UTILITY OF DOPPLER ULTRASOUND PORTAL VEIN VELOCITY MEASUREMENT IN THE EVALUATION OF NON-ALCOHOLIC FATTY LIVER DISEASE: A CASE-BASED APPROACH OF THE LITERATURE

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is becoming one of the most frequent liver diseases worldwide, and great difficulties are encountered nowadays for finding non-invasive, sensitive, specific and reproducible workup methods to help these patients. The aim of our study is to assess literature findings on the utility of evaluating portal vein velocity with Doppler ultrasonography (US) in patients affected with NAFLD, and to discuss a series of such cases vs different US findings. A relationship between portal vein velocity and stage or progression on NAFLD would suggest that such an alternative, non-invasive and reproducible examination could be used in the management of non-cirrhotic NAFLD patients. The basis of such a relationship would be the presumed reduction in liver and hepatic vascular compliance, associated with an increase in hepatic vascular resistance in such patients. Although previously considered a non-reproducible and operator-dependent examination, Doppler US assessment of the portal venous velocity is now viewed by more and more practitioners as a useful tool at least in what follow-up and detection of complications in NAFLD patients is concerned.

Keywords: ultrasound, liver fibrosis, non-alcoholic fatty liver disease.

1. INTRODUCTION

The prevalence of adult obesity has registered for a long period of time a constant and dramatic increase worldwide [1]. Disorders such as biliary and cardiovascular diseases, dyslipidemia, hyperinsulinemia, type 2-diabetes mellitus and nonalcoholic fatty liver disease (NAFLD) are known as frequent comorbidities in obese patients. Furthermore, the elevated prevalence and incidence of patients with NAFLD in current practice urges the need to develop simple, non-invasive, dynamic, cost-effective, repetitive and reproducible methods for the control and management of such patients. In what liver diseases are concerned, differences in the elasticity of liver can help facilitate the diagnosis of diffuse diseases that cause fibrosis [2,3] by measuring portal vein velocity with Doppler US. Such indirect work-up methods should be developed as, lately, especially in Europe, a constant decrease in the number of liver biopsies (LB) was registered, in favor of non-invasive modalities of evaluation (biological tests or elastographic evaluation), in patients with chronic liver disease [4]. MRI is considered, in the vast majority of literature studies, the most sensitive and reliable method of detecting, quantifying, and distinguishing hepatic steatosis from other lesions of the liver [5,6].

On the other hand, Doppler US is a method of investigation frequently used because of the non-invasive assessment of the hemodynamics of hepatic vessels. Some studies have demonstrated quantitative hepatic blood flow changes in patients with parenchymal liver diseases and have reported significant results about the relation between the degree of fat accumulation and portal vein hemodynamics [5-8]. Consequently, it has been shown that, because of the decreased compliance of the liver affected by NAFLD, the normal triphasic flow form in hepatic veins becomes monophasic [9]. Again, a fatty tissue overload in the liver, a compensatory increase in hepatic arterial flow has been described, secondary to elevated pressure on portal triad structures, and decreased resistivity index [10].

Therefore, our article aims at assessing further literature findings on the relationship between portal venous velocity measured by Doppler US and the degree, progression and severity of NAFLD. This approach comes to discuss three cases of NAFLD patients with typical but different changes in the portal venous velocity patterns.
2. MATERIALS AND METHODS

US examinations were performed with a General Electric Logiq E9 device on a 3.5 MHz convex probe. Patients rested in supine or left lateral position and were examined from subcostal or intercostal approaches. Usually, a proper doppler ultrasonogram of the portal venous velocity requires sample gate adjustment between 6-10 mm, directly proportional to the diameter of the portal vein, and subsequently, portal vein spectral analyses should always be examined for at least 2-3 cycles [11].

