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Intravesical Pressure Monitoring: A Practical Method for Predicting Diuresis in Patients with Congestive Heart Failure

ABSTRACT

Background: Intra-abdominal pressure (IAP) may increase in acute decompensated heart failure (ADHF) due to fluid accumulation in the splanchnic system, contributing to renal venous congestion and impaired diuresis. This study aimed to evaluate the predictive value of IAP for early diuretic response in patients with ADHF.

Methods: This prospective, single-center study included 83 patients (mean age 71.6 ± 13.6 years, 58.9% female) admitted to the intensive care unit for ADHF. Patients requiring renal replacement therapy or in refractory shock were excluded. Guideline-directed medical therapy including intravenous loop diuretics was administered. The IAP was measured intravesically via Foley catheter and pressure transducer before treatment initiation. Elevated IAP was defined as >8 mm Hg. Abdominal perfusion pressure (APP) was calculated as mean arterial pressure minus IAP.

Results: Patients were divided into elevated IAP (n = 44) and normal IAP (n = 39) groups. Baseline demographics were comparable. The APP was significantly lower in the elevated IAP group (65.3 vs. 74 mm Hg; *P* = .008). The IAP showed a moderate negative correlation with 24-hour and 48-hour urine output (R^2 = 0.192 and 0.131). Each 1 mm Hg increase in IAP was associated with a 213 mL and 310 mL decrease in urine output at 24 and 48 hours, respectively.

Conclusion: Intravesical IAP measurement may serve as a practical tool to predict short-term diuretic response in ADHF. Elevated IAP and reduced APP could help identify patients requiring intensified decongestive strategies.

Keywords: Cardiorenal syndromediuresis, heart failureintra-abdominal pressure, intravesical pressure

ORIGINAL INVESTIGATION

INTRODUCTION

Congestive heart failure (CHF) is a growing public health problem characterized by frequent hospitalizations, high morbidity, and mortality. Acute decompensated heart failure (ADHF), often accompanied by overt symptoms such as congestion, fluid retention, and impaired renal function, remains the most common cause of hospitalization among patients with heart failure. Large observational studies across broad patient populations have demonstrated a significant reduction in life expectancy following ADHF-related hospitalizations.[1]

Recent evidence has identified intra-abdominal pressure (IAP) as a crucial yet frequently overlooked factor contributing to insufficient decongestion and cardiorenal dysfunction in heart failure. Even mild elevations in IAP may compromise renal perfusion and reduce diuretic response, ultimately worsening clinical outcomes.[2,3] Clinical studies have further demonstrated that higher IAP is associated with decreased urine output, worse renal prognosis, and increased mortality.[3-5]

Importantly, IAP is a modifiable parameter. Both pharmacologic decongestion and mechanical interventions such as paracentesis have been shown to lower IAP and thereby improve urine output and renal function.[6] This positions IAP as a potential tool for individualized assessment of volume status and diuretic responsiveness in patients with ADHF.

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Despite its clinical relevance, routine measurement of IAP is not yet standard practice in heart failure units. Traditional assessment tools, such as physical examination and natriuretic peptides, may fail to detect abdominal congestion—particularly in patients with preserved ejection fraction or obesity. In this context, the transvesical method of IAP measurement, validated in acute heart failure, represents a noninvasive, bedside, and reproducible alternative for clinical use.[7]

This study aimed to evaluate the prognostic value of intravesical IAP measurement in patients admitted with ADHF. Specifically, the study investigated whether elevated IAP is associated with reduced diuretic response and impaired renal function. Integrating this physiological parameter into routine clinical assessment may support the development of individualized and targeted decongestion strategies in the management of ADHF.

METHODS

Study Design and Patient Population

This prospective, single-center observational study was conducted to evaluate the prognostic significance of intravesical IAP measurement in patients admitted with ADHF. Consecutive adult patients (≥18 years old) with New York Heart Association functional class III or IV symptoms were enrolled between June 1, 2023, and January 31, 2024, following admission to the cardiac intensive care unit (CICU) of a tertiary referral center.

