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PORTAL HYPERTENSION INDEX AND LIVER VASCULAR INDEX IN PREDICTION OF ESOPHAGOGASTRIC VARICES IN EGYPTIAN BUDD CHIARI SYNDROME PATIENTS

By

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Abstract

BCS is a clinical condition caused by hepatic venous outflow obstruction mainly due to an underlying thrombotic disorder. BCS patients are found to have portal hypertensive gastropathy (PHG) together with esophageal varices (OV) with or without gastric varices. Esophageal varices represented the main source as well as the main independent predictor for bleeding unrelated to invasive therapy for BCS. So, the intensification of prophylaxis for the first or recurrent bleeding might decrease bleeding on anticoagulation therapy.

This study evaluated portal hypertension index and liver vascular index in the prediction of esophagogastric varices in Egyptian patients with Budd Chiari syndrome.

A total of 50 patients with BCS were subjected to upper GI endoscopy for the presence and grading of oesophageal varices and accordingly were divided into GI: variceal group and GII non-variceal group. More subgrouping of the GI was according to the varices size into SGIa (small varices) and SGIb (large varices). Ultrasound with Doppler evaluated the sonographic parameters and indices of portal hypertension.

The results showed that PHTN index was higher in OV patients than in those without (P: <0.001), with a highly significant difference between groups (P=0.000). LVI was lower in OV patients than in those without (P: <0.001), with a highly significant difference between groups (P=0.000).

Keywords: Budd-Chiari syndrome; Portal hypertension Index; Liver vascular Index, Esoph-ago-gastric varices.

Introduction

Budd-Chiari syndrome (BCS) is a clinical condition caused by hepatic venous outflow obstruction located anywhere from the small hepatic veins to the junction of the inferior vena cava and the right atrium, regardless of the obstruction cause (Zahn *et al,* 2010). The BCS patients usually have an underlying thrombotic disorder that can be divided into genetic factors or factor V Leiden, prothrombin gene mutation and acquired disorders or antiphospholipid antibody syndrome (Chait *et al,* 2005).

Rosenberg and Friedman (2004) in Canada divided hepatic venous outflow obstruction into three categories due to the obstruction level: a- Veno-occlusive disease (VOD) at sinusoids and terminal venue's level, b- Budd-Chiari syndrome (BCS) from hepatic veins to inferior vena cava superior end, and c-Venous obstruction at heart level referred to as congestive hepatopathy (CH). Their evolution and severity varied due to cause, and degree of obstruction, with a wide clinical BCS presentation ranged from asymptomatic to fulminant hepatic failure (Menon *et al,* 2004). Dabbous *et al.* (2013) in Egypt added that most of the BCS patients had portal hypertensive gastropathy (PHG) together with oesophageal varices (OV) with or without gastric varices (GV). But, of the most common fatal complications of portal hypertension was GI bleeding due to OVs with significant morbidity and mortality (Jalan and Hayes, 2000). Darwish *et al.* (2009) in Western countries reported that variceal size was one of the critical factors responsible for first hemorrhage anticoagulation & TIPS placement

which must be treated. Esophageal varices were main cause for bleeding (Rautou *et al,* 2011). The anticoagulation therapy was indicated with the large or medium-sized OVs with red signs should undergo band ligation before anticoagulation therapy (Ageno *et al,* 2012).

Screening for portal hypertension in patients with BCS needs a cheap high sensitive, specific, and accepted by patients, but, upper gastrointestinal endoscopy didn't meet all the demands (Piscaglia *et al,* 2001). But, the duplex Doppler sonography decreased in portal flow velocity and an increase in portal vein diameter (Zironi *et al,* 1992). Besides, increased Doppler impedance indices were indicated in portal hypertension (PH) for hepatic and splenic arteries with 97% sensitivity & 93% specificity at cut-off value of 12 cm/s (Iwao *et al,* 1997). Piscaglia *et al.* (2001) reported that Doppler US detected varices in >50% of risky PH patients. Bintintan *et al.* (2015) reported the value of Doppler indexes for detection of esophageal varices in patients with liver cirrhosis which portal hypertension index showed 93.8% sensitivity and 50% specificity to predict large varices in cirrhotic patients at a cut of value > 1.23. Tarzamni *et al.* (2008) suggested the PH index >2.08 and spleen size >15.05 cm, identified patients with a low probability of large OV who didn't need upper gastrointestinal endoscopy, and that diagnosing PH and gastroesophageal varices were true diagnostic value in Budd Chiari syndrome patients.

