HETEROGENEOUS ADAPTATION OF THE PATELLOFEMORAL JOINT TO SHORT- AND LONG-TERM ANTERIOR CRUCIATE LIGAMENT DEFICIENCY

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INTRODUCTION

Focal chondral or osteochondral defects occur at an earlier age, are more severe and cover a larger portion of the cartilage surface on the patella compared to the femoral groove in the human patellofemoral joint (Meachim and Emery, 1974, Hjelle et al., 2002). The majority of research investigating the etiology of patellofemoral cartilage defects has focused on joint biomechanics. Less attention has been given to other possible factors such as the different material properties and/or histological architecture of the cartilages (Froimson et al., 1997). We have shown that differences exist between healthy feline patellar and femoral groove cartilages in tissue thickness, chondrocyte shape and chondrocyte volumetric fraction in both magnitude and depth distribution (Clark et al., 2003). Furthermore, under identical applied loads, changes to all of these parameters differ in magnitude and depth distribution. We hypothesized that these differential histological parameters may result in predisposing one of the surfaces to altered biosynthetic activity and/or structural damage compared to the other.

The purpose of this study was to investigate if the cartilages of the feline patellofemoral joint adapt differently to anterior cruciate ligament (ACL)-deficiency, and to elucidate where, throughout the depth of the cartilage, this adaptation begins. We wanted to systematically quantify feline patellofemoral histology in short- (4 months) and long-term (67 months) ACL-deficient knees.

METHODS

Unilateral ACL-transection was carried out on twelve skeletally mature male cats. The animals were then allowed free movement until sacrifice either 4 (n=6) or 67 (n=6) months post surgery. The experimental femur and patella were dissected free and histologically fixed using a ruthenium hexammine trichloride fixative solution (Hunziker et al., 1982). Full thickness osteochondral blocks (3mm x 1mm) were harvested from the same anatomical sites in experimental specimens. 0.5μm thick sections were cut and stained with toluidine blue for light microscopy. Cartilage thickness and chondrocyte shape, clustering, size and volumetric fraction were quantified.

RESULTS

Feline patellofemoral cartilages adapted differently to ACL-deficiency: the cartilage of the femoral grooves had little microscopic pathology compared to the patellae (Figure 1).

4 months after surgery the patellar articular cartilage from the experimental joints was thicker, contained larger chondrocytes more frequently arranged in clusters and had a larger volumetric fraction of chondrocytes compared to normal controls (Figure 1). Interestingly, the majority of these changes were only observed in the middle layer (Figure 1).

At 67 months post surgery we found significant differences in patellae cartilage compared to normal controls including increased thickness, rounded and clustered

