Print ISSN: 2321-6379
Online ISSN: 2321-595X

Original Article

Study of Non-Invasive Predictors of Esophageal Varices in Chronic Liver Disease

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Abstract

Introduction: Chronic liver disease is a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis.[1]

Aims and Objectives: The aims of the study were to study the ultrasonographic parameters and platelet count, to study platelet count and spleen diameter ratio in prediction of esophageal varices in chronic liver disease.

Materials and Methods: The study comprised 100 portal hypertensive patients who were admitted in Mahatma Gandhi Memorial Hospital between February 2017 and October 2018.

Discussion: Cirrhosis is the most advanced form of liver disease and variceal hemorrhage is one of its lethal complications. Over half of the patients with cirrhosis will develop varices. The risk of bleeding once OV formed is 20–35% within 2 years.

Conclusion: Ultrasonography of abdomen is a simple, convenient, and non-invasive method for assessing the severity of portal hypertension in patients and to predict the severity of esophagogastric varices indirectly.

Key words: Chronic liver disease, Esophageal varices, Portal hypertension

INTRODUCTION

Chronic liver disease is a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis.[1] Portal hypertension is the significant complicating feature of decompensated cirrhosis and is responsible for the development of ascites and esophageal varices, results in the development of collaterals to bypass the increased resistance to flow within the portal vein to return blood to systemic circulation.[2] PH refers to elevated HVPG >5 mm of hg. The increased hepatic resistance to blood flow caused by architectural disruption combined with intrahepatic vasoconstriction and vasodilatation in splanchnic vascular bed with increased splanchnic blood flow is the underlying pathology. Portal hypertension becomes clinically significant when the PPG increases above the threshold value of 10 mm Hg (e.g., formation of varices) or 12 mm Hg (e.g., variceal bleeding and ascites). PPG values between 6 and 10 mm Hg represent subclinical portal hypertension.[3,4] Bleeding from ruptured esophageal or gastric varices is the main complication of portal hypertension and a major cause of death. Most cirrhotic patients develop esophageal varices, with a lifetime incidence as high as 90%.[5] As per existing guidelines in a case of cirrhosis of liver, we are screening with upper gastrointestinal endoscopy to look for any esophagogastric varices present or not and grade the severity of varices and to start the prophylactic B. blockers. Doubts are expressed regarding the cost-effectiveness of universal screening with upper gastrointestinal endoscopy. A study done by Spiegel *et al.* published in journal HEPATOLOGY[6] concluded… "Empiric β blocker therapy for the primary prophylaxis of variceal hemorrhage is a cost-effective measure as the use of screening endoscopy to guide the therapy adds significant cost with only marginal increase in effectiveness." In this setting, if we can predict the severity of portal hypertension by a low cost and non-invasive method then we can use the upper gastrointestinal endoscopy for only high risk patients. Although the occurrence of esophageal varices and the

Access this article online

www.ijss-sn.com

Month of Submission : 07-2020
Month of Peer Review : 08-2020
Month of Acceptance : 08-2020
Month of Publishing : 09-2020

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time of gastrointestinal bleeding in portal hypertension cannot be exactly predicted, there are some endoscopic, ultrasonographic, laboratory parameters. and clinical signs associated with high risk of bleeding. Some studies have shown good correlation between ultrasonographic findings and platelet count and severity of esophagogastric varices. "In this study, we make an attempt to predict the esophageal varices based on ultrasonographic findings, platelet count, and platelet count spleen diameter ratio and its correlation with upper GI endoscopy.

Aims and Objectives

The aims of the study were as follows:

1. To study the ultrasonographic parameters and platelet count.
2. To study platelet count and spleen diameter ratio in prediction of esophageal varices in chronic liver disease.

MATERIALS AND METHODS

The study comprised 100 portal hypertensive patients who were admitted in Mahatma Gandhi Memorial Hospital between February 2017 and October 2018.

A detailed clinical history was recorded regarding age, sex, and duration of symptoms such as jaundice, distension of abdomen, hematemesis, and melena. All patients underwent complete clinical examination including detailed examination of gastrointestinal system. Routine biochemical investigations and liver function tests were done in every patient. Every recruited patient underwent ultrasonography and fiberoptic upper gastrointestinal endoscopy. Platelet count spleen diameter ratio was calculated.

