ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 02 | February - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

EVALUATION OF NON-INVASIVE MARKERS IN PATIENTS WITH ESOPHAGEAL VARICES IN CHRONIC LIVER DISEASE

General Medicine

Jainendra Jain Resident, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

G.N. Saxena* Professor, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India *Corresponding Author

Puneet Rijhwani Professor, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Deepak Gupta Professor, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Vaishali Sharai Resident, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Pushpendra Singh Chouhan Resident, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Manjeet Resident, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Naveen Yadav Resident, Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

ABSTRACT

Background and Aim: Non-invasive biomarkers can reflect systemic inflammation and severity of varices in CLD patients. It has been hypothesized that combination of various markers can be useful in monitoring varices in chronic liver disease(CLD) patients. This study was aimed to associate the platelet count / splenic diameter ratio in small and large esophageal varices in CLD patients and assess its relationship with clinical and laboratory parameters of CLD. **Methods:** A hospital based observational study done in 150 CLD patients who fitting in inclusion and exclusion criteria and attending the OPD/IPD of Mahatma Gandhi Hospital, Jaipur. The PC/SD ratio was based on serum platelet count and splenic diameter. This ratio was used to measure the severity of esophageal varices and disease activity. **Results:** CLD patient with large esophageal varices had a significantly low PC/SD ratio ($p<0.01$*) and serum albumin ($p<0.01$*), along with increase in APRI and MELD score. **Conclusion:** The results of this study found that some non-invasive markers could predict the severity of varices in CLD was significantly associated with. The present study demonstrated that PC/SD ratio decreased in patient with CLD and positively correlated with disease severity.

KEYWORDS

varices, PC/SD ratio, Portal hypertension, MELD score

INTRODUCTION

Chronic liver disease (CLD) is a progressive impairment of liver functioning for more than six months, including clotting factor synthesis, protein detoxification, & bile excretion. CLD leads to fibrosis & cirrhosis by inflammation, destruction, & regeneration of liver parenchyma.[1] The spectrum of etiologies is broad for chronic liver disease, which includes hepatotrophic viruses, chemicals, alcohol & drug abuse, autoimmune disorders, cholestasis, & metabolic diseases,[2,3] & is a major cause of morbidity & mortality in many countries.[4,5] CLD is a major cause of mortality & morbidity in many countries.[4,5]

Cirrhosis is a final stage of chronic liver disease that results in disruption of liver architecture, the formation of widespread nodules, vascular reorganization, neo-angiogenesis, & deposition of an extracellular matrix. The underlying mechanism of fibrosis & cirrhosis at a cellular level is the recruitment of stellate cells & fibroblasts, resulting in fibrosis, while parenchymal regeneration relies on hepatic stem cells. Liver fibrosis is caused by a continuous excessive deposition of the extracellular matrix (ECM) in response to chronic liver injury.[2,3] Cirrhosis result in development of portal hypertension. Hyperdynamic circulation, ascitis, hepatic encephalopathy, and esophagogastric varices are consequences of portal hypertension. 50% of cirrhotic ascites patients develop varices. LC has a bad prognosis and is cancer-prone.[7]

A hepatic venous pressure gradient (HVPG) measurement is the "gold standard" for the evaluation of the presence & severity of portal hypertension. Patients with cirrhosis may have either subclinical portal hypertension (PH) (HVPG is limited to 6–10 mmHg) or clinically significant PH (CSPH) (HVPG > 10 mmHg), which is further classified as severe (HVPG > 12 mmHg) & very severe PH (HVPG > 16 mmHg). In advanced cirrhosis, PH results in a profound hemodynamic derangement, which, in turn, leads to marked splanchnic vasodilation, & usually high HPVG > 16 mmHg.[8]

