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Evaluation of ultrasonography measurement of superior vena cava diameter in comparison to central venous pressure in guiding of fluid therapy in patients with hypovolemic shock

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Abstract

Invasive placement of a central venous catheter is the norm for measuring central venous pressure (CVP), which is essential in perioperative care (CVC). Clinical evaluation of the jugular venous pressure allows for a non-invasive estimation of CVP. Superior vena cava (SVC) diameter and collapsibility with breathing have been emphasised in echocardiographic estimates of CVP. The goal of this study is to compare CVP values to ultrasound measurements of SVC diameter and collapsibility index in directing fluid treatment for patients with hypovolemic shock. On admission, patients underwent hemodynamic monitoring of intravascular volume by measuring central venous pressure, taking a non-invasive blood pressure reading, and calculating urine output. Additionally, SVC ultrasonography was used to determine the diameter and collapsibility of the saphenous vein. Conclusions Patients with a central venous pressure (CVP) of 10 or higher had substantially greater values for their SVC maximum and minimum, and smaller values for their SVC coefficient of variation (CI). There was a positive association between CVP and maximum and minimum dSVC, and a negative correlation between CVP and SVC-CI. The optimal SVC-CI cutoff for separating patients with and without CVP10 was 36%, with a sensitivity of 27% and a specificity of 87.5 %. Independent predictors of CVP10 were shown to be lower SVC-CI, which aided in the decision to discontinue fluid infusion. Overall, the CVP is still the gold standard for guiding fluid resuscitation in the critically unwell. Estimating CVP non-invasively by measuring SVC diameter and collapsibility during positive pressure breathing seems intriguing. Predicting high CVP using SVC-CI might help you decide when to stop giving the patient fluids.

Key Words: Shock, hypovolemia,CVP, SVC, shock monitoring.

1. Introduction

To put it simply, shock is a potentially fatal consequence of poor circulation. [1]. Distributive, hypovolemic, cardiogenic, and obstructive shock are the primary four types of shock. Shock has been diagnosed, but its pathogenesis, in the case of undifferentiated shock, remains unknown [2]. In patients with hypovolemic shock, severe hypovolemia causes peripheral perfusion to be compromised. Ischemic harm to essential organs, which may lead to multi-system organ failure, can occur in these people if they are not treated [3]. In the impoverished countries, children with diarrhoea are particularly at risk for hypovolemic shock. [4]. Mesenteric and coronary ischemia, which may induce abdomen or chest discomfort, are two potential outcomes of severe hypovolemic shock. [5]. In hypovolemic shock, several different laboratory results may be off. [6]. It is common practise to evaluate fluid balance by measuring central venous pressure (CVP) [5]. The diameter and collapsibility of the inferior vena cava or internal jugular vein may be used as surrogates for CVP [7]. For patients in hemorrhagic shock, early use of blood products over crystalloid resuscitation leads in improved outcomes. Anti-fibrinolytic therapy to individuals with major bleed seems to minimise mortality [8]. The precise fluid deficit cannot be identified for individuals in hypovolemic shock owing to fluid losses. Therefore, it is advised to start with 2 litres of isotonic crystalloid solution infused rapidly as an effort to immediately restore tissue perfusion [9]. A central venous catheter is inserted via the internal jugular vein and guided to the superior vena cava (SVC) just above the right atrium in order to get a reading of the CVP [10]. Researchers concluded that CVP was a subpar indicator of fluid responsiveness. There were also problems in obtaining reliable CVP readings. [11]. Hypovolemia and vasodilation are two conditions that might bring about a drop in CVP. A lower central venous pressure would result from a reduction in venous return, which any of these would accomplish. [12]. Reduced contractility, valve anomalies, and dysrhythmias may all contribute to increase CVP in heart failure. Overly positive PEEP in ventilated patients raises pulmonary arterial resistance, which in turn raises central venous pressure [13]. The CVP's clinical value lies in its ability to evaluate the cardiovascular system [14]. The CVP is a therapeutically appealing, though non-specific, indication of fluid status due to its ease of assessment. Therefore, other indices must be utilised, such as the inferior vena cava collapsibility index (IVC CI), for more precise evaluation of volume status [15]. Clinical measurement of jugular venous pressure or ultrasound evaluation of the inferior vena cava is two indirect methods of determining CVP (IVC). It may be measured immediately with a simple manometer linked to a central venous catheter. Right above the left atrium, the transducer should be positioned on the patient's mid chest, at the mid-axillary line. One typical error in CVP monitoring is failing to take into consideration the influence of positive end expiratory pressure (PEEP) during positive pressure breathing. Preload, afterload,

