Noninvasive estimation of intravascular volume status in cirrhosis by dynamic size and collapsibility indices of the inferior vena cava using bedside echocardiography

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Key words
central venous pressure, cirrhotic cardiomyopathy, echocardiography, inferior vena cava, inferior vena cava collapsibility, intravascular volume status.

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Abstract

Background and Aim: Echocardiographic assessment of the inferior vena cava diameter (IVCD) and collapsibility index (IVCCI) is a noninvasive estimate of intravascular volume status (IVS) but requires validation for cirrhosis. We evaluated IVC dynamics in cirrhosis and correlated it with conventional tools such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and right atrial pressure (RAP).

Methods: A total of 673 consecutive cirrhotic patients were screened by echocardiography, and 125 patients underwent right heart catheterization with recording of hepatic venous pressure gradient (HVPG), RAP, pulmonary artery (PA) pressure, and PCWP. CVP data were available for 80 (64%) patients, and finally, 76 patients (84% male, 50% ethanol related, mean age 52.1 years, 57.8% with ascites) with complete data were enrolled.

Results: The mean CVP measured was 12.8 ± 4.8 mmHg, and IVCCI was 29.5 ± 10.9%. The IVCD ranged from 0.97 to 2.26 cm and from 0.76 to 1.84 cm during expiration and inspiration, respectively, with a mean of 1.8 ± 0.9 cm. The mean IVCD correlated with RAP (r = 0.633, P = 0.043) but not with HVPG (r = 0.344, P = 0.755), PCWP (r = 0.562, P = 0.072), or PA pressure (r = 0.563, P = 0.588). A negative linear correlation was observed between the CVP and the IVCCI (r = −0.827, P = 0.023) in all patients and stratified for those with (r = −0.748, P = 0.039) and without ascites (r = −0.761, P = 0.047). A positive correlation was observed between CVP and IVCDmax (r = 0.671, P = 0.037) and IVCDmin (r = 0.612, P = 0.040).

Conclusions: IVCD and collapsibility index provides noninvasive IVS assessment, independent of HVPG or ascites, with the potential for calculating fluid requirements in cirrhosis.

Introduction

Cirrhosis is associated with a hyperdynamic circulation characterized by increased cardiac output and reduced systemic vascular resistance (SVR), with a normal or even low mean arterial pressure (MAP).1,2 Optimizing intravascular volume is essential in managing patients with cirrhosis to avoid acute kidney injury induced by hypovolemia and reduce the risk of developing hepatorenal syndrome (HRS) in decompensated cirrhosis.3 However, the presence of cirrhotic cardiomyopathy and ascites and the poor correlation of central venous pressure (CVP) with volume status make clinical assessment difficult.4 CVP is one of the indices of intravascular volume status (IVS) and is an early goal of the goal-directed therapy approach.5,6 However, CVP and pulmonary capillary wedge pressure (PCWP), often used to measure static hemodynamics, are not reliable markers of circulatory volume.7,8 In addition, patients with ascites and peripheral edema may still be relatively underfilled in terms of intravascular volume as some 40–50% of the extracellular fluid volume can be in the microcirculation.

Recently, noninvasive methods to assess IVS, such as ultrasonography,9 transthoracic echocardiography (TTE) with tissue Doppler imaging (TDI),10 and transesophageal echocardiography (TEE),11,12 have been reported. They have reported a correlation between inferior vena cava diameter (IVCD) and CVP. Another study reported a correlation between internal jugular vein (IJV) sonographic diameter and CVP as a surrogate marker of IVS.13 In this study, we assessed the utility of point-
of-care echocardiographic assessment of IVCD and collapsibility to assess the volume status in cirrhotic patients compared with conventional invasive parameters such as CVP and PCWP.

