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Ultrasonographic Optic Nerve Sheath Diameter as a Non-Invasive Marker of Systemic Congestion in Decompensated Heart Failure

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ABSTRACT

Background: Assessing fluid status in decompensated heart failure (DHF) is challenging. This study evaluates ultrasonographic optic nerve sheath diameter (ONSD), a non-invasive technique, as a marker for systemic venous congestion.

Objective: To assess the value of ONSD and its relationship with other clinical and sonographic markers of congestion in DHF patients.

Patients and Methods: This cross-sectional study included sixty patients with DHF who underwent comprehensive point-of-care ultrasound (ocular, cardiac, lung, IVC) and clinical evaluation, including central venous pressure (CVP) measurement.

Results: Enlarged ONSD was highly prevalent (96.7%) and demonstrated a strong, direct relationship with invasively measured CVP ($r=0.797$, $p<0.001$). ONSD size also correlated significantly with sonographic and clinical signs of systemic fluid overload, including lung congestion (LUS score, $r=0.938$), a dilated IVC ($r=0.871$), and cardiac function (EF%, $r=-0.692$).

Conclusion: Ultrasonographic ONSD measurement is a simple, reliable, non-invasive marker of systemic congestion in DHF. It serves as a powerful tool to complement a multi-organ ultrasound approach to patient care.

Keywords: Optic Nerve Sheath Diameter (ONSD); Decompensated Heart Failure (DHF); Venous Congestion; Point-of-Care Ultrasound (POCUS); Central Venous Pressure (CVP).

INTRODUCTION

Decompensated heart failure (DHF) represents a global health challenge and a leading cause of hospitalization for adults worldwide. The clinical course of DHF is frequently marked by recurrent episodes of fluid overload, which drive high rates of morbidity, mortality, and costly rehospitalizations, thereby imposing a significant burden on healthcare systems [(1)].

The cornerstone of effective DHF management is the accurate assessment and management of systemic congestion. Indeed, residual congestion at the time of hospital discharge is one of the strongest predictors of adverse post-discharge outcomes, including early readmission and death. Consequently, the ability to reliably quantify a patient's volume status is paramount to tailoring decongestive therapy, preventing end-organ damage and improving patient prognosis [(2)].

Traditionally, clinicians have relied on a combination of physical examination findings, such as jugular venous distention, peripheral edema, and pulmonary rales, alongside daily weight measurements and fluid balance calculations. However, these clinical signs have been shown to have limited sensitivity and specificity, often failing to accurately reflect the true underlying state of hemodynamics. Invasive hemodynamic monitoring, particularly the measurement of central venous pressure (CVP) via a central venous catheter, has long been considered a more objective method for assessing right-sided filling pressures [(3)]. Despite this, its use is associated by inherent risks, including catheter-related bloodstream infections, thrombosis, and pneumothorax, making it unsuitable for routine use in the broad population of DHF patients. Furthermore, the utility of CVP as a standalone guide for fluid management remains a subject of ongoing debate [(4)].

The rise of point-of-care ultrasound (POCUS) has precipitated a fundamental change in the bedside assessment of hemodynamics and fluid status. This non-invasive, repeatable, and readily available technology allows to directly visualize the downstream consequences of cardiac dysfunction. Multi-organ scanning protocols have become increasingly integrated into clinical practice. Standard assessments include measuring the diameter and respiratory collapsibility of the inferior vena cava (IVC) to estimate right atrial pressure, and performing lung ultrasound (LUS) to quantify extravascular lung water through the identification of B-lines [(5)]. While these techniques have profoundly enhanced the quantification of fluid overload, the pathophysiological consequences of venous hypertension are systemic and are not confined to these compartments, leaving a potential blind spot in our assessment: the brain.

A growing body of evidence supports the concept of "congestive encephalopathy", which posits that elevated systemic venous pressure directly impacts cerebral hemodynamics. The transmission of high CVP through the valveless internal jugular veins impedes venous outflow from the cranial vault, leading to cerebral venous stasis [(6)].

This can, in turn, precipitate an increase in intracranial pressure (ICP), a phenomenon that has historically been difficult to assess non-invasively and may contribute to neurological symptoms in DHF patients, such as headache. The ultrasonographic measurement of the Optic Nerve Sheath Diameter

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(ONSD) has emerged as a validated, reliable surrogate for ICP in various clinical settings, including traumatic brain injury and neurocritical care [(7)].