This study aimed to evaluate the PTHN index and LVI in the prediction of esophagogastric varices in selected Egyptian patients with Budd Chiari syndrome.

Materials and Methods

This study was a cross-sectional study. A total of 50 patients (ages from16 to 56 years and 36 were females) with Budd Chiari syndrome were selected from Tropical Medicine Department, Ain Shams University Hospitals, or attended the BCS outpatient clinic from 2020 to 2022.

Eligibility criteria: Patients were considered eligible if they fulfilled the following criteria: adults aged between 18-60 years, Egyptian nationality, patients diagnosis as primary BCS (after Budd-Chiari protocol study), and patients accepted participation, patients without any co-morbid who neither underwent sclerotherapy or band ligation of esophageal varices nor receive any vasoactive treatment as primary or secondary prophylaxis against esophageal varices, or underwent any interventional modality for BCS.

Patients were subjected to: complete history taking and clinical Examination. Laboratory investigations for CBC, liver profile (AST, ALT, albumin, total and direct bilirubin, PT, PTT, INR), renal function tests (BUN, create, Na, K), hepatitis markers: HB surface antigen (HBs Ag) and HC virus antibody (HCV/Ab) by 3rd generation ELISA, ascitic fluid analysis for ascetic patients (total proteins, ascitic fluid albumin and SAAG by estimation of serum albumin & ascitic fluid albumin), Thrombophilia workup to clarify the BCS etiology.

Patients were evaluated by upper GI endoscopy for oesophageal varices grades and were divided into two groups, GI variceal and GII non-variceal, the GI according to varices size were subdivided into SGIa (with small varices) and SGIb (with large varices).

Abdominal Ultrasonography with Color Doppler: After an overnight fasting, Liver size was measured as the span of the right lobe in mid-clavicular line on oblique view and classified as shrunken (< 11cm), average (11-15cm), or enlarged (> 15cm) after Kuntz and Dieter (2006), liver echogenicity, hepatic veins status, IVC and portal vein (diameter, patency, flow direction & flow velocity). PV is normally up to 13mm in diameter measured from the inner to outer wall during suspended respiration, portal vein flow velocity (cm/s) and portal vein diameter, Hepatic artery resistance index (RI), measured in the intrahepatic main branches (Piscaglia *et al,* 2001). RI was calculated over a cardiac cycle formula: RI = (Peak systolic velocity- end diastolic velocity)/systolic velocity**,** splenic ar-

tery resistance index (RI), was measured intraparenchymally, near to hilum**,** portal hypertension index = (hepatic artery RI×0.69× splenic artery RI×0.87)/portal vein mean velocity (Piscaglia *et al,* 2001), liver vascular index was calculated as the ratio of portal venous velocity to hepatic arterial pulsatility index (Iwao *et al,* 1997). Hepatic arterial Pulsatility index = Peak systolic velocity-end diastolic velocity/mean velocity. Splenic size was measured in a coronal plane, and was classified according to its longest axis into normal up to 12-13cm**,** splenic vein diameter & patency normal splenic vein diameter less than 10 mm. Ascites status was reported as either mild, moderate or marked ascites. Presence or absence of portosystemic collaterals e.g. left gastric vein, paraumbilical vein, porta-hepatic collaterals, lienorenal collaterals or splenic hilar collaterals by Doppler examination.

