**Clinical significance of isolated gastric varices in liver cirrhotic patients: A single-referral-centre retrospective cohort study**

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**Key words**
bleeding, gastric varices, liver cirrhotic patients, mortality rate.

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**Abstract**

**Introduction:** Gastric varices (GVs) occur in 10–30% of liver cirrhotic patients, with a mortality rate of up to 45%. Rupture of isolated GVs (IGVs) is less prevalent but often results in more severe hemorrhage and a higher risk of mortality than rupture of esophageal varices (EVs). However, there is no clear consensus yet about the optimal management for incidentally discovered IGVs.

**Objective:** To determine the clinical significance of IGVs in liver cirrhotic patients.

**Methods:** This was a retrospective cohort endoscopy database study within a 2-year period (2016–2017). All study subjects were liver cirrhotic patients with OVs or GVs. The exclusion criteria were noncirrhotic portal hypertension, presence of malignancy, absence of varices, and incomplete data. Statistical analysis was performed using IBM SPSS 23.

**Results:** A total of 153 patients were included in this study. IGVs were found in 13 (8.49%) patients, whereas OVs were found in 112 (73.20%) patients and gastro- OVs were found in 28 (18.30%) patients. Child-Pugh class C (CP C) score was the strongest independent risk factor for variceal bleeding in bivariate analysis (hazard ratio [HR]: 10.21, 95% confidence interval [CI]: 4.15–25.12, \( P = 0.001 \)) and multivariate analysis (HR: 12.49, 95% CI: 4.95–31.54, \( P = 0.001 \)); however, the presence of IGVs was not an independent risk factor. CP C score was also the only significant risk factor associated with 1-year mortality in liver cirrhotic patients on multivariate analysis (HR: 26.77, 95% CI: 6.01–119.34, \( P = 0.001 \)).

**Conclusion:** The presence of IGVs has no clinical significance in the occurrence of 1-year rebleeding and in patient survival.

**Introduction**

Variceal bleeding is the leading cause of death in cirrhotic patients, with a mortality rate of up to 20%. The prevalence of gastric varix (GV) bleeding has been documented to be around 10–30% of variceal bleeding cases. The Sarin classification is the most frequently used classification system for GVs, based on which further clinical management is determined. Gastroesophageal varix type 1 (GOV1) is an esophageal varix (OV) that extends into the lesser curvature, whereas GOV2 extends from the esophagus into the fundal area. Isolated GV type 1 (IGV1) is confined in the fundus, whereas IGV2 is located elsewhere in the stomach. GV bleeding is still considered a challenging situation when treating liver cirrhotic patients.

Portal pressure has been considered as the best parameter for predicting the development of varices and OV bleeding; however, because GVs tend to have lower portal pressure than OVs, it is difficult to predict the bleeding occurrence and to ensure the efficacy of beta-blocker treatment in clinical practice. The major risk factors for GV bleeding are the location of the varices (IGVs are the most predominant), a large varix size, the presence of red spots, and severe liver disease. IGV bleeding is still considered to be less frequent than OV bleeding; however, it is more severe with a higher mortality rate. With respect to the type of varices, but the management of GV bleeding is far less studied than that of OV bleeding. Therefore, the current study aimed to determine the clinical significance of IGVs in liver cirrhotic patients.

**Methods**

This was a retrospective cohort endoscopy database study performed at Dr. Cipto Mangunkusumo National General Hospital.
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a government-owned national referral hospital, from January 2016 to December 2017.

All subjects of this study were liver cirrhotic patients with OVs or GVs found during an esophagogastroduodenoscopy (OGD) procedure. All cases were index cases of OGD. The diagnosis of cirrhosis was reported by the physician in the medical records and was made using standard diagnostic methods such as ultrasonography examination combined with liver stiffness measurement (using Fibroscan) or other imaging examinations, such as abdominal computed tomography or magnetic resonance imaging. The exclusion criteria were noncirrhotic portal hypertension, presence of malignancy (hepatocellular carcinoma and extrahepatic malignancy), and incomplete data. Patients were retrospectively followed up for 1 year for variceal bleeding and mortality risk. One year was set for the monitoring of variceal bleeding owing to the progression rate and mortality risk.5,6

The patients’ characteristics, including gender, age, etiology of liver disease, Model for End-stage Liver Disease (MELD) score, and Child-Pugh (CP) score, were collected for analysis. The patients were divided into three groups based on the presence of varices: OV, IGV, and the GOV groups.