Eligible participants included both de novo ADHF cases and those with acute exacerbation of chronic heart failure. Inclusion criteria required the presence of at least 2 clinical signs of systemic congestion, including peripheral edema, pulmonary congestion (e.g., orthopnea, paroxysmal nocturnal dyspnea, pulmonary rales), elevated jugular venous pressure, ascites, hepatomegaly, or rapid unexplained weight gain.[8,9]

Patients were excluded if they required invasive or noninvasive mechanical ventilation, had undergone abdominal or thoracic surgery within the prior 3 months, had contraindications to Foley catheter insertion, or were on renal replacement therapy. All patients provided written informed consent before participation.

HIGHLIGHTS

- Elevated intra-abdominal pressure (IAP) is independently associated with reduced diuretic response and worsening renal function in patients with acute decompensated heart failure.
- Each 1 mm Hg increase in IAP led to a significant decrease in urine output, exceeding the effects of increasing diuretic dosage.
- Transvesical IAP measurement is a reliable, non-invasive bedside method that can support individualized assessment of volume status and guide decongestion strategies.

Out of 137 patients screened, 54 were excluded based on these criteria: 17 had recent cardiothoracic surgery, 19 required mechanical ventilation, 13 were on dialysis, and 5 had a history of urinary surgery. Ultimately, 83 patients were included in the final analysis. This study was conducted in accordance with the principles of the Declaration of Helsinki (2013 revision) and was approved by the local ethics committee (Approval number: 2023/09/694, Date: May 30, 2023). Written informed consent was obtained from all participants before any study-related procedures were performed.

Pharmacological Treatment

Hemodynamic stabilization and decongestion were achieved following the 2021 ESC Guidelines and the 2023 Focused Update on heart failure management.[8,9] All patients received intravenous loop diuretics as first-line therapy, administered as either boluses or continuous infusions at the discretion of the treating physician. Vasodilators (e.g., nitroglycerin or nitroprusside) were used in patients with preserved blood pressure and signs of congestion, while inotropic agents were reserved for cases of low cardiac output or cardiogenic shock.

Guideline-directed medical therapy—including angiotensin-converting enzyme inhibitors, angiotensin receptor-neprilysin inhibitors, beta-blockers, and mineralocorticoid receptor antagonists—was initiated or continued as tolerated. In patients with refractory congestion, sequential nephron blockade using thiazide-type diuretics or ultrafiltration was considered.[9]

Intra-abdominal Pressure Measurement

Intra-abdominal pressure was assessed using the transvesical technique, a validated and minimally invasive method recommended for use in non-ventilated patients with heart failure.[10-12] Patients were positioned supine, and a Foley catheter was inserted under sterile conditions into the urinary bladder. A pressure transducer was connected to the catheter system and zeroed at the midaxillary line at the level of the iliac crest to ensure accurate pressure calibration.[10,11] To standardize the procedure, 25 mL of sterile saline was instilled into the bladder, and the drainage tubing was clamped to establish a continuous fluid column. The IAP was measured in millimeters of mercury (mm Hg) at end-expiration. Based on emerging evidence from heart failure populations, elevated IAP was defined as values >8 mmHg, which have been associated with impaired renal perfusion, diuretic resistance, and adverse clinical outcomes in patients with ADHF.[12,13] Abdominal perfusion pressure (APP) was calculated by subtracting the measured IAP from the mean arterial pressure (MAP), in accordance with current guidelines for evaluating visceral organ perfusion in critically ill patients.[13]

Data Collection and Variable Definitions

Data were collected by heart failure specialists and included patient demographics, comorbidities, and ongoing pharmacologic therapies. Comprehensive transthoracic echocardiographic evaluations were performed according to current guideline suggestions. Blood work, including serum creatinine (Scr), lactate, and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, was obtained upon admission.