and ventricular compliance may all be affected by PEEP. Depending on pulmonary compliance and fluctuations in intrathoracic cavity pressure, PEEP may provide artificially high CVP readings by increasing flow resistance [16]. The superior vena cava (SVC) is a frequent choice for a central venous catheter insertion [17]. Inadequate fluid delivery has been linked to an increase in postoperative complications, according to a recent multicenter retrospective research. Thus, excessive fluid infusion may raise the danger of pulmonary and peripheral tissue edoema retarding the recovery of respiratory and intestinal function, while the conservative fluid treatment may cause an unstable hemodynamic profile, multiorgan hypoperfusion, and longer hospital stay [18]. When it comes to directing volume expansion, traditional static hemodynamic indicators like central venous pressure (CVP) and pulmonary capillary wedge pressure have shown to be mostly ineffective. Moreover, the dynamic indices of stroke volume variation (SVV) [19] or pulse pressure variation (PPV)[20] are widely used and accepted as robust indicators to predict preload responsiveness in mechanically ventilated patients, but they require a costly sophisticated device or invasive catheterization to obtain accurate results. With the increased accessibility of ultrasound instruments in perioperative settings, ultrasonography has been advocated for volume evaluation due to its benefit in noninvasiveness, reproducibility, and low learning curve [21]. [21]. SVC measures needed transesophageal echocardiography (TEE) technology, which presently limits its normal clinical use; nonetheless, SVC collapsibility index (SVCCI) and SVC change throughout the cardiac cycle (SVCV) have demonstrated encouraging outcomes in mechanically ventilated patients [22]. With the development of SVC acquisition by transthoracic echocardiography (TTE) method, it is, thus, feasible to estimate volume responsiveness by monitoring SVC fluctuation utilising a noninvasive technique [23]. The central venous pressure (CVP) is a frequently used indicator of the state of the intravascular volume. However, CVP measurements may be impacted by the use of positive pressure ventilation. Recently, the stroke volume variation has been employed as reference during intraoperative anaesthetic management in the operating room [24]. When the intrathoracic pressure is higher than the SVC pressure, the SVC collapses to some degree or entirely during positive pressure ventilation. However, the abdominal segment of the IVC expands in response to an increase in intrathoracic pressure. Critically sick patients' intravascular volume and fluid responsiveness may be estimated by measuring their SVC's collapsibility [25]. The non-invasive assessment of right ventricular systolic pressure (RVSP) relies heavily on an accurate calculation of the CVP, and errors in this estimation might result in either an under- or an overestimation of RVSP. However, there are no available statistics that directly compare SVC indices to CVP. Transthoracic echocardiography has challenges imaging the SVC in adult patients, hence this isn't covered in guideline statements from echocardiography organisations either. The SVC may be clearly seen on transoesophageal echocardiography (TOE) in almost all individuals[26]. Therefore, the purpose of this research is to assess whether or not SVC diameter and collapsibility index values obtained using ultrasound are as useful as CVP measurements in directing fluid treatment for patients with hypovolemic shock.

2. Patients and methods

2.1Study design

An observational cross-sectional study was carried out in the intensive care unit (ICU), Benha University Hospital and approved by The Ethical Committee of Benha University.

2.2Study patients

This study included 100 patients of both sex with hypovolemic shock admitted to the ICU in Benha University Hospital. A written informed consent was taken from patient's relatives. All patients had a functioning central venous catheter inserted and SVC diameter using US was measured.

