

MR TAGGING BASED CARDIAC-INDUCED LIVER DEFORMATION ANALYSIS IN MURINE MODEL OF NON-ALCOHOLIC FATTY LIVER DISEASE

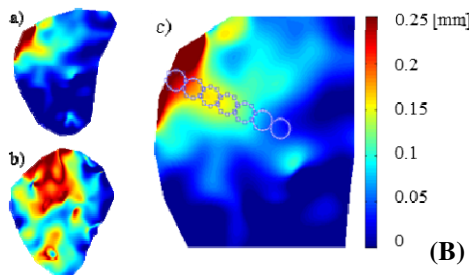
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Introduction: The assessment of tissue stiffness becomes a routine diagnostic method in chronic liver diseases and is usually performed using transient elastography to determine the degree of fibrosis [1, 2]. However, by the anatomical arrangement of organs the liver is periodically and reversibly deformed by heart during consecutive cardiac cycles. This deformation is propagated from the heart-liver point of osculation through the organ and the range of this strain may vary regarding the organ condition. The aim of this work was to find abnormalities in cardiac-induced liver deformation in the murine model of non-alcoholic fatty liver disease (NAFLD) using MR Tagging and to verify the method feasibility.

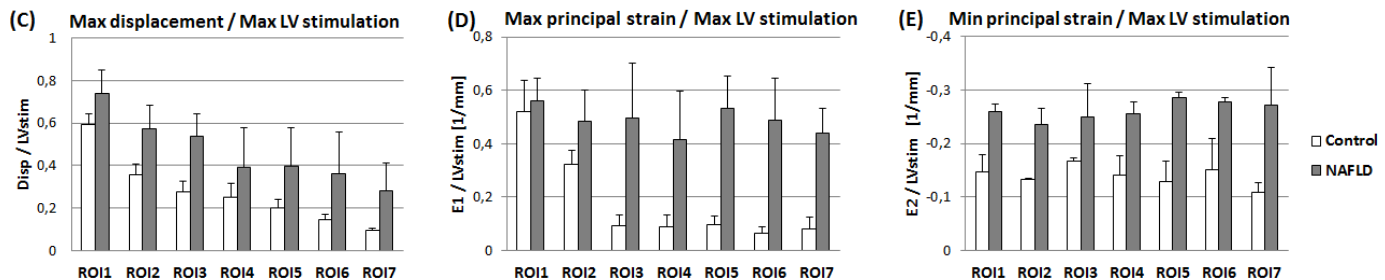
Subjects and Methods: Two groups of 6-month C57BL/6J mice were examined: control, standard AIN-93G diet (n=3); NAFLD, 45% kcal high-fat diet (n=3). The animals underwent MRI procedure: ECG-gated FLASH-cine sequence with SPAMM tagging module (9.4T Bruker BioSpec, Germany; TE=1.5ms, TR=8.5ms, 192x192 data matrix, FOV 30x30mm², 1mm slice thickness, tagging grid with 0.2 mm tag line, 0.6 mm tag span, 20 frames/cardiac cycle, sagittal projection), Figure A.

Peak-combination HARP algorithm [3, 4] was self-implemented in Matlab (Mathworks, USA) and employed for image analysis to compute internal tissue displacements Disp (mm), principal strains (%): first E₁ (stretching) and second E₂ (compression). The maximum deformation was assessed within 7 regions of interest (ROI) of ~0.6mm diameter, placed along the direction of motion (Figure B. c). The LV declinations (LVstim, mm) were computed over the whole cardiac cycle and the values of Disp, E₁ and E₂, were normalized to maximum LV stimulation.



Results: The example images of inter-tissue displacement maps are presented in Figure B. The deformation wave spreading from the left lobe of the liver seemed to be symmetric and regular in the control group (Fig. B. a) while for the NAFLD group the map represent inhomogeneity of motion (Fig. B. b).

Figures (C), (D) and (E) show results for Disp, E₁ and E₂, respectively, divided by LVstim (mean values \pm SD). While going deeper inside the organ the deformation fades for the controls, in the NAFLD strain is sustained within remote regions. Moreover, the tissue stretching E₁ near the point of osculation (ROI1) seem to be preserved in NAFLD, but the deformation wave is further propagated deeper. There are noticeable increase in tissue compression E₂ within all the assessed regions for the NAFLD group compared to healthy subjects.



Discussion: Despite small study population good consistency within controls points to good reproducibility of the MR Tagging-based deformation analysis. The method revealed liver stiffness changes in NAFLD mice. The increased regional displacements and tissue compression in response to cardiac stimulation may reflect structural tissue changes caused by increased fat deposition [5]. At the same time the preserved maximum stretch suggests non-fibrotic stage of the pathology [2, 5]. The results may suggest the routine is helpful not only for liver fibrosis assessment but maybe for evaluation of fat deposition degree.

Conclusions: The preliminary results of the study show potential usability of cardiac-induced liver deformation assessment by MR Tagging for differentiating NAFLD versus healthy organ.

References:

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