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ORIGINAL ARTICLE

Gall Bladder Wall Thickness as Noninvasive Screening Parameter of Oesophageal Varices in Compensated Cirrhotic Patients

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Abstract

Background: **About 40% of people with hepatic cirrhosis also developed oesophageal varices (OV). The most serious portosystemic shunts caused by portal hypertension are OV.**

Aim and objectives: **The goal of the study was to establish whether or not the gall bladder wall thickness (GBWT) is a reliable, noninvasive predictor of EV in people who have cirrhosis.**

Patients and methods: **From August 15, 2022, through March 1, 2023, 120 patients with chronic liver disorders and liver cirrhosis were recruited from the inpatient and outpatient clinics at Sayed Galal Hospital. Two groups of patients were created, one with OV and one without. Sixty cirrhotic patients without OV (non-OV group) and 60 cirrhotic cases with OV (OV group) were studied.**

Results: **Revealed that the area under the curves (AUCs) for both the GBWT and the platelet count to splenic diameter ratio were highly selective ($P < 0.001$), but that there was no statistically significant difference among the GBWT and the platelet count to splenic diameter ratio ($P > 0.05$). There was a significant correlation among OV and [Child−pugh score, portal hypertension gastropathy (PHG), platelet count, international normalized ratio (INR), albumin, white blood cell, hemoglobin, GBWT (mm), spleenic diameter (mm), portal vein (PV) diameter (mm), and platelet count to spleenic diameter ratio, GBWT (mm), spleenic diameter (mm), and PV diameter (mm)].**

Conclusion: **Given the correlation between GBWT and OV size, it may be possible to employ this non-invasive technique for predicting the existence of OV. The ratio of platelets to splenic longitudinal diameter (PLT/SLD) did not differentiate between OV patients and healthy controls.**

Keywords: Cirrhosis, Esophageal varices, Gall bladder wall thickness

1. Introduction

Hepatitis B and C virus infection, alcoholism, and autoimmune liver disorders are the principal causes of liver cirrhosis.[1]

Portal hypertension can cause ascites, spontaneous bacterial peritonitis, and portosystemic collaterals. This condition is considered to be a problem of clinical significance. It has been discovered that oesophageal varices, often known as OV, progress at a rate of 12% in cirrhotic individuals who have clinically significant portal hypertension.[2]

There is a high recurrence incidence after the initial bleeding incident,[3] and the mortality rate at six weeks is as high as 37%, even though it has decreased over the years.[4]

All of the guidelines recommend performing a screening for OV at the same time as liver cirrhosis is being diagnosed. The diagnostic method that is regarded as being the 'gold standard' is called an upper gastrointestinal (GI) endoscopy. Patients with decompensated cirrhosis who do not have OV are required to have endoscopic reexamination once every year, while patients with compensated disease

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are only required to undergo the procedure once every 2–3 years.[5]

In nations with lesser health care standards, recurrent endoscopic controls of cases with severe liver fibrosis or liver cirrhosis are not usually generally available despite being justified. Therefore, it is of great interest to find noninvasive predictors of portosystemic collaterals. Unfortunately, the gall bladder also drains venous blood straight into the liver via a network of tiny arteries.[6]

Small veins bring blood to the cystic duct, where it meets the common bile duct and portal venous system.[7]

The study set out to determine if GBWT might be used to predict OV in cirrhotic individuals without resorting to invasive procedures.

1.1. Patients and methods

This study involved 120 cases of chronic liver diseases with liver cirrhosis, selected from inpatient and outpatient clinic of Sayed Galal hospital from 8-15-2022 to 3-1-2023. Group I consisted of 60 cirrhotic patients who did not have OV (the non-OV group), and group consisted of 60 cirrhotic patients who did have OV (the OV group). Blood tests, an endoscopy of the upper digestive tract to look for ovarian cysts (OV), and an ultrasound of the abdomen were all performed. Inclusion criteria: patient diagnosed with liver cirrhosis and Exclusion criteria: patients with chronic calculary cholecystitis, cases with hepatocellular carcinoma (HCC) and other malignancies and nonalcoholic fatty liver disease (NAFLD) (where grade III with Non visualized gall bladder wall).

