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Original Article



**Ultrasound Guided Measurement of Inferior Vena Cava Diameter, Common Carotid Artery Diameter versus Central Venous Pressure for Estimation of Intravascular Volume Status in Septic Shock Patients**

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# **ABSTRACT**

**Background:** In critically ill patients, the fluid resuscitation is crucial and is the first step in treatment protocol. Invasive CVP measurement was used to guide fluid resuscitation. However, it is time consuming and invasive maneuver. Ultrasound measurement of inferior vena cave and common carotid arteries are suggest to predict the fluid response as well as CVP or even better. The current work designed to compare inferior vena cava diameter and common carotid artery diameter with central venous pressure for estimation of intravascular volume status in septic shock.

Patients and Methods: The study included 60 subjects with septic shock who received vasopressor support. CVP and ultrasound were performed before and after fluid challenge test. Values of inferior vena cava and common carotid arteries were documented and compared to CVP values. This was performed after full clinical assessment by detailed clinical examination and laboratory workup.

**Results:** The MAP was significantly increased and HR decreased after than before procedure. In addition, CVP, (inferior vena cava), ICVmax, ICV min and common carotid artery diameter (CCAD) were significantly increased, IVC CI (%) was significantly reduced after procedure than basal values. There was significant decrease of IVC<sub>max</sub>, IVC<sub>min</sub> and CCAD while there was significant increase of IVC CI% with lower  $(\leq 8)$  than higher  $(> 8)$  CVP values. CVP was positively and significantly correlated with IVC<sub>max</sub>, IVC<sub>min</sub> and CCAD, while it was inversely correlated with IVC CI%. The AUC was over 0.75 for IVCmax, IVCmin and CCAD for prediction of CVP while it was lower than 0.7 for IVC CI%. These data reflected the better predictive power of IVCmax, IVCmin and CCAD. The best cutoff value was 1.35, 1.25, 10.45 and 4.15 for IVCmax, IVCmin, IVC CI% and CCAD successively.

**Conclusion:** IVC and CCA diameters measurement by US may replace CVP measurement for estimation of intravascular volume status in septic shock patients.

**Keywords:** Septic Shock; Fluid responsiveness; Challenge test; Ultrasound; Central venous pressure.

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#### **INTRODUCTION**

In critically ill patients, the determination of intravascular volume (IVV) is of utmost importance to accurately diagnose the status of shock. This could be challenging due to overlapping of clinical manifestations of hypovolemia, cardiogenic shock and obstructive shock **(1-3)**. However, the determination of the IVV in the critically ill patients or those with trauma can be extremely difficult. This can affect the treatment plan as the fluid loading is the initial resuscitation step of hemodynamically unstable patients **(4,5)** .

Both under-resuscitation with inadequate perfusion and over-resuscitation increase the morbidity and mortality in critically ill patients who were admitted to the intensive care unit (ICU). Traditionally, the fluid management can be guided by the cardiac filling pressures (central venous pressure (CVP) and pulmonary artery occlusion pressure) **(6,7)**. The CVP had been used for guiding of fluid management for decades. However, it is not devoid of complications (e.g., cardiac injury, arrhythmias, injury of the vascular nerve, pneumothorax, hemothorax, bleeding locally, hematoma, infection, thrombus formation, pulmonary embolism and postphlebitis syndrome **(8,9)**. In addition, CVP as an accurate measure of volume status has been recently challenged. There was poor relation between CVP and blood volume. In addition, it is difficult to use to guide clinical fluid management decision **(8)** .

Alternatively, the measurement of inferior vena cava (IVC) or common carotid artery diameters by ultrasound has been used to assess the volume status **(10)**. During inspiration, there was collapsibility of IVC, and distension occurred during expiration. This can reflect volume status by ultrasound diameter determination **(11, 12)**. Thus, ultrasound measurement of IVC diameter is considered a non-invasive, readily available method to provide a rapid guiding of fluid resuscitation **(13)**. Common carotid arteries (CCA) provides blood supply to the brain (internal arteries) and head and neck (external arteries). The average diameter of CCA is 6.5 and 6.1 mm in adult men and women respectively **(14,15)**. CCA reacts to IVV expansion with marked dilation in fluid demanding patients **(16)** .

