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RESEARCH ARTICLE

ROLE OF DOPPLER EVALUATION OF PORTAL VEIN HEMODYNAMICS IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE (A PROSPECTIVE STUDY OF 100 SUBJECTS)

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ABSTRACT

Aim: To evaluate the hemodynamics of portal vein in patients with non-alcoholic fatty liver disease (NAFLD) with help of Doppler sonography.

Materials and Methods: In a prospective case-control study carried out in the Department of Radio-diagnosis SSG hospital, Vadodara between April 2016 to November 2016 on an outpatient basis, 100 subjects with previous 4-6 hours of fasting were assessed by senior radiologists with many years of experience in the field of abdominal sonography and Doppler on a Mylab-40 ultrasound machineusing a low frequency, 3-5 MHz convex transducer. They were examined with help of Gray-scale and Doppler (color and spectral wave) sonography for evaluating the hemodynamics of the main portal vein. These subjects were divided into 2 groups of test (64 subjects with various grades of fatty infiltration of liver) and control (36 healthy subjects) based on presence or absence of fatty infiltration of the liver. The test group was further divided into 3 subgroups (group 1, 2 and 3) on the basis of the degree of fatty infiltration of the liver assessed on the gray-scale images. The fatty infitration of the liver was assessed on Gray-scale images by comparing its echogenicity with that of the right renal cortex and was graded as mild moderate and severe. The portal vein pulsatility index (VPI) and time-averaged mean flow velocity (MFV) were calculated for each subject. VPI was calculated as (peak maximum velocity - peak minimum velocity) /peak maximum velocity).

Results: VPI and MFV values were, respectively, 0.32 +/- 0.06 and 16.8 +/- 2.6 cm/second in the control group, 0.27 +/- 0.07 and 14.2 +/- 2.2 cm/second in the group with grade 1 fatty infiltration, 0.22 +/- 0.06 and 12.2 +/- 1.8 cm/second in the group with grade 2 fatty infiltration, and 0.18 +/- 0.04 and 10.8 +/- 1.5 cm/second in the group with grade 3 fatty infiltration. There was a negative inverse correlation between the grade of fatty infiltration and both VPI (f - 55.3, p <0.001) and MFV (f - 43.9, p <0.001).

Conclusion: The pulsatility index and the mean flow velocity decreases as the degree of fatty infiltration increases indicating that fatty infiltration leads to impedance to blood flow in the hepatic parenchyma and can later cause changes of chronic hepatitis and cirrhosis. The exact pathology of liver cirrhosis is incompletely understood. However, few suggest it may be due to increasing fibrosis consequent to increased lipid deposition.

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INTRODUCTION

The prevalence of adult as well as childhood obesity is increasing tremendously nowadays (World Health Organization, 1998). The spectrum of NAFLD ranges from simple steatosis, through fibrosis, to cryptogenic cirrhosis. NAFLD is the most common cause of elevated liver enzyme concentrations and is currently the third leading cause of liver cirrhosis (Wajchenberg, 2000; Mulhall *et al*., 2002; Brunt, 2007; Jensen and Johnson, 1996; Bjorntorp, 1990; Kissebah *et al*., 1982; Fujioka *et al*., 1987; Peiris *et al*., 1988). Many reports suggest that the portal venous hemodynamics are altered in patients with fatty liver disease. (Bolondi *et al*., 1991; Aube *et al*., 2004; O'Donohue *et al*., 2004) Ultrasonography has a sensitivity of 82 to 89 % and a specificity of 93 % in detecting
fatty infiltration of the liver. (Aube *et al*., 2004) Doppler sonography is used as a non-invasive method in the assessment of hemodynamics of hepatic vascular flow in patients with liver disease. In this study, we evaluate the effects of hepatic fatty infiltration on portal vein hemodynamics.

MATERIALS AND METHODS

A prospective study carried out between April 2016 to November 2016 on 100 subjects visiting the out-patient department of SSG hospital, were divided into test (64 subjects) and control groups (36 subjects) on the basis of presence or absence of fatty infiltration of the liver. The test group was further subdivided into three groups (Group 1, 2 and 3) on basis of severity of fatty infiltration of the liver as mild, moderate and severe. The patients and controls were recruited from subjects undergoing sonographic examinations of unrelated organ systems (genitourinary, musculoskeletal, and so forth) without any abnormality other than hepatic steatosis.