The anatomical basis for this measurement is sound: the optic nerve sheath is a direct meningeal extension that contains cerebrospinal fluid within a subarachnoid space continuous with that of the cranium. As such, when ICP rises, the pressure is transmitted directly to this space, causing the distensible sheath to expand in a measurable way [(8)].

Despite a compelling physiological rationale and the validation of ONSD in other fields, its specific role within the context of DHF remains incompletely defined. While individual links between ONSD and CVP have been explored, the precise relationship between ONSD and a comprehensive panel of modern invasive and non-invasive markers of congestion has not been fully interpreted in a dedicated DHF cohort. It is unknown how ONSD measurement compares and correlates with established POCUS markers like the LUS score and IVC dynamics in these patients. Therefore, the present study was undertaken to assess the value of ONSD measurement as an integrated marker of cardiorespiratory and systemic congestion, aiming to validate its utility as a novel tool that captures the global fluid status of patients with DHF.

PATIENTS AND METHODS

Study Design and Setting: This cross-sectional, observational study was conducted at Menoufia University Hospitals, a tertiary care center, over a one-year period from March 2024 to March 2025.

Sample Size Justification The sample size was calculated using G*Power 3.1.9.7 based on the correlation (r=0.422) between ONSD and CVP reported by **Chen *et al.*** [(9)]. A minimum of 39 participants was required to detect this correlation with 80% power at α=0.05. Accounting for improving statistical power and generalizability, we enrolled 60 patients.

Patient Population The study included sixty (60) consecutive adult patients admitted to the Emergency Department or Cardiac Care Unit with a primary diagnosis of decompensated heart failure.

- **Inclusion Criteria:** Patients aged 18 years or older with a formal diagnosis of decompensated heart failure.
- **Exclusion Criteria:** Age under 18 years; history of intracranial pathology (e.g., brain tumor, recent stroke, significant head trauma); diagnosed optic nerve disease (e.g., optic neuritis, glaucoma); recent ophthalmologic surgery; presence of an ocular prosthesis; or inability to provide informed consent.

Data Collection: Comprehensive clinical, demographic, and radiological data were systematically collected for each patient upon admission.

- **Clinical and Demographic Data:** Baseline information included age, sex, and Body Mass Index (BMI). Vital signs (Mean arterial pressure, heart rate, peripheral oxygen saturation) were recorded. A detailed medical history, including duration of heart failure, prior cardiac events, and New York Heart Association (NYHA) functional classification, was obtained. Clinical assessment included evaluation for congested neck veins and measurement of Central Venous Pressure (CVP).
- **Ultrasonography Protocol:** A comprehensive point-of-care ultrasound examination was performed by a trained radiologist who was blinded to the patient's CVP values. All scans were conducted using a standardized protocol with the patient in a semi-recumbent position (30 degrees).
  - **Optic Nerve Sheath Diameter (ONSD):** A high-frequency linear transducer (7-12 MHz) was placed on the closed eyelid. ONSD was measured bilaterally at a distance of 3 mm posterior to the globe. The average of the measurements of both eyes was recorded as the mean ONSD.
  - **Inferior Vena Cava (IVC):** Using a low-frequency curvilinear transducer (2-5 MHz) in the subcostal view, the maximal and minimal IVC diameters were recorded. The IVC Collapsibility Index was calculated as [(Max. Diameter – Min. Diameter) / Max. Diameter] × 100.
  - **Echocardiography:** Left Ventricular Ejection Fraction (EF%) was calculated from M-mode tracings using the Teichholz formula. The presence and size of pericardial effusion were qualitatively graded.
  - **Lung Ultrasound (LUS):** A standardized 8-zone protocol was used to quantify B-lines. The total number of B-lines across all zones constituted the LUS score, which was graded for severity. The presence and size of pleural effusion and any lung consolidation or collapse were also documented.
  - **Abdominal Ultrasound:** The liver was assessed for hepatomegaly, and the peritoneal cavity was scanned for the presence and grade of ascites.

