Original article

Clinical evaluation & follow up of 69 Iraqi patients with Portal Hypertension

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Abstract

Background: Portal hypertension (P.H.T.) is a serious medical issue, it results from pathological increase in portal circulation pressure.-Variceal bleeding is responsible for 10-13% of upper gastrointestinal (G.I.T.) bleeding; Mortality after variceal bleeding reaches 50% during first six weeks in cirrhotic patients.

Objective: To study P.H.T. in Iraqi patients.

Methods: during the period of three months patients discovered to have esophageal & gastric varices were fully assessed clinically & endoscopically.

Results: During the study period 69 patients were found to have P.H.T.

91.3% of patient's with P.H.T have hepatic etiology, 47.6% of these were due to hepatitis B, C & alcoholism, 69.5% presented with hematemesis , advanced liver disease was found in 88.8% .

Thrombocytopenia, advanced variceal grade, and red signs were significantly more in bleeders group, while child's class was not significant.

Conclusions:

1. Hepatic etiology was most prevalent in P.H.T patients , with hepatitis B,C and alcoholism important preventable causes .
2. Hematemesis is the commonest presentation .
3. Advanced liver disease was associated more with bleeding .
4. Thrombocytopenia, higher variceal grade, and red signs were associated significantly with bleeding in P.H.T.

Introduction:

Portal hypertension (P.H.T) is a frequent clinical syndrome caused by pathological increase in hydrostatic pressure in the portal venous system.

The importance of this syndrome is defined by the frequency of seriousness and it's complication.

Any pathological processes that interfere with blood flow at any level within the portal system can provoke P.H.T., this may be hepatic or extra- hepatic.

Schistosomiasis is the commonest cause of P.H.T. worldwide [1], while liver cirrhosis is the commonest cause in Europe and North America[2].

P.H.T. is the most frequent clinical syndrome of cirrhosis; it is present in more than 80% of cirrhotic patients [2].

Bleeding is the most notorious

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complication of P.H.T., it's the cause of upper gastrointestinal bleeding in 10% of patients[3] while in cirrhotic patients it's responsible for more than 80% of upper gastrointestinal bleeding leading to initial mortality of 25-50%.

Bleeding due to P.H.T is mostly from esophageal varices, but gastric varices and portal hypertensive gastropathy contribute to this also. The current study was conducted aiming to throw a light on P.H.T. in Iraqi patients, and studying various factors associated with P.H.T. and its complications.

Patients and Methods

During a period of three months (from the first of June to the end of August 2000), 69 patients discovered to have esophageal and/or gastric varices during upper endoscopy, carried at the Center of Gastrointestinal and Liver Diseases in Baghdad, referred for different causes, were included in this study.

Each patient was fully assessed including history and physical examination, the Child's score was assessed according to the Child-Pugh classification[1].

Description of endoscopic findings were as follow :

1. **Esophageal varices:** These were described according to Paquet into 4 grades[4].
   - **Grade I:** Small varices without luminal prolapse .
   - **Grade II:** Moderate size showing luminal prolapse with minimal obscuring of gastro-esophageal junction.
   - **Grade III:** Large varices showing prolapse substantially obscuring the gastro-esophageal junction.
   - **Grade IV:** Very large varices completely obscuring the junction.

Red signs were observed and include cherry- red spots, red wale markings (longitudinal red streaks) and the varix-on-varix sign[4].