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Homogeneity of the groups was ensured using the following exclusion criteria: positive viral hepatitis markers (hepatitis B surface antigen and anti-HCV antibody), diabetes mellitus, cirrhosis or chronic parenchymal liver disease, alcohol use (>40 g of ethanol per week), splenomegaly, systemic arterial hypertension, cardiac disease, presence of free intra-abdominal fluid, non-homogenous fatty infiltration, vascular malformations or space-occupying lesions of the liver, chronic obstructive lung disease and known gastrointestinal malignancy or disease. Cardiac diseases were excluded via medical history, physical examination, and chest radiography. Acute or chronic liver diseases and malignancy were also eliminated by appropriate clinical, laboratory, and imaging tests. All subjects were examinedwith help of low frequency 3-5MHz transducer on Mylab-40 ultrasound machine by a single senior radiologist having years of experience in the field of abdominal sonography in the left decubitus and supine positions. The subjects had fasted overnight and were studied in the morning between 9 and 12 AM. All segments of the liver were carefully scanned. The measurements were performed at the main portal vein just proximal to its bifurcation. The Doppler gate length was 5 mm. The angle between the Doppler ultrasound beam and the long axis of the portal vein was maintained between 0and 60 degrees. The Doppler gain and filter were set to optimize sensitivity without producing excessive noise. Flow velocity range settings were optimized in each case. During the measurements, the subjects were asked to hold their breath for 5 to 8 seconds mid-inspiration, and measurements were taken during this period. Spectral waveform of portal venous flow was recorded for at least 5 seconds. For characterization of the portal vein blood flow, the values of peak maximum velocity (Vmax, in cm/second), peak minimum velocity (Vmin, in cm/second), and time-averaged mean portal vein flow velocity (MFV, in cm/second) were obtained in all patients and control subjects. Portal vein pulsatility index (VPI) was calculated as (peak maximum velocity - peak minimum velocity)/peak maximum velocity. This index is similar to the resistance index used to characterize arterial waveforms. Fatty infiltration of the liver parenchyma by was assessed on gray-scale images by comparing the echogenicity of the liver with the adjoining right renal cortex. Control subjects had normal hepatic echotexture and beam attenuation. Grade 1patients had a subtle increase in echogenicity of the liver parenchyma, with a slight decrease indelineation of the portal vein walls and normal delineation of the diaphragm. Grade 2 patients had a moderate diffuse increase in liver echogenicity, with a moderately decreased delineation of the portal vein walls and of the diaphragm. Grade 3 patients had a marked increase in hepatic parenchymal echogenicity, with poor or absent delineation of the portal vein walls and diaphragm.Gray-scale settings such as frequency, focus, gain, and tissue harmonic imaging were optimized by the sonographer on a case-by-case basis.

Statistical analysis was performed using SPSS for Windows, version 11.5 (SPSS, Chicago, IL). Data are expressed as the mean 6 standard deviation. Analysis of variance (ANOVA) was used for comparison of the means. When ANOVA yielded statistically significant results, the Tukey honestly significant difference test was applied for multiple comparisons. Correlations between the fatty infiltration grades and other variables were performed using Spearman rank correlation. Partial correlation analysis was used to eliminate the confounding effects of BMI. All tests of significance were 2-tailed. A p value of <0.05 was considered statistically significant.

RESULTS

All patients with Non-alcoholic fatty liver disease (test group) were overweight i.e. they had a BMI of more than 25 (normal for Indian population was less than 22.9). Thephasicity of portal venous flow was less in the control group as compared to the test group.

Table 1. Demographics and Doppler flow characteristics of subjects

	Study group	Control group	P-value
Total number (n)	64	36	
Age (mean)	49.17 +/- 10.28	43.24 +/- 10.75	>0.05
Sex (m:f)	34:33	18:15	>0.05
Body mass index (bmi)	30 +/- 2.84	22 +/- 1.58	<0.001
Vpi	0.24 +/- 0.06	0.32 +/- 0.06	<0.001
Mfv (in cm/second)	12.8 +/- 0.04	16.8 +/- 2.6 cm/s	<0.001

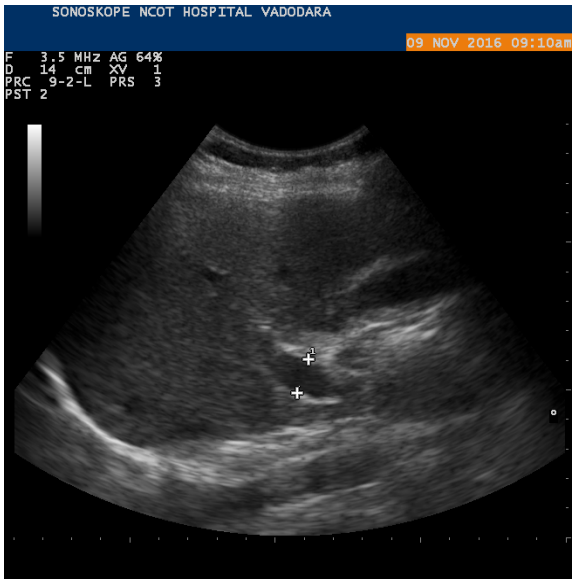
VPI: pulsatility index, MFV: mean flow velocity.

