Accuracy of left gastric vein hemodynamic changes as screening test for gastroesophageal varices in cirrhotic liver patient using color Doppler ultrasound.

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Abstract: Background: Portal hypertension is one of complication of cirrhosis which results in the development of spontaneous porto-systemic collaterals at a number of anatomic sites as a response to increased pressure. **Objective:** To test the accuracy of left gastric vein (LGV) color Doppler homodynamic changes as a screening tool for the presence and severity of gastroesophageal varices (GEV) in cirrhotic patients. **Patients and methods:** One hundred consecutive cirrhotic patients were included in this study. All patients underwent endoscopy before ultrasonic examination. The method of left gastric vein identification unified for all patients. Measurements of diameter, flow direction and flow velocity in the left gastric vein (LGV) as well as the presence of paraesophageal varices were done in all patients using ultrasonography Doppler study. **Results:** According to presence of oesophageal varices (OV), 53 patients had OV and 47 patients had no OV. According to presence of gastric fundal varices (FV), only 9 patients had varices. Moreover, only 3 patients had gastric forniceal varices. The Mean diameter of LGV was 6±1.5 mm with mean flow velocity 15.7 ± 6.7 cm/s. **Conclusion:** The results suggest that portal hemodynamics changes in cirrhotic patients are characterized by passive congestion and increased blood flow. However, these 2 features had different preponderances in different parts of the portal venous system. Flow velocity, direction and diameter of the left gastric vein done by ultrasonic Doppler study may be play a role in evaluation of portal hypertension and relation with the development and size of varices.

[Abdelgawad Saied, Mohamed Elfiomy, Ashraf Elbahrawy, Atif Abouelfotoh, Ahmed Elwasseif, Nabil Rafaat, Assem El-Sherif. Mokhtar Ragab, *Ehab A. Helal,* Sad Rezk Abdul Wahed. **Accuracy of left gastric vein hemodynamic changes as screening test for gastroesophageal varices in cirrhotic liver patient using color Doppler ultrasound.** *J Am Sci* 2014;10(4):75-85]. (ISSN: 1545-1003). http://www.jofamericanscience.org. 10

Key Words: Left gastric vein; Hemodynamics; Gastroesophageal varices; Liver cirrhosis; Color Doppler ultrasound.

1.Introduction:

Hepatic cirrhosis is defined anatomically as a diffuse process with fibrosis and nodule formation. Whatever the causes are, the end result is the same (cirrhosis) (1). Portal hypertension is one of complication of cirrhosis which results in the development of spontaneous porto-systemic collaterals at a number of anatomic sites as a response to increased pressure. The most clinically significant of these are the gastroesophageal varices because of their propensity to rupture and cause life-threatening hemorrhage (2). Incidence of first variceal hemorrhage ranges from 20 to 40% within 2 years. Recurrent bleeding occurs in 30% to 40% of patients within the next 2 to 3 days and in up to 60 % within 1 week (3). The left gastric vein and to a lesser extent the short gastric veins are the major communications between the portal vein and gastroesophageal varices in patients with portal hypertension (4).

Current guidelines recommend screening all cirrhotic patients by endoscopy, to identify patients at risk of bleeding who should undergo prophylactic treatment. However, since the prevalence of varices in cirrhotic patients is variable, universal screening would imply a large number of unnecessary endoscopies and a heavy burden for endoscopy units. In addition, compliance to screening programs may be hampered by the perceived unpleasantness of endoscopy (5).

Predicting the presence of esophageal varices by non-invasive means might increase compliance and would permit to restrict the performance of endoscopy to those patients with a high probability of having varices. Over the years, several studies have addressed this issue by assessing the potential of biochemical, clinical and ultrasound parameters, transient elastography and CT scanning.

The ultrasonographic examination is a simple, inexpensive, accurate, and noninvasive technique. It has been widely used to investigate the relationship between OV and hemodynamics associated with portal hypertension and liver cirrhosis (6).

In cirrhotic patients, because of portal outflow obstruction (i.e., elevated intrahepatic portal vascular

resistance), increased blood flow in the splenic vein cannot enter the liver via the PV, and a considerable percentage of splenic vein flow is forced to bypass the liver. One of the most important shunting routes is the LGV, which may normally arise from the PV and splenic vein. When increased flow in the splenic vein is prominent, the diversion of a large quantity of portal flow via the LGV would result in more severe esophageal varices and might trigger the occurrence of esophageal varices bleeding[7].

In this study, we investigated the accuracy of left gastric vein (LGV) color Doppler homodynamic changes as a screening tool for the presence and severity of gastroesophageal varices (GEV) in cirrhotic patients.