2.3Inclusion criteria

ASA I–III, ICU patients above 18 years old, non-intubated, non-ventilated with hypovolemic nonhemorrhagic shock (mean arterial BP <65 mmHg and tachycardia (defined as heart rate>100 beats/minute)

2.4Exclusion criteria:

Patients under 18 years, patients with severe orthopnea, morbid obese BMI above 50kg/m2, suspected or diagnosed raised intraabdominal or intrathoracic pressures as known pregnancy, portal hypertension, or mediastinal mass, valvular heart disease , extended cervico facial cellulitis, venous thrombosis, ongoing hemodialysis on an internal jugular vein cathter, intracerebral hemorrhage or increased intracranial pressure, atrial fibrillation.

2.5Patients' examination

All patients were subjected to full assessment, as age, sex, body weight, and height. Mean age of studied cases was 51.2 years, ranged from 19 to 80 years. They were 74 males and 26 females. Full history was reported, past medical history including diseases as diabetes mellitus, hypertension, chronic kidney disease, chronic liver disease, cardiac history, cerebrovascular stroke, history of previous allergy, history of any drug or toxin intake, and in case of traumatized patient, mode and the time of trauma was taken in consideration. Past surgical history including any recent surgeries. Complete clinical examination was done. Measurement of the patient's hemodynamic parameters was evaluated by ABCDE

Measurement of CVP and SVC diameter and CI by ultrasound: as all the readings of SVC diameter and CVP measurements were recorded concomitantly. All ultrasonographic examinations were performed with the patients in supine position by the same physician throughout the study. All the readings were taken by the researcher. As he sought specialized training in use of

bed side ultrasonography by taking POCUS Course. Supine chest radiography and transthoracic echocardiography were done to exclude cardiogenic and obstructive shock.

2.6 Statistical Analysis

The collected data was analysed using Statistical package for Social Science (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). Student T test was used to assess the statistical significance of the difference between two study groups. Correlation analysis was used to assess the strength of association between two quantitative variables. The ROC Curve (receiver operating characteristic) provides a useful way to evaluate the sensitivity and specificity for quantitative diagnostic measures that categorize cases into one of two groups. The optimum cut off point was defined as that which maximized the AUC value. Logistic regression analysis was used for prediction of risk factors. A p is significant if <0.05 at confidence interval 95%.

3. Results

The present study was conducted on 100 cases who were admitted to the ICU with hypovolemic shock. Patients' features are shown in **table 1**. Out of all studied cases, 49% received vasoactive drugs, while 51% did not., mean CVP was 6.9 cm H2O, ranged from 0 to 11 cmH2O; most of cases had CVP less than 10 cmH2O (76%), while only 24% had CVP equal or more than 10 cmH2O. Regarding the SVC diameters , the maximum diameter mean was 1.5 mm, while the minimum diameter mean was 1.1 mm and the SVC Collapsibility index mean was 28.9 and it ranged from 6.7% to 62% **(table 1).** High CVP was significantly associated with higher dSVC max, dSVC min, lower SVC-CI% **(table 2, figure 1).**

CVP showed significant positive correlation with age, BMI, SBP, DBP, MAP, dSCV max, dSVC min, significant negative correlation with HR, and SVC-CI. While, the SVC-CI% showed significant negative correlation with age, SBP, DBP, MAP, dSVC max, dSVC min, significant positive correlation with HR **(table 3, figure 2).**

Receiver operating characteristic (ROC) curve of SVC-CI was conducted for discrimination between patients with CVP<10 and patients with CVP≥10. The best cut-off point of the SVC-CI was 36.8% with (Sensitivity 27.6% and Specificity 87.5%). **Table 4** shows various sensitivities and specificities of different cut off values.

Logistic regression analysis was conducted to predict factors affecting CVP, using age, gender, BMI, SBP, DBP, MAP, HR and SVC-CI as covariates. Higher SBP, DBP, MAP, lower HR, SVC-CI were associated with prediction of high CVP in univariable analysis. While in multivariable analysis, only lower HR, SVC-CI were considered independent predictors for CVP≥10, in order to make decision to stop fluid infusion **(table 5).**

Table (1) Vital signs, CVP, SVC assessment of all studied cases.