Upper Gastrointestinal Endoscopy: All were performed blindly at Ain Shams Endoscopy Unit to detect the presence or absence of OV. Endoscopic OV was classified into small or large varices (small varices ≤ to 5 mm, large > 5mm). Gastric varices and portal hypertensive gastropathy were recorded. Ability of Doppler indices (liver vascular index and portal hypertension index) and esophageal varices grades were assessed.

Ethical consideration: The study was done according to the ethical guidelines of 1975 Declaration of Helsinki (6^{th} Revision, 2008), with ethical approval number: FMASU 56/ 2020 (4/2/2020). Written informed consent from the participated patients was obtained after explaining the aim of the study.

Statistical analysis: Data was tabulated and analyzed by using the SPSS statistical package version 16. The patients' demographics and clinical characteristics were compared by Student t, $\chi 2$, or Fisher exact tests according to their variable type. Qualitative data was presented as frequency and percentages. Quantitative variables were presented as mean± standard deviation (SD), median and range. P value less than 05 was considered significant.

Results

CBC, liver functions, liver enzymes, kidney functions, and serum electrolytes, showed a highly significant difference between the OV subgroups (small OV, large OV) compared to non OV ones as to albumin level (P<0.001) & total bilirubin level (P<0.001), and a highly significant difference between small and large O.V subgroups as to Albumin level, total, direct bilirubin, platelets, and WBCs count.

Sonographic data showed a progression from non OV via small OV & large OV associated with a highly significant differences, with more liver coarseness and cirrhotic configuration, higher PVD (P=0.00), lower PVV(P=00), higher occurrence of Porto-systemic collaterals, and ascites. Splenic size, in post hoc analysis, showed highly significant difference between small &large OV subgroups without significant difference between non OV and small OV subgroup.

Doppler indices*:* PHNT index was highly significantly in large OV subgroup than in small OV subgroup (P =0.00) than in non OV group (P =0.00). But, LVI was highly significantly lower in large OV subgroup than in small OV one (P =0.00) than in non OV group (P =0.00). HAPI showed highly significant difference between the non OV group (lower values) as compared to small (P =0.003) & large (P= 0.00) OV subgroups, without significant differences (P= 0.131) when comparing the small and large (P= 0.00) OV subgroups.

HARI showed highly significant difference between non OV group (lower values) and both small (P=.004) & large (P=.005) OV subgroups, without significant difference between small and large subgroups (P= 0.980). SARI was highly significantly in small OV subgroup than in non OV group (P=0.017) and in large OV subgroup than in small one (P=0.00).

PHTN index at a cut-off point of > 1.13 showed 100% sensitivity and 88.89 % specificity to predict presence of esophageal varices with 94.1% positive predictive value and

100% negative predictive value. LVI showed 96.87% sensitivity & 100% specificity to predict the presence of esophageal varices with 100% positive predictive value, and 94.7% negative predictive value at a cut-off point of ≤13.39.

PHTN index at a cut-off point of > 1.84 showed 100% sensitivity & 87.5% specificity in differentiation between small OV and large OV with 88.9% positive predictive value & 100% negative predictive value. LVI showed 87.5% sensitivity and 100% specificity in differentiation between small OV and large OV with 100% positive predictive value & 88.9% negative predictive value at a cut-off point of ≤10.17.

Details were given in tables (1, 2, 3 & 4) and figures (1 & 2).

Table 1: Comparison between GIa (Small OV), GIb (Large OV) and GII (Non OV) regarding laboratory data