2. **Gastric varices:** These are classified by Sarin et.al. into the following[5]:
   - **A- Gastroesophageal varices (G.O.V.):** These are always associated with esophageal varices and extend beyond the gastroesophageal junction, they are subdivided into:
     - **(1) -G.O.V. type 1:** These extend as a continuation of esophageal varices along the lesser curvature of stomach, and are more or less straight.
     - **(2) -G.O.V. type 2**: These extend into the fundus of stomach, these are long and tortuous.
   - **B- Isolated Gastric varices (I.G.V.):** These are seen in the absence of esophageal varices and included:
     - **(1) -I.G.V. type 1:** These are located in the fundus and fall short of the cardia by few centimeters.
     - **(2) -I.G.V. type 2**: These include ectopic isolated varices present anywhere in the stomach (such as in the antrum, pylorus or body).
3. **Portal Hypertensive Gastropathy (P.H.T.G.):** These are divided according to McCormack et. al.[6] subdivision into:
   - **A-Mild:** The mucosal findings include a fine pink speckling or secarlatina rash, superficial erythema on the surface of rugae, and the most distinctive mosaic or snakeskin mucosal pattern.
   - **B-Severe:** Include discrete cherry red spots and a diffuse hemorrhagic gastritis.

The location of changes of P.H.T.G. is divided into: diffuse, antral, and proximal to the antrum (fundus and body) [7].

The severity of bleeding was assessed according to Gostout CJ[8] to mild, moderate, severe.

Results:

During the three months period of the study, 1406 patient consulted the Center of Gastrointestinal and Liver Diseases, 69/1406 were found to have P.H.T. (4.9%), of these males were 47/69 (68.1%) with an age range of 3-73 years and mean of 50.4 ± 16.3 years, while females were 22/69 (31.8%) with an age range of 15-80 years and mean of 42.8 ± 15.8 years (Table 1).

Table 1 shows that the aetiology in 63/69 (91.3%) patients was due to hepatic causes with viral hepatitis (B and C) in 22/63 (34.9%), undetermined aetiology in 26/63 (41.2%), While 6/69 (8.6%) patients were due to extrahepatic causes, and portal vein thrombosis due

to umbilical sepsis in 2/6 (33.3%).48/69 (69.5%) presented with upper G.I.T. bleeding and 38/69 (55%) with hematemesis and melena, while 7/69 (10.1%) with melena only.

The time of occurrence of the bleeding episode was found more during 0:00-8:00 hour (58.3%) (Table 3).

Table (4) demonstrates hepatic causes of P.H.T.; and their child's score were Child A = 7/63 (11.1%), Child B - 28/63 (44.4%), and Child C = 28/63 (44.4%).By comparing the child's score between the bleeders group and non-bleeders group we find child C patients are more in the bleeders group but not reaching statistical significance (P>0.05).

The correlation between severity of bleeding and child's score demonstrates that severe bleeding was associated with higher score (child C) reaching statistical significance (P< 0.03) as shown in(Ttable 5).

Upper endoscopy demonstrates esophageal varices in 67/69 (97.1%) with 28/69 (40.5%) have red signs, while I.G.V.I. seen in 2/69 (2.8%), O.G.V. 11 in 22/69 (31.8%), and P.H.T.G. in 29/69 (42%), duodenal ulcer seen in 9/69 (13.0%) as it is shown in (Table 6).

Tables (7) show the different variables comparing the bleeder and non-bleeder groups, and demonstrate that the lower platelet count, the advanced esophageal varices grade, and the presence of red signs reached statistical significance.

During hospitalization 5/69 (7.2%) patients died, all were of hepatic aetiology, with child C in 4/5 (80%), the immediate cause of death was persistent bleeding from esophageal varices in two patient in spite of sclerotherapy, while another two died due to encephalopathy precipitated by bleeding, and a fifth patient died due to persistent bleeding from duodenal ulcer (Table 8).

