ORIGINAL ARTICLE

Comparison of inferior vena cava collapsibility and central venous pressure in assessing volume status in shocked patients

Monira T Ismail [a,b,*], Afaf A. El-iraky [a], Emad El-Din A. Ibrahim [c], Tarek H. El.Kammash [d], Ahmed E. Abou-Zied [a]

[a] *Emergency Medicine department, Faculty of Medicine, Suez Canal University, Ismailia, Egypt*
[b] *Medical Science Department, Sulaiman Al-Rajhi University, Saudi Arabia*
[c] *Anesthesia and intensive care department, Faculty of Medicine, Suez Canal University, Ismailia, Egypt*
[d] *Diagnostic radiology department, Faculty of Medicine, Suez Canal University, Ismailia, Egypt*

A R T I C L E   I N F O

A B S T R A C T

Introduction: Determination of intravascular volume status in patients admitted to the emergency centre is critical. Physical signs of hypovolaemic, distributive, cardiogenic, and obstructive shock frequently overlap, making an accurate diagnosis of shock state difficult. This is problematic because fluid loading is considered the first step in haemodynamically unstable patients' resuscitation. Yet, multiple studies have shown that only approximately 50% of haemodynamically unstable patients in the intensive care unit and operating theatre respond to a fluid challenge. This study aims to provide an accurate estimation of intravascular volume status using bedside noninvasive methods as an essential part of the assessment of volume status in shocked patients.
Methodology: This is a cross-sectional analytical study conducted on 102 shocked patients presented to the emergency centre. IV fluid boluses were standardized to be administered at 500 mL every 30 min over 120 min, as clinically indicated. Concurrent measurements of inferior vena cava collapsibility index (IVC-ci) were performed shortly before the initiation of IV bolus (i.e., time 0), and then at 30, 90, and 120 min, we measured both venous collapsibility index (CI) and central venous pressure (CVP). At each session, we recorded patient demographics, fluid responsiveness, and vital sign assessments.
Results: We discovered that IVC-ci at cut-off point 40 has a sensitivity of 93.3% and specificity of 70.7% with an AUC of 0.908 and a good 95% CI (0.84–0.975), implying that IVC-ci of 40% or higher can indicate fluid responsiveness in shocked patients. CVP, despite having a good sensitivity of 88.6%, high specificity of 100%, and a significant p-value, is not a reliable detector of fluid responsiveness due to its small AUC value and low 95% CI.
Conclusion: IVC-ci could be a good tool with moderate reliability for detecting fluid responsiveness because it is a less invasive and fast method.

Introduction

Haemodynamic support for patients in shock is crucial to prevent worsening organ dysfunction, resuscitation should be started while the investigation to determine the cause is ongoing [1]. Determination of intravascular volume status in patients admitted to the emergency centre (EC) is critical [2]. An accurate diagnosis of shock state can be challenging because physical signs of hypovolaemic, distributive, cardiogenic, and obstructive shock frequently overlap [3]. Clinical determination of the intravascular volume in critically ill and injured patients can be extremely difficult. This is problematic because fluid loading is considered the first step in haemodynamically unstable patients' resuscitation. Yet, multiple studies have shown that only approximately

50% of haemodynamically unstable patients in the intensive care unit (ICU) and operating theatre respond to a fluid challenge [4]. Traditional invasive intravascular volume assessment modalities, such as pulmonary artery and central venous pressure (CVP) catheters, which provide physiologic data, such as cardiac output and right atrial pressure, are time-consuming and carry significant risks [5].

Traditionally, CVP has been assumed to accurately reflect the intravascular volume and has played a central role in guiding fluid management decisions for decades [6]. This invasive method has several complications, such as arrhythmias, cardiac chamber injury, vascular-nerve injury, pneumothorax, haemothorax, local bleeding, haematoma, infection, thrombosis, occlusion, pulmonary embolism, and post-phlebitis syndrome, which may occur with catheter placement [7].

---

* Corresponding author.
E-mail address: Monirataha77@yahoo.com (M.T. Ismail).

Over the last decade, the long-held belief that CVP accurately reflects volume status has been challenged. Recent literature reviews have revealed a poor relationship between CVP and blood volume, which is sometimes difficult to apply in clinical practice [8]. A recent systematic study revealed a poor relationship between CVP and blood volume, as well as the inability of CVP/ΔCVP to predict the haemodynamic response to a fluid challenge. CVP should not be used to make clinical fluid management decisions [6].

The inferior vena cava (IVC) is the largest vein in the venous system with low pressure. To a certain extent, vein expansion reflects venous pressure variations. This variation also reflects the excess of the intravascular volume. As a result, the IVC diameter may be a useful diagnostic tool in the assessment of hypovolaemia and hypervolaemia [7]. There is growing interest in researching the venous collapsibility index (VCI) as a noninvasive, easily repeatable, and portable alternative to traditional invasive haemodynamic monitoring approaches [4]. Studies in intensive care unit (ICU) patients revealed that measurements of the respiratory variation in IVC diameter can be used to predict fluid responsiveness in mechanically ventilated patients. There has been no research on the accuracy and feasibility of the caval index to predict fluid responsiveness in the EC [8].

This study aims to provide an accurate estimation of intravascular volume status using bedside noninvasive methods as an essential part of the assessment of volume status in shocked patients.

