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Inferior Vena Cava Collapsibility versus Central Venous Pressure as a Guide of Fluid Management in Septic Patients

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Abstract: Objective: Fluid infusion, the most critical step in the resuscitation of patients with septic shock, needs preferably continuous invasive hemodynamic monitoring. The study was planned to evaluate the efficacy of ultrasonographically measured inferior vena cava collapsibility index (IVC CI) in comparison to central venous pressure (CVP) in predicting fluid responsiveness in septic shock. Materials and Methods: Thirty-six patients of septic shock requiring ventilatory support (invasive/noninvasive) were included. Patients with congestive heart failure, raised intra-abdominal pressure, and poor echo window were excluded from the study. They were randomly divided into two groups based on mode of fluid resuscitation – Group I (CVP) and Group II (IVC CI). Primary end-points were mean arterial pressure (MAP) of ≥65 mmHg and CVP >12 mmHg or IVC CI <20% in Groups I and II, respectively. Patients were followed till achievement of end-points or maximum of 6 h. Outcome variables (pulse rate, MAP, urine output, pH, base deficit, and ScvO2) were serially measured till the end of the study. Survival at 2 and 4 weeks was used as secondary end-point. **Results:** Primary end-point was reached in 31 patients (15 in Group I and 16 in Group II). Fluid infusion, by either method, had increased CVP and decreased IVC CI with resultant negative correlation between them (Pearson correlation coefficient –0.626). There was no significant difference in the amount of fluid infused and time to reach end-point in two groups. Comparison in outcome variables at baseline and end-point showed no significant difference including mortality. **Conclusion:** CVP and IVC CI are negatively correlated with fluid resuscitation, and both methods can be used for resuscitation, with IVC CI being noninferior to CVP.

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Key Words: Central venous pressure, fluid responsiveness, hypovolemia, septic shock, sonographic inferior vena cava variability

1. Introduction:

Undoubtedly, one of the most important principles of treating patients with shock since long time is intravenous fluids. Surprisingly, dosing intravenous fluid during resuscitation of shock remains largely empirical. Too little fluid can lead to organ hypo-perfusion and progress to organ dysfunction. However, over-dosing of fluid will impede oxygen delivery and compromise patient outcome. Recent data suggest that early aggressive resuscitation of critically ill patients may limit and/or reverse tissue hypoxia, progression to organ failure, and improve outcome. [(1)] Protocol of early goal-directed therapy reduces organ failure and improves survival in patients with septic shock [(2)].

Central venous pressure (CVP) monitoring is a mainstay of estimating intravascular fluid status and cardiac preload in critically ill and injured patients [(3)] As bedside ultrasound is increasingly available and incorporated into Critical Care, a number of protocols have been developed for the evaluation of patients in shock, respiratory distress, and cardiac arrest, for example the Bedside Lung Ultrasound in Emergency (BLUE) protocol focuses solely on lung ultrasound for the diagnosis of pneumothorax, pulmonary edema, pulmonary consolidation, and effusions [(4)].

Many studies were performed to evaluate the correlation between IVC diameter and CVP. Many of these studies have been used complex imaging and measuring techniques such as formal trans-esophageal echocardiography and caval indices. Most of these studies were based physiologically on frank-starling low which stated that within limit increase of preload will raise cardiac output even in compromised heart [(5)].

Recently, all guidelines in fluid management and perfusion protocols suggest using of no single

parameter or technique to evaluate fluid responsiveness and perfusion. As no single test has near 100% specificity or sensitivity, so combination of different clinical and laboratory data can help good assessment and will guide fluid management safely and with more accurate decisions. Many clinical parameters can be used to guide perfusion as; mean arterial pressure, cerebral and abdominal perfusion pressures, urine output, mentation, capillary refill, skin perfusion/mottling, cold extremities (and cold knees), blood lactate, arterial PH, BE, and HCO_3, mixed venous oxygen saturation ($SmvO_2$), mixed venous Pco_2, tissue Pco_2, skeletal muscle tissue oxygenation (StO2) [6].