2. Patients and methods:

One hundred consecutive cirrhotic patients (as proven by clinical and laboratory data, ultrasonography findings, histopathological assessment of liver tissue or APRI score) were included in this study. The study was conducted at El-Hussein hospital, Al-Azhar university. All patients without past history of upper GIT bleeding, endoscopic or surgical intervention for management of portal hypertension, history of vasoactive drugs (beta blockers, nitrates, somatostatin or vasopressin), during the period of 6 months before inclusion, as well as patients with thrombosis of portal or hepatic venous systems were excluded.

Patients were subjected to full clinical assessment with special reference to history of chronic liver disease, past history of schistosomiasis and anti-schistosomal therapy, attacks of hematemsis and/or melena, hepatic encephalopathy, and associated comorbidity. In addition the presence of jaundice, ascites, splenomegaly and dilated para-umblical veins were searched for during physical examination.

Complete blood count, alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum albumin, serum bilirubin, blood urea, serum creatinine and INR were tested for all patients. Anti-body to hepatitis C virus (HCV Ab) and hepatitis B surface antigen were tested for patients when was possible by third generation ELISA. Antinuclear antibody (ANA) and anti-smooth muscle antibody (ASMA) were tested in patients negative for HCV Ab and HBs Ag.

In patients with decompensated liver disease, cirrhosis was diagnosed on the basis of clinical, laboratory, and ultrasonography data. Patients with compensated liver disease (n=37), diagnoses of cirrhosis based on histopathological assessment of liver tissue (n=10), or APRI score (n=27) (8). Severity of liver cirrhosis evaluated according to Child-Turcotte-Pugh classification (9).

Abdominal ultrasonography and Color Doppler ultrasonography was performed for all patients using ALOKA prosound series α7 (Germany), with 3.5 MHz convex probe after overnight fast. Patients were assessed in supine position during quite respiration. Splenic longest axis was also measured and classified into, normal (≤ 12 cm)(10) mildly enlarged (13-15 cm), moderate (16-18) or massive (>18) (11). Ascites, if present, was presented as mild, moderate or marked (10).

In Color Doppler ultrasonography, measurements of diameter, flow direction and flow velocity in the LGV were done in all patients. The site of left gastric vein destination at portal circulation, as well as the presence of para-esophageal varices was assessed. The LGV usually originates from the portal-splenic vein junction or its vicinity and runs to the gastro-esophageal junction. It was identified longitudinally by ultrasonography in a left oblique scan in the epigastric region. Blood flow measurement was made in the straight portion of the LGV, usually within 5 cm from its origin. The diameters of the LGV were calculated from the inner surface within the vessel as seen in a longitudinal view. The sample volume was selected from 2 to 5 mm widths to include the width of the vessel. Flow direction was assessed according to the uperiopward or downward position of the Doppler waveform over the baseline (hepatopetal, bidirectional, or hepatofugal). The beam-vessel angle was less than 60° in every patient. Flow velocity was calculated as an average value of three consecutive measurements. The radiologist was blind to any information on the endoscopic findings of varices and the portal pressure.

Upper GIT endoscopy was done for all patients using PENTAX EPM 3500 (Japan). Esophageal varices were graded according to the criteria of Japanese Research Society for Portal hypertension and endoscopic finding of portal vein hypertension into, no varices, in straight and small caliber varices (F1), moderately enlarged beady varices (F2), or markedly enlarged nodular or tumor-shaped varices (F3). Gastric varices were classified into cardiac or forniceal type. Portal hypertensive gastropathy was depicted as present or absent.

3. Results:

One hundred consecutive cirrhotic patients were included in this study. 50 male patients and 50 female patients with mean age 52.6 ± 9.1 (31 – 70) years. Hepatitis C virus (HCV) was the cause of cirrhosis in 84 patients, Hepatitis B virus (HBV) in 3 patients, autoimmune with anti smooth antibody muscle antibody (ASMA) in 3 patients and cryptogenic in 10 patients. 37 patients were child A, 23 were child B while 40 patients were child C (Table 1).

There are 86 patients had history of hepatic coma, 43 patients had ascites, 75 patients had splenomegaly (Table 2). The mean of S. Alb was 3 ± 0.8 mg/dl, the mean of INR was 1.3±0.3 and mean of creatinine 0.9 ±0.4. 74 patients could be assessed but 26 patients couldn`t be assessed. Mean diameter of LGV was 6±1.5 mm with mean flow velocity 15.7 ± 6.7 cm/s. 55 patients had hepatopetal direction of flow, 16 patients had hepatofugal direction and only 3 patients had bidirection flow of LGV. 60 patients had para-oesophageal varices (PEV) by color Doppler ultrasound and 40 patients didn`t have.

There are 42 patients had portal hypertensive gastropathy.

According to presence of OV, 53 patients had OV and 47 patients had no OV. According to presence of gastric cardiac varices, only 9 patients had varices. And according to presence of gastric forniceal varices only 3 patients had varices. LGVD was increased more with increasing age, child C, presence of ascites, increasing S. bilirubin, decreased S. alb, decreased hemoglobin and thrombocytopenia.