Considering that EV are one of the most common & potentially life-threatening complications of liver cirrhosis, there is a need for the identification of non-invasive markers for fast orientation in EV detection, & further risk & treatment assessment.[24] Taking into account the high cost of repeated upper endoscopies, especially in resource limited settings, simple non-invasive scores could potentially be of great interest in the daily clinical practice of developing countries. Several clinical, laboratory, and ultrasound factors have been examined as non-invasive alternatives to endoscopy. [25] [24,26-30] Other predictive scores include age–bilirubin–INR–creatinine (ABIC), Glasgow alcoholic hepatitis score (GAHS), and Lille model. [31] The ABIC score categorises AH patients into high-, moderate-, and low-risk groups based on 90-day and 1-year death risk. [32] The Lille score examines serum bilirubin following a 7-day regimen of corticosteroids and helps decide whether to discontinue or continue treatment. [33]

Child–Turcotte–Pugh (CTP) & MELD scores are routinely utilised for liver cirrhosis patients. Johnson et al.[34] developed the albumin-bilirubin (ALBI) score derived from two laboratory variables, bilirubin & albumin, without using factors evaluated subjectively (such as ascites & encephalopathy) or otherwise obtained.[34] In recent years, Roayaie et al.[35] proposed modifying the ALBI score by incorporating platelet count into ALBI in order to measure of liver function reserve.

The PALBI model has recently been used as a predictor of mortality in patients with cirrhosis related complications.[36] So, the present study was conducted to investigate series of non-invasive biochemical & radiological markers for prediction of esophagealvarices in patients with chronic liver disease.

MATERIALS AND METHODS

This hospital based observational study is conducted in all patients (outpatient & Inpatient) diagnosed as Chronic Liver Disease and evaluated under the Department of General Medicine, Mahatma Gandhi Hospital, Jaipur.

Inclusion criteria:

a. Diagnosed cases of Chronic Liver Disease with esophageal varices irrespective of etiology.
b. Age more than 18 year

Exclusion criteria:

a. Patients aged less than 18 years.
b. Patients with active gastro-intestinal bleed on admission.
c. Patients with Hepatocellular carcinoma detected by ultrasonography &/or elevated alpha-feto protein.
d. Patients with known primary coagulation disorders or recent history of anti-platelets drugs.
e. Patients with past history of porto-spleenic surgical interventions and unfit for endoscopic procedures.

RESULTS

Table 1: Distribution of study subjects according to small & large varices

Varices	N	%
Small (Group A)	90	60
Large (Group B)	60	40
Total	150	100

Table 1 shows the total 150 subjects included in study, 90 (60%) subjects had small varices & 60 (40%) had large varices

Table 2: Sign & symptoms among the study subjects

Variables	Small Varices (Group A)		Large Varices (Group B)		p value
	N=90	%	N=60	%	
Jaundice	51	56.67	31	51.67	0.72
Pedal edema	41	45.56	26	43.33	0.81
Palpable spleen	6	6.67	22	36.67	<0.01*
Ascites	43	47.78	44	73.33	<0.01*

*: statistically significant

There was a statistically significant difference present in no. of subjects with palpable spleen & with ascites, in Group A & Group B (p<0.01). In group B, subjects with palpable spleen were 36.67% & with ascites were 73.33%, whereas in group A, this number was 6.67% & 47.78%, respectively. (Table 2)

Table 3: CBC profile among the study subjects

Variables	Small Varices (Group A)		Large Varices (Group B)		p value
	Mean	SD	Mean	SD	
Hemoglobin (g/dl)	11.76	1.93	10.51	1.72	0.14
WBC (103/mm3)	4.81	1.45	4.47	2.03	0.11
Platelets (109/l)	121.32	51.19	87.08	41.84	0.006*

*: statistically significant

There was a statistically significant difference present in mean levels of Platelet in Group A & Group B, i.e., 121.32±51.19 & 87.08±41.84, respectively (p=0.006). (Table 3)