Table 2. Demographics and Doppler flow characteristics of test subjects in subgroups (n=64)

	GROUP 1 Mild fatty infiltration	GROUP 2 Moderate fatty infiltration	GROUP 3 Severe fatty infiltration	p-value
Total number	27	16	21	
AGE	49.12 +/- 3.42	49.25 +/-11.48	48.84 +/- 10.24	>0.05
BMI	29 +/- 3.54	30 +/- 1.12	32 +/- 6.68	<0.001
VPI	0.27 +/- 0.07	0.22 +/- 0.06	0.18 +/- 0.04	<0.001
MFV (cm/second)	14.2 +/- 2.2	12.8 +/- 1.8	10.8 +/- 1.5	<0.001

BMI: body mass index, VPI: pulsatility index, MFV: mean flow velocity.

Demographics of both the test and control groups was similar with respect to the age and sex (p>0.05). The body mass index (BMI) was higher in the test group as compared to control group (p<0.001). The VPI (pulsatility index) and the MFV (mean flow velocity was lower in the test subjects as compared to the control group (p<0.001). Within the test subjects the VPI and the MFV was lowest within the subgroup 3 having severe fatty infiltration. Sonographic index of the fatty liver was negatively co-related with MFV (f - 43.9, p <0.001) and VPI (f - 55.3, p <0.001).



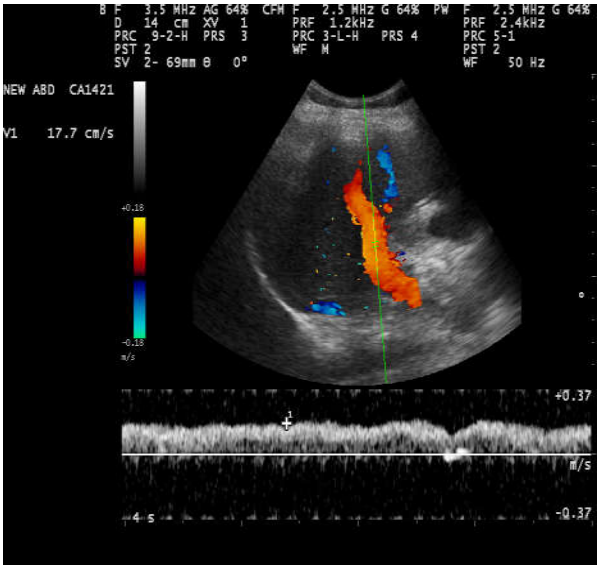
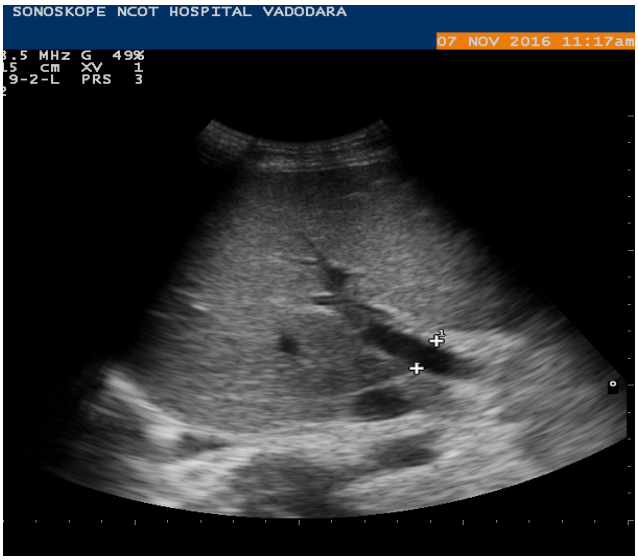


Fig 1a, b showing gray scale image of a 42 yrs old control subject with a body mass index of 23 showing normal liver echotexture and mean portal venous velocity of 17.7 cm/sec



