



Bedside ultrasound to assess acute central venous pressure change during treatment of decompensated heart failure

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ABSTRACT

Background: Accurate volume status assessment is crucial for the treatment of acute decompensated heart failure (ADHF). Volume status assessment by physical exam is often inaccurate, necessitating invasive measurement with right heart catheterization (RHC), which carries safety, pragmatic (scheduling, holding anticoagulants, etc.), and financial burdens. Therefore, a reliable, non-invasive, cost-effective alternative is desired. Previously, we developed an ultrasound (US) based technique to measure internal jugular vein (IJV) compliance during RHC which was used for single time point central venous pressure (CVP) predictions. We now aim to apply this technique to track acute changes in CVP during diuresis for ADHF in patients with an in-dwelling pulmonary artery catheter (PAC).

Methods: We used an observational, prospective study design and recruited 15 patients from the cardiac critical unit (CCU) being treated for ADHF (systolic or diastolic) with intravenous (IV) diuretics with/without inotropic agents who underwent Swan- Ganz catheter/PAC insertion for continuous CVP monitoring. 13 of 15 patients received milrinone infusions. US images of the IJV were obtained at end-expiration and during the strain phase of Valsalva at multiple 2–3 hours intervals. Change in IJV cross-sectional area (CSA) (ImageJ) was used as a measure of IJV compliance. Patients unable to perform the Valsalva maneuver were excluded.

Results: Calculated percentage change (%Δ) in CSA of IJV was plotted against CVP. An inverse relationship was observed between CVP and %Δ in CSA of IJV. The data was fit with a polynomial regression curve ($R^2 = 0.36$, root mean square error = 3.19). Fivefold cross-validation showed a stable model for predicting CVP based on CSA ($R^2 = 0.31$, root mean square error = 3.18)

Conclusion: Serial portable US assessment of IJV compliance can act as a surrogate measure of CVP and, therefore, can provide reliable information on acute hemodynamic changes in ADHF.

1. Introduction

Congestive heart failure (CHF) is a clinical syndrome that is associated with significant morbidity and mortality and poses a serious financial burden [1]. It impacts nearly 6.2 million Americans and constitutes the primary diagnosis for hospital discharge in ~ 1 million and a secondary diagnosis in ~ 2 million hospitalizations annually [1]. While patients admitted with CHF have about 20–30% risk of death within a year, the inpatient mortality for CHF exacerbation ranges from ~ 4% to 12% and as high as ~ 20% in high-risk subgroups [2–4]. Discharge from CHF-related hospitalization is followed by a readmission within 30 days in about 24% of cases [5].

Acute decompensated heart failure (ADHF) results from elevated ventricular filling pressures and manifests as pulmonary and systemic congestion [6]. The accurate assessment of filling pressures is the key to optimal management, achieving euvolemic status and thereby reducing readmission risk. The non-invasive evaluation of filling pressures through physical examination is carried out by measuring the height of the right internal jugular vein (IJV) meniscus, which is a manometric measure of right atrial pressure (RAP) and, therefore, reflective of left ventricular filling pressures. However, this estimation of central venous pressure (CVP) by physical examination is accurate in < 50% of cases and frequently is either unobtainable or prone to subjectivity [7,8]. This subjective assessment of volume status often underestimates true

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intravascular hemodynamics and could potentially lead to inadequate diuresis that increases the risk for early readmission with ADHF [9]. On the other hand, invasive right heart catheterization (RHC) is considered the gold standard for volume assessment in ADHF, however, it carries both safety and financial concerns [10]. Therefore, a non-invasive, safe, and reliable alternative for accurate assessment of CVP (i.e. RAP) is needed.

Previously, we developed a technique to predict RAP using bedside ultrasonic assessment of jugular venous compliance that revealed an IJV cross-sectional area (CSA) change $\leq 66\%$ between end-expiration and strain phase of Valsalva correlates with RAP ≥ 12 mmHg with the sensitivity of 77%, specificity of 75% and negative predictive value of 94% [11]. However, our previous RAP predictions were at a single time point. In this current study, we aimed to correlate serial ultrasound measurements of IJV compliance with acute changes in CVP, obtained with an in-dwelling pulmonary artery catheter (PAC) in patients undergoing diuresis for ADHF, to determine if this technique can reliably provide CVP predictions in a serial fashion for continued hemodynamic monitoring to act as an alternative to RHC/indwelling PAC.

