Original Research Article

Evaluate the role of endoscopy and ultrasonography in patients of portal hypertension

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Received: 23 July 2019
Revised: 29 September 2019
Accepted: 03 October 2019

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ABSTRACT

Background: Aim of the study was to evaluate the role of endoscopy and ultrasonography in pediatric patients suffering from portal hypertension. Subjects: children under 12 years of age hospitalized with any symptom or sign suggestive of portal hypertension.

Methods: The study was conducted in 30 children with portal hypertension. They were divided into two groups on the basis of site of lesion: extrahepatic (extra hepatic portal vein obstruction) and intrahepatic (chronic liver diseases).

Results: Mean age of children with EHPVO (extra hepatic portal vein obstruction) was 4 years and 4 months while that of children with CLD (chronic liver diseases) mean age was 8 years and 4 months. Endoscopic findings in patients with EHPVO have severe grade of varices as compared to those with CLD. In patients with EHPVO, the most common USG finding was nonvisualisation of the main portal vein or either branch (75-85%). Portal vein cavernoma was seen in 75% of these patients. Portosystemic collaterals were visualized in 23 patients in which the left gastric collateral (60%) was the most common followed by short gastric collaterals in 11 children (55%).

Conclusions: Endoscopy and ultrasonography are new and better modalities to assess the diagnosis and severity of portal hypertension. Extra hepatic portal vein obstruction is the commonest cause followed by intrahepatic obstruction (Chronic liver diseases).

Keywords: Collaterals, Endoscopy, Liver, Portal hypertension, Ultrasonography, Varices

INTRODUCTION

Portal hypertension is defined as the presence of wedged hepatic venous pressure greater than 4 mmHg above the inferior vena cava pressure of splenic pulp pressure of more than 15 mmHg or portal vein pressure of more than 30 cm of saline as measured at surgery.¹² Despite the large variety of causes scant work is available on the etiology, presentation, clinical course and diagnostic modalities of portal hypertension in children.

Therefore, this study was designed to evaluate the, utility of ultrasound and endoscopy in pediatric patients with portal hypertension.

METHODS

The study was conducted in children under 12 years of age who were hospitalized in the pediatric department of Chacha Nehru Bal Chikitsalaya (Affiliated to Maulana Azad Medical College) in Geeta Colony, Delhi.
Inclusion criteria

- Patients with a history of hematemesis and/or melana
- Splenomegaly ascites
- Distension of abdomen or another clinical feature suggestive of portal hypertension.

Exclusion criteria

- Children below 1 month of age

A detailed history including the presenting complaints, past history, duration of illness, natal and postnatal history of omphalitis or exchange transfusion, family history of similar illness, history of drug intake and detailed nutritional history were recorded. A detailed general physical examination was carried out in each patient and recorded. Special efforts were made to look for Kaysor-Fleisher ring by slit lamp examination, in patients with cirrhosis or chronic liver disease. An abdominal ultrasound was done by using a Realtime Ultrasound as PHILIPS ENVISOR with an electronic linear and Convex Probe of 3.5-5 MHz in thirty patients. The patients were fasted for 6-8 before the procedure. Younger children less than 5 years were sedated with syrup phenargan (0.5 mg/kg). Features carefully determined included the splenic size, echo pattern of liver, presence of ascites, patency and diameter of splenoportal axis, presence of portosystemic collaterals and associated gastro esophageal varices. The above findings were recorded. Percutaneous needle biopsy of liver was done in patients in whom the prothrombin time was within normal limits before or after giving injection vitamin k, 1mg intramuscular daily for 3 days.

Written consent was obtained from the parents or guardians. Liver biopsy was done with liver biopsy needle (Menghini’s) after sedating the child with intravenous Midazolam 0.1 mg/kg stat. The child was put in the supine position. The part was cleaned with spirit and tincture iodine and all aseptic precautions were taken. First local anesthesia was given in the 8th or 9th intercostal space with 1% lignocaine in the right midaxillary line. Then the needle was pushed medially towards the liver and the tissue was taken out and preserved in 10% formalin for histopathological examination.

The area was sealed with tincture benzoin and vital monitoring was done for 12-24 hours. The biopsy material was sent for histopathological examination at the pathology department in MAMC New Delhi. It was stained by Eosin and Hematoxylin stain. Statistical analysis was done included Student’s ‘t’ test, Fisher’s exact and McNemar’s test. Fiberoptic endoscopy was done in all patients clinically suspected of portal hypertension for the presence of esophageal varices. The endoscope used was FUJINON EG-250 WR5 fiberoptic endoscope. The patients were fasted for at least 6 to 8 hours before endoscopy. All endoscopies were performed after conscious sedation of the child with injection Midazolam 0.1 mg/kg/IV stat. If varices were seen, they were graded according to the modified conn’s criterion.

