ORIGINAL RESEARCH PAPER INTERNATIONAL JOURNAL OF PURE MEDICAL RESEARCH

**KEYWORDS:** Extra hepatic portal venous obstruction, Thrombophilia

# A STUDY ON CLINICO-LABORATORY PROFILE IN CHILDREN WITH EXTRA HEPATIC PORTAL VENOUS OBSTRUCTION

Volume - 9, Issue - 5, May- 2024 ISSN (O): 2618-0774 | ISSN (P): 2618-0766

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INTERNATIONAL JOURNAL OF PURE MEDICAL RESEARCH

**ABSTRACT-**

**Context:** EHPVO is a common cause of portal hypertension in children. Umbilical sepsis, umbilical vein catheterization, history of NICU admissions, dehydration and abnormal thrombophilic parameters are common etiological causes with most common presentation as hematemesis followed by splenomegaly. Objective: To study clinical profile and laboratory features at presentation in children with EHPVO. Method: Children with clinical profiles suggestive of EHPVO like upper GI bleeding and abdominal lump attending the pediatric OPD, and specialty OPD and admitted in wards were enlisted. A detailed history was taken to find out the risk factors causing EHPVO. Clinical examination and anthropometric evaluation of the cases were recorded. Complete hemogram, anemia profile, liver function tests, levels of thrombophilic factors like protein C and S, antithrombin III, and factor V were done. Result: Among 50 participants, 27 (52.9%) were males and 23 (47.1%) were females and the mean age group being 8.09 ± 3.09 years. Hematemesis (92.20%) and splenomegaly (90.20%) were the most common clinical features and growth retardation was seen in 64% of cases. Anemia was found in all the cases, Leukopenia (45.1%), thrombocytopenia (82.4%), and folate deficiencies (15.7%). Deranged SGOT, ALP and PT-INR were observed in 72.54%, 76.4% and 11.7% cases. Most common etiology observed was idiopathic (45%) followed by abnormal thrombophilic parameters in 33.3%, umbilical sepsis in 25.49%, umbilical vein catheterization and history of NICU admissions in 19.6% and dehydration due to recurrent diarrhea and vomiting in 11.7%. while amongst the thrombophilic factors studied protein S deficiency (15.69%) was found to be the most common cause followed by protein C deficiency and antithrombin III deficiency (13.73% each) and factor 5 deficiency in 7.84% cases. Conclusion: The etiology of EHPVO in the majority of patients remain still unclear. It is commonly associated with impaired somatic growth. The risk of EHPVO increases in the presence of thrombophilia, umbilical sepsis, umbilical vein catheterisation and dehydration. The investigations showed abnormal anemia profile and liver function test.

**INTRODUCTION-** Portal hypertension is abnormally increased portal venous pressure in the portal vein and its branches.1 It is defined as a hepatic venous pressure gradient greater than 5 mmHg.2,3 The causes for portal hypertension are classified as originating in the portal venous system before it reaches the liver (prehepatic causes), within the liver (intrahepatic) or between the liver and the heart (post-hepatic).[4]

Extra- hepatic portal vein obstruction (EHPVO) is a vascular disorder of liver defined by obstruction of extra hepatic portal vein with or without involvement of intrahepatic portal vein, splenic vein or superior mesenteric vein. EHPVO is one of the most common cause of portal hypertension in children (70-80%) of all types of Portal hypertension[4,5].

Clinical presentation: Acute form - abdominal pain, low grade fever, transient ascites (10- 20%) following surgery or bleed. Chronic form – UGI - bleed (MC) in 60 – 70%5,6 splenomegaly, growth retardation ( 51% ), hypersplenism (5 -10%), anaemia , biliopathy (90- 100%) due to bile duct compression from dilated venous collaterals due to portal biliopathy, immunological defects usually being cell mediated defects, mild hepatic dysfunction, cognitive and psychomotor changes.[6,7,8]

EHPVO is a major cause of Portal hypertension (54%) and upper gastrointestinal bleeding in children (68–84%) from the developing world,9,10 20-30% of all variceal bleeds in India, with most of these children belonging to low and lower-middle socio-economic strata. The usual age of presentation is 4-7 years.