Methods

This is a cross-sectional analytical study, which was conducted in Suez Canal University hospital in Ismailia city on 102 shocked patients presented to EC, Collected within one year from December 2017 up to December 2018. The study was conducted at the EC at SCUH, Ismailia, Egypt. The EC at SCUH is a 100-bed divided into general assessment area, trauma yard, observational rooms and 10 -bed unit as a resuscitation room, working 3 days per week (Saturday, Monday and Wednesday). During the other 4 days, emergency patients are directed to Ismailia General Hospital unless a major traumatic accident or a disaster occurs. The estimated EC visit volume is around 400 patients per day. Emergency centres provide care for all kind of cases, including road traffic injury victims and patients with medical, surgical, obstetrics or paediatric emergencies. Moreover, it provides emergency care service for the inhabitants of 5 governorates in Egypt; therefore, it is usually crowded with shocked patients. There's increasing number of patients presenting to EC, which necessitates applying an accurate and rapid diagnostic tool for shocked patient in order to stop wasting resources and provide the best care for patients in need within the ideal time [10].

We randomly selected adult patients (≥18 years old) who were admitted to the EC with signs and symptoms of any type of shock, requiring fluid resuscitation and central venous catheterization for invasive haemodynamic monitoring. We excluded patients who met the following criteria: Patients who refused to participate in the study, patients with intra-abdominal pressure over 12 cmH2O, if ultrasonography was unable to view the IVC, and patients in late pregnancy (second and third trimesters) because it increases intra-abdominal pressure in intubated and mechanically ventilated patients.

Before the ultrasonographic examination, the patient provided written, informed consent for participation, either by himself/herself if conscious or by a legal guardian if the patient was unconscious and delayed consent after regaining consciousness. The approval of hospital administration and the chief of the EC was sought in the case of an unconscious patient.

Patients who did not meet the exclusion criteria were assessed for the following signs of shock: systolic blood pressure >90 mmhg, heart rate <100beat/min, capillary refill <2 s. Blood pressure was measured by means of a noninvasive cuff. We included patients exhibiting one or more signs of shock according to guidelines of ACLS and ATLS (advanced

cardiac life support and advanced trauma life support, respectively) in 2010.

We started intravenous fluid resuscitation of 20 ml/kg of crystalloid (normal saline as a bolus 1–2 litre NaCl 0.9%) within 120 min. Our targets were: a systolic blood pressure of 70:90 mmHg, a MAP (mean arterial pressure) >60 mmHg and >80 mmHg in head trauma, a CVP of 8:12 mmH2O, and a urine output >0.5 ml\hr. The patient was reassessed after the first bolus, and if vital signs did not improve, another bolus was administered. Ipon reassessment if there is no improvement, a vasopressor and/or one or two of the following inotropes was used: Norepinephrine: (0.05–1 mcg/kg/min), Epinephrine: (0.05–0.5 mcg/kg/min), Dopamine (5–20 mcg/kg/min).

The patients' haemoglobin and haematocrit were also assessed; if haemoglobin was less than 7 gm/d and haematocrit less than 25%, the patient required a transfusion of packed RBCs (red blood cells). In trauma patients, we employed the damage control resuscitation protocol, which includes controlled hypotension, haemostasis resuscitation (as previously described), and damage-controlled surgery. In cardiogenic shock, fluids are cautiously administered, guided by CVP.

We determined the age, sex, pulse, blood pressure, respiratory rate (RR), oxygen saturation (SO2), capillary refill, and urine output for all patients.

We used the model of ultrasound machine DP-2200Plus, Digital Ultrasonic Diagnostic Imaging System. Mindray Building, Keji 12th Road South, Hi-tech Industrial Park, China, 2017; Imaging mode: B, 2B, $B + M$, M; gray scales: 256; Display: 10 inches non-interlaced; Transducer frequency: 2.5 ∼ 10 MHz; Transducer connector: 1 standard, 2 optional; Beam-forming: Digital Beam-forming (DBF); Dynamic Receiving Focusing (DRF); Up to 16 zone transmitting focusing; Real-time Dynamic Aperture (RDA); Dynamic Frequency Scanning (DFS); Dynamic Receiving Apodization (DRA); Scanning angle: from 67 to 120° (depending on transducers); Scanning depth (mm): from 21.6 to 248 (depending on transducers) with a curved array transducer.

1) We measured the IVC diameter, while the patient was in a supine position.
2) The IVC was examined from a subcostal view in a longitudinal section. The maximal and minimal diameters were measured during a normal respiratory cycle; no breathing instructions were given. We examined the IVC, where its vessel walls were visualized best, preferably no further than 3 cm caudal to the right atrium junction.

We measured the diameter of IVC at the maximum of expiration (IVC-max) and at the maximum of inspiration (IVC-min) to calculate the collapsibility index.

The IVC was examined from a subxiphoid view in a parasagittal plane. The maximal and minimal diameter were measured during a normal respiratory cycle; no breathing instructions were given. We examined the IVC, where its vessel walls were visualized best, preferably no further than 3 cm caudal to the right atrium junction [9]. Measuring was done in M-Mode.

Subcostal Longitudinal view: View improves with the patient taking a deep inspiration. The transducer is placed right lateral to the subxiphoid, with the transducer indicator pointing toward 12:00 and the energy directed toward the left atrium.

Transhepatic coronal view: The liver parenchyma provides an excellent acoustic window and may be used when a subcostal view of the IVC is unobtainable, consider measuring in M-Mode.

We measured the diameter of IVC at the maximum of expiration (IVC/max) and at the maximum of inspiration (IVC/min) to calculate the collapsibility index.