Variants		GIa (Small OV) N= 16	GIb (Large OV) N= 16	GII (Non OV) N= 18	Test value	P- value	Sig.
HB	M±SD	11.98±2.14	11.41±1.57	12.13±2.80	0.470•	0.628	NS
	Range	7- 15	9.8- 16	7- 17.5			
WBC	M±SD	8.13±3.62	5.90±2.67	10.32±4.64	5.817•	0.006	HS
	Range	2.3- 13.6	3- 11.1	4.1- 22			
PLT	Median (IQR)	211.5 (132.5- 299.5)	105.5 (70- 212.5)	216.5 (150-320)	11.863≠	0.003	HS
	Range	112- 520	42-373	126- 790			
AST	Median (IQR)	37.5 (29 – 81)	38 (31-51)	63.5 (38- 91)	2.721≠	0.256	NS
	Range	25 – 223	22- 498	14- 454			
ALT	Median (IQR)	33 (21.5- 58)	23 (13.5- 56.5)	40.5 (20- 88)	1.403≠	0.496	NS
	Range	6 – 195	3.1- 553	5- 425			
Albumin	M±SD	2.91±0.27	2.10±0.19	3.76±0.21	226.551•	< 0.001	HS
	Range	2.5-3.3	1.8- 2.4	3.4- 4.1			
Bilirubin (total)	Median (IQR)	2.35 (1.95- 2.7)	6.05 (3.8-10.9)	1.4 (1.2- 1.6)	43.514≠	< 0.001	HS
	Range	1.7- 2.9	2.9- 20	0.4- 1.6			
Bilirubin (direct)	Median (IQR)	0.9 (0.6- 1.45)	4 (2.15- 5.55)	0.55 (0.4- 0.8)	32.616≠	< 0.001	HS
	Range	0.09- 1.8	1.2- 13	0.1- 1			
PT	M±SD	15.31±3.61	16.11±3.70	14.56±2.94	0.882•	0.421	NS
	Range	11-25	12- 27	11- 23			
PTT	M±SD	39.29±14.55	43.75±12.88	37.17±13.67	1.003•	0.374	NS
	Range	20- 67	21- 76	20- 65			
INR	M±SD	1.72±0.79	1.82±0.65	1.50±0.21	1.261•	0.293	NS
	Range	1.1- 4	1.2 – 3.75	1.1- 1.85			
NA	M±SD	131.00±5.62	131.06±7.41	130.61±4.46	0.030•	0.971	NS
	Range	122- 142	120-142	123-140			
K	M±SD	3.98±0.69	4.10±0.57	4.02±0.68	0.139•	0.87	NS
	Range	2.5- 5.5	3 – 5.2	3.3- 6.3			
Creatinine	M±SD	1.03±0.82	0.98±0.41	0.83±0.19	0.670•	0.517	NS
	Range	0.6- 4	0.4- 2	0.5- 1.2			
BUN	Median (IQR)	13.5 (8- 22.5)	14.5 (12- 27)	14 (10-20)	1.625≠	0.444	NS
	Range	6-70	7- 39	7- 25			
HBsAg	No	16 (100.0%)	16 (100.0%)	18 (100.0%)	2.168	0.338	NS
	Yes	0 (0.0%)	0 (0.0%)	0 (0.0%)			
HCV Ab	No	16 (100.0%)	16 (100.0%)	18 (100.0%)	1.172	0.557	NS
	Yes	0 (0.0%)	0 (0.0%)	0 (0.0%)			
Ascitic total proteins	M±SD	2.78±1.18	3.18±0.94	3.35±1.09	1.070•	0.352	NS
	Range	1- 4.9	1.4- 5.1	1.8- 5			
SAAG	M±SD	1.43±0.50	1.44±0.83	1.19±0.41	0.628•	0.539	NS
	Range	0.6 – 2.5	0.4 – 3.2	0.5 – 1.7			

*P > 0.05: Non significant; P < 0.05: Significant; P < 0.01: Highly significant

Table 2: Post hoc analysis between groups regarding Doppler indices

Variants	GII vs. GIa	GII vs. GIb	GIa vs. GIb
Portal HTN index (HA R.I X 0.69) X (SA R.I X 0.87)	0.000	0.000	0.000
LVI	0.000	0.000	0.000
HA P.I	0.003	0.000	0.131
HA R.I	0.004	0.005	0.980
SA R.I	0.017	0.000	0.000