Etiologically, EHPVO is a heterogenous disease and the cause varies with respect to age and geographic location. Etiological factors being Congenital anomalies: Portal vein atresia, associated cardiovascular, urinary, limb anomalies, cleft lip & palate Systemic prothrombotic states: Genetic: MTHFR deficiency, prothrombin gene mutation, factor-V Leiden, protein-C, S, antithrombin-III deficiency,5 Acquired: Diarrhea, Nephrotic syndrome, oral contraceptives, ACLA or APLA Local conditions, Infections: Omphalitis, liver abscess, pyelephlebitis, pancreatitis, cholangitis, neonatal sepsis, necrotizing enterocolitis Surgery: Billroth-II.[9]

Most commonly the entire length of the portal vein is obstructed by the thrombus, the most common site being the portal vein formation [39%], followed by the entire length of portal vein [34%], splenic vein [16%] and the entire spleno-renal axis [11%].9 Portal cavernoma is the pathognomonic feature of chronic EHPVO where the portal vein is grossly replaced by a sheath of variably sized tortuous channels in a connective tissue matrix.

**METHODS:** This was a hospital-based prospective observational study was conducted in the Department of Pediatrics at Sawai Man Singh Medical College & Hospital, Jaipur from May 2021 to May 2022 in 50 subjects. The sample size was calculated at a 95% confidence level alpha error of 0.05 expecting 85.9% of children of EHPVO presenting with hemorrhage. Children with clinical profiles suggestive of EHPVO like upper GI bleeding and abdominal lump were enlisted and a detailed history was taken. Clinical examination and anthropometric evaluation were done and recorded. Complete hemogram and anemia profile, liver function tests were done. Levels of thrombophilic factors like protein C and S, antithrombin III, and factor V were done. The data thus collected was recorded in a preformed proforma and was used for further correlation and data analysis.

**INCLUSION CRITERIA** were children < 18 years of age,USG and Triple-phase CT findings suggestive of EHPVO. While exclusion criteria included patients diagnosed with other causes of portal

hypertension like NAFLD, hepatitis, budd-chiari syndrome and children with bleeding disorders not involving the liver.

**STATISTICAL ANALYSIS:** The statistical analysis was carried out using the Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, version 22.0 for Windows) and MS - Excel (Microsoft Inc. version 2010). Sample data collected are described in terms of mean, standard deviation, frequencies a number of cases, and percentages were appropriate. The means of two groups were compared using Student's t-test whereas the means of several groups were compared using one-way ANOVA. Categorical data were compared using the Chi-square test or Fisher exact test whichever was applicable. The proportions were compared using the Z test. A probability value (p- value) less than 0.05 was considered statistically significant with a 95% confidence interval (two-sided).

**RESULT:**

**Table1: Baseline demographic of the patients**

| | | | SEX | | Total |
|---|---|---|---|---|---|
| | | | Female | Male | |
| Age | 0-5 years | Count | 2 | 11 | 13 |
| | | % of Total | 3.9% | 21.6% | 25.5% |
| | 6-10 years | Count | 9 | 15 | 24 |
| | | % of Total | 17.6% | 29.4% | 47.1% |
| | 11-18 years | Count | 13 | 1 | 14 |
| | | % of Total | 25.5% | 2.0% | 27.5% |
| Total | | Count | 24 | 27 | 51 |
| | | % of Total | 47.1% | 52.9% | 100.0% |

| | Mean | sd | Median | Minimum | Maximum |
|---|---|---|---|---|---|
| AGE | 8.09 | 3.60 | 8.00 | 1 | 14 |

As shown in table, majority of patients in our study belongs to 6-10 years (47.1%) followed by 27.5% in 11-18 years age group and the least (25.5%) in < 5 years.

**Table 2: Etiology of EHPVO and their age and gender wise distribution**

| | | AGE | | | SEX | | Total |
|---|---|---|---|---|---|---|---|
| | 0-5 years | 6-10 years | 11-18 years | | Female | Male | |
| umbilical vein catheterisation | N | 3 | 6 | 1 | 2 | 8 | 10 |
| | % | 23.1% | 25.0% | 7.1% | 8.3% | 29.6 % | 19.6% |
| umbilical vein sepsis in past | N | 4 | 8 | 1 | 4 | 9 | 13 |
| | % | 30.8% | 33.3% | 7.1% | 16.7% | 33.3 | 25.5% |
| NICU admission for sepsis | N | 4 | 5 | 1 | 2 | 8 | 10 |
| | % | 30.8% | 20.8% | 7.1% | 8.3% | 29.6 % | 19.6% |
| Intra-abdominal infection | N | 3 | 3 | 0 | 3 | 3 | 6 |
| | % | 23.1% | 12.5% | 0.0% | 12.5% | 11.1 % | 11.8% |
| dehydration | N | 3 | 3 | 0 | 1 | 5 | 6 |
| | % | 23.1% | 12.5% | 0.0% | 4.2% | 18.5 % | 11.8% |

