

Comparison of Lumbar Artery Anatomy and Degenerative Disc Disease in Healthy controls and patients with low back pain

Combination of dynamic, contrast enhanced perfusion and MR-Angiogram in the lumbar spine using a 3Tesla MRI

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Purpose:

Every year an estimate of 50 to 91 billion dollars (1998) is spent on health related expenditures caused by back pain in the United States [1,2], with degenerative disc disease (DDD) as the major cause. The viability of the chondrocytes in the nucleus pulposus (NP) of the intervertebral disc (IVD) is crucially dependent on oxygen and glucose delivery for proper synthesis and maintenance of the extracellular matrix (ECM) [3,4]. The (endplate) subchondral blood flow is a product of the vertebral body arterial network that arises from the segmental lumbar arteries. Not many studies have been conducted to show the arterial anatomy of the lumbar region with most being performed at autopsy [5]. The disruption of blood flow to the subchondral endplate has been described as a possible cause for DDD [6,7,8]. The use of high field strength MR would allow a fast data acquisition with high SNR; and as such high-resolution MR-Angiography images can be obtained to allow for evaluation of vessels up to 1 mm in diameter. In this study we evaluated the blood supply to the vertebral bodies via the segmental arteries in correlation with DDD classified via high resolution T2 images and T2 mapping of the intervertebral disc in patients with a history of back pain, compared to healthy controls.

Methods:

Thirty-one subjects were enrolled into this study. The First group contained 8 healthy subjects (19–26 yrs.) without any history of back pain or back injury. The second group contained 23 subjects (24–58 yrs.) with a degenerative disc disease and/or a low back pain. These subjects were referred either by spine surgeons at our institution or by word of mouth. All patients had lab results including creatinine and GFR measurements prior to their MRI. IRB consent was signed. All images were acquired with a Philips Achieva 3T system using Philips' 6-element SENSE torso coil. T1- and T2-weighted anatomical scans covering the spinal column from L1 to S1 were performed in the sagittal plane using a TSE sequence (TR/TE=900ms/10ms and TR/TE=2940/120, respectively), followed by DCE-MRI (Omniscan®, 0.05 mmol/kg). Then the contrast-enhanced MR-angiography was performed using the same dosage. The MRA sequence was acquired from a single, 50mm thick coronal slice using the FFE sequence in dynamic mode, to collect real-time images every 0.5s. As soon as the contrast arrived in the descending aorta, the actual 3D angiographic scan was started by the operator immediately (TR/TE=5.1/1.78ms, voxel size=0.8x0.8x1.5mm³, SENSE factor=4, and 50 coronal slices). Segmental vessels on MRA were graded as patent, stenotic or occluded. Discs were graded as grade 0 for a high and homogenous signal-intensity on T2 image of the NP, grade I for mildly to moderately inhomogeneous signal-intensity of the NP, and grade II for severely decreased and inhomogeneous signal-intensity of the NP.

Results:

The healthy control group showed grade 0 discs on all IVD levels from L2-S1. They also showed bilaterally patent segmental vessels from L2-L4 level. The L5 segmental vessels are not consistently described in the literature and are not seen in every human. However 11 of the 31 subjects showed at least 1 segmental L5 artery which was at least stenotic, mostly in the patent group. 5 of the subjects with segmental L5 arteries were seen among the 8 controls (63%). 6 were seen among the 23 patients (26%). 12 subjects showed at least one grade I or II disc at one of the levels L2-S1 in combination with at least one stenotic or occluded segmental artery. 10 subjects showed at least one grade I or II disc while their L2-L4 segmental arteries were bilaterally patent. 1 subject showed grade 0 discs at all levels while having one occluded segmental artery. Table 1 summarizes the results of disc grade and segmental artery status.