Patients with child C, splenomegaly, increase bilirubin, low S. albumin and thrombocytopenia had more hpatofugal direction than others. Patients with Child C had more incidence of detection of PEV, in contrast patients with Child B. OV were present and were increasing in grades with increasing of age, male more than females, child class C more than A, presence of ascites, splenomegaly, increasing S. bilirubin, low S. albumin, increasing INR and thrombocytopenia.

Gastric forniceal varices were present more frequent with male than female, presence of ascites and increasing S. bilirubin. Gastric cardic varice had highly statistics significance similar to OV, but ALT had additional significant relation to cardiac varices rather than OV. There was highly significant relation between OV and LGV values. OV were found with more increase in LGVD and hepatofugal direction by color Doppler ultrasound and There was no relation between left gastric vein values and gastric forniceal varices. The gastric cardiac varices were found with increasing LGVD and hepatopfugal direction.

The percentage ratio of patients with hepatofugal direction was increased with presence of portal hypertensive gastropathy. In the same line, grading of PEV were found more with portal hypertensive gastropathy. LGV diameter, direction, presence of PEV by color Doppler parameter was found to be the best parameter to predict OV with sensitivity 100%, specificity83.3%, PPV88.9% and NPV100%.

The results of this study are tabulated through the following tables:

Table 1: Basic data of studied patients

Basic data	***n* (%)**
Sex	
Male	50 (50%)
Female	50 (50%)
Etiology of cirrhosis	
HCV	84 (84%)
HBV	3 (3%)
Autoimmune	3 (3%)
Cryptogenic	10 (10%)
Child class	
A	37 (37%)
B	23 (23%)
C	40 (40%)

Table 2: Clinical data of studied patients

Clinical data	*n* (%)
History of hepatic coma	
No	86 (86%)
Yes	14 (14%)
Ascites	
No	57 (57%)
Minimal	1 (1%)
Mild	9 (9%)
Moderate to Marked	33 (33%)
Splenomegaly	
No	25 (25%)
Mild	38 (38%)
Moderate	34 (34%)
Massive	3 (3%)

Table 3: Left gastric vein color Doppler values among studied patients.

LGV color Doppler values:	Mean ± SD	Range
LGV diameter.	6 ± 1.5 mm	3 – 11 mm
LGV flow velocity.	15.7 ± 6.7 cm/s	4 – 48.6 cm/s
Number of PEV by Doppler.	1 ± 1.3 column	0 – 5 columns
Max. O.V. column size.	0.3 ± 0.5 cm	0 – 2 cm
	Number	%
Grading of PEV by Doppler		
No	60	60
Mild	4	4
Moderate	5	5
Large	31	31
Direction of LGV flow.		
Not seen	26	26
Hepatopetal	55	55
Hepatofugal	16	16
Bidirectional	3	3
Site of LGV termination		
Not seen	26	26
Portal	55	55
Splenic	19	19

LGV=Left gastric vein, PEV=Para esophageal varices, O.V.=esophageal varices.

Table 4: Relation between basic data, clinical and laboratory values and left gastric vein diameter.

	LGV diameter			
	Normal	increased	X^2	*P*
Sex M F	 2(28.6%) 5(71.4%)	 34(50.7%) 33(49.3%)	1.5	0.4
Age	47.6 ± 4.6	54.3 ± 7.6	3.7	0.03
Etiology of cirrhosis HCV HBV Autoimmune. Crypt.	 7(100%) 0(0%) 0(0%) 0(0%)	 54(80.5%) 1(1.5%) 2(3%) 10(15%)	11.2	0.08
Child class A B C	 6(85.7%) 0(0%) 1(14.3%)	 28(41.8%) 20(29.9%) 19(28.3%)	27.2	0.000
Hepatic coma No Yes	 7(100%) 0(0%)	 61(91%) 6(9%)	8.5	0.01
Ascites No Minimal Mild Marked	 7(100%) 0(0%) 0(0%) 0(0%)	 46(68.7%) 0(0%) 7(10.4%) 14(20.9%)	36.1	0.000
Splenomegaly No Mild Moderate Massive	 2(28.6%) 3(42.8%) 2(28.6%) 0(0%)	 16(23.9%) 29(43.3%) 20(29.8%) 2(3%)	4.3	0.6
ALT	50.1±24.3	44.2±2.66	0.24	0.8
AST	57.7±25.9	62.2±37.2	2.4	0.09
T. Bilirubin	0.8 ± 0.2	1.7 ± 1.1	6.8	0.002
S. Albumin	3.7 ± 0.6	3.1±0.8	9.4	0.000
INR	1.2 ± 0.3	1.3±0.3	2.3	0.1
S. Creatinine	0.8 ± 0.2	0.9±0.3	0.4	0.7
Blood urea	27.1±4.7	32.4±8.1	1.2	0.3
Hemoglobin	13.3 ± 1.7	10.9±2.6	3.1	0.05
WBCs $\times 10^3$	6.5 ± 2.1	5.9±2.4	0.3	0.7
Platelet$\times 10^3$	181±88	127±85	4.5	0.01
APRI	2 ± 3.1	1.8±2.2	1.5	0.2

INR=International normalize ratio, APRI=AST Platelet Ratio Index. ALT=Alanine aminotranseferase, AST=Aspartate aminotransferase.