On the basis of history, umbilical sepsis was found to be the most common factor responsible for EHPVO in 25.5% patients followed by umbilical vein catheterization in 19.6% and history of NICU admissions in 19.6%. History of repeated admission for recurrent diarrhea or vomiting leading to dehydration was observed in 11.6% patients

**Table 3: Clinical profile of patients with EHPVO:**

| | | | Age | | | SEX | | Total |
|---|---|---|---|---|---|---|---|---|
| | | | 0-5 years | 6-10 years | 11-18 years | Female | Male | |
| Pallor | | Count | 10 | 21 | 13 | 23 | 21 | 44 |
| | | % within Age | 76.9% | 87.5% | 92.9% | 95.8% | 77.8% | 86.3% |
| Upper GI bleeding episodes | 0 | Count | 2 | 2 | 0 | 1 | 3 | 4 |
| | | % within Age | 15.4% | 8.3% | 0.0% | 4.2% | 11.1% | 7.8% |
| | 1 | Count | 4 | 3 | 0 | 3 | 4 | 7 |
| | | % within Age | 30.8% | 12.5% | 0.0% | 12.5% | 14.8% | 13.7% |
| | 2 | Count | 0 | 1 | 0 | 0 | 1 | 1 |
| | | % within Age | 0.0% | 4.2% | 0.0% | 0.0% | 3.7% | 2.0% |
| | 3 | Count | 7 | 18 | 14 | 20 | 19 | 39 |
| | | % within Age | 53.8% | 75.0% | 100.0% | 83.3% | 70.4% | 76.5% |
| Lower GI bleeding episodes | 0 | Count | 5 | 6 | 3 | 8 | 6 | 14 |
| | | % within Age | 38.5% | 25.0% | 21.4% | 33.3% | 22.2% | 27.5% |
| | 1 | Count | 3 | 1 | 0 | 1 | 3 | 4 |
| | | % within Age | 23.1% | 4.2% | 0.0% | 4.2% | 11.1% | 7.8% |
| | 2 | Count | 1 | 1 | 0 | 0 | 2 | 2 |
| | | % within Age | 7.7% | 4.2% | 0.0% | 0.0% | 7.4% | 3.9% |
| | 3 | Count | 4 | 16 | 11 | 15 | 16 | 31 |
| | | % within Age | 30.8% | 66.7% | 78.6% | 62.5% | 59.3% | 60.8% |
| Short stature | | Count | 6 | 10 | 6 | 12 | 10 | 22 |
| | | % within Age | 46.2% | 41.7% | 42.9% | 50.0% | 37.0% | 43.1% |
| petechiae | | Count | 5 | 8 | 9 | 13 | 9 | 22 |
| | | % within Age | 38.5% | 33.3% | 64.3% | 54.2% | 33.3% | 43.1% |
| recurrent infections | | Count | 4 | 10 | 7 | 9 | 12 | 21 |
| | | % within Age | 30.8% | 41.7% | 50.0% | 37.5% | 44.4% | 41.2% |
| pain abdomen | | Count | 3 | 9 | 6 | 11 | 7 | 18 |
| | | % within Age | 23.1% | 37.5% | 42.9% | 45.8% | 25.9% | 35.3% |
| abdominal lump | | Count | 2 | 7 | 5 | 6 | 8 | 14 |
| | | % within Age | 15.4% | 29.2% | 35.7% | 25.0% | 29.6% | 27.5% |
| growth retardation | | Count | 7 | 11 | 7 | 14 | 11 | 25 |
| | | % within Age | 53.8% | 45.8% | 50.0% | 58.3% | 40.7% | 49.0% |

As shown in table 3, hematemesis (92.2%) was the most common symptom reported by the patients, followed by pallor (86.3%), melena (45.2%) and short stature (43.1 %). Pain abdomen (35.3%) and abdominal lump (27.5%) were among the least reported symptoms in EHPVO patients. Amongst all the patients, females had more complains of bleeding episodes, pallor, petechiae, short stature and pain abdomen than males while history of recurrent infections and complain of abdominal lump were more common amongst males. Adolescent group of 11-18 years were the most symptomatic amongst the three age groups The presenting symptoms of the patients in our study was neither found to be statistically significant for age nor for sex.

