Hepatic Artery Doppler Indices in Children with Extra Hepatic Portal Vein Obstruction

Jesudoss Randhir*, Satyabhama Chandrasekaran[†], Malathi Sathiyasekaran[‡], Jayanthi Venkataraman^{§,||}

*Department Gastroenterology Stanley Medical College and Hospital [†]Precision Diagnostics [‡]Paediatric Gastroenterologist, Sundaram Medical Foundation, [§]Global Health City, # 439, Cheran Nagar, Perumbakkam and ^{||}Department of Gastroenterology, Stanley Medical College and Hospital, Chennai, India

Doppler measurement provides information on the hemodynamics in the hepatic artery and the portal venous system. **Aim:** To study the hepatic artery hemodynamics in children with extra hepatic portal vein obstruction. **Materials and methods:** Hepatic artery indices were studied using Doppler indices in 15 children (<12 years) with extra hepatic portal hypertension (EHPVO) and obliterated esophageal varices. The hepatic artery resistive index, the arterial acceleration time and the acceleration index were used to determine the flow pattern within the hepatic artery. Controls were 15 healthy age-sex matched children, belonging to the same socioeconomic strata in absence of liver disease. **Results:** The mean age of the children was 8.43 ± 3.2 years and male female ratio was 4:1. All the children had obliterated esophageal varices. The hepatic artery resistive index in the children with EHPVO was normal and similar to controls. The hepatic arterial early systolic acceleration index was significantly higher in cases compared to controls (436 ± 290 vs 214 ± 100 ; P value <0.004). The hepatic arterial acceleration time though low in the cases (86 ± 35 cm/s) was not statistically different from the controls (128 ± 14 cm/s). **Conclusion:** There was a significant increase in hepatic arterial early systolic acceleration in children with chronic EHPVO. The latter may be responsible for an increase in hepatic arterial in flow velocity in a slow flow system despite a normal resistive index. (J CLIN EXP HEPATOL 2013;3:102–105)

Doppler sonography of the portal venous system, provides considerable information on the portal hemodynamics in various types of portal hypertension.^{1,2} While there are several studies that have reported portal vein and hepatic artery hemodynamics in healthy individuals³ those with acute portal vein thrombosis,⁴ therapeutic portal vein embolization,⁵ and hepatic artery stenosis after liver transplant,⁶ very few studies have looked into the portal hemodynamics in children with long standing extra hepatic portal vein obstruction (EHPVO).^{7,8}

Congestive index and venous pulsatility index in the portal vein, both in adult and pediatric patients are determinants of the hemodynamic status of the portal system.^{8,9} These indices are not reliable in portal vein thrombosis and portal cavernoma as there is a poor correlation

between the size of the portal vein and portal pressure.^{7,10} Also, the transformation of the portal vein to a cavernoma makes measurement of these indices difficult.

Hepatic artery indices are likely to be more informative¹¹ under such circumstances. One can get a clue to the hemodynamics of the blood flow in the hepatic artery. Till date, there are no published studies that have reported on hemodynamics in hepatic artery in patients with cavernomatous transformation of the portal vein.

McNaughton et al¹² describes the normal hepatic arterial waveform in the hepatic artery to be pulsatile with low resistance. The peak height of the wave corresponds to the peak systolic velocity (V_1), and the trough to end-diastolic velocity (V_2). The normal resistive index (RI) within the hepatic artery ranges from 0.55 to 0.8. Any measured RI above or below the normal range represents a disease state.^{13–15}

The aim of the present study was to determine the hepatic artery resistive index, the acceleration time and the acceleration index in children with EHPVO.

MATERIALS AND METHODS

The Liver Unit at Stanley Hospital predominantly caters to the adult population. The present study included 15 children with EHPVO who were referred to the Liver Clinic for management of variceal bleed. All these children were on propranolol in a dose of one mg/kg as secondary prophylaxis.

Keywords: Doppler indices, extra hepatic portal venous obstruction, hepatic artery index

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Address for correspondence: Dr. V. Jayanthi, Consultant Gastroenterologist and Hepatologist, Global Health City, # 439, Cheran Nagar, Perumbakkam, Chennai 100, India.

E-mail: drjayanthi35@yahoo.co.in

Abbreviations: EHPVO: extra hepatic portal vein obstruction; RI: resistive index; HARI: hepatic artery resistive index; ESA: early systolic acceleration; HAAT: hepatic artery acceleration time; HAAI: hepatic artery acceleration index; HABR: hepatic arterial buffer response

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Children with normal platelet count and normal liver function tests and a confirmed diagnosis of EHPVO on ultrasound and Doppler were included in the study. Those who had bled recently or were actively bleeding and were hemodynamically unstable or had liver cirrhosis in addition to portal vein thrombosis or portal biliopathy were excluded from the study. Controls were age and sex matched healthy children attending the Well Baby Clinic in the pediatric department of the same hospital.

Formal ethics committee approval of Stanley Medical College Hospital was obtained.

Technique

Doppler study was done using a 3.5 MHz convex sector probe (Toshiba Core Pro-vision 2000) in the morning of the test after a 4-h fast. The recordings in the hepatic artery were taken on at least 3 occasions in a fasting state, and during breath holding while the child was on secondary prophylaxis with propranolol. Waveforms were obtained, using an angle of insonation of less than 60°. The hepatic artery acceleration time and index was studied in the intra-hepatic branch of the right division of the right hepatic artery bifurcating within the liver parenchyma.^{16,17}

Definition

Hepatic Artery Resistive Index (HARI): peak systolic velocity minus end-diastolic velocity divided by the peak systolic velocity.