The objective of this study was to compare inferior vena cava diameter and common carotid artery diameter with central venous pressure for estimation of intravascular volume status in septic shock.

## **PATIENTS AND METHODS**

This was a prospective cross sectional trial. It was conducted at Al-Azhar University Hospitals and approved by the local research and ethics committee of Al-Azhar faculty of medicine. Sixty patients with septic shock were included from the critical care unit.

We included patients between 20 and 60 years of age, either men or women, who had septic shock and on norepinephrine support less than 1.5 mcg/kg/min with spontaneous breathing. But, we excluded patients who refused to share in the study or uncooperative. Patients with other types of shock than septic shock were also excluded. In addition, we excluded patients with valvular heart disease, cardiomyopathy, pulmonary hypertension, increased intra-abdominal pressure, who had surgery for carotid artery, those with advanced liver disease, renal failure, obesity or on mechanical ventilation (MV).

To determine the sample size, the study of **Badry et al. (10)** was used as a guide. The Epi-Info STATCALC was used to calculate the sample size, taking into account 95% CI (two sided), 80% power and error margin of 5% and calculated odds ratio of 1.115. The calculation produced a 56 as a sample size. Taking into account the dropout rate, the sample increased to 60. However, all completed the study.

All subjects were clinically evaluated by complete history taking and physical examination. The general clinical examination concentrated on the vital signs and complexion. The body mass index was calculated after the measurement of weight and height, from the equation: BMI= weight/squared height and obesity was decided at BMI 30 kg/m<sup>2</sup> or above. The routine laboratory work-up consisted of complete blood count, renal function tests, liver function tests (enzymes, albumin, bilirubin, gammaglutamyl transferase (GGT), prothrombin time and international normalized ratio (INR), ESR, CRP and procalcitonin.

The intravascular volume status was assessed before and after fluid challenge test by 500 ml crystalloid administration within  $20 - 30$  minutes. The ultrasound study was performed using a SonoSite M-Turbo Model M-MSK equipped with a 13–6-MHz Transducer. A cardiac probe (1-5 MHz, phased array) was used for IVC imaging. All ultrasound examinations were performed by single experienced sonographer, blinded to CVP measurement. The examination was performed while the patient was in the supine position.

The IVC was measured in the Subcostal Inferior Vena Cava (SIVC) view. The IVC was first recognized in a transverse plane **(Figure 1).** The probe was set in a subxiphoid position perpendicular to the skin. Then it was moved to the right to visualize the IVC in the centre of the field. After that, the probe was rotated by 90° to obtain a longitudinal plane **(Figure 2)**. In this plane, IVC was seen on the longitudinal axis draining into the right atrium. In addition, the right hepatic vein, the last tributary to join the IVC intra-abdominally was visualized in this view.

The IVC was confirmed in the PWM. Then, the Mmode was used to visualize the plane just distal to the right hepatic vein, nearly 2-3 cm from the junction between the right atrium with IVC. This was performed to be sure of the intra-abdominal measurement of the IVC calibre and to avoid antra-thoracic region as a standard. The M-mode was used to capture a 10-s cine loop of the IVC over two or three respiratory cycles.

Then the maximum diameter of IVC  $(D_{\text{max}})$  and the minimum diameter of IVC  $(D_{min})$  were recorded. Three measurements were performed and the average was entered for statistical analysis.. Then caval index (dIVC) was calculated from the equation:  $dIVC = [(D<sub>max</sub> - D<sub>min</sub>) /$  $((D_{max} + D_{min})/2)] \times 100\%$ . The dIVC is expressed as a percentage (extending from 0 to 100%). The 0% spectrum indicates minimal IVC collapse (i.e., volume overload) and the other end of the spectrum is 100% which indicates almost complete IVC collapse (i.e., volume depletion).