2. Materials and Methods:

This prospective, randomized observational study was carried out in the Intensive Care Unit (ICU) of Abu Qir specialized hospital. Selection of patients was done from the emergency department, medical and surgical wards. Fifty patients with septic shock and fulfilled inclusion and exclusion criteria during 1-year period from first July 01, 2015, to June 30, 2016, were enrolled in the study [Figure 1]. The criteria for inclusion were fulfillment of two out of four criteria for systemic inflammatory response syndrome, probable or suspected septic etiology, systolic blood pressure <90 mmHg or systolic arterial pressure (SAP) <100 mmHg despite adequate fluid challenge (20 ml/kg of normal saline infused over half hour). Criteria for exclusion were pregnancy or other causes of raised intra-abdominal pressure, patients in whom USG could not be done because of poor echo window or dressings, acute coronary syndrome, cardiac dysrhythmias (as a primary diagnosis) or valvular heart diseases, congestive heart failure, pulmonary embolism, status asthmaticus, contraindication to central venous catheterization.

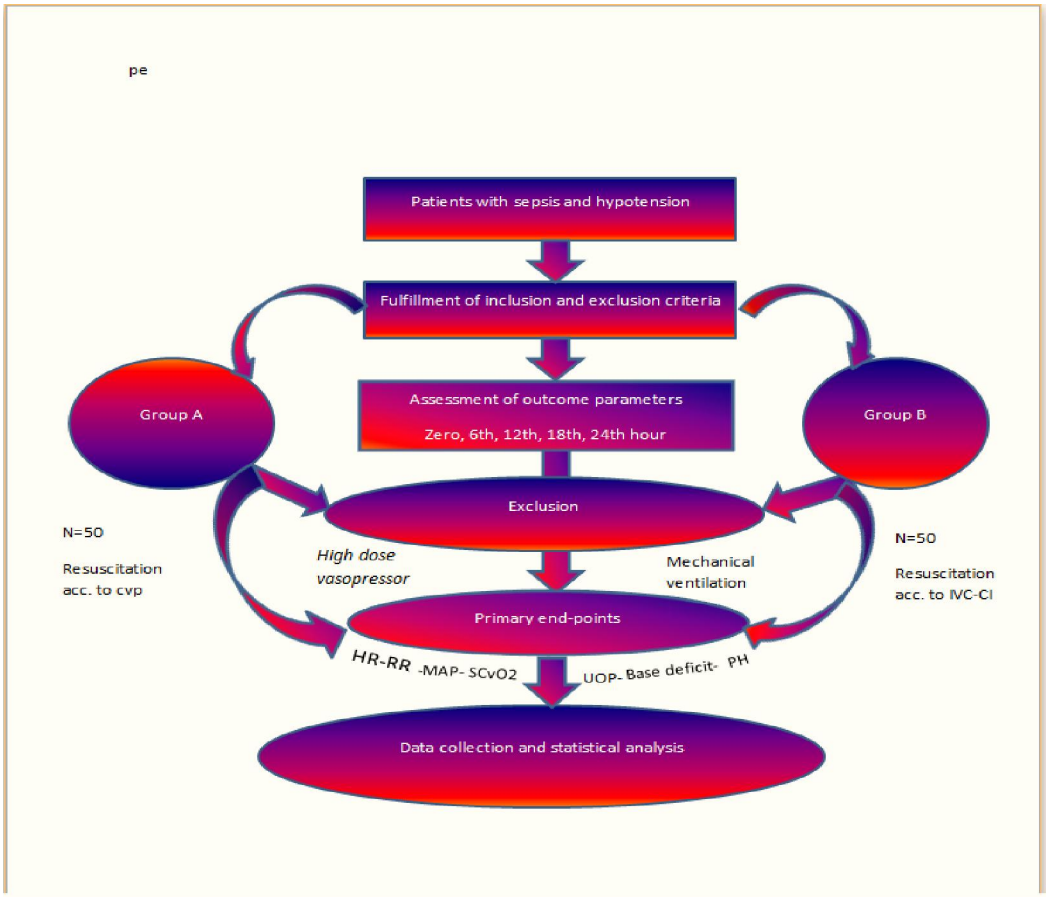

Figure 1

After admission to the ICU, patients were subjected to full clinical and laboratory investigations including lactate and central venous catheters were inserted. Echocardiography of patients was done to assess cardiac contractility and to rule out congestive heart failure. Patients were divided into two groups (Groups A and B) depending on the method of fluid resuscitation, and randomization was done by MRN. Group A patients (25) were resuscitated according to CVP and Group B patients (25) were resuscitated

