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Research Article

Predictors of Upper Gastrointestinal Bleeding in Egyptian Patients with Non-Cirrhotic Portal Hypertension

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Abstract

Non-cirrhotic portal hypertension (NCPH) represents infrequent conditions that cause portal hypertension in the absence of cirrhosis. This study evaluated predictors of upper gastrointestinal bleeding in Egyptian patients with NCPH.

One hundred Egyptian patients suffering from NCPH without a previous history of upper GIT bleeding were included and followed up for two years. All underwent laboratory investigations, Colour Doppler ultrasonography, and upper gastrointestinal tract (GIT) endoscopy. They were classified into two groups: Group I, which included 34 patients with bleeding during the follow-up period, and Group II, which included 66 patients without bleeding.

Budd Chiari syndrome was the commonest aetiology (73%), followed by extrahepatic portal vein thrombosis (13%), schistosomiasis (3%), venocclusive disease (1%), arterio-venous fistula (1%), congenital hepatic fibrosis (1%) and idiopathic NCPH (8%).

The mean splenic size (cm), portal vein (mm), and splenic vein (mm) diameters were higher in bleeders than in non-bleeders ($P < 0.01$). The mean portal vein flow velocity (cm/sec) and platelet count/spleen diameter ratio were lower in bleeders than in non-bleeders ($P = 0.003$ and 0.001, respectively). The presence of three or more variceal columns, larger grades of esophageal varices, and risk signs were significantly detected in the bleeders group. Stepwise logistic regression analysis showed that splenic vein diameter (> 10 mm) (OR = 2.64, $P = 0.008$), platelet count/spleen diameter (mm) ratio (< 1000) (OR = 0.999, $P = 0.009$) and number of columns of oesophageal varices (≥ 3) (OR = 27.2, $P = 0.001$) were independent risk factors for upper GIT bleeding.

In conclusion, splenic vein diameter, platelet count/spleen diameter ratio, and the number of columns of oesophageal varices were independent predictors of upper GIT bleeding in NCPH.

Keywords: Budd Chiari Syndrome; Portal Vein Thrombosis; Upper Git Bleeding; Splenic Vein Diameter; Platelet Count/Spleen Diameter Ratio

Abbreviations

NCPH: Non-Cirrhotic Portal Hypertension; IPH: Idiopathic Portal Hypertension; NCPF: Non-Cirrhotic Portal Fibrosis; EHPVT: Extra Hepatic Portal Venous Thrombosis; JAK II: Janus Tyrosine Kinase-2; CD: Cluster of Differentiation; PVV: Portal Vein Flow Velocity; GOV: Gastro-Oesophageal Varices; IGV: Isolated Gastric Varices; PHG: Portal Hypertensive Gastropathy; SD: Standard Deviation; ROC: Receiver Operating Characteristic; AUC: Area Under the Curve; MTHFR: Methyl Tetrahydrofolate Reductase Mutation; FVLM: Factor V Leiden Mutation; 1ry APA: Primary Antiphopholipid Antibody Syndrome; MPD: Myeloproliferative Disorder; 2ry APA: Secondary Antiphospholipid Antibody Syndrome; PGM, Prothrombin Gene Mutation; PNH: Paroxysmal Nocturnal Haemoglobinuria

Introduction

Non-cirrhotic portal hypertension (NCPH) represents a relatively infrequent group of conditions that causes portal hypertension in the absence of cirrhosis. As with cirrhotic portal hypertension, most cases of NCPH are caused by increased portal venous outflow resistance, although, rarely, an increased flow in the portal circulation may be responsible [1,2].

Although the development of varices and subsequent bleeding are the main manifestations of portal hypertension from all causes [3,4], there are unique features related to NCPH. The clinical manifestations of pre-sinusoidal portal hypertension are characteristically devoid of ascites and encephalopathy, whereas ascites is a

cardinal feature of post-sinusoidal obstruction. The prognosis of portal hypertension caused by pre-sinusoidal conditions is relatively better than that of any cause of portal hypertension [5].

