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ULTRASHORT TIME-TO-ECHO MRI OF THE CARTILAGENOUS ENDPLATE AND RELATIONSHIP TO DEGENERATIVE DISC DISEASE AND SCHMORL'S NODES

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Introduction: The vertebral endplate is composed of an inner bony and outer cartilaginous endplates (CEP). The CEP supplies the intervertebral disc (IVD) with nutrients and metabolites, and is instrumental for metabolism, exchange of waste products and biomechanics of the disc [1]. Lumbar disc degeneration on MRI is a risk factor for the development of low back pain [2]. It has been previously hypothesized that changes in disc mechanics may be initiated by damage to the endplate [3, 4]. Similarly, CEP defects may be involved in the formation of Schmorl's nodes (SNs) (i.e. invagination of IVD material into the adjacent endplates) [5], which associated with severity of lumbar disc degeneration [6]. The ultrashort time-to-echo (UTE) MRI is an imaging technique that enables improved visualization of tissues with short T2 relaxation that appear dark in signal on conventional T2-weighted (T2W) imaging. By employing this technique in the lumbar spine, we believe that the CEP, which appears hypointense in T2W MRI, may be observed as continuous high-signal and may thus be differentiated from the bony endplate. Although cadaveric studies have addressed the feasibility of UTE in assessing the CEP [7], studies addressing such technology in live human subjects have not been addressed. We hypothesize that the early diagnosis of CEP defects may provide useful information for understanding the pathogenesis of disc degeneration and SNs in humans. Therefore, the objectives of this study were two-fold: 1) to assess the feasibility in detecting the presence of CEP defects in human subjects and 2) to assess the presence of CEP in degenerated and non-degenerated vertebral lumbar levels, and in the setting of SNs.

Materials and Methods: 18 subjects (10 males, 8 females; mean age=37.3 years; range=17-61 years, ±SD=13.7 years) were recruited to undergo MRI scans following approval from the Institutional Review Board. 9 subjects were recruited from the ongoing DDD Cohort Study, with known presence of disc degeneration or SNs; and 9 subjects were volunteers. Each subject was imaged on a 3T Achieva scanner (Philip Healthcare, Best, The Netherlands). 3D UTE images, with parameters: FOV=240×240×97.2 mm^3, TR/TE=4.8ms/0.14ms, acquisition voxel size=0.6×0.6×2.4 mm^3, reconstructed voxel size=0.6×0.6×1.2 mm^3 were obtained with Torso XL coil with radial trajectory; and conventional 3D T2W images with parameters: FOV=240×240×61.2 mm^3, TR/TE =2000ms/120ms, acquisition voxel size=0.6×1.03×2.4 mm^3, reconstructed voxel size=0.6×0.6×1.2 mm^3, were obtained for reference and comparison. 108 IVDs from T12/L1 to L5/S1 were evaluated. All IVDs were graded for disc degeneration according to the Schneiderman classification based on 3D T2W sagittal images and scores at each level were summated to illustrate the global severity of disc degeneration. All levels were evaluated for the presence of SNs based on 3D T2W sagittal images, defined as localised vertebral endplate indentations at either the rostral or caudal endplates or both. Based on the UTE images, CEP defects were defined as discontinuity of high-signal over 4 consecutive slices (4.8mm). Images were independently assessed by two raters. Assessment of associations between CEP defects and DDD and SNs was performed.

Results: In the UTE images, CEP defects (Fig.1A, D and E) were found in 65 of 108 (60.2%) vertebral levels, which were not noted on conventional T2W MRI. Although the overall presence of CEP defects on UTE were not found to be level dependent (p=0.633), younger subjects exhibited CEP defects and this was largely significant in the upper and mid-lumbar regions (T12/L1 p<0.001, L1/2 p=0.001, L2/3 p=0.023, L3/4 p=0.017, L4/5 p=0.589, L5/S1 p=0.935). In adjusted multivariate analyses, CEP was significantly associated with younger individuals (OR: 0.94, 95% CI: 0.91 – 0.98, p<0.001), SNs (OR: 8.90, 95% CI: 2.27 – 34.85, p=0.002), the presence (OR: 4.12, 95% CI: 1.18 – 14.38, p=0.027) and severity (OR: 2.18, 95% CI: 1.11 – 4.26, p=0.023) of disc degeneration, and was marginally associated with males (OR: 2.29, 95%CI: 0.970 – 5.40, p=0.059).

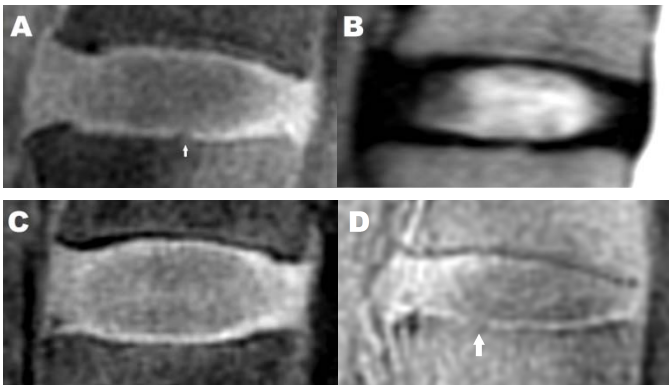

Fig.1. (A) UTE image of an L3/4 IVD level, showing a CEP defect (arrow), which was observed as discontinuity of signal over 4 consecutive slices. (B) Corresponding T2W image at the same resolution. (C) UTE image of an L2/3 IVD level with normal CEP. (D) UTE image of T12/L1 IVD level with CEP defect in caudal side (E) UTE image of an L2/3 IVD level with SNs demonstrating continuous CEP signal in the rostral SN (black arrows- bony endplate; white arrows- continuous CEP), but CEP defects in the caudal SN (blue arrow- discontinuous CEP signal).

Discussion: Our study is the first to assess UTE in lumbar spine in vivo in human subjects and illustrates its feasibility in detecting CEP defects. We noted that CEP defects occurred more frequently in younger individuals, which may suggest that CEP changes occur early on and may potentially heal in time. Our study further noted that CEP defects were significantly associated with the presence and severity of disc degeneration as well as the presence of SNs. Although further studies are needed to better characterize the effects of SN variants upon CEP defects, the current study broadens the understanding of the role of endplate defects and the association with degenerative disc changes.

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