Table 5: Relation between basic data, clinical and laboratory values and left gastric vein flow direction parameters.

	LGV flow direction				
	Petal	Fugal	Bi	X^2	*p*
Sex M F	 27(49.1%) 28(50.9%)	 8(50%) 8(50%)	 1(33.3%) 2(66.7%)	0.3	0.9
Age	53.8±7.8	54.4±7.6	48.3±2.1	1.6	0.19
Etiology HCV HBV Autoimm. Crypt.	 45(81.8%) 1(1.8%) 1(1.8%) 8(14.6%)	 13(81.3%) 0(0%) 1(6.3%) 2(12.4%)	 3(100%) 0(0%) 0(0%) 0(0%)	2.5	0.87
Child class A B C	 27(49.1%) 16(29.1%) 12(21.8%)	 4(25%) 4(25%) 8(50%)	 3(100%) 0(0%) 0(0%)	9.8	0.04
Hepatic coma No Yes	 49(89.1%) 6(10.9%)	 16(100%) 0(0%)	 3(100%) 0(0%)	3.7	0.15
Ascites No Minimal Mild Marked	 39(70.9%) 0(0%) 7(12.7%) 9(16.4%)	 11(68.8%) 0(0%) 0(0%) 5(31.2%)	 3(100%) 0(0%) 0(0%) 0(0%)	6.9	0.14

Splenomegaly					
No	14(25.5%)	2(12.5%)	2(66.7%)		
Mild	28(50.9%)	4(25%)	0(0%)	14.9	0.02
Moderate	11(20%)	10(62.5%)	1(33.3%)		
Massive	2(3.6%)	0(0%)	0(0%)		
ALT	41.9±26.8	49.8±24.3	69.6±4.9	1.4	0.2
AST	59.5±39.1	64.3±25.3	89.3±14.2	2.4	0.07
T.Bilirubin	1.5±0.9	2.1±1.6	0.7±0.3	4.9	0.003
S. Albumin	3.2±0.8	2.8±0.8	4.1±0.1	8.5	0.000
INR	1.3±0.3	1.4±0.4	1±0.1	2.5	0.07
S. Creatinine	0.9±0.3	0.9±0.4	0.6±0.2	0.8	0.5
Blood urea	31.4±7.5	32.3±8.6	39±12.1	0.8	0.5
Hemoglobin	11.2±2.8	10.9±2.1	11.7±3.4	0.07	0.9
WBCs $\times 10^3$	6.1±2.4	5.6±2.2	4.9±0.95	0.5	0.7
Platlet $\times 10^3$	132±63	87±46	386±184	23.2	0.000
APRI	1.8±2.6	2.2±0.9	0.7±0.2	1.5	0.2

INR=International normalize ratio, APRI=AST Platelet Ratio Indix. ALT=Alanine aminotranseferase, AST=Aspartate aminotransferase.

Table 6: Relation between left gastric vein values and esophageal varices.

	Esophageal varices				X^2	*P*
	No OV	F1	F2	F3		
LGV diameter:						
Normal	6(12.8%)	0(0%)	1(3.6%)	0(0%)	16.2	0.01
Increased	36(76.7%)	7(63.7%)	15(53.6%)	9(64.3%)		
LGV flow direction:						
Hepatopetal	36(85.7%)	7(100%)	11(68.8%)	1(11.1%)		
Hepatofugal	4(9.5%)	0(0%)	4(25%)	8(88.9%)	27.6	0.000
Bidirection	2(4.8%)	0(0%)	1(6.2%)	0(0%)		
Site of termination:						
Portal	29(61.7%)	7(63.4%)	12(42.9%)	7(50%)	16.6	0.01
Splenic	13(27.7%)	0(0%)	4(14.3%)	2(14.3%)		
Grading of PEV:						
No	34(72.3%)	9(81.8%)	9(32.1%)	8(57.1%)		
Mild	3(6.4%)	0(0%)	0(0%)	1(7.1%)	27.2	0.001
Moderate	3(6.4%)	1(9.1%)	1(3.6%)	0(0%)		
Large	7(14.9%)	1(9.1%)	18(64.3%)	5(35.7%)		

LGV=left gastric vein, PEV=Para esophageal varices by doppler.

Table 7: Relation between left gastric vein values and gastric forniceal varices.