Serum creatinine levels were recorded at admission, and the estimated glomerular filtration rate (GFR) was calculated using the following formula: GFR = 141 × min (Scr/κ, 1)$^{\alpha}$ × max (Scr/κ, 1) – 1.209 × 0.993^{age} × 1.018 [if female] × 1.159 [if black]. Here, Scr is in mg/dL, and κ, α, age, 1.018, and 1.159 are constants that vary based on gender and ethnicity.[14]

Worsening renal function was defined as an increase in Scr levels exceeding 0.3 mg/dL, aligning with the criteria established by the Kidney Disease: Improving Global Outcomes guidelines for acute kidney injury. This definition is widely accepted in clinical practice to identify significant renal impairment.[15]

Statistical Analysis

All statistical analyses were performed using R Studio version 4.3.1 (R Project, Vienna, Austria) and packages "rms," "Hmisc," and "ggplot2." Normally distributed continuous data were expressed as mean and standard deviation values, whereas non-normally distributed data were expressed as medians and interquartile ranges, and categorical data were described as absolute and percentage values. Independent samples *t*-test and Mann–Whitney *U* test were used for comparisons of independent continuous data groups, and Pearson χ^2 or Fisher's exact tests were used for comparisons of categorical data groups.

The primary outcomes were defined as the urinary volume in the first 24 hours and total 48 hours. Multiple linear regression analysis was performed to examine the relationship between IAP and the primary outcomes. The IV diuretic dosage, baseline creatinine, and requirement of inotropes were used for adjustment in models. Moreover, a generalized linear model and proportional odds model were used for modeling the dependence and were further compared with ordinary least squares regression.

In addition, a generalized linear model (proportional odds) was applied to evaluate urine output not only as a continuous variable but also as an ordinal outcome (e.g., low, intermediate, and high response categories). This approach allowed for a more nuanced assessment of diuretic response across different levels of urine output. Furthermore, the results obtained from the proportional odds model were compared with those derived from the OLS regression to verify the robustness and consistency of the findings. The parallel use of these 2 statistical frameworks enhanced the reliability and interpretability of the results.

For all statistical analyses, 2-tailed probability (*P*) values of less than .05 were deemed to indicate statistical significance.

Artificial Intelligence Usage Disclosure

During the preparation of this article, the authors did not use artificial intelligence-assisted technologies, such as large language models, chatbots, or automated text/image generators.

RESULTS

This single-center study included 83 patients (58.9% female and mean age 71.6 ± 13.6 years) with decompensated heart failure admitted to the CICU. Most common comorbidity was hypertension (65%), followed by ischemic heart disease (57.8%), diabetes mellitus (42.1%), atrial fibrillation/flutter (39.7%), and chronic obstructive pulmonary disease (27.7%). Patients were stratified by IAP levels (high IAP, 53%; normal IAP, 47%). Baseline clinical, echocardiographic characteristics, and hematologic parameters of the patients were similar in both groups. (Table 1). Median length of stay at the CICU was 10 days (interquartile range [IQR] 7-14) for the normal IAP group and 8 days (IQR 5-11) for the high IAP group (*P* = .149). In-hospital mortality was observed in 4 patients (9.1%) in the high IAP, 5 patients (12.8%) in the normal IAP group (*P* = .728) (Table 1).

At baseline, Scr level was 1.39 (IQR 1.11-1.94) mg/dL in patients with high IAP and 1.42 (IQR 0.99-1.88) mg/dL in patients with normal IAP (*P* = .544). In addition, eGFR values were 42 (IQR 33.2-58.3) mL/min/m^2 and 43.6 (IQR 29.8-56.1) mL/min/m^2, respectively (*P* = .418). During hospitalization, WRF (Worsening renal function) developed in 12 patients (27.3%) in the high IAP group, while WRF developed in 7 patients (17.9%) in the normal IAP group (*P* = .313). Six patients (13.6%) in the high IAP group and 1 (2.6%) patient in the normal IAP group required renal replacement therapy (*P* = .07). The APP was lower in the high IAP group compared to the normal IAP group (65.3 mm Hg [56.8-78.5] vs. 74 mm Hg [64-78.5]; *P* = .008) (Table 1).

