Evaluation of the changes in renal artery flow in patients with cirrhotic liver disease pre and post meal in comparison to healthy subjects

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Purpose: It is well known that cirrhotic liver disease influences vascular flow in the hepatic as well as mesenteric vasculature [1-3]. The aim of this study was to investigate to what extent renal flow is altered by hepatic disease and to what extent renal flow is altered by a meal challenge in patients with portal hypertension, compared to healthy subjects.

Methods: In this IRB-approved and HIPAA-compliant study, 10 patients (55.1±13.2years, 80.8±21.6kg) with portal hypertension, evidenced by the presence of varices and splenomegaly, as well as 6 healthy subjects (32.2±10.1years, 85.7±8.7kg) were imaged after written informed consent was obtained. Pre-meal studies were conducted after at least 5 hours of fasting. Following the pre-meal scan, subjects ingested 574mL EnSure Plus® (Abbott Laboratories, Columbus, OH; 700cal, 28% from fat, 57% from carbohydrates) orally. The post-meal acquisition was started 20 minutes after the meal.

Studies were conducted on a clinical 3T scanner (Discovery MR 750, GE Healthcare, Waukesha, WI) with a 32-channel body coil (NeoCoil, Pewaukee, WI). 4D velocity mapping was achieved using a radially undersampled phase contrast acquisition (5-point PC-VIPR) with increased velocity sensitivity performance and comprehensive coverage of the upper abdomen [4,5]. Radial 4D flow MRI image parameters included: imaging volume: 32x32x24cm, 1.3mm acquired isotropic spatial resolution, TR/TE=6.4/2.2ms. All subjects received 0.03mmol/kg of gadofosveset trisodium (Lantheus, N. Billerica, MA) to maximize SNR performance, prior to the pre-meal scan. Pre- and post meal challenge 4D flow MRI was adjusted for optimal imaging conditions and differed in the venc: (pre=100cm/s, post=120cm/s) and flip angle (pre=16°, post=14°) (figure 1). Cut-planes for flow measurements were placed at 5 predefined locations (supra-celiac aorta (SCAo), supra-renal aorta (SRAo) right and left renal artery (RRA, LRA) and infra-renal aorta (IRAo)). Measurements were done with EnSight Software (CEI, Apex, NC).

Results: Blood flow in the SC Ao after meal significantly (p<0.05) increased in patients by 10% (3.46 to 3.82 L/min) as well as 34% (3.53 to 4.75) in healthy subjects. In contrast, flow in the SRAo after a meal increased in patients on average by 45% but decreased by 7% in healthy subjects. Renal flow significantly increased in patients by 37% (0.53 to 0.66 L/min) whereas it decreased by 23% (0.59 to 0.40) in the healthy control group (figure 2).

Discussion: In this work, we demonstrated that a meal influences not only hepatic and mesenteric but also renal flow under healthy conditions as well as in patients suffering from cirrhotic liver disease. However, results are different in both groups. A meal triggers a higher cardiac output and thus an increased flow in the supra-celiac aorta. In healthy subjects a meal causes a significant increase in blood flow to the liver and gut. This increase is even higher than the increase in flow in the SC Ao and thus the flow in the SRAo and the renal arteries decreases. In patients with cirrhotic liver disease the resistance in the liver seems to be too high to allow a significant increase in flow to the liver. Hence, the increased flow in the SC Ao passes the celiac trunk and as our results suggest the superior mesenteric artery and increases the flow to the kidneys and the IRAo.

Conclusion: Portal hypertension influences blood flow not only in the liver, but also in entire mesenteric vasculature. Further, this work suggests an unexpected influence of portal hypertension on the response of renal blood flow to a meal challenge. Additional investigation is needed to understand the influence of portal hypertension on renal blood flow with meals.