Table 1  Baseline characteristics of patients

| Esophageal varices (EV) (n = 112) | Isolated Gastric varices (IGV) (n = 13) | Gastroesophageal varices (GOV) (n = 28) |
|----------------------------------|--------------------------------------|--------------------------------------|
| Male, n (%)                      | 83 (74.1%)                           | 6 (46.2%)                           | 22 (78.6%)                           |
| Age, mean ± SD                   | 51 ± 11.02 years                     | 56 ± 9.48 years                     | 52 ± 10.33 years                     |
| Etiology, n (%)                  |                                       |                                      |                                      |
| HBV                              | 56 (50%)                             | 6 (46.2%)                           | 11 (39.3%)                           |
| HCV                              | 34 (30.4%)                           | 3 (23.1%)                           | 4 (14.3%)                            |
| NBNC                             | 22 (19.6%)                           | 4 (30.8%)                           | 13 (46.4%)                           |
| Albumin (g/dL), mean ± SD        | 3.41 ± 0.95                          | 3.17 ± 0.40                         | 3.26 ± 0.54                          |
| Bilirubin (mg/dL), median (min-max) | 1.35 (0.16–24.27)                 | 1.26 (0.47–7.50)                    | 1.44 (0.35–4.77)                     |
| INR, mean ± SD                   | 1.20 ± 0.27                          | 1.19 ± 0.13                         | 1.17 ± 0.51                          |
| Creatinine (mg/dL), mean ± SD    | 1.10 ± 0.79                          | 0.76 ± 0.14                         | 0.97 ± 0.46                          |
| Natrium (mEq/L), mean ± SD       | 137 ± 5.32                           | 139 ± 5.13                          | 137 ± 4.14                           |
| Hemoglobin (g/dL), mean ± SD     | 11.11 ± 2.44                         | 10.64 ± 2.01                        | 9.85 ± 1.86                          |
| Platelet (/uL), median (min-max) | 101 (23–389)                         | 83 (50–133)                         | 93 (32–361)                          |
| Child-Pugh score, mean ± SD      | 7.32 ± 2.42                          | 6.84 ± 1.81                         | 7.32 ± 1.83                          |
| Child-Pugh score category, n (%) |                                       |                                      |                                      |
| A                                | 57 (50.9%)                           | 7 (53.8%)                           | 10 (35.7%)                           |
| B                                | 33 (29.5%)                           | 5 (38.5%)                           | 15 (53.6%)                           |
| C                                | 22 (19.6%)                           | 1 (7.7%)                            | 3 (10.7%)                            |
| MELD score, mean ± SD            | 9.85 ± 4.35                          | 9.54 ± 2.85                         | 10.39 ± 3.90                         |
| MELD category, n (%)             |                                       |                                      |                                      |
| <14                              | 97 (86.6%)                           | 12 (92.3%)                          | 23 (82.1%)                           |
| ≥14                              | 15 (13.4%)                           | 1 (7.7%)                            | 5 (17.9%)                            |
| Type of varices, n (%)           |                                       |                                      |                                      |
| EV                               | 112 (100%)                           |                                    |                                      |
| IGV                              | 13 (100%)                            |                                    |                                      |
| GOV                              | 0 (0%)                               |                                    |                                      |
| Grade of varices, n (%)          |                                       |                                      |                                      |
| Small (grade 1–2)                | 62 (55.4%)                           | 7 (53.8%)                           | 9 (32.1%)                            |
| Large (grade 3–4)                | 50 (44.6%)                           | 6 (46.2%)                           | 19 (67.9%)                           |
| Presence of red spot, n (%)      | 11 (9.8%)                            | 1 (7.7%)                            | 6 (21.4%)                            |

Results

The characteristics of the study participants are shown in Table 1. Of 328 cirrhotic patients who underwent OGD, only 153 patients were eligible to be included in the final analysis based on the exclusion criteria. The flowchart of sample recruitment is shown in Figure 1. The patients had a male
predominance and a mean age of 51 years. The most frequent etiology of liver disease was hepatitis B virus infection. CP A score was found in 74 (48%) patients, CP B score in 53 (35%) patients, and CP C score in 26 (17%) patients. There were 41 patients (26.8%) with GVs and 112 patients with OVs (73.2%). IGV1s were found in 13 (8.5%) patients, whereas GOVs were found in 28 (18.3%) patients. There were no patients with IGV2. During the follow up, about 92.1% (139/151) of the patients were documented to be using propranolol. OV band ligation was performed in 78.6% (88/112) of the patients with OVs and 92.9% (26/28) of the patients with GOVs. All patients with IGVs (13/13, 100%) and 71.4% (20/28) of the patients with GOVs were treated with cyanoacrylate injection.

