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Bedside internal jugular vein ultrasonography as a prognostic tool in COPD exacerbations

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Abstract

Aims: To evaluate if internal jugular vein (IJV) diameter changes, assessed by bedside ultrasonography, are associated with the severity and prognosis of Chronic Obstructive Pulmonary Disease (COPD) exacerbations, and to determine their utility in guiding clinical management in the emergency department. **Material and methods:** This prospective, observational pilot study included 45 patients presenting to the emergency department with a COPD exacerbation. Patients underwent right IJV ultrasonography at presentation and 2 hours post-treatment. IJV diameters (IJVMAX, IJVMIN, IJVCOMP) and derived collapsibility indices (INDEX1, INDEX2) were measured by a single, blinded physician. Outcomes analyzed were hospital/ICU admission and 1-month mortality. **Results:** 45 patients (mean age 64.96 years) were included. Following treatment, IJVMIN decreased ($p=0.026$) and collapsibility (INDEX1) increased significantly ($p=0.002$). 1-month mortality (n=4) was associated with higher post-treatment IJVCOMP2 ($p=0.041$). Patients admitted to the ICU (n=9) had significantly higher IJV diameters and lower collapsibility indices ($p<0.05$). For predicting ICU admission, IJVMIN (cut-off ≥1,05cm) had an AUC of 0.861 with 86.1% specificity. **Conclusions:** Bedside IJV ultrasonography helps predict severity and monitor treatment response in COPD exacerbations. IJV parameters are associated with ICU admission and mortality, suggesting they are valuable, non-invasive decision support tools for emergency physicians.

Keywords: COPD Exacerbation; Internal jugular vein; Ultrasonography; Emergency Department; Prognosis

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common and significant respiratory disease characterized by progressive airflow limitation, presenting with symptoms such as cough, sputum production, and dyspnea. It is reported that approximately 10% of individuals over the age of 40 have COPD, with the prevalence increasing with age [1]. A study conducted in Turkey determined the prevalence of COPD to be as high as 19.1%, a figure that clearly underscores the public health importance of the disease [2]. Causing over 120,000 deaths annually in the United States, COPD was ranked as the third leading cause of death worldwide prior to the COVID-19 pandemic [3]. According to the Global Burden of Disease (GBD) 2021 data, COPD is the eighth leading cause of disability in Turkey, affecting approximately 787 per 100,000 people and resulting in about 35 deaths per 100,000 people annually [4].

COPD is characterized by frequent outpatient visits and recurrent hospitalizations, imposing a significant burden on the healthcare system, particularly during periods of exacerbation. The fundamental pathophysiology

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of the disease is based on expiratory airflow limitation, which is known to develop in airways smaller than 2 mm in diameter [5] . This obstruction arises from mechanisms such as the constriction of airway smooth muscles, inflammation-induced thickening and fibrosis of small airway walls, loss of airways due to parenchymal destruction, and dynamic airway collapse resulting from reduced elastic recoil pressure [6,7] .

An increased respiratory rate shortens the expiratory time, preventing the complete exhalation of the tidal volume. This phenomenon, defined as "dynamic hyperinflation," leads to an increase in end-expiratory lung volume and causes a sensation of dyspnea, even without accompanying hypoxemia [8]. A COPD exacerbation is a clinical event characterized by a worsening of symptoms over a period of up to 14 days, often triggered by infections, air pollution, or other airway irritants, and is associated with increased local and systemic inflammation. It typically presents with tachypnea, tachycardia, increased cough, sputum production, and dyspnea [9]. During an exacerbation, the intensification of air trapping and hyperinflation leads to increased intrathoracic pressure, which in turn elevates pulmonary vascular resistance and imposes a greater load on the right heart [10]. This rise in intrathoracic pressure affects not only the pulmonary circulation but also systemic venous return, potentially causing marked distension in central venous structures like the internal jugular vein (IJV) [11]. In clinical practice, jugular venous distension is frequently observed in patients experiencing a COPD exacerbation and is regarded as a physical sign indicating a disruption of cardiopulmonary equilibrium. We hypothesize that pulmonary hypertension in patients with COPD can be predicted by evaluating structural and functional changes in the IJV [12].

In the GOLD 2024 report, the severity of COPD exacerbations is classified as mild (dyspnea on VAS <5, respiratory rate <24/min, heart rate <95/bpm, resting SpO_2 ≥92% with a desaturation of ≤3%, and CRP <10 mg/L), moderate (presence of at least three previous criteria with hypoxemia – $PaO_2 \leq 60$ mmHg and/or hypercapnia – $PaCO_2 > 45$ mmHg), or severe (In addition to the moderate criteria, the presence of new-onset hypercapnia accompanied by acidosis) according to the Rome classification [13].

Most current assessment methods rely on clinical observation and symptom scoring. However, these parameters may not always accurately reflect the patient's current hemodynamic status, thereby complicating admission or discharge decisions in the emergency department. Consequently, there is a growing need for quantitative, rapid, and non-invasive methods.