Multiple linear regression analysis demonstrated that IAP is an independent predictor of diuresis volume for the first 24 hours and total 48 hours (Figures 1 and 2). One-unit increase in IAP was associated with a decrease in urine output of 213 mL (95% CI: 355-71) within 24 hours and 310 mL (95% CI: 569-51) within 48 hours (Tables 2 and 3).

Temporal trends of the relationship with IAP and diuresis volume were also demonstrated a reduced diuresis in the high IAP groups (Figure 3).

The multivariable logistic regression analysis conducted on the variables in Table 4 revealed that an increase in IAP from 6 mm Hg to 9 mm Hg was a stronger predictor of urine output compared to variables such as Scr levels, inotrope use, and the increase in furosemide dose from 80 mg to 200 mg. An increase in IAP from 6 to 9 mm Hg was independently associated with a 640 mL reduction in urine output (95% CI: −1065 to −215; *P* = .003), highlighting its significant impact on urine output.

The analysis results showed that the increase in IAP from 6 mm Hg to 9 mm Hg was strongly associated with renal function deterioration and a decreased response to diuretic therapy. The odds ratio for this association was 0.42 (95% CI: 0.25-0.69; *P* < .001), emphasizing the negative effect of elevated IAP on urinary output. Figure 4 illustrates the results of this multivariable regression analysis, demonstrating the negative impact of increased IAP on urine output compared to other variables, even after adjusting for clinical factors (Figure 5).

Table 5 comparison revealed that the proportional odds model demonstrated a slightly higher explanatory power (R^2 = 0.242) compared to the OLS regression model (R^2 = 0.192), indicating that categorizing urine output into

Table 1. Baseline Characteristics and In-Hospital Clinical Outcomes of the Patients Stratified According to IAP Level

Variables	High IAP (n = 44)	Normal IAP (n = 39)	*P*
Age, years	73 ± 11.5	70.1 ± 15.5	.568
BMI, kg/m^2	24.6 ± 2.36	25.1 ± 3.35	.476
Hypertension, %	31 (70.5)	23 (59)	.274
Diabetes, %	20 (45.5)	15 (38.5)	.520
Atrial fibrillation, %	20 (45.5)	13 (33.3)	.260
Ischemic, %	29 (65.9)	19 (48.7)	.113
Previous CABG, %	8 (18.2)	4 (10.3)	.362
Previous PCI, %	4 (9.1)	3 (7.7)	.819
Stroke, %	3 (6.8)	1 (2.6)	.619
COPD, %	11 (25)	12 (30.8)	.558
Malignancy, %	0	2 (5.1)	.218
Medication on admission, %	31 (70.5)	22 (56.4)	.184
Beta blocker	30 (68.2)	21 (53.8)	.180
ACEI	20 (45.5)	12 (30.8)	.170
MRA	20 (45.5)	19 (48.7)	.939
SGLT2 inhibitors	28 (63.6)	22 (56.4)	.502
Loop diuretic	23 (52.3)	16 (41)	.306
Statin			
Ejection fraction, %	30 (20-45)	30 (25-45)	.107
Systolic BP, mm Hg	110 ± 35.1	116 ± 32	.279
SpO_2, %	88.6 ± 8.47	90.4 ± 6.98	.445
IVC, cm	2.14 ± 0.6	2.15 ± 0.19	.983
Estimated SPAP, mm Hg	41.6 ± 10.4	40.4 ± 10.8	.640
Hemoglobin, g/dL	12.2 ± 2.49	12.4 ± 2.87	.725
CRP	13.6 (6-50)	22 (9.6-46)	.483
Nt-pro BNP, ng/L	9576 (4379-33 109)	8711 (3803-26 053)	.523
Pleural effusion, %	39 (88.6)	33 (84.6)	.590
Ascites, %	16 (36.4)	11 (28.2)	.428
Length of stay, days (in CICU)	10 (7-14)	8 (5-11)	.149
Creatinine on admission, mg/dL	1.39 (1.11-1.94)	1.42 (0.99-1.88)	.544
eGFR (mL/min/m^2)	42 (33.2-58.3)	43.6 (29.8-56.1)	.418
Worsening renal function, %	12 (27.3)	7 (17.9)	.313
Lactate, mmol/L	2.21 (1.55-3.3)	1.83 (1.13-2.32)	.087
IAP, mm Hg	9 (9-11)	6 (6-7)	<.001
MAP, mm Hg	80.1 ± 22.3	86.9 ± 21.5	.151
APP, mm Hg	65.3 (56.8-78.5)	74 (64-78.5)	.008
HCO_3, mmol/L	23.4 ± 5.22	22.7 ± 5.53	.578
Sodium, mEq/L	137 ± 4.51)	137 ± 3.89	.783
Potassium mEq/L	4.75 ± 0.7)	4.83 ± 0.9	.539
IV furosemide dose, mg	160 (100-200)	120 (80-200)	.209
Inotrope, %	6 (13.6)	3 (7.7)	.490
In-hospital mortality, %	7 (14.9)	4 (10.2)	.747