Inferior vena cava collapsibility index (IVC-ci) was calculated as follows:

Inferior vena cava collapsibility index (IVC-ci) = (IVC/max diameter–IVC min diameter)/ (IVC/max diameter) * 100 [9].

Our study protocol was as follows:

1) Patients evaluated at time 0 (baseline) for blood pressure, pulse, RR, SO2, capillary refill time, MABP, IVC-ci, IVC/max, and IVC/min.

2) Folly's catheter (urinary) inserted to measure both urine output and intra-abdominal pressure.

3) Start fluid through wide pore peripheral cannula and prepare for central line insertion.

4) Fluid protocol: we give patients two liters of crystalloid solution in 2 h (120 min) by giving 500 ml every 30 min.

5) Patients reevaluated at 30, 90, and 120 min for blood pressure, pulse, RR, SO2, capillary refill time, MABP, IVC-ci, IVC-max, IVC-min, and CVP.

6) Special consideration of patients with cardiogenic shock, obstructive shock, and patients with CVP >15. Fluid was administered cautiously to these patients and decreased to 200 ml every 30 min guided by CVP.

7) According to MABP, the study population separated into two groups, fluid responders whose MABP after resuscitation is 70 or higher and fluid non-responders whose MABP less than 70 after resuscitation.

8) Each type of shock managed according to international guidelines and our policies in the university hospital. These international guidelines as follows: The American College of Surgeons guidelines used for hypovolemic shock. The American College of Surgeons and ATLS guidelines used to treat haemorrhagic shock. Camping guidelines for sepsis and septic shock. The AHA guidelines used for cardiogenic shock.

Intra-abdominal pressure measurement: Before measuring the intra-abdominal pressure, we drained the bladder using a Foley urinary catheter while the patient was placed in a supine position. Then, 50–100 ml of isotonic fluid was injected into the bladder in sterile conditions and the distal portion was clamped. Then, an 18-gauge needle was inserted into the output of the urinary catheter. The needle was connected to a three-way system and a water manometer.

After being filled with sterile fluid, the patient side of the manometer was opened. The manometer's "0″ point was positioned with the patient's pubic symphysis and the point where the liquid column was measured in centimeters. Thus, the bladder pressure and intra-abdominal pressure were measured in cmH2O units. We excluded patients with intra-abdominal pressure over 12 cmH2O from the study.

Central line insertion: Central venous line placement typically performed at one of these four sites, the right or left internal jugular vein (IJV), or the right or left subclavian vein.

Inferior vena cava measurements: IV fluid boluses were standardized to be administered at 500 mL every 30 min over 120 min, as clinically indicated. Concurrent measurements of inferior vena cava collapsibility index (IVC-ci) were performed shortly before the initiation of IV bolus (i.e., time 0), and then at 30, 90, and 120 min, we measured both venous CI (collapsibility index) and CVP. At each session, we recorded patient demographics, fluid responsiveness, and vital sign assessments.

Special consideration of patients with cardiogenic shock, obstructive shock, and patients with CVP >15. Fluid was administered cautiously to these patients and decreased to 200 ml every 30 min guided by CVP.

According to MABP, the study population is separated into two groups, fluid responders whose MABP after resuscitation is 70 or higher and fluid non-responders whose MABP less than 70 after resuscitation.

We obtained data using a fill-in questionnaire, entered it into a computer spreadsheet, and analysed it using appropriate statistical software to determine the statistical inference of the variables under consideration.

Data was then imported into SPSS 16 (Statistical Package for Social Sciences) for analysis. Based on the data type, the following tests were performed to examine differences for significance, chi-square test, *t*-test, multi-variant regression analysis, and one-way ANOVA with the least significant difference. We performed chi-square and nonparametric tests to compare categorical variables. P-value was set at <0.05 for significant results. Spearman's correlation coefficient was used to estimate different correlations between variables. Sensitivity, specificity, and ICV-ci as a modality in early diagnosis of shock, fluid responsiveness detection, and monitoring of shocked patients were measured based on the cut-off point.

This study was approved by the Suez Canal University Faculty of Medicine Ethics Committee. Each patient or one of his/her legal guardians provided written informed consent. Handwritten signatures, fingerprints, and personal stamps were accepted. The approval of hospital management and the chief of the EC were sought in the case of an unconscious patient.

Results

amongst the 102 spontaneously breathing patients (57.8% male and 42.2% female) with ACF included in this analysis, 43.1% (44 patients) were responders while 56.9% (58 patients) were non-responders. amongst responder patients, 25% were admitted to ICU compared with 96.6% in non-responders. 70.5% of responder patients were admitted to the ward (in-patient) and 4.5% were transferred to the operating theatre. In non-responders, 1.7% died and 1.7% were transferred to the operating theatre

In this study, CVP at time 0 cannot be determined since it requires time for preparation and insertion of 20 to 30 min, thus, we started resuscitation through the peripheral line until the central line is administered. CVP at time 30–90–120 showed a significant difference between shock types.

In this study population, there was a significant difference between shock types in IVC-ci at baseline (time 0), indicating that we can use IVC-ci as a diagnostic measure for shock type before starting resuscitation compared to CVP, which requires time for insertion.

Table 1 shows IVC-ci at baseline and during resuscitation in different shock types of the central line. This table shows that there was a significant difference between types of shock in IVC-ci at baseline (time 0), indicating that we can use IVC-ci as a diagnostic measure to differentiate between types of shock before starting resuscitation.