		Cases N=100
SBP (mmHg)	mean±SD (Minimum-maximum)	98.4±16.3 (60-132)
DBP (mmHg)	mean±SD (Minimum-maximum)	54.5±10.5 (37-75)
MAP (mmHg)	mean±SD (Minimum-maximum)	68.7±11.7 (46-95)
Pulse rate (bpm)	mean±SD (Minimum-maximum)	116.1±34.5 (66-183)
CVP (cm H_2O)	mean±SD (Minimum-maximum)	6.9±2.3 (0-11)
CVP<10	N, %	76(76%)
CVP≥10	N, %	24(24%)
dSVC max (mm)	mean±SD (Minimum-maximum)	1.5±0.6 (0.5-2.60)
dSVC min (mm)	mean±SD (Minimum-maximum)	1.1±0.5 (0.2-2.40)
SVC-CI (%)	mean±SD (Minimum-maximum)	28.9±13.1 (7.6%-62%)

Table (2) Comparison of SVC among studied cases according to CVP.

		CVP<10	CVP≥10	*p*
		N=76	**N=24**	
dSVC max	mean±SD (Minimum-maximum)	1.3±0.4 (0.5-2.3)	2.2±0.3 (1.7-2.6)	**<0.001**
dSVC min	mean±SD (Minimum-maximum)	0.9±0.4 (0.2-1.7)	1.6±0.3 (1.1-2.4)	**<0.001**
SVC-CI%	mean±SD (Minimum-maximum)	29.8%±13.9% (9%-62%)	26.3%±10% (7.6%-45%)	**0.025**

Table (3) Correlation of CVP and SVC-CI % with other studied parameters.

	CVP		**SVC-CI%**	
	coefficient	***p***	***coefficient***	***p***
Age	0.324	**0.001**	-0.232	**0.020**
BMI	0.273	**0.006**	-0.009	0.927
SBP	0.804	**<0.001**	-0.366	**<0.001**
DBP	0.830	**<0.001**	-0.309	**0.002**
MAP	0.883	**<0.001**	-0.356	**<0.001**
HR	-0.927	**<0.001**	0.402	**<0.001**
dSVC max	0.891	**<0.001**	-0.377	**<0.001**
dSVC min	0.848	**<0.001**	-0.640	**<0.001**
SVC-CI%	-0.431	**<0.001**	-	-

Table (4) Validity of SVC-CI for prediction of CVP ≥10.

SVC-CI	**Sensitivity**	**Specificity**
27.8%	50.0	54.2
28.8%	44.7	54.2
29.5%	43.4	54.2
30.4%	38.2	58.3
30.9%	38.2	66.7
31.3%	36.8	66.7
32.3%	36.8	70.8
34.0%	30.3	83.3
35.5%	28.9	83.3
36.8%	27.6	87.5
37.8%	26.3	87.5
39.0%	25.0	87.5
40.5%	21.1	91.7
41.9%	19.7	91.7
42.9%	18.4	91.7
44.0%	18.4	95.8
45.5%	14.5	100

Table (5) Regression analysis for prediction of CVP≥10 in order to stop fluid infusion.

	Univariable		**Multivariable**	
	p	***OR(95% CI)***	***p***	***OR(95% CI)***
Age	0.133	1.018(0.9950.995-1.042)		
Gender	0.226	0.665(0.3440.344-1.287)		
BMI	0.066	1.067(0.9960.996-1.144)		
SBP	**<0.001**	1.064(1.0381.038-1.092)	0.536	1.031(0.9350.935-1.138)
DBP	**<0.001**	1.144(1.0821.082-1.21)	0.488	1.072(0.8810.881-1.305)
MAP	**<0.001**	1.123(1.0741.074-1.174)	0.835	0.972(0.7420.742-1.272)
HR	**<0.001**	0.935(0.9040.904-0.967)	**0.006**	0.942(0.9030.903-0.983)
SVC-CI%	**0.023**	0.987(0.9650.965-0.998)	**0.045**	0.867(0.5980.598-0.945)

OR, odds ratio; CI, confidence interval.