Ethical Considerations:

The study protocol was approved by the Ethical Committee of the Faculty of Medicine, Menoufia University, IRB Approval date and number 3/2024 RADIO20, Written informed consent was obtained from all participants after a thorough explanation of the study. Participation was voluntary, and

subjects could withdraw at any time without impact on their medical care. The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

Data were analyzed using IBM SPSS software version 27. Descriptive statistics were presented as mean, standard deviation (SD), frequency, and percentage. The relationship between mean ONSD and other variables was assessed using the independent samples t-test, one-way ANOVA, or Pearson correlation coefficient (r), as appropriate. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

The study cohort comprised 60 patients diagnosed with decompensated heart failure. The baseline demographic, clinical, and key ultrasonographic characteristics are summarized in **Table 1**. The mean age of the participants was 68.67 ± 8.60 years, and 51.7% (n=31) were male. The majority of patients were in advanced functional classes, with 60.0% (n=36) classified as NYHA Class IV.

An abnormal mean ONSD (defined as ≥5 mm) was observed in 96.7% (n=58) of patients, with a cohort mean of 5.90 ± 0.39 mm. Signs of significant systemic and pulmonary congestion were prevalent across the cohort.

Table (1): Baseline Characteristics of the Study Population (n=60)

Characteristic	Value
Demographics	
Age (years), Mean ± SD	68.67 ± 8.60
Sex, Male n (%)	31 (51.7%)
BMI (kg/m²), Mean ± SD	27.54 ± 3.36
Clinical Data	
NYHA Class IV, n (%)	36 (60.0%)
Congested Neck Veins, n (%)	50 (83.3%)
CVP (mmHg), Mean ± SD	14.23 ± 3.04
Ultrasonographic Data	
Mean ONSD (mm), Mean ± SD	5.90 ± 0.39
LVEF (%), Mean ± SD	40.65 ± 6.37
IVC Diameter (mm), Mean ± SD	25.16 ± 2.80
IVC Collapsibility (%), Mean ± SD	36.47 ± 12.52
LUS Score, Mean ± SD	15.90 ± 4.61

A strong, statistically significant positive correlation was found between Mean ONSD and invasively measured CVP (r=0.797, p<0.001) **[Figure 1].**

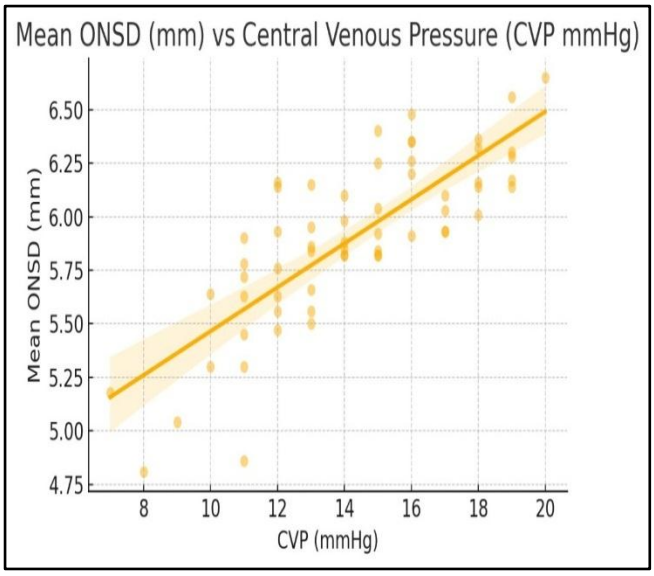

Figure (1): Scatterplot graph for correlation between Central Venous Pressure (CVP) and Mean ONSD (mm).

Mean ONSD also demonstrated highly significant correlations with all other sonographic markers of fluid overload. The strongest relationship was observed with the Lung Ultrasound (LUS) Score (r=0.938). Furthermore, Mean ONSD was strongly correlated with IVC diameter (r=0.871) and negatively correlated with both IVC collapsibility index (r=-0.702) and LVEF (r=-0.692). These correlations are detailed in **Table 2**.

Table (2): Correlation between Mean ONSD and Key Quantitative Variables |

Variable	Pearson Correlation (r)	p-value
CVP (mmHg)	0.797	<0.001*
LUS Score	0.938	<0.001*
IVC Diameter (mm)	0.871	<0.001*
IVC Collapsibility Index (%)	-0.702	<0.001*
EF (%)	-0.692	<0.001*
O_2 Saturation (%)	-0.676	<0.001*

**Statistically significant*

Clinically and sonographically apparent signs of end-organ congestion were also significantly associated with a higher mean ONSD. As shown in **Table 3**, patients with congested neck veins, lung consolidation, hepatomegaly, and higher grades of pleural effusion and ascites all had significantly higher mean ONSD values.