P > 0.05: Non significant; P< 0.05: Significant; P < 0.01: Highly significant, *: Chi-square test; •: One Way ANOVA test

Table 3: Comparison between GIa (Small OV) and GIb (Large OV) and GII (Non OV) regarding sonographic parameters

Grade of O.V		GIa (Small OV)	GIb (Large OV)	GII (Non OV)	Test value	P- value	Sig.
Liver echogeniciaty	Homogenous	0 0.0%)	0 (0.0%)	5 (27.8%)	25.952*	< 0.001	HS
	Coarse	10 (62.5%)	4 (25.0%)	13 (72.2%)			
	Cirrhotic	6 (37.5%)	12 (75.0%)	0 (0.0%)			
Liver size (cm)	M±SD	18.73±1.24	18.19±3.00	18.79±1.49	0.429•	0.654	NS
	Range	16- 21	11- 22	16- 21			
HVO	Patent	0 (0.0%)	0 (0.0%	0 (0.0%)	–	–	–
	Occluded	16 (100.0%)	16 (100.0%)	18 (100.0%)			
Number of HVO	1	0 (0.0%)	1 (6.2%)	0 (0.0%)	3.180*	0.528	NS
	2	2 (12.5%)	4 (25.0%)	4 (22.2%)			
	3	14 (87.5%)	11 (68.8%)	14 (77.8%)			
Rt. H. V	No	2 (12.5%)	3 (18.8%)	0 (0.0%)	3.472*	0.176	NS
	Yes	14 (87.5%)	13 (81.2%)	18 (100.0%)			
Middle H.V	No	0 (0.0%)	1 (6.2%)	2 (11.1%)	1.857*	0.395	NS
	Yes	16 (100.0%)	15 (93.8%)	16 (88.9%)			
Lt. H.V	No	0 (0.0%)	2 (12.5%)	2 (11.1%)	2.068*	0.356	NS
	Yes	16 (100.0%)	14 (87.5%)	16 (88.9%)			
IVC Patency	Patent	10 (62.5%)	8 (50.0%)	10 (55.6%)	0.510*	0.775	NS
	Occluded	6 (37.5%)	8 (50.0%)	8 (44.4%)			
PV patency	Patent	16 (100.0%)	15 (93.8%)	17 (94.4%)	0.991	0.609	NS
	Occluded	0 (0.0%)	1 (6.2%)	1 (5.6%)			
PV flow direction	Petal (towards liver)	16 (100.0%)	13 (81.2%)	18 (100.0%)	6.782*	0.034	S
	Fugal (away from liver)	0 (0.0%)	3 (18.8%)	0 (0.0%)			
PVD (mm)	M±SD	12.63±0.55	14.09±0.59	10.62 ± 0.77	123.381•	0	HS
	Range	11.8- 13.3	13.4- 15.3	9 -11.7			
PVV (cm/sec)	M±SD	15.44±1.15	10.75±1.34	18.56±1.08	182.513•	0	HS
	Range	13-17	9- 12.5	17- 21			
Porto systemic collat-erals	Absent	6 (37.5%)	3 (18.8%)	14 (77.8%)	12.566*	0.002	HS
	Present	10 (62.5%)	13 (81.2%)	4 (22.2%)			
Splenic size	M±SD	13.68±1.42	16.45±3.77	13.79±1.60	6.562	0.003	HS
	Range	11.7- 15.7	11- 24	12- 17.5			
SVD	M±SD	8.64±1.29	10.11 ± 2.88	8.49±1.64	3.175	0.051	NS
	Range	7- 11	6- 16	7- 12			
Splenic vein patency	Patent	16 (100.0%)	16 (100.0%)	18 (100.0%)	–	–	–
	Occluded	0 (0.0%)	0 (0.0%)	0 (0.0%)			

*P > 0.05: Non significant; P < 0.05: Significant; P < 0.01: Highly significant

Table 4: Comparison between GIa (Small OV) and GIb (Large OV) and GII (Non OV) regarding studied Doppler indices