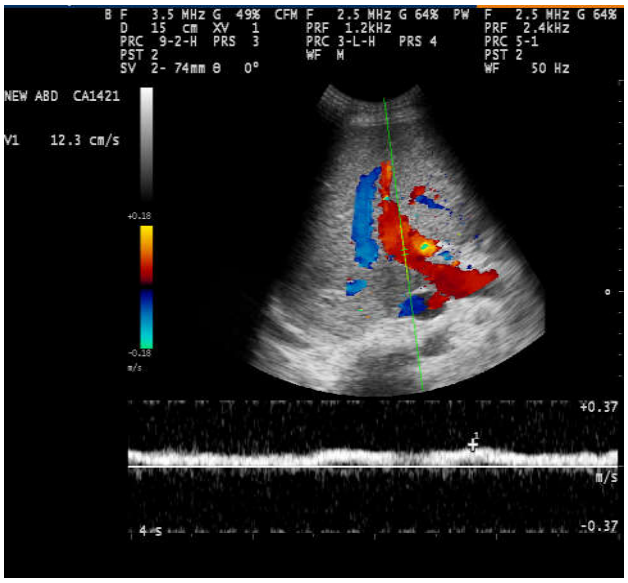
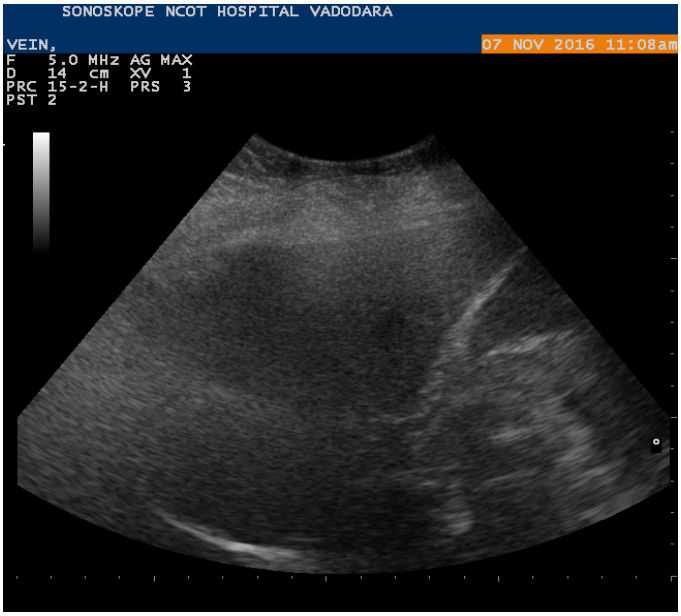


Fig 2a, b showing gray scale image of a 46 yrs old test subject with a body mass index of 29 showing bright liver echotexture (grade 1fatty changes) and mean portal venous velocity of 12.8 cm/sec



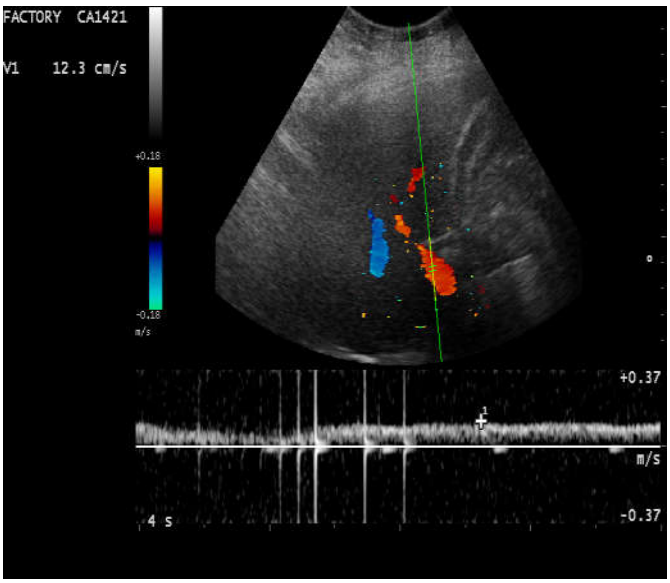
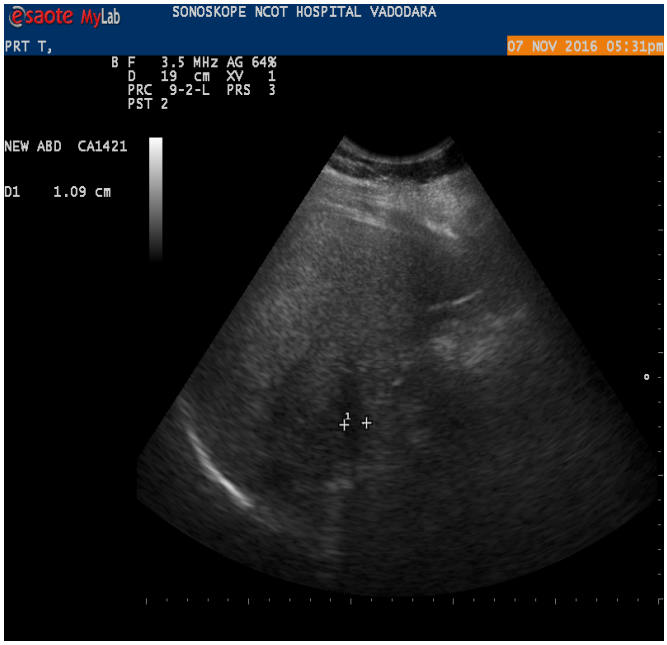