There was no statistically significant correlation between mean ONSD and patient demographics, including age, sex, or BMI. Similarly, mean ONSD did not significantly differ based on chronic markers such as NYHA functional class or the history of previous cardiac events.

Table (3): Relationship between Mean ONSD and Key Categorical Variables

Variable	Mean ONSD (mm) ± SD	p-value
Congested Neck Veins		
Present	5.96 ± 0.36	0.004*
Absent	5.58 ± 0.40	
Pleural Effusion		
Large	6.42 ± 0.15	
Moderate	6.10 ± 0.20	<0.001*
Small	5.81 ± 0.32	
None	5.55 ± 0.45	
Ascites		
Large	6.43 ± 0.18	
Moderate	6.18 ± 0.17	<0.001*
Small	5.89 ± 0.27	
None	5.56 ± 0.36	
Lung Consolidation		
Present	6.15 ± 0.28	<0.001*
Absent	5.78 ± 0.38	
Hepatomegaly		
Present	6.02 ± 0.37	0.010*
Absent	5.76 ± 0.37	

**Statistically significant*

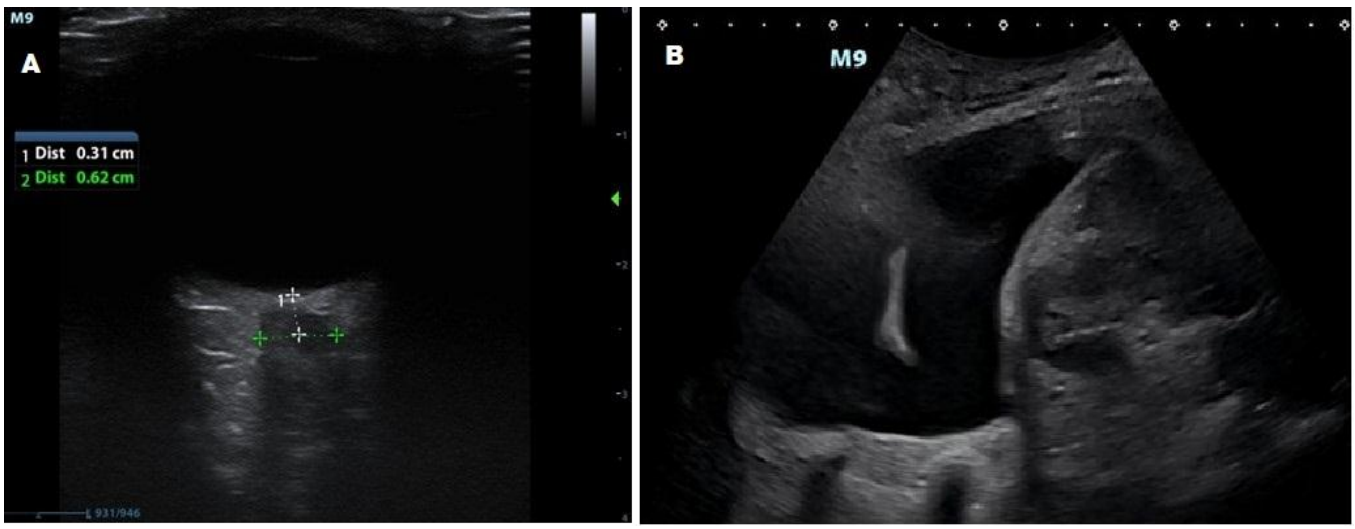

Figure (2): (A) Transverse orbital ultrasound image shows the optic nerve sheath at 6.2 mm, 3 mm behind the globe**, (B)** A lung ultrasound image displays lung collapse and pleural effusion indicating severe fluid retention.

Figure (3): (A) A transverse orbital ultrasound image shows the optic nerve sheath at 6.4 mm, 3 mm behind the globe, **(B)** An echocardiography image reveals preserved systolic function (EF 55%), demonstrating ONSD utility in detecting congestion in HFpEF, where EF alone may not capture disease severity.