**Risk factor for 1-year variceal bleeding in cirrhotic patients.** The risk factors for 1-year variceal bleeding are shown in Table 2. In bivariate analysis, the significant factors were CP B score, CP C score, GOVs, presence of red spots, and a history of repeated variceal bleeding. In multivariate analysis, only CP C score and GOVs were significant risk factors (Table 3). The CP C score was the strongest independent risk factor for 1-year variceal bleeding (HR: 12.49, 95% CI: 4.15–25.12).

**Table 2** Bivariate analysis of risk factors associated with variceal bleeding

| Variables                        | No       | Yes       | HR (95% CI)   | P value |
|----------------------------------|----------|-----------|---------------|---------|
| Gender, n (%)                    |          |           |               |         |
| Female                           | 32 (76.2%) | 10 (23.8%) | 1             | —       |
| Male                             | 82 (73.9%) | 29 (26.1%) | 1.06 (0.74–1.53) | 0.743   |
| Age, n (%)                       |          |           |               |         |
| ≤60 years                        | 84 (71.2%) | 34 (28.8%) | 1             | —       |
| >60 years                        | 30 (85.7%) | 5 (14.3%)  | 0.45 (0.177–1.16) | 0.089   |
| Child-Pugh Score, n (%)          |          |           |               |         |
| A                                | 66 (89.2%) | 8 (10.8%)  | 1             | —       |
| B                                | 35 (66%)  | 18 (34%)  | 4.48 (1.94–10.33) | 0.001   |
| C                                | 13 (50%)  | 13 (50%)  | 10.21 (4.15–25.12) | 0.001   |
| MELD Score, n (%)                |          |           |               |         |
| <14                              | 104 (78.8%) | 28 (21.2%) | 1             | —       |
| ≥14                              | 10 (47.6%) | 11 (52.4%) | 3.76 (1.86–7.60) | 0.001   |
| Type of varices, n (%)           |          |           |               |         |
| EV                               | 90 (80.4%) | 22 (19.6%) | 1             | —       |
| IGV                              | 9 (69.2%)  | 4 (30.8%)  | 1.49 (0.51–4.31) | 0.467   |
| GOV                              | 15 (53.6%) | 13 (46.4%) | 2.51 (1.26–4.99) | 0.009   |
| Grade of varices, n (%)          |          |           |               |         |
| Small                            | 65 (83.3%) | 13 (16.7%) | 1             | —       |
| Large                            | 49 (65.3%) | 23 (34.7%) | 2.20 (1.14–4.30) | 0.020   |
| Red spot, n (%)                  |          |           |               |         |
| Negative                         | 106 (78.5%) | 29 (21.5%) | 1             | —       |
| Positive                         | 8 (44.4%)  | 10 (55.6%) | 3.29 (1.59–6.81) | 0.001   |
| Portal hypertensive gastropathy, n (%) |          |           |               |         |
| Negative                         | 15 (75%)  | 5 (25%)  | 1             | —       |
| Positive                         | 99 (74.4%) | 34 (25.6%) | 1.16 (0.45–2.96) | 0.764   |
| Bleeding, n (%)                  |          |           |               |         |
| Never                            | 46 (100%) | 0 (0%)    | 1             | —       |
| First bleeding                   | 23 (82.1%) | 5 (17.9%)  | 1.49 (0.51–4.31) | 0.467   |
| Reoccurrence of bleeding         | 45 (57%)  | 34 (43%)  | 2.51 (1.26–4.99) | 0.009   |

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Population study (N= 153 patients)
confidence interval [CI]: 4.95–31.54, \( P \leq 0.001 \)). The presence of GOVs was also shown to be an independent risk factor associated with the bleeding incidence after 1 year in multivariate analysis (HR: 2.95, 95% CI: 1.40–6.19, \( P \leq 0.004 \)) (Table 3). With respect to the factors associated with variceal bleeding, the Kaplan–Meier plot shows that the CP score and the type of varices significantly contributed to the bleeding incidence, with a \( P \) value in log-rank test of 0.001 and 0.022, respectively. After 1 year of follow up, the probability (risk) of bleeding was 75.1% in patients with CP C score and 49.7% in patients with GOVs (Figs 2 and 3).