The common causes of NCPH include idiopathic portal hypertension (IPH), non-cirrhotic portal fibrosis (NCPF), and extrahepatic portal venous thrombosis (EHPVT). Other causes include schistosomiasis, hepatic venous outflow tract obstruction, and congenital hepatic fibrosis [6,7].

Variceal bleeding in NCPH has lower mortality as compared with cirrhosis because of better liver functions in NCPH [8].

This work aimed to evaluate the predictors of upper GIT bleeding in Egyptian patients with NCPH.

Patients and Methods

This prospective study was conducted on consequently recruited one hundred Egyptian patients with NCPH without previous history of upper GIT bleeding who were admitted to the Tropical Medicine Department at Ain Shams University Hospital, during the period from January 2012 to June 2022.

Patients with concomitant infection with viral hepatitis B or C, cirrhotic portal hypertension, hepatocellular carcinoma, upper GIT bleeding due to ulcers or erosions, those who received non-selective beta-blockers, had any prior interventional radiological procedures [angioplasty ± stenting or trans-jugular intrahepatic portosystemic shunt (TIPS)], or liver transplantation were excluded. Informed written consent was obtained from each patient before inclusion.

The study protocol was approved by the Research Ethical Committee of the Faculty of Medicine, Ain Shams University (Approval Number: 699) according to the ethical guidelines of the 1975 Declaration of Helsinki.

All the included patients had: (a) A complete clinical evaluation; (b) Laboratory investigations: CBC, liver profile, viral hepatitis markers (HBs Ag, HB core Ab, HCV Ab) using the ELISA technique; (c) Thrombophilia workup to clarify the underlying etiology of vascular liver disease. It was done for patients with Budd-Chiari syndrome (BCS) and extrahepatic portal vein thrombosis (EPVT) as follows: anticardiolipin antibodies, lupus anticoagulant, antinuclear antibodies, protein C, S, antithrombin III, factor V Leiden G1691A mutation, prothrombin gene G20210A mutation, methylene tetrahydrofolate reductase C677T mutation by PCR, Janus tyrosine kinase-2 (JAK II) V617F mutation by PCR (to exclude myeloproliferative disorders) and flow cytometry for CD55 and CD59 (to exclude paroxysmal nocturnal hemoglobinuria) [9]; (d) Abdominal ultrasonography: for liver size, echogenicity, spleen size, portal vein diameter and ascites; (e) Color Doppler ultrasonographic study: using a color Doppler unit with a 3.5 MHz convex probe for confirmation of portal vein (PV) patency and diameter, mean PV flow velocity (mean PVV) (cm/sec), PV direction of flow, splenic vein patency and diameter, presence of portosystemic collaterals and patency of hepatic veins; (f) Platelet count/spleen diameter ratio: calculated as: platelet count/ maximum spleen bipolar diameter by ultrasound in mm [10]; (g) Ultrasonography guided liver biopsy: When indicated to confirm the diagnosis of the underlying liver disease; and (h) Upper GIT endoscopy using the Pentax video endoscope EG 3440. Oesophageal varices were classified into small, medium, or large [11]. Red color signs were classified into Cherry Red Spots, Red Wale Markings, and Hematocystic Spots [12]. Gastric varices were classified into either gastro-oesophageal varices (GOV) or isolated gastric varices (IGV) [13]. Portal hypertensive gastropathy (PHG) was classified into either mild or severe [14].

Patients who were diagnosed as BCS or EPVT began anticoagulation therapy involving unfractionated heparin or low-molecular-weight heparin followed by oral anticoagulation therapy with warfarin (initially 3 mg, with increasing dosage until a target INR of 2-3 was obtained). Before starting anticoagulation, band ligation was planned for medium and large-sized oesophageal varices or those with risk signs. Also, cyanoacrylate (Histoacryl®) injection was done for those with signs of risky gastric varices [15].

In the current work, variceal bleeding was diagnosed according to Baveno consensus definitions [14]. All patients were informed about the manifestations of GIT bleeding and instructed to discontinue anticoagulation therapy under such circumstances or if any bleeding was noted. Bleeding was diagnosed by the presence of hematemesis and/or melena or gastric aspirate containing blood. Upper GIT endoscopy was done as early as possible (after resuscitation within 24 hours). Variceal bleeding was confirmed if there was spurting or oozing from the varix, adherent clot, or white nipple on the varix or if there was blood in the stomach with no other potential source of bleeding [14].