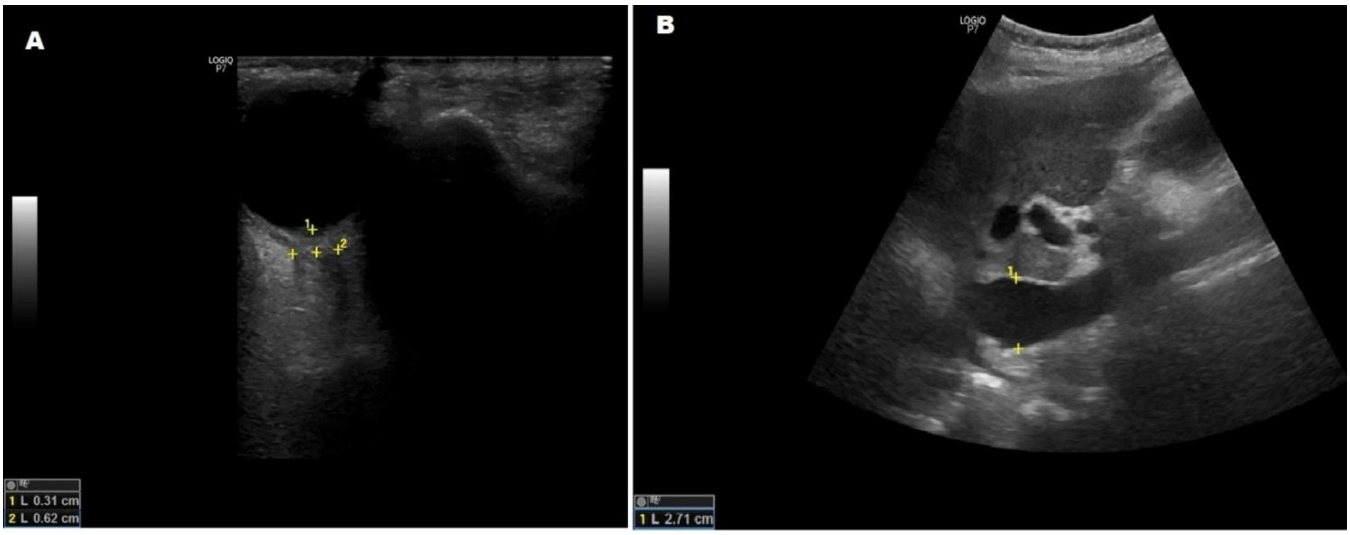

Figure (4): (A) A transverse orbital ultrasound image shows a hypoechoic optic nerve, measured at 6.2 mm, 3 mm behind the posterior globe margin, **(B)** An abdominal ultrasound image shows a dilated IVC (27 mm) with minimal inspiratory collapse indicating significant fluid overload.

DISCUSSION

This study provides compelling evidence that ultrasonographic measurement of the Optic Nerve Sheath Diameter (ONSD) serves as a powerful, non-invasive parameter joining the full spectrum of systemic and pulmonary congestion in patients with decompensated heart failure (DHF). The remarkably high prevalence (96.7%) of an enlarged ONSD in our cohort is a critical finding. It moves beyond the traditional view of DHF as a purely cardiopulmonary syndrome and underscores that its hemodynamic consequences frequently and profoundly extend to the intracranial compartment. This lends strong, quantitative support to the emerging concept of "congestive encephalopathy", where systemic venous hypertension directly impacts CSF physiology, and highlights the urgent need for accessible, non-invasive tools to monitor this often-overlooked aspect of heart failure [(6)].

The cornerstone of our research is the strong, direct, and highly significant correlation between ONSD and invasively measured CVP ($r=0.797$, $p<0.001$). This finding provides robust, direct evidence for the underlying physiological hypothesis: as central venous pressures rise in DHF, this pressure is transmitted retrograde through the valveless jugular veins, increasing dural sinus pressure. This creates a functional obstruction or "bottleneck" to both cerebrospinal fluid (CSF) absorption via the arachnoid granulations and cerebral venous outflow. The resulting compensatory rise in intracranial pressure (ICP) causes the distensible, fluid-filled optic nerve sheath, an anatomical extension of the intracranial subarachnoid space, to expand [(8)]. Our data, therefore, firmly position ONSD as a reliable non-invasive

surrogate for CVP, offering a safe, repeatable, and readily available alternative to central venous catheterization and its associated risks of infection, thrombosis, and pneumothorax.

