The Degenerative Joint Disease – Morphological Aspects in the Wild Boar

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Abstract
The aim of the study was to outline some data about the morphology of the pathological wild boar articular cartilage. The study was carried out on articular cartilage samples from wild boar femoral head and condyles, 1 - 10 years old. The samples were processed and stained by usual histological techniques. The samples examination revealed different stages of articular cartilage injury and various morphological aspects that suggest the degenerative joint disease (DJD) occurrence. The chondrocyte clones, the extracellular matrix alteration, the cartilage fissures, the cartilage erosions and the subchondral bone exposure were the observed morphopathological aspects. These aspects are the specific pathological lesions for DJD, as described in the specialized literature for other species.

Keywords: degenerative joint disease, morphology, wild boar

1. Introduction

The articular cartilage is strictly adapted for wear and tear stress, so that any homeostasis disruption could lead to severe damages that may finalize to pathological states, as degenerative joint disease (DJD). The DJD is a degenerative progressive disease of the synovial articulation that involves all joint components. The DJD ends with the cartilage, synovia and bone structure alteration and with changes in the joint anatomy structure and function [1, 2, 3, 4].
The articular cartilage injury can be observed in domestic animals and in the wild ones. Because the articular cartilage specific morphological aspects are slightly investigated in the wild animals the present paper tries to outline some data about the morphology of the wild boar articular cartilage, as a starting point for further studies. Taking into account the variability phenomenon in biology, we consider that this study is also necessary in order to offer a large data base for the morphology of the articular cartilage.

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2. Materials and methods

The study was carried out on articular cartilage samples from wild boar femoral head and condyles, 1 - 10 years old, from Muvi Impex SRL, Scandia Română SA Sibiu slaughter house. The samples were processed by usual histological techniques and stained by hematoxilin–eosin, Masson's trichrome and Mallory's trichrome methods [5] and they were processed in the Histology laboratory of the Veterinary Medicine Faculty from Timisoara.

3. Results and discussion

On microscopic examination the cartilage aspects were different, according to the severity of the illness. The upper layer was irregular and

sometimes fissured in both in the early and in the advanced stage of the disease (Fig.1).

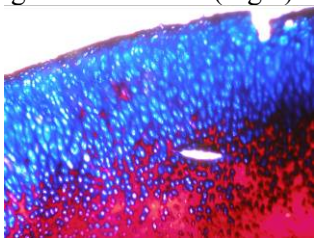

Figure 1. Photomicrograph of the wild boar femoral head. Fissure in the superficial zone. Mallory's trichrome staining, 100x.

In the middle cartilaginous layer the interterritorial matrix was sometimes fragmented and the chondrocytes, that are often polymorphous, tend to group in clones (Fig. 2). An increase in the sulphated compounds secretion in extracellular matrix was observed around the chondrocytes from injured areas (Fig. 2). The chondrocyte clones and the extracellular matrix alteration were the main features of the cartilage morphology changes of the examined samples.

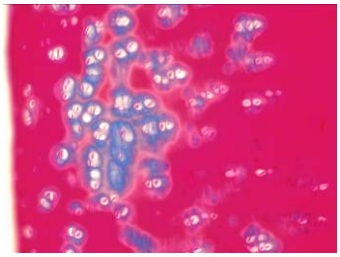

Figure 2. Photomicrograph of the wild boar femoral head. The superficial and the transition zone. Mallory's trichrome staining, 200x.

When the superficial layer is damaged, the extracellular matrix of the middle layer is not homogenous, it is poor in glucosaminoglycans and rich in collagens and the chondrocytes are small and axially grouped. (Fig. 2, 3).

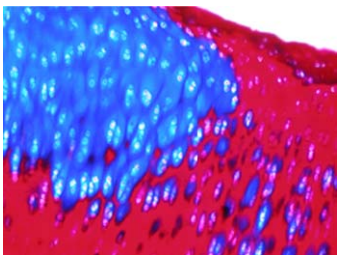

Figure 3. Photomicrograph of the wild boar femoral head. Chondrocytic clones in both superficial and transition layers. Mallory's trichrome staining, 200x.