Ultrasound (US) assessment of IJV diameters indirectly monitors intrathoracic pressure changes. In COPD exacerbations, it is postulated that changes in IJV diameters, associated with the increased load on the right heart, may correlate with disease severity. This study aims to evaluate whether changes in IJV diameters are associated with the severity and prognosis of COPD exacerbation and to investigate whether these changes can provide guidance for clinical classification and patient management.

This study is novel in its focus on a topic that has been sparsely addressed in the literature and its exploration of integrating a non-invasive and practical method like US into COPD management. It is anticipated that our findings will shed light on future advanced research in this field and make a significant contribution to the literature.

Material and method

The study was conducted without the approval of the ethics committee as it evaluated the educational activity, and publicly available data were used. Ethical approval was granted by the Gaziantep University Clinical Research Ethics Committee (Decision No: 2024/215, Date: July 31, 2024), and the study was conducted in accordance with the ethical principles of the Declaration of Helsinki

Study design

This prospective, observational pilot study was conducted in the emergency department of a tertiary care hospital between July 1, 2024, and December 31, 2024. The sample size was determined based on a preliminary power analysis with 80% power and a 5% significance level, resulting in the inclusion of 45 patients. A statistician determined that the minimum number of patients required to find a statistically significant difference in the strong relationship between the MMRC score and pulmonary artery pressure after treatment, compared to a medium-level relationship (ρ=0.8-0.5), was 38 (α=0.05, 1-β=0.90). It was recommended to increase this number by 10% to account for potential patient dropouts. The analysis was performed using Gpower 3.1.

Inclusion criteria were as follows: presenting to the emergency department; diagnosed with COPD exacerbation according to GOLD criteria; aged 18 years or older; fully conscious individuals able to provide cooperation and comply with study procedures. Exclusion criteria were as follows: pregnancy, presence of a neck mass or catheter; pericardial effusion; receiving invasive mechanical ventilation support; intellectual disability, individuals with anatomical limitations precluding ultrasonographic assessment.

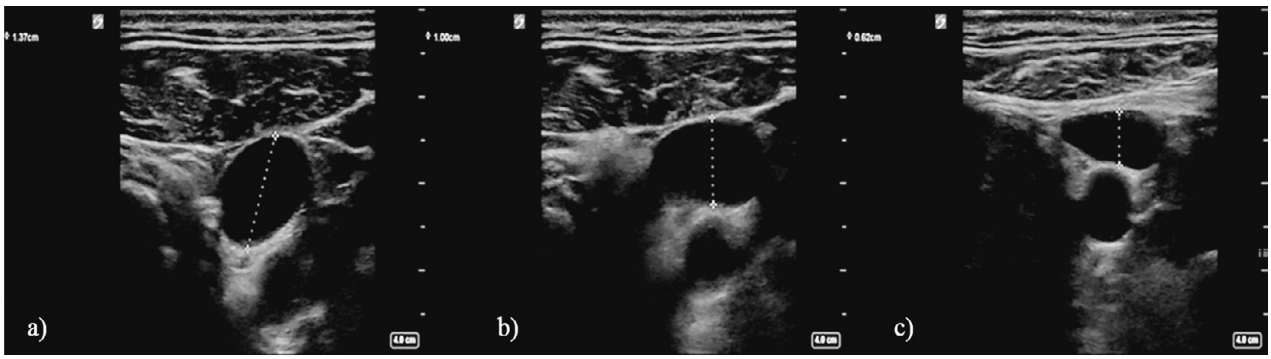

Fig 1. Ultrasonographic measurement technique of the internal jugular vein (IJV): a) Measurement of the maximum anterior-posterior diameter (IJV-MAX); b) Measurement of the minimum diameter during inspiration (IJV-MIN), showing venous collapsibility; c) Assessment of compressibility (IJV-COMP) with standardized controlled compression; note the collapsed vein above the intact, circular carotid artery.

The maximum (IJVMAX), minimum (IJVMIN), and compression-induced (IJVCOMP) diameters of the internal jugular vein were measured. Based on these measurements, two collapsibility indices were calculated: INDEX1=(IJVMAX–IJVMIN)/IJVMAX and INDEX2=(IJVMAX–IJVCOMP)/IJVMAX. The same parameters were re-measured after treatment and designated as IJVMAX2, IJVMIN2, and IJVCOMP2, and the post-treatment indices were calculated as follows: INDEX-POST1=(IJVMAX2–IJVMIN2)/IJVMAX2 and INDEX-POST2=(IJVMAX2–IJVCOMP2)/IJVMAX2

Data collection

Patient selection was based on a comprehensive assessment of medical history, hospital records, and physical examination. Individuals with suspected decompensated heart failure were excluded to ensure that any venous findings could be primarily attributed to COPD. Consequently, the final study population consisted exclusively of patients whose chronic comorbidities were in a compensated state.

After obtaining informed consent from the patients, their baseline vital signs (blood pressure, respiratory rate, heart rate, temperature, SpO_2), demographic data (age, gender, comorbidities), and laboratory values (arterial blood gas, complete blood count, CRP) were recorded.