ACEI, angiotensin-converting enzyme inhibitor; APP, abdominal perfusion pressure; BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; HCO_3, Bicarbonate; IAP, intra-abdominal pressure; IVC, inferior vena cava; MAP, mean arterial pressure; MRA, mineralocorticoid receptor antagonist; NT-proBNP, N-terminal pro B-type natriuretic peptide; PCI, percutaneous coronary intervention; SGLT2-I, sodium-glucose cotransporter-2 inhibitor; SPAP, systolic pulmonary artery pressure; SpO_2, peripheral capillary oxygen saturation.

ordinal groups provided additional discriminatory value. The likelihood ratio statistics also supported an adequate fit for both models. Importantly, across both modeling approaches, increased IAP consistently emerged as the strongest predictor of reduced urine output and renal function deterioration, maintaining statistical significance even after adjusting for Scr, inotrope use, and diuretic dose. These findings highlight that the detrimental impact of elevated IAP on diuretic responsiveness is robust and independent of other clinical factors, underscoring its relevance as a key hemodynamic marker in acute heart failure management.

DISCUSSION

This prospective observational study demonstrated a significant association between IAP, measured via the transvesical method, and both early diuretic response and renal function changes in patients hospitalized with ADHF. These findings suggest that IAP is not merely a passive marker of congestion but may represent an independent and modifiable determinant of renal hypoperfusion and inadequate decongestion.

The deleterious impact of elevated IAP on renal function can be explained through several pathophysiological mechanisms, including impaired abdominal venous return, increased renal venous pressure, reduced GFR, and the development of intrarenal edema. In a preclinical model of CHF, Abu-Saleh et al[2] demonstrated that increasing IAP to 10-14 mm Hg resulted in reduced renal plasma flow, proximal tubular injury, and elevated urinary neutrophil gelatinase-associated lipocalin levels—a sensitive biomarker of early tubular damage—thereby supporting the hypothesis of direct parenchymal insult from elevated IAP.[2]

In this cohort, each 1 mm Hg increment in IAP was associated with an average decrease in 24-hour urine output by 213 mL, and by 310 mL over 48 hours. Additionally, IAP elevations