**Methods**

In this observational study, we reviewed the echocardiographic data of 673 consecutive individuals with cirrhosis who were screened between 1 August 2015 and 31 December 2016 at the Institute of Liver and Biliary Sciences, New Delhi. The inclusion criteria were as follows: patients with cirrhosis defined by clinical, biochemical, histological, or ultrasonographic criteria, aged between 18 and 65 years. The exclusion criteria were: patients >65 years, chronic renal disease, pregnancy and peripartum cardiacopathy, hypertension, coronary artery disease, valvular heart disease, sick sinus syndrome/pacemaker, thyroid dysfunction, portal vein thrombosis, transjugular intrahepatic portosystemic shunt (TIPS) insertion, hepatocellular carcinoma, severe anemia, or declining consent to participate. The normal IVCD was measured both during inspiration and expiration using M-mode echocardiography. Of the 673 patients who met the study criteria, 125 patients also underwent right heart catheterization to measure hepatic venous pressure gradient (HVPG) prior to potential liver transplant workup, prior to TIPS, or as a part of clinical assessment of beta-blocker response, thus providing invasive assessment data of HVPG, right atrial (RA) pressure, and PCWP. The CVP data were available for 80 patients, and finally, 76 patients (73.6% male, 57.8% with ascites, 39.4% with prior portal vein thrombosis, transjugular intrahepatic portosystemic shunt (TIPS) insertion, hepatocellular carcinoma, severe anemia, or declining consent to participate. The normal IVCD was measured both during inspiration and expiration. The IVC was visualized, with individuals in the supine position, in the Trendelenburg position. The IVC was monitored using a subcostal four-chamber view (midline, inferior to the xiphoid, angling to the right) and turning the probe antclockwise to 90°, with a slight tilt to the right to achieve subcostal IVC view. Once the 2D image of IVC entering the RA was acquired, the M-mode line was placed through IVC, 1 cm caudal from its junction with the hepatic vein, and a tracing was obtained. IVC and aorta diameter were measured at end-expiration and end-inspiration in two-dimensional long-axis view. All evaluations were performed in the supine position. The IVC collapsibility index (IVCCI) was the difference between the maximum and minimum IVCDs divided by the maximum IVCD, expressed as a percentage (IVCD_{max}−IVCD_{min}/IVCD_{max}×100%). The following parameters were recorded: heart rate (HR), MAP, left ventricular end systolic diameter (LVESD), left ventricular end diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF), cardiac index, and SVR. SVR was calculated by (MAP – mean right atrial pressure [RAP]) × 80/cardiac output. LVESD, LVEDD, and left atrium (LA) diameter were assessed by M-mode. LVEF was measured through the biplane two-dimensional mode using Simpson’s method. Cirrhotic cardiomyopathy was diagnosed if systolic or diastolic dysfunction, together with supporting criteria such as electrophysiological abnormalities or abnormal serum markers, was present. LVDD was defined and classified according to the ASE guidelines as given below:

- Grade 1: e' < 8 cm/s, E/e' ratio < 8, E/A ratio < 0.8, and deceleration time (DT) >200 ms;
- Grade 2: e' < 8 cm/s, E/e' ratio 9–15, E/A ratio 0.8–1.5, and DT 160–200 ms; and
- Grade 3: e' < 8 cm/s, E/e' ratio > 15, E/A ratio > 2, and DT <160 ms.

All echocardiographic studies were performed by the same observer (Jelen S Khumuckham), and intraobserver coefficients of variability in the echocardiography laboratory were 5% for M-mode and 10% for two-dimensional and Doppler values.

**Central venous pressure assessment.** After central catheterization of the internal jugular vein (IJV) using the Seldinger technique, CVP was measured using an electronic transducer with the patient placed in a 15° Trendelenburg position.

**Cardiac catheterization assessment.** Hepatic venous catheterization was performed for the indications cited above from the right femoral route using a 7 Fr double-lumen balloon-tipped Swan-Ganz catheter, and the parameters recorded included the free hepatic venous pressure (FHVP), wedged hepatic venous pressure (WHVP), IVC pressure, RV pressure, pulmonary artery (PA) pressure, and PCWP. The HVPG level was calculated as the difference between the WHVP and FHVP readings.

**Statistical analysis.** Statistical analysis was conducted using student’s t test for normally distributed data and Mann Whitney U test for nonparametric data. In addition, χ² analysis with correction for small numbers and ANOVA were also carried out along with agreement of IVCD assessment with invasive monitoring parameters by Pearson correlation. Data are expressed as mean ± SD or percentages. Statistical significance was considered to be at or below the 5% level. Data were analyzed with SPSS software version 22 (IBM SPSS Statistics, Armonk, USA).

