Micro-CT quantification of subchondral endplate changes in intervertebral disc degeneration

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Introduction: Subchondral bone changes in Osteoarthritis (OA) have been studied intensively in the past decades1. Subchondral bone remodeling has been associated with OA progression due to compromised nutrient and gas diffusion and reduced structural support of the overlaying cartilage2. Also the intervertebral disc (IVD) is dependent on nutrient provision through a cartilage layer with underlying subchondral bone. Moreover, IVD degeneration shows many similarities to OA. However, little is known about the subchondral bone changes in IVD degeneration. Increased subchondral bone thickness, radiologically seen as sclerosis of the endplates, has been suggested to compromise the diffusion from the vertebra to the IVD and negatively influence the nutritional state of the IVD3. The aim of this study is to determine the subchondral bone changes during IVD degeneration by micro-CT techniques.

Materials and Methods: Twenty-seven L4-L5 IVDs were obtained at autopsy within 48h after death of the patients, average age 53.7 yrs, range 33-90.8 yrs. Midsagittal slices of IVDs were stored in 4% formalin and scored according the Thompson degeneration score by three observers4. IVDs slices were subsequently scanned using a micro-computed tomography (micro-CT) scanner (Skyscan 1076, Skyscan, Kontich, Belgium) at a voxel size of 17.8 μm. Per scan 12 standardized cylindrical regions of interest (ROI) were selected with CTAnalyser, (Skyscan). Six ROIs contained the bony endplate and trabecular (endplate ROIs) and 6 ROIs were selected 1.3 mm (75 pixels) below or above the endplate which only contained trabeculae (trabecular ROIs). Osteophytes and calcifications of the growth plate were not included in the ROIs. Four ROIs were selected at the anterior side of each IVD, four in the center of the IVD and four at the posterior side (Fig 1).

Bone in the ROIs was identified by automated thresholding segmentation5. With 3D-Calculator6 bone volume as percentage of the total volume (BV/TV) of the ROI, structure model index (SMI), trabecular connectivity (CON) and Trabecular Thickness (TbTh) were determined. It should be noted that the endplate itself is part of the endplate ROI and as such the parameters might reflect exclusive effects of the endplate itself.

Results: Macroscopical grading according to the Thompson score resulted in 5 grade I, 5 grade II, 7 grade III, 6 grade IV and 4 grade V IVDs. BV/TV in the endplate ROIs is significantly increased in grade II, III and V compared to grade I (p=0.003, p=0.004 and p=0.019 respectively), while the trabecular ROIs showed a significant decrease in BV/TV in grade III and IV compared to grade II (p=0.022 and p=0.001 respectively) (Fig 2). The TbTh in the endplate ROIs showed a significant increase in grade II, III, and V compared to grade I (p=0.001 from all three comparisons). In the trabecular ROIs no differences in TbTh were observed (Fig 3).

Neither SMI nor CON showed statistically significant differences between degeneration grades.

In advanced stages of IVD degeneration higher BV/TV were found in the anterior and posterior endplate ROIs when compared to the center ROI. In grade III a significant higher BV/TV in the anterior endplate ROI was found when compared to the center ROI (p<0.01). In grade IV BV/TV of both anterior and posterior endplate ROIs were significantly higher (p=0.013 and p=0.030 respectively). In grade V a significant higher BV/TV in the anterior endplate ROI was found when compared to the center ROI (p=0.023). In grade IV BV/TV of both anterior and posterior endplate ROIs were significantly higher (p=0.013 and p=0.030 respectively). In grade V a significant higher BV/TV was found in the posterior endplate ROI (p=0.039).

Discussion: This study clearly shows that there are subchondral endplate changes in different stages of IVD degeneration. Since the Trabecular thickness increase is only found in the endplate ROI and not in the trabecular ROI (Fig 3) the thickness increase can be fully attributed to the endplate only. Furthermore we find a loss of bone volume fraction (BV/TV) in the trabecular ROIs vs. an increase in the endplate ROI (Fig 2), again showing indirectly that the endplate itself increased drastically in the different stages of IVD degeneration. The increase in endplate thickness in the advanced stages of IVD degeneration (grade III, IV and V) is especially seen in the anterior and posterior edges of the vertebrae. Biomechanical loading of the anterior and posterior edges of the vertebrae is increased in IVD degeneration and could very well be responsible for the increase in bone volume in these regions of the IVD7. The increase in endplate thickness most likely negatively influences the diffusion and nutrition of the IVD8. However, it is not clear if the endplate plays a role in the etiology of IVD degeneration or is a result of changed biomechanics in a degenerating IVD.