The outcome of the study is the occurrence of the first attack of upper GIT bleeding; evidenced by hematemesis and/or melena; during the follow-up period (2 years) and prior to any indicated radiological intervention (angioplasty ± stenting or TIPS).

Recruited patients were classified into two groups: Group I: thirty four patients with upper GIT bleeding; and Group II: sixty-six patients without bleeding.

Statistical analysis

Qualitative data were presented as numbers and percentages while quantitative data were presented as the mean, standard deviations (SD), and ranges. The comparison between the two groups with quantitative data was performed by using an independent t-test. The Chi-square test or Fisher exact test was used for qualitative data. The confidence interval was set to 95% and the accepted margin of error was set to 5%. So, the P-value was considered as

P > 0.05: Nonsignificant, P < 0.05: Significant, and P < 0.01: Highly significant.

Different risk factors for bleeding were analysed using the univariate analysis. Those variables showing significant association with bleeding were introduced in a multivariate binary logistic regression analysis to show the independent risk factors. Enter mode had been selected, and variables showing inconsistency by significance or odds ratio were subsequently excluded (16). The sensitivity, specificity, and positive and negative predictive values were determined for several cutoff values and a receiver operating characteristic (ROC) curve was constructed [17].

Results

Regarding the patients' characteristics and sociodemographic data, the mean age of included patients was 35.6 ± 7.3 years with a male-to-female ratio of 1:1. Forty-two percent were from Cairo, 36% from Delta and 22% from Upper Egypt.

BCS was the commonest etiology of NCPH in the current study (73%), followed by EPVT (13%), schistosomiasis (3%), venocclusive disease (1%), arterio-venous fistula (1%), congenital hepatic fibrosis (1%) and idiopathic NCPH (8%) (Table 1).

Patients were classified into two groups according to the occurrence of upper GIT bleeding in the follow-up period: Group I (bleeders, n = 34) and Group II (non-bleeders, n = 66).

The socio-demographic data of the studied patients and their clinical presentation showed a non-significant impact on the occurrence of upper GIT bleeding (P > 0.05), apart from splenomegaly which was significantly detected in bleeders group *versus* non-bleeders group (P = 0.045) (Tables 2).

Etiology	No.	%
Budd Chiari syndrome	73	73%
MTHFR	15	20.6%
FVLM	12	16.4%
1ry APA	10	13.7%
MPD	9	12.3%
2ry APA	6	8.2%
PGM	4	5.5%
Anti thrombin III deficiency	3	4.1%
PNH	1	1.4%
Protein C deficiency	1	1.4%
Behcet	1	1.4%
Protein S deficiency	1	1.4%
Mixed etiology	5	6.8%
Idiopathic cause	5	6.8%
Extrahepatic Portal vein thrombosis	13	13%
MTHFR	4	30.8%
FVLM	3	23.1%
MPD	2	15.4%
1ry APA	1	7.8%
Mixed etiology	3	23.1%
Schistosomiasis	3	3%
Rare disorders	3	3%
Venocclusive	1	1%
Arterio-venous fistula	1	1%
Congenital hepatic fibrosis	1	1%
Idiopathic NCPH	8	8%

Table 1: Etiology of non-cirrhotic portal hypertension (NCPH) among the studied patients (n = 100).

MTHFR: Methyl Tetrahydrofolate Reductase Mutation; FVLM: Factor V Leiden Mutation; 1ry APA: Primary Antiphopholipid Antibody Syndrome, MPD: Myeloproliferative Disorder; 2ry APA: Secondary Antiphospholipid Antibody Syndrome; PGM: Prothrombin Gene Mutation; PNH: Paroxysmal Nocturnal Haemoglobinuria

Anticoagulation therapy had non-significant impact on the occurrence of upper GIT bleeding among patients with vascular liver diseases (73 Budd-Chiari and 13 extrahepatic portal vein thrombosis) (Table 3,4).