Table 4: Comparison of non-invasive parameters in study population

Variables	Small Varices (Group A)		Large Varices (Group B)		p value
	Mean	SD	Mean	SD	
Mean portal vein size	13.19	0.30	14.60	0.48	0.005*
Mean spleen diameter	107.63	14.44	13.81	1.41	<0.01*
PC/SD	1405.6	291.57	128.12	13.09	<0.01*

*: statistically significant

There was a statistically significant difference present in mean levels of non-invasive parameters, i.e., portal vein size, spleen diameter & PC/SD, among Group A & B. (Table 4)

Table 5: APRI & MELD score among the study subjects

Variables	Small Varices		Large Varices		p value
	Mean	SD	Mean	SD	
APRI	1.29	0.72	2.30	0.88	0.003*
MELD	11.3	3.21	15.8	4.39	0.002*

*: statistically significant

There was statistically significant difference present in APRI score (p=0.003) & MELD score (p=0.002), between Group A & B. (Table 5)

DISCUSSION

In this study, a total of 150 subjects, 111 were males and 39 were females. Hence there was male dominancy in the present study.

In this study, there was a statistically significant difference present in no. of subjects with palpable spleen & with ascites, in Group A & Group B (p<0.01).Splenomegaly is recognized as one of the diagnostic signsof cirrhosis & portal hypertension.In present study group B (large varices), subjects with palpable spleen were 36.67% & with clinically detectable ascites was present in 73.33%, whereas in group A, this number was 6.67% & 47.78%, respectively.

There was a statistically significant difference present in mean levels of non-invasive parameters, i.e., portal vein size, spleen diameter & PC/SD, among Group A & B. these findings were similar to results of Sarangapani A et al., (2010)[96], Kumar P et al., (2020)[108].

CONCLUSION

In light of the growing number of patients requiring care, it is urgent that a non-invasive method be developed for predicting the likelihood that a patient may develop varices. The bipolar diameter of the spleen is commonly measured in cirrhotic patients using ultrasound because it is readily available, repeatable, and noninvasive. Despite the study's limited sample size, the authors conclude that the platelet count/spleen diameter ratio is useful for distinguishing between minor and big esophageal varices. Endoscopy could be avoided in patients who do not have a high risk of missing esophageal varices if the platelet count/spleen diameter ratio was used instead. Since ultrasonography is already performed on cirrhotic patients on a yearly or biannual basis as part of the surveillance programme for hepatocellular carcinoma, adopting this method of using non-invasive diagnostics will inevitably reduce the cost of managing cirrhotic patients. Therefore, the current study shows that patients at risk of developing large-sized varices can be screened utilising clinical, haematological, biochemical, and radiological markers. Large esophageal varices are strongly associated with the presence of a clinically visible spleen, ascites, platelet count, APRI score, and MELD score. Furthermore, it indicates that indirect indications of portalhemodynamics such as portal vein size, spleen diameter, and PC/SD can be utilised effectively as a screening test without putting patients through esophagogastroduodenoscopy.

REFERENCES

1. Bataller R, Brenner DA. Liver fbrosis. J Clin Invest. American Society for Clinical Investigation 2005;115(2):209–18.
2. Friedman SL. Liver fbrosis – from bench to bedside. J. Hepatol. 2003;38:S38–53.
3. Jaeschke H. Cellular adhesion molecules: regulation and functional signifcance in the pathogenesis of liver diseases. Am J Physiol. 1997;273(3 Pt 1):G602–11.
4. Pinzani M, Marra F. Cytokine receptors and signaling in hepatic stellate cells. Semin Liver Dis. Copyright © 2001 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, USA. 2001;21(3):397–416.
5. Iwamoto T, Terai S, Hisanaga T, et al. Bone-marrow-derived cells cultured in serum-free medium reduce liver fbrosis and improve liver function in carbon-tetrachloride-treated cirrhotic mice. Cell Tissue Res. Springer-Verlag 2013;351(3):487–95