2. Methods

We conducted a pilot study using an observational, prospective study design that was approved by the University of Pittsburgh Institutional Review Board. Informed consent was obtained from all participants. The supporting data of this study are available from the corresponding author on reasonable request.

2.1. Study population

Patients admitted with ADHF to the coronary care unit (CCU) at the University of Pittsburgh Presbyterian Hospital were enrolled in the study if they met the following criteria: (i) active treatment with intravenous (IV) diuretics, (ii) placement of an indwelling PAC/Swan-Ganz catheter for continued CVP monitoring, and (iii) ability to consent and perform Valsalva maneuver. Unlike previous studies, patients on inotropic agents (e.g., dobutamine, milrinone) and mechanical circulatory support (MCS) (e.g., IABP, Impella) were not excluded since most patients with an indwelling Swan-Ganz catheter have advanced/end-stage heart failure requiring inotropic or MCS; however, patients on dialysis without treatment with IV diuretics were excluded (Fig. 1). Demographic data and baseline clinical characteristics such as gender,

age, body mass index (BMI), ejection fraction (EF), B-type natriuretic peptide (BNP) levels, serum creatinine, treatment with inotropes/vasopressors, and comorbidities were collected.

2.2. Ultrasound imaging

Two-dimensional imaging of either one of the IJV was performed at the bedside using a portable ultrasound imaging system (iLook 25; Sonosite Corp) with a standard vascular probe (broadband 10–5 MHz linear array transducer designed for vascular access). The patients were supine with the head end of the bed elevated to 45 degrees. A clear cross-sectional view of the IJV was obtained in the center of the ultrasound screen and two sets of still-frame images of the IJV were captured, first at end-expiration during a relaxed breathing pattern and second during the strain phase of Valsalva (Fig. 2). CVP at rest was also simultaneously measured to correlate with the percentage CSA change of IJV. The Valsalva maneuver was standardized with a simple manometer attached to a short disposable tube and the patient was instructed to forcefully expire and generate a pressure of at least 30 mm Hg. The still images were uploaded in bitmap (bmp) format using software provided with the ultrasound system (Infraview; Sonosite Corp) and analyzed using Image J software (National Institutes of Health). Jugular vein cross-sectional measurements were obtained in both sets of images by manually tracing the luminal edge of the vessel on the ultrasound image.

2.3. Data collection

The study was conducted on CCU patients with indwelling PAC undergoing treatment for ADHF with IV diuretics and meeting the inclusion criteria. Initial ultrasound images were obtained preferably within 24 hours of PAC insertion. Serial images were obtained at regular intervals of 1–3 hours during the daytime. An average of 6 sets of images were obtained for each patient. Pulmonary artery pressures, cardiac profiles, and net volume status were also recorded at the time of each image collection.

2.4. Statistical analysis

Categorical data are presented as percentages, and the continuous data are reported by median (IQR). Percentage change in CSA of IJV between end-expiration and strain phase of Valsalva was calculated using the formula:

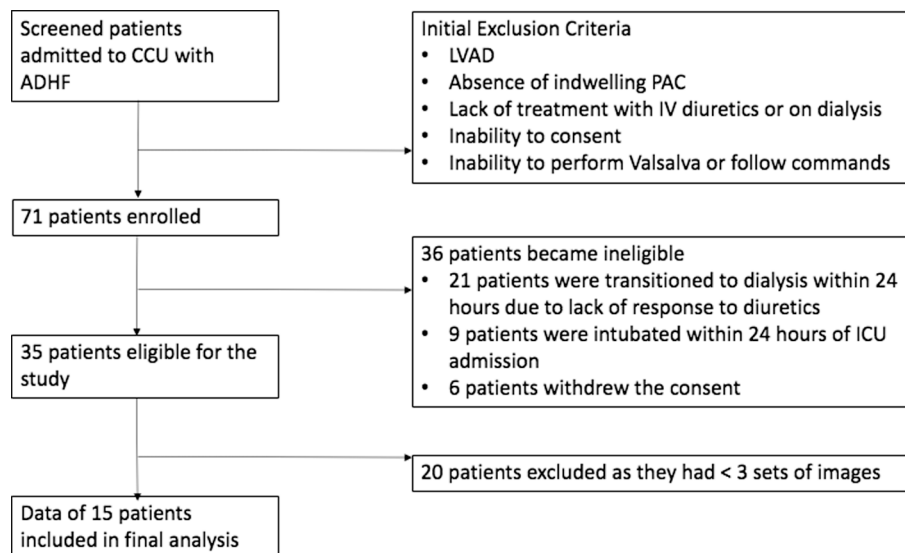


Fig. 1. Study design flow chart. ADHF indicates acute decompensated heart failure; LVAD: left ventricular assist device; PAC: pulmonary artery catheter; CCU: cardiac care unit; ICU: intensive care unit.