Inclusion Criteria

Cases of portal hypertension admitted in the Department of General Medicine and Gastroenterology in MGM Hospital.

Exclusion Criteria

The following criteria were excluded from the study:

1. Cases of portal hypertension who are on blockers.
2. Cases of portal hypertension who underwent EST or EVL.
3. Cases of portal hypertension who underwent TIPS or shunt surgery.
4. Hepatocellular carcinoma.
5. Primary hematological disorders.
6. Active gastrointestinal bleeding on admission.
7. Previously known gastrointestinal bleeding.
8. Unstable medical condition.

Study Proforma

Laboratory testing, ultrasonography, and fiberoptic upper gastrointestinal endoscopy were done in every recruited patient.

Laboratory Testing

Hematological and biochemical workup included measurement of hemoglobin, total leukocyte count, platelet count, prothrombin time, and serum concentrations of bilirubin (total and conjugated), serum albumin, alanine aminotransferase, and aspartate aminotransferase. For each patient, a modified Child-Pugh score was calculated.45 All patients were tested for HBsAg and antibodies to hepatitis C virus to determine the cause of liver cirrhosis. Tests for other causes of cirrhosis (serum ceruloplasmin and slit lamp examination for Wilson's disease, tests for autoantibodies for autoimmune liver disease, and iron studies for hemochromatosis) were carried out only if there was a suggestive clinical clue.

Ultrasonography

(a) Measurement of liver size: Liver size is measured using sagittal approach in the midclavicular line. It is measured from diaphragm to the inferior border on b-mode image.

(b) Measurement of splenic size: Spleen size was measured by placing the patient in supine position, using 2–5 MHz curvilinear transducer in the coronal plane of section, posteriorly in one of the lower left intercostal spaces. The patient was examined in various degrees of inspiration to maximize the window to the spleen. The spleen parenchyma is extremely homogenous and it has uniform mid to low echogenicity. When the spleen enlarges, it can be more echogenic. A maximum cephalocaudal measurement exceeding 13 cm indicates enlargement with high degree of reliability.

(c) Measurement of portal vein diameter: The portal venous supply to the left lobe of liver can be visualized using an oblique, cranially angled subxiphoid view (recurrent subcostal oblique projection). The main and right portal veins are best seen in the sagittal or oblique sagittal plane. It is measured in supine position, during quiet respiration where the portal vein crosses anterior to the IVC43.

(d) Presence of collaterals: Five major sites of portosystemic venous collaterals are

1. Gastroesophageal junction between coronary and short gastric veins and systemic esophageal veins.
2. Paraumbilical vein-connects left portal vein to the systemic epigastric veins near umbilicus.
3. Splenorenal and gastrorenal.

4. Intestinal – regions in which GIT becomes retroperitoneal collaterals form (e.g.: ascending, descending colon, duodenum, and liver).

Duplex Doppler provides additional information. Increase of <20% in the diameter of the portal vein with deep inspiration indicates portal hypertension with 81% sensitivity and 100% specificity.

Ultrasonography is the preferred initial investigation because of its low cost and high accuracy [Table 1].

Endoscopy

Endoscopy is important to assess semi-quantitatively the number, appearance and size of any esophageal varices

I. Esophageal varices
- Grade I: Small varices without luminal prolapsed [Table 2].
- Grade II: Moderate-sized varices with luminal prolapsed with minimal obscuring of gastroesophageal junction.
- Grade III: Large varices showing luminal prolapsed substantially obscuring of gastroesophageal junction [Table3].
- Grade IV: Very large varies completely obscuring GE junction
- Grade 1 and 2 are considered as small varices and Grade 3 and 4 as large varices 47.

II. Gastric varices [Table 4]
These are classified as continuation of esophageal varices along the lesser curve of the stomach (GOV-1) or in the fundus (GOV-2); more rarely "Isolated gastric varices" may be found in the fundus (IGV-1) or in the rest of stomach (IGV-2) [Table 5]. The prevalence of gastric varices in portal hypertension is about 20%. They cause 5–10% of all episodes of upper gastrointestinal bleeding in portal hypertension.