Table 1
The age, sex and aetiology distribution of the study group

Aetiology	N0.	Female	Mean (Range) Years	Male	Mean (Range) Years
Hepatic					
Sinusoidal					
Chronic hepatitis B	**12**	**1**	**55**	**11**	**57.8 (32-73)**
Chronic hepatitis C	**10**	**2**	**29 (15-43)**	**8**	**48 (25-66)**
Alcoholic	**8**	**-**		**8**	**51 (36-65)**
Immune	**2**	**2**	**42.9 (30-55)**	**-**	
Hepatic malignancy	**2**	**-**		**2**	**62.5 (62-63)**
Undetermined	**26**	**12**	**43.5 (22-65)**	**14**	**50 (15-72)**
Postsiusoidal					
Budd-chiari syndrome*	**3**	**3**	**33.3 (19-46)**	**-**	**-**
Pre-hepatic (Portal vein thrombosis)					
Umbilical sepsis	**2**	**-**		**2**	**6 (3-9)**
Pancreatic carcinoma	**1**	**-**		**1**	**65**
Supra renal carcinoma	**1**	**1**	**80**	**-**	**-**
Undetermined	**2**	**1**	**43**	**1**	**42**
Total	**69**	**22**	**42.8 (15-80)**	**47**	**50.4 (3-73)**

Table 2
The presenting symptoms of the study group

Aetiology	N0.	Hematemesis & Melena	Hematemesis	Melena	Ascites	Encephalopathy	Jaundice	Abdominal pain
Hepatic								
Sinusoidal								
Chronic hepatitis B	12	7 (58.3%)	-	1 (8.3%)	2 (16.6%)	-	2 (16.6%)	-
Chronic hepatitis C	10	8 (80%)	-	1 (10%)	-	-	1 (10%)	-
Alcoholic	8	5 (62.5%)	1 (20%)	1 (20%)	-	1	-	-
Immune	2	2 (100%)	-	-	-	-	-	-
Hepatic malignancy	2	2* (100%)	-	-	-	-	-	-
Undetermined	26	12 (46.1%)	1 (3.8%)	4 (15.3%)	6 (23%)	-	3 (11.5%)	-
Postsiusoidal								
Budd-chiari syndrome	3	-	-		3 (100%)	-	-	-
Pre-hepatic (Portal vein thrombosis)								
Umbilical sepsis	2	2 (100%)	-	-	-	-	-	-
Pancreatic carcinoma	1	-	-	-	-	-	-	1 (100%)
Supra renal carcinoma	1	-	-	-	-	-	-	1 (100%)
Undetermined	2	-	1 (50%)	-	-	-	-	1 (50%)
Total	69	38 (55%)	3 (4.3%)	7 (10%)	11 (15.9%)	1 (1.4%)	6 (8.6%)	3 (4.3%)

* In one case, the bleeding was due to duodenal ulcer

Table 3
The time of occurance of bleeding

Time of bleeding occurance	N0.	%
0.00 - 8.00 hour	28	58.3
8.00 - 16.00 hour	18	37.5
16.00 - 24.00 hour	2	4.1
Total	48	100%

Table 4
The characteristics of bleeding and non-bleeding groups of hepatic aetiology of portal hypertensive patient in regard to aetiology and the child's score

Aetiology	N0.	Bleeding group				non- bleeding group			
		N0.	A	B	C	N0.	A	B	C
Hepatic									
Sinusoidal									
Chronic hepatitis B		**8**	**-**	**3**	**5**	**4**	**-**	**1**	**3**
Chronic hepatitis C		**9**	**2**	**5**	**2**	**1**	**-**	**1**	**-**
Alcoholic		**7**	**2**	**1**	**4**	**1**	**-**	**-**	**1**
Immune		**2**	**-**	**-**	**2**	**-**	**-**	**-**	**-**
Hepatic malignancy		**2***	**-**	**2**	**-**	**-**	**-**	**-**	**-**
Undetermined		**17**	**3**	**6**	**8**	**9**	**-**	**7**	**2**
Postsiusoidal									
		-	**-**	**-**	**-**	**3**	**-**	**2**	**1**
		45 (71.4%)	**7 (15.5%)**	**17 (37.7%)**	**21 (46.6%)**	**18 (28.5%)**	**-**	**11 (61.1%)**	**7 (38.8%)**

~~12~~
~~10~~
~~8~~
~~2~~
~~2~~
~~26~~

*** One case of bleeding due to duodentalulcer**
P > 0.05

Table 5
The corrolation of severity of bleeding to the child's score in the group of hepatic causes of portal hypertension