The vascular structures were investigated with gray scale, color Doppler and spectral Doppler USG. Usually, on color Doppler, the lumen should be fully filled with homogeneous color and the flow direction should be towards the liver (as shown in Fig. 1); on spectral exam, the sample window should be located in the center of the lumen with the flow cursor placed parallel to the portal flow and the transducer oriented along the longitudinal axis of the main portal vein (as shown in Fig. 2) [12]. Portal flow examination was performed with an angle held between 30–60 degrees, always after deep inhalation and breath holding. Maximum (Vmax) and minimum (Vmin) velocities (cm/s) were recorded in each patient. In all patients, the craniocaudal size of the liver
was also measured on the midclavicular line in the sagittal plane (Fig. 3), and liver elasticity (Fig. 4) was obtained through Two Dimensional Shear Wave Elastography (2D-SWE). The bipolar diameter of the spleen and splenic vein were measured in all cases and correlated with portal vein caliber and liver elasticity.

3. RESULTS AND DISCUSSIONS

1st case: P.M., a 62 year-old male patient diagnosed in 2012 with NAFLD, type 2 diabetes (insulin dependent), essential arterial hypertension, and obesity grade 1. Abdominal US showed severe steatosis (hyperchogenic liver and hepatomegaly) of the liver, portal venous caliber of 12.2 mm, and a spleen bipolar diameter of 127 mm, with a spleenic venous caliber of 9.7 mm. Liver stiffness assessed by 2D-SWE was of E mean of 12.64 kPa. As shown in Figure 5, Doppler US portal vein velocity showed the following hemodynamic changes: decreased portal blood flow velocity and reduction in portal flow volume – typical changes found in NAFLD patients with increased liver stiffness. The proven alterations of hepatic blood flow patterns in such patients are probably linked to reduced vascular compliance of the liver, caused by fatty infiltration. Furthermore, poor portal venous velocity can be suspected in such patients with elevated portal and spleen venous calibers and splanomegaly.

2nd case: Z.I., a 69 year-old patient diagnosed in 2010 with NAFLD, mixed dyslipidemia, and obesity grade 1. Abdominal US showed intense liver steatosis (shown as a high echogenicity index of the liver and hepatomegaly), portal venous caliber within 14.9 mm, and splanomegaly with a spleen bipolar diameter of 124 mm and a spleenic venous caliber of 10.2 mm. Liver stiffness assessed by 2D-SWE was of E mean of 11.61 kPa. Despite severe US assessed liver steatosis, doppler US (Fig. 6) showed slightly decreased peak portal vein velocity.

3rd case: M.A, a 68 year-old male patient diagnosed in 2015 with NAFLD, grade 2 essential arterial hypertension, type 2 diabetes and obesity grade 3. Abdominal ultrasonography showed hepatomegaly with an increased echogenicity index and indirect signs of portal hypertension: portal venous caliber of 14.6 mm, splanomegaly with a bipolar spleen diameter of 150 mm and a spleenic vein caliber of 11.2 mm. Liver stiffness assessed by 2D-SWE was of E mean of 12.63 kPa. As shown in Figures 7 and 8, portal venous velocity and volume flow are seriously impaired for a non-cirrhotic patient. As deductible, the most important comorbidity of this patient is the severe obesity with a BMI of 34.
38.9 kg/sqm, which apparently influences the parameters of portal venous velocity, despite a relatively modest increase in liver elasticity. Abdominal obesity should be therefore seen as an important disorder that can influence hepatic fat accumulation, hence affecting portal venous velocity and flow, as also demonstrated by significant studies in the field [11].

Fig. 7 and 8. Spectral Doppler US assessed portal venous velocity of a patient with severe indirect signs of portal hypertension showing only slightly decreased portal venous velocity and preserved volume flow: Vmax (18.2 cm/s), Volume Flow= 619.0 ml/min (Image courtesy of St. Spiridon Emergency Hospital of Iasi, Institute of Gastroenterology and Hepatology)

4th case: M. I., a 37 year-old male patient newly diagnosed with NAFLD and mixed dyslipidemia (April 2016). Abdominal ultrasonography showed a moderately increased echogenicity index of the liver with a normal portal venous caliber of 12 mm, a normal spleen bipolar diameter of 114 mm and a normal spleenic vein caliber of 7.8 mm. Liver stiffness assessed by 2D-SWE 2D-SWE was very proximal to normal values, with an E mean of 9.05 kPa. Despite the normal organic parameters evaluated by ultrasonography and only a slightly increase in liver stiffness, Doppler US assessed that portal venous velocity and volume flow are visibly impaired, as shown in Figures 9 and 10.