III. Portal hypertensive gastropathy (PHG)
2 types of gastric mucosal changes are seen in portal hypertensive gastropathy [Table 6]. Mosaic pattern of gastric mucosa indicates mild PHG and cherry red spots in gastric mucosa reflect severe PHG.

Statistical Analysis

This is an observational study where 100 patients were included, of which 50 are cases (with esophageal varices) and 50 are controls (without esophageal varices). The cases were again divided into large and small varices based on endoscopic findings. Detailed history taking and clinical examination were done.

Descriptive statistics of normally distributed variables is reported as mean and SD and that of non-normally distributed variables were subjected to Mann–Whitney test and median with range was calculated and $P < 0.05$ is taken as significant. All variables which were found to be significant on univariate analyses were included as candidate variables for logistic regression analysis to identify independent predictors for the presence of esophageal varices and their size.

RESULTS

Age Distribution

Median age with range among 50 cases was 43.18 (range 24–86), and among 50 controls was 42.16 (range 22–68),

Table 1: Age distribution among cases and controls

Age	Cases	Controls	Large varices	Small varices
20–30	6	10	3	3
31–40	24	15	12	12
41–50	12	14	6	6
51–60	4	7	3	1
61–70	3	4	1	2
>70	1	0	1	0
Total	50	50	26	24

Table 2: Sex distribution among cases and controls

	Females		Males		Total	
	Number	%	Number	%	Number	%
Cases	11	22	39	78	50	100
Controls	8	16	42	84	50	100
Large varices	6	23	20	77	26	52
Small varices	5	21	19	79	24	48

Table 3: Distribution of various etiologies among cases and controls

Etiology	Patients
Alcoholic liver disease	62
Hepatitis B	10
Hepatitis C	2
Non-alcoholic fatty liver	6
Wilsons disease	1
Cause remained unknown	19

Table 4: Distribution of cases and controls according Child-Pugh score

Column 1	Class A	Class B	Class C	Total
Cases	4	18	28	50
Controls	7	23	20	50
Large	3	10	13	26
Small	1	8	15	24
Total	15	59	76	

Table 5: Sensitivity, specificity, positive, and negative predictive values for significant parameters for presence of varices

Parameters	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Portal vein diameter (>13.05 mm)	65%	54%	55.58%	45.26%
Spleen Diameter(>15.4cm)	78.80%	64%	56.06%	44.63%
Platelet Count(<102000)	82.69%	58%	59.68%	41.03%
Platelet count/splenic diameter(<815)	80.77%	64%	56.67%	44.01%

Table 6: Sensitivity, specificity, positive, and negative predictive values for the significant parameters for the presence of large varices

Parameters	Positive predictive value	Negative predictive value
Portal vein diameter(>14.m)	60.43%	40.32%
Spleen diameter(>16.25cm)	52.84%	47.85%
Platelet count(<93500)	54.29%	46.42%
Platelet count/spleen diameter(<548)	58.36%	42.28%

for large varices it is 44.46 (range 29–86) and for small varices is 41.79 (range 24–65).

Sex Distribution

Of 100 patients, 81 were male and 19 were female. Among cases 39 were male and 11 were female (no. of males/females in large varices is 20/6 and small varices is 19/5) and in controls 42 were male and 8 were female.

Distribution of Patients Based on Etiology

Alcoholic liver disease is the most common etiology in this study corresponding to 62% of cases followed by hepatitis B with 10%.

Relationship of cases and controls based on Child-Pugh score:

Child-Pugh score was calculated for all the patients with most of the patients with varices fall in Group C and without varices in Group B

Platelet count shows highest sensitivity for the detection of esophageal varices with 82.69% followed by platelet count/splenic diameter of 80.77%. Specificity is highest for splenic diameter and platelet count/splenic diameter.

Platelet count/splenic diameter shows highest sensitivity of 88% and specificity is highest for splenic diameter with 69.23% for detection of large varices.

DISCUSSION

Cirrhosis is the most advanced form of liver disease and variceal hemorrhage is one of its lethal complications. Over half of the patients with cirrhosis will develop varices. The risk of bleeding once OV formed is 20–35% within 2 years.

The reported mortality rate from first episode of variceal bleeding is 17–57%. Of those who survive the initial episode of bleeding and who do not receive active treatment, the risk of recurrent bleeding is approximately 66% and usually occurs within 6 months of the initial bleeding episode.