In the areas where the extracellular matrix was fragmented, the already formed chondrocytic clones may separate and migrate in the articular cavity [6].

In the middle layer was observed the absence of the fibril organization (Fig. 2, 3) and the presence of a thin glycocalyx around chondrocytes. In association with the reduced metacromasia, these aspects reveal the metalloproteinase`s intervention in the pericellular macromolecules catabolism [7]. Following the metalloproteinase`s action on the extracellular matrix the cartilage hydration changes and lead to the local altered biochemistry [8] that disturb the protoglycans synthesis.

The matrix swelling, the beginning of the chondrocyte multiplication and the clone proliferation are the modifications of the chondron that were also observed (Fig. 4). These aspects could be interpreted as a reaction to the lack of the cartilaginous tissue, as a failed healing local reaction or as a consequence of the contact of the cartilage deep layers with the synovial fluid.

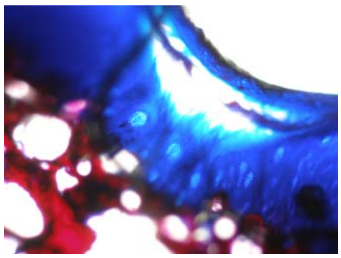

Figure 4. Photomicrograph of the wild boar femoral condyle. Chondrocytic clones in the deep layer. Mallory's trichrome staining, 40x.

The loss of the chondrocyte organization, the rarefaction and the degenerescence of the extracellular matrix were seen only in advanced forms of the disease (Fig. 4, 5, 6).

Figure 5. Photomicrograph of the wild boar femoral condyle. Extracellular matrix fragmentation in different layers and variable chromic aspects. Mallory's trichrome staining, 40x.

According to the data from specialized literature [8, 9], the decrease of the proteoglycans amount in the interterritorial matrix and the consecutive increase of the proteoglycans synthesis and their release in the pericellular matrix, were the specific early changes observed in the degenerative cartilage. As described by Fassebender *et al*., 1982, mentioned by Todhunter [8], we also observed the absence of the metacromasia, the chondrocite clones and often the cartilage fissures, in early degenerative cartilage stages.

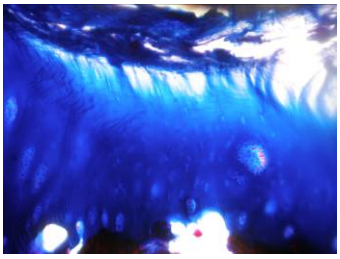

Figure 6. Photomicrograph of the wild boar femoral condyle. Chondrocytic clones in the deep layer. Mallory's trichrome staining, 400x.

Some studies demonstrated that the chondromalacia causes the cartilage injury because of a poor collagen network support [10]. The chondromalacia lead to the increase of the subchondral bone stiffness, so that the bone becomes more sensitive to the mechanical forces.
In the deeper layers, depending on the injury severity, the extracellular matrix was dense and homogenous or emphasized variable chromic aspects. There were observed multiple chondrons (2-6 cells), groups of cells in division, necrotic chondrocytes or empty lacunae (Fig. 7).

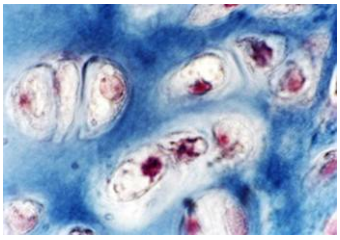

Figure 7. Photomicrograph of the wild boar femoral condyle. Variability of the cell morphology: axial proliferation and chondrocyte necrosis. Mallory's trichrome staining, 900x.