Fig 3a, b showing gray scale image of a 56 yrs old test subject with a body mass index of 30 showing grade II fatty changes and mean portal venous velocity of 12.3 cm/sec



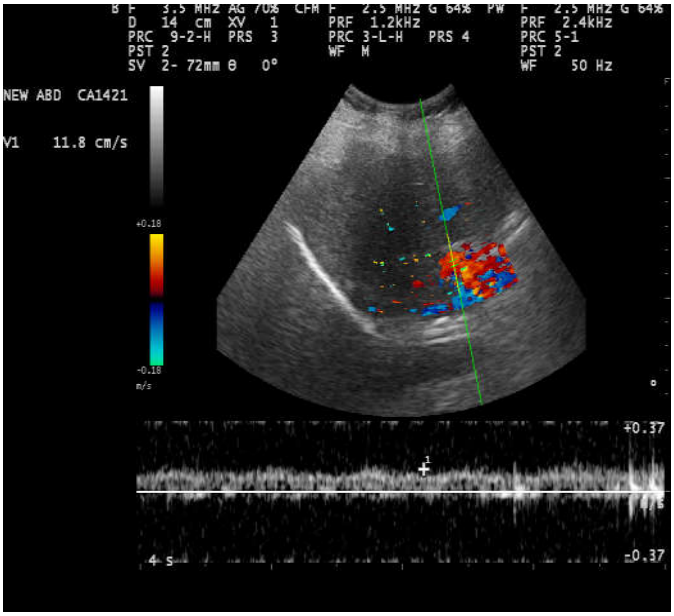


Fig 4a, b showing gray scale image of a 52 yrs old test subject with a body mass index of 30 showing grade III fatty changes and mean portal venous velocity of 11.8 cm/sec

DISCUSSION

The portal venous flow is described as continuous, hepatopetal flow showing phasic variation. (Schneider *et al*., 2005) The VPI (pulsatility index) and the MFV (mean flow velocity) of the portal vein in normal healthy persons is 0.48 (Dietrich *et al*., 1998). In this study, patients with hepatic steatosis were divided into 3 groups according to the grayscale appearance of the liver parenchyma. Duplex sonography, on the other hand, is able to demonstrate vascular hemodynamic changes, in addition to the grade of steatosis. Fatty liver is visualized on gray-scale images as an increase in parenchymal echogenicity and can be graded as mild, moderate, and severe. (O'Donohue *et al*., 2004; Schneider *et al*., 2005; Dietrich *et al*., 1998) The relative contribution of intrahepatic fat deposition and inflammation to the alteration of portal vein hemodynamics is still unclear. However, it can proposed that hepatic fatty infiltration leads to impaired compliance (impedance) to the portal venous flow leading to alteration in the portal venous hemodynamics on the basis of the finding that the pulsatility index (VPI) and the MFV (mean flow velocity) was lower in the test group (comprised of subjects with varying degrees of fatty infiltration). Schneider *et al*. (2005) found a negative co-relation between degree of hepato-steatosis and portal vein flow modulation in patients with hepatitis c and a negative co-relation between fatty infiltration and portal vein MFV. In our study the subjects did not have chronic hepatic disease and were anti HCV antibody negative. However, negative co-relation between portal vein hemodynamics and degree of fatty infiltration in the liver. Similar study design, by Balki *et al* (2008) showed negative co-relation between hepatic-steatosis and portal venous VPI, MFV, Vmax and V min.

Conclusion

The portal vein pulsatility index and mean flow velocity decreased with increasing fatty infiltration of the liver. This may be due to increase impedance to portal venous blood flow secondary to increased fat deposition within the liver parenchyma.

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