		Bleeders		Non-bleeders		Chi-square test		Sig.
		No.	%	No.	%	X^2	P-value	
Age	Mean ±SD	34.5 ± 10.59		32.6 ± 8.56		0.346	0.649	NS
Gender	Male	18	52.9%	32	48.5%	1.076	0.284	NS
	Female	16	47.1%	34	51.5%			
Residence	Cairo	15	44.1%	27	40.9%	2.733	0.715	NS
	Delta	13	38.2%	23	34.8%			
	Upper Egypt	6	17.6%	16	24.2%			
Alcoholic	No	34	100%	66	100%	NA	NA	
	Yes	0	0%	0	0%			
Smoking	No	23	67.6%	46	69.7%	2.274	0.243	NS
	Yes	11	32.4%	20	30.3%			

Table 2: Comparison between bleeders (n = 34) and non-bleeders (n = 66) regarding socio-demographic data.

NA: Not Available; NS: Nonsignificant

	Bleeders			Non-bleeders		Chi-square test		Sig.
	No.		%	No.	%	X^2	P-value	
Symptoms								
Right upper quadrant pain	No	7	20.6%	8	12.1%	0.645	0.413	NS
	Yes	27	79.4%	58	87.9%			
Ascites	No	8	23.5%	9	13.6%	0.682	0.451	NS
	Yes	26	76.5%	57	86.4%			
Lower limb edema	No	23	67.6%	44	66.7%	0.018	0.898	NS
	Yes	11	32.4%	22	33.3%			
Signs								
Palpable liver	No	10	29.4%	14	21.2%	2.251	0.154	NS
	Yes	24	70.6%	52	78.8%			
Palpable spleen	No	5	14.7%	25	37.9%	3.788	0.045	**S**
	Yes	29	85.3%	41	62.1%			
Abdominal tenderness	No	25	73.5%	48	72.7%	3.483	0.674	NS
	Yes	9	26.5%	18	27.3%			
Prominent veins on abdomen	No	28	82.4%	54	81.8%	1.277	0.293	NS
	Yes	6	17.6%	12	18.2%			

Table 3: Comparison between bleeders (n = 34) and non-bleeders (n = 66) regarding clinical data.

	Before starting Anticoagulation		After starting anticoagulation		McNemmar test		Sig.
	No.	%	No.	%	X^2	P-value	
Non-bleeders	72	83.7%	66	76.7%	1.235	0.395	NS
Bleeders	14	16.3%	20	23.3%			
Total	86	100%	86	100%			

Table 4: Impact of anticoagulation on upper GIT bleeding among patients with vascular liver diseases (Budd-Chiari and extrahepatic portal vein thrombosis).

Regarding laboratory data, thrombocytopenia was highly significantly detected in bleeders *versus* non-bleeders (Mean platelet count was 102.45 ± 99.23 x 10^3/µL *versus* 260.66 ± 178.23 x 10^3/µL, respectively, *P* = 0.005) (Table 5).

Doppler ultrasonographic findings revealed that the mean splenic size (cm), portal vein (mm) and splenic vein (mm) diameters were highly significantly increased in bleeders *versus* non-bleeders (17.54 ± 2.15 *vs.* 12.76 ± 2.32 cm, 14.87 ± 2.64 vs. 11.57 ± 1.34 mm and 11.58 ± 2.45 vs. 8.73 ± 1.87 mm, respectively, P < 0.01). The mean portal vein flow velocity (cm/sec) was significantly decreased in bleeders versus non-bleeders (14.23 ± 3.12 vs. 17.89 ± 4.86 cm/sec, respectively, P = 0.003) (Table 6).