In hypovolaemic shock, IVC-ci 0 was 75.4 ± 27.5, which is nearly collapsed. In distributive shock (septic and anaphylactic) IVC-ci 0 was flat but not total collapsed as in hypovolaemic shock (septic 54.2 ± 16.1, anaphylactic 65.3 ± 28.9), and some readings were normal. In cardiogenic shock, IVC-ci 0 was distended (28.5 ± 12.4) with minimal collapsibility. IVC-ci 0 was severely distended in obstructive shock with essentially no collapsibility (7.5 ± 10.2).

Table 2 shows that amongst the study population, there was a significant difference in IVCmax0 in different types of shock. In hypovolaemic shock, IVC-max was flat (12.4 ± 2.1) while in distributive shock (septic and anaphylactic) it was flat to normal (13.5 ± 1.8 and 12.5 ± 2.2), respectively. In cardiogenic and obstructive shock, it was distended (21.1 ± 2.7 and 23.1 ± 4.03), respectively.

Table 3 shows the differences in haemodynamic parameters variations between responders and non-responders at baseline and during resuscitation in all study populations.

Respiratory rate

At baseline, the RR was 25.48 ± 2.9 in non-responders and 21.8 ± 2.4 in responders, with a significant difference between the two groups. After resuscitation, (RR120) was 23.4 ± 2.7 in non-responders and 17.9 ± 1.9 in responders, with a significant difference.

Oxygen saturation: In all types of shock (total study population), there was a significant difference in SO2 between responders and non-responders at baseline and after resuscitation.

MABP: At baseline and after resuscitation, there was a significant difference in MABP readings between responders and non-responders.

At baseline, responders had an IVCci0 of 83.8 ± 22.08, whereas non-responders had an IVCci0 of 39.9 ± 14.8, with a significant difference between the two groups. After resuscitation, IVCci120 was 45.4 ± 12.2

Table 1

IVC-ci at baseline and during the course of resuscitation in different types of shock of central line.

		Hypovolaemic	Septic	Cardiogenic	Anaphylactic	Obstructive	p-value
IVC ci	0	75.4 ± 27.5	54.2 ± 16.1	28.5 ± 12.4	65.3 ± 28.9	7.5 ± 10.6	<0.05*
	30	54.6 ± 17.9	50.8 ± 14.9	26.4 ± 11.9	54.5 ± 23.8	7 ± 9.8	<0.05*
	90	45.3 ± 9.9	44.09 ± 9.14	18.7 ± 15.9	47 ± 16.8	0	<0.05*
	120	41.7 ± 11.3	41.5 ± 9.3	13.1 ± 16.6	41.8 ± 12.4	0	<0.05*

Table 2

IVC-max at baseline and during the course of resuscitation in different types of shock.

Hypovolaemic			Septic	Cardiogenic	Anaphylactic	Obstructive	p-value
IVC maximum	0	12.4 ± 2.1	13.5 ± 1.8	21.1 ± 2.7	12.5 ± 2.2	23.1 ± 4.03	<0.05*
	30	14.2 ± 1.5	14.2 ± 1.6	21.8 ± 2.7	14.5 ± 1.7	23.3 ± 2.3	<0.05*
	90	16.4 ± 1.14	15.9 ± 1.4	23.4 ± 2.5	16.7 ± 0.9	24.05± 1.3	<0.05*
	120	17.3 ± 2.6	16.7 ± 1.4	24.3 ± 1.8	17.5 ± 1.3	24.05 ± 1.3	<0.05*

Table 3

Difference in variations of haemodynamic parameters between responders and non-responders.

		Total study population	responder	Non responder	p-value
Age		62.9 ± 18.5	53.6 ± 18.03	70 ± 15.6	<0.05*
Pulse	0	115.8 ± 5.3	112.2 ± 3.6	116.6 ± 14.8	<0.05*
	30	113.5 ± 6.13	109.09 ± 4.6	116.8 ± 4.9	<0.05*
	90	107.6 ± 6.7	101.8 ± 4.4	112.1 ± 4.3	<0.05*
	120	104.9 ± 8.6	96.5 ± 4.6	111.4 ± 4.5	<0.05*
RR	0	23.9 ± 3.2	21.8 ± 2.4	25.48 ± 2.9	<0.05*
	30	23.3 ± 3.4	20.9 ± 2.5	25.1 ± 2.9	<0.05*
	90	22± 3.5	19.2 ± 2.2	24.1 ± 2.7	<0.05*
	120	21.06 ± 3.6	17.9 ± 1.9	23.4 ± 2.7	<0.05*
So^2	0	92.9 ± 2.8	94.7 ± 2.6	91.5 ± 2.01	<0.05*
	30	93.06 ± 2.7	95.04± 2.3	91.5 ± 1.89	<0.05*
	90	93.8 ± 2.3	95.6 ± 1.9	92.5 ± 1.5	<0.05*
	120	94.2 ± 2.2	96.06 ± 1.7	92.8 ± 1.4	<0.05*
MABP	0	52.7 ± 5.5	57.5 ± 3.6	49.1 ± 3.7	<0.05*
	30	54.7 ± 6.8	61.2 ± 3.5	49.8 ± 4.01	<0.05*
	90	62.1 ± 7.7	70.2 ± 2.19	56.1 ± 3.9	<0.05*
	120	65.8 ± 10.07	76.5 ± 3.9	57.7 ± 3.37	<0.05*

Pulse: A pulse at baseline (pulse0) was observed in both non-responders (116.6 ± 14.8) and responders (112.2 ± 3.6) in the study population, with no significant difference. After resuscitation, (pulse120) was (96.5 ± 4.6) in responders and (111.4 ± 4.5) in non-responders, with a significant difference between the two groups.