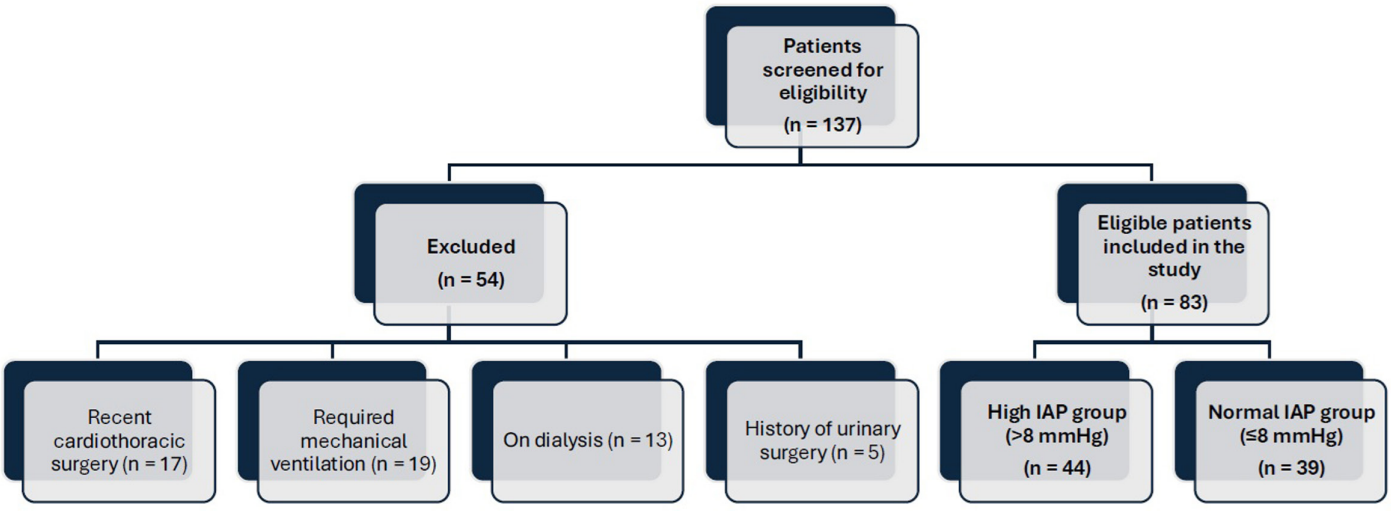

Figure 1. Flow diagram of patient selection and study groups

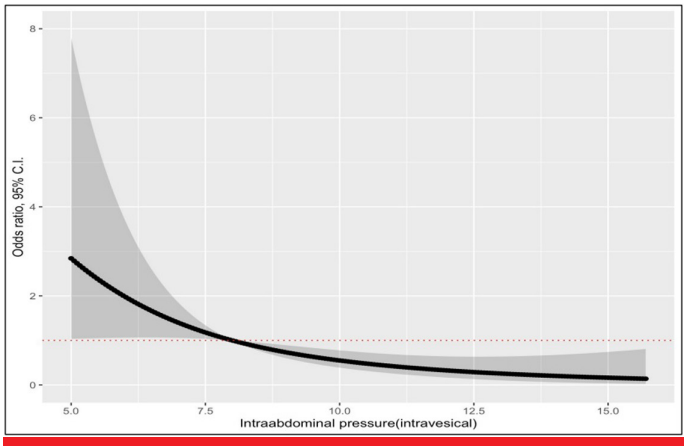

Figure 2. The Relationship Between Urinary Output and Intravesical Pressure. This figure illustrates the correlation between urinary output and intravesical pressure in patients.

from 6 to 9 mmHg corresponded to a loss of diuresis up to 640 mL, which exceeded the effect of escalating furosemide doses. These results underscore IAP as not only a marker of volume overload but also a direct hemodynamic stressor limiting effective diuretic response.

The clinical relevance of IAP in diuretic resistance has been previously demonstrated. Nguyen et al[6] reported a marked improvement in diuretic response following paracentesis in patients with elevated IAP. Similarly, Mullens et al[12] described the detrimental role of abdominal venous congestion in renal dysfunction and subsequent adverse clinical outcomes in ADHF.