	Forniceal varices			
	absent	present	X^2	*P*
LGV diameter:				
Normal	7(7.2%)	0(0%)	2.4	0.3
Increased	64(66%)	3(100%)		
LGV flow direction:				
Hepatopetal	53(74.6%)	2(66.7%)		
Hepatofugal	15(21.2%)	1(33.3%)	0.4	0.8
Bidirection	3(4.2%)	0(0%)		
Site of termination:				
Portal	52(53.6%)	3(100%)	3.7	0.2
Splenic	19(19.6%)	0(0%)		
Grading of PEV:				
No	60(61.7%)	0(0%)		
Mild	4(4.1%)	0(0%)	7.2	0.065
Moderate	5(5.2%)	0(0%)		
Large	28(28.9%)	3(100%)		

LGV=left gastric vein, PEV=Para esophageal varices by doppler.

Table 8: Relation between left gastric vein values and gastric cardiac varices.

	Cardiac varices			
	absent	present	X^2	*P*
LGV diameter:				
Normal	7(7.7%)	0(0%)	7.6	0.02

Increased	58(63.7%)	9(100%)		
LGV flow direction:				
Hepatopetal	52(80%)	3(33.3%)		
Hepatofugal	10(15.4%)	6(66.7%)	10.3	0.006
Bidirection	3(4.6%)	0(0%		
Site of termination:				
Portal	48(52.8%)	7(77.8%)	5.8	0.055
Splenic	17(18.7%)	2(22.2%)		
Grading of PEV:				
No	55(60.4%)	5(55.6%)		
Mild	4(4.4%)	0(0%)	2.2	0.5
Moderate	5(5.5%)	0(0%)		
Large	27(29.7%)	4(44.4%)		

LGV=left gastric vein, PEV=Para esophageal varices by doppler.

Table (9): Sensitivity, specificity, PPV and NPV of LGV parameters in detecting esophageal varices.

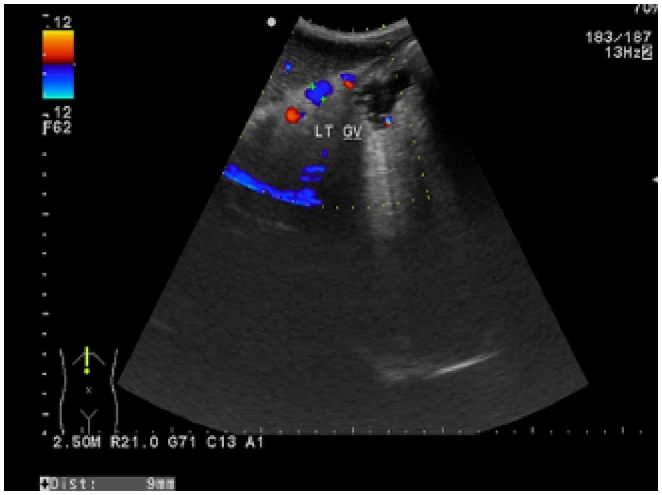
Parameters	Sensitivity	Specificity	PPV	NPV
LGV diameter.	96.8 %	14.3 %	45.5 %	85.7 %
LGV flow direction.	37.5 %	85.7 %	66.7 %	64.3 %
PEV by Doppler.	50.9 %	72.3 %	67.5 %	56.7 %
LGV diameter and flow direction.	92.3 %	50 %	66.7 %	85.7 %
LGV diameter and PEV.	100 %	31.3 %	64.5 %	100 %
LGV flow direction and PEV.	57.1 %	96.2 %	88.9 %	80.6 %
LGV diameter, flow direction and PEV.	100 %	83.3 %	88.9 %	100 %
LGV diameter, flow direction, PEV and Chlid A.	100 %	0 %	50 %	NaN
LGV flow direction, PEV and Child A.	16.7 %	85.7 %	50 %	54.5 %
LGV diameter, PEV, Child A.	100 %	0 %	12.5 %	NaN
LGV diameter and Child A.	66.7 %	0 %	7.1 %	0 %
LGV flow direction and Child A.	5.3 %	60 %	14.3 %	33.3 %
PEV and Child A.	4 %	50 %	11.1 %	25 %

LGV=left gastric vein, PEV=Para Esophageal Varices, PPV=Positive Predictive Value, NPV=Negative Predictive Value.

Table (10): Sensitivity, specificity, PPV and NPV of LGV parameters in detecting gastric varices.

Parameters	Sensitivity	Specificity	PPV	NPV
LGV diameter.	100 %	11.5 %	18.2 %	100 %
LGV flow direction.	50 %	80.6 %	33.3 %	89.3 %
LGV diameter and flow direction.	100 %	36.8 %	33.3 %	100 %

LGV=left gastric vein, PPV=Positive Predictive Value, NPV=Negative Predictive Value.



