Post-traumatic fetlock osteoarthritis in Standardbred racehorses : Is it the culprit of long-term progression to degenerative joint disease?

Bertuglia A[1], Pagliara E[1], Ricci A[1], Brkljaca Bottegaro N[2].

[1]Department Veterinary Science, Turin, Italy, [2]Faculty of Veterinary Medicine Clinics, Zagreb, Croatia.

Introduction:

Osteoarthritis (OA) begins many years before structural changes are detectable, since DJD spans over a lifetime in man. In racehorses progression from post-traumatic OA stage to DJD seems much shorter. The purpose of this study was to assess changes in inflammatory and structural biomarkers in serum (S) and synovial fluid (SF) in a cohort of STBRs diagnosed with post-traumatic fetlock OA over the racing career of the animals. We hypothesised that biomarkers assay could demonstrate the progression of degenerative status in the joints after post-traumatic OA, better than clinical and radiographic assessment.

Materials and methods:

Thirty-seven STBRs between 18-24 months of age, diagnosed with fetlock joint OA as a cause of lameness, were included in the study. Horses were observed over a period of 5-years of racing activity. Six sound, age-matched STBRs were used as healthy controls. Blood sampling, SF sampling from affected joints, lameness and radiologic examinations were performed at the first lameness episode and repeated yearly. Samples were processed for IL-1ß, IL-6, TNF-α, COMP and CTX-II using ELISA kits. A semi-quantitative radiographic-based score was employed to define the severity of OA. A mixed linear model analysis was employed for multiple comparisons between C and OA groups over the timeframe of the study.

Results:

Twenty-five horses fulfilled the study requirements. Significant differences between C and OA were detected for all the biomarkers in SF at T4 and T5. In S those differences were not recorded for IL-6. Concentrations of inflammatory cytokines in OA decreased at T2, followed by an increase with time in SF and S, and attaining a significant difference to baseline at T4 (IL1ß, TNF-α) and T5 (IL1ß, IL-6, TNF-α). Structural biomarkers showed an increasing trend during consecutive assessments with a significant difference to T1 in S, and SF at T3, T4 and T5. A significant progression over time was evident for radiographic score in the OA group, but not for clinical assessment. In a multivariate analysis only TNF-α values in SF significantly and independently contributed in explaining radiological changes at T4 and T5.

Discussion/Conclusions:

Both inflammatory and structural biomarkers were increased in the S and SF of OA-affected STBRs, demonstrating that long-term increased concentration of biomarkers is a disease-effect. Biomarkers could predict structural progression of traumatic OA with a better accuracy than clinical and radiological assessment. TNF- in the SF was correlated with radiological changes, raising the suggestion that cartilage degradation is up regulated by inflammatory stimuli. In conclusion, this study underlines that early post-traumatic fetlock OA and the DJD status are closely interdependent processes.