated centrally on the humeral head (Fig. 3); thin flakes of fibro-chondroid tissue covering splits and enfoldments of the cartilage and the sub-chondral bone. These changes were seen in the medial part of the humeral condyle (Figs 4, 5), in the posterior part of the femoral head (Figs 6, 7) and laterally to the intercondyloid eminence of the proximal tibia. The size of the flakes varied; on the proximal tibia the flake covered an area of about $8 \times 20$ mm.
The thickness of the cartilage varied; and in some areas had a soft and moistened consistency. This finding was specially noticeable on the humeral head and on the proximal tibia. Penetrating fissures were found at different locations in the majority of the joints.

*The elbow joint.* In the weight-bearing area of the medial part of the humeral condyle, oval lesions resembling osteochondritis dissecans, extending over an area of about $4 \times 10$ mm, were found in case 2. In the left elbow joint, the flap of

![Fig. 4. Case 2. The left humeral condyle. A defect similar to an osteochondritis dissecans is partly covered by a flake of fibrochondroid tissue.](image)

Subchondral bone tissue is seen in the defect.

![Fig. 5. The same specimen as Fig. 4. The flake of fibrochondroid tissue is folded aside.](image)
cartilage, covering the sub-chondral bone, continued into a thin flake of chondroid tissue, similar to that described above (Figs 4, 5). The proximal part of the ulna was somewhat curved, most apparent in the antero-posterior view (Fig. 8). The medial coronoid process was not horizontally directed as normal, but anteriorly inclined (underdeveloped). In the anterior part of the process, fragments and fissures were seen (Fig. 9). A massive wall of fibrous tissue rimmed the dorsal and medial area of the process (Fig. 9).

In both dogs the radial head was latero-medially inclined (Figs 9, 10). The joint surface was divided by a broad split, antero-posteriorly directed (Fig. 10). The depth of the split was 5–10 mm, and it was partly filled with connective tissue. The joint surface medial to the split was partly fragmented, and splits between fragments were filled with connective tissue.
Histological findings

The histological picture varied, but some abnormal structure was found in all joints examined. The main changes were related to the superficial layer of the joint cartilage; however, the deeper layer as well as sub-chondral bone and epiphyseal plates were partly involved (Figs 11–16).

The joint cartilage was of normal, or increased, thickness. In some joints (the humeral head, the humeral condyle, the femoral head and the proximal tibia) flakes of fibroid formations appeared to develop from the superficial layer of the joint cartilage (Fig. 16). These flakes were partly invaded by vessels and partly degenerated. In areas in which the thickness of the joint cartilage had increased, the cartilage was partially replaced by fibrous tissue throughout the height of the cartilage (Figs 12, 16). In some areas fibrous development within the hyaline cartilage seemed to occur, with no vessels or fibrocytes being visible (Fig. 15). At
other locations vessels and fibrocytes were present in the transformed fibro-chondroid tissue. In the deeper layer of the joint cartilage, there were areas of degeneration consisting of small chondrocytes with pycnotic nuclei (Fig. 14). Clusters of unopened cartilage cells (Fig. 14), as well as necrotic tissue were also seen.
Enfolded cartilage and cystic formations and cracks, vertically and horizontally directed, penetrating into the epiphyseal bone, were common findings (Figs 13, 14).

Generally, the ossification of the bone trabeculae appeared normal. Osteoblast activity varied, but was present in the majority of sections taken. Remnants of chondroid matrix were present centrally in the trabeculae. In some sections the trabeculae were scarce and thin, and appeared fractured with a few intertrabecular cells (Fig. 12). In other sections, the trabeculae were thicker with an abundance of cells present between the trabeculae. At some locations amorphous masses of necrotic tissue were seen between the bone trabeculae (Fig. 12). This tissue was present in the epiphysis as well as in the diaphysis.

Islets of degenerated cartilage were present in areas beneath degenerated joint cartilage.

The columns of cartilage of the epiphyseal plates as well as the primary spongiosa, were in most areas of normal height and orderly arranged. At some locations, however, degenerated hyaline cartilage was present at the epiphyseal side of the growth plates, thus leading to irregularity of the vesicular cartilage as well as the metaphyseal trabeculae (Fig. 13).