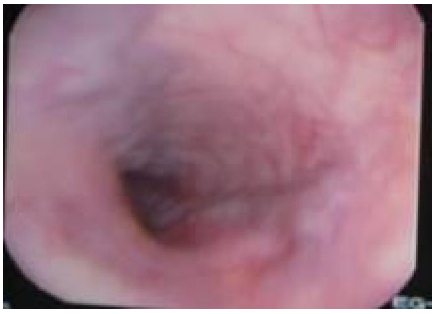


Fig. 1: A-Color Doppler ultrasound shows the hepatopetal flow direction of LGV (measures 9 mm).
B- Upper G.I.T endoscopy shows F1 straight and small caliber varices.

4. Discussion

Liver cirrhosis represents a tremendous health burden in Egypt. Portal hypertension and the development of esophageal varices "OV" and gastric varices "GV" that carries the risk of bleeding is a major complication of liver cirrhosis. Oesophageal varices are detected in approximately 50% of cirrhotic patients, at their diagnosis. OV is more common in Child-Pugh class C patients compared to Child-Pugh class A patients (5). Once OV is formed it can bleed at a rate of 5–15% per year. The risk of bleeding is higher in patients with large varices >5mm diameter, higher Child-Pugh score, and those with red wall markings on varices at endoscopy.

Current guidelines recommend that all cirrhotic patients should undergo screening endoscopy at diagnosis to identify patients with varices (5). Inter-observer variation in detection and grading had been reported by many researchers (12-14). The current guidelines result in a significant economic burden, especially that up to 50% of patients may not develop OV up to10 years after the initial diagnosis (15).

Many investigators explored the usefulness of different clinical, laboratory, and imaging parameters in the screening of OV. Platelet count, Child-Pugh score, platelet count / spleen diameter ratio, Liver and spleen elastography, variable abdominal ultrasound indices as portal vein diameter and flow speed were all tried but sensitivity and specificity were not enough to recommend the use of any of these tests (16).

Esophageal varices were documented by upper endoscopy in 53 patients 53%, Zardi *et al.,* (16) reported an incidence of 57% of OV in patients with liver cirrhosis, where 84 % of the studied patients had HCV as the cause of cirrhosis. The demographic characteristics of patients showed that older patients had higher grades of OV (p= 0.04) and also had cardiac varices (p 0.006) but not forniceal varices. This coincides with other researchers (17) who reported in a large prospective study (582 patients without history of bleeding) that older patients had higher grades of OV. On the other hand this is conflicting with (18) who reported that age didn`t affect presence of OV. However their study design was retrospective, which might explain the conflicting results.

Likely male patients were more prone to have both OV (p 0.002) & Forniceal varices (p 0.04). This is in the same line with Barrera *et al.* (19) who correlates between male and high risk esophageal varices (HREV) in cirrhotic patients. On the other hand, Agha *et al.* (20) reported that gender didn`t affect presence or absence of OV in schistosomiasis. This controversy may be due to type of patients which are cirrhotic patients in (19) who coincide with our patients and bilharziasis in (20) who is opposite to my results.

Low albumin and thrombocytopenia were common finding in OV and cardiac varices. While increased bilirubin and ascites were common finding in OV and forniceal varices. Patients with ascites were more prone to have OV and more high grades of OV, this results is similar to results of Bota *et al.*(21) who found that ascites is more frequent present with presence of OV and specifically with high grades of OV.

Splenomegaly correlated with presence of OV and grading of OV. Furthermore, the grade of OV increased head to head in relation to the size of the spleen. Thrombocytopenia and hyperbilirubinemia were independent risks for presence of OV. This is parallel to (22-24). Moreover in one study, (25) concluded that splenomegaly and thrombocytopenia are the best noninvasive predictors for OV.

Although, the results showed that serum albumin was decreased with presence of OV and increasing grading of OV. But it cannot be reliably taken as a risk for OV, as there are many factors affecting albumin level in blood. This result is in the same line with (26) who reported that hupoalbuminemia is significantly decreased with OV, but it is better to add spleen size to it as predictors for OV.

In this study it was found that there was no relation between white blood cells (WBCs) and OV. This result is similar to results of Alcantara *et al.* (27) who reported that there is no statistically significant correlation between white blood cells and OV. High International normalize ratio (INR) was a risk of OV existence. And it is also affected by many factors. Likely more than one study had reported similar conclusion (21, 27).

Patients with higher Childs' classification were more frequent to have higher grade of OV and cardiac varices but not forniceal varices. Patients with large grades of OV had high child classification more than patients with absence OV or low grades of OV. Like my results there are numerous studies concluded that high Child's score is an isolated risk factor for both OV detection and grade (28). Most of studies didn`t explore cardiac and forniceal varices, but they dealt with them as one unit (gastric or fundal varices). And it has been reported that high Child ` s classification is a risk factor for presence of fundal varices (29).