Furthermore, our results demonstrate that ONSD is not an isolated marker but is linked to the entire cascade of downstream congestion, acting as a global integrator of fluid status. The correlation with the Lung Ultrasound (LUS) score (r=0.938) is particularly striking and clinically significant. It suggests an intimate cardiorespiratory-intracranial hemodynamic link, reflecting a vicious cycle where left-sided failure causes pulmonary edema (visualized as B-lines), which in turn increases intrathoracic pressure. This elevated intrathoracic pressure further impedes venous return to the heart and, critically, impairs venous drainage from the brain, thus completing a feedback loop that elevates ONSD. This multi-organ perspective is highly consistent with findings from studies in other fluid-overloaded states. For instance, our findings in a DHF cohort dramatically reinforce the conclusions drawn by **Simenc *et al.*[10]** and **Mowafy and Elsayed[11]** in pre-eclamptic patients, suggesting that the link between ONSD and systemic edema is a universal physiological response to fluid overload, independent of the primary pathology. Similarly, the observation that ONSD changes directly track with CVP and IVC metrics during fluid removal, as shown by **Chen *et al.*[9]** in post-cardiac surgery patients, aligns perfectly with our results.

The strong correlations with a dilated, non-collapsible IVC and the presence of hepatomegaly and ascites confirm that ONSD mirrors the severity of systemic, right-sided venous congestion and its end-organ consequences. This demonstrates that ONSD acts as a broad indicator, integrating pressure information from both the right and left sides of the circulation into a single measurement. A crucial finding was the significant negative correlation between ONSD and LVEF (r=-0.692), mechanistically linking poor systolic function to the downstream consequence of increased ONSD. However, the fact that ONSD was elevated regardless of whether patients had reduced or preserved ejection fraction is clinically paramount. It suggests ONSD is a pure marker of congestion, the final common pathway in all DHF phenotypes. This is particularly valuable for assessing fluid status in heart failure with preserved ejection fraction (HFpEF), a condition where volume assessment can be notoriously difficult due to normal pump function. Our data strongly supports the work of **Beaubien-Souligny *et al.*[12]** who proposed ONSD as a key cerebral marker of congestion in HFpEF patients, and suggests that ONSD could help clinicians identify HFpEF patients who require aggressive diuresis despite a normal LVEF. Further supporting this, **Kazancıoğlu and Batçık[13]** studied patients undergoing CABG with differing EF values and found that perioperative ONSD changes.

Lastly, ONSD did not correlate with chronic markers like NYHA functional classification, duration of illness, or a history of prior cardiac events. This lack of correlation is a significant strength, suggesting that ONSD is not a static marker of chronic disease severity but rather a dynamic indicator of the patient's acute hemodynamic state and current fluid burden. This enhances its potential clinical utility for real-time, goal-directed therapy. One can propose a clinical workflow where a baseline ONSD measurement is performed upon admission, administers a diuretic bolus, and re-measures ONSD hours later. A significant decrease would provide immediate, objective feedback on therapeutic efficacy, potentially allowing for more rapid and precise adjustment of treatment compared to waiting for changes in body weight or cumulative urine output. As demonstrated by **Chen *et al.*[9]** who showed ONSD measurements decreased in parallel with successful fluid removal, serial ONSD measurements could be a key tool to guide the intensity of diuresis, help achieve safe euvolemia, and avoid the complications of both under- and over-diuresis [9]. The finding that ONSD is independent of patient demographics such as age, sex, or BMI further solidifies its robustness as a universally applicable biomarker.

LIMITATIONS

This study has several limitations. Its single-center, cross-sectional design captures associations at a single point in time and limits generalizability. Furthermore, without a gold-standard invasive ICP measurement, the precise relationship between CVP, ICP, and ONSD in DHF remains inferred. Finally, like all ultrasound measurements, ONSD is operator-dependent, although a standardized protocol was used to minimize variability.

CONCLUSION

This study provides compelling evidence that ultrasonographic measurement of the Optic Nerve Sheath Diameter is a simple, non-invasive technique that provides a wealth of information about the hemodynamic status of patients with decompensated heart failure. Our findings demonstrate that ONSD is not merely an isolated ocular finding but a powerful integrated marker that strongly correlates with central venous pressure, the severity of systemic venous congestion, and the degree of pulmonary edema. Its independence from common demographic variables enhances its potential as a unifying tool for assessing fluid overload. While further longitudinal studies are required to validate these findings and explore their prognostic and therapeutic implications, ONSD measurement stands as a promising new frontier in the ultrasound assessment of heart failure, offering a valuable window into this complex and challenging patient population.

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