**Risk factor for 1-year mortality in cirrhotic patients.** There are various risk factors associated with the 1-year mortality rate of liver cirrhotic patients (Table 4). The bivariate analysis showed that CP B score, CP C score, and MELD score \( \geq 14 \) were significant factors. However, the multivariate analysis showed that the CP C score was the only significant risk factor associated with the 1-year mortality rate of liver cirrhotic patients (HR: 26.77, 95% CI: 6.01–119.34, \( P \leq 0.001 \)). Among the factors associated with mortality, the Kaplan–Meier plot showed that only CP score significantly contributed to bleeding, with a \( P \) value of 0.001 in the log-rank test. After 1 year of follow up, the probability (risk) of mortality in patients with CP C score was 61.2% (Fig. 4).

**Discussion**

To our knowledge, there is no clear recommendation yet about the management of asymptomatic or silent GVs (especially IGVs). Furthermore, there is also no recommended algorithm for predicting the long-term clinical outcome of patients because the prevalence of GVs is much lower than that of OVs. However, GOVs or IGVs have been known to have a higher bleeding tendency with a higher mortality than OVs in liver cirrhotic patients. In our cohort study, the presence of GVs was found in 26% of the patients. This result is consistent with those of previous studies reporting that the prevalence of GVs in subjects with portal hypertension varies between 15 and 55%.2,3,7,8

| Variables       | HR (95% CI)      | \( P \) value |
|-----------------|------------------|--------------|
| Child-Pugh C score | 12.49 (4.95–31.54) | 0.001        |
| GOV             | 2.95 (1.40–6.19)  | 0.004        |
On the basis of pathogenesis, GOVs and IGVs tend to have more blood supply than OVs owing to the complexity of potential vascular anastomosis involving the left gastric vein, azygos vein, and inferior phrenic vein. Therefore, GV rupture can cause massive bleeding and might lead to a high mortality rate because of difficulty in controlling bleeding.9 The other main issue in clinical practice with respect to GVs is that they do not correlate well with the measurement of hepatic vein pressure gradient (HVPG), which is usually considered as the gold-standard method for confirming and monitoring clinically significant portal hypertension during treatment. In the presence of variceal bleeding, it has been shown that IGVs have significantly lower portal pressure than OVs.10 More interestingly, contrasting findings have been found in this study as GV (especially GOVs) bleeding in the 1-year period was more frequent than OV bleeding (46.4% vs 19.6%). In our database, GOVs were more frequently found in CP B score patients, whereas OVs were more frequent in CP A score patients. IGVs were also predominant in patients with CP A score. Therefore, the severity of the liver condition might be the reason why GOVs tended to have more bleeding in our cohort. Among our patients with IGVs, those with CP A score were predominant in number, followed by those with CP B score and only one patient with CP C score. The complex natural history, development, and lower bleeding incidence of GVs have somewhat become forgotten issues; however, the variability among many research findings serves as a reminder of the importance of performing routine follow up. Noninvasive treatments, such as use of nonselective beta-blockers, might lower the HVPG; however, they do not reduce the GV’s bleeding and mortality rates.11

Another major drawback is that studies have shown an increased risk of variceal bleeding associated with an increased variceal size, the presence of red spots, and a high CP score classification.12 Sarin et al. reported that GVs are five times more likely to occur in patients with bleeding and that patients with IGVs have a significantly higher bleeding risk than those with GOVs.2 Our study showed different results, in that we observed that IGVs do not increase the 1-year bleeding risk, but the bleeding risk increases with the presence of GOVs and in patients with CP C score. The different results in portal hypertension might be explained by the fact that many non-cirrhotic portal hypertension cases were included by Sarin et al. In our retrospective cohort database, all patients were cirrhotic. Another important finding in this study is that the presence of red spots was not a risk factor for 1-year variceal bleeding.