APRI score had no relation to OV and fundal varices. This is head to head to (30) who concluded that APRI score hadn`t impressiveness to be predictor for OV. Most of clinical and laboratory predictors for OV were also predictors to cardiac varices. This may be explained that cardiac varices originated mainly as extensions to OV.

In this study, demographic characteristics of patients with left gastric vein dilatation showed that older patients were found to have greater left gastric vein diameter (LGVD) (p 0.03). This observation might be a reflection to the fact that older patients were more frequent to have portal hypertension as proved by having OV. Neither the cause of cirrhosis, nor the co existence of Bilharziasis had a relation to LGVD. Which point that none of these factors carries any additional load to the readily existing portal hypertension.

In this study LGVD and LGV direction of flow showed a relation to patients laboratory markers. Independent predictors of high LGVD were low hemoglobin (p=0.05), low platelet (p=0.01), decreased albumin (p=0.002) and high bilirubin (p=0.002). LGV direction showed also correlation with platelet count, bilirubin and serum albumin. This may be due to correlation of these parameters with OV. The results showed that LGVD increased head to head in relation to the grade of OV (p=0.01).This is in line with (31) who reported that the diameter of the LGV trunk increased with increasing varix size.

The results also showed that the direction of flow in the LGV had a positive relation to the grade of OV as 85.7% of patients without varices showed centripetal flow versus 9.5% showing hepatofugal flow. While patients with grade F3 showed 88.9% hepatofugal flow versus only 11.1% hepatopetal flow. Similar results were reported in more than one study as the hepatofugal flow and speed were related to the development of higher grades of OV (2, 31, 32).

Moreover, in one study the author concluded that the velocity of the flow is more important than the diameter in predicting high grade OV (29). Parallel to these results (33) who concluded that rapid hepatofugal flow in the LGV velocities more useful than LGVD in predicting recurrence of varices following endoscopic treatment. Unlike the previous studies both LGVD and speed of flow showed to be the most relevant tests to the portal vein diameter. Moreover LGVD proved to be a good predictor of the advanced OV as it detected 64% of patients with stage 2 and 3 OV and it reported the sensitivity of 75% in detecting OV (16). Similar data were also reported by (34).

Another observation in this study, that the detection of para-oesophageal varices, grading of para-oesophageal varices, number of para-oesopgageal varices columns and the maximum diameter of these varices were all positively linked to the grade of OV. This is on the same line with (35) who concluded that the detection of para-oesophageal varices is a sensitive marker that is linked to bleeding OV. In the same line, Para-oesophageal varices had the same haemodynamics as the LGV and both were able to predict early variceal recurrence after sclerotherapy (36).

Anatomically, Porto-systemic Collaterals are divided into periesophageal collateral veins and para-oesophageal collateral veins. Periesophageal collateral veins were thought to be more important predictor of OV than para-oesophageal collaterals (37-39). On the other hand only LGV flow direction and presence of para-oesophageal varices by Doppler correlated with the presence of gastric varix. This is in contrast with (33) who reported that hepatofugal flow correlated with OV but not forniceal varices. The existence of other non tested shunts as gastro-renal or para-umblical collateral can explain the dysconcordance of the results of this study. Additionally, lack of studies testing haemodynamics of gastric varices hindered our trial to find relevant data, as many studies excluded the patients with gastric varices from analysis (31) or either low number of patients with gastric varices were recruited in the study only 8 patients. Sato *et al.* (40) like our study only 12 patients had gastric varices. We faced the same situation in our study as only 12 patients had gastric varices.

The complex anatomy of the portal-systemic circulation and the presence of other types of shunts that were not included in different studies can explain the conflicting results between all studies. To overcome this limitation, we studied the combined "bivariate" analysis of different parameters on the detection of both OV and gastric varices. The concomitant detection of elevated LGVD and detected para-oesophageal varices highly correlated with detection of OV. This observation proved to be also true for gastric cardiac varices but not to gastric forniceal varices. It is relevant to say that anatomically, forniceal varices are connected to splenic vein and short gastric veins.

In view of these results we studied the sensitivity and specificity of each individual test and a formula of more than one test in peruse of the most sensitive and probably specific formula in detection OV and fundal varices (FV). LGVD proved to be a sensitive test in detecting OV 96.7% but the specificity was only 14.2%.The sensitivity was even higher with FV 100% and specificity of 11.4%. In another study the LGVD reported a sensitivity of 75% in detecting OV, the same sensitivity of 75% was reported in an additional study (16).

Direction of flow had a sensitivity of 37.5% and a specificity of 85.7% in detecting OV. Unlike our results (34) reported a higher sensitivity"83%" for flow direction in detecting OV. However in their study almost 43% of the patients had history of haematemesis, which was one of the exclusion criteria in the current study. On the other hand in my study the utility of elevated LGVD in predicting fundal varix

showed a sensitivity of 50% and a specificity of 80.6%.