The variability of chondrocyte morphology reveals the cartilage degeneration [11]. The territorial and pericellular matrix of the chondrocyte was fissured (Fig. 4, 5, 6), as it was also described by Van Bree, 1993 [12], who observed this feature, but in the radial layer. At the basis of the profound fissures there were sometimes abnormal chondrocyte multiplications, which are forming clusters, markedly stained for glucosaminoglycans, as observed also by Collins *et al*., 1960, mentioned by Sledge [13]. These morphological and histochemical aspects confirm and demonstrate that the synthesis of the extracellular matrix ceases while the degradative activity remains elevated as osteoarthritis progresses.
Meachim *et al*., 1979, mentioned by Poole [14] demonstrate that in the older cartilages, the progressive accumulation of the heavy keratinsulphate molecules made it stiff and generates the interterritorial matrix expansion in the territorial one and the progressive suffocation of the chondron. The altered chondrocyte nutrition will determine the degenerative changes in cartilage, emphasized by the profound fissure corresponding to the superficial ones.
We observed that the connection between the spongious bone and the calcified cartilage was weak and discontinuous (Fig.5, 6). Sometimes the superficial erosions were related to the thickness of the calcification zone (Fig.5), fact that was considered by others [14] a consequence of the mechanical load increase in the cartilage.
In the subchondral bone incomplete osseous lamellae and large myeloid cavities can be seen (Fig. 8), that sometimes were filled with connective haemorrhagic tissue or with fibrous connective tissue.

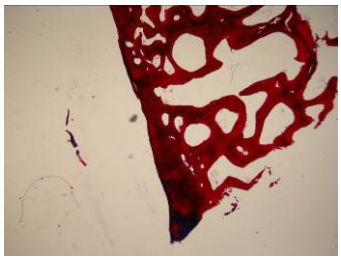

Figure 8. Photomicrograph of the wild boar femoral condyle. Incomplete osseous lamellae and large myeloid cavities. Mallory's trichrome staining, 40x.

The progressive erosion of the articular cartilage may end with the exposure of the subchondral bone that allows injury spreading into the bone (Fig. 4, 5, 8).
The advanced degenerative states were accompanied by osseous rarefaction. The bone resorption or sclerosis is a consequence of the

microfractures of the subchondral trabeculae, as a first trauma effect or as a secondary mechanical cartilage properties alteration [8]. Radin and Rose, 1986, mentioned by Todhunter [8], consider that the subchondral microfractures and the consecutive bone sclerosis are the primary osteoarthritis causes.

According to specialized literature [13], we found out that vertical fissures of the cartilage are related to the age of the animal and do not lead obligatory to osteoarthritis.

However the proteoglycan synthesis in degenerative cartilage was observed, that was not sufficient to re-establish the healthy state. Carney *et al*., 1985, and Pelletier *et al*., 1987, mentioned by Todhunter [8], demonstrated that the increased proteoglycan synthesis does not compensate either quantitatively or qualitatively the true needs of the cartilage, because of the reduced aggregation capacity.

In early degenerative joint disease stages the progression of the matrix degradation may be moderated by increasing the matrix compounds synthesis, therefore the collagen and glucosaminoglycan content will be stabilized for a while. In advanced stages of the degenerative joint disease the chondrocyte ability to maintain the matrix homeostasis ceases and the cartilage will be eroded.

We agree that also in the wild boar degenerative joint disease the degradation of the cartilage begins at the surface, but it also could be possible to start at the basis (the microfracture presence), as the specialized literature emphasize [15].

4. Conclusions

The samples examination revealed different stages of articular cartilage injury and various morphological aspects that suggest the occurrence of the primary degenerative joint disease.

The articular cartilage of the wild boar degenerative joint disease presented chondrocytic clones, extracellular matrix alteration, cartilage fissures and erosions, subchondral bone exposure and epiphyseal bone rarefaction, as described in the specialized literature for other species.

The severe forms of osteoarthritis were noticed mainly in older animals, which emphasize the role of cartilage aging in articular pathology.

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