2. Methods

Each participant underwent the following: comprehensive clinical examination and thorough history collection to include cardiovascular, chest, abdominal and neurological examination, Laboratory investigations, abdominal ultrasound for assessment of: liver status and size, GBWT, spleen diameter, portal vein (PV) diameter (Device ultrasound used was GE logiq p7), and upper endoscope for detection and grading of OV.

2.1. Statistical analysis

The recorded information was analyzed using SPSS (SPSS Inc., Chicago, IL, USA) version 23.0 for the social sciences. Quantitative information was shown using averages, SD, and limits. Quantitative and percentage data were also provided for qualitative characteristics. The Spearman's rank correlation coefficient, scatter plot, receiver operating characteristic (ROC) curve, and probability (*P* value) were among the statistical analyses conducted.

3. Results

Table 1 showed a comparison between non-OV group and OV group in this point age in non-OV group 58.47 ± 3.66 and OV group 58.41 ± 3.12. Gender in Non-OV group women 25 (41.7%) men 35 (58.3%) and OV group women 24 (40.0%) men 36 (60.0%). Child–pugh score in non-OV group 60 (100.0%) A group and OV group 13 (21.7%) A group 42 (70.0%) B group 5 (8.3%) C group. Ascites in Non-OV group, no cases had ascites but in OV group 5 (8.3%) had ascites

Table 1. Comparison among non-oesophageal varices group and oesophageal varices group regarding baseline characteristics.

Baseline characteristics	Non-OV Group ($n = 60$)	OV Group ($n = 60$)	Test value	$n = -$value
Age (years)				
Mean ± SD	58.47 ± 3.66	58.41 ± 3.12	*t*:0.094	0.925
Range	50–65	50–65		
Sex				
Female	25 (41.7%)	24 (40.0%)	χ^2:0.034	0.853
Male	35 (58.3%)	36 (60.0%)		
Child–pugh score				
A	60 (100.0%)	13 (21.7%)		
B	0	42 (70.0%)	x^2:77.260	<0.001**
C	0	5 (8.3%)		
Ascites				
No	60 (100.0%)	55 (91.7%)	χ^2:5.217	0.022*
Yes	0	5 (8.3%)		
PHG				
No	60 (100.0%)	13 (21.7%)	χ^2:77.260	<0.001**
Yes	0	47 (78.3%)		

The data are existing as mean standard deviation and number (%).
Using as: *t*-test for independent samples; chi-square test, denoted by χ^2.
PHG, portal hypertension gastropathy.
Significance *P* value of * is less than 0.05 & *P* value of ** is less than 0.01.

and 55 (91.7%) no ascites. PV in non-OV group in normal blood pressure and OV group 47 (78.3%) suffered from portal hypertension gastropathy (PHG) but the rest of cases were not.

Table 2 showed a comparison among non-OV group and OV group according to ultrasound parameters. GBWT (mm) in non-OV group 2.92 ± 0.31 and OV group 4.43 ± 0.48. Spleenic diameter (mm) in non-OV group 115.58 ± 4.27 and OV group 143.80 ± 10.52. PV diameter (mm) in non-OV group 11.64 ± 0.57 and OV group 13.48 ± 0.94.

Table 3 presented that, there was −ve relationship among GBWT and alanine transaminase (ALT), spleenic diameter and [Albumin, white blood cell (WBC), fasting blood glucose (FBG), low-density lipoprotein (LDL) and alkaline phosphatase (ALP)], PV diameter and (FBG, LDL, and ALT) and Platelet count to spleenic diameter ratio, and [age, international normalized ratio (INR), albumin, WBC, HB, total cholesterol, LDL, high-density lipoprotein (HDL), ALT, aspartate transaminase (AST), and ALP].