Individuals arriving at the emergency department underwent chest X-rays from a posterior-anterior view to detect conditions that elevate intrathoracic pressure, such as pneumothorax,large pleural effusion. Those with initial findings were not included in the study.

Each patient underwent right IJV US twice: once upon presentation and 2 hours after the administration of the standard COPD exacerbation treatment protocol. The treatment protocol was administered in the emergency department according to the patient's clinical severity and consisted of standard approaches, including bronchodilators, systemic steroids, oxygen therapy, and antibiotics when indicated.

US measurements were performed using a Fujifilm Sonosite X-Porte machine. The patients were positioned in a 45° semi-recumbent position with their heads slightly turned to the contralateral side. A 6–13 MHz high-frequency linear probe was used. The IJVMAX, IJVMIN, and IJVCOMP values were acquired using both 2D and M-mode imaging (fig. 1). To measure the IJVCOMP, a standardized method of manual compression was employed using the linear transducer. Following the methodology described by Parikh et al [15], this technique involved applying vertical compression to displace the skin surface to a depth of approximately 2 cm. This standardized displacement depth was maintained to ensure a consistent compression technique across all patients.

All measurements were performed by an emergency medicine specialist with 5 years of experience in US, who was blinded to the clinical findings. Prior to the study, this physician received one month of specialized training in jugular vein measurements from a radiologist with over 10 years of experience. To ensure standardization, all US assessments were performed by the same physician.

Outcome endpoints

The associations between the changes in pre- and post-treatment IJV diameters and indices and the following outcomes were analyzed: fospital admission (to a general ward or the intensive care unit); discharge from the emergency department; 1-month mortality.

Statistical analysis

Data were summarized using mean, standard deviation, and median (minimum–maximum) for numerical variables, and frequency and percentage for categorical variables. The normality of distribution was assessed using the Shapiro-Wilk test. For comparisons between two groups, the independent samples t-test or Mann-Whitney

U test was used, while one-way ANOVA or the Kruskal-Wallis test was employed for multiple group comparisons. For paired measurements, the paired t-test or the Wilcoxon signed-rank test was applied. The Chi-square test was used for categorical variables, Spearman's correlation analysis was used for assessing correlations between variables, and Receiver Operating Characteristic (ROC) analysis was used to determine cut-off values. The Bonferroni correction was applied for multiple comparisons. All analyses were performed using SPSS version 22.0, and a p-value of <0.05 was considered statistically significant.

Results

A total of 54 patients were evaluated, and 9 were excluded [invasive mechanical ventilation (n=2), neck mass (n=1), intellectual disability (n=1), and incomplete data (n=5)]. Of the 45 patients included in the study, 88.9% (n=40) were male and 11.1% (n=5) were female. The mean age was 64.96 ± 10.23 years. At least one comorbidity was present in 44.4% of the patients.

When pre- and post-treatment IJV diameters and their derived indices were compared, the minimum diameter (IJVMIN) showed a significant decrease (p=0.026), while INDEX1 showed a significant increase (p=0.002) (Table I). These findings suggest a reduction in intrathoracic pressure and an increase in venous collapsibility following treatment.

During the study period, 4 patients (8.88%) died. In patients who died, the post-treatment IJVCOMP2 value was found to be statistically significantly higher compared to the surviving group (p=0.041) (Table II).Regarding the other parameters, no significant difference was detected between the deceased and survivor groups. This finding indicates that IJVCOMP2 is associated with mortality.

According to the COPD exacerbation severity classification adapted from the Rome classification, notable differences were observed in IJVMAX, IJVMIN, IJVCOMP, INDEX1, IJVMAX2, IJVMIN2, and IJVCOMP2 parameters. in patients grouped as mild, moderate, and severe (p<0.05 (Table III)). The IJVMAX value was 1.06±0.32 (median 0.93) in the mild group and 1.77±0.62 (median 1.82) in the severe group (p=0.043). The INDEX1 parameter was found to be 47.88±12.94 (median 46.74) in the mild group and 24.13±8.34 (median 26.07) in the severe group (p=0.008). In the other parameters, the severe group also showed significantly higher values compared to the mild and moderate groups. In post-hoc analyses, intergroup comparisons are indicated by different letters (a, ab, b), with no significant difference found between groups sharing the same letter.

Data regarding hospital admissions were analyzed. Eighteen patients (40%) were admitted to the ward, 9 (20%) were admitted to the intensive care unit (ICU), and the remaining 18 (40%) were discharged from the emergency department. When comparing patients admitted to the ICU (n=9) with those not admitted to the ICU (i.e., admitted to the ward or discharged, n=36), the IJV diameters (IJVMAX, IJVMIN, IJVCOMP, IJVMAX2, IJVMIN2, IJVCOMP2) were found to be statistically significantly higher in the ICU group (p<0.05). Furthermore, collapsibility parameters such as INDEX1 and INDEX2 showed lower values in the ICU group. These results indicate that IJV diameters are associated with the need for ICU admission and that collapsibility is reduced in these patients (Table IV).