RESULTS

The study was conducted in 30 children with portal hypertension. On the basis of clinical presentation and various laboratory parameters the patients were divided in to two groups (1) Extrahepatic (Extra hepatic portal vein obstruction 66.6 % (n=20), (2) Intrahepatic Chronic liver disease 33.3% (n=10). Age range from 1.25 to 12 years. Mean age of children with EHPVO was 4 years and 4 months while that of children with CLD mean age was 8 years and 4 months. All 3 children with portal hypertension where less than 4 years has EHPVO (Table 1).

Table 1: Age distribution of children with portal hypertension.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Total</th>
<th>EHPVO</th>
<th>CLD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Number</td>
<td>Number</td>
</tr>
<tr>
<td>1-2</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>2-4</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>4-6</td>
<td>8</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>6-8</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>8-10</td>
<td>9</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>10-12</td>
<td>7</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>30</td>
<td>20</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Mean Age</td>
<td>4 years</td>
<td>8 Years</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 months</td>
<td>4 months</td>
<td></td>
</tr>
</tbody>
</table>

In cases of Extrahepatic portal vein obstruction (EHPVO) (M:F) (ratio 3:1) whereas in patients with chronic liver disease. Grade 3 varices was the most common finding in patients with EHPVO and chronic liver disease (CLD). Generally, the EHPVO patients tended to have severe grade of varices. Esophagitis was present in 5(25%) cases of EHPVO as compared to 1(10%) of the patient with CLD, gastric varices were seen in 4(13.3%) of cases of portal hypertension, all of them were cases of EHPVO (Table 2).

In patients with EHPVO, the most common USG finding was neovascularization of the main portal vein or either branch (75-85%). Portal vein cavernosa was seen in 75% of these patients. Portosystemic collaterals were visualized in 23 patients in which the left gastric collateral (60%) was the most common followed by short gastric collaterals in 11 children (55%). Spleno-portal shunt was seen in 2(10%) patients. Ascites was detected in 4(20%) patients. In all children with CLD, splenoportal axis was patent. Left gastric collaterals were seen in 5(10)(50%) and short gastric collateral were visualized in 4(40%) patients. Liver parenchyma was hyperchoic in 10(10)(100%) of cases and ascites was also seen in 10(10)(100%) patients (Table 3).

The most common site of block in the EHPVO was at the junction of superior mesenteric vein and splenic vein.
9(45%), followed by the block at the distal part of portal vein 6(30%). The complete splenoportal axis was blocked in 4(20%) patients. The splenic vein block was in one (5%) case (Table 4).

### DISCUSSION

Portal hypertension usually develops as a result of obstruction to the portal blood flow which could be either in the liver (intrahepatic) or outside the liver. Intrahepatic type of block is usually caused by conditions affecting the liver parenchyma while the extrahepatic type is usually caused by obstruction in the venous channels which together comprises the portal tract. Portal hypertension later on will cause an increase of pressure in the portal venous system, with its consequences like enlargement of spleen and opening up of porto-systemic collateral channels. Most appropriate diagnostic method would be measurement of pressure in the venous system either by measuring splenic pulp pressure (more than 15 mm Hg) or measuring pressure in esophageal varices (more than 30 cm of saline) through endoscopy (Boyer 1982).

However, in the clinical practice, as these methods are invasive and difficult, so usually not recommended for diagnosis. So, the diagnosis of portal hypertension entirely depends on demonstration of collaterals either by radiology or by endoscopy. Endoscopy of UGI tract and ultrasonography is more helpful for demonstration of varices. In this study 20 out of 30 children (66.6%) had intrahepatic portal hypertension, ie a block in the main portal vein and /or its branches which is similar with other studies which ranges from 57.7% to 85.7%. The incidence of chronic liver disease resulting in portal hypertension as 33.3% in this study. Seven cases (23%) were of chronic active hepatitis, 2 cases (6.6%) of postnecrotic cirrhosis, one case (3.3%) of Wilson’s disease. This incidence of chronic liver disease (33.3%) is in concordance with earlier workers who reported this 22 to 30% cases. The major causes of chronic liver disease resulting in portal hypertension reported in the Western studies are Alpha-1 antitrypsin deficiency, secondary biliary cirrhosis and idiopathic cirrhosis from 3.7 to 4.5%, 3.7 to 30% and 11 to 30% respectively. In Indian scenario however, ICC is reported to be commonest (53%) cause of CLD followed by chronic active hepatitis (6.3%) and cryptogenic cirrhosis (5.3%) and outflow venous obstruction (2.6%).