according to IVC-CI. CVP was measured by central venous catheters inserted in either subclavian or internal jugular vein with its tip positioned in superior vena cava just proximal to the right atrium and confirmed by echocardiography and CXR. It was measured at zero point which corresponds with phlebostatic axis. Phlebostatic axis was taken as the line where a coronal plane midway between the back and sternum (in practice, the midaxillary line) intersects a cross-sectional plane through the fourth intercostal space. The CVP was measured in the expiratory and inspiratory phases of respiration using a column of saline which was later on converted into mmHg by dividing it by 1.3. The IVC assessment was made using hand-carried USG unit (Mindray M7, 1–5 MHz phased array probe) with the patient in the supine position using an acoustic window inferior to the xiphoid, angling to the right. The cross-sectional image of the IVC was visualized at the right atrial/hepatic vein/IVC junction and then rotated so that a long axis view of the IVC was obtained. M mode was applied at approximately 1 cm distal to the IVC-hepatic vein junction where the anterior and posterior walls were clearly visualized. For the sake of simplicity, maximum and minimum diameters were measured in each respiratory cycle. IVC-CI was calculated as (maximum diameter on expiration − minimum diameter on inspiration)/maximum diameter on expiration and expressed in percentage [Figure 2].

Both of these variables were recorded in each patient every two hourly till 24 h. Patients were given a fluid bolus 500 ml of crystalloid half-hourly after measuring CVP and IVC-CI till target levels of CVP or IVC-CI were achieved in respective groups. Vasopressors were started within 2 hours in situations of non-achievement of desired MAP.

Primary end-points were MAP of ≥65 mmHg and CVP >12 mmHg or IVC-CI <20% in Groups A and B, respectively. Patients were observed FOR 24 h. Outcome variables (pulse rate [PR], MAP, urine output, pH in arterial blood gas, base deficit and $ScvO_2$) were serially measured in both groups at 0, 6th, 12th, 18th, h, and end of the study. Lactate at baseline and end of the study was measured.