Table 4

Sensitivity & Specificity of CVP, IVC-ci, IVC-max &IVC-min at time 30 for all study population.

Time30	Area under the curve	Cut off point	Sensitivity	Specificity	p-value	CI
CVP	0.116	3	88.6%	100%	<0.05*	0.03–0.197
IVC collapsibility	0.908	40	93.3%	70.7%	<0.05*	0.84 −0.975
IVC maximum	0.258	13	61.4%	100%	<0.05*	0.159–0.358
IVC minimum	0.143	5	79.5%	98.3%	<0.05*	0.06–0.227

in responders and 29.05 ± 15.9 in non-responders, with a significant difference.

By studying the ROC curves for CVP, IVC-ci, IVC/max, and IVC/min at 30 min. (Fig. 1). We can observe that IVC-ci at cut-off point 40 has a sensitivity of 93.3% and specificity of 70.7%, with AUC of 0.908 and a good 95% CI (0.8–0.975) (tab:4), implying that IVC-ci of 40% or higher can detect fluid responsiveness. CVP, despite having a good sensitivity of 88.6%, high specificity of 100%, and significant p-value, is not a reliable detector of fluid responsiveness due to its small AUC and low 95% CI. However, IVC/max has good sensitivity, high specificity, and significant p-value, but its relatively small AUC of 0.189 and low 95% CI (0.097–0.281) make it unreliable for detecting fluid responsiveness. Also, IVC/min is an unreliable measure for fluid responsiveness due to its small AUC and low 95% CI9 (Table 4).

This table shows that IVC-ci at cut-off point 40 has a sensitivity of 93.3% and specificity of 70.7% with an AUC of 0.908 and a good 95%

CI (0.84–0.975). IVC/max has good sensitivity, high specificity, and significant p-value put with a negligible AUC of 0.189 and low 95% CI (0.097–0.281). We also discovered that IVCci0 at cut-off point 40 has a sensitivity of 93% and specificity of 65.5%, indicating that IVC-ci of 40% or higher can detect fluid responsiveness. Furthermore, we discovered that IVC-ci at 120 min (after fluid resuscitation) cut-off point 38 has 86.4% sensitivity and 63.8% specificity, indicating that IVC-ci of 38% or higher can detect fluid responsiveness. In our study, we discovered that CVP is *inversely* related to IVC-ci with high statistical significance, this means that when CVP rises, IVC-ci falls.

Discussion

Point-of-care or focused ultrasonography is currently a crucial bedside technique within critical care and emergency medicine for answering time-dependant targeted clinical questions. For acute illness, it has
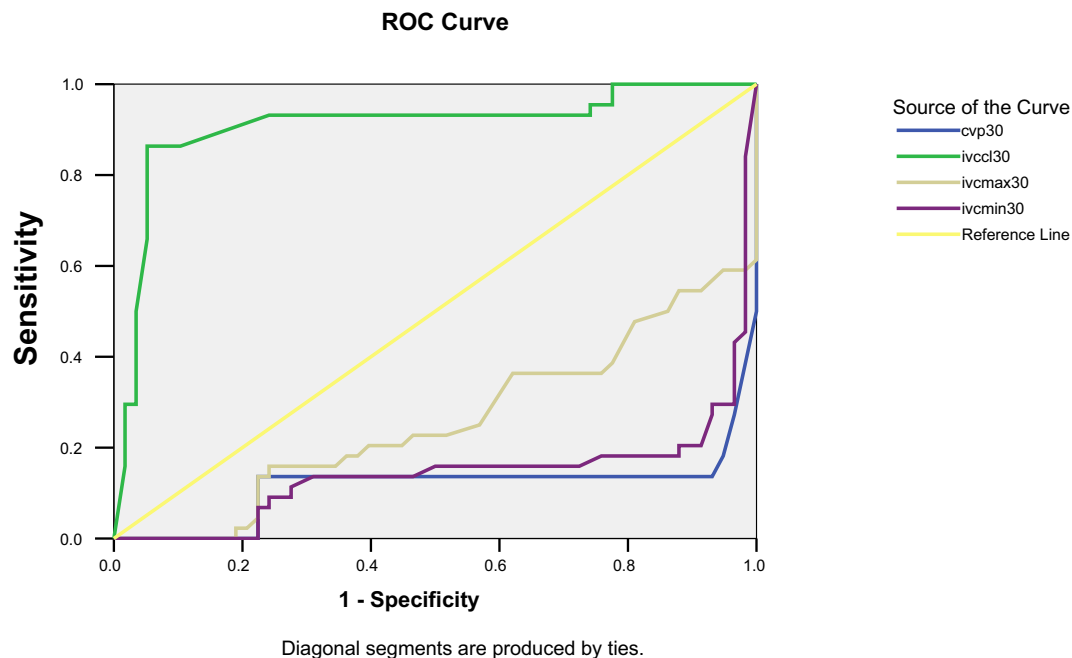
ROC Curve

Diagonal segments are produced by ties.

Fig. 1. ROC curve of CVP, IVC-ci, IVC-max and IVC-min at time 30.

several advantages over traditional imaging modalities. It is safe, rapid, noninvasive, and is delivered to the patient's bedside. In specific clinical settings, bedside sonography is an adjuvant to clinical examinations to rule in or rule out the primary diagnoses [10]. Accurate estimation of intravascular volume status is vital in the management of the critically ill. Invasive procedures can lead to various complications, and the VCI is increasingly being recognized as a potential noninvasive replacement or source of adjunct information. Nonetheless, questions have been raised concerning its effectiveness [5].