Ethical clearance was obtained from the Institutional Ethics Committee and was performed in accordance with the Declaration of Helsinki. All authors had access to the manuscript data and have approved the final manuscript.

**Echocardiographic assessment.** All the echocardiographic tests were conducted by a cardiologist who was blinded to the patients’ CVP using a 3.5 MHz probe (ALOKA Medical Systems, Tokyo, Japan). The TTE evaluation was performed as per the American Society of Echocardiography (ASE) guidelines. The IVC was visualized, with individuals in the supine position, using a subcostal four-chamber view (midline, inferior to the xiphoid, angling to the right) and turning the probe antclockwise to 90°, with a slight tilt to the right to achieve subcostal IVC view. Once the 2D image of IVC entering the RA was acquired, the M-mode line was placed through IVC, 1 cm caudal from its junction with the hepatic vein, and a tracing was obtained. IVC and aorta diameter were measured at end-expiration and end-inspiration in two-dimensional long-axis view. All evaluations were performed in the supine position. The IVC collapsibility index (IVCCI) was the difference between the maximum and minimum IVCDs divided by the maximum IVCD, expressed as a percentage (IVCD_{max}−IVCD_{min}/IVCD_{max}×100%). The following parameters were recorded: heart rate (HR), MAP, left ventricular end systolic diameter (LVESD), left ventricular end diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF), cardiac index, and SVR. SVR was calculated by (MAP – mean right atrial pressure [RAP]) × 80/cardiac output. LVESD, LVEDD, and left atrium (LA) diameter were assessed by M-mode. LVEF was measured through the biplane two-dimensional mode using Simpson’s method. Cirrhotic cardiomyopathy was diagnosed if systolic or diastolic dysfunction, together with supporting criteria such as electrophysiological abnormalities or abnormal serum markers, was present. LVDD was defined and classified according to the ASE guidelines as given below:

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**Results**

Demographic data. Of 76 individuals enrolled for the study, 64 (84.2%) were male, with the etiology of cirrhosis being ethanol related (50%), non-alcoholic steatohepatitis (39.4%), and chronic viral hepatitis (10.5%). The demographics of individuals are outlined in Tables 1 and 2. The mean age of individuals was 52.1 years. The average corrected body mass index (BMI) was 22.9 kg/m², which ranged between 14.1 and 29.8 kg/m². Of the 76 patients, 36 (47.3%) met criteria for grade 1 LVDD, and 4 met criteria for grade 2 LVDD (5.2%). The criteria for cirrhotic cardiomyopathy were met in 22(28.9%) patients.

CVP and IVCD assessment. The mean CVP measured was 12.8 ± 4.8 mmHg, with an IVCCI of 29.5 ± 10.9%. The CVP was found to be less than 8 mmHg among 12 (15.7%) patients,
while 42 (55.2%) patients had CVP between 8 and 12 mmHg, and 22 (28.9%) patients had CVP greater than 12 mmHg. During inspiration, the IVCD decreased in every individual to a variable extent of patients without and with ascites.20,21

There was a statistically significant correlation in the mean CVP pressure and the IVCCI and cardiac catheterization (Figs 1a–c). A strong positive correlation was demonstrated between the CVP and the IVCDmax between groups as determined by one-way ANOVA (P = 0.001). This is presented in Table 3.

A Pearson correlation was conducted to determine the relationship between the CVP and the IVCCI (%), and the maximum and minimum IVCD. A strong negative linear correlation was observed between the CVP (10.3 ± 4.4 mmHg) and the IVCCI, which was statistically significant (r = −0.827, P = 0.023) in all patients and stratified for those with ascites (r = −0.748, P = 0.039) and without ascites (r = −0.761, P = 0.047) (Figs 1a–c). A strong positive correlation was demonstrated between the CVP and the IVCDmax (r = 0.671, P = 0.037) and IVCDmin (r = 0.612, P < 0.040). (Fig. 2a,b) The correlation coefficients for IVCDmax, IVCDmin, and CVP remained significant even in the presence of ascites.