The two sections of costo-chondral junctions revealed columns of vesicular cartilage orderly arranged, and so was the primary spongiosa. In one section a transverse fissure separated the columns of vesicular cartilage.

Fig. 11. Case 2. The left humeral head, histological section. A protuberance of fibrochondroid tissue is prominent from the joint surface (conf. Fig. 2). The epiphyseal bone trabeculae are scarce, thin and partly fractured. H&E, ×8.5.
FIG. 12. Case 2. The left humeral head, histological section, revealing fibrous development of parts of the joint cartilage. The epiphyseal bone trabeculae are scarce, thin and partly fractured. Few intertrabecular cells are visible. Amorphous masses of partly necrotic tissue in some areas (arrows). H&E. x 8.5.

FIG. 13. Case 2. The left radial head, histological section, revealing cracks, degenerated cartilage and cystic formations. The epiphyseal cartilage as well as the primary spongiosa appear normal, except in the area beneath the degenerated and cystic part of the epiphysis. H&E. x 8.5.
DISCUSSION

The pathological processes of the cases reported were primarily related to the joint cartilage. In the majority of the joints small protuberances or thin flakes of chondroid tissue were proliferating from the joint surface. Further, fibroid replacement and degeneration affected the deeper layer of the joint cartilage as well as the epiphyseal bone trabeculae, and led to malformation of the medial coronoid process of the ulna and the radial head.

Congenital dislocations or luxations of the elbow joint are reported by Fox (1964), Campbell (1969, 1979), Pass & Ferguson (1971), Grøndalen (1973), Stevens & Sande (1974), Carrig et al. (1977), Bingel & Riser (1977) and Milton et al. (1979). More common causes of elbow lameness in young dogs, especially of larger breeds, are ununited anconeal process, fragmentation or fissures of the coronoid process or osteochondritis dissecans of the humeral condyle (Olsson, 1977; Grøndalen, 1979; Grøndalen & Grøndalen, 1981; Berzon & Quick, 1980; Denny & Gibbs, 1980; Robins, 1980; Tirgari, 1980; Mason et al., 1980).

According to Olsson (1977) the three latter lesions are different manifestations of the generalized disease osteochondrosis. Osteochondrosis is defined as a disturbance in the endochondral ossification (Ljunggren & Reiland, 1970).

According to this definition, all lesions of the epiphyseal plates and the basal layer of joint cartilage may be regarded as osteochondrosis. The changes in the presented cases resemble the changes described as osteochondrosis. However, the
fibroid degeneration and the superficial proliferation differ, and thus it does not seem appropriate to include this condition into the osteochondrosis syndrome.

Chondrodysplasia is a term used in connection with hereditary dwarfism in the Alaskan Malamute (Fletch et al., 1973). They reported the changes to be related to the cartilage of the growth plate.

In the litter presented, six out of seven litter mates appeared to be more or less affected, and the possibility of inheritance was assessed. The mating was therefore
repeated, but the dam did not conceive. At the following heat the dam was disabled because of a ruptured cruciate ligament, and the owner refused to mate her.

Inadequate supply or unbalanced amounts of vitamins and minerals during the period of growth may lead to disturbances of cartilage and bone formation. In the presented cases, however, the principal changes were localized to the superficial layer of the joint cartilage, thus differing from the changes seen in classic forms of nutritional skeletal disease of young animals (Rickets, secondary hyperpara-
thyroidism, hyper- or hypo-vitaminosis A). Deficiencies of trace elements as copper, manganese, magnesium and silicon, may lead to disturbance in growth of the skeleton (Hurley & Asling, 1963; Underwood, 1971; Carlisle, 1977; Chou et al., 1979), but the changes described in the experimentally induced deficiencies, do not correspond to the findings in these dogs.

The possibility of polyarthritis as a reason for the changes, may be excluded since the joint capsules appeared normal.

The conditions, previously described, concerning disturbances of the cartilage of the growing animal are, according to the author, different from the lesions described in this article, and the neutral word chondropathy is chosen for the condition.

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REFERENCES