In this study, we evaluated the diagnostic value of measuring the IVC index in the assessment of intravascular volume status in shocked patients. We also examined the reliability of the IVC index in monitoring the efficacy of resuscitation in shocked patients and determining which patient will benefit the most from this strategy. In our study, there was a significant difference (P-value < 0.05*) between types of shock in IVC-ci and IVC/max at baseline (time0), which indicates that we can use them as a diagnostic measure to differentiate between shock types before starting resuscitation. Our result was consistent with Agarwal D. et al., who had a meta-analysis in 2012 of data from five studies on the sonographic measurement of the IVC in assessing fluid status in the EC. They discovered that the maximum IVC diameter is lower (6.3 mm 95% CI 6–6.5 mm) in patients with hypovolaemia than in patients with euvolaemia [8]. In 2018, Elbaih et al. discovered significant diagnostic reliability of RUSH of each shock type in polytrauma patients, with a total accuracy of 95.2% (The RUSH examination includes an IVC measurement.) [10].

In this study, we also discovered that IVCci0 and IVCci30 at cut-off point 40 had a sensitivity of 93.3% and specificity of 70.7% with an AUC of 0.908 and a good 95% CI (0.84–0.975), indicating that IVC-ci of 40% or higher can detect fluid responsiveness. Furthermore, we discovered that IVCci120 (after fluid resuscitation) at cut-off point 35 has 86.4% sensitivity and 63.8% specificity with an AUC of 0.848 and a good 95% CI (0.769–0.927), implying that IVC-ci of 35% or less can detect fluid unresponsiveness. However, IVC/max has good sensitivity, high specificity, and significant p-value, but its small AUC of 0.189 and low 95% CI (0.097–0.281) make it unreliable for detecting fluid responsiveness. Also, IVC/min is an unreliable measure for fluid responsiveness due to its small AUC and low 95% CI.

Muller et al. (2012) discovered that IVC-ci moderately predicted fluid responsiveness in spontaneously breathing ACF patients. Fluid responsiveness cannot be excluded in patients with a low IVC-ci value (<40%), while patients with IVC-ci above 40% are more likely to respond to fluid challenges. Then, despite its simplicity of use, IVC-ci should be used with caution in ACF patients who are spontaneously breathing [11]. Kent et al. (2013) supported the same result, concluding that IVCD measurements and IVC–CI calculations were found to be reliable markers of both clinical responses to volume resuscitation and intravascular volume status. Hypovolaemic patients were more likely to be diagnosed when IVC–CI >50%. However, when IVC–CI <20%, the patient may either have hypervolaemia or euvolaemia [12].

In 2014, de Valk et al. discovered that a caval index < 36.5% in a patient with signs of shock predicts the absence of an adequate response to a 500 ml NaCl 0.9% fluid challenge with a reliability of 92%. Aggressive fluid therapy might not be indicated or even harm these patients. However, it is impossible to predict the response to a fluid challenge for a patient with a caval index >36.5%. This is reflected by the low positive predictive value (48%) and weak correlation between caval index and fluid responsiveness. An explanation for the absence of a blood pressure response might be that these patients represent a group requiring more volume therapy than 500 ml. In future studies, we will investigate this issue more, and in our study, we gave more fluid and assessed the patient for a longer time [13].

Our results were also supported by Airapetian et al. (2015), who discovered that IVC/max was not predictive of fluid responsiveness. In contrast, he found that IVC-ci >42% may predict an increase in CO after fluid infusion in spontaneously breathing patients in the ICU [14]. Sawe HR et al. (2016) also agreed that ultrasound measurement of the CI can predict the blood pressure response in patients requiring intravenous fluid resuscitation and may be useful in the early identification of patients who will benefit most from volume resuscitation, and those who will likely require other interventions. They discovered the optimal CI cut-off values of 45%, 52%, and 53% for predicting MAP rises of 5, 8, and 10 mmHg per litre of fluid, respectively. The sensitivity and specificity of CI of 50% for predicting a 10 mmHg increase in MAP per litre were 88% (95% CI, 81%–93%) and 73% (95% CI, 67%–79%), respectively [15].

According to Preau et al. (2017), the CI of the IVC during deep standardized inspiration is a simple, noninvasive bedside predictor of fluid responsiveness in non-intubated patients with sepsis-related acute circulatory failure. When such index is superior or equal to 48%, fluid responsiveness is predicted with a sensitivity of 84% and a specificity of 90% [16].

Elbaih et al. (2018) discovered that IVC-ci shows 100% specificity and sensitivity in predicting fluid responsiveness when greater or equal to 50%. They also revealed that bedside ultrasonography evaluation of IVC-ci may be a useful bedside approach for EC physicians. The physician may be able to obtain a bedside assessment of intravascular volume by the IVC-ci assessment during normal respiration (>46.4%). Bedside ultrasonography of the IVC, in conjunction with common clinical markers, may be a useful adjunct in the evaluation of patients with EC [17].

Bortolotti et al. (2018) support our results in spontaneously breathing patients with cardiac arrhythmias, indicating that the CI and inspiratory diameter of the IVC assessed during deep inspiration are noninvasive bedside tools for predicting fluid responsiveness in acute circulatory failure due to infection. A cut-off point $\geq 39\%$ for IVC-ci predicts response to fluid with a specificity of 88%, a sensitivity of 93%, and a negative predictive value of 93% [18].