	Bleeders		Non-bleeders		Independent t-test		Sig.
	Mean	SD	Mean	SD	T	p-value	
ALT (7-40 IU/L)	34.53	17.66	44.27	26.88	-0.947	0.359	NS
AST (7-37 IU/L)	39.67	12.35	48.47	33.32	-0.855	0.396	NS
Albumin (3.5-5.3 gm/dL)	3.11	0.50	3.32	0.61	0.761	0.485	NS
Total bilirubin (0.2-1.3 mg/dL)	1.22	0.79	1.13	0.65	-0.580	0.567	NS
Direct bilirubin (0-0.3 mg/dL)	0.22	0.12	0.21	0.14	-0.824	0.445	NS
Prothrombin time	15.92	3.89	15.44	2.34	0.620	0.547	NS
Partial thromboplastin time	35.12	5.41	30.74	5.35	-1.125	0.234	NS
INR	2.26	0.25	1.99	0.19	1.624	0.141	NS
RBCs count × 10^6/µL	4.99	0.79	5.22	0.78	1.218	0.642	NS
Haemoglobin (g/dL)	10.87	2.24	11.44	2.22	1.315	0.423	NS
WBCs count x 10^3/µL	6.99	2.72	8.69	4.15	-1.888	0.165	NS
Platelets x 10^3/µL	102.45	99.23	260.66	178.23	2.934	0.005	HS
Na (135-148 mmol/L)	130.81	4.32	131.32	3.84	-0.486	0.645	NS
K (3.5-5.3 mmol/L)	4.08	0.51	4.21	0.48	-0.998	0.326	NS
Creatinine (0.4-1.4mg/dl)	0.94	0.28	0.87	0.21	0.651	0.534	NS
BUN	10.39	1.63	10.68	1.45	-0.623	0.525	NS

Table 5: Comparison of bleeders (n = 34) and non-bleeders (n = 66) regarding laboratory data.

NS: Non-Significant; HS: Highly Significant

The mean platelet count/spleen diameter ratio was highly significantly decreased in bleeders *versus* non-bleeders (811.76 ± 276.13 *versus* 1879.47 ± 1267.23, respectively, *P* = 0.001) (Table 6).

A comparison between the two groups concerning Upper GIT endoscopic findings is shown in table 7. The presence of three or more variceal columns, larger grades of oesophageal varices, and risk signs were highly significantly detected in the bleeders' group (P < 0.001).

Table 8 and figure 1 show sensitivity, specificity, positive and negative predictive values of the best cutoff values for platelet count (< 122 × 10^3/µL), spleen size (> 17 cm), portal vein diameter (> 12 mm), splenic vein diameter (> 10 mm) and platelet count/spleen diameter ratio (< 1000) as significant risk factors for occurrence of upper GIT bleeding in NCPH.

The significant variables in univariate analysis were included in a stepwise multivariate logistic regression analysis which showed that splenic vein diameter (> 10 mm) (OR = 2.64, P = 0.008), platelet count/spleen diameter (mm) ratio (< 1000) (OR = 0.999, P = 0.019) and number of columns of oesophageal varices (≥ 3) (OR = 27.2, P = 0.001) were independent risk factors for occurrence of variceal bleeding in NCPH. The performance of this prediction model is displayed by the Receiver Operating Characteristic (ROC) curve. The Area under the Curve (AUC) was 0.952 and the 95% confidence interval (95% CI) = 0.901 – 1.002, denoting a very good predictive ability (Table 9 and Figure 2).

		Bleeders		Non-bleeders		Chi-square test		Sig
		No.	%	No.	%	X2	P-value	
Number of columns of oesophageal varices	1	0	0%	20	30.3%	26.247	0.000	HS
	2	5	14.7%	30	45.5%			
	3	22	64.7%	9	13.6%			
	4	7	20.6%	7	10.6%			
Grade of oesophageal varices	Small	0	0%	39	59.1%	43.269	0.000	HS
	Medium	7	20.6%	24	36.4%			
	Large	27	79.4%	3	4.5%			
Risk signs	Negative	5	14.7%	66	100%	48.458	0.000	HS
	Positive	29	85.3%	0	0%			
Gastroesophageal varices	Negative	25	73.5%	60	91%	3.527	0.062	NS
	Positive	9	26.5%	6	9.1%			
Portal hypertensive gastropathy	Severe	13	38.2%	0	0%	16.164	0.000	HS
	Mild	14	41.2%	38	57.6%			
	Negative	7	20.6%	28	42.4%			

Table 7: Upper GIT endoscopic findings among bleeders (n = 34) and non-bleeders (n = 66).