Cardiac hemodynamic studies and IVC assessments. On right heart catheterization studies, a Pearson correlation coefficient was calculated between the mean IVCD and RAP (r = 0.633, P = 0.043) and PCWP (r = 0.562, P = 0.072), HVPG (r = 0.344, P = 0.755), or PA pressure (r = 0.563, P = 0.588). The correlation coefficients for IVCDmax and IVCDmin as a function of RA pressure were 0.686 and 0.767, respectively.

| Variables | Preascitic cirrhosis (n = 32) | Cirrhosis with ascites (n = 44) | P value |
|-----------|-------------------------------|-------------------------------|--------|
| Age (years) | 46.8 ± 7.6 | 49.1 ± 9.8 | 0.048 |
| Hypertension | 5% | 7% | 0.265 |
| Smoker | 3% | 2% | 0.658 |
| Diabetes mellitus | 18% | 22% | 0.063 |
| Presence of esophageal varices | 22 | 34 | |
| Prior variceal bleeding | 13 | 17 | |
| Child-Pugh score | 9.8 ± 1.0 | 10.3 ± 0.91 | 0 |
| MELD score | 14.2 ± 2.5 | 17.4 ± 2.3 | 0.001 |
| MELD Na | 16.4 ± 3.06 | 18.1 ± 3.06 | 0 |
| Serum creatinine (mg/dL) | 0.71 ± 0.24 | 0.84 ± 0.22 | 0.314 |
| INR | 1.5 ± 0.31 | 1.6 ± 0.31 | 0.259 |
| MAP (mmHg) | 83.7 ± 11.5 | 80.3 ± 12.9 | 0.378 |
| HR (bpm) | 78.1 ± 14.2 | 88.9 ± 11.2 | 0.056 |
| Central venous pressure (mmHg) | 10.3 ± 2.4 | 14.3 ± 2.8 | 0.045 |
| Echocardiographic parameters | | | |
| IVCDmax (cm) | 1.7 ± 0.31 | 2.5 ± 1.6 | 0.024 |
| IVCDmin (cm) | 1.25 ± 0.31 | 1.42 ± 0.24 | 0.364 |
| IVC collapsibility (%) | 32.2 ± 5.1 | 21.7 ± 2.9 | 0.036 |
| LVEF (%) | 55–60 | 55–60 | 0.9 |
| LVEDV (mL) | 75.0 ± 8.1 | 73.9 ± 5.7 | 0.342 |
| LVESV (mL) | 32.7 ± 4.2 | 28.7 ± 4.7 | 0.413 |
| LV end-diastolic posterior wall thickness (cm) | 1.0 ± 0.2 | 1.0 ± 0.2 | 0.452 |
| LV end-diastolic septal thickness (cm) | 1.1 ± 0.3 | 1.0 ± 0.2 | 0.433 |
| LV end diastolic diameter (cm) | 4.6 ± 0.6 | 4.8 ± 0.5 | 0.056 |
| SVRI (dynes. s m2/cm5) | 2042 ± 416 | 1098 ± 566 | 0.004 |

Hemodynamic assessment and cardiac catheterization

| Variables | Preascitic cirrhosis (n = 32) | Cirrhosis with ascites (n = 44) | P value |
|-----------|-------------------------------|-------------------------------|--------|
| CO (L/min) | 5.4 ± 1.9 | 6.1 ± 1.7 | 0.041 |
| Therapy at the time of inclusion (%) | | | |
| β-blockers | 0 | 0 | |
| Furosemide | 5 | 38 | |
| Spironolactone | 10 | 38 | |

CO, cardiac output; CVP, central venous pressure; HR, heart rate; HVPG, hepatic venous pressure gradient; INR, XXX; IVCD, inferior vena cava diameter; LV, left ventricle; LVEDV, LV end diastolic volume; LVEF, left ventricular ejection fraction; LVESV, LV end systolic pressure; LVEDD, left ventricular systolic diameter; MAP, mean arterial pressure; MELD, model for end-stage liver disease; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; SVI, stroke volume index; SVRI, systemic vascular resistance index.