Since cirrhotic patients with large esophageal varices are at a high risk for bleeding, preventive efforts have concentrated on identifying cirrhotic patients with large varices.

In 1997, The American College of Gastroenterology (ACG) recommended screening endoscopy for cases with established cirrhosis who were candidates for medical therapy.

Also, in 1998, The American Association for the study of liver disease (AASLD) recommended screening endoscopy for varices and to be in particular routine in child Class B and C patients, but in child class A to be limited to patients with evidence of portal hypertension (thrombocytopenia or large portal vein/collaterals on abdominal imaging).

Prophylactic therapy initiated when large varices were discovered on screening endoscopy, had shown a decrease in the incidence of bleeding and an effect on bleeding – related mortality.

It was estimated that 100 screening endoscopies need to be performed to prevent 1–2 cases of variceal bleeding.

Therefore, identification of clinical features that can accurately predict esophageal varices and help identifying patients at greatest risk is important to improve the yield and cost-effectiveness of endoscopic screening.

Bleeding occurs in significant proportion of patients with severe PHG which accounts for most nonvariceal bleeding episodes in patients with cirrhosis and portal hypertension. PHG bleeding is a serious complication, which is usually chronic and insidious but occasionally massive and life-threatening. Overt hemorrhage from the gastric mucosa occurred in 60% of patients with severe PHG with a cumulative risk of bleeding of 75% over a 5-year follow-up period.

Several studies in the past have shown independent parameters such as splenomegaly, ascites, Spider nevi, Child's grade, platelet count, prothrombin time/activity, portal vein diameter, platelet count/ spleen diameter ratio, serum albumin, and serum bilirubin as significant predictors for the presence of esophageal varices. Our study found that 50% of the cirrhotic patients had EV diagnosed by endoscopy. This result is similar to the range of 24–80% showed in literature and reminds us that a significant part of cirrhotic patients are unnecessarily submitted to this procedure.

Relationship of esophageal varices with clinical and laboratory parameters:

a) Ascites and hepatic encephalopathy: In a study done by Fook-Hong NG *et al.* showed that low platelet count and presence of ascites were the significant independent predictors for high-grade EGV.

In present study, ascites and hepatic encephalopathy were not significantly associated with the presence of varices. Similar results were obtained by Cherian *et al.* in predicting esophageal varices.

a) Serum albumin, total bilirubin, prothrombin activity:

In a study done by D'Amico *et al.* showed that a serum albumin concentration of < 3.3 g/dL was predictors of esophageal varices.

In a cross-sectional study done by Schepis *et al.* has shown that prothrombin activity of 70% was used as an independent predictor of esophageal varices with an odds of 9.85. In our study, we did not get significance for serum albumin and prothrombin activity in prediction of esophageal varices. Similar results were obtained by Cherian *et al.*, where no significance was obtained for the above parameters.

No studies in the past have shown that total bilirubin as a predictor of esophageal varices. The present study also did not show any statistical significance for the prediction of esophageal varices based on total bilirubin levels.

4. Child-Pugh score:

The Child-Pugh score consists of two clinical features and three laboratory parameters and is used to assess the prognosis of chronic liver disease.

The Child-Pugh score was originally developed in 1973 to predict surgical outcomes in patients presenting with bleeding esophageal varices.

In our study, Child-Pugh score was not significantly associated with presence of esophageal varices but most of the cases belong to class C and controls (no esophageal varices) belong to Class B.

The study done by Jijo *et al.* shows significance and has a highest sensitivity of 95% for Child-Pugh Class B and C in predicting esophageal varices and postulated an algorithm where patients with Child-Pugh Class B and C were given primary prophylaxis and for Class A, they have seen platelet count and spleen diameter and then initiated prophylaxis accordingly.

5. Platelet count

Pathogenesis of thrombocytopenia includes productive, consumptive, or distributional mechanisms. It is commonly believed to be due to pooling and destruction of platelets in the spleen which may be mediated by platelet-associated IgG. Reduced levels of thrombopoietin either due to impaired production or rapid degradation may also add to thrombocytopenia.

