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Inferior vena cava peak velocity versus collapsibility index to predict volume status in patients with gastrointestinal bleeding: A pilot study

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Abstract:

OBJECTIVES: Patients presenting to the emergency department (ED) with hemorrhagic shock secondary to acute gastrointestinal (GI) bleeding represent a substantial proportion of the critically ill population. Accurate evaluation of intravascular volume status in these patients is crucial for guiding fluid resuscitation. However, the limitations of invasive methods and inferior vena cava (IVC) diameter/collapsibility measurements in assessing intravascular volume have been widely discussed in the literature. Despite their validation, noninvasive hemodynamic monitoring techniques remain underutilized in the ED. Therefore, the present study aimed to investigate the predictive value of IVC blood flow velocity measured by Doppler ultrasonography in intravascular volume assessment and to compare it with the IVC collapsibility index. Electrical velocity measurement was used as a surrogate marker of volume deficit.

METHODS: Eighteen patients presenting with a preliminary diagnosis of acute GI bleeding were included in the study. The study population consisted solely of patients with suspected hypovolemia; therefore, no normovolemic control group was included in the study. Demographic, vital, and laboratory data were recorded, along with IVC diameter, collapsibility, IVC peak velocity measured by continuous-wave Doppler, and noninvasive hemodynamic monitoring parameters at admission, at the 30th and 60th min, and immediately after completion of fluid replacement. All data were subjected to statistical analysis.

RESULTS: A significant correlation was observed between initial IVC peak velocity and stroke volume variation at admission ($P = 0.009$). The measurement demonstrated a specificity of 100% and a sensitivity of 84%, although the confidence interval was wide due to the small number of patients without volume deficit. No statistically significant differences were detected at the other time points. This can be explained by the ongoing bleeding and the lack of a valid gold standard.

CONCLUSION: Measurement of IVC peak velocity was found to be a significant predictor of hypovolemia, although it showed limited utility for volume monitoring. Furthermore, it demonstrated superior performance compared to IVC diameter and collapsibility measurements. However, the study does not evaluate its ability to distinguish hypovolemia from true normovolemia.

Keywords:

Continuous wave Doppler, inferior vena cava, peak velocity, stroke volume variation, volume status

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Box-ED section**What is already known on the study topic?**

- Inferior vena cava (IVC) diameter and collapsibility are used for volume status but have limited reliability in acute gastrointestinal (GI) bleeding.

What is the conflict on the issue? Has it importance for readers?

- The accuracy of IVC collapsibility is debated; emergency physicians need faster, more reliable tools for fluid resuscitation decisions.

How is this study structured?

- A single-center prospective pilot study comparing continuous wave (CW) Doppler-derived IVC peak velocity with IVC diameter, collapsibility, and noninvasive hemodynamic monitoring in GI bleeding patients.

What does this study tell us?

- IVC peak velocity predicts hypovolemia with high specificity and better accuracy than IVC collapsibility, but has limited role in serial monitoring.

Introduction

Early diagnosis and effective fluid replacement during acute hemorrhage are crucial for reducing mortality and morbidity. Inadequate management of hypovolemic shock can result in hypoperfusion and multiple organ dysfunction. Therefore, accurate and timely evaluation of the adequacy of fluid resuscitation is essential for successful treatment. A significant proportion of patients presenting to the emergency department (ED) are in hypovolemic shock. Among this critical group, gastrointestinal (GI) bleeding is one of the most frequently encountered conditions, often leading to hemorrhagic shock due to acute blood loss.^[1-4]

In the contemporary management of hemorrhagic shock, ultrasonographic methods are widely used alongside physical examination and laboratory testing. However, the reliability of ultrasonographic measurements may be compromised in certain clinical conditions, such as cardiac arrhythmias, age-related changes in vascular elasticity, heart failure, valvular pathologies, and pregnancy, which involve hormonal alterations. Furthermore, the short intravascular half-life of crystalloid fluids presents another significant challenge that limits the effectiveness of fluid resuscitation.^[5-12]

Therefore, investigating new, practical, and reliable noninvasive methods for assessing fluid resuscitation has become a priority in both clinical practice and current literature. In emergency medicine, there is an increasing demand for methods that can be rapidly applied without

compromising patient comfort. To address this need, several devices based on varying principles have been developed to facilitate non-invasive measurements. Compared to invasive techniques, noninvasive methods are advantageous in emergency settings due to their rapid applicability, lack of complication risk, and operator independence. Among these devices, those utilizing electrovelocimetry to measure parameters such as cardiac output (CO), stroke volume (SV), SV variation (SVV), and pulse pressure variation (PPV) are now widely employed for evaluating fluid responsiveness. The ICON device (Osypka Medical), used in our study, is one of the validated instruments in this category.^[13-17]