Table I. Internal jugular vein diameters

	Mean±SD	Median (Q1-Q3)	p-value
IJVMAX	1.29±0.45	1.20 (0.93–1.60)	0.981§
IJVMAX2	1.26±0.43	1.19 (0.99–1.46)	
IJVMIN	0.85±0.43	0.74 (0.53–1.10)	**0.026*§**
IJVMIN2	0.76±0.42	0.65 (0.44–0.94)	
IJVCOMP	0.56±0.37	0.44 (0.28–0.88)	0.051§
IJVCOMP2	0.51±0.39	0.43 (0.2–0.58)	
INDEX1	36.62±15.20	34.96 (25.25–46.74)	**0.002*‡**
INDEX-POST1	43.49±16.79	45.28 (29.86–53.85)	
INDEX2	59.61±16.54	58.87 (49.47–74.8)	0.089‡
INDEX-POST2	63.19±17.35	63.96 (50.85–77.27)	

IJVMAX: Pre-treatment Internal Jugular Vein Maximum Diameter, IJVMIN: Pre-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP: Pre-treatment Internal Jugular Vein Compression Diameter, INDEX1: (IJVmax-IJVmin)/IJVmax, INDEX2: (IJVmax-IJVcomp)/IJVmax, IJVMAX2: Post-treatment Internal Jugular Vein Maximum Diameter, IJVMIN2: Post-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP2: Post-treatment Internal Jugular Vein Compression Diameter, INDEX-POST1: Post-treatment Index 1, INDEX-POST2: Post-treatment Index 2; ‡Paired t-test, §Wilcoxon test.

Table II. Relationship between measurement parameters and mortality

	Deceased		Survived		
	Mean ± SD	**Median (Q1–Q3)**	**Mean ± SD**	**Median (Q1–Q3)**	**p-value**
IJVMAX	1.61 ± 0.73	1.56 (1.14–2.07)	1.26 ± 0.41	1.20 (0.93–1.49)	0.406§
IJVMIN	1.21 ± 0.59	1.20 (0.81–1.62)	0.81 ± 0.40	0.74 (0.53–0.98)	0.163§
IJVCOMP	0.95 ± 0.56	0.99 (0.61–1.30)	0.52 ± 0.34	0.41 (0.28–0.66)	0.108§
INDEX1	25.63 ± 8.17	27.24 (19.18–32.07)	37.69 ± 15.36	36.24 (25.26–47.87)	0.131‡
INDEX2	45.04 ± 16.49	37.45 (36.46–53.62)	61.03 ± 16.04	59.45 (50.98–75.00)	0.064‡
IJVMAX2	1.65 ± 0.48	1.59 (1.35–1.97)	1.22 ± 0.41	1.19 (0.98–1.39)	0.069§
IJVMIN2	1.10 ± 0.44	0.95 (0.84–1.37)	0.72 ± 0.41	0.63 (0.44–0.90)	0.069§
IJVCOMP2	0.80 ± 0.31	0.74 (0.55–1.05)	0.48 ± 0.39	0.40 (0.20–0.57)	**0.041***§
INDEX-POST1	34.27 ± 8.44	33.97 (28.40–40.13)	44.39 ± 17.19	46.45 (29.86–55.56)	0.254‡
INDEX-POST2	51.66 ± 10.70	48.91 (44.88–58.44)	64.32 ± 17.54	64.10 (55.47–77.53)	0.166‡

IJVMAX: Pre-treatment Internal Jugular Vein Maximum Diameter, IJVMIN: Pre-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP: Pre-treatment Internal Jugular Vein Compression Diameter, INDEX1: (IJVmax-IJVmin)/IJVmax, INDEX2: (IJVmax-IJVcomp)/IJVmax, IJVMAX2: Post-treatment Internal Jugular Vein Maximum Diameter, IJVMIN2: Post-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP2: Post-treatment Internal Jugular Vein Compression Diameter, INDEX-POST1: Post-treatment Index 1, INDEX-POST2: Post-treatment Index 2; ‡Paired t-test, §Wilcoxon test.

The performance of IJV measurement parameters in predicting admission to the intensive care unit was evaluated with ROC analysis (fig 2). For the IJMIN parameter, a cut-off value of ≥1.0500 cm yielded a sensitivity of 77.8%, a specificity of 86.1%, and an Area Under the Curve (AUC) of 0.861. Similarly, other parameters such as IJVMAX (cut-off ≥1.5050 cm, sensitivity 77.8%, specificity 83.3%) and IJCOMP (cut-off ≥0.8250 cm, sensitivity 77.8%, specificity 86.1%) were also found to be significant in predicting ICU admission.

Discussion

In this prospective observational study, we evaluated changes in IJV diameters via bedside US in emergency department patients with COPD exacerbation, hypothesizing that these measurements reflect intrathoracic pressure variations and associated hemodynamic shifts. Our findings support this hypothesis, demonstrating that IJV parameters can objectively quantify disease severity, treatment response, and prognostic indicators, potentially aiding clinical decision-making in resource-limited settings.