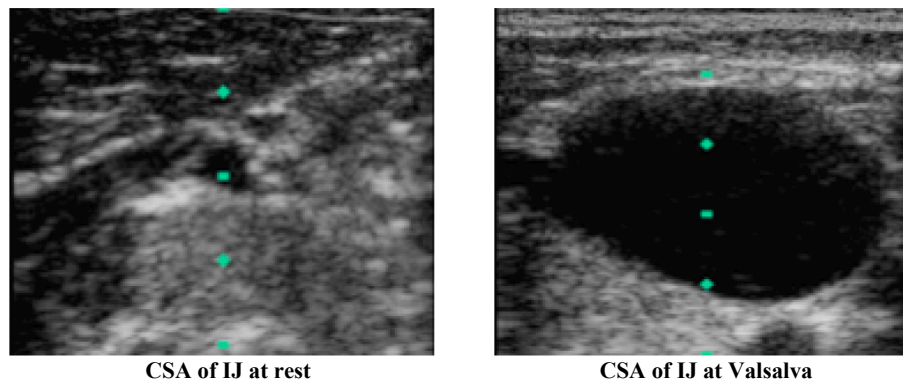


Fig. 2. Ultrasound images of study subjects. The left side image shows the cross-sectional area (CSA) of the internal jugular vein (IJV) at rest while the right-side image shows the CSA of IJV at Valsalva. The patient has a difference of > 89.5% in CSA of IJV between rest and Valsalva corresponding to central venous pressure (CVP) < 10 cm.

Percentage change in IJV compliance (% Δ) = [CSA of IJV during Valsalva (V2) – CSA of IJV at end-expiration (V1)] ÷ CSA of IJV at end-expiration (V1) × 100

$$\% \Delta = \frac{V2 - V1}{V1} \times 100$$

The calculated percentage change in IJV compliance was then plotted against CVP for comparison for each study subject. The data were fit to a polynomial regression curve at every datapoint. We used several non-linear models including the growth exponential, cubic spline, and polynomial model. We used fivefold cross-validation to calculate the internal validity of each model and then chose the model with less variability in R-square.

A receiver operating characteristic (ROC) curve was also generated from the IJV compliance and the area under the curve (AUC) was tested for a significant difference from 0.5 (null hypothesis) using CVP > 10 as a gold standard to indicate volume overload. We used 10 mmHg as the cut point as it represents the upper normal limit of CVP. CVP higher than 10 mmHg indicates volume overload and values <10 mmHg represent normal volume state. This finding is based on the biomechanical properties of veins that allow maximum compliance at lower pressures (i.e. at normal volume state or CVP < 10 mmHg) whereas at extreme pressures (i.e. at a hypervolemic state or CVP > 10 mmHg) the compliance and hence, the variation in IJV-CSA between rest and Valsalva decreases [11]. Thus, this cut point has clinical significance in the determination of volume status at a given time point.

We used a Youden test to calculate the optimum cut point of CSA. The diagnostic utility of the percentage change in IJV compliance to detect elevated CVP was assessed by calculating the sensitivity, specificity, and positive likelihood ratio (LR). Statistical analysis was performed with Stata 16.1 (StataCorp, College Station, TX).

3. Results

A total of 15 patients meeting the inclusion criteria were assessed in our study. The studied cohort had a median age of 66 years and 67% were males. All patients were managed for cardiogenic shock and had heart failure with reduced ejection fraction (HFrEF, EF < 50%) with most of them (73.3%) having EF of 10–15%. 53.3% of the cohort had isolated systolic dysfunction, while 46.7% had combined systolic and diastolic heart failure. Ischemic cardiomyopathy was present in 73.3% of the patients. 87.67% of the patients were on inotropic support (either dobutamine or milrinone), 13.3% had MCS (either IABP or Impella), and 6.67% were just on IV diuretics. Severe pulmonary hypertension was noted in 13.3% of patients. Other demographic characteristics and clinical variables are shown below (Table 1).

The patients were studied using CVP and CSA measures for a median

Table 1
Baseline Patient Characteristics.