Table 4 showed that, there was negative correlation between GBWT and (age, INR, albumin, WBC, HB, FBG, AST, and ALP), spleenic diameter and (INR, ALT, AST, and ALP), PV diameter and (age, INR, albumin, WBC, HB, FBG, LDL, AST, and ALP) and Platelet count to spleenic diameter ratio and (total cholesterol, LDL, and ALT).

Table 5 showed that, there was a statistically significant difference among presence of OV and (Child−pugh score, PHG, Platelet count, INR, Albumin, WBC, HB, GBWT (mm), Spleenic diameter (mm), PV diameter (mm) and Platelet count to spleenic diameter ratio.

Areas under the curves (AUCs) with P values ($P < 0.001$) for both GBWT and Platelet count to spleenic diameter ratio can be seen in Table 6, however, a comparison of the two indices reveals no statistically significant difference ($P > 0.05$) between them Fig. 1.

4. Discussion

In this study, we hypothesized that noninflammatory GBWT evaluated by ultrasound might be utilized as a noninvasive predictor of OV in cirrhotic cases. We

Table 2. Comparison among nonoesophageal varices group and oesophageal varices group regarding ultrasound parameters.

Ultrasound parameters	Non-OV Group ($n = 60$)	OV Group ($n = 60$)	Test value	P value
GBWT (mm)				
Mean ± SD	2.92 ± 0.31	4.43 ± 0.48	*t*:20.644	<0.001**
Range	2.1–3.4	3.2–5.2		
Spleenic diameter (mm)				
Mean ± SD	115.58 ± 4.27	143.80 ± 10.52	*t*:19.257	<0.001**
Range	107–123	125–160		
PV diameter (mm)				
Mean ± SD	11.64 ± 0.57	13.48 ± 0.94	*t*:12.928	<0.001**
Range	10.3–12.4	11.4–14.9		

Significance P value of ** is less than 0.01.
PV, portal vein.

Table 3. Correlation between GBWT (mm), Spleenic diameter (mm), PV diameter (mm) and Platelet count to spleenic diameter ratio, using Pearson correlation coefficient among non-oesophageal varices group.

Parameters	GBWT (mm)		Spleenic diameter (mm)		PV diameter (mm)		Platelet count to spleenic diameter ratio	
	r value	P value	r value	P value	r value	P value	r value	P value
Age (years)	0.088	0.501	0.252	0.053	0.154	0.239	−0.072	0.586
INR	0.052	0.695	0.154	0.240	0.002	0.986	−0.007	0.956
Albumin	0.089	0.500	−0.073	0.579	0.066	0.614	−0.038	0.772
WBC	0.045	0.731	−0.065	0.621	0.014	0.914	−0.017	0.896
HB	0.057	0.664	0.175	0.181	0.021	0.873	−0.039	0.770
FBG	0.011	0.933	−0.167	0.201	−0.002	0.986	0.027	0.839
Total cholesterol	0.154	0.241	0.135	0.304	0.112	0.395	−0.162	0.217
LDL	0.019	0.885	−0.089	0.498	−0.006	0.962	−0.019	0.884
HDL	0.229	0.078	0.400	0.002*	0.205	0.116	−0.243	0.062
ALT	−0.035	0.788	0.057	0.667	−0.044	0.739	−0.009	0.948
AST	0.151	0.250	0.096	0.465	0.150	0.253	−0.181	0.166
ALP	0.084	0.525	−0.050	0.705	0.078	0.553	−0.089	0.499

r-Pearson correlation coefficient.
ALP, alkaline phosphatase; ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; FBG, fasting blood glucose; PV, portal vein.

Table 4. Correlation between GBWT (mm), spleenic diameter (mm), PV diameter (mm) and platelet count to spleenic diameter ratio, using Pearson correlation coefficient among oesophageal varices group.