IVC diameter and collapsibility are widely used indicators but have significant limitations. Although IVC diameter and collapsibility index have been employed for many years to assess fluid responsiveness, it is now recognized that these measurements yield reliable results only in patients with hypovolemia. In contrast, measuring blood flow velocity (peak velocity) across the IVC using Doppler ultrasonography (US) provides a more dynamic hemodynamic parameter, capable of reflecting intravascular volume changes in real time. However, very few studies have evaluated IVC blood flow velocity as a tool for assessing patients' volume status. The pilot study conducted by Biassucci *et al.* served as a reference point for the design of our study.^[18,19]

In our study, we preferred CW Doppler over pulse-wave (PW) Doppler for a more accurate detection of subtle changes in venous flow velocity. While PW Doppler excels in distinguishing depth and low flow velocities, we chose CW Doppler due to the nonpulsatile nature of venous flow and the potential for cursor misalignment with respiratory effort. In addition, when the IVC is nearly or completely collapsed, using PW Doppler becomes more challenging. By performing all measurements from the subxiphoid region, we aimed to minimize the limitations of CW Doppler in deeper tissues. The Diagnostic Ultrasound, 6th Edition, edited by Rumack CM and Levine D, provided guidance for this methodological choice.^[20]

In this study, we aimed to evaluate the predictive value of peak blood flow velocity across the IVC, measured by US, in assessing intravascular volume status and to compare it with the IVC collapsibility index. We hypothesized that IVC peak velocity would outperform the IVC collapsibility index in predicting hypovolemia.

Methods**Study setting**

This single-center, prospective, observational cohort study was conducted at the Department of Emergency

Medicine, Aydın Adnan Menderes University Hospital, between October 15, 2023, and April 15, 2024.

The study was approved by the Ethics Committee of Aydın Adnan Menderes University Faculty of Medicine (Date: October 11, 2023, Decision No: 2023/174) and was supported by the Adnan Menderes University Scientific Research Projects Coordination Unit (Project No: TPF-23052). Written informed consent was obtained from all participants through face-to-face interviews.

Study population

Acute GI bleeding was selected as the model of acute hemorrhage for this study. This choice was made because GI bleeding cases are frequently encountered in the ED, represent a patient group for whom diagnosis and management are time-critical, and allow for direct monitoring of fluid responsiveness.

Patients over 18 years of age presenting to the ED with suspected hypovolemic shock secondary to acute hemorrhage from GI bleeding were included in the study. The study included only patients with suspected hypovolemia due to acute GI bleeding; therefore, no normovolemic control group was included in the study. Exclusion criteria included age under 18 years, pregnancy, known heart failure (clinical history, patients with ejection fraction [EF] below 45% and tricuspid annular plane systolic excursion [TAPSE] <17 mm), atrial fibrillation/flutter, tricuspid regurgitation (minimal and Grade 1 tricuspid regurgitation are included), peripheral vascular disease, and portal vein thrombosis (because we anticipate that this will affect the volume of the IVC). The diagnosis of GI bleeding was confirmed endoscopically in all included cases. Upper/lower GI source differentiation and hemoglobin levels were not used as inclusion or exclusion criteria, as they were not relevant to the primary objective of dynamic volume status assessment. Patient management was performed by primary physicians; no additional laboratory tests were obtained, and no interventions were made regarding treatment during the study.

Data collection and ultrasonographic measurements

At baseline, the shock index was assessed in the enrolled patients. Arterial blood gas parameters, vital signs (temperature, heart rate, blood pressure, and oxygen saturation), anthropometric measurements (height, weight, and body mass index), and Glasgow Coma Scale scores were recorded.

To determine the eligibility criteria, left ventricular EF, TAPSE, and the presence of tricuspid regurgitation were evaluated using parasternal long-axis, parasternal short-axis, and apical four-chamber views.