The Early Systolic Acceleration (ESA) in the hepatic artery is the slope of a tangent of the initial systolic up-sweep of the arterial waveform i.e. the duration of upstroke from end-diastole to peak systole, measured using calipers. The time corresponding to ESA was computed and this was referred to as the **Hepatic Artery Acceleration Time (HAAT)** in sec. The value used in this study was obtained by taking an average of three measurements.

The **Hepatic Artery Acceleration Index (HAAI)** is the ratio between the acceleration of the Doppler spectral waveform and the relative peak systolic velocity. The systolic acceleration was calculated as a change in the distance between the beginning of systolic flow and the peak systolic velocity (cm/sec), divided by the acceleration time. The acceleration index is expressed in frequency units as KHz/sec or in velocity units as cm/sec.²

Statistical Analysis

Student *t*-test was applied for case and controls and *P* value <0.05 was considered significant.

RESULTS

The mean age of 15 children was 8.4 ± 3.2 years (range: 5–15 years). There were 12 boys. All the children were from lower socioeconomic strata attending the government hos-

pital. The mean number of endotherapy sessions at the time of Doppler study was 7 ± 4.3 and follow up after obliteration of esophageal varices was 22 ± 12 months (range: 5 months to 8 years).

On Doppler imaging, the liver size and echotexture was normal. The portal vein was replaced by multiple tortuous vessels at the porta hepatis with a characteristic "flat" Doppler waveform (Figure 1). Four children had lienorenal collaterals. The mean hepatic artery resistive index (HARI) in patients (0.64 ± 0.04) and healthy controls (0.68 ± 0.02) was similar. The hepatic artery acceleration time (HAAT), a marker of the time required for the acceleration, though shorter in the study group (86 ± 35 cm/s) was not statistically significant from the controls (128 ± 14 cm/s). However, the hepatic arterial early systolic acceleration index (HAAI) was significantly higher in the cases (436 ± 290 KHz/s) compared to controls (214 ± 100 KHz/s) ($P < 0.004$) (Figure 2).

DISCUSSION

Doppler sonography is a noninvasive method to assess the splanchnic venous and arterial vasculature.¹⁸ However, a caution is indicated when interpreting small changes in the measurement of the Doppler ultrasound indices i.e. even in normal conditions substantial variability exists in hepatic arterial measures.¹⁹ The present study in a small group of 15 children has given us important information on hepatic arterial hemodynamics in children with EHPVO. There was a significant increase in hepatic arterial early systolic acceleration index, with no significant change in the resistive index.

The relationship between the portal vein and the hepatic artery blood flow was reported by Ternberg and Butcher even as early as in 1965.²⁰ The obstruction in portal vein (slow flow) serves as a mechanical interposition in the path of a rapid flowing arterial (hepatic artery) stream.

Figure 1 The portal vein replaced by multiple tortuous vessels at the porta hepatis with a characteristic "flat" Doppler waveform.

Figure 2 Doppler spectral wave showing the hepatic arterial early systolic acceleration.

This results in an increase in hepatic arterial blood flow secondary to a decrease in resistive index within the artery.

Several studies in acute portal vein thrombosis have shown a relationship between hepatic artery resistance and blood flow velocity. Sheen et al had shown a low HARI of 0.57 ± 0.11 in patients with portal vein thrombosis viz a viz a HARI of 0.75 ± 0.08 amongst controls.⁴ Platt et al in a study of 35 patients with suspicious portal vein thrombosis showed a reduction in hepatic artery resistive index (HARI). He concluded from the study that reduction in HARI could be an objective parameter for portal vein thrombosis.²¹ Kito et al⁵ in a study of hepatic artery resistance and flow velocity in patients undergoing right portal vein embolization prior to extended liver resection showed a decrease in resistive index to 0.5 or less within the hepatic artery with a significant increase in blood flow velocity in the artery.

An earlier study amongst cirrhotics, had shown an increase in hepatic arterial resistance and reduction in blood flow in the hepatic artery²² and this was significant in advanced cirrhosis. While the mechanism of an increase in hepatic arterial resistance in cirrhosis liver is comprehensible, the hemodynamic changes in EHPVO is less clear, where the sinusoidal pressure is low or normal.

Based on 'hepatic arterial buffer response (HABR) theory,' hepatic arterial blood flow varies inversely with portal blood flow to maintain total blood flow.²³ In EHPVO, the cavernoma around the fibrosed portal vein is likely to result in retrograde flow of blood in the portal vein resulting in 'liver steal syndrome'. The flow pattern in the hepatic artery is not known.

Doppler studies on HARI in chronic EHPVO are meager. The present study did not show any difference in resistive index between case and controls. Are factors contributing to the hepatic artery resistive index (an increase or a decrease) different in acute and chronic portal vein thrombosis? This at present is not known. The increase in HAAI in our children due to an increase in early

systolic acceleration was the only significant buffering factor in this series. Can the early systolic acceleration be the factor for an increase in hepatic artery blood flow? This needs to be established in larger series of cases.

There are a few limitations in this study; one of them is the small sample size. The second issue was the difficulty in assessing the portal vein indices in presence of a cavernoma, and thirdly calculation of pulsatility index in the hepatic artery would have been more informative. The results of this study must be viewed as preliminary and further evaluation of larger series is required to arrive at a precise conclusion.

CONFLICTS OF INTEREST

All authors have none to declare.

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