With ultrasonography, a correct diagnosis of EHPVO and CLD (Hyperechoic liver) was made in 20/20 (100%) and 10/10 patients respectively. USG proved very useful in identification of the site and nature of block in the splenoportal axis. In our study ultrasonography revealed that block at the junction of superior mesenteric vein and splenic vein was the most common site (45%) followed by distal part of portal vein (30%). These results were similar to previous studies. A successful diagnosis of portal cavernoma was possible in 50% of EHPVO patients. These results are in conformity with an earlier study. The frequency of detection of gastroesophageal collaterals with USG is reported to range from 12 to 85%, depending on the size of the coronary vein and the esophageal varices. In this study USG detected short and left gastric collaterals in 55% and 60% cases which is

<table>
<thead>
<tr>
<th>Site of blocks</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Block at the junction of superior mesenteric vein and splenic vein</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>II Block at the distal part of portal vein.</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>III Complete splenoportal axis block</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>IV Isolated splenic vein block.</td>
<td>2</td>
<td>10</td>
</tr>
</tbody>
</table>

### Table 3: Ultrasonographic findings in patients with portal hypertension.

<table>
<thead>
<tr>
<th>Ultrasonographic Findings</th>
<th>EHPVO (n=20)</th>
<th>CLD (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main portal vein not visualized</td>
<td>17</td>
<td>85</td>
</tr>
<tr>
<td>Right branch not visualized</td>
<td>15</td>
<td>75</td>
</tr>
<tr>
<td>Left branch not visualized</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td>Portal vein cavernoma</td>
<td>15</td>
<td>75</td>
</tr>
<tr>
<td>Left gastric collaterals</td>
<td>12</td>
<td>60</td>
</tr>
<tr>
<td>Short gastric collaterals</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>Splenorenal shunt</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Ascites</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>Hyperechoic liver</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Table 4: Pattern of blocks observed by ultrasonography Patients with EHPVO (n=21).

similar to the previous studies.\(^\text{10}\) On upper GI endoscopy, GI Hemorrhage was the commonest symptom in extrahepatic group 95\%(19/20). This observation is comparable with the other studies.\(^\text{4,7,11,12}\)

Similarly previous studies reported that children with EHPVO bleed at a young age 49\% of children with EHPVO bleed before the age of 3 years 4, however in our study the mean age (±S.D.) at the time of presentation was 4 years 4 months while the mean age at the time of first bleed was 3.7±3.15 years. In children with Chronic liver disease 60\%(6/10) had upper GI bleeding, the mean age of presentation was 8.1±2.92 year, patients with Chronic liver disease, therefore bleed at a significantly later age. These observations are in concordance with earlier studies.\(^\text{13}\) Upper GI hemorrhage was more frequent in EHPVO in comparison with CLD (95\% Vs 60\%). Similar observations (80\% Vs 32\%) have been made earlier.\(^\text{4}\) There was a definite relationship between the size of varices and the risk of upper GI hemorrhage 3/4 cases with grade IV varices, 14/15 cases with grade III varices, 7/9 cases with grade II varices, 1/2 cases with grade I varices bleed. Almost similar observation has been made in adults with portal hypertension by other studies.\(^\text{14}\) All our patients with a major endoscopic sign, i.e. cherry red spot, i.e. varices that appeared covered with small dilated blood vessels had variceal bleeding. In 55\% of our patients with EHPVO had nonspecific liver changes in the form of mild portal fibrosis, mononuclear infiltrate and Kupffer cell hyperplasia. In the remaining (45\%), no significant pathology found.\(^\text{7}\) Amongst nine children with CLD, 7 had changes of chronic active hepatitis, i.e loss of limiting plate, piece meal necrosis and fibrosis, in remaining two patients finding of post-necrotic cirrhosis were observed.

**CONCLUSION**

Extra hepatic portal vein obstruction is the commonest cause followed by intrahepatic obstruction (Chronic liver diseases). Ultrasonography is a useful non-invasive investigation for the diagnosis, type of portal hypertension and the site of block. Upper gastrointestinal endoscopy is very useful in the diagnosis of severity of portal hypertension as well as detecting the source of upper gastrointestinal bleeding along with therapeutic treatment.

**Funding:** No funding sources  
**Conflict of interest:** None declared  
**Ethical approval:** The study was approved by the Institutional Ethics Committee

**REFERENCES**