Figure (2): Identification of IVC in longitudinal view.

A 12 MHz liner probe was used to visualize the CCA. It was performed by a short axis view, which was perpendicular to the skin in the transverse plane, on the neck lateral to the cricoid cartilage on the other side of the CVP line insertion. The patient then positioned supine with the head of the bed  $30^{\circ}$  elevated in the AP (anteroposterior) CCAD. The CCAd was measured in mm from intimal-to- intimal edge using the frozen B-Mode image at the end of expiration. The transducer position was marked on the skin and each measure was performed 3 times and the average was documented (Figure 3).

The corrected CFT was calculated form the equation:  $CFT$  = systole time/cycle time. The systole time was measured from the start of systolic upstroke to the start of the dicrotic notch. The cycle time was defined as the duration of the cycle CBF. It was calculated as blood flow  $=\pi \times$  (carotid diameter)  $2/4 \times VTI \times$  Heart rate, where VTI specifies the velocity time integral. VTI of the Doppler

signal was measured using manual tracings. The carotid diameter was measured at the level of the sample gate (Figure 4).

CVP was measured in all patients while they are in the supine position in cmH2O using water- manometer at the end of expiration. The CVP was measured and documented by the ICU residents blinded to the study protocol and the sonographers were unaware to the CVP readings (Figure 5).

The primary outcome was the comparison between IVC diameter and common CCAD with CVP for estimation of IVV status in septic shock patients regarding facility, efficacy, and invasiveness. The secondary outcomes were the use of IVC collapsibility index for estimation of IVV status as non-invasive monitoring in septic shock patients.



Figure (3): Short-axis view of the right CCA; (a) In the middle third of the neck, (b) In the distal third of the neck. scm: sternocleidomastoid muscle. jv: internal jugular vein. tir: thyroid right lobe. cca: CCA. sc. subclavian artery. ia: innominate artery



Figure (4): Corrected Carotid Flow Time; Carotid systole time (measured from the start of the systolic upstroke to the start of the dicrotic notch (left)). Carotid cycle time (measured from start of one systolic upstroke to the next (right)). Velocity time integral tracing of the spectral Doppler signal (left), carotid diameter (right).



Figure (5): CVP Measurement

### **Statistical Analysis:**

The collected data were coded and fed to the statistical package for social sciences software package for windows, version 20.0 (IBM Inc., Chicago, Armonk, USA). The arithmetic mean (measure of central tendency) and standard deviation (SD) were measured to express the continuous normally distributed data. On the other side, the relative frequencies and percentages were used to express the categorical variables. Independent-samples "*t*"-test was used to test significant differences between two means. The Mann Whitney "U" test was used for twogroup comparisons in non-parametric data. The Chisquare  $(x^2)$  test was used to test associations between qualitative parameters. The Pearson's correlation coefficient (*r*) test was used to assess the correlation between two sets of variables. Receiver operating characteristic (ROC) curve was built to estimate the overall predictive power and best cut-off value, sensitivity

and specificity at this cut-off value of studied parameters.

### **RESULTS**

In the current work there were 32 males and 28 females. Their age ranged between 20 and 60 years. All had septic shock and all received vasopressor support in the form of norepinephrine (table 1). Table (2) presented the hemodynamics and ultrasonography measurements before and after fluid administration. These results reveal that, the MAP significantly increased and HR significantly decreased after procedure than their corresponding values before procedure. In addition, CVP, ICVmax, ICV min and CCAD were significantly increased after the procedure, while IVC CI (%) was significantly reduced after the procedure than corresponding basal values.