		GIa (Small OV)	GIb (Large OV)	GII (Non OV)	Test value•	P- value	Sig.
PHTN (HA R.IX0.69) X (SA R.IX0.87)	M±SD	1.64±0.21	2.76±0.61	1.13±0.17	80.06	0	HS
	Range	1.39- 2.12	1.86- 3.85	0.91-1.56			
LVI	M±SD	12.32±1.10	8.43±1.40	15.52±0.91	163.246	0	HS
	Range	10.24- 14.41	6.57- 10.68	13.57-16.81			
HA P.I	M±SD	1.26±0.05	1.29±0.08	1.20±0.03	11.517	0	HS
	Range	1.18- 1.36	1.17- 1.38	1.16- 1.29			
HA R.I	M±SD	0.69±0.05	0.69±0.08	0.62±0.08	6.077•	0.004	HS
	Range	0.6- 0.78	0.5- 0.81	0.48- 0.78			
SA R.I	M±SD	0.61±0.05	0.70±0.03	0.57±0.06	35.276•	0	HS
	Range	0.53- 0.69	0.62- 0.75	0.5- 0.7			

*P > 0.05: Non significant; P < 0.05: Significant; P < 0.01: Highly significant

Discussion

Budd Chiari Syndrome (BCS) is associated with a risky complications and death due to portal hypertension and liver failure (Valla, 2009). Its management was achieved via anticoagulation therapy along with control of prothrombotic condition and improved hepatic outflow obstruction (Slakey *et al,* 2001). Oesophageal varices and portal hypertensive gastropathy with or without gastric varices were found in most BCS patients due to resultant portal hypertension of greatest concern in BCS patients due to the risk of bleeding with high mortality (Nafeh *et al,* 2001) with more substantial morbidity and mortality than other gastrointestinal bleeding causes (Gameel *et al,* 2004).

In the present study, 32/50 BCS variceal patients had a mean age of (28.22±8.27) compared to 18 without variceal ones with a mean

ages (25.17±7.70). Female patients were 22 (68.8%) in variceal (OV), and 14 (77.8%) in non variceal (non OV), but without significant difference. This agreed with Elkenawy *et al.* (2020) in Egypt didn't find significant difference between variceal and non-variceal cirrhotic patients as regards age, & sexes

In the present study, the WBC (7.01±3.33) was significantly lower in OV group than in non OV ones (10.32±4.64). This agreed with Gue *et al.* (2004) in Singapore who found a significant inverse correlation between low WBC & esophageal varices grade 2 or more. Qamar and Grace (2009) attributed leucopenia in portal hypertension to splenic sequestration. Besides, The OV group had significantly lower PLT count than the non OV group that agreed with Arulprakash *et al.* (2010) in India and Mahmoud *et al.* (2014) in Egypt they reported a decrease in platelet count in patients with varices as compared to those without varices, and thrombocytopenia was much higher in patients with OVs than those without. Platelet count depended on many factors not only the portal hypertension (Thabut *et al,* 2003). Suk (2012) found that low platelet predicted oesophageal varices' size, and thrombocytopenia included productive, consumptive, or distributional was due to the spleen destruction.

In the present study, albumin was significantly lower in the OV group than in non-OV ones, but total bilirubin and direct bilirubin were higher in the OV group (P=0.00). Barrera *et al.* (2009) found higher total bilirubin in oesophageal varices patients. This agreed with Muhammad *et al.* (2012), they found that serum albumin of 2.8g/dl or less gave very high sensitivity and specificity in the OV prediction. Berzigotti *et al.* (2012) reported that esophageal varices patients had significantly higher bilirubin, and lower albumin. Elkenawy *et al.* (2020) found that serum albumin, and serum bilirubin were significantly different between variceal and non-variceal patients (P = 0.000).