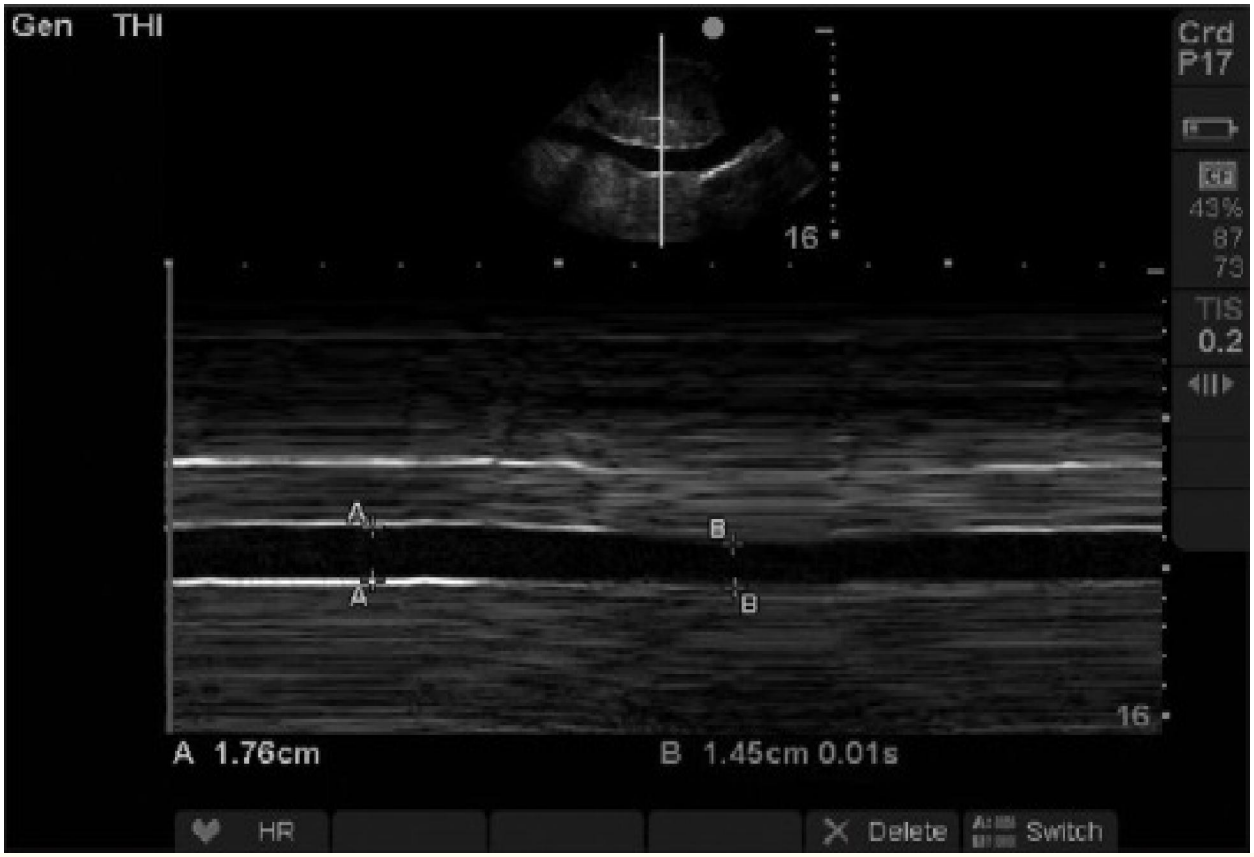

Figure 2

Calculation of inferior vena cava collapsibility index ([A–B]/B) (%) using ultrasonography.

IVC-CI = IVC_e – IVC_i/IVC_e

Statistical analysis

Both descriptive and analytical statistics were used in the study as appropriate. The values were

expressed as mean ± standard deviation. Correlation between CVP and IVC-CI was calculated by spearman correlation coefficient. Paired *t*-test was used to calculate any difference in outcome parameters after fluid resuscitations in both groups. Unpaired *t*-test was used for comparison of intergroup data. $P < 0.05$ was considered statistically significant.

3. Results:

Fifty patients of septic shock on a ventilator were randomly divided into two groups based on mode of fluid resuscitation – Group A (CVP) and Group B (IVC-CI). Two patients were excluded during the study due to mechanical ventilation implementation. There was no significant difference between two groups in baseline characters (age, Acute Physiology and Chronic Health Evaluation [APACHE] score on presentation, and mean fluid infused) during the study [Table 1].

Table (1): Comparison between the two studied groups according to demographics data, APACHE score, Lactate level and IV fluids

	Group A (n = 24)		**Group B** (n = 24)		**Test of Sig.**	**P**
	No.	%	No.	%		
Gender						
Y	13	54.0	14	58.0		0.774
X	11	46.0	10	42.0		
Age (years)						
Min. – Max. Mean ± SD. Median	39.0 – 60.0 50.60 ± 6.02 51.0		35.0 – 59.0 53.52 ± 5.39 55.0		t=1.807	0.077
APACHE score	22+/- 6		20+/-8		-	0.86
Lactate level (mg/dl)						
Baseline						
Min. – Max.	21.0 – 64.0		20.0 – 78.0		225.0	0.089
Mean ± SD.	36.24 ± 10.99		43.88 ± 15.84			
Median	34.0		45.0			
After 24 hrs.						
Min. – Max.	10.0 – 32.0		14.0 – 67.0		166.5*	0.012*
Mean ± SD.	19.83 ± 6.53		28.17 ± 12.72			
Median	19.0		24.50			
(P1)	<0.001*		<0.001*			
Tot IV fluids (liter)						
Min– Max.	3.7 – 7.5		4.6 – 8.5			0.089
Mean ± SD.	5.02 ± 0.9278		5.95 ± 0.8699			
Median	4.9		5.8			

X: female. Group A: Central Venous Pressure (CVP) group.
Y: male. Group B: Inferior Vena Cava (ICV) group.
P: p value for comparing between the two groups.
p_1: p value for **Wilcoxon signed ranks test** for comparing between base line and after 24 hours. *: Statistically significant at $p \leq 0.05$.