Categorical Characteristics	Number (%) n = 15
Male	10 (66.7)
HFrEF	15 (100)
EF 10–25%	11 (73.3)
EF 25–45%	4 (26.7)
Ischemic Cardiomyopathy	11 (73.3)
Systolic Dysfunction	8 (53.3)
Tricuspid Regurgitation (mild-moderate)	10 (66.7)
Pulmonary Hypertension	
None	9 (60.0)
Mild-moderate	4 (26.7)
Severe	2 (13.3)
Atrial Fibrillation	10 (66.7)
CAD	11 (73.3)
HTN	12 (80.0)
DM	6 (40.0)
COPD	5 (33.3)
OSA	4 (26.7)
CKD stage 3–5	5 (33.3)
Inotropic Support	13 (86.7)
Mechanical Circulatory Support	2 (13.3)
Continues Characteristics	Median (IQR)
Age	66 (50–68)
BMI	28.3 (22.9–29.8)
Total Cholesterol	132 (104–156)
LDL	63 (57–90)
HDL	30 (19–42)
SBP on admission	104 (92–133)
DBP on admission	66 (54–78)
Temp on admission	36.5 (36.1–36.7)
RR on admission	22 (18–28)
Creatinine on admission	1.23 (1.2–1.6)
BNP on admission	2434 (1664–5000)
Average time interval between insertion of PA catheter and first reading	1 (1–3)
Average number of readings	5 (3–8)
Average hour of monitoring	15 (5–29)

of 4 (IQR = 2–6) repeated times during the course of the study. The total number of measurements for these patients was 90. The median (IQR) of CVP and CSA was 10 (7–13) and 96.1 (34.5–256.0), respectively. We used a growth model to predict the CVP based on CSA. In this model (Figs. 3 and 4, red line), the CVP was imputed using this equation: CVP = 6.48 + 7.38 × 0.99^{CSA}. This model had an R² of 0.36 with a root mean square error of 3.19. Fivefold cross-validation showed a stable model for predicting CVP based on CSA (R² = 0.31, root mean square error = 3.18). The variance of R² for this model was 0.02 vs. 0.04 for a

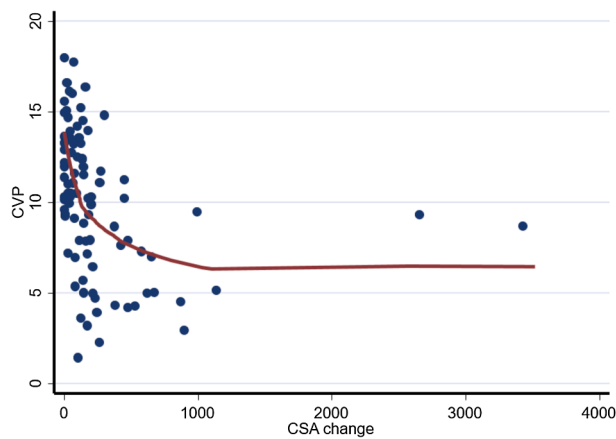


Fig. 3. Plotted change in cross-sectional area (CSA) of the IJV against central venous pressure (CVP). The data was fit with a nonlinear regression (red curve) showing an inverse exponential relationship between change in CSA and CVP ($CVP = 6.48 + 7.38 \times 0.99^{CSA}$). Removing CSA outliers (>2000) generate a similar equation ($CVP = 6.01 + 7.75 \times 0.99^{CSA}$).

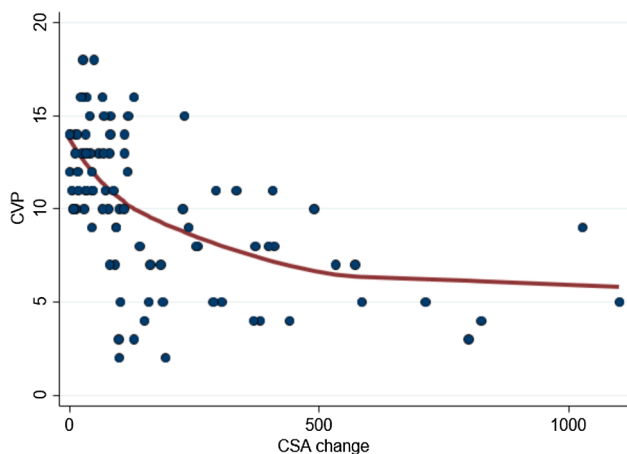


Fig. 4. Plotted change in cross-sectional area (CSA) of the IJV against central venous pressure (CVP). The data was fit with a nonlinear regression (red curve) showing an inverse exponential relationship between change in CSA and CVP.

polynomial model and 0.03 for a cubic spline model.