Post-treatment decreases in IJVMIN (p=0.026) and IJVCOMP (p=0.051), alongside an increase in INDEX1 (p=0.002), align with reduced air trapping and dynamic hyperinflation, key pathophysiological features of COPD exacerbations that elevate intrathoracic pressure and impair venous return [6,7]. No change in IJVMAX (p=0.981) suggests this parameter may represent a more stable anatomical baseline, less influenced by acute pressure fluctuations. These observations underscore US's role in providing quantifiable insights beyond subjective clinical assessments, such as dyspnea scores or vital signs [14].

Our results echo prior studies linking IJV metrics to cardiopulmonary dynamics. For instance, Parikh et al (2019) reported a negative correlation between IJV collapsibility and pulmonary artery occlusion pressure, indicating that reduced collapsibility signals heightened intrathoracic pressure – consistent with our INDEX1 findings [15]. Similarly, in COPD cohorts, transcutaneous Doppler assessment of jugular venous flow has predicted pulmonary hypertension, with valve incompetence

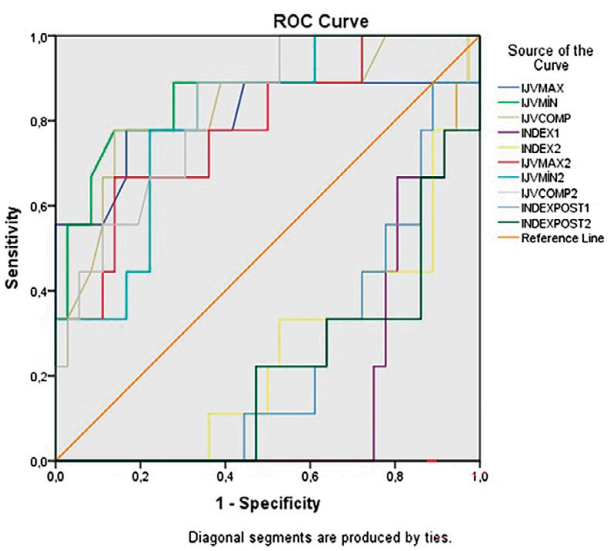

Fig 2. ROC analysis evaluating the performance of IJV measurement parameters in predicting ICU admission.

exacerbating retrograde flow during pressure surges [12]. Doepp et al (2008) found a higher prevalence of IJV valve incompetence in COPD patients with pulmonary hypertension, attributing it to chronic intrathoracic pressure elevations [16]. Matsuyama et al (2001) further demonstrated that jugular vein US estimates mean pulmonary artery pressure in COPD, supporting IJV as a non-invasive proxy for right heart strain [12]. Our findings suggest that bedside IJV US has the potential to provide an indirect and non-invasive estimation of pressures, such as right atrial pressure (RAP) or pulmonary artery occlusion pressure, which normally require invasive methods like pulmonary artery catheterization. This is a critical advantage for rapidly assessing a patient's hemodynamic status in busy and resource-limited environments such as the emergency department.

Higher post-treatment IJVCOMP2 in deceased patients (p=0.041) implies persistent venous distension despite therapy, potentially marking refractory hyperinflation and poor prognosis. This positions IJVCOMP2

Table III. Relationship between Rome classification and ınternal jugular vein diameters

Parameter	Rome Classification	Mean±SD	Median (Q1-Q3)	p-value
IJVMAX	Mild	1.006±0.32	0.93 (0.9–1.1)[a]	
	Moderate	1.27±0.38	1.21 (0.94–1.49)[ab]	**0.043*§**
	Severe	1.77±0.62	1.82 (1.52–2.17)[b]	
IJVMIN	Mild	0.56±0.21	0.55 (0.46–0.6)[a]	
	Moderate	0.83±0.7	0.80 (0.54–1)[ab]	**0.006*§**
	Severe	1.36±0.53	1.37 (1.1–1.88)[b]	
IJVCOMP	Mild	0.34±0.11	0.37 (0.31–0.4)[a]	
	Moderate	0.55±0.36	0.45 (0.23–0.77)[ab]	**0.024*§**
	Severe	0.93±0.43	0.95 (0.88–1)[b]	
INDEX1	Mild	47.88±12.94[a]	46.74 (40.86–50)	
	Moderate	35.74±14.83[ab]	35.22 (22.47–44.33)	**0.008*‡**
	Severe	24.13±8.34[b]	26.07 (15.13–31.25)	
INDEX2	Mild	67.06±12.75	65.45 (55.91–78.48)	
	Moderate	59.47±17.18	60.55 (46.74–75)	0.120‡
	Severe	49.17±14.34	46.45 (37.4–59.45)	
IJVMAX2	Mild	0.98±0.2	0.99 (0.9–1.11)[a]	
	Moderate	1.26±0.38	1.27 (1–1.46)[ab]	**0.020*§**
	Severe	1.67±0.59	1.59 (1.14–2.3)[b]	
IJVMIN2	Mild	0.47±0.17	0.50 (0.3–0.63)[a]	
	Moderate	0.77±0.4	0.73 (0.44–0.9)[ab]	**0.013*§**
	Severe	1.11±0.52	0.95 (0.77–1.74)[b]	
IJVCOMP2	Mild	0.27±0.12	0.23 (0.2–0.34)[a]	
	Moderate	0.50±0.37	0.46 (0.2–0.66)[ab]	**0.015*§**
	Severe	0.87±0.5	0.74 (0.54–1.2)[b]	
INDEX-POST1	Mild	52.21±14.87	49.49 (45.28–63.03)	
	Moderate	42.48±17.7	44.76 (29.07–55.56)	0.141‡
	Severe	35.43±9.36	33.97 (27.57–44.79)	
INDEX-POST2	Mild	72.42±10.2	74.15 (63.96–81.13)	
	Moderate	62.94±18.29	62.73 (50.85–77.53)	0.054‡
	Severe	50.64±14.03	48.91 (41.94–66.33)	