Figure 3 Varices bleeding within 1-year according to type of varices.
Screening for the presence of varices has been recommended for all liver cirrhotic patients; however, the invasive nature of the endoscopy procedure and HVPG measurements have made routine follow-up more difficult. Noninvasive tool such as transient elastography (Fibroscan) has not been approved yet for use in routine variceal screening and management. Practical novel procedures that can be performed in one-stop visit, such as endoscopic ultrasound, have expanded the field of hepatology by enabling assessment of varices and their collaterals, liver biopsy, direct measurement of portal pressure, and sclerotherapy even in the acute bleeding setting.

The CP score classification is a well-known scoring system for assessing liver disease severity, in which a higher score is associated with a greater mortality rate. Varices are known to be significantly more prevalent in CP C score than in CP A score. CP C score was found to be the strongest risk factor for increased bleeding and 1-year mortality in the presence of varices. This is supported by a previous study in which CP C score was found to be a risk factor for the presence of varices, progression to large varices, and variceal bleeding.

The current study had some limitations. First, this was a retrospective study. Nevertheless, this study provided insights from our practice that might help improve the management of patients in the future. Second, only 153 patients were eligible for the study analysis after the application of the exclusion criteria. However, only 11 patients had incomplete data. Therefore, from our statistical analysis, the low proportion of missing data (only 11 of 164 patients or only 6.7% of the sample) means less estimation bias. Third, the IGVs group had a small sample size; however, this might be unavoidable because the prevalence of IGVs is lower than that of OVs and GOVs. Fourth, the images were reviewed again through the computer database and the written report. However, all endoscopic procedures were performed by a senior and experienced consultant. Larger prospective studies are needed to develop specific recommendations for nonbleeding IGVs.

Conclusion
IGVs have no clinical significance in cirrhotic patients. The presence of GOVs seemed to be an important factor for the occurrence of 1-year bleeding, but not for survival, in liver cirrhotic patients. The most important risk factor for 1-year bleeding and patient’s survival was the CP C score.

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Table 4: Bivariate analysis of risk factors associated with mortality after 1 year in cirrhotic patients

| Variables | Survivor | Non survivor | HR (95% CI) | P value |
|-----------|----------|--------------|-------------|---------|
| Gender, n (%) | | | | |
| Female | 37 (88.1%) | 5 (11.9%) | 1 | — |
| Male | 93 (63.8%) | 18 (16.2%) | 1.37 (0.51–3.68) | 0.538 |
| Age, n (%) | | | | |
| ≤60 years | 102 (86.4%) | 16 (13.6%) | 1 | — |
| >60 years | 28 (80%) | 7 (20%) | 1.46 (0.60–3.55) | 0.403 |
| Child-Pugh Score, n (%) | | | | |
| A | 72 (97.3%) | 2 (2.7%) | 1 | — |
| B | 45 (84.9%) | 8 (15.1%) | 6.36 (1.35–29.97) | 0.019 |
| C | 13 (50%) | 13 (50%) | 26.77 (6.01–119.34) | 0.001 |
| MELD Score, n (%) | | | | |
| <14 | 117 (88.6%) | 15 (11.4%) | 1 | — |
| ≥14 | 13 (61.9%) | 8 (38.1%) | 4.12 (1.74–9.74) | 0.001 |
| Type of varices, n (%) | | | | |
| EV | 90 (80.4%) | 22 (19.6%) | 1 | — |
| IGV | 12 (92.3%) | 1 (7.7%) | 0.36 (0.48–2.67) | 0.317 |
| GOV | 28 (100%) | 0 (0%) | — | — |
| Grade of varices, n (%) | | | | |
| Small | 55 (82.1%) | 12 (17.9%) | 1 | — |
| Large | 58 (85.3%) | 10 (14.7%) | 1.23 (0.54–2.80) | 0.628 |
| Red spot, n (%) | | | | |
| Negative | 116 (85.9%) | 19 (14.1%) | 1 | — |
| Positive | 14 (77.6%) | 4 (22.2%) | 1.59 (0.54–4.67) | 0.401 |
| Portal hypertensive gastropathy, n (%) | | | | |
| Negative | 17 (85%) | 3 (15%) | 1 | — |
| Positive | 113 (85%) | 20 (15%) | 0.91 (0.27–3.06) | 0.877 |
| Bleeding in 1 year, n (%) | | | | |
| No | 97 (85.1%) | 17 (14.9%) | 1 | — |
| Yes | 33 (84.6%) | 6 (15.4%) | 1.05 (0.42–2.67) | 0.913 |
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References