Ultrasonographic measurements were performed by two operators: the principal investigator, a faculty member, and a 4th-year emergency medicine resident. The principal investigator was a certified WINFOCUS instructor in Türkiye, whereas the resident had completed both basic and advanced US training provided by the Emergency Medicine Association of Türkiye. Before the study, the resident underwent 4 h of didactic training conducted by the principal investigator and completed supervised measurements on 10 patients. Although both operators received standardized training, no formal interobserver reliability analysis was conducted.

Ultrasonographic measurements were conducted using a Sonosite Edge II system (FUJIFILM, Sonosite, Inc.) with a sector probe. IVC measurements were repeated at admission, 30 min, 60 min, and postfluid replacement. All images were recorded in video format and stored in the device memory [Supplementary Figure 1].

Inferior vena cava collapsibility measurement

Measurements were taken in the supine position from the subxiphoid region using a sector probe, approximately 2 cm proximal to the atriocaval junction. M-mode was used, and recordings were obtained for at least three respiratory cycles.

Blood flow velocity across inferior vena cava

In the supine position, peak velocity was measured using continuous-wave (CW) Doppler mode with a sector probe, 2 cm proximal to the atriocaval junction, for at least three respiratory cycles. The average of the measured values was recorded. Respiratory effort was not quantitatively standardized; however, operators instructed patients to maintain normal tidal breathing to reduce variability.

In this study, the ICON device (Osypka Medical GmbH, Berlin, Germany), which is based on the principle of noninvasive electrical velocity measurement, was used as a surrogate reference test, although it is not a gold standard method. Parameters including CO, SV, SVV, PPV, corrected flow time (FTc), thoracic fluid content (TFC), stroke index and systemic vascular resistance (SVR) were measured at admission, at the 30th and 60th min of blood product replacement, and immediately after completion of the transfusion. No clinical outcome data (intensive care unit admission, mortality, or rebleeding) were collected, as the study focused exclusively on early hemodynamic measurements.

Statistical analysis

Data were analyzed using Jamovi for Windows version 2.4.8. Categorical variables are expressed as n (%). Normally distributed numerical variables are presented as mean $\pm$ standard deviation, whereas nonnormally

interquartile distributed numerical variables are reported as median with interquartile range. Comparisons between categorical variables were performed using the Chi-square test. When any expected cell count is under five or more than 20% of cells have expected counts below five, Fisher's exact test is used. For multiple comparisons of continuous variables, repeated measures ANOVA or the Friedman test was applied. *Post-hoc* analyses of continuous variables were conducted using the Greenhouse-Geisser correction, Tukey's *post hoc* test, or the Durbin-Conover test. To determine the predictive value of ultrasonographic measurements in identifying hypovolemia, receiver-operating characteristic (ROC) curve analysis was performed, and the area under the curve (AUC), sensitivity, specificity, positive predictive value, and negative predictive value (NPV) were calculated. Results were given with 95% confidence interval (CI), and *P* values. *P* < 0.05 was considered statistically significant.

Results

Eighteen patients were included in the study, comprising 9 (50%) females and 9 (50%) males [Table 1 and Figure 1]. The statistical analyses of these data are presented in tables along with their corresponding descriptions. All patients were clinically considered to have potential intravascular volume loss due to active GI bleeding and

required erythrocyte suspension transfusion. Details of volume replacement therapy, urine output, and cumulative fluid balance are presented in Table 2.

According to our surrogate reference test (SVV), 13 patients (72.2%) had a volume deficit, whereas 5 patients (27.8%) did not. In patients with volume deficit, the IVC peak velocity was 71 ± 11.6 cm/s, compared to 54.5 ± 6.21 cm/s in patients without a volume deficit (*P* = 0.009). Conversely, based on initial IVC ultrasonographic measurements, 13 patients (72.2%) were identified as hypovolemic, whereas 5 patients (27.8%) were not. The IVC peak velocity in hypovolemic patients was 68.1 ± 12.9 cm/s, whereas in nonhypovolemic patients, it was 62.0 ± 12.3 cm/s (*P* = 0.377).