IJVMAX: Pre-treatment Internal Jugular Vein Maximum Diameter, IJVMIN: Pre-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP: Pre-treatment Internal Jugular Vein Compression Diameter, INDEX1: (IJVmax-IJVmin)/IJVmax, INDEX2: (IJVmax-IJVcomp)/IJVmax, IJVMAX2: Post-treatment Internal Jugular Vein Maximum Diameter, IJVMIN2: Post-treatment Internal Jugular Vein Minimum Diameter, IJVCOMP2: Post-treatment Internal Jugular Vein Compression Diameter, INDEX-POST1: Post-treatment Index 1, INDEX-POST2: Post-treatment Index 2; ‡Paired t-test, §Wilcoxon test. Different superscript letters indicate statistically significant differences between groups in post-hoc analysis ($p<0.05$).

as a candidate biomarker for mortality risk stratification, akin to findings in heart failure where low IJV collapsibility predicts elevated right atrial pressure (RAP) [15-17]. Teke et al (2023) observed no mortality difference in jugular compliance during COPD exacerbations but noted diameter changes correlating with outcomes, partially aligning with our data [11].

Per Rome classification, significant differences in IJVMAX, IJVMIN, IJVCOMP, INDEX1, IJVMAX2, IJVMIN2, and IJVCOMP2 across severity groups ($p<0.05$) highlight IJV's sensitivity to exacerbation gradients [9]. Non-significant INDEX2, INDEX-POST1, and INDEX-POST2 ($p=0.054$ for INDEX-POST2) may stem from measurement variability, small sample size, or individual treatment responses, warranting larger cohorts for validation.

ICU-admitted patients exhibited elevated pre- and post-treatment diameters with reduced collapsibility ($p<0.05$), indicating worse hemodynamic compromise and invasive care needs [18-20]. This aligns with evi-

Table IV. Relationship between patient disposition (admission vs. discharge) and measurement parameters

Parameter	Patient evolution	Mean±SD	Median (Q1-Q3)	p-value
IJVMAX	Discharged	1.14±0.38	0.94[a] (0.89–1.35)	
	Ward Admission	1.20±0.27	1.15[a] (0.97–1.40)	**0.011*§**
	ICU Admission	1.78±0.56	1.98[b] (1.52–2.17)	
IJVMIN	Discharged	0.66±0.35	0.57[a] (0.46–0.80)	
	Ward Admission	0.78±0.25	0.77[a] (0.55–0.95)	**0.002*§**
	ICU Admission	1.36±0.48	1.45[b] (1.10–1.75)	
IJVCOMP	Discharged	0.42±0.29	0.39[a] (0.23–0.50)	
	Ward Admission	0.49±0.27	0.42[a] (0.23–0.66)	**0.008*§**
	ICU Admission	0.97±0.44	0.98[b] (0.88–1.12)	
INDEX1	Discharged	44.01±14.42[a]	45.17 (33.33–55.84)	
	Ward Admission	35.40±15.06[b]	35.86 (21.43–44.33)	**0.004*‡**
	ICU Admission	24.28±7.05[b]	25.25 (19.35–29.17)	
INDEX2	Discharged	64.13±15.25[a]	65.38 (55.56–75.00)	
	Ward Admission	61.01±16.80[b]	62.16 (49.47–79.09)	**0.044*‡**
	ICU Admission	47.77±14.26[b]	43.43 (37.40–59.45)	
IJVMAX2	Discharged	1.14±0.39	1.06[a] (0.90–1.20)	
	Ward Admission	1.19±0.31	1.24[ab] (0.99–1.37)	**0.026*§**
	ICU Admission	1.65±0.52	1.55[b] (1.25–2.10)	
IJVMIN2	Discharged	0.58±0.34	0.54[a] (0.30–0.65)	
	Ward Admission	0.73±0.30	0.73[ab] (0.50–0.98)	**0.005*§**
	ICU Admission	1.16±0.53	0.90[b] (0.88–1.74)	
IJVCOMP2	Discharged	0.36±0.31	0.26[a] (0.17–0.40)	
	Ward Admission	0.45±0.22	0.51[ab] (0.24–0.58)	**0.003*§**
	ICU Admission	0.91±0.52	0.75[b] (0.54–1.20)	
INDEX–POST1	Discharged	51.20±14.90[a]	49.75 (39.64–63.64)	
	Ward Admission	41.71±16.88[b]	44.76 (29.07–53.85)	**0.011*‡**
	ICU Admission	31.62±13.15[b]	32.46 (27.57–40.14)	
INDEX–POST2	Discharged	71.21±14.59[a]	76.52 (63.87–81.82)	
	Ward Admission	62.85±15.56[a]	58.41 (54.17–76.92)	**0.003*‡**
	ICU Admission	47.84±16.67[b]	48.98 (41.94–61.60)	