Comparing cases with CVP more than 8 to those with values lower than 8 revealed that, there was significant decrease of IVCmax, IVCmin and CCAD while there was significant increase of IVC CI% with lower than higher CVP values (Table 3). CVP was positively and significantly correlated with IVCmax, IVCmin and CCAD, while it was inversely correlated with IVC CI%. The correlation was powerful for all variables (Table 4).

The area under the curve (AUC) was over 0.75 for IVCmax, IVCmin and CCAD for prediction of CVP while it was lower than 0.7 for IVC CI%. These data reflected the better predictive power of IVC<sub>max</sub>, IVC<sub>min</sub> and CCAD. The best cutoff value was 1.35, 1.25, 10.45 and 4.15 for IVC<sub>max</sub>, IVC<sub>min</sub>, IVC CI% and CCAD successively (Table 5 and figures 6 through 9).



**Table (1):** Demographic data among the study population

Table (2): Hemodyanmics and ultrasonography measurements before and after fluid administration

<b>Variable</b>	<b>Pre-procedural</b>	Post-procedural	<b>Test (paired)</b>	P value
$MAP$ (mmHg)	$53.75 + 3.05$	$62.02 + 3.02$	14.923	$< 0.001*$
HR (beat/minute)	$125.7 \pm 8.34$	$95.32 + 8.28$	20.03	$< 0.001*$
$CVP$ (cmH <sub>2</sub> O)	$1.97 + 0.74$	$6.86 + 1.44$	23.425	$< 0.001*$
$IVC$ max $(cm)$	$0.98 \pm 0.1$	$1.31 \pm 0.27$	8.859	$< 0.001*$
$IVC \text{ min (cm)}$	$0.79 \pm 0.1$	$1.18 \pm 0.23$	12.044	$< 0.001*$
IVC CI $\left(\frac{9}{6}\right)$	$14.44 \pm 3.65$	$11.34 \pm 3.35$	4.836	$< 0.001*$
$CCAD$ (mm)	$2.83 + 0.34$	$3.82 + 0.84$	8.431	$< 0.001*$

Table (3): Relation between Post-procedural CVP (cmH2O) and ultrasonography parameters among the study population



Table (4): Correlation between CVP (cmH<sub>2</sub>O) and ultrasonography parameters among the study population



Table (5): Accuracy of ultrasonography parameters to predict CVP (cmH2O) among the study population.





# **DISCUSSION**

Sepsis is a life-threatening condition due to multi-organ dysfunction with a deregulated response to infection. This can results grave complications (e.g., septic shock). Septic shock is a subset of infection with circularity and cellular/metabolic dysfunction with a higher risk of mortality. The first step in treatment is fluid resuscitation to improve blood pressure **(17)** . When the cardiac preload in the low volume status, the fluid challenge will lean to an increase in the stroke volume (SV). In addition, it determines the optimal amount of next fluids. It is a crucial issue during the continued inquiry **(18)** .

The hemodynamic support in shock is of utmost importance to prevent deterioration of organ function. It must be started immediately during the search of the cause is ongoing **(19)** . Traditionally, the invasive intravascular volume assessment modalities are used (e.g., CVP and pulmonary artery pressure) to provide data about cardiac output and right atrial pressure. However, these means are time-consuming and carry and significant risks or complications **(20,21)** .

The current work aimed at comparing IVC and CCA diameters with CVP for estimation of intravascular volume status in a septic shock.

In the current work, by inclusion criteria, all patients had septic shock and all receive vasopressors in the form of norepinephrine. However, this is different than those reported by **Badry** *et al***. (10)** who reported vasopressor use for 21.8% of patients. **Schefold** *et al***. (22)** reported vasopressor use for 83% of patients. As well, **Hilbert** *et al.* **(16)** reported the percentage to be 80%.