Parameters	GBWT (mm)		Spleenic diameter (mm)		PV diameter (mm)		Platelet count to spleenic diameter ratio	
	r value	*P* value	r value	*P* value	r value	*P* value	r value	*P* value
Age (years)	−0.150	0.253	0.007	0.960	−0.153	0.244	0.188	0.149
INR	−0.242	0.062	−0.063	0.631	−0.186	0.154	0.157	0.232
Albumin	−0.044	0.736	0.143	0.276	−0.063	0.635	0.020	0.881
WBC	−0.178	0.173	0.114	0.387	−0.099	0.453	0.052	0.691
HB	−0.121	0.356	0.195	0.136	−0.152	0.245	0.083	0.529
FBG	−0.103	0.435	0.174	0.183	−0.095	0.468	0.073	0.582
Total cholesterol	0.042	0.752	0.208	0.111	0.005	0.970	−0.115	0.380
LDL	0.027	0.837	0.174	0.184	−0.011	0.932	−0.105	0.423
HDL	0.019	0.886	0.078	0.554	0.026	0.842	0.002	0.989
ALT	0.091	0.487	−0.018	0.890	0.058	0.660	−0.051	0.699
AST	−0.078	0.555	−0.192	0.142	−0.108	0.412	0.165	0.209
ALP	−0.062	0.638	−0.197	0.132	−0.005	0.967	0.128	0.331

ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; FBG, fasting blood glucose; INR, international normalized ratio; PV, portal vein.

Table 5. Multiple logistic regressions for the variables connected with the presence of oesophageal varices.

Parameters	OR	95% C.I.		*P* value
		Lower	Upper	
Age (years)	2.334	2.090	2.604	0.115
Sex	2.541	1.969	3.275	0.168
Child–pugh score	1.955	1.862	2.053	0.047*
Ascites	0.710	0.287	1.764	0.122
PHG	6.791	3.094	18.434	0.040*
Platelet count	2.002	1.853	2.161	0.014*
INR	0.954	0.188	2.197	0.011*
Albumin	3.801	2.228	6.481	0.039*
WBC	9.679	3.600	18.685	0.021*
HB	2.911	2.177	3.894	0.008*
FBG	9.008	4.762	25.299	0.135
Total cholesterol	2.217	1.985	2.474	0.109
LDL	2.414	1.871	3.111	0.259
HDL	1.857	1.769	1.950	0.139
ALT	0.675	0.272	1.675	0.416
AST	6.452	2.939	17.513	0.066
ALP	1.902	1.760	2.053	0.133
GBWT (mm)	0.907	0.179	2.087	0.017*
Spleenic diameter (mm)	3.611	2.116	6.157	0.024*
PV diameter (mm)	9.195	3.420	17.751	0.039*
Platelet count to spleenic diameter ratio	2.765	2.068	3.699	0.003*

ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; C.I, confidence interval; FBG, fasting blood glucose; INR, international normalized ratio; PV, portal vein; OR, odds ratio; PHG, portal hypertension gastropathy.
Significance *P* value of * is less than 0.05.

reasoned that the GBWT would be useful in the diagnosis of portal hypertension and OV since the gall bladder receives its drainage via the PV.[8]

The current analysis uncovered a disparity in GBWT that was statistically significant ($P < 0.001$) among the cirrhotic group with OV (group II) and the cirrhotic group without OV (group I). Cases with liver cirrhosis and OV had a mean GBWT of 4.43 ± 0.48 mm, whereas those without OV had a GBWT of 2.92 ± 0.31 mm.

Consistent with the findings of Shamsi *et al.*, who found that the mean GBWT of cases without OV (2.7 ± 0.1 mm) was substantially lower ($P < 0.01$) than that of cases with OV (5.6 ± 0.2 mm), we find the same result. It was once believed that low albumin levels caused GBWT in cases with chronic liver disease.[9]

It has been suggested that chronic liver disease cases who do not have hypoalbuminemia or ascites can develop congestive cholecystopahty owing to portal hypertension, which causes edema and congestion in the gallbladder wall. Patients who have congestive gastropathy have a thicker stomach wall.