Aetiology	Bleeding group			
	N0.	A	B	C
Mild	9 (20%)	4 (44.4%)	3 (33.3%)	2 (22.2%)
Moderate	26 (57.7%)	3 (11.5%)	11 (42.3%)	12 (46.1%)
Severe	10 (22.2%)*	-	3 (30%)	7 (70%)
Total	45	7 (15.5%)	17 (37.7%)	21 (46.6%)

* One case of bleeding due to duodenal ulcer
P < 0.03 (mild & mod- severe)

Table 6
The details of upper endoscopy findings in the study group

Aetiology	N0.	Esophageal varices			Red signs	Isolated gastric varices I	Esophago- gastric varices II	Portal hypertensive gastropathy			Duodenal ulcer	Duodenitis
		II	III	IV				N0.	Mild	Severe		
Hepatic												
Sinusoidal												
Chronic hepatitis B	12	3	7	2	6	-	4	4	3	1	-	3
Chronic hepatitis C*	10	-	7	3	5	-	3	4	2	2	1	1
Alcoholic	8	2	3	3	4	-	1	3	2	1	3	1
Immune	2	-	2	-	2	-	-	-	-	-	-	-
Hepatic malignancy	2	1	1	-	-	-	-	1	1	-	1**	-
Undetermined	26	7	12	6	8	1	9	14	8	6	4	5
Postsiusoidal												
Budd-chiari syndrome	3	3	-	-	-	-	-	-	-	-	-	-
Pre-hepatic (Portal vein thrombosis)												
Umbilical sepsis	2	-	2	-	2	-	2	-	-	-	-	-
Pancreatic carcinoma	1	-	-	-	-	1	-	-	-	-	-	-
Supra renal carcinoma	1	-	1	-	-	-	1	1	1	-	-	-
Undetermined	2	2	-	-	1	-	2	2	2	-	-	-
Total	69	18 (26%)	35 (50.7%)	14 (20.2%)	28 (40.5%)	2 (2.8%)	22 (31.8%)	29 (42%)	19	10	9 (13%)	10 (14.4%)

*** Gastric polyp (antral); ** In one case, the bleeding was due to duodenal ulcer**

Table 7

The discription of bleeding and non- bleeding groups of patients with portal hypertention

	Bleeding group (n =48)	Non-Bleeding group (n =21)	P value
Age mean □ SD Rang	47.5 □ 3 - 73	49.1 □ 5 - 80	>0.05
sex Male Female	35 13	12 9	>0.05
children score A	10	3	>0.05
B	17	11	
C	21	7	
Serum bilirubin	3.7 □ 5.8	3.9 □ 5.8	>0.05
Serum albumin	3.0 □ 0.5	2.9 □ 0.6	>0.05
Prothrombin time	17.8 □ 3.4	17.8 □ 4.0	>0.05
Platelet count x 10^9	88.1 □ 57.3	142.7 □ 50	<0.001
Esophageal varices II	7	11	<0.001
III	27	8	
IV	13	1	
Isolated gastric varices I	1	1	>0.05
Esophago- gastric varices II	17	5	>0.05
Portal hypertensive gastropathy	19	11	>0.05
Red signs	25	3	<0.01
Ascites Moderate	28	12	>0.05
Severe	4	3	
Encephalopathy I - II	9	2	>0.05
III - IV	5	1	
Hepatic hydrothorax	6	-	-
Spontaneous bacterial peritonitis	4	1	>0.05
Portal vein thrombosis	8	3	>0.05

Table 8

The description of in-hospital mortality group

Aetiology	Age	Sex	N0.	Child's score	Cause of death
Chronic hepatitis B	60	Male	1	C	Persistent bleeding asophageal varices
Alcoholic	52	Male	1	C	Encephalopathy + bleeding esophageal varices
Immune	54	Female	1	C	Encephalopathy + bleeding esophageal varices
Hepatic malignancy	65	Male	1	C	Persistent bleeding asophageal varices
Undetermined	63	Male	1	B	Persistent bleeding duodenal ulcer

Discussion:

During the 3 months period (1st of June to the end of August 2000), 1406 patients were evaluated for different complaints, 69/1406 (4.9%) were found to have esophageal and /or gastric varices, and were considered to have portal hypertension.