ROC analysis was performed to determine the predictive value of admission IVC peak velocity in identifying volume deficit [Figure 2]. According to this analysis, when an admission IVC peak velocity threshold of 64.2 cm/s was applied, the sensitivity for predicting volume deficit was

Table 1: Descriptive statistics of patients' demographic characteristics, vital signs, blood gas values, hemorrhagic shock parameters, and echocardiographic findings at admission

Category	Parameter	Value, n (%) or mean±SD
Demographics	Age (years)	69.1±15.1
	Sex	
	Female	9 (50)
	Male	9 (50)
	Height (cm)	164±7.9
Vital signs	Weight (kg)	68±11.8
	BMI (kg/m ²)	25.2±3.6
	SBP (mmHg)	112±20.2
	DBP (mmHg)	63.1±11.8
	MAP (mmHg)	79.5±13.2
	HR (/min)	100±15.1
	Oxygen saturation (%)	98 (1.75)
	Temperature (°C)	36 (0.275)
	GCS	15 (0)
	pH	7.38±0.06
Blood gas parameters	pO ₂ (mmHg)	29.3 (12.6)
	pCO ₂ (mmHg)	40.5 (4.55)
	HCO ₃ (mmol/L)	23.4±4.5
	Lactate (mmol/L)	2.45 (1.67)
	Base deficit (mmol/L)	-0.55±5.17
Hemorrhagic shock indicators	Shock index	0.966±0.279
	Hb level at admission	7.68±2.80
	Lowest Hb level recorded	3.70
Source of bleeding	Upper GI	15 (83.3)
	Lower GI	3 (16.7)
Echocardiography	EF (%)	64.4±9.76
	TAPSE (mm)	21.5 (5.5)

TAPSE: Tricuspid annular plane systolic excursion, Hb: Hemoglobin, GI: Gastrointestinal, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, HR: Heart rate, GCS: Glasgow Coma Scale, EF: Ejection fraction

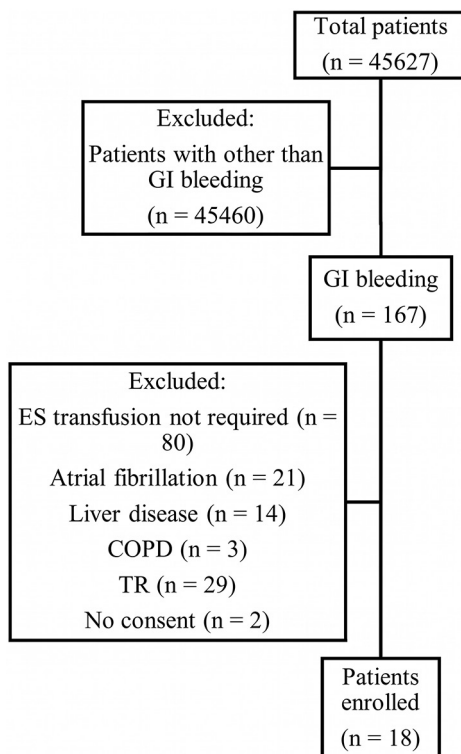


Figure 1: Flowchart of patient enrollment. GI: Gastrointestinal, ES: Erythrocyte suspension, COPD: Chronic obstructive pulmonary disease, TR: Tricuspid regurgitation

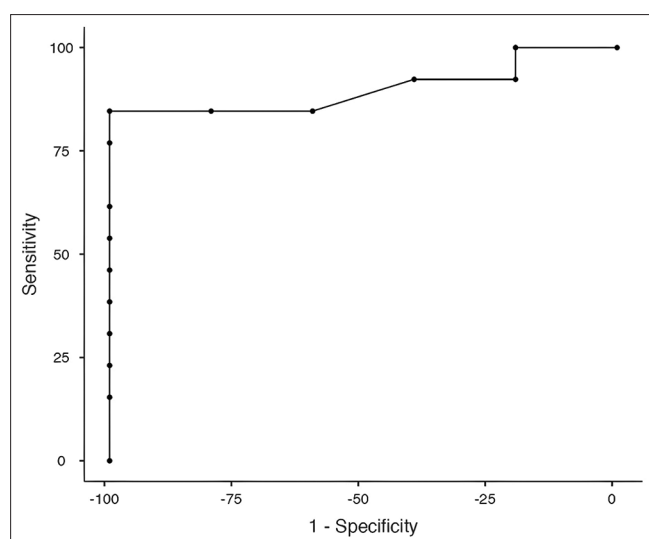


Figure 2: Receiver-operating characteristic curve of admission inferior vena cava peak velocity at a threshold of 64.2 cm/s for predicting volume deficit

Table 2: Descriptive statistics of patients' volume replacement therapy, urine output, and fluid balance data