Operating characteristic (ROC) curves for the predictors of volume status (CVP) using noninvasive IVC parameters are shown in Table 4. The collapsibility indices (maximum and minimum
IVCCI had the best area under the curve (AUC). The IVCDmax size cut-off value with optimum predictive use for CVP above or below 10 mmHg was 2.0 cm, and the optimal IVCCI max cut-off value was 40%. The IVCDmin <1.5 cm predicted CVP <10 mmHg and indicated the need for further volume resuscitation, with a sensitivity of 91%, specificity of 79%, and 96% negative predictive value. The most notable finding is that the IVC size measurements and the collapsibility indices had an excellent negative predictive value.

**Determination of accuracy of IVC measurements to classify volume status.** All patients were categorized by IVCDmax and IVCCI into one of nine subgroups as shown in Table 5. The mean CVP was calculated for each subgroup, and the percentages of patients falling within the traditional CVP ranges of 0–5, 5–10, 10–15, and >15 mmHg were determined. They were grouped according to whether their collapsibility was high (>55%), low (<35%), or normal (35–50%) and whether their IVC was small (<1.7 cm), normal (1.7–2.1 cm), or large (>2.1 cm). The specific subgroup’s IVCCI and size were assigned according to current guidelines.14 Within each classification of size, there was an increase in mean CVP as collapsibility decreased. On the contrary, when grouped by IVCCI, there was no significant change in mean CVP between patients with small-sized IVCDs.

When collapsibility was high and IVC was small or normal, CVP was between 0 and 5 mmHg in 72% and between 5 and 10 mmHg in 24% cases. Thus, this subgroup indicated hypovolemia and the potential need for volume expansion depending on the clinical scenario. When collapsibility was high with a large IVC, or IVCCI was normal, and IVC was small or normal, CVP was between 0 and 10 mmHg 87% of the time. If collapsibility was normal and the IVC was large, CVP was between 10 and 15 mmHg. On the other hand, if the IVC was large with low IVCCI, the CVP was between 10 and 20 mmHg.

**Discussion**

Overall, our findings support the use of point-of-care noninvasive echocardiographic measurements of IVCD or collapsibility to estimate CVP or RAP as a surrogate marker for IVS. Although CVP monitoring is a useful tool for guiding fluid management, it requires placement of a central venous catheter, which is an invasive procedure and is associated with complications. bedside sonography/echocardiography has emerged as a potentially useful tool, optimizing hemodynamic measurements in cirrhosis with careful interpretation of right ventricular function integrated with cardiac output and perfusion pressure.23

Initially, IVS assessed noninvasively by the IVC ultrasound was focused on the correlation of the mean IVCD with the CVP.21 These results are comparable to our findings. There is an inverse relationship of the IVCCI to the CVP at extremes of intravascular fluid volume. Brennan et al. documented that the combination of both IVCD and collapsibility indices improve evaluations of the IVCCI with clinically important categories of RAP.22

**Accuracy of noninvasive assessment of CVP.**

Several studies have evaluated the correlation between RA pressure and different IVC parameters with variable accuracy.23,24 A good correlation between the IVCCI and RA pressure (0.57 < r ≤ 0.76) has been reported.25 Although there is a correlation between IVCD and RAP, because of the variability and overlap between patients with normal and elevated RAP, it can only be used as a surrogate marker for dynamic assessment rather than an absolute index of RAP.26 An increase of RAP beyond a certain level may cause only minimal increases in IVCD and the degree of IVCCI. Thus, IVC dimensions and collapsibility can be used to detect elevated CVP, but they have limited utility in identifying the magnitude of CVP elevation. As per the ASE guidelines, IVCD ≤2.1 cm that collapses >50% with inspiration suggests normal RAP of 3 mmHg (range, 0–5 mmHg), whereas IVCD>2.1 cm that collapses ≤50% with inspiration suggests high RAP of 15 mmHg (range, 10–20 mmHg). In scenarios where IVCD and collapse have contradictory values, an intermediate value of 8 mmHg (range, 5–10 mmHg) may be used.27

Stawicki et al.28 demonstrated that the IVCCI strongly correlates with low (<20%) and high (>60%) CVP values and suggested that the closer the IVCCI is to 0 or 100%, the higher is the probability that the patient is either volume-overloaded or volume-depleted, respectively. The ability to predict CVP values precisely is of untested clinical gain, taking into account the poor performance of CVP as a marker of intravascular volume and fluid responsiveness. A very high IVCCI (often associated with a very low CVP) may serve as a rational sign that it is possible to give more fluid without precipitating volume overload and reduction in IVCCI, a marker of successful volume repletion.29