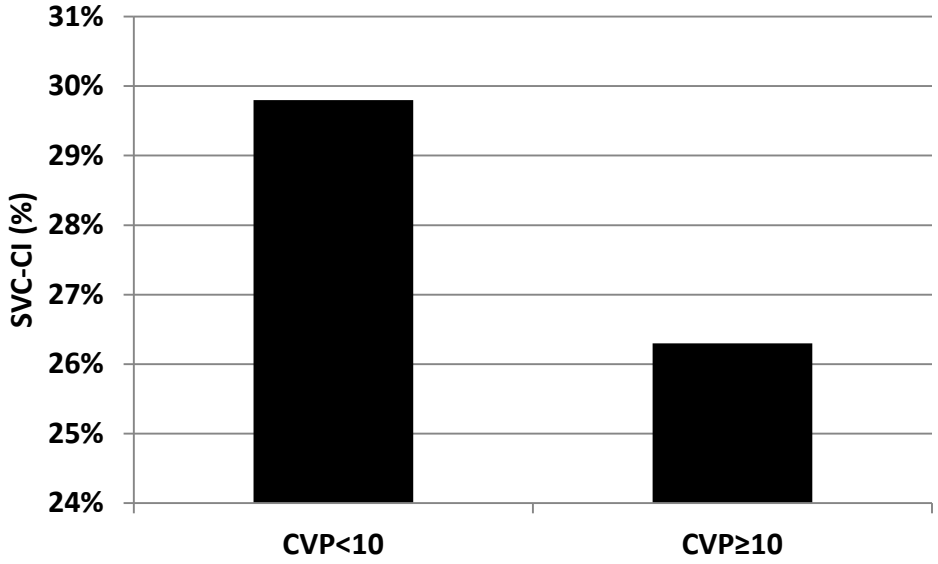

Fig. (1) The SVC CI among studied cases according to CVP.

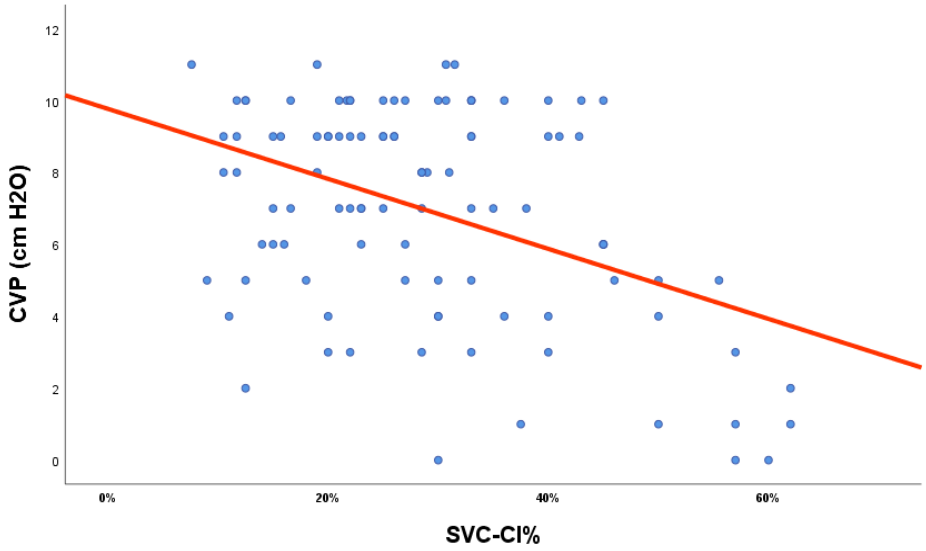

Fig. (2) Correlation of CVP with SVC CI.