Removing two CSA outliers (>2000) generates a similar equation ($CVP = 6.01 + 7.75 \times 0.99^{CSA}$) with R^2 of 0.37 and root mean square error of 3.20. Fivefold cross-validation showed a stable model for predicting CVP based on CSA ($R^2 = 0.35$, root mean square error = 3.19). The variance of R^2 for this model was 0.03.

A ROC curve was generated from those patients ($n = 15$) with both indwelling PAC and IJV imaging (Fig. 5). The ROC curve was generated using percentage change in IJV CSA during Valsalva and resting end-expiration. In all measurements, the optimum CSA cut point for $CVP < 10$ was 89.5 (Fig. 2). This cut-point had a sensitivity of 83% and specificity of 79%, with an area under the curve (AUC) of 0.89 and a positive likelihood ratio (LR) of 3.6 for this CVP.

4. Discussion

In this study, we sought to determine whether our technique could track acute change in volume status during active diuresis in critically ill patients admitted with ADHF. We found that our technique could successfully be incorporated in the management of heart failure patients, who were on inotropic and/or mechanical ventilatory support, to assess the acute variations in their systemic congestion. Our technique has the

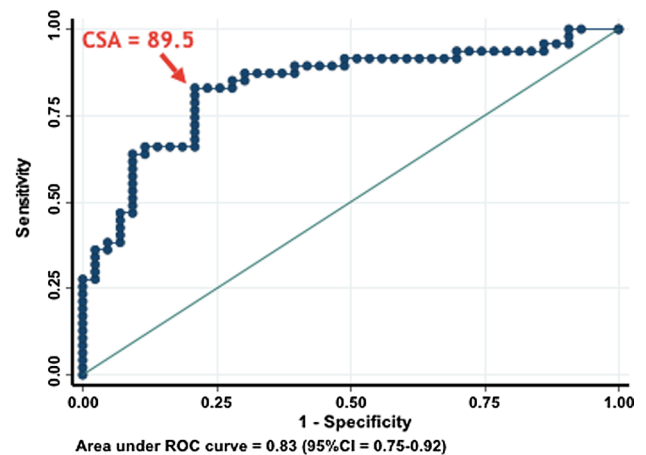


Fig. 5. ROC curve of internal jugular vein (IJV) cross-sectional area (CSA) compared with right atrial pressure (RAP) representing optimal RIJCV CSA (red arrow). An increase in right IJV CSA of $> 89.5\%$ with Valsalva - predicted elevated CVP (≤ 10 mm Hg) with sensitivity of 83% and specificity of 79% (AUC: 0.83; 95% CI, 0.75–0.92; $P < 0.05$). AUC indicates area under the curve; CI, confidence interval; ROC, receiver operating characteristic.

advantages of being non-invasive, easily implementable, and cost-effective, and constitutes a steep learning curve. Since volume status assessment by physical examination is very unreliable in critically ill patients, especially those requiring inotropic and circulatory support, and RHC confers safety concerns associated with the physiological burden of indwelling instrumentation; our technique would help address both issues.

Historically, patient-reported symptoms (dyspnea, orthopnea, PND) combined with clinical signs of volume overload (peripheral edema, JVD, S3 gallop, or bibasilar crackles) have been routinely used to diagnose ADHF [12,13]. Findings of pulmonary vascular congestion and edema on chest imaging are also of assistance in diagnosis but need to be distinguished from acute respiratory distress syndrome due to pulmonary and systemic causes. Similarly, certain cardiac biomarkers such as BNP or its variants are widely used as a marker of myocardial wall stretch in hypervolemic state, however, its utility is limited in certain conditions such as end-stage renal disease, pulmonary pathology, etc. when its value is not reflective of true volume status [14]. Other techniques used for hemodynamic assessment include bedside ultrasound measurement of hepatic venous flow [15] and inferior vena cava collapsibility index [16–18] as measures of RAP and CVP, respectively; however, none of these parameters or techniques have shown absolute efficacy in predicting accurate volume assessment, leading to ineffective diuresis resulting in the advancement of heart failure requiring multiple hospitalizations for decompensated states. To obtain more accurate hemodynamic profiles, RHC is often pursued, but as an invasive procedure, this is associated with medical complications (infection, arrhythmias, bleeding, etc.) and financial burdens [19–21]. Attempts have been made in the past to devise a non-invasive alternative to predict volume status that is safe, cost-effective, and reliable.