Previous studies have consistently demonstrated that patients with cirrhosis have diastolic dysfunction.30–32 The presence of cirrhotic cardiomyopathy may ultimately confound these readings, and the IVC and intracardiac pressures may not

### Table 3 Comparison of mean arterial pressure, heart rate, IVC collapsibility index, mean CVP pressure, and maximum and minimum IVC diameter of patients in the three groups of intravascular volume states

| Parameters                          | CVP < 8 mmHg (n = 12) | CVP 8–12 mmHg (n = 42) | CVP > 12 mmHg (n = 22) | ANOVA (P-value) |
|-------------------------------------|------------------------|-------------------------|------------------------|-----------------|
| Mean arterial pressure (mmHg)       | 70.2 ± 14.2            | 75.0 ± 16.3             | 72.63 ± 12.4           | 0.625           |
| Heart rate (per minute)             | 90.7 ± 12.9            | 89.6 ± 24.9             | 92.70 ± 21.5           | 0.065           |
| Mean CVP pressure (mmHg)            | 6.4 ± 1.8              | 10.6 ± 2.4              | 15.7 ± 3.5             | 0.001           |
| IVCD_{max} (cm)                     | 1.6 ± 1.9              | 1.8 ± 2.56              | 2.3 ± 1.6              | 0.004           |
| IVCD_{min} (cm)                     | 1.0 ± 0.7              | 1.4 ± 0.51              | 2.1 ± 0.9              | 0.001           |
| IVCCI (%)                           | 51.5 ± 7.5             | 30.2 ± 10.1             | 20.7 ± 8.9             | 0.001           |

CVP, central venous pressure; IVC, inferior vena cava; IVCCI, IVC collapsibility index; IVCD_{max}, maximum IVC diameter; IVCD_{min}, minimum IVC diameter.

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Systemic Hemodynamics in Cirrhosis

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Figure 1 (a) Correlation between central venous pressure and inferior vena cava collapsibility index in all subjects ($n = 76$). (b) Correlation between central venous pressure and inferior vena cava (IVC) collapsibility index in patients with ascites.

Fluid management strategy in cirrhosis. Incorporation of a goal-directed sonographic protocol, including assessment of the IVC, has been shown to improve the accuracy of fluid assessment and resuscitation in critically ill noncirrhotic patients with shock. Point-of-care sonography evaluating cardiac contractility and IVC collapsibility in patients with suspected sepsis improves fluid management and, possibly, clinical decisions. On the other hand, our study determined that a small-diameter IVC with high collapsibility correlates with low-volume states. However, the treating clinician needs to gauge the need for fluid resuscitation in individual patients as a dynamic continuum. All patients with small IVC and high IVCCI need not be proposed as a sensitive and accurate method to evaluate subclinical systolic dysfunction in various groups of disease. Our data serve to corroborate these findings in stable cirrhotic patients. In patients with decompensated liver cirrhosis and ascites, with creatinine >1.5, there was an impairment in IVCCI (IVC collapsibility <50%). This is further affected by the presence of cirrhotic cardiomyopathy and sepsis states, which alter volume status in critically ill cirrhotic patients. Sampaio et al. reported that IVCD and IVCCI are of value in the prediction of IVS in liver cirrhosis with renal dysfunction. On the other hand, the study of Kitamura and Kobayashi found that interpretation of caval indices is difficult due to factors that restrict the physiological variability of the IVC, such as cirrhosis, external compression of the IVC, and elevated intra-abdominal pressure due to ascites.

Figure 2 (a) Correlation between expiratory inferior vena cava (IVC) diameter and central venous pressure. (b) Correlation between inspiratory IVC diameter and central venous pressure.

In clinical practice, it is difficult to distinguish between portopulmonary hypertension from vascular overload using IVCD only without right heart catheterization.

In contrast, resting LV systolic functional impairment is not apparent when measured by conventional methods such as LV ejection fraction, partly because of reduced afterload due to a low SVR. Recently, 2D speckle-tracking strain analysis has been accurately reflect intravascular volume (IVV) any longer. In clinical practice, it is difficult to distinguish between portopulmonary hypertension from vascular overload using IVCD only without right heart catheterization.