1. Mallet M, Rudler M, Thabut D. Variceal bleeding in cirrhotic patients. *Gastroenterol. Rep.* 2017; 5: 185–92.
2. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: A long-term follow-up study in 568 portal hypertension patients. *Hepatology.* 1992; 16: 1343–9.
3. Wani ZA, Bhat RA, Choudhury A. Gastric varices: classification, endoscopic and ultrasonographic management. *J. Res. Med. Sci.* 2015; 20: 1200–7.
4. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J. Portal hypertensive bleeding in cirrhosis: risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology.* 2017; 65: 310–35.
5. Lo GH, Lai KH, Cheng JS, Chen MH, Chiang HT. A prospective, randomized trial of butyl cyanocrylate injection versus band ligation in the management of bleeding gastric varices. *Hepatology.* 2001; 33: 1060–4.
6. Haq I, Tripathi D. Recent advances in the management of variceal bleeding. *Gastroenterol. Rep. (Oxf).* 2017; 5: 113–26. https://doi.org/10.1093/gastro/gox007.
7. Watanabe K, Kimura K, Matsutani S, Ohto M, Okuda K. Portal hemodynamics in patients with gastric varices. A study in 230 patients with esophageal and/or gastric varices using portal vein catheterization. *Gastroenterology.* 1988; 95: 433–40.
8. Mudawi H, Ali Y, Tahir ME. Prevalence of gastric varices and portal hypertensive gastropathy in patients with Symmers’ periportal fibrosis. *Ann. Saudi Med.* 2008; 28: 42–4.
9. Komori K, Kubokawa M, Ihara E et al. Prognostic factors associated with mortality in patients with gastric fundal variceal bleeding. *World J. Gastroenterol.* 2017; 23: 496–504.
10. Morrison J, Mendoza-Elias N, Lipnik A et al. Gastric varices bleed at lower portosystemic pressure gradients than esophageal varices. *J. Vasc. Interv. Radiol.* 2018; 29: 636–41.
11. Mishra SR, Sharma BC, Kumar A, Sarin SK. Primary prophylaxis of gastric variceal bleeding comparing cyanoacrylate injection and beta-blockers: a randomized controlled trial. *J. Hepatol.* 2010; 54: 1161–7.
12. Pagliaro L, D’Amico G, Sörensen T et al. Prevention of first bleeding in cirrhosis. A meta-analysis of randomized trials of nonsurgical treatment. *Ann. Intern. Med.* 1992; 117: 59–70.
13. Pu K, Shi J, Wang X et al. Diagnostic accuracy of transient elastography (FibroScan) in detection of esophageal varices in patients with cirrhosis: a meta-analysis. *World J. Gastroenterol.* 2017; 23: 345–56.
14. Paternostro R, Reiberger T, Bucsics T. Elastography-based screening for esophageal varices in patients with advanced chronic liver disease. *World J. Gastroenterol.* 2019; 25: 308–29.
15. Adler D. New common ground: EUS-guided portal pressure measurements as a bridge between endoscopy and hepatology. *Gastrointest. Endosc.* 2017; 85: 1002–4.
16. Huang J, Samarasena J, Tsujino T et al. EUS-guided portal pressure gradient measurement with a simple novel device—a human pilot study. *Gastrointest. Endosc.* 2017; 85: 996–1001.
17. Durand F, Valla D. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. *J. Hepatol.* 2005; 42: 100–7.
18. Zaman A, Becker T, Lapidus J. Risk factors for the presence of varices in cirrhotic patients without a history of variceal hemorrhage. *Arch. Intern. Med.* 2001; 161: 2564–70.
19 Merli M, Nicolini G, Angeloni S et al. Incidence and natural history of small esophageal varices in cirrhotic patients. *J. Hepatol.* 2003; 38: 266–72.

20 Cales P, Desmorat H, Vinel J, Caucanas J, Ravaud A, Gerin P. Incidence of large oesophageal varices in patients with cirrhosis: application to prophylaxis of first bleeding. *Gut.* 1990; 31: 1298–302.

21 Thapa P, Maharjan D, Tamang T, Shrestha S. Clinical correlation between Child Pugh’s score and oesophageal varices in upper gastrointestinal endoscopy in cirrhotic patient. *J. Kathmandu Med. Coll.* 2015; 4: 135–9.