Thus, platelet count depends on multiple factors not just portal hypertension.[47] Garcia- Tsao *et al.* (180 patients), Pilette *et al.* (116 patients), and K. C. Thomopoulos *et al.* (184 patients) reported a low platelet count to be an independent risk factor for the presence of varices. Mohammad Khuram *et al.* (200 patients) found OV in 146 with 121 having thrombocytopenia (94.5%).

We report that platelet count of <10200/mm^3 is 82.67% sensitive and 58% specific predictor of OV with positive predictive value of 59.64% and negative predictive value of 41.02 % in predicting presence of varices and a platelet count of 93500/mm^3 is 75% sensitive, 65.37% specific with 54.28, and 46.43 positive and negative predictive values, respectively, in predicting large varices.

Similar results were obtained in a study done by Cherian *et al.* with platelet count of 90000/mm^3 with 59.3% sensitivity, 64.2% specificity, and 47.5 PPV and 74.2 is NPV.

Chalasani *et al.* (346 patients) found that a platelet count <88,000 was an independent risk factor for the presence of large varices.

In retrospective analysis of 143 patients with compensated cirrhosis, Schepis *et al.* reported OV in 63 patients (44%) with platelet count of <100,000 as predictor of OV.

Most of the studies in the past have shown platelet count as a significant individual predictor of esophageal varices. In the present study, platelet count has shown highest sensitivity of 82.69 in predicting presence of esophageal varices among all the parameters studied with odds of 6.65.

Relations of Esophageal Varices with Ultrasonographic Parameters

Upper GI endoscopy of the study population revealed that a total of 50 patients had developed gastroesophageal varices.

Ultrasonography showed that median portal vein diameter (PVD) of the patients with gastroesophageal varices (GEV) was 13.9 mm with range of 8–18 mm and without gastroesophageal varices (GEV-0) was 12.1 mm with range of 7.8–16 mm. This difference was statistically significant ($P < 0.0322$).

Radiologically, median spleen diameter of the patients with OV was 16 cm with range of 8–26 cm and spleen size in the no varices group was 13.8 cm with range of 9–19 cm, and the difference was highly significant ($P < 0.001$). Hence, it can be concluded that gastroesophageal varices developed in cirrhotic patients with portal vein diameter more than 13.9 mm and larger than 16 cm spleen size.

These observations were more or less similar to other studies. In the study by Prihatini *et al.*, portal vein diameter 11.5 mm and spleen size of 10.3 cm were predictive factors for esophageal varices in liver cirrhosis. Here, spleen size and portal vein diameter was smaller than our study.

Portal vein diameter and spleen size for development of gastroesophageal varices were also nearer to Cherian *et al.* study (portal vein 14 mm and spleen size 17 cm). Thomopoulos *et al.* showed that the majority of patients with gastroesophageal varices had spleen size more than 13.5 cm which was nearly similar to ours.

In our study, as the portal vein diameter and spleen size increased, gastroesophageal varices also transformed to higher grades. Median portal vein diameter and spleen size with range in higher grade varices were 14.7 mm (8.5–16.8 mm) and 17.2 cm (8–26 cm), respectively.

In a study by Schepis *et al.,* portal vein diameter 13 mm was associated with higher grade varices.

Sharma and Aggarwal had noted that a clinically palpable spleen was associated with high grade varices; however, they did not measure the splenic size radiologically.

The fact that different studies conclude different best cutoff values of the portal vein diameter may be explained at least in part by that the physique of Asian populations is smaller than that of European populations. It has been reported that the normal mean portal vein diameter in Chinese populations is ± 1.3 mm, while it is 11.0 ± 0.3 mm in French populations.

Our data showed that spleen diameter and portal vein diameter measured by ultrasonography were independent predictors for the presence of varices.

Contrary to what was suggested in previous reports, no correlation between splenomegaly and EV was found in other studies. These differences may be due to the variations among studies regarding the etiology and the stage of liver cirrhosis studied.

Moreover, splenomegaly is found more frequently in post hepatitis cirrhosis than in alcoholic cirrhosis.

Some of these patients with splenomegaly and dilated portal vein may not have EV. One of the possible explanations for this result could be the development of spontaneous intra-abdominal shunts that decrease the blood flow of varices while maintaining congestive splenomegaly and dilated portal vein.

