

Effect of Cirrhosis on Portal Venous Flow Reserve

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Background: Morphologic liver imaging features have been proposed as indicators of cirrhosis including surface nodularity, caudate lobe enlargement, ascites, expanded gallbladder fossa, right posterior hepatic notch, enlarged hilar periportal space, splenomegaly, varices, dilated cisterna chili and vascular changes such as small hepatic veins or recannalized umbilical vein. But in spite of all these "signs" cirrhosis often goes undiagnosed on CT/MRI scans until it reaches the point of end-stage and irreversibility. Accordingly, a physiological indicator might be able to identify changes of cirrhosis even when characteristic morphological features are not present. Since portal flow increases post-prandially, a cirrhotic liver may have less ability to increase flow following a meal, i.e. decreased flow reserve. We have been including a 2D PC portal flow measurement routinely with all liver MRIs performed for the past 5 years, which includes patient with known cirrhosis as well as potential liver donors and other patients with normal livers being imaged for incidental findings on CT and US scans. Using these data, we test the hypothesis that the portal flow is greater in patients with normal livers post-prandially compared to cirrhotic livers.

Purpose: This study compares flow in the main portal vein (ml/sec) in cirrhotic and noncirrhotic patients during fasting and postprandial states.

Materials and methods: Patients who underwent dynamic gadolinium-enhanced 3D MR imaging at 1.5 T were routinely imaged with 2D phase contrast MRA with the following parameters: TR/TE/flip= 24.0 ms/7.3 ms/30, matrix = 512 x 512, slice thickness – 5mm, venc = 30 cm. Volume flow in the portal vein was measured using CV flow analysis software (Medis, Netherlands) by defining a region of interest encompassing the portal vein. A total of 97 patients were imaged including 67 patients with normal livers, (50 fasting, 17 post prandial) and 30 patients with biopsy proven cirrhosis (25 fasting, 5 post-prandial). Fasting status was determined by evaluating the stomach contents on axial/coronal SSFSE images as well as the size of the gallbladder, if present. Patients who had altered portal venous flow such as portal venous thrombosis or TIPS were excluded from the study.

Results: We observed that fasting, noncirrhotic and cirrhotic patients had similar mean portal venous flow velocities of -13 ml/s and -13 ml/s, respectively. However, while noncirrhotic patients had increased mean PV flow of -16 ml postprandially as expected, cirrhotic patients had decreased PV flow of -7 ml/s post-prandially.

Conclusion: Along with morphologic features of cirrhosis, decreased portal venous flow observed in postprandial, cirrhotic patients serves as a useful physiologic indicator of cirrhosis that can be easily analyzed on MRI. In this study, post-prandial status was based on observation of the stomach contents with no effort to use a standardized meal. However, in the clinical routine setting, the patient should be imaged pre and post a standardized meal e.g. sustacal.

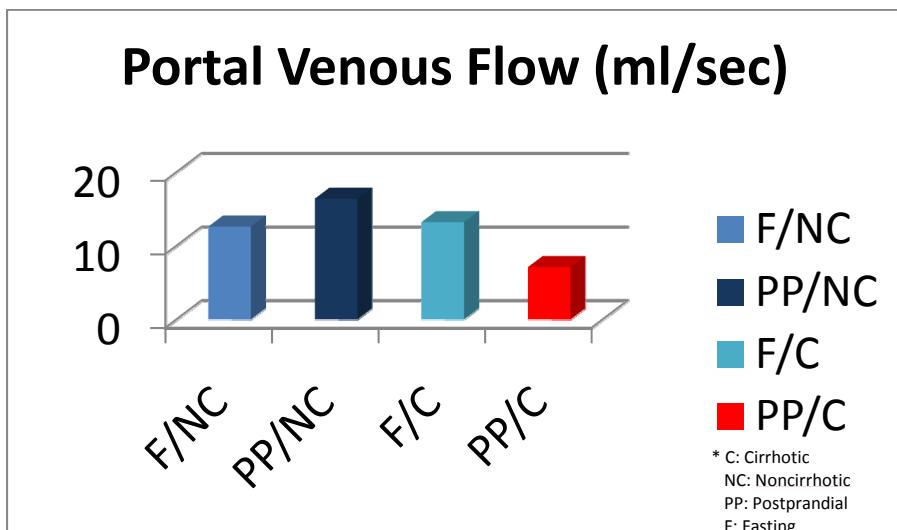


Figure 1. Graph showing decreased PV flow in cirrhotic patients post-prandially in contrast to noncirrhotic patients with increased PV flow. Fasting, noncirrhotic and cirrhotic patients had similar mean portal venous flow velocities.

Figure 2. Examples of PV flow velocity measurements on 2D Phase contrast MRA