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without ascites, independent of HVPG. IVCD and BMI, RA pressure, and PCWP in cirrhosis with and noninvasive point-of-care tests such as IVCD and collapsibility. Patients who are septic, in shock, or mechanically ventilated using mum and maximum IVCDs but an inverse relation with IVCCI. In cirrhosis. There is a positive relationship of CVP with mini-

index can provide a useful guide for noninvasive IVS assessment a steadfast formula for calculating AUC, area under receiver operating characteristic curve; cut-off, parameter value for optimal performance; CVP, central venous pressure; IVCDmax, maximum inferior vena cava diameter; IVCDmin, minimum inferior vena cava diameter; NPV, negative predictive value; PPV, positive predictive value.

| Parameter     | AUC     | Cut-off | Sensitivity | Specificity | PPV  | NPV  |
|---------------|---------|---------|-------------|-------------|------|------|
| IVCDmax       | 0.712   | 2.0 cm  | 73          | 85          | 62   | 90   |
| IVCDmin       | 0.678   | 1.5 cm  | 86          | 79          | 59   | 96   |
| IVCCImin      | 0.773   | 20%     | 70          | 82          | 57   | 90   |
| IVCCImax      | 0.791   | 40%     | 78          | 84          | 62   | 90   |

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Table 4 Area under receiver operating characteristic curve for prediction of volume status CVP above or below 10 mmHg

Table 5 Tabulation of patients as per IVC size (small [<1.7 cm], normal [1.7–2.1 cm], or large [>2.1 cm]) and collapsibility (high [>55%), low [<35%), or normal [35–50%]) with measured CVP as per 3 volume status subgroups

| Volume status | CVP < 8 mmHg (decreased IVS) (n = 12) | CVP 8–12 mmHg (euvolemic) (n = 42) | CVP > 12 mmHg (increased IVS) (n = 22) |
|---------------|--------------------------------------|-----------------------------------|--------------------------------------|
| Collapsibility (decreased IVS) (n = 12) | CVP < 8 mmHg (decreased IVS) (n = 12) | CVP 8–12 mmHg (euvolemic) (n = 42) | CVP > 12 mmHg (increased IVS) (n = 22) |
| Size (cm)     | <1.7                                 | 1.7–2.1                           | >2.1                                 |
| No. of patients | 4                                    | 6                                 | 2                                   |
| Mean CVP (mmHg) | 5                                    | 6                                 | 8                                   |
| 0–5 mmHg (%)   | 72                                   | 80                                | 50                                  |
| 5–10 mmHg (%)  | 24                                   | 20                                | 50                                  |
| 10–15 mmHg (%) | 4                                    | 0                                 | 0                                   |
| >15 mmHg (%)   | 0                                    | 0                                 | 0                                   |

CVP, central venous pressure; IVC, inferior vena cava; IVS, intravascular volume status.

volume resuscitated unless there is a clinical indication with impaired hemodynamics. In clinically hypovolemic states, an anticipated change in IVCD can be used to predict a patient’s response to vol-

e expansion. The limitations of the study were the exclusion of ventilated patients, those on hemodialysis, or overt heart failure that limits the applicability of this approach in critically ill cirrhotic patients. In addition, we tested asymptomatic patients who were clini-
cally stable. These data need further validation in scenarios like shock or sepsis, which will alter cardiac hemodynamics. In such situations, noninvasive IVC parameters need to be interpreted in terms of clinical status, such as tissue perfusion, renal function, and pulmonary fluid volume. Future prospective studies could be focused on finding a steadfast formula for calculating fluid requirements in cirrhosis patients who are septic, in shock, or mechanically ventilated using noninvasive point-of-care tests such as IVCD and collapsibility.

In conclusion, the dynamic IVC size and collapsibility index can provide a useful guide for noninvasive IVS assessment in cirrhosis. There is a positive relationship of CVP with minimum and maximum IVCDs but an inverse relation with IVCCI. Our findings corroborate the correlations of echocardiographic IVCD and BMI, RA pressure, and PCWP in cirrhosis with and without ascites, independent of HVPG.

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