**Table 4: Signs observed in patients with EHPVO**

| Presentation | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| SIGNS | | | Age | | | | SEX | | | Total |
| | | | 0-5 years | 6-10 years | 11-18 years | Chi-Square /Fisher's test | Female | Male | Chi-Square/Fisher's test | |
| | | | | | | P value | | | | |
| HEPATOMEGALY | No | N | 10 | 16 | 10 | 0.805 | 20 | 16 | 0.073 | 36 |
| | | % | 76.9% | 66.7% | 71.4% | | 83.3% | 59.3% | | 70.6% |
| | Yes | N | 3 | 8 | 4 | | 4 | 11 | | 15 |
| | | % | 23.1% | 33.3% | 28.6% | | 16.7% | 40.7% | | 29.4% |
| SPLEENOMEGALY | Absent | N | 1 | 3 | 1 | 0.022* | 2 | 3 | 0.896 | 5 |
| | | % | 7.7% | 12.5% | 7.1% | | 8.3% | 11.1% | | 9.8% |
| | Mild | N | 6 | 3 | 1 | | 4 | 6 | | 10 |
| | | % | 46.2% | 12.5% | 7.1% | | 16.7% | 22.2% | | 19.6% |
| | Moderate | N | 6 | 16 | 7 | | 14 | 15 | | 29 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | % | 46.2% | 66.7 % | 50.0% | | 58.3 % | 55.6 % | | 56.9% |
| | Mass ive | N | 0 | 2 | 5 | | 4 | 3 | | 7 |
| | | % | 0.0% | 8.3% | 35.7% | | 16.7 % | 11.1 % | | 13.7% |
| ASCITIS | Absent | N | 10 | 17 | 6 | 0.078 | 15 | 18 | 0.771 | 33 |
| | | % | 76.9% | 70.8 % | 42.9% | | 62.5 % | 66.7 % | | 64.7% |
| | Mild | N | 2 | 3 | 2 | | 3 | 4 | | 7 |
| | | % | 15.4% | 12.5 % | 14.3% | | 12.5 % | 14.8 % | | 13.7% |
| | Mod erate | N | 1 | 3 | 3 | | 4 | 3 | | 7 |
| | | % | 7.7% | 12.5 % | 21.4% | | 16.7 % | 11.1 % | | 13.7% |
| | Mass ive | N | 0 | 1 | 3 | | 2 | 2 | | 4 |
| | | % | 0.0% | 4.2% | 21.4% | | 8.3% | 7.4 % | | 7.8% |

*significant at p value <0.05

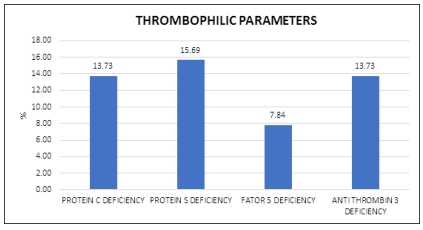
On clinical examination splenomegaly was found in 90.20% patients which had female predominance. The study depicted statistically significant difference for splenomegaly among cases for different age groups (P value = 0.022). Ascites was found in 36% patients and hepatomegaly in only 29.4% cases.