Parameter	Value, n (%) or mean±SD
Fluids and urine output prior to admission (mL)	
Crystalloid	400 (800)
ES-colloid	0 (50)
Total fluid	400 (800)
Urine	62.5 (225)
Fluids and urine output at 30 min (mL)	
Crystalloid	450 (800)
ES-colloid	100±72.9
Total fluid	568 (726)
Urine	87.5 (350)
Fluids and urine output at 60 min (mL)	
Crystalloid	500 (763)
ES-colloid	200 (115)
Total fluid	735 (644)
Urine	288 (465)
Fluids and urine output after replacement (mL)	
Crystalloid	744±564
ES-colloid	320 (270)
Urine	401±327
Cumulative values	
Total fluid intake	1123±605
Total urine output	438 (575)
Balance	743±671
Ratio of ES-colloid to total fluid (%)	43±22.5

ES: Erythrocyte suspension

84.6% (95% CI: 54.55-98.08), specificity was 100% (95% CI: 42.82-100), positive predictive value was 100%, NPV was 71.4% (95% CI: 41.13-89.95), and accuracy was 88.9% (95% CI: 65.29-96.63), AUC was 0.900.

IVC peak velocity values obtained at 30 min, 60 min, and immediately after transfusion did not show statistically

significant differences between volume-deficient and non-volume-deficit groups (all $P > 0.05$). Similarly, serial measurements of IVC diameter, collapsibility, and electrovelocimetric parameters (including SVV, FTc, TFC, SV, CO, and CI) did not demonstrate meaningful changes over time [Table 3].

At admission, comparison of IVC and SVV groups (volume deficit vs. no volume deficit) revealed no significant difference ($P = 0.268$) [Table 4].

Discussion

In our pilot study, we found that IVC peak velocity showed promise in outperforming the IVC collapsibility index in predicting volume deficit. Considering the measurement methods mentioned in the recent literature, we investigated the feasibility of measuring blood flow velocity across the IVC using CW Doppler as an alternative method.^[21,22]

Among the 18 patients included in this study, a comparison of admission IVC peak velocity values with admission SVV values, considered the surrogate reference test, revealed a statistically significant correlation. According to the ROC analysis results, admission IVC peak velocity achieved high sensitivity (84.6%) and specificity (100%) in predicting volume deficit. However, the wide CI reflects the small number of patients ($n = 5$) in the non-volume-deficit group, and the specificity estimate should be interpreted with caution.

The clinical significance of IVC peak velocity in predicting hypovolemia may be attributed to compensatory mechanisms such as tachycardia and vasoconstriction induced by neurohormonal and neurohumoral responses to volume loss. These physiological changes reduce IVC diameter and, according to the Bernoulli principle, increase blood flow velocity. In our study, although heart rate decreased and IVC diameter increased over time, IVC peak velocity demonstrated an upward trend (although not statistically significant), which may reflect the intravascular volume expansion provided by fluid resuscitation. However, delayed neurohormonal and neurohumoral responses in the acute phase may have limited this relationship.^[23]

Although IVC diameter and the collapsibility index are widely used in emergency practice, recent literature has highlighted significant limitations of these methods, including controversies regarding the optimal measurement site and concerns about their reliability. In our study, when we compared IVC diameter and collapsibility measurements with SVV as the reference test, their predictive value for identifying hypovolemia

Table 3: Statistical evaluation of patients' vital signs, ultrasonographic measurements, and electrovelocimetric measurements at admission, 30 min, 60 min, and after infusion

Parameter	Initial	30 min	60 min	After infusion	P
Vital findings					
SBP (mmHg)	112±20.2	120±24.0	118±22.2	123±24.4	0.095*
DBP (mmHg)	63.1±11.8	63.3±12.5	66.0±11.5	67.4±10.6	0.473**
MAP (mmHg)	79.5±13.2	80.4±15.1	84.0±12.9	86.0±13.1	0.113*
HR (bpm)	100±15.1	91.1±13.6	89.2±13.5	88.1±17.4	0.004*
Shock index	0.97±0.28	1.18±0.51	1.23±0.53	1.45±0.43	0.104**
Ultrasonographic measurements					
IVC min (cm) ^a	0.74±0.39	0.85±0.33	0.98±0.47	1.07±0.45	0.01**
IVC max (cm) ^b	1.30±0.52	1.43±0.46	1.64±0.48	1.79±0.40	<0.001*
CI (%)	43.6±20.0	40.3±11.8	41.3±22.0	36±14	0.836**
IVC peak velocity (cm/s)	66.4±12.7	70.4±18.7	70.4±18.2	73.3±20.2	0.327*
Electrovelocimetric measurements					
SVV (%)	17.9±8.26	17.6±6.38	18.1±8.09	20.1±9.48	0.756**
FTc (ms)	311±24.4	319±15.7	309±25.9	321±21.1	0.183*
TFC	25.5±3.84	27.2±7.03	27.1±6.44	27.2±7.03	0.825**
SV (mL)	63.5±13.7	63.9±12.8	60.7±14.8	63±12.7	0.446*
SI (mL/m ²)	37.2±6.46	37±7.49	36.4±9.98	36±10.5	0.511**
CO (L/min)	5.79±1.63	5.72±1.27	5.38±1.53	5.10±1.20	0.942**
SVR (dynes/s/cm ⁻⁵) ^c	1119±507	1103±400	1286±440	1347±406	0.002**