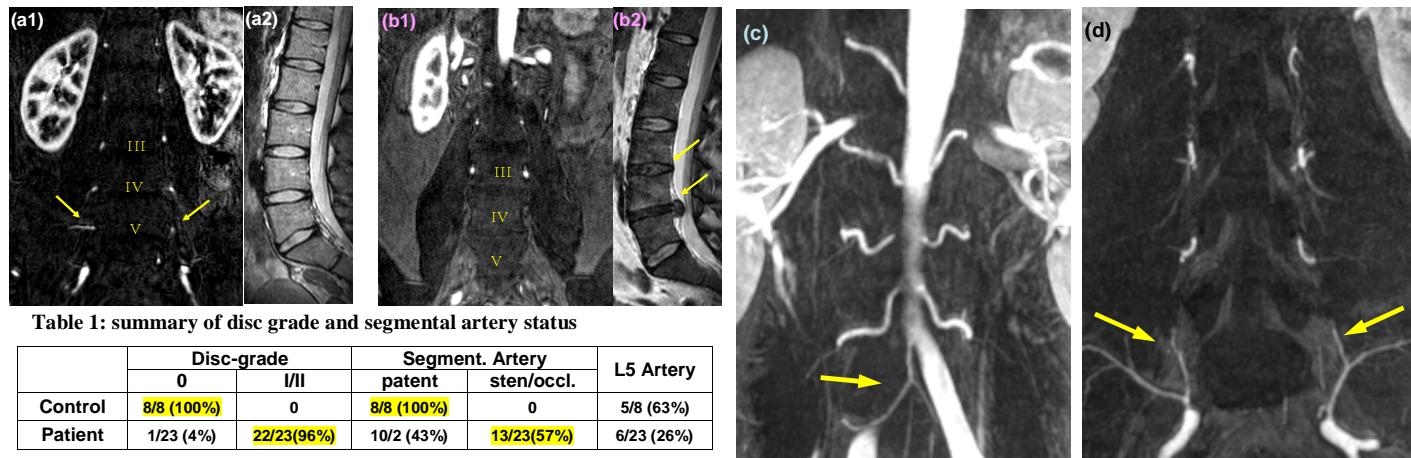


Figure 1. (a1) shows a control with bilateral patent segm. arteries from L1-L5 and corresponding T2 weighted image with healthy IVD. (b1) bilaterally occluded L4-and absent L5 segm. arteries and grade I/II IVD at L3/L4 and L4/L5 respectively. (c) Contrast-enhanced MIP showing the origin of the L5-segm. artery from the middle sacral or A. iliocalis (d)

Discussion:

We demonstrated a method for studying the vascular anatomy and dynamics of the spine in one scanning session. Spinal arterial anatomy is not fully known, especially the role of the L5 segmental arteries. The role that segmental vessel stenosis plays in the development of degenerative disc disease is of interest. In addition, with at least 40% of all degenerative discs at the L5-S1 level, the lumbosacral vascular anatomy may be a contributor. Kauppila et al. described a correlation of DDD and back pain with calcification of the abdominal aorta [6]. Our study demonstrated that vascular disease is seen more frequently with age and the presence of degenerative disc disease. The origin of the L5 segmental arteries in our subjects has consistently been either the middle sacral artery which derives from the bifurcation area of the aorta, or the proximal section of the lumbar arteries. It is not known whether lack of L5 vertebral vessels provides a predisposition to L5-S1 degenerative disease. There are also regions that show DDD without apparent stenosis or occlusion in the neighboring segmental vessels. Bibby et al. described a higher vulnerability of the IVD to nutrient supply due to its avascular nature [9]. Considering this fact it is possible that the IVD would show degeneration sooner than MRA would show an apparent stenosis. We hope to gain a better understanding of this finding by combining with the perfusion study in the future.

References: [1] Errico T. Clin Orthop relat Res 2005; 435:106-117. [2] Luo X et al. Spine. 2003; vol. 29, no. 1:79-86. [3] Grunhagen T, et al. JBJS 2006; 88:30-35. [4] Horner H. et al. Spine 2001; 26, 23: 2543-2549. [5] Caglar et al. Surg Neurol 2004; 61:29-33. [6] Kauppila L. et al. Spine 1997; 22,14: 1642-1649. [7] Wallace A. et al. Spine 1994 ; 19,12: 1324-1328. [8] Iwahashi M. et al. Spine 2002 ; 27 (13) 1396-1401. [9] Bibby et al. Joint Bone Spine 2001, 68 : 537-42.

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