In the present study, liver echogenicity degrees differed in a highly significant fashion, with the OV group having coarser and cirrhotic configurations (P=0.00). This agreed with Ma *et al.* (2020) who reported that liver rough surface was independent predictors of OV. Also, portal vein diameter showed high significance in variceal group than in non-variceal one. But, Shastri *et al.* (2014) reported that portal vein >13mm had 84% sensitivity to diagnose the oesophageal varices.

Achim *et al.* (2016) showed that PVD was significantly higher in cirrhotic patients as compared to controls, but the portal vein diameter didn't correlate with the esophageal varices size. Salman *et al.* (2020) reported that PVD had the highest diagnostic value to detect oesophageal varices in post-HCV cirrhoic patients at cut-off values of ≥12.5mm (99% sensitivity & 94% specificity). Portal vein diameter ≥13.74, ≥14.35, & ≥ 14.65mm gave a good diagnostic oesophageal varices value of grades 2, 3, & 4. But, Wicaksono *et al.* (2022) showed that in post HBV & HCV, the PVD alone didn't predict the OV degree. Zhou *et al.* (2015) in China reported that the patterns of portosystemic collaterals and the LPV & SV diameters were associated with cirrhosis Child-Pugh classifications.

In the present study, portal vein velocity (PVV) was lower in variceal patients than in non-variceal ones with a highly significant difference between the non-OV group & small OV and large OV subgroups (P= 0.000). Mahmoud *et al.* (2014) and Heikal (2020) reported that median values of PVV in varic eal patients were significantly low than in non-variceal ones. Besides, Elkenawy *et al.* (2020) reported that PVV decreased significantly in grades 2 & 3 OV without significant between them compared with Grade 1 OV (P= 0.004 & 0.000, respectively). However, Abdallah *et al.* (2021) found a significant difference in PVV between large & small OV patients**.** Others didn't find optimal OV prediction PVV (Schepis *et al,* 2001; Rezayat *et al,* 2014; Chakrabarti *et al,* 2016). This controversy may be due to the false-positive velocities secondary to most cirrhotic patients have porto-systemic shunts arising from por-

tal hypertension, which varied in complexity (Baik, 2010), or Doppler angle closer to 90° degree with respect to the flow direction (Park *et al,* 2012).

In this study, porto-systemic collaterals showed a highly significant difference between the variceal and non-variceal ones (p=0.001). Mahmoud *et al.* (2014) reported that optimum diagnostic cut-off value of splenic diameter to predict OVs was > 14.03cm with 90.16% sensitivity & 60% specificity. Salahshour *et al.* (2020) found that prediction of OV and variceal haemorrhage achieved with high specificity and accuracy depended upon porto-systemic collaterals. Also, the present splenic size, didn't show significant difference between variceal & non-variceal ones (P=0.115) or between non-variceal and small-variceal ones (P=0.894), but with a highly significant difference between non variceal group and large OV ones or between small &large OV subgroups (P=.003). Salman *et al.* (2020) found that splenic size was a significant discriminator for oesophageal varices in patients with post-HCV cirrhotic at ≥13.5cm cut-off value. Madhotra *et al.* (2002); Mahran *et al.* (2006); Chang *et al.* (2007) and Berzigotti *et al.* (2012) found that splenic size was an independent predictor of oesophageal varices.

The present study showed that portal hypertension index was high in patients with OV than in those without (P <0.001), with high significant difference between non OV, small OV & large OV groups (P=0.000). PHTN index at a cut-off point of > 1.13 had 100% sensitivity & 88.89% specificity in predicting oesophageal varices with 94.1% positive predictive value & 100% negative predictive value. Also, PHTN index at a cut-off point of > 1.84 had 100% sensitivity & 87.5% specificity to differentiate between small & large OV with 88.9% positive predictive value & 100% negative predictive value**.** This agreed with Tarzamni *et al.* (2008) reported that PHTN index was significantly higher in cirrhotic patients with OV irres-pect of size. They suggested endoscopic evaluation for O.V in patients with compensated cirrhosis with portal hypertensive index > 2.08 and spleen size > 15.05 cm. Mahmoud *et al.* (2014) found that the PHTN index at optimum diagnostic cut-off value of > 2 predicted OVs with 36.1% sensitivity & 100% specificity.