Fig. 9 and 10. Spectral Doppler US assessed portal venous velocity of a young patient newly diagnosed with NAFLD with normal-borderline US organic parameters but showing decreased portal venous velocity and affected volume flow: Vmax (20.5 cm/s), Volume Flow= 507.5 ml/min (Image courtesy of St. Spiridon Emergency Hospital of Iasi, Institute of Gastroenterology and Hepatology)
5th case: N.D., a 49 year-old male patient, diagnosed in 2012 with non-alcoholic steatohepatitis, morbid obesity (with a BMI of 49 kg/sqm), previously diagnosed with grade 3 essential arterial hypertension and type 2 diabetes non-adherent to treatment; despite inconsistent follow-up with a hepatology consultant, the patient was recently admitted in the gastroenterology unit (April 2016) with severe abdominal pain and edematous status. Abdominal ultrasonography showed indirect elements of severe portal hypertension, with a portal venous caliber of 16.6 mm, a spleen bipolar diameter over 200 mm and a splenic venous caliber of 14.7 mm, and mild ascites. Liver stiffness assessed by 2D-SWE was very proximal to normal values, with an E mean of 20.03 kPa. All these paraclinical imagistic parameters plead for the diagnosis of cirrhosis secondary to chronic and uncontroled NAFLD. As shown in Figure 11, Doppler US proves spontaneous portosystemic shunts and abnormal blood flow direction in the portal vein (bidirectional), with higher than normal portal venous velocity parameters, elevated volume flow and a patent umbilical vein.

![Spectral Doppler US assessed portal venous velocity of a cirrhotic patient liver cirrhosis secondary NAFLD showing higher than normal portal venous velocity and increased volume flow: V_max (30.0 cm/s), Volume Flow= 1653 ml/min (Image courtesy of St. Spiridon Emergency Hospital of Iasi, Institute of Gastroenterology and Hepatology)](image)

Balci et al. [7] reported findings on 105 patients with hepatosteatosis, concluding that the pulsatility index and mean portal venous velocity decreased, as the severity of fatty infiltration increased. Nevertheless, other statistically significant studies found no correlation between the degree of hepatic fat fraction and portal venous velocity, even if the difference in portal venous velocity between the patients with NAFLD and the normal subjects is statistically significant [11]. However, one of the main downsides of the vast majority of studies is that the fatty infiltration score is unitarily determined solely on the basis of gray-scale images of the liver parenchyma, even if other more reliable radiologic methods do exist. In a descriptive study on patients with NAFLD, Erdogmus et al. [7] also reported a decrease in portal venous velocity indexes related to an increase in fat accumulation. Other studies, performed on very large cohorts of patients, state that portal venous maximum velocity is notably lower in patients with fat accumulation than in healthy individuals [13].

Cioni et al. [14] have long ago reported that normal portal venous maximum velocity ranges between 20 and 33 cm/s, while Dietrich et al. [15] have established a correlation between the degree of fat accumulation on liver biopsy and portal venous velocity and maximum flow in some patients with chronic liver disease. On the other hand, in relation with the pathology of portal velocity changes in NAFLD patients, Mohammadi et al. [16] have relatively recently reported that the values of liver blood flow parameters are inversely correlated with the degree of fatty infiltration, an important component of this relationship being the increased intra-abdominal pressure in patients with higher BMI values that lower hepatic blood flow. This last statement is of great interest in the present analysis, as most of NAFLD patients with portal venous velocity changes are also affected by various degrees of obesity.

4. CONCLUSIONS

Both series of cases presented and the vast majority of studies prove that portal vein velocities in patients with NAFLD are visibly lower than in healthy subjects. However, one of the main technical downsides is that portal vein flow patterns are not changed by the degree of hepatic fat accumulation, the latter being almost
uniformly subjectively assessed. Otherwise, Doppler US should be dealt with as a basic diagnostic method for the noninvasive evaluation of portal venous flow patterns. As NAFLD is a chronic condition that may advance to cirrhosis, it requires thorough follow-up and periodic workup. Thus, at least the hemodynamic changes in the liver seen through the changes in portal venous velocity parameters can be easily monitored with the above-described method.

References