In the current work, there was significant improvement of hemodynamic after procedure than corresponding values before procedure. These results agree with **Badry** *et al***. (10)** who reported that the MAP was increased significantly at 30 min, 1hr, 2hrs, 3hrs, 6hrs, 12hrs, 24hrs, and 48hrs as compared to base line value. In addition, **Rashwan** *et al.*  $^{(23)}$  reported that the MAP (mmHg) was 56 ±5.9 on admission and significantly increased to 63.2 after fluid administration. As well, **Hilbert** *et al***. (16)** reported that there was significant increase of MAP after than before fluid administration. Furthermore results are consistent with **Khan** *et al.* <sup>(24)</sup>.

Regarding heart rate, our results are in accordance with **Badry** *et al***. (10)** who reported that the HR was significantly decreased at 30 min, 1hr, 2hrs, 3hrs, 6hrs, 12hrs, 24hrs, and 48hrs as compared to base line value. In addition, **Rashwan** *et al***. (23)** reported that the heart rate on admission was  $105\pm 8$ , and significantly reduced to  $84\pm 7$  after fluid administration.

Our results regarding CVP are in line with **Badry** et al. <sup>(10)</sup> who reported that serial CVP showed a gradual increase from baseline value of  $2 \pm 0.8$  cmH<sub>2</sub>O to  $11.2 \pm 2.4$  cmH<sub>2</sub>O. As well, **Rashwan** *et al***. (23)** reported that the mean CVP was 3.71.7 (cmH2O) on admission and markedly increased to  $8.5 \pm 1.4$  (cmH<sub>2</sub>O) after fluid administration. On the other hand, our results in contrast with **Soliman** *et al***. (25)** who reported that CVP did not have significantly changed

before and after fluid boluses. This may be explained by different inclusion and exclusion criteria.

Regarding ultrasound results, our results supported by **Badry** *et al.* **(10)** who reported that the mean values of IVC maximum (IVC max) and IVC minimum (IVC min) diameters were increased significantly at different times after fluid administration as compared to base line value. However, our results disagree with **Soliman** *et al***. (25)** who reported that respiratory variation of (IVC-d) showed no significant change before and after fluid challenge. Again, this could be attributed to different inclusion and exclusion criteria.

Regarding CCAD (mm) among the study population, our results matched with **Badry** *et al***. (10)** who reported that the baseline value of CCAD was 2.9±0.4mm and this value increased significantly at all times after fluid administration till it reached to 5.1±0.7mm. As well, our results are in line with **Rashwan** *et al***. (23)** who reported that the mean diastolic CCA diameter was 5.40.6(mm) on admission and significantly increased from to 6.60.5 (mm) after fluid administration. Also, our results supported by **Hilbert** *et al***. (16)** . However, our results disagree with **Khan**  $et$   $al.$  <sup>(24)</sup> who reported that there was no significant difference between before and after volume expansion in the responder group regarding carotid artery blood flow.

Regarding the relation between post-procedural CVP (cmH2O) and ultrasonography parameters, Our results supported by **Badry** *et al***. (10)** who reported significant changes between group with values lower than 8 and those above 8 regarding all ultrasonography parameters. Also, **Nagdev** *et al.* **(26)** aimed to determine whether a greater than or equal to 50% decrease in IVC diameter was associated with a CVP of less than 8 among adult emergency department patients undergoing central venous catheterization. They reported that IVC CI in Population with CVP  $>8$  ranged from 16.3 -24.9 while in Population with CVP  $<8$  the IVC max (cm) ranged from 59.1 to 77.1. Furthermore, **Ilyas (27)** reported that IVC max, IVC min, and IVC-collapsibility Index were found to have better results in identifying CVP >10.