The present study showed that liver vascular index was lower in patients with OV than in those without (P <0.001), with high significant difference between the non OV; small OV and large OV ones (P=0.000). The RO-curve showed that LVI had 96.87% sensitivity & 100% specificity to predict oesophageal varices with 100% positive predictive value & 94.7% negative predictive value at a cut-off point of ≤13.39. The LVI had 87.5% sensitivity & 100% specificity to differentiate between small OV and large OV with 100% positive predictive value and 88.9% negative predictive value at a cut-off point of ≤10.17. Besides, Tarzamni *et al.* (2008) found that LVI (P < 0.0005) was significantly lower in patients with OV irrespective of size and in patients with large varices (P< 0.0005). Mahmoud *et al.* (2014) reported significant lower values in LVI to detect patients with varices than in those without varices. But, Hekmatnia *et al.* (2011) reported that OV grade was not significant with LVI (P>0.05).

In the present study, hepatic artery pulsatility index (HA P.I) was higher in patient with varices than those without (P <0.001), with high significant difference between non OV ones (low) compared to both small (P = 0.003) and large (P= 0.00) OV, without significant difference (P=0.131). Berzigotti *et al.* (2012) showed that HAPI had a high value in patients with OV compared with those without OV, but without significance differences. Masoud *et al.* (2018) reported that the HA P.I significantly increased in esophageal varices patients Abdallah *et al.* (2021) found significant difference in HA P.I between small OV & large OV patients being lower in the former than in the latter (P= 0.022). Others didn't find that HA P.I predicted esopha-

geal varices (Taourel *et al,* 2008; Mahmoud *et al,* 2014; Chakrabarti *et al,* 2016).

In the present study, hepatic artery resistive index (HA R.I) was higher in the variceal group (0.69±0.07 vs. 0.62±0.08). In post hoc analysis, HA R.I showed highly significant difference between non OV group (lower values) and both small (P=.004) and large (P=.005) OV subgroups, but without significant difference. This agreed with Masoud *et al.* (2018), found that hepatic artery resistance index (0.76±0.12 vs. 0.65±0.04) was highly significantly elevated in varices patients compared to those without the OVs.

Salman *et al.* (2020) reported that post-HCV cirrhotic patients with esophageal varices had higher HA RI than non-variceal ones. But, Taourel *et al.* (2008) and Chakrabarti *et al.* (2016) found that HARI was not helpful in predicting esophageal varices.

In the present study, the splenic artery resistive index (SA R.I) was higher in patients with OV (0.65±0.06 vs. 0.57±0.06). In the post hoc analysis, SA R.I increased with advancement of OV to significantly higher in the small OV subgroup than in non-OV ones (P=0.017) and in large OV subgroup than in small one (P=0.00). This agreed with Tarzamni *et al.* (2008) who found that SA R.I was significantly higher in cirrhotic patients with OV irrespective of size. Besides, Abdallah *et al.* (2021) found significant difference in SA R.I between large & small OV patients was lower in large OV than in small OV ones. But, Berzigotti *et al.* (2012) Mahmoud *et al.* (2014) and Chakrabarti *et al.* (2016) didn't find significant difference in SA R.I between the variceal and non-variceal groups.

Conclusion

The portal haemodynamic parameters proved effective predictors of OVs in cirrhotic individuals and could be used as non-invasive imaging to reduce upper GI endoscopy. So, we can reduce exposure to frequent invasive procedures.

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Explanation of figures

Fig. 1: ROC curve for validity of portal hypertension index and liver vascular index to differentiate between GI (OV) & GII (non O.V)

Fig. 2: ROC curve for validity of portal hypertension index and liver vascular index to differentiate between GIa (small) & GIb (Large)