Reviewer’s report

**Title:** Intrahepatic blood flow assessment by Doppler ultrasonography: relationship among hepatic vein, portal vein, hepatic artery and portal pressure intraoperatively measured in patients with portal hypertension

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**Reviewer:** Annalisa Berzigotti

**Reviewer’s report:**

This paper by Zhang and colleagues was aimed at clarifying the pathophysiological factors associated with abnormalities in hepatic veins Doppler pattern in patients with cirrhosis and portal hypertension. In 60 patients requiring surgery for portal hypertension the authors assess simultaneously this Doppler parameter and portal pressure as measured directly and invasively; they also measure portal vein velocity and hepatic artery pulsatility index (HAPI) by Doppler; non-invasive measurements were also performed in a group of healthy controls.

The results are consistent with a moderate linear correlation between portal pressure and flattening of flow in the hepatic veins; similarly, portal vein velocity decreased and HAPI increased in patients with abnormal hepatic vein flow pattern. It is concluded that in patients with PHT, monophasic HV waveform indicates higher portal pressure and that it can be hypothesized that parenchymal fibrosis and fat infiltration reduce HV compliance being the cause of the abnormality of Doppler HV waveform.

The paper explores an interesting issue, which has been previously evaluated by other authors but that is still poorly understood. However, the study has some major limitations, which are listed below:

1) A main flaw of the study is the lack of data about inferior vena cava pressure and other parameters related to systemic hemodynamics. Absolute portal pressure is obviously an indicator of the severity of portal hypertension, but from a pathophysiological point of view the consequences of portal hypertension depend on the portal pressure gradient, which is calculated as the difference between portal pressure and inferior vena cava pressure (Bosch et al. Seminars Liver Diseases 2006). Therefore, the lack of IVC pressure measurement decreases the reliability of the results.

2) As the authors acknowledge, right heart catheterization or at least echocardiography should have been performed to rule-out heart valvular diseases or other conditions which can directly affect HV Doppler pattern. Moreover, the assessment of cardiac output, systemic arterial pressure and systemic vascular resistance would have given data on the presence of hyperdynamic syndrome, which could also be a determinant of abnormality of HV
Doppler flow.

3) The study population is poorly described (see table 1): data on etiology of cirrhosis, prevalence and size of gastroesophageal varices, previous episodes of bleeding from varices, ascites, … should be added to support the hypothesis that abnormalities in HV Doppler pattern depend principally from portal pressure increase. Indeed, ascites increases intrabdominal pressure which could flatten HV Doppler pattern.

4) Design of the study: the study lacks a clear hypothesis, which should be better defined.

5) Study protocol: both portal pressure and central venous pressure can be affected by drugs used in general anesthesia; the authors should carefully comment on the protocol of anesthesia used, specifying if this was the same in all patients, and in which moment PP and Doppler were performed. Were the patients ventilated? How was respiration suspended?

6) The results show that flattened HV Doppler flow pattern indicated more severe portal hypertension; however, all patients had severe portal hypertension since this was an inclusion criteria, and differences in PP between patients with normal and abnormal flow were very small (2-3 mmHg); how do the authors suggest to interpret the finding of abnormal HV Doppler pattern in a patients with known clinically significant portal hypertension? Did the patients with more flattened HV Doppler pattern show a different outcome on follow-up (decompensation, death, bleeding…), so supporting a clinical usefulness of the Doppler finding?

7) Discussion: a referral to “liver stiffness” is made in some sentences. In the methods and results no mention on direct liver stiffness measurement is made; was liver stiffness evaluated (transient elastography? ARFI? Other methods?) or is HAPI used as the only surrogate of liver stiffness? If it is so, the authors should better clarify the background for the use of Doppler impedance of hepatic artery as an indicator of liver stiffness.

8) Conclusions: Since no correlation was observed between HV Doppler flow pattern and histological findings it is difficult to accept the conclusion (see last lines of the abstract), which should be changed.

Minor comments

Histological findings might be better explained in the results section and not only in table 1; moreover, the lack of correlation between portal pressure and histological findings should be described in the results section and not in the discussion.

The paper would improve if the authors transform the semi-quantitative data of HV Doppler flow pattern into a more objective and numerical index, such as the damping index (Kim et al. Liver International 2007)
Level of interest: An article of limited interest

Quality of written English: Acceptable

Statistical review: No, the manuscript does not need to be seen by a statistician.

Declaration of competing interests:
I declare that I have no competing interests.