Our results showed that significant correlation between CVP (cmH2O) and all ultrasonography measurements. This was in line with **Mahrous** *et al***. (13)** who reported that CVP and IVC-CI had a statistically significant correlation. Also, our results in line with **Maghraby** *et al.* **(28)** who reported that CVP demonstrated a significantly positive correlation with IVC diameter in expiration and inspiration but the correlation with IVC-C was inverse. Furthermore, **Khalil** *et al.* <sup>(29)</sup> who aimed to assess the IVV status in critically ill patients using IVC diameter and correlating with CVP. They reported that correlation CVP and max IVC diameter were correlated. The correlation was moderate and significant ( $r = 0.53$ ,  $p < 0.001$ ). The CVP and min IVC diameter correlation was also moderate and significant ( $r = 0.58$ ,  $p < 0.001$ ). As well, our results in line with **Rashwan** *et al.*  $^{(23)}$  who reported that the correlation between CCA diameter before fluid administration had a significant strong proportional correlation with CVP (P <  $0.001$ ,  $r = 0.8$ ). The increased diameter of CCA after fluid administration showed a moderate positive correlation

with the increase in the CVP ( $P < 0.001$ ,  $r = 0.4$ ). Also, our results matched with **Thanakitcharu** *et al.* **(30)** who aimed to conduct a prospective, cross-sectional study to evaluate the IVC diameter as a guidance for estimating the volume status in critically-ill patients by bedside ultrasonography, focusing on correlations between CVP and IVC-CI and IVC diameter, they reported that The highest significant correlation was found between the CVP and IVC-CI ( $r = -$ 0.612,  $p < 0.001$ ). Furthermore our results agreed with **Ilyas (27)** who reported that the IVC Collapsibility Index demonstrated a strong negative correlation with invasive CVP, indicating that the IVC Collapsibility Index's value decreases as CVP rises. On the other hand, our results disagree with **Govender** *et al***. (31)** who aimed to determine if is there an association between CVP measurement and US assessment of the IVC, they reported that there was a weak negative correlation between CVP and IVC-CI, which was not statistically significant for all participant.

Regarding predictive power of ultrasonography measurements, out results are in line with **Ismail** *et al***. (8)** who aimed to provide accurate measurement of IVV status using a non-invasive methods in shocked patients. They showed that, IVC-ci at 40 as a cut-off point yielded a 93.3% and 70.7% of sensitivity and specificity, respectively. The area under the curve (AUC) was 0.908 reflecting a high accuracy of the test. This indicated that the IVC-ci of 40% or higher indicate fluid responsiveness in shocked patients. However, CVP was not considered a reliable indicator of fluid responsiveness irrespective of its good sensitivity and specificity (88.6% and 100.0%, respectively). This was due to small AUC and low 95% confidence interval.

**Nagdev** *et al***. (26)** reported that the sensitivity and specificity of IVC diameter to determine CVP <10 cmH<sub>2</sub>O were 77% and 91%, and 90% and 89% for CVP>10 cmH2O, respectively. **Schefold** *et al***. (22)** showed that IVC diameter  $<$  2 cm estimated a CVP of  $<$  10 mmHg with a sensitivity of 85%, specificity of 81%, and positive predictive value (PPV) of 87% (95% confidence interval (CI) 71% to 95%). Moreover**, Stawicki** *et al***. (32)** reported that the sensitivity of caval index (IVC CI) greater than 50% to predict the CVP less than 8 mmHg was 91%, and the specificity was 94%. **Marik** *et al***. (33)** used the carotid artery Doppler US to evaluate the hemodynamically unstable patients and reported a significant increase in the CCA diameter in the fluid responders. **Bapat** *et al***. (34)** reported an increase in the brachial artery diameter in response to volume loading by a passive leg raising (PLR) method. The CCA diameter before fluid administration showed a significant strong positive correlation with CVP  $(P < 0.001, r = 0.8)$  and the increase in the CCA diameter after fluid loading had a significant moderate positive correlation with the increase in the CVP ( $P < 0.00$ ,  $r = 0.4$ ).

**Conclusion:** IVC and CCA diameters measurement by US may replace CVP measurement for estimation of intravascular volume status in septic shock patients. Also, caval index can be used to predict fluid responsiveness.

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