GBWT with a cut-off value greater than or equal to 3.1 mm had a sensitivity of 76.5%, specificity of 74.3%, positive predictive value (PPV) of 87.8%, negative predictive value (NPV) of 56.5%, and receiver operating characteristic analysis calculated

Table 6. Receiver-operating characteristic (ROC) curve for prediction of oesophageal varices using the GBWT (mm), spleenic diameter (mm), PV diameter (mm) and platelet count to spleenic diameter ratio.

Parameters	Cut-off	Sen.	Spe.	PPV	NPV	AUC	*P* value
GBWT (mm)	≥3.1	76.5%	74.3%	87.8%	56.5%	0.846	<0.001**
Spleenic diameter (mm)	≥120	71.4%	82.9%	91.3%	56.9%	0.861	<0.001**
PV diameter (mm)	≥12	69.4%	82.9%	90.8%	52.7%	0.826	<0.001**
Platelet count to spleenic diameter ratio	≤1439.7	75.3%	88.6%	94.1%	59.6%	0.845	<0.001**

Significance *P* value of ** is less than 0.01.
NPV, negative predictive value; PV, portal vein; PPV, positive predictive value.

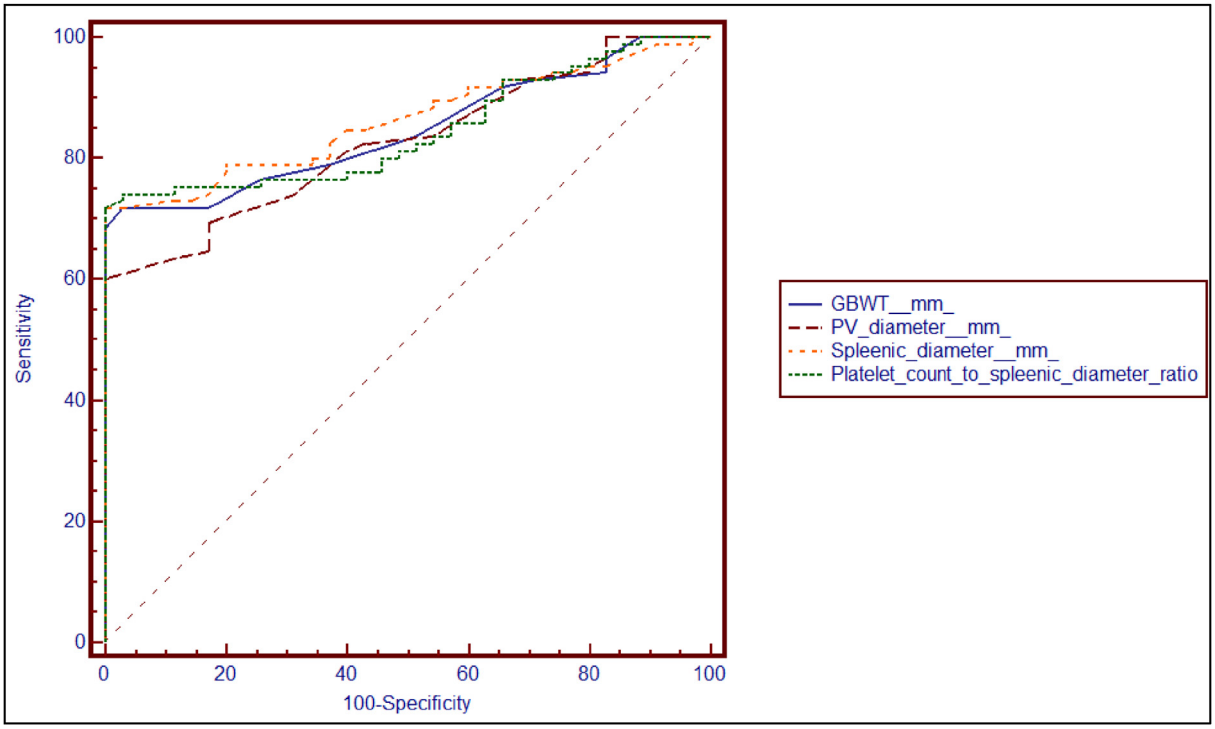

Fig. 1. Receiver operating characteristic curve for prediction of oesophageal varices using the GBWT (mm), spleenic diameter (mm), PV diameter (mm) and platelet count to spleenic diameter ratio. PV, portal vein.