A similar study by Fayadh MH, and Al-Karboli TA, during the whole year of 1996 showed that P.H.T. exist in (7%) of patients admitted to the center of G.I.T. and Liver Diseases in Baghdad for different complaints [9].

In comparative analysis of the aetiology of P.H.T. by different studies, it's observed that hepatic causes are the main aetiological factors[1),(2),(4),(9),(10)], this was comparable to our result as seen in table (1). However analyzing the different hepatic causes chronic viral hepatitis (B&C) comprise 34.9% which is near to the result of Kassir Z, and AL-Rawi F, (36.9%) [10]. While undermined aetiology was the commonest group (41.2%), which is very high compared to the European figure of 5-10% which is obviously related to lack of means of proper investigations [11].

Extra hepatic causes "portal vein thrombosis (P.V.T)" was found in (8.6%) and (33.3%) of them were due to umbilical sepsis, while unknown causes comprise 33.3%, these results are comparable to a study by Fayadh MH, and AL-Karboli TA,[9] It's estimated that in about half of patients with P.V.T. the aetiology remains obscure, even after full investigation [12], while Yamada RM et. al., in a study of P.V.T. in children in Brazil, couldn't find the aetiology of P.V.T. in 73.1% [13].

Males are more predominant in our study group which is similarly reported locally in other studies [9,14]. However, it was not possible to find similar results abroad [1,4,12,15].

The mean age of children with P.V.T. in our study group was 6 years, which is similar to studies carried in Italy [16], United States of America[4], and Brazil[13].

Table (2) show that the commonest presentation in our study group was hematemesis and melena (55%), while (10.1%) presented with melena only. Mann et. al., reported that (75.5%) of patients with P.H.T. presented with hematemesis, with (24.5%) presenting with melena only[17], similarly Yamada et. al. reported hematemesis as the main presentation (57.6%) of P.H.T.[13]. This goes in line with the accepted fact that upper G.I.T. bleeding, mainly hematemesis is the commonest presentation of P.H.T [4,12].

It is obvious from our study that patients with hepatic causes of P.H.T. present with bleeding more than extra-hepatic causes (71.4% and 50%) respectively.

Table (3) demonstrate that the early hours of the day (0:00-8:00. hour) were the most common time for bleeding episode in our study group (58.3%), which is similar to other studies by McCormick et. al. [4], and Garcia-Pagan et. al.[18].

Bleeding due to varices is associated with poor liver function (advanced child's score) [4,12,15] Kim et. al. and Mann et. al., concluded similar results in Japan[19] and U.S.A.[17], our study shows that child's C is more in bleeder group (46.6%) than non bleeder group (38.8%) although not reaching statistical significance (P>0.05) as seen in table (4). Another study in Yugoslavia demonstrated that child's C does not correlate with bleeding incidence[20]. Our study demonstrated child's A in 15.5%, child's B in 37.7% and child's C in 46.6% of the bleeder group nearly similar results were demonstrated by the Veterans Affairs Cooperative Variceal Sclerotherapy Group[21], and by Mann et.al. [17].

Red signs were observed more in the bleeder group reaching statistical significance (P<0.05), this was similarly suggested by other studies[4,19,20].

Duodenal ulcer was observed In (14.3%) all of hepatic aetiology similarly other studies In Iraq show duodenal ulcer In (10.3% -18.7%) of patients with chronic liver disease [10,25,26]. A study In U.S.A. showed (13.5%) in of patients undergoing liver transplantation have doudenal ulcer [7], and in Britain (11%) of cirrhotic patients have peptic ulcer[11], while a study in Italy by Giacobbe *et. al.* observed peptic ulcer in (18.2%) [29] in patients with cirrhosis.