Sig: Significance; NS: Non-Significant; HS: Highly Significant

	Cutoff point	AUC	Sensitivity	Specificity	PPV	NPV
Platelet count (× 10^3/µL)	< 122	0.828	80.8%	76.5%	72.4%	83.9%
Spleen size (cm)	> 17	0.784	56%	94.1%	87.5%	74.4%
Splenic vein diameter (mm)	> 10	0.789	53.8%	97.1%	93.3%	73.7%
Platelet/spleen ratio	< 1000	0.906	92%	73.5%	71.9%	92.6%
Portal vein diameter (mm)	> 12	0.789	84.2%	74.2%	66.7%	88.5%

Table 8: Cutoff values of significant risk factors for variceal bleeding in non-cirrhotic portal hypertension.

AUC: Area Under the Curve, PPV: Positive Predictive Value; NPV: Negative Predictive Value

	B (SE)	P-value	OR (95% CI)
Splenic vein diameter (> 10 mm)	0.970 (0.37)	0.008	2.64 (1.28 – 5.42)
Three or more variceal columns	3.302 (1.01)	0.001	27.2 (3.8 – 195.3)
Platelet count / spleen diameter (mm) ratio (< 1000)	-0.001 (0.000001)	0.019	0.999 (0.998 – 1.00)
Constant	-10.419 (3.815)	0.006	

Table 9: Independent risk factors for variceal bleeding in non-cirrhotic portal hypertension.

B (SE): Regression Coefficient (Standard Errors of B); OR: Odds Ratio; CI: Confidence Interval

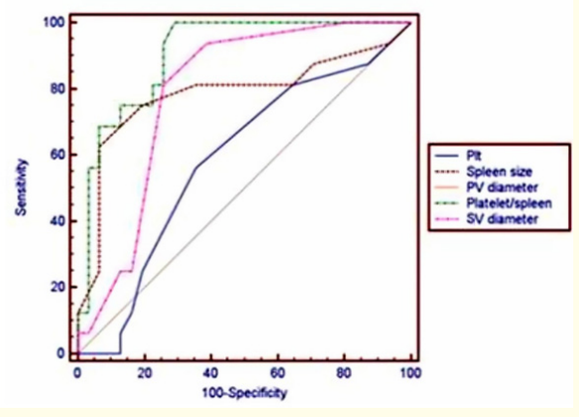

Figure 1: Receiver Operating Characteristic (ROC) curve showing sensitivity and specificity of the best cutoff values for platelet count, spleen size, portal vein diameter, splenic vein diameter, and platelet/spleen ratio as significant risk factors for upper GIT bleeding in the studied patients.

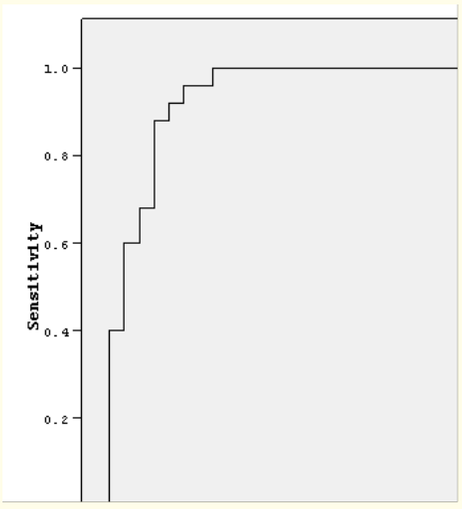

Figure 2: ROC curve displaying the discriminating ability of the proposed model for bleeding prediction (Area under the curve, AUC=0.952 and 95% Confidence interval, CI = 0.901 - 1.002).

Discussion

Non-cirrhotic portal hypertension (NCPH) may present with gastrointestinal hemorrhage in 20-60% of cases [5]. Oesophageal varices are seen in 85-90% of patients and these varices are usually of high grade at the time of diagnosis [18]. while gastric varices are seen in about 25% [19]. Variceal bleeding in NCPH has lower mortality as compared with cirrhosis because of better liver functions [5,6].