Table (3): Correlation between different parameters in all patients at (24 hours) (n= 48)

		CVP	**D-min**	**D-max**	**IVC- index**	**lactate**
CVP	**r_s**		0.101	-0.352	-0.253	0.387
	p		0.494	0.014*	0.082	0.007*
D-min	**r_s**			0.119	-0.869	0.118
	p			0.421	<0.001*	0.426
D-max	**r_s**				0.350	-0.118
	p				0.015*	0.424
IVC- index	**r_s**					-0.143
	p					0.331
lactate	**r_s**					
	p					

*: Statistically significant at $p \leq 0.05$. D-min: minimal diameter. rs: spearman correlation coefficient.
D-max: maximal diameter. IVC: inferior vena cava CVP: central venous pressure.

With fluid infusion, CVP values increased and IVC-CI values decreased in both groups. Correlating CVP and IVC-CI among patients, we found moderately negative correlation with spearman correlation coefficient -0.253 in total observations [table 3].

With resuscitation, PR, MAP, pH, and base deficit improved significantly in both groups, but urine output and $ScvO_2$ increased in Group B only [Table 4].

Table (4): Hemodynamic parameters at baseline and at the end of the study

	Baseline	End of study	P 1
Heart rate (B/m) Group A Group B P	125.6±19.3 126.12±17.56 0.946	108.13±13.5 106.6±15.4 0.566	0.003* 0.002*
MAP (mmHg) Group A Group B P	46.80 ± 5.52 51.40 ± 7.10 0.08	72.92 ± 6.03 73.0 ± 3.90 0.059	<0.001* <0.001*
PH Group A Group B P	7.27±0.21 7.29±0.18 0.06	7.32±0.23 7.34±0.12 0.057	0.020* 0.010*
Base deficit (mmol/l) Group A Group B P	-9.42±5.8 -12.9±6.8 0.082	-6.54±6.10 -9.3±6.81 0.12	0.015* 0.035*
ScvO2 (%) Group A Group B P	69.5±12.50 69.66±13.30 0.931	71.15±12.23 76.20±11.25 0.481	0.215 0.032*
UOP (ml/hr.) Group A Group B P	37.34±36.42 42.26±51.81 0.461	59.61±47.20 80.64±70.61 0.214	0.420 0.019*

P: p value for comparing between the two groups. *: Statistically significant at $p \leq 0.05$.
p_1: p value for **Wilcoxon signed ranks test** for comparing between base line and after 24 hours.
Group A: Central Venous Pressure (CVP) group. Group B: Inferior Vena Cava (ICV) group.

4. Discussion

The incidence of sepsis and septic shock is increasing even with advancing scientific researches. Septic shock and multiple organ dysfunction syndromes still remain a medical challenge for both primary care physicians and intensivists. The treatment includes mainly supportive measures. Mortality rises proportionately with the progression of organ failure that emphasizes the need for its prevention.

After the invention of the ICUs in the 1950s, the hemodynamic monitoring has traveled a long way with many crossroads. More invasive techniques became popular by time. CVP has been utilized and recommended since long for fluid resuscitation in septic shock. However, since CVP is incapable of predicting the exact volume status, it should not be used alone to make clinical decisions regarding fluid management. In the recent time, the pendulum has swung toward noninvasive, simple techniques which are less time consuming, with more safety and accuracy [(7), (8)].

Bedside ultrasonography is a commonly used technique that is readily available in the ED, intensive care unit, and elsewhere. It has even been used in pre-hospital settings. Ultrasonography is safe, non-invasive, and portable, and images are readily interpreted by a broad variety of specialists. Accurate measurement of internal structures, especially blood vessels as the IVC in our study, is readily achieved with ultrasound [(9)].

Changes in intra-abdominal and intrathoracic pressures (e.g., during positive-pressure ventilation or Valsalva maneuver) are also well known to alter the CVP and changes IVC diameters. Our study population composed of patients who are spontaneously breathing.

Previous studies generally support the correlation of IVC diameter to CVP. Many of these studies, however, used complex imaging and measuring techniques such as formal trans-esophageal echocardiography and caval indices.

In 2009 **Atkinson et al**, declared that the use of ultrasound-guided IVC assessment for fluid management in critically ill emergency patients is only validated when used in conjunction with other information such as the Abdominal and Cardiac Evaluation with Sonography in Shock protocol (ACES protocol) This study confirmed the usefulness of IVC assessment to guide IV fluids but was not specific to septic patients [(10)].