Previously, our team demonstrated that this bedside ultrasound technique to assess right IJV compliance accurately measured the patients' volume status (RAP) and predicted 30-day readmission for ADHF [11]. Our current study is based on the same principle but expands on this work as follows: We measured RAP continuously via an indwelling PAC. We also obtained serial IJV-CSA readings in the same subjects undergoing active diuresis and our study cohort was comprised of critically ill patients in the CCU with most patients on inotropes and MCS, in contrast to the former study where such patients were excluded. These factors could potentially explain why the cut-point was different between these two studies as some of these inotropes (e.g. milrinone, dopamine etc) have vasodilatory effects on vessels. Moreover, the

sensitivity and specificity of this study improved compared to the prior study (83% vs 77% and 79% vs 75%, respectively), primarily due to the increased prevalence of elevated CVP since our cohort consisted of patients with cardiogenic shock or end-stage heart disease. Our study also proves that this technique can accurately assess RAP in a serial fashion, and the assessment of IJV compliance using portable ultrasound correlates with CVP measurements through indwelling PAC at every time point. Hence, this technique could serve as a rapid, safe, cost-effective, and non-invasive alternative to RHC for tracking volume changes in patients with ADHF undergoing diuresis. Also, by providing accurate volume assessment, this technique could help guide diuresis and discharge planning.

A similar principle of using physiological variation in IJV-CSA to calculate CVP has been applied in several other studies that have yielded comparable results [17,22,23]. Notably, Zamboni et al, also studied IJV-CSA images obtained through the US in conjunction with a simultaneous electrocardiogram (ECG) recording and collaborated them with CVP that was measured manually through triple lumen subclavian catheters. In contrast to our study where IJV-CSA images and CVP were simultaneously collected, Zamboni et al, noticed a lag time between obtaining CSA images of IJV and CVP. Although CVP and IJV-CSA time-series signals were poorly correlated in their study (mean $r = -0.018$, $SD = 0.357$), the autocorrelation counterparts of their real-time data showed highly positive correlation (mean $r = 0.725$, $SD = 0.215$). Through refined regression analysis using the mean IJV-CSA value and selected autocorrelation R-values as predictors, Zamboni et al, predicted mean CVP with decent accuracy ($R^2 = 0.612$ as opposed to our $R^2 = 0.31$). More studies are required to alleviate this discrepancy between the R^2 value and to overcome the knowledge gaps.

Our study includes several limitations. It is a single-center study and thus needs validation in a larger multi-center study. Since indwelling PAC was a pre-requisite for inclusion into the study, the subjects included were a severely sick cohort with either end-stage heart failure or cardiogenic shock and on treatment with inotropic therapy or MCS. Also, many patients also had renal failure and were transitioned to dialysis quickly upon their admission to CCU either because of lack of response to IV diuretics or inability to tolerate bolus diuretics due to low blood pressures necessitating continuous renal replacement therapy and excluding them from the study. A discrepancy between right- and left-sided filling pressures (R-L mismatch) can occur in certain conditions such as severe pulmonary hypertension and has the propensity to confound CVP values [24]. Only a small proportion of the cohort (13.3%) had severe pulmonary hypertension to be analyzed separately, preventing detailed analysis of the cohort; however, their exclusion did not significantly alter the study results, including CSA cut point, suggesting that study results will be applicable to the majority of ADHF patients. Lastly, the study was performed in a small number of patients in a CCU setting with readily available resources; namely, there was bedside ultrasound and personnel to perform multiple/serial ultrasound measurements and calculate CVP based on IJV compliance that, in turn, requires calculation by manually measuring CSA of IJV by uploaded images in Image J, as current portable ultrasound machines lack this feature. Replicating the same techniques on medical units with a disproportionate ratio of patients to providers may raise pragmatic issues.

5. Conclusions

Our current study provides a non-invasive, safe, and rapid technique that can act as a surrogate for RHC. This methodology has promising prospects in terms of reliability, operation, and cost-effectiveness and could potentially serve as a point-of-care daily examination in patients being managed for ADHF to direct diuresis and discharge.

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Disclosures

The authors have no relationship with the industry and have no disclosures.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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