Values are presented as mean±SD. *Repeated measures ANOVA, **Friedman, ^aSignificant differences among initial–60 min, 30 min–after infusion, and initial–after infusion values, ^bSignificant differences among 60 min–after infusion and 30 min–after infusion values, ^cSignificant differences among initial–60 min, 30 min–60 min, and 30 min–after infusion values. SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, HR: Heart rate, IVC: Inferior vena cava, CI: Cardiac index, SVV: Stroke volume variation, FTc: Corrected flow time, TFC: Thoracic fluid content, SV: Stroke volume, SI: Stroke index, CO: Cardiac output, SVR: Systemic vascular resistance

Table 4: Statistical evaluation of admission inferior vena cava peak velocity measurements according to sex, initial inferior vena cava group, initial stroke volume variation group, initial corrected flow time group, and initial shock index group

Variable	Category	n (%)	IVC peak velocity (cm/s) (mean±SD)	P*
Sex	Female	9 (50.0)	66.3±12.9	0.967
	Male	9 (50.0)	66.5±13.3	
Initial IVC group	Volume deficit present	13 (72.2)	68.1±12.9	0.377
	Volume deficit absent	5 (27.8)	62.0±12.3	
Initial SVV group	Volume deficit present	13 (72.2)	71.0±11.6	0.009
	Volume deficit absent	5 (27.8)	54.5±6.21	
Initial FTc group	Normal	13 (72.2)	65.5±13.9	0.666
	Low	5 (27.8)	68.6±9.85	
Initial SI group	Shock present	10 (55.6)	65.1±13.8	0.646
	Shock absent	8 (44.4)	68.0±11.9	

*Student's t-test. IVC: Inferior vena cava, SVV: Stroke volume variation, FTc: Corrected flow time, SI: Shock index, SI: Stroke index

was found to be insufficient [Table 4]. Contributing factors include age-related changes in vascular elasticity, the ellipsoid anatomical structure of the IVC, the accuracy of the ultrasonographic measurement site, right and left heart dysfunction, and cardiac arrhythmias. These limitations have been discussed in previous studies by Di Nicolò *et al.*, Biasucci *et al.*, and Tedaldi *et al.*^[5,18,24] Despite employing broad exclusion criteria to minimize such confounders, our results still indicated that IVC diameter and the collapsibility index are inadequate for identifying hypovolemia.

Furthermore, IVC peak velocity measurements were repeated at the 30th and 60th min and after fluid replacement. When these values were compared with SVV and IVC groups at the corresponding time points, no statistically significant differences were observed. Based on these results, we believe that CW Doppler-derived IVC peak velocity measurement may have limited utility in monitoring intravascular volume status. Despite administering an average of 1123 ± 605 mL of total fluids (43 ± 22.5% of which was erythrocyte suspension), no statistically significant change was observed in shock index measurements. This is attributable to ongoing GI bleeding, suggesting that fluid resuscitation was suboptimal. Consequently, IVC peak velocity measurements may not have adequately reflected the expected changes in volume status during monitoring. Therefore, further studies with more appropriate resuscitation strategies, hemorrhage control, and larger sample sizes are warranted. In addition, SVV measured by electrical velocimetry is the surrogate reference test we use to assess intravascular volume status. Therefore, the relationship between IVC peak velocity and SVV may indicate a possible physiological link rather than reflecting absolute diagnostic accuracy. This limitation of the reference test is also a possible reason for the lack of significant results in serial measurements.

The small number of cases within SVR subgroups limited the statistical power of our study. Nonetheless, the upward trend observed in SVR, particularly after

the 30th min, may be explained by vascular filling associated with fluid replacement and the onset of a vasoconstriction response.