**Table 5: Anemia profile in EHPVO patients**

| | | | | | AGE | | SEX | Total |
|---|---|---|---|---|---|---|---|---|
| | | | 0-5 years | 6-10 years | 11-18 years | Female | Male | |
| Anemia | Normal Hb | N | 0 | 0 | 0 | 0 | 0 | 0 |
| | | % | 0.0% | 0.0% | 0.0% | 0.0% | 0.0% | 0.0% |
| | Mild | N | 3 | 3 | 1 | 1 | 6 | 7 |
| | | % | 23.1 % | 12.5 % | 7.1% | 4.2% | 22.2% | 13.7 % |
| | Moder ate | N | 4 | 7 | 4 | 9 | 6 | 15 |
| | | % | 30.8 % | 29.2 % | 28.6 % | 37.5 % | 22.2% | 29.4 % |
| | Severe | N | 6 | 14 | 9 | 14 | 15 | 29 |
| | | % | 46.2 % | 58.3 % | 64.3 % | 58.3 % | 55.6% | 56.9 % |
| TLC | Normal | N | 12 | 13 | 3 | 9 | 19 | 28 |
| | | % | 92.3 % | 54.2 % | 21.4 % | 37.5 % | 70.4% | 54.9 % |
| | Leucop enia | N | 1 | 11 | 11 | 15 | 8 | 23 |
| | | % | 7.7% | 45.8 % | 78.6 % | 62.5 % | 29.6% | 45.1 % |
| Platlets | Normal | N | 6 | 3 | 0 | 2 | 7 | 9 |
| | | % | 46.2 % | 12.5 % | 0.0% | 8.3% | 25.9% | 17.6 % |
| | Throm bocyto penia | N | 7 | 21 | 14 | 22 | 20 | 42 |
| | | % | 53.8 % | 87.5 % | 100.0 % | 91.7 % | 74.1% | 82.4 % |
| B12 Deficiency | No | Count | 12 | 20 | 8 | 18 | 22 | 40 |
| | | % within Age | 92.3 % | 83.3 % | 57.1 % | 75.0 % | 81.5% | 78.4 % |
| | Yes | Count | 1 | 4 | 6 | 6 | 5 | 11 |
| | | % within Age | 7.7% | 16.7 % | 42.9 % | 25.0 % | 18.5% | 21.6 % |
| Folate deficiency | No | Count | 12 | 20 | 11 | 20 | 23 | 43 |
| | | % within Age | 92.3 % | 83.3 % | 78.6 % | 83.3 % | 85.2% | 84.3 % |
| | Yes | Count | 1 | 4 | 3 | 4 | 4 | 8 |
| | | % within Age | 7.7% | 16.7 % | 21.4 % | 16.7 % | 14.8% | 15.7 % |

According to our study anemia was found in 100% patients of which severe anemia (7gm/dl) was found in 56.9% patients, moderate anemia (7-10gm/dl) in 29.4% and mild anemia(10-12gm/dl) in 13.7% patients. Leukopenia (4000/mm3 ) in 45.1% patients, thrombocytopenia (1.5 ×103 /mm3 ) in 82.4% patients and B12 and folate deficiency in 21.6% and 15.7% respectively.

**Figure 1:** Thrombophilic Parameters




On investigating the patients for thrombophilic parameters protein S deficiency was found to be the most common risk factor (15.69%) followed by protein C deficiency in 13.73% patients. Factor 5 deficiency was found in least number of cases of only 7.84% patients.

## DISCUSSION

As discussed in table1 In our study, a total of 27 males (52.94%) and 23 females (47.06%) were recruited. A maximum of 15 (29.4%) males out of 27 belonged to 6–10 years age group. Among females the maximum cases of 13 (25.5%) were seen in 11-18 years age group. The mean age of our study group was 8.09 ± 3.6 years with the youngest case being 9 months while the maximum age was 14 years. Of all the cases two were infants (<1 year). Alina grama et al (2021)11 evaluated 63 EHPVO children in Romania and reported that 33 (52.38%) to be males with 5.14 ± 4.9 years mean age. Singh Swati et al (2021)12 in wadia hospital, Mumbai studied 81 patients, 51 (62.29%) of which were males. Batia Weiss et al (2010)1 studied 30 children of which 13 (43.3%) were males and found 4.8 ± 4.6 years as the mean age.

In our study the most common risk factor identified for EHPVO was history of umbilical sepsis in 13 (25.5%) patients out of a total of 50 cases, followed by past history of umbilical vein catheterisation in 10 (19.6%) and NICU admission for sepsis in10 (19.6%). This was similar to a study by Jena SK et al (2017)13 where neonatal umbilical sepsis was identified as the commonest risk factor in 10 (16.1%) patients and umbilical vein catheterization contributed to 9.6% of the cases. In contrast to our study Grama A. et al (2021)11 in their study reported umbilical vein catheterization to be responsible for 72.02% cases followed by bacterial infections in (47.62%) and dehydration in 19.08% cases.

our study reported the most common clinical feature of EHPVO to be hematemesis (92.15%), followed by anaemia (72.55%) and abdominal lump (52.94%) and pain abdomen (35.29%), ascites (34.5%) to be the least common features. Amongst upper GI bleeds maximum number of cases 39 (76.5%) presented with ≥3 episodes. This was similar to the study by Grama A. et al (2021)11 who also reported maximum number of cases (35%) with ≥3 episodes of upper GI bleeding. Chawla Y. et al (2014)14 evaluated abdominal pain (91%), ascites (38%), splenomegaly (37%) and growth retardation (50%) patients which was in contrast to our study.