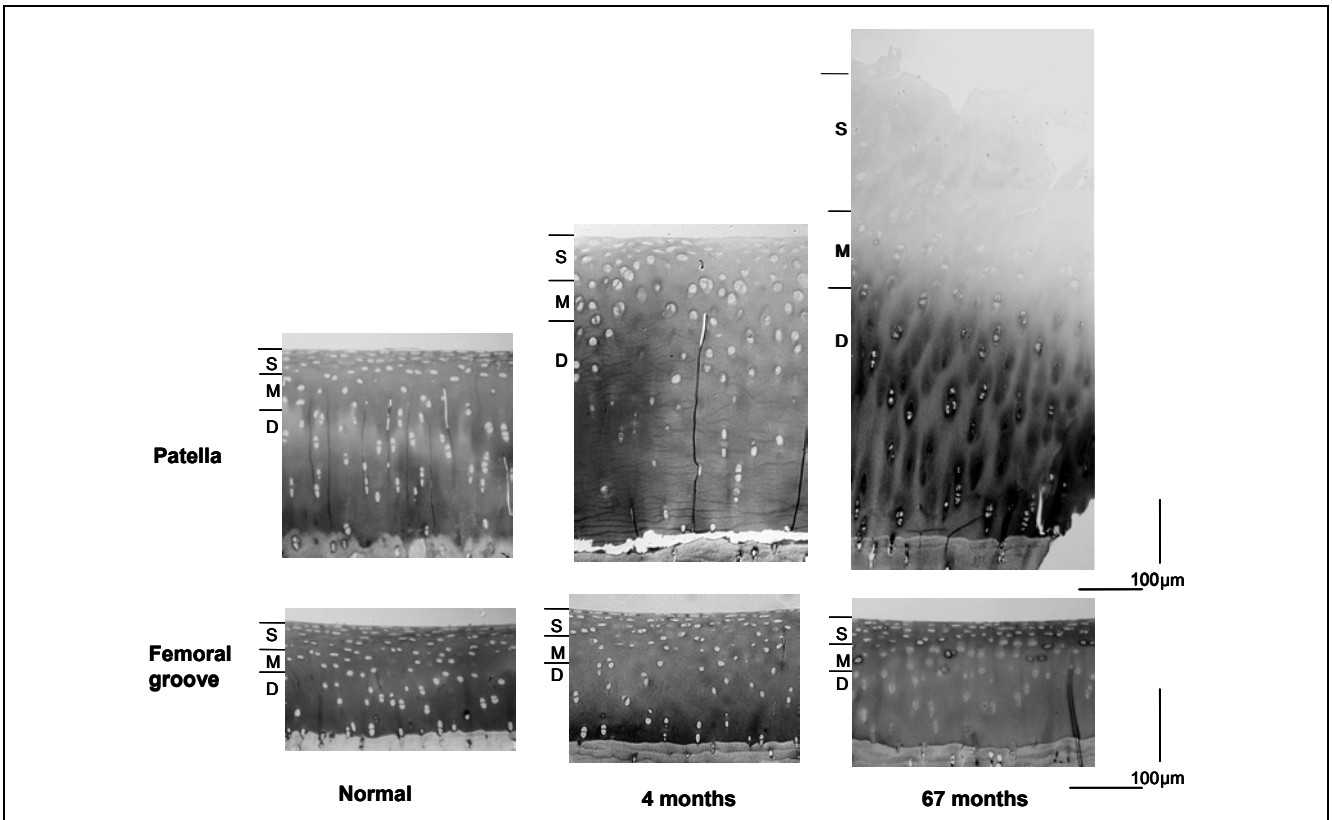

Figure 1: Comparison of normal, 4 or 67 months post ACL- transection articular cartilage. The superficial (S), middle (M) and deep (D) histological layers are indicated for each section. Sections taken from the feline patella and femoral groove, 0.5µm thick, stained with toluidine blue and photographed at 100x magnification.

superficial layer chondrocytes, a fibrillated and fissured cartilage surface and uneven proteoglycan staining throughout cartilage depth (Figure 1). These changes were most apparent in the middle and superficial layers (Figure 1).

DISCUSSION

Cartilage adaptation to ACL-deficiency was heterogeneous within the patellofemoral joint: the femoral groove cartilage had little pathology compared to the patellae. We hypothesize that this is due, in part, to the differential histological parameters (see intro) of the surfaces in healthy cartilage. Furthermore, patellar cartilage adaptation was first apparent in the middle layer 4 months after surgery. We have shown that the largest tissue strains and changes to chondrocyte volume and shape occur in the middle layer of healthy patellar tissue under static load (Clark et al., 2003). Furthermore, the middle layer of patellar cartilage contains the largest and most spherical chondrocytes throughout tissue depth. We speculate that these properties of middle layer patellar tissue may enable it mechanically and/or biologically to respond most quickly to changes that occur in the patellofemoral joint with ACL-deficiency.

REFERENCES

Clark A et al. (2003) *J. Biomech.* 36, 553.
Froimson M et al. (1997) *OA. Cart.* 5, 377.
Hjelle K et al. (2002) *J. Arthro. Surg.* 18, 730.
Hunziker E et al. (1982) *J. Ultrastructure Res.* 81, 1.
Meachim G and Emery I (1974) *Ann. Rheum. Dis.* 33, 39.

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