Relationship of Esophageal Varices with Platelet Count and Splenic Diameter

The parameter connects thrombocytopenia to splenomegaly to introduce a variable that takes into consideration the decreased platelet count most likely attributed to hypersplenism caused by portal hypertension.

This method uses two easily obtained parameters, which are part of the routine in a cirrhotic patient and, thus, would not increase costs.

The platelet count to spleen diameter ratio, proposed by Giannini *et al.* in 2003, reported that the platelet count/spleen diameter ratio to be the only independent variable associated with presence of OV on multivariate analysis and identified a cutoff value of 909, giving a PPV of 96% and NPV of 100%. Moreover, it appears to be the best noninvasive predictor of EVs that have been developed so far.

Several studies have been performed in an attempt to validate this new parameter as a new noninvasive screening tool for EVs.

In the present study, on univariate analysis, a platelet count-spleen diameter ratio of 608 was significantly associated with the presence of esophageal varices and it was found significant even in multivariate analysis with odds of 10.92 (CI-4.07-29.26).

Similar results in univariate analysis were found in study done by Jijo *et al.* but did not found significance in multivariate analysis.

CONCLUSION

Ultrasonography of abdomen is a simple, convenient, and non-invasive method for assessing the severity of portal hypertension in patients and to predict the severity of esophagogastric varices indirectly.

Patients having
- Portal vein diameter >13.9 mm,
- Spleen size >16 cm and
- Platelet count of <98000/microL

Platelet count and spleen diameter ratio <608 were found to have varices which were indirect evidences of severity of portal hypertension. The above said parameters tend to predict varices when they occur in combination than they occur individually.

These predictors may be of help
- To the physicians practicing in rural areas where endoscopy facilities are not readily available, in helping them to initiate appropriate primary pharmacological prophylaxis in these patients.
- In an urban setting where the endoscopy workload is high, a noninvasive predictor, as in this study, can help one to initiate drug therapy while waiting for the endoscopy procedure.

SUMMARY

Chronic liver disease is a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis.[1]

Portal hypertension is the significant complicating feature of decompensated cirrhosis. Most cirrhotic patients develop esophageal varices, with a lifetime incidence as high as 90%, and the reported mortality from variceal bleeding ranges from 17% to 57%.[7-10] Of those who survive the initial episode of bleeding and who do not receive active treatment, the risk of recurrent bleeding is approximately 66% and usually occurs within 6 months of the initial bleeding episode.

As per existing guidelines in a case of cirrhosis of liver, we are screening with upper gastrointestinal endoscopy to look for any esophagogastric varices present or not and grade the severity of varices. And then we start the prophylactic measures like propranolol to prevent the first bleed.

To restrict upper GI endoscopy to those patients who have ultrasonographic or laboratory indicators able to predict the presence of esophageal varices would result in a better risk/benefit ratio for the endoscopic study.

In this study, we make an attempt to predict the esophageal varices based on ultrasonographic findings, platelet count and platelet count spleen diameter ratio, and its correlation with upper GI endoscopy.

The present study was carried out in the Department of Medicine, Mahatma Gandhi Memorial Hospital, Warangal, between February 2017 and October 2018.

A hundred patients included in the study, of which 50 are cases (with esophageal varices) and 50 are controls (without esophageal varices). The cases were again divided into 26 large varices and 24 small varices based on endoscopic findings. Laboratory testing, ultrasonography, and fiberoptic upper gastrointestinal endoscopy were done in every recruited patient.

We conclude that ultrasonography of abdomen is a simple, convenient, and non-invasive method for assessing the severity of portal hypertension along with laboratory testing in patients with chronic liver disease and to predict the severity of esophagogastric varices indirectly.

Patients having portal vein diameter >13.9 mm, spleen size >16 cm, and presence of collaterals on ultrasonography and platelet count of <98000/microL, platelet count and spleen diameter ratio < 608 were found to have varices which were indirect evidences of severity of portal hypertension. The above said parameters tend to predict varices when they occur in combination than they occur individually.

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How to cite this article: Sammaiah P. Study of non-invasive predictors of esophageal varices in chronic liver disease. Int J Sci Stud 2020;8(6):75-81.

Source of Support: Nil, **Conflicts of Interest:** None declared.