However, Oakley et al. conducted a meta-analysis in 2017 and discovered that respiratory variation in IVC diameter has limited ability to predict fluid responsiveness, particularly in spontaneously ventilating patients. When employing IVC ultrasound to aid in treatment decisions, the clinical context should be taken into account. In that analysis, 17 studies were involved. The sensitivity and specificity for positive IVC ultrasound as a predictor of fluid responsiveness were 0.63 and 0.73, respectively [19].

In 2018, Orso et al. conducted a meta-analysis for caval index using 20 studies: The pooled area under the curve, logarithmic diagnostic odds ratio, sensitivity, and specificity were 0.71 and 0.75, respectively. He also stated that ultrasound evaluation of IVC diameter and its respiratory variations does not seem to be a reliable method for predicting fluid responsiveness [20]. In our study, CVP shows a significant correlation with IVC–CI ($r = -0.827$, 0.891, and 0.882 at 30, 90, 120 min, respectively) and P-value ≤ 0.001, we revealed an inverse relationship between CVP and IVC-ci. Nagdev et al. (2010) discovered that during a respiratory cycle, 50% collapse or more of the IVCD was highly associated with a low CVP [21]. This result is consistent with that observed in a study conducted by Stawicki et al., in 2014, who discovered that measurements of IVC–CI by bedside ultrasonography can provide a useful guide to noninvasive volume status assessment in surgical ICU patients. CVP appears to best correlate with IVC–CI in the setting of low ($\leq 20\%$) and high ($\geq 60\%$) collapsibility ranges [22].

Worapratya et al. (2014) discovered that the caval index calculated from the IVC diameter measured by bedside ultrasound in the emergency room correlates well with CVP. The correlation of the CVP measurement with the ultrasound IVC caval index was $r = -0.721$ ($P = 0.000$) by two-dimensional mode ultrasound and $r = -0.647$ ($P = 0.001$) by M-Mode [23]. Similarly, Stawicki et al. (2015) discovered that the dynamic change in IVC-ci as a measure of responsiveness to fluid bolus is inversely related to changes observed in CVP [5]. Ilyas et al. (2017) support the same results. They discovered that there was a strong negative correlation between CVP and IVC CI (%), which was statistically significant ($r = -0.827$, $n = 100$, $p < 0.0005$) [24].

Abdelwahab et al. (2017) discovered a significant relationship between CVP and sonographic IVC measurements (IVC diameter and caval index) in spontaneously breathing patients. Although a statistically significant correlation was found between IVC measurements (IVC diameter and caval index) and CVP in spontaneously breathing and mechanically ventilated patients ($p < 0.001$), regression coefficients were higher in spontaneously breathing ($r = 0.74$ for IVC diameter and -0.76 for caval index) than mechanically ventilated patients ($r = 0.4$ for IVC diameter and -0.47 for caval index) [25]. Nazemi et al. (2017) also re-

vealed that caval index >50% has a sensitivity of 94% and a specificity of 97% in predicting low CVP (8 mmHg).

It appears that portable IVC sonography of emergency patients provides a noninvasive and fast approach to estimate the CVP and haemodynamic condition of the patient [26]. In this study, we agree with Elbaih et al. (2018), who discovered that CVP shows a significant correlation with IVC–CI with $r = -0.843$ and P-value ≤ 0.001, we revealed an inverse relationship between CVP and IVC-ci [17].

Because our study was conducted in a single hospital, the general applicability of our results may be limited, regardless of whether Suez Canal University Hospitals serve five governorates. The study population was also relatively small; therefore, we are unable to make firm conclusions. Lastly there was only one researcher involved in the study who was therefore unable to be blinded. Patients who need permissive hypotension should be removed from similar studies, as this type of patient selection is '' selection bias''.

Conclusion

Based on our findings, bedside ultrasonography of IVC and IVC-ci could be a good tool with moderate reliability for detecting fluid responsiveness because it is a less invasive and fast method compared to CVP. We may state that an IVC-ci of 40% or higher is a good predictor of fluid responsiveness. These results are valuable for hypovolaemic and distributive shock (septic and anaphylactic) patients but not for cardiogenic shock patients.

Our study also shows that IVC-max can differentiate between shock types, which can aid in decision-making. We discovered an inverse correlation between CVP and IVC-ci, allowing us to use IVC-ci to evaluate CVP noninvasively.

Based on our study, we recommend the following: Bedside ultrasound and measurement of IVC should be provided for any shocked patient in the EC, as this can aid in differentiating between types of shock and making decisions. It can also detect fluid responsiveness, which aids in patient monitoring and early planning for suitable management. We further recommend further research into IVC ultrasound for each type of shock and other applications. Ultrasound devices should be available in each EC. As part of their training course, all emergency physicians should learn how to use point-of-care ultrasound.

Dissemination of results

Results of this study were presented locally at the Emergency Centre scientific day with staff members at Suez Canal University.

Author contribution

Authors contributed as follow to the conception or design of the work, the acquisition, analysis, or interpretation of data for the work; and drafting the work or revising it critically for important intellectual content: MTI and AE contributed 30%; EI contributed 20%; TE and AA 10% each. All authors approved the version to be published and agreed to be accountable for all aspects of the work.

Declaration of Competing Interest

The authors declared no competing interests.

References

[1] Taha M, Elbaih A. Pathophysiology and management of different types of shock. Narayana Med J 2017;6(1):14–39.