4. Discussion

When intravascular volume decreases to an unsafe level, a condition known as hypovolemic shock sets place [3]. When comparing our results to those of Cowie et al., who found that the median CVP in their sample was 10 mmHg with a range of 2 to 19 mmHg, we find that the mean CVP in our sample was 6.9 cm H2O, with a range of 0 to 11 cm H2O; most cases had CVP less than 10 cmH2O (76 percent), and only 24 percent had CVP equal to or greater than 10 cm H2O. (27). Patients with high CVP were shown to have considerably greater SBP, DBP, MAP, and significantly lower HR than those with normal CVP. In all instances where the CVP was 10 or greater, vasoactive medications were administered. Among patients requiring mechanical breathing, Rahim-Taleghani et al. discovered a positive association between HCO3 and CVP and a negative correlation between pH and anion gap. CVP was negatively correlated with just pH among individuals who were not receiving mechanical breathing [28]. The current investigation found that elevated CVP in patients was substantially linked to increased SVC maximum and minimum and decreased SVC CI. Cowie et al. discovered a small but statistically significant relationship between CVP and the collapsibility index of the superior vena cava [27]. CVP was shown to have a positive relationship with age, BMI, SBP, DBP, MAP, dSCV max, dSCV min, and a negative relationship with HR and SVC-CI in the current research. According to Cowie et al., there is no correlation between SVC diameter and CVP [27]. According to our findings, there was a positive relationship between SVC-CI percent and HR and a negative relationship between age, SBP, DBP, MAP, dSVC max, and dSVC min. Prior to VE, the collapsibility of the SVC varied from 0% to 100%, was

only marginally connected with the greatest diameter of the SVC, and was unrelated to CVP. The greatest diameter of the superior vena cava (SVC) was not correlated with central venous pressure (CVP), similar to what Cowie et al. There was a very significant relationship between maximum SVC diameter and body mass index. CVP did not correlate with either age or body size in a way that could be considered statistically significant [27]. The optimal cutoff value of SVC diameter to predict fluid responsiveness was found to be 29% by Charbonneau et al., with a sensitivity of 54% and a specificity of 94%. SVC was shown to have a weak relationship with CI. To distinguish between respondents and non-respondents, an SVC >36% has a 42% sensitivity and a 100% specificity [29]. SVC-CI was tested using a receiver operating characteristic curve to differentiate between CVP10 and CVP10 patients. While Shalaby et al. discovered a substantial association between CVP and the two ultrasonography measures (IVC CI and IVCdmax) investigated, the optimal cut-off point for the SVC-CI was 36.8% (Sensitivity 27.6% and Specificity 87.5%). When comparing the two ultrasonography parameters for predicting CVP 10 cm H2O, others have discovered that the inferior vena cava collapsibility index (IVC CI) had the better performance. Predicting fluid responsiveness using the IVC collapsibility index and the IVC diameter was shown to have a higher diagnostic accuracy [30]. To predict parameters impacting CVP, age, gender, BMI, SBP, DBP, MAP, HR, and SVC-CI were used in a logistic regression analysis. Fluid infusion was stopped whenever CVP reached a value12 cm. Prediction of CVP equal to or greater than 10 was linked with higher SBP, DBP, MAP, lower HR, and SVC-CI in univariate analysis. Multivariate analysis only included lower HR and SVC-CI as predictors for CVP10 when deciding whether or not to discontinue fluid infusion. Ultrasound SVC measures were shown to be predictive predictors of fluid responsiveness, with the minimum SVC diameter being somewhat more effective than the SVC variation and the maximum SVC diameter, as discovered by Cheng and colleagues. A minimum SVC diameter of 1.135 cm was found to be the most sensitive and specific cutoff value, respectively (87.2% and 88.0%). Further, the smallest SVC diameter had an AUC of 0.929. After implementing volume increases, the SVC fluctuation was greatly reduced. Conversely, there was no discernible change in CVP either before or after the fluid challenge [31]. It may be concluded that the respiratory variation of SVC is preferable than dIVC and SVCV in predicting volume responsiveness since SVC-CI showed a higher correlation coefficient with cardiac output, a bigger AUC, and a smaller grey zone [32]. Our findings should be interpreted with caution due to the study's limitations, which include its observational character and the paucity of analogous research that compare measures of SVC diameter and collapsibility index with CVP. It is also suggested that future research include data collection both before and after fluid infusion.

According to the results, CVP is still the most often utilised variable to direct fluid resuscitation in critically sick patients. Estimating CVP non-invasively by measuring SVC diameter and collapsibility during positive pressure breathing seems intriguing. The SVC-CI may be utilised as a predictor for the CVP.

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