Again **Ferrada et al**, in 2012 proved that evaluation of the IVC diameter via limited transthoracic echocardiogram (LTTE) offers a rapid, non-invasive way to evaluate fluid status in critically ill patients either ventilated or not regardless ventilator parameters [(11)].

Also another study done by **Wiwatworapan et al**, in 2012 critically ill medical patients indicated that the measurement of the IVC diameter has a good correlation with CVP but there was not specific to patients with sepsis[(12)].

In our study, statistics showed that the maximal diameter of IVC correlated more than minimal diameter and caval indices with CVP with a p value of <0.014, r_s=0.352 for maximal diameter and p value =0.082, r_s=-0.253 for the collapsibility caval index. Also it stated that a minimal IVC diameter (< 12 mm) predicted a central venous pressure ≤8 mm Hg with sensitivity of 50%, specificity of 90.91%. The study revealed negative correlation between CVP and IVC-CI (-0.622) in the whole study sample; with intravenous fluid resuscitation CVP increases and IVC-CI decreases. Hence, our study confirmed that there is no difference between both methods of resuscitation.

Studies using different ultrasound techniques have reported r- values range 0.660–0.860 for CVP and IVC-CI correlations. In summarizing the results of these studies; **Robert Lorenzo** highlighted the potential value of use of ultrasonography in evaluation of IVC respiratory changes in fluid resuscitation decisions, but encouraged further studies [(13)].

Other studies as those done by **Jue et al**, in 1992 documented that the correlation between inferior vena cava diameter at expiration and mean right atrial pressure was 0.58 and the correlation between inspiratory change in inferior vena cava diameter and mean right atrial pressure was poor (r = 0.13). Despite these correlations, they prove that an inferior vena cava diameter of ≤ 12 mm predicted a right atrial pressure of 10 mm Hg or less with specificity 100%, but sensitivity was only 25%. An inferior vena cava diameter > 12 mm had no predictive value for right atrial pressure [(14)].

These results was parallel to our study result in prediction of low CVP with minimal diameter less than 12mm but with higher specificity and lower sensitivity, the difference probably owing to the patients who were mechanically ventilated and the use of different techniques to measure mean right atrial pressure as pulmonary catheter.

Another study on surgical ICU (SICU) patients was done by S. **Peter Stawicki** et al, in 2009 who stated that IVC-CI appears to correlate best with CVP in the setting of low (<0.20%) and high (>0.60%) collapsibility ranges and so measurements of IVC-CI by ultrasound can provide a useful guide to noninvasive volume status assessment in SICU patients. despite these results meets our study in the benefits of ivc measurement as a guide of fluid managements, it was done on surgical patients. Also extremes of values of the IVC-CI make it of no value in measurements between 20% and 60% [(15)].

In 2010, **Nagdev et al**, reported that 50% collapse of the IVC diameter during a respiratory cycle is strongly associated with a low CVP. The correlation between caval index and central venous pressure was -0.74 (95% confidence interval [CI] -0.82 to -0.63). The sensitivity of caval index greater than or equal to 50% to predict a central venous pressure less than 8 mm Hg was 91% (95% CI 71% to 99%), the specificity was 94% (95% CI 84% to 99%) these results goes with our result in confirmation of good correlation between IVC –CI and CVP in non-ventilated septic patients [(16)].

In 2012 **R. lorenzo**, reported that the IVC respiratory collapse is correlated to cvp but with lower r_s values than our study that ranged from 0.490 to 0.560, this came back to the single measures taken by the research team at one given time [(17)].

Prekker et al, in 2013 declared that among spontaneously breathing patients without vasopressor support, the maximal inferior vena cava diameter is a more accurate estimate of central venous pressure than the inferior vena cava collapsibility index as the maximal inferior vena cava diameter correlated moderately with central venous pressure (r_s= 0.58), whereas the inferior vena cava collapsibility index showed poor correlation (r_s = 0.16), these results agree to some extent with our study results but there is difference in specificity and sensitivity of IVC diameter to predict low CVP, the difference may came back to patients who were not supported by any vasopressors in prekker study [(18)].