Based on these results LGVD proved to have a high sensitivity but very low specify in detecting both OV and FV. But the direction of flow showed an opposite pattern with low sensitivity and high specificity for detecting both OV and FV. The concomitant detection of elevated LGVD and direction of flow showed a sensitivity of 50.9% and a specificity of 72.3% for detecting OV, while it showed` a sensitivity of 92.3% and a specificity of 50% in detecting FV. The combined detection of high LGVD and presence of paraoesophageal varices by Doppler recorded a sensitivity of 100% and a specificity of 31.2%. While the triple detection of high LGVD, abnormal direction of flow and presence of paraoesophageal varices recorded 100% senstivity and 83% specificity.

We used color doppler ultrasound to elicit left gastric vein parameters to can predict OV, but there are another route to elicit left gastric vein parameters, the most accurate one of them is percutaneous transhepatic splenoportography which give direct information about collaterals of portosystemic shunts, OV, left gastric vein, short gastric veins, splenic vein and portal vein (41). Its use is not reliable as it doesn`t demonstrate well direction of flow of left gastric vein, in addition to this it is invasive maneuver, may be more cost than upper GIT endoscopic study and need more skills to be done.

On the opposite side, it give more details about porto venous system as whole than color Doppler, it can evade tense ascites and gaseous distention which hinder color Doppler ultrasound and can safely avoid anomalies of left gastric vein and OV. But still color Doppler is better than any other way to elicit left gastric vein parameters which give more data than percutaneous transhepatic splenoportography specially direction of flow which is extremely important to predict OV.

Conclusion

In conclusion, The results suggest that portal hemodynamics changes in cirrhotic patients are characterized by passive congestion and increased blood flow. However, these 2 features had different preponderances in different parts of the portal venous system. Flow velocity, direction and diameter of the left gastric vein done by ultrasonic Doppler study may be play a role in evaluation of portal hypertension and relation with the development and size of varices.

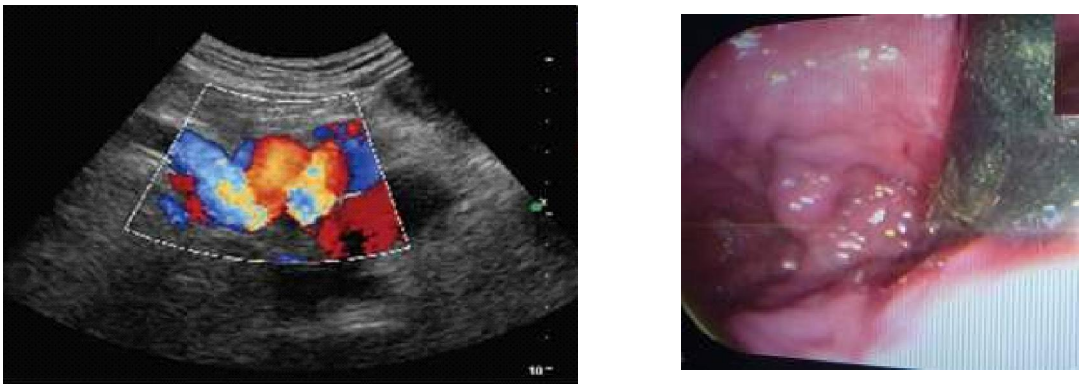


Fig. 2: A. Color Doppler ultrasound of para-oesophageal varices with color aliasing.
B- Upper G.I.T endoscopy shows F3 markedly enlarged nodular or tumor-shaped cardiac varices.

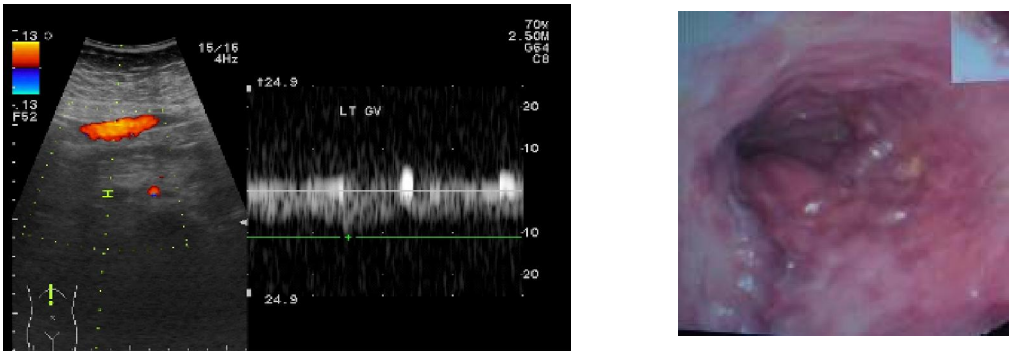


Fig.3: A- Color Doppler and spectral analysis shows hepatofugal flow and velocity (11 cm/sec) at left gastric vein.
B- Upper G.I.T endoscopy shows F2 moderately enlarged beady varices.

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