IJVMAX: Pre-treatment Internal Jugular Vein Maximum Diameter; IJVMIN: Pre-treatment Internal Jugular Vein Minimum Diameter; IJVCOMP: Pre-treatment Internal Jugular Vein Compression Diameter; INDEX1: (IJVmax-IJVmin)/IJVmax; INDEX2: (IJVmax-IJVcomp)/IJVmax; IJVMAX2: Post-treatment Internal Jugular Vein Maximum Diameter; IJVMIN2: Post-treatment Internal Jugular Vein Minimum Diameter; IJVCOMP2: Post-treatment Internal Jugular Vein Compression Diameter; INDEX-POST1: Post-treatment Index 1; INDEX-POST2: Post-treatment Index 2; *$p<0.05$; ‡One-way ANOVA; §Kruskal-Wallis test. Different superscript letters indicate statistically significant differences between groups in post-hoc analysis ($p<0.05$).

dence that up to 70% of readmissions after COPD exacerbations result from decompensation of comorbidities, such as heart failure or pneumonia, emphasizing the need for systematic differential diagnosis in acute care settings [21].

ROC analyses reinforced this, with IJVMIN (cut-off ≥1.05 cm, AUC=0.861) and IJVCOMP (cut-off ≥0.825 cm) showing strong predictive value for ICU admission (sensitivity 77.8%, specificity 86.1%). These cut-offs could enhance risk stratification, complementing studies where IJV ultrasound estimates fluid status in dyspnea or heart failure [22].

Our study has several limitations, primarily stemming from its single-center, pilot design with a small sample size (n=45), which restricts the generalizability of our findings. Despite standardization and blinding protocols, operator-dependent measurements may have introduced variability. In this study, spectral Doppler measurements were excluded because obtaining dependable and consistent Doppler angles was technically challenging, primarily due to the significant respiratory motion artifacts observed in patients with severe tachypnea. Additionally, the short 1-month follow-up period limits our ability to assess long-term outcomes. While we identified potential confounders, congestive heart failure (CHF) warrants specific mention as it can independently affect internal jugular vein (IJV) diameters. However, congestive heart failure (CHF) was diagnosed in only four patients, and none of these individuals were among the four who succumbed.This finding strongly suggests that severe outcomes in our cohort were primarily driven by the chronic obstructive pulmonary disease (COPD) exacerbation itself rather than pre-existing cardiac failure. To address these limitations, future large-scale, multicenter trials are needed to validate our preliminary findings, ideally incorporating automated measurements and subgroup analyses to definitively isolate the effects of such comorbidities. A further significant limitation is the lack of direct comparison with invasive 'gold standard' methods, such as right heart catheterization, or advanced imaging techniques like cardiac MRI.While such methods would provide definitive hemodynamic data, their routine use in the emergency department for patients in acute respiratory distress is precluded by ethical concerns, patient safety risks, and practical constraints. Therefore, our study was deliberately designed to evaluate a rapid, non-invasive, bedside alternative suitable for the emergency setting, rather than to replace established invasive monitoring. In relation to the compression technique, we employed a standardized manual compression method involving a vertical displacement of approximately 2 cm, as detailed in previous studies [15]. However, we recognize the limitation posed by the absence of a pressure manometer to precisely measure the applied force. Consequently, variations in the compliance of neck tissues and the physical constitution of patients may influence the degree of vein compression.

In summary, bedside IJV US emerges as a practical, non-invasive adjunct for monitoring intrathoracic pressure in COPD exacerbations, guiding severity assessment and management. These insights, corroborated by existing literature, advocate for its routine adoption in emergency settings, pending broader validation [9,11,12,15,21].

Conclusion

This study demonstrated that internal jugular vein parameters, assessed by bedside US in patients presenting to the emergency department with COPD exacerbation, provide significant contributions to predicting disease severity and monitoring response to treatment. The significant changes in IJV diameters after treatment, particularly the increases in collapsibility indices and the association of certain diameter measurements with mortality and the need for intensive care, suggest that this method could be a valuable decision support tool in clinical practice.