The size of oesophageal varices is one of the strongest risk factors for variceal rupture [20,21] thus, all patients must be classified according to their risk status for presence of varices and appropriate prophylactic measures should be taken to prevent hemorrhage [22,23].

The current study aimed to evaluate predictors for upper GIT bleeding in a cohort of 100 Egyptian patients with NCPH. Among these patients, 34 patients (34%) suffered from bleeding during the two-year follow-up period while 66 patients (66%) were non-bleeders.

Among the studied cohort, Budd Chiari syndrome was found to be the commonest etiology of NCPH (73%), followed by extrahepatic portal vein thrombosis (13%), periportal fibrosis due to schistosomiasis (3%), veno-occlusive disease (1%), arterio-venous fistula (1%), congenital hepatic fibrosis (1%) and idiopathic NCPH (8%).

In the present study, portal vein diameter was highly significantly increased in patients with variceal bleeding. This is in agreement with what was reported by *Prihatini., et al.* [24] and *Sarwar., et al.* [25] However, this finding was not observed by *Shabestari., et al.* [26] and *Ismail., et al.* [27] Several factors can affect the results of portal vein diameter measurement such as techniques of examination, status of fasting, experience of the examiner and position of the patient [28].

The splenic size and splenic vein diameter were highly significantly increased in patients with variceal bleeding. This is in agreement with *Tarzamni., et al.* [29] and *Sarangapani., et al.* [30], respectively. *Cunningham., et al.* [31] found that spleen size >17.2 cm had a sensitivity of 78.6% and a specificity of 64.3% for the prediction of high-risk varices when they investigated 44 patients with NCPH.

The mean portal vein flow velocity was significantly lower in patients with variceal bleeding than in those without bleeding. This is supported by *Korner* [32] and *Chiu.,* et al. [33].

In our studied cohort of NCPH, we investigated the platelet count/spleen diameter (mm) ratio as a parameter linking thrombocytopenia to spleen size. We found that this ratio was highly significantly lower in patients with variceal bleeding than in those without bleeding. ROC curve showed that the best cutoff level was <1000 with sensitivity, specificity, negative predictive, and positive predictive values of 92%, 73.5%, 92.6%, and 71.9% respectively. *Baig.,* et al. [34]. reported a cutoff value of 1014, which gave positive and negative predictive values of 95.4% and 95.1% respectively. The platelet count/spleen diameter ratio was deemed to be an appropriate parameter as it normalizes platelet count to splenic sequestration [30].

Endoscopy was proved to be a powerful tool for the determination of bleeding risks in NCPH. The current study has shown that large varices were more likely to bleed than small ones. These results are matching to those of *Limquiaco.,* et al. [28]. and *Benedeto-Stojanov.,* et al. [35].

Other significant endoscopic predictors of bleeding from oesophageal varices noted in the current study were the multiple variceal columns and the presence of risk signs. This is in agreement with *Limquiaco.,* et al. [28]. These signs correspond to the dilated blood-filled channels lying within and beneath the squamous epithelium due to high variceal pressure [28].

The current study demonstrated that platelet count <122.000, platelet count/ spleen diameter ratio cutoff <1000, portal vein diameter >12 mm, splenic vein diameter >10 mm and spleen size >17 cm were significant risk factors of variceal bleeding in NCPH because they represented the median values and offered the best discrimination. This is partially in agreement with *Sarangapani* et al. (30) who found that platelet count/spleen diameter ratio cutoff 909, platelet count <120.000, PV diameter >13 mm and splenic vein diameter >13.8 mm were the significant predictors.

From the multivariate analysis in the current study, it was found that splenic vein diameter (> 10 mm), platelet count/spleen diameter (mm) ratio (< 1000), and number of columns of oesophageal varices (≥ 3) were independent risk factors for the occurrence of variceal bleeding in patients with NCPH. This prediction model showed high performance thus denoting a very good predictive ability.

Conclusion

In conclusion, splenic vein diameter (> 10 mm), platelet count/ spleen diameter ratio (< 1000) and number of columns of esophageal varices (≥ 3) are predictors of variceal bleeding in Egyptian patients with NCPH.

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