[2] Citilcioglu S, Sebe A, Ay MO, et al. The relationship between inferior vena cava diameter measured by bedside ultrasonography and central venous pressure value. Pakistan J Med Sci 2014;30(2):310–15.

[3] Seif D, Mailhot T, Perera P, et al. Caval sonography in shock: a noninvasive method for evaluating intravascular volume in critically ill patients. J Ultrasound Med 2012;31(12):1885–90.

[4] Marik PE, Monnet X, Teboul JL. Hemodynamic parameters to guide fluid therapy. Ann Intensive Care 2011;21(1):1 1.

[5] Stawicki SP, Kent A, Patil P, et al. Dynamic behavior of venous collapsibility and central venous pressure during standardized crystalloid bolus: a prospective, observational, pilot study. Int J Crit Illness Injury Sci 2015;5(2):80–4.

[6] Stone MB, Huang J. Inferior vena cava assessment: correlation with CVP and plethora in tamponade. Glob Heart 2013;8(4):323–7.

[7] Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? a systematic review of the literature and the tale of seven mares. Chest 2008;134(1):172–8.

[8] Agarwal D, Soucy Z, Surana A, et al. Role of inferior vena cava diameter in assessment of volume status: a meta-analysis. Am J Emerg Med 2012;30(8):1414–19 e1.

[9] Izakovic M. Central venous pressure—evaluation, interpretation, monitoring, clinical implications. Bratislava Med J 2008;109(4):185–7.

[10] Elbaih AH, Housseini AM, Khalifa MEM. Accuracy and outcome of rapid ultrasound in shock and hypotension (RUSH) in Egyptian polytrauma patients. Chin J Traumatol 2018;21(3):156–62.

[11] Muller L, Bobbia X, Toumi M, et al. Respiratory variations of inferior vena cava diameter to predict fluid responsiveness in spontaneously breathing patients with acute circulatory failure: need for a cautious use. Critical Care 2012;16(5):R188.

[12] Kent A, Bahner DP, Boulger CT, et al. Sonographic evaluation of intravascular volume status in the surgical intensive care unit: a prospective comparison of subclavian vein and inferior vena cava collapsibility index. J Surg Res 2013;184(1): 561–566.

[13] deValk S, Olgers TJ, Holman M, et al. The Caval Index: an adequate non-invasive ultrasound parameter to predict fluid responsiveness in the emergency department? BMC Anesthesiol 2014;14:114.

[14] Airapetian N, Maizel J, Alyamani O, et al. Does inferior vena cava respiratory variability predict fluid responsiveness in spontaneously breathing patients? Critical Care 2015;19:400.

[15] Sawe HR, Haeffele C, Mfinanga JA, et al. Predicting fluid responsiveness using bedside ultrasound measurements of the inferior vena cava and physician gestalt in the emergency department of an urban public hospital in sub-Saharan Africa. PLoS One 2016;11(9):e0162772.

[16] Preau S, Bortolotti P, Colling D, et al. Diagnostic accuracy of the inferior vena cava collapsibility to predict fluid responsiveness in spontaneously breathing patients with sepsis and acute circulatory failure. Crit Care Med 2017;45(3):e290–7.

[17] Elbaih AH, Housseini AM. Validity of shock index, modified shock index, central venous pressure, and inferior vena cava collapsibility index in evaluation of intravascular volume among hypovolemic Egyptian patients. Am J Diagnostic Imag 2018;4(1):5–13.

[18] Bortolotti P, Colling D, Colas V, et al. Respiratory changes of the inferior vena cava diameter predict fluid responsiveness in spontaneously breathing patients with cardiac arrhythmias. Ann Intensive Care 2018;8(1):79.

[19] Long E, Oakley E, Duke T, et al. Does respiratory variation in inferior vena cava diameter predict fluid responsiveness: a systematic review and meta-analysis. Shock 2017;47(5):550–9.

[20] Orso D, Paoli I, Piani T, et al. Accuracy of ultrasonographic measurements of inferior vena cava to determine fluid responsiveness: a systematic review and meta-analysis. J Intensive Care Med 2020;35(4):354–63.

[21] Nagdev AD, Merchant RC, Tirado-Gonzalez A, et al. Emergency department bedside ultrasonographic measurement of the caval index for noninvasive determination of low central venous pressure. Ann Emerg Med 2010;55(3):290–5.

[22] Stawicki SP, Adkins EJ, Eiferman DS, et al. Prospective evaluation of intravascular volume status in critically ill patients: does inferior vena cava collapsibility correlate with central venous pressure? J Trauma Acute Care Surg 2014;76(4):956–63.

[23] Worapratya P, Anupat S, Suwannanon R, et al. Correlation of Caval index, inferior vena cava diameter, and central venous pressure in shock patients in the emergency room. Open Access Emerg Med 2014;6:57–62.

[24] Ilyas A, Ishtiaq W, Assad S, et al. Correlation of IVC diameter and collapsibility index with central venous pressure in the assessment of intravascular volume in critically ill patients. Cureus 2017;9(2):e1025.

[25] Abdelwahab HW, El-Wahab RA. Correlation of bedside ultrasonographic measurement of IVC diameter and caval index with CVP in spontaneously breathing and mechanically ventilated patients. J Emerg Med Critical Care 2017;3(1):2.

[26] Nazemi S, Ahmadinezhad M, Rafiee M, et al. The comparison of the caval index with the invasive method for estimating low CVP in ICU-admitted patients. Int J Med Res Health Sci 2017;6(2):49–52.