At last, in 2016 a study done by **Manjri Garg** confirmed that IVC-CI is significantly negatively correlated with CVP, and either method can be used in fluid management. This study agrees with our study

result of the negative correlation between IVC-CI and CVP, also in supporting using either method for resuscitation [19].

Limitations of the study

Our study is limited in various ways. The range of subjects was less broad than expected because there were a lot of patients excluded for the selection criteria so it couldn't be applied on all ICU patients. Also the absence of CVL in many spontaneously breathing patients made the study on this group of patients limited. Also some patients had a poor subcostal echocardiographic window such as obese patients or orthopnic patients and patients with surgical laparotomy in whom ultrasonography is difficult.

Technical difficulties in the IVC imaging included tachypnic patients because of too rapid respiratory variations in IVC to be captured and measured by the ultrasound. All these factors limit the routine use of the IVC diameter and caval indices instead of CVP in all ICU patients.

Conclusion and future work

Based on our observational study which compared using central venous pressure with inferior vena cava collapsibility index as a guide of fluid management in sepsis patients, we recommend using any of both methods to guide fluid therapy as there is no significant difference in primary outcome between both groups.

We can -despite our limited study- implement non-invasive inferior vena cava diameter and collapsibility measurements in practice to guide fluid goals in septic patients rather than invasive central venous pressure changes monitoring which may carry risks of mechanical complication of central line insertion.

Further studies with large number of subject need to be performed to rule in or out these results.

Financial support and sponsorship:

Nil.

Conflicts of interest:

There are no conflicts of interest.

Brief Introduce

Objective: Intravenous fluids is the most challenging step in the treatment of patients with septic shock, that needs continuous monitoring of hemodynamics and response feedback. The study was structured to assess the efficacy of ultrasonographically measured inferior vena cava collapsibility index (IVC-CI) in comparison to the traditional central venous pressure (CVP) in guiding fluid management in septic patients. **Materials and Methods:** Fifty patients diagnosed by septic shock requiring minimal dose vasopressors support were included. Patients with congestive heart failure, raised intra-abdominal pressure, and poor echo window were excluded from the study. They were randomly divided into two groups based on method of fluid resuscitation – Group A (CVP) and Group B (IVC-CI). Primary end-points were mean arterial pressure (MAP) of ≥65 mmHg and CVP >12 mmHg or IVC-CI <20% in Groups A and B, respectively. Patients were followed till achievement of end-points or maximum of 24 hours. Outcome variables (pulse rate, MAP, urine output, PH, and base deficit, and $ScvO_2$, lactate) were serially measured till the end of the study. **Results**: Fluid infusion, by any of the two methods, had increased CVP and decreased IVC-CI with resultant negative correlation between them (spearman correlation coefficient -0.253). There was no significant difference in the amount of fluid infused and time to reach end-point in two groups. Comparison in outcome variables at baseline and end-point showed no significant difference. **Conclusion:** CVP and IVC-CI are negatively correlated with fluid resuscitation, and both methods can be implemented for resuscitation, with IVC-CI being nearly equal to CVP but with no complications that may be related to CVC.

Key Words: Central venous pressure, fluid management, hypovolemia, septic shock, sonographic inferior vena cava collapsibility.

References:

1. Levy MM, Macias WL, Russell JA, Williams MD, Trzaskoma BL, Silva E, Vincent JL. Failure to improve during the first day of therapy is predictive of 28-day mortality in severe sepsis. Chest. 2004; 120 S.
2. Rivers, Emanuel, Bryant Nguyen, Suzanne Havstad, Julie Ressler, Alexandria Muzzin, Bernhard Knoblich, Edward Peterson, Michael Tomlanovich, and Early Goal-Directed Therapy Collaborative Group. 2001. "Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock." New England Journal of Medicine 345 (19): 1368–77.
3. De Lorenzo RA, Morris MJ, Williams JB, Haley TF, Straight TM, Holbrook-Emmons VL, et al. Does a simple bedside sonographic measurement of the inferior vena cava correlate to central venous pressure? J Emerg Med 2012; 42(4):429-36.
4. Lichtenstein D, Mezière G. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: The BLUE Protocol. Chest 2008; 134; 117-25.
5. Richard klabunde, cardiovascular physiology concepts, second edition, Lippincott Williams & wilkins,2012;4:69-75.
6. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived

variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. Crit Care Med 2009; 37(9):2642-7.
7. Nagdev, Arun D., Roland C. Merchant, Alfredo Tirado-Gonzalez, Craig A. Sisson, and Michael C. Murphy. 2010. "Emergency Department Bedside Ultrasonographic Measurement of the Caval Index for Noninvasive Determination of Low Central Venous Pressure." Annals of Emergency Medicine 55 (3): 290–95.
8. Rivers, Emanuel, Bryant Nguyen, Suzanne Havstad, Julie Ressler, Alexandria Muzzin, Bernhard Knoblich, Edward Peterson, Michael Tomlanovich, and Early Goal-Directed Therapy Collaborative Group. 2001. "Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock." New England Journal of Medicine 345 (19): 1368–77.
9. Strode CA, Rubai BJ, Gerhardt RT, Christopher FL, Bulgrin JR, Kinkler ES Jr, et al. Satellite and mobile wireless transmission of focused assessment with sonography in trauma. Acad Emerg Med 2003; 10: 1411–4.
10. Atkinson, P. R.T., D. J. McAuley, R. J. Kendall, O. Abeyakoon, C. G. Reid, J. Connolly, and D. Lewis. 2009. "Abdominal and Cardiac Evaluation with Sonography in Shock (ACES): An Approach by Emergency Physicians for the Use of Ultrasound in Patients with Undifferentiated Hypotension." Emergency Medicine Journal 26 (2): 87–91.
11. Ferrada, Paula, Rahul J Anand, James Whelan, Michel A Aboutanos, Therese Duane, and Ajai Malhotra. 2012. "Qualitative Assessment of the Inferior Vena Cava: Useful Tool for the Evaluation of Fluid Status in Critically Ill Patients," no. April: 13–16.
12. Wiwatworapan, Weerapan, Nuchapa Ratanajaratroj, and Buncha Sookananchai. 2012. "Correlation between Inferior Vena Cava Diameter and Central Venous Pressure in Critically Ill Patients." Journal of the Medical Association of Thailand 95 (3): 320–24.
13. De Lorenzo RA, Morris MJ, Williams JB, Haley TF, Straight TM, Holbrook-Emmons VL, et al. Does a simple bedside sonographic measurement of the inferior vena cava correlate to central venous pressure? J Emerg Med 2012; 42(4):429-36.
14. Jue, John, William Chung, and Nelson B. Schiller. 1992. "Does Inferior Vena Cava Size Predict Right Atrial Pressures in Patients Receiving Mechanical Ventilation?" Journal of the American Society of Echocardiography 5 (6): 613–19.
15. Stawicki, S. Peter, Benjamin M. Braslow, Nova L. Panebianco, James N. Kirkpatrick, Vicente H. Gracias, Geoffrey E. Hayden, and Anthony J. Dean. 2009. "Intensivist Use of Hand-Carried Ultrasonography to Measure IVC Collapsibility in Estimating Intravascular Volume Status: Correlations with CVP." Journal of the American College of Surgeons 209 (1): 55–61.
16. Nagdev, Arun D., Roland C. Merchant, Alfredo Tirado-Gonzalez, Craig A. Sisson, and Michael C. Murphy. 2010. "Emergency Department Bedside Ultrasonographic Measurement of the Caval Index for Noninvasive Determination of Low Central Venous Pressure." Annals of Emergency Medicine 55 (3): 290–95.
17. De Lorenzo RA, Morris MJ, Williams JB, Haley TF, Straight TM, Holbrook-Emmons VL, et al. Does a simple bedside sonographic measurement of the inferior vena cava correlate to central venous pressure? J Emerg Med 2012; 42(4):429-36.
18. Matthew E. Prekker, Nathaniel L. Scott, Danielle Hart, Mark D. Sprenkle, James W. Leatherman. 2013. "Point-of-Care Ultrasound to Estimate Central Venous Pressure: A Comparison of Three Techniques*." Critical Care Medicine 41 (3): 833–41.
19. Garg Manjri, Sen, Jyotsna Goyal, Sandeep, Chaudhry and Dhruva. Comparative evaluation of central venous pressure and sonographic inferior vena cava variability in assessing fluid responsiveness in septic shock. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine 2016; 20:708-713.

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