Duodenitis was observed in (15.8%), similarly another study in Iraq showed doudenitis in (15%)[30].

Analysis of laboratory results showed that only thrombocytompenia was more in the bleeder group reaching statistical "significance (P< 0.001), this was suggested by other studies[6,31].

Our study demonstrates in-hospital mortality in (10.4%), where (4.1%) were due to persistent variceal bleeding in spite of sclerotheray, and another (4.1%) were due to encephalopathy precipitated by variceal bleeding, while (2%) due to persistent bleeding duodenal ulcer in spite of endoscopic therapy. A review by Dotsenko et. al. of 6 years experience in endoscopic sclerotherapy using absolute alcohol showed (17.5%) in-hospital mortality, (9.45%) were due to persistent bleeding in spite of sclerotherapy, (6.7%) due to encephalopathy precipitated by bleeding in spite of sclerotherapy, and (1.3%) due to esophageal perforation precipitated by sclerotherapy[32]. A large study of 152 patients at King's College Hospital showed (1%) in-hospital mortality after sclerotherapy[33]. While a study from Singapore using ethanolamine showed a high in-hospital mortality (48.5%) [34], others studies from India[35] and U.S.A.

The study showed that patient with child's C class have 75% risk of bleeding, this was similar to the results of Italian study[22].

Comparing the severity of bleeding between mild and moderate-sever bleeding, child's C was more in the latter group reaching statistical significance (P< 0.03) as shown in table (5). This goes in line with other studies [4,12,23].

Analyzing table (6) & (7) ,we conclude that advanced grade of esophageal varices was more in bleeders group reaching statistical significance (P<0.001), this was similarly suggested by other studies[4,20]. Isolated gastric varices I (I.G.V.I) was demonstrated in (4.16%) of patients and in (50%) of them they were the cause of bleeding, other studies show that I.G.V.I. is seen in (8%) of P.H.T. patients[24], while esophago-gastric varices II (O.G.V.II) is seen in (31.8%) of P.H.T. patients. AL-Alusi F, and Fayadh HM, showed O.G.V.II in (11.3%)[25], while in Saudi Arabia it was found in (21.5%) of P.H.T. patients[26]. Gastric varices were found in (2%-70%) of P.H.T. patients in different studies abroad [5,7,24].

Portal hypertensive gastropathy (P.H.T.G.) was observed in (42,0%) with mild form in (65.5%) and severe form in (34.4%), all patients with P.H.T.G. had esophageal varices. A study in Iraq showed P.H.T.G. in (47%) [25], and in Saudi Arabia the incidence was (46%), all of them had esophageal varices [26]. A study in Italy showed P.H.T.G. in (27.3%) with mild form in (77.5%) and (22.4%) severe form[27].

A study in U.S.A. showed P.H.T.G. in (28.3%) with antral distribution in (43.4%), proximal to the antrum in (47.8%), and diffuse in (8.6%), interestingly, 21.7% of these patients had P.H.T.G. without esophageal or gastric varices [7].

Conclusions:

Hepatic aetiology was found in (91.3%) of patients with portal hypertension (P.H.T.), and of these (47.6%) was due to chronic hepatitis B, C and alcoholism which are preventable diseases.

Upper gastrointestinal (G.I.T.) bleeding was the commonest presentation in P.H.T. (69.5%). Most patients presented with advanced liver disease (child's class B&C =88.8%).

Severity of bleeding was significantly more in advanced liver disease (child's class C). Gastric varices and portal hypertensive gastropathy (P.H.T.G.) are not uncommon, and are important cause of bleeding. Upper G.I.T. bleeding was the main cause of in-hospital mortality; mortality was associated with child's class C in 80% of cases. In comparison between bleeder and non bleeder groups thrombocytopenia, higher varices grade, and red signs were significantly correlating factor, while other variables including child's class were of no significance.

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