AUC of 0.846 for the detection of OV, while GBWT with a cut-off value of 3.95 mm had a sensitivity of 92%, specificity of 95% and PPV of 86. In a prior study, the cut-off value for GBWT was 4.35 mm, which resulted in a sensitivity of sixty percent and a specificity of 90% for the occurrence of OV in pediatric cases. This value was determined by utilizing a sensitivity of 60% and a specificity of 90%.[10]

While a greater threshold was employed, the sensitivity was similar to what we found. Since Alcantara's study was based on data from infants with cirrhosis due to causes other than biliary atresia and autoimmune hepatitis, the varied patient cohorts may also account for the discrepancy.[10]

Since portal hypertension and GBWT were found to be linked in a small Chinese study, it seemed reasonable to assume that GBWT might serve as a predictor of OV as well.

Platelet count to spleenic diameter ratio was found to be significantly lower in cirrhotic individuals with varices than in those without varices ($P < 0.001$). We also discovered that platelets/splenic longitudinal diameter (PLT/SLD) had a sensitivity of 75.3%, specificity of 88.6%, PPV of 94.1%, and NPV of 59.6% for the diagnosis of OV and a sensitivity of 96%, specificity of 89%, PPV of 98.5% and NPV of 77.6% for the diagnosis of big varices, when the cutoff value was 1175.5.

Abo-Alsoud *et al.* found that 638.9 was the optimal cutoff value for identifying O.V., with 100% sensitivity, 97.5% specificity, 95.2% PPV, and 100% NPV. According to studies comparing the mean platelet count to spleen diameter in cirrhotic people with and without varices.[11]

Similar to our findings, Esmat *et al.* discovered that a platelet/spleen diameter limit of 1326.6 properly predicted OV with 96.3% sensitivity, 83.3% specificity, 96.3% PPV and 83.3% NPV.[12]

In contrast to the findings of Qamar and colleagues and Hassan and colleagues, who discovered that there was no significant difference in the (PLT/SLD) ratio between OV cases and controls, the present data suggest that there is such a difference. According to the findings of our research, 78.3% of the instances involving OV involved children B and C, whereas all of the cases involving non-OV involved children A. It is possible that this is due to the fact that earlier studies were conducted on individuals with Child 'A' and early 'B' liver cirrhosis, who had less platelet dysfunction. In the present study, the results of multivariate logistic regression analysis demonstrated that GBWT, platelet count, PV diameter, spleen diameter, and the ratio of platelet diameter to spleen diameter were all capable of functioning as independent predictors of OV.[13,14]

Tsaknakis *et al.*'s discovery that GBWT, ascites, platelet count, and spleen diameter independently predicted OV is supported by our data.[15]

The tiny sample size and unknown proportion of ultra-sonographers were among the study's drawbacks. However, such shortcoming is addressed by a few key elements of the process.

In the first place, the endoscopy and ultrasonography were done on the same day, reducing the possibility of a shift in the endoscopic results due to time or people. Second, we examined patients both retrospectively and prospectively, so we caught all the relevant clinical and laboratory data.

4.1. Conclusion

Since the size of an OV correlates with GBWT, this noninvasive technique may be used to predict the occurrence of OV. Patients with and without OV did not vary significantly in terms of PLT/SLD ratio, indicating that these approaches cannot be employed in the prediction of the existence of varices. It is possible that the varying degrees of liver illness in our patients are responsible for this disparity.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article.

Conflicts of interest

The authors declared that there were no conflicts of interest.

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