

Quantitative liver MRI combining Phase Contrast imaging, Elastography, and DWI: assessment of test-retest and post-prandial effect. Prospective study at 3T.

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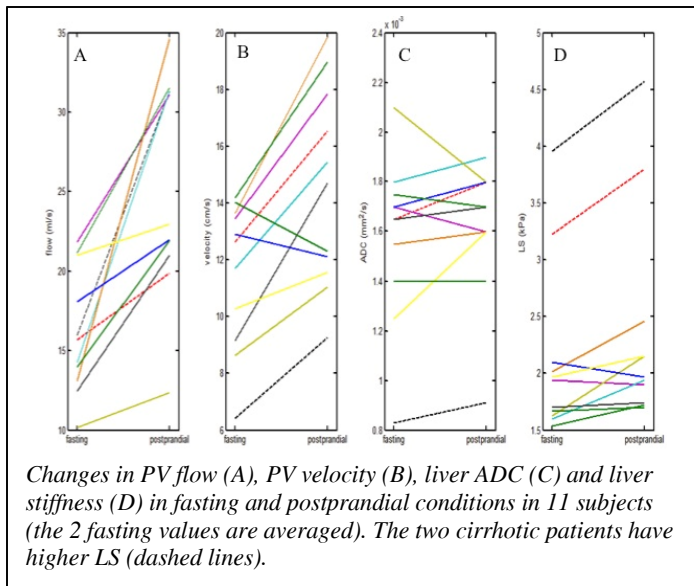
Purpose: Techniques such as MR Elastography (MRE), phase contrast (PC) and diffusion-weighted imaging (DWI) have potential for non-invasive detection of liver fibrosis, cirrhosis and portal hypertension associated with chronic liver disease. Since portal flow and liver stiffness (LS) may be altered by food intake [1, 2], changes in LS, portal vein (PV) flow, PV velocity and liver ADC (which is affected by flow) might be observed and may lead to decreased reproducibility. This prospective study quantifies reproducibility (in fasting conditions) and post-prandial changes in PV flow/velocity, LS, and liver ADC at 3T.

Methods: 9 healthy volunteers and 2 patients with HCV and cirrhosis (M/F 8/3, mean age 35 y) were enrolled in this prospective IRB approved study. All subjects underwent 3T MRI (MR750, GE Healthcare), including 2D PC (pulse triggered with $V_{ENC}=50$ cm/s, slice perpendicular to the portal vein), axial SS EPI DWI (free breathing, 16 b-values from 0 to 800 mm^2/s) and MRE (4 slices through the liver). All subjects were initially scanned twice after 6 hours of fasting to assess reproducibility of each technique (subjects were removed from the scanner and re-scanned). The subjects were then scanned again in postprandial conditions, 20 min. after a 700 Kcal liquid meal (Ensure Plus). To quantify PV flow and velocity, a ROI was drawn in the PV on PC images. Mean LS and liver ADC were obtained by placing a ROI in the right hepatic lobe on LS maps and diffusion images. A mono-exponential fitting was used to compute the ADC. The coefficients of variation (CV) for PV flow, PV velocity, LS and liver ADC were computed for the two scans in fasting state. Wilcoxon paired tests were performed to assess differences in these metrics before and after caloric intake (average from the 2 fasting scans was used for comparison).

	PV Flow	PV Velocity	ADC	LS
Fasting #1	16.1 ± 3.9	11.6 ± 3.0	1.5 ± 0.4	2.1 ± 0.8
Fasting #2	16.3 ± 4.5	11.5 ± 2.3	1.6 ± 0.3	2.1 ± 0.7
CV	17.4%	11.7%	10.1%	4.5%
Postprandial	25.5 ± 6.9	14.5 ± 3.5	1.6 ± 0.3	2.4 ± 0.9
p*	0.001	0.007	0.3	0.01

PV flow, PV velocity, liver ADC and LS (liver stiffness) for all subjects (mean ± SD) in fasting and post-prandial states. CV is calculated to assess reproducibility in fasting conditions. PV Flow (ml/s), PV Velocity (cm/s), ADC ($\times 10^{-3}$ mm^2/s), LS (kPa). *: comparison of average fasting values and postprandial values.

Results: PV flow, PV velocity, liver ADC and LS showed good to excellent reproducibility in fasting state, with CVs ranging from 4.5%-17.4% (Table). PV flow, PV velocity and LS were all significantly higher in postprandial state (Table, Fig.). The two cirrhotic patients showed an average increase of 16.6% in LS (4.0 to 4.6 kPa for 1st patient, and 3.2 to 3.8 kPa for 2nd patient), while the healthy volunteers had an average increase of 10.3% in LS. Average increase in PV flow and PV velocity was 61.5% and 27.5%, respectively. Liver ADC did not significantly change before and after caloric intake.



Discussion: We observed that caloric intake increases PV flow/velocity (as expected and shown previously [1]) and LS (as shown [2] recently) with no effect on liver ADC. The optimal cut-off stiffness value of 2.93 kPa presented by Yin et al [3] to detect patients with chronic liver disease is able to fully differentiate patients from healthy volunteers. A larger number of patients with liver fibrosis is needed to assess the clinical significance of PV flow/velocity and LS changes after caloric intake.

Conclusion: These results indicate that caloric intake is a factor to consider in interpreting PC-based PV flow/velocity and MRE-based hepatic stiffness measurements. The difference in LS between fasting and postprandial states is elevated in patients with liver disease and the diagnostic significance of this observation needs to be verified further.

References

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 3. Yin, M., et al.. Clin Gastroenterol Hepatol, 2007. 5(10): 1207-1213
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