In reference to table 4, our study found splenomegaly in 46 (90.2%) of which massive splenomegaly was seen in 12 (21%) children. This

was similar to the study by Goyal S. et al (2014)15 who reported splenomegaly in 49 out of 53 patients (92.5%), upper GI bleeding in 46 (86.8%) children and growth retardation in 30 (56.6%) EHPVO patients. Weiss B et al (2010)1 depicted splenomegaly only in 13 (43.3%) patients.

Mean Hb levels in our study was 6.78 ± 2.46 gm/dL with severe anaemia found in all the age groups. Females were reported to be more severely anaemic 6.12 ± 2.41 gm/dl as compared to males. Anaemia could be possibly due to frequent variceal bleeds before presentation and poor nutritional status. Mean total leukocyte count (TLC) was 5,164.7 ± 3,060.83/mm3 and leukopenia was found in 45.1% patients (p value=0.002) with maximum number of patients in 11-18 years age group. Leukopenia and lymphopenia were found to be statistically significant for age. This finding may be due to progressive hyper splenic state. This was similar to the results reported by Jean SK et al (2017)13 in their study where anaemia was found in 90.3% and leukopenia in 40.3% patients with EHPVO. Singh I.K. et al (2011)16 reported mean haemoglobin of 9.48 ± 1.45 gm/dl and the mean haemoglobin ranged from 6.6 to 11.8 gm/dl and mean TLC count reported was 3,608.33 ± 1,528.83/mm3. Thrombocytopenia was found in 82.4% cases in our study with mean platelet count as 99,176.5 ± 58,212.27/mm3 (p value= 0.005) which is similar to that evaluated in the study by Singh I.K. et al (2011)16 where mean platelet count was found to be 87,083.33 ± 44,626.87/mm3 in EHPVO patients. While In contrast Jean S.K et al (2017)13 had reported thrombocytopenia only in 40.3% cases. Vitamin B12 and folate deficiency was found in 21.6% (p value=0.140) and 15.7% (p value= 0.035) cases respectively with maximum number of cases found in < 5 years age group. The deficiencies were predominantly seen in males as 18.5% and 14.8% respectively of Vitamin B12 and folate. With increasing age the folate deficiency was found to increase in the patients and was found to be statistically significant for age (p value= 0.035) in our study.

Amongst the thrombophilic factors studied protein S deficiency (15.69%) was found to be the most common cause followed by protein C deficiency and antithrombin III deficiency which were seen in 13.73% each, factor 5 deficiency was reported only in 7.84% of the cases in our study. Jean S.K et al (2017)13 in their study found protein C deficiency in 19.1% patients followed by protein S deficiency in 17% cases and anti thrombin III deficiency in 12.7% cases of the 47 patients studied.

Limitation of our study was being a single-centre study with small sample size. Our patients had severe clinical presentation and this could be due to referral bias as our centre is tertiary care referral centre. Our study has not included follow up and outcome of patients so longterm follow-up studies would be more fruitful in assessing the profile and risk factors of EHPVO in children. We also could not assess the genetic mutations due to financial constraints.

## CONCLUSION

EHPVO is a common cause of portal hypertension in children. Umbilical sepsis, umbilical vein catheterization, history of NICU admissions, dehydration due to recurrent diarrhea and vomiting, and abnormal thrombophilic parameters (Protein S deficiency (15.69%) followed by Protein C deficiency and antithrombin III deficiency (13.73%) each) are common etiological causes but we could not find the predisposing cause in 45% of patients. The most common presentation was hematemesis (92.2%) followed by pallor (86.3%) and short stature (43.1%). The abdominal lump was the least common presenting complaint seen in 27.5% of children. Growth retardation was seen in 64 % of children and 43.1% were stunted. Splenomegaly was seen in 90.2% of children. Anemia was seen in all children with EHPVO, whereas Pancytopenia was observed in 45% of children.

