Study of the Effect of Excessive Tibial Plateau Angle on Degenerative Changes of Canine Cranial Cruciate Ligament

Abstract of the Doctoral Thesis

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Canine cranial cruciate ligament rupture (CrCLR) is a very common orthopaedic disease of the canine stifle. Excessive tibial plateau angle (eTPA), which increases the tensile force in the cranial cruciate ligament (CrCL) is recognized as one of the risk factors of CrCLR. To date, there are no known studies investigating the relationship between the eTPA and degenerative changes in the CrCL. The objectives of this study were to generate an animal model for eTPA and to evaluate the degenerative changes in CrCLs in dogs with eTPA, including changes in the ECM components (type I [COLI], type II [COLII], and type III collagen [COLIII]) and the expression of sry-type HMG box 9 (SOX9) with chondroid metaplasia. We generated an animal model of eTPA and evaluate degenerative changes of the CrCL and the caudal cruciate ligament (CaCL).

We generated an animal model of eTPA and evaluate degenerative changes of the CrCL and the caudal cruciate ligament (CaCL). Seven mature female Beagles were included. Cylindrical osteotomy was performed bilaterally in the proximal tibia. The TPA was increased to approximately 40° in the left tibia (eTPA group) and remained unchanged in the right tibia (control group). The dogs were subjected to exercise stress beginning 3 months postoperatively and were euthanized 12 months postoperatively, and the CrCLs and CaCLs were collected. All specimens were stained with haematoxylin and eosin (HE) to assess the cell morphology and subjected to immunostaining to evaluate COLI, COLII, and COLIII, and the SOX9 expression. Peak vertical force (PVF) was assessed in each dog preoperatively and every odd numbered month postoperatively using force plate analysis. There was no difference between the eTPA group and the control group in the postoperative PVF. Macroscopic CrCL injury was absent in 6 dogs, but was present in one in the eTPA group, which was excluded from the analysis. The cell density decreased and the percentage of round cells increased in the eTPA group. In the eTPA group, COLII, COLIII, and SOX9 expression was significantly increased, and COLI deposition was decreased and the expression in the cytoplasma of the ligamentocytes increased compared to the control group. Moreover there was no difference between the eTPA group and the control group in the CaCL.

This study showed that the expression of the SOX9 increases in the CrCL of the stifle with the eTPA.

Then we confirmed that the tensile force in the CrCL increases with the eTPA in our ex vivo study. It is reported that tensile force increasing promotes the expression of the SOX9. Therefore it is suggested that eTPA may increase the expression of the SOX9, promote the synthesis of cartilage matrix such as COLII and COLIII, which lead to the tensile strength decrease. The collagen composition may change, which COLI, the principal tensile-resistant fiber decrease and COLIII tending to form thin fibers, and chondroid metaplasia may be promoted in the CrCL of stifles with eTPA, and these changes were not observed in the CaCL. Because COLI is the principle tensile resistant fibre, and cartilage-like tissue is more vulnerable to disruption under normal tensile forces, eTPA may be one risk factor for CrCLR.