Due to the non-invasive, rapid, and bedside applicability of the method, it is anticipated that US could enable more effective clinical decision-making in the risk stratification and treatment management processes of patients experiencing a COPD exacerbation. In light of these findings, the inclusion of IJV measurements in the routine assessment protocols for COPD management may be recommended. To enhance the generalizability and robustness of these findings, future multicenter,standardized protocols large-scale prospective studies are warranted. Incorporating IJV ultrasonography into routine COPD management could transform emergency care by enabling rapid, non-invasive assessment of disease severity.

Conflict of interest: none

References

1. Liu Y, Carlson SA, Watson KB, Xu F, Greenlund KJ. Trends in the prevalence of chronic obstructive pulmonary disease among adults aged ≥18 years – United States, 2011–2021. MMWR Morb Mortal Wkly Rep 2023;72:1250-1256.
2. Özdemir T, Yilmaz Demirci N, Kiliç H, Koç O, Kaya A, Öztürk C. An epidemiologic study of physician-diagnosed chronic obstructive pulmonary disease in the Turkish population: COPDTURKEY-1. Turk J Med Sci 2020;50:132-140.

3. Ahmad FB, Anderson RN. The leading causes of death in the US for 2020. JAMA 2021;325:1829-1830.
4. World Health Organization. Global health estimates: leading causes of DALYs, https://www.who.int/data/gho/data/themes/mortality-and-global-health-estimates/global-health-estimates-leading-causes-of-dalys; 2024 [accessed 27 August 2024].
5. McDonough JE, Yuan R, Suzuki M, et al. Small-airway obstruction and emphysema in chronic obstructive pulmonary disease. N Engl J Med 2011;365:1567-1575.
6. Gagnon P, Guenette JA, Langer D, et al. Pathogenesis of hyperinflation in chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis 2014;9:187-201.
7. O'Donnell DE, Laveneziana P. The clinical importance of dynamic lung hyperinflation in COPD. COPD 2006;3:219-232.
8. O'Donnell DE, Lam M, Webb KA. Spirometric correlates of improvement in exercise performance after anticholinergic therapy in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1999;160:542-549.
9. Celli BR, Fabbri LM, Aaron SD, et al. An updated definition and severity classification of chronic obstructive pulmonary disease exacerbations: the Rome proposal. Am J Respir Crit Care Med 2021;204:1251-1258.
10. O'Donnell DE, Webb KA, Neder JA. Lung hyperinflation in COPD: applying physiology to clinical practice. COPD Res Pract 2015;1:4.
11. Teke KB, Doğan NÖ, Özturan İU, Yılmaz S, Yaka E, Pekdemir M. Prognostic value of jugular venous diameters and compliance in patients with exacerbation of chronic obstructive pulmonary disease. J Med Ultrasound 2024;32:238-243.
12. Matsuyama W, Ohkubo R, Michizono K, et al. Usefulness of transcutaneous Doppler jugular venous echo to predict pulmonary hypertension in COPD patients. Eur Respir J 2001;17:1128-1131.
13. Global Initiative for Chronic Obstructive Lung Disease (GOLD). 2025 GOLD report, https://goldcopd.org/2025-gold-report/; 2024 [accessed 12 August 2025].
14. Guevarra K, Greenstein Y. Point-of-care ultrasonography in the critical care unit: an update. Curr Cardiol Rep 2025;27:54.
15. Parikh R, Spring M, Weinberg J, Reardon CC, Farber HW. Use of ultrasound-measured internal jugular vein collapsibility index to determine static intracardiac pressures in patients with presumed pulmonary hypertension. Ann Intensive Care 2019;9:124.
16. Doepp F, Bähr D, John M, Hoernig S, Valdueza JM, Schreiber SJ. Internal jugular vein valve incompetence in COPD and primary pulmonary hypertension. J Clin Ultrasound 2008;36:480-484.
17. Pellicori P, Kallvikbacka-Bennett A, Dierckx R, et al. Prognostic significance of ultrasound-assessed jugular vein distensibility in heart failure. Heart 2015;101:1149-1158.
18. Alzaabi O, Guerot E, Planquette B, et al. Predicting outcomes in patients with exacerbation of COPD requiring mechanical ventilation. Ann Intensive Care 2024;14:159.
19. Moin EE, Seewald NJ, Halpern SD. Use of life support and outcomes among patients admitted to intensive care units. JAMA 2025;333:1793-803.
20. Hawkins NM, Nordon C, Rhodes K, et al. Heightened long-term cardiovascular risks after exacerbation of chronic obstructive pulmonary disease. Heart 2024;110:702-709.
21. Celli BR, Fabbri LM, Aaron SD, et al. Differential Diagnosis of Suspected Chronic Obstructive Pulmonary Disease Exacerbations in the Acute Care Setting: Best Practice. Am J Respir Crit Care Med 2023;207:1134-1144.
22. Tzadok B, Shapira S, Tal-Or E. Ultrasound of jugular veins for assessment of acute dyspnea in emergency departments and for the assessment of acute heart failure. Isr Med Assoc J 2018;20:308-310.