## REFERENCES


1. Weiss B, Shteyer E, Vivante A, Berkowitz D, Reif S, Weizman Z, et al. Etiology and long-term outcome of extrahepatic portal vein obstruction in children. World J Gastroenterol. 2010;16:4968–72. "Portal Hypertension - Liver and Gallbladder Disorders". MSD Manual Consumer Version. Retrieved 11 March 2021.
2. D'Souza, Donna. "Portal hypertension | Radiology Reference Article | Radiopaedia.org". Radiopaedia. Retrieved 11 March2021 –"Portal hypertension | Disease | Overview | Genetic and Rare Diseases Information Center (GARD) an NCATS Program"
3. . rarediseases.info.nih.gov. Retrieved 2016-01-08.
4. Sarin SK, Sollano JD, Chawla YK, Amarapurkar D, Hamid S, Hashizume M, et al. Consensus on Extrahepatic portal vein obstruction. Liver International. 2006;26:512-9.
5. Abd El-hamid N, Taylor RM, Marinello D, Mufti GJ, Patel R, MieliVergani G, et al. Aetiology and management of extrahepatic portal vein obstruction in children: King's College Hospital experience. J Pediatr Gastroenterol Nutr. 2008;47:630–4. 10.
6. Gürakan F, Eren M, Koçak N, Yüce A, Ozen H, Temizel IN, et al. Extrahepatic portal vein thrombosis in children: Etiology and longterm follow-up. J Clin Gastroenterol. 2004;38:368–72. 11.
7. . Bloom, S.; Kemp, W.; Lubel, J. (2015-01-01). "Portal hypertension: pathophysiology, diagnosis and management". Internal Medicine Journal. 45 (1): 16–26.
8. Peter L, Dadhich SK, Yachha SK. Clinical and laboratory differentiation of cirrhosis and extrahepatic portal venous obstruction in children. J Gastroenterol Hepatol. 2003;18:185-9.
9. Fagundes ED, Ferreira AR, Roquete ML, Penna FJ, Goulart EM, Figueiredo Filho PP, Bittencourt PF, Carvalho SD, Albuquerque W. Clinical and laboratory predictors of esophageal varices in children and adolescents with portal hypertension syndrome. J Pediatr Gastroenterol Nutr 2008;46(2):178–183
10. Primignani M. Portal vein thrombosis, revisited. Dig Liver Dis. 2010;42: 163–70.
11. Grama A, Pîrvan A, Sîrbe C, Burac L, Ştefănescu H, Fufezan O, Bordea MA, Pop TL. Extrahepatic Portal Vein Thrombosis, an Important Cause of Portal Hypertension in Children. J Clin Med. 2021 Jun 18;10(12):2703. doi: 10.3390/jcm10122703. PMID: 34207387; PMCID: PMC8235032.
12. Singh, S., Bhamre, R., Shetty, N.S., Meshram, H., Shah, S.K., & Shah, I.B. (2021). Correlation of endoscopic findings with Doppler ultrasound in portal hypertension in children. Clinical and Experimental Hepatology, 7, 191 – 1
13. Jena, S.K., Mohanty, M., Patra, U.C., & Behera, S. (2017). A study on demographic and clinical profile of children with extra hepatic portal venous obstruction and with special reference to thrombophilic factors. International Journal of Community Medicine and Public Health, 4, 640-645.
14. Chawla YK, Bodh V. Portal vein thrombosis. J Clin Exp Hepatol. 2015 Mar;5(1):22-40. doi: 10.1016/j.jceh.2014.12.008. Epub 2015 Jan 6. PMID: 25941431; PMCID: PMC4415192.
15. Goyal S, Dixit VK, Jain AK, Mishra OP, Jena SK, Ghosh J. Revisiting extra hepatic portal vein obstruction in children from the north Indian gangetic plain. Indian J Pediatr. 2014 May;81(5):429-33. doi: 10.1007/s12098-013- 1257-7. Epub 2013 Oct 10. PMID: 241138
16. Singh, I. K., Bhatnagar, V., Gupta, A. K., & Seith, A. (2011). Correlation of splenic volume with hematological parameters, splenic vein diameter, portal pressure and grade of varices in extrahepatic portal vein obstruction in children. Pediatric Surgery International, 27(5), 467– 471. doi:10.1007/s00383-010-2847-3