Other hemodynamic parameters obtained through noninvasive monitoring (FTc, TFC, SV, CO, and CI) did not show statistically significant differences. This may be attributed to the small sample size, preserved cardiac function of the patients, insufficient fluid resuscitation, and ongoing bleeding.

Limitations

Although our study achieved the required population size based on power analysis, it was conducted on a relatively small sample compared to existing literature.

The study population consisted of patients with suspected hypovolemia due to acute GI bleeding and did not include a true normovolemic control group. This makes it impossible to demonstrate the diagnostic performance of IVC peak flow velocity between hypovolemic and normovolemic patients.

SVV obtained from an electrical velocimetry device was used as a surrogate reference test, although it is not a validated gold standard for assessing intravascular volume in spontaneously breathing ED patients or in the context of active hemorrhage.

Ongoing bleeding during the study period may have influenced serial hemodynamic measurements, potentially masking true physiological responses to transfusion. The timing of the postreplacement measurement was recorded as occurring immediately after transfusion, but no standardized time interval was applied, which may have contributed to variability.

The small sample size resulted in wide CIs, particularly for specificity estimates, and limits the statistical power to detect meaningful differences in serial measurements.

Finally, this was a single-center pilot study conducted in a specific clinical population; the generalizability of the findings is limited, and larger, controlled studies using validated reference standards are required to determine the true diagnostic value of IVC peak velocity.

Conclusion

In this prospective pilot study of patients presenting with suspected acute GI bleeding, IVC peak blood flow velocity measured by CW Doppler demonstrated a significant association with SVV-defined volume deficit at admission, whereas conventional IVC measurements showed limited discriminatory value. However, the study design does not allow assessment of the ability

of peak velocity to distinguish hypovolemia from true normovolemia, as all enrolled patients were clinically suspected to have intravascular volume loss.

Serial Doppler and ultrasonographic measurements did not reliably track changes during transfusion, likely influenced by ongoing bleeding, variable clinical responses, and the use of SVV as a surrogate reference standard. Therefore, the clinical role of IVC peak velocity in dynamic volume monitoring remains uncertain.

Overall, IVC peak velocity may serve as a supplementary parameter for assessing volume deficit at presentation among patients with suspected GI bleeding, but its diagnostic value should be interpreted cautiously. Further studies, including normovolemic controls, validated reference standards, and standardized resuscitation protocols, are required to clarify its potential utility in emergency settings.

Author contributions statement

YEÖ: Formal analysis (equal); Project administration (lead); writing – original draft (lead); writing – review and editing (equal). YEÖ, OCB: Conceptualization, Methodology. AMS, OCB, ED: writing – original draft (equal). YEÖ: Formal analysis (equal); Methodology (lead). AMS, OCB, ED: Investigation (equal); Writing – original draft (supporting). YEÖ, OCB, AMS, ED: Investigation (equal); Writing – review and editing (equal).

Conflicts of interest

None Declared.

Ethical approval

The study was conducted with the approval of the Aydın Adnan Menderes University Faculty of Medicine Noninvasive Clinical Research Ethics Committee. (Date: 11/10/2023, Decision No: 2023/174).

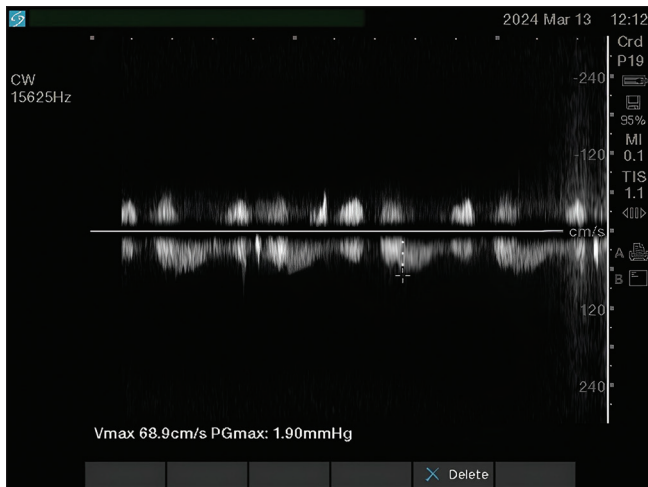
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Supplementary Figure 1: Example of inferior vena cava peak velocity measurement using continuous-wave Doppler