According to current WSACS guidelines, intra-abdominal hypertension (IAH) is defined as a sustained IAP ≥12 mm Hg, with normal values ranging from 5 to 7 mm Hg in critically ill individuals. The IAP ≥8 mm Hg is considered elevated, and values exceeding 20 mm Hg with evidence of new organ dysfunction define abdominal compartment syndrome. The IAH is further stratified into 4 grades: grade I (12-15 mm Hg), grade II (16-20 mm Hg), grade III (21-25 mm Hg), and grade IV (>25 mm Hg).[13] Although these thresholds are well established in surgical and intensive care settings, emerging evidence suggests that clinically relevant effects may occur at lower IAP levels in ADHF. In fact, Mullens et al[12] observed that

Table 2. Multiple Linear Regression Analysis on Diuresis Volume in the First 24 Hours

			95% CI		
Predictor	**Estimate**	**SE**	**Lower**	**Upper**	***P***
IAP (mm Hg)	−213.54	70.81	−355.19	−71.88	.004
Admission creatinine (mg/dL)	−63.25	217.16	−497.65	371.14	.772
IV diuretic dosage (mg)	3.01	2.01	−1.01	7.02	.140
Requirement of inotropes (0/1)	−757.58	793.83	−2345.48	830.33	.344

R^2: 0.192. IAP, intra-abdominal pressure; IV, intravenous; SE, standard error; R^2, coefficient of determination.

Table 3. Multiple Linear Regression Analysis on Diuresis Volume in the Total 48 Hours

			95% CI		
Predictor	**Estimate**	**SE**	**Lower**	**Upper**	***P***
IAP (mm Hg)	-310.41	129.26	−569.68	−51.1	.020
Admission creatinine (mg/dL)	26.90	369.74	−714.71	768.5	.942
IV diuretic dosage (mg)	3.60	3.71	−3.84	11.0	.336
Requirement of inotropes (0/1)	−727.16	1407.36	−3549.96	2095.6	.608

R^2: 0.131.
IAP, intra-abdominal pressure; IV, intravenous; SE, standard error; R^2, coefficient of determination.

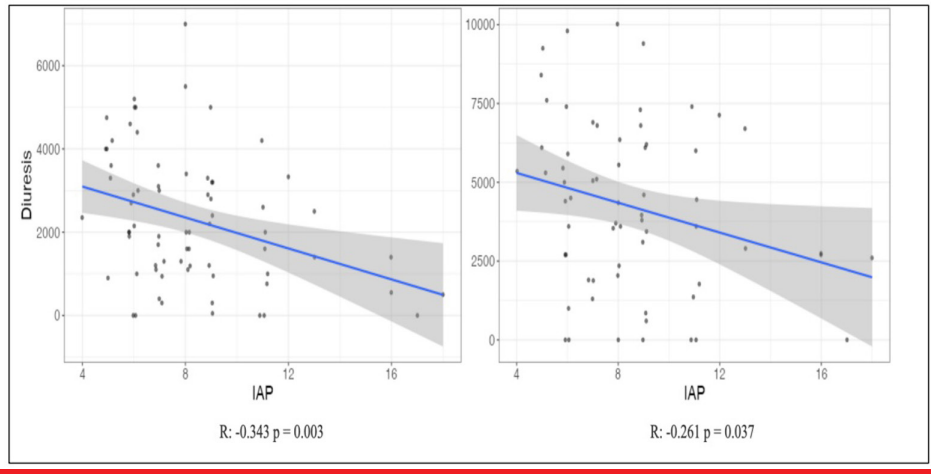

Figure 3. Scatterplot and linear regression lines showing the relationship between 24-hour and 48-hour diuresis and intra-abdominal pressure. This scatterplot presents the relationship between 24-hour and 48-hour diuresis volumes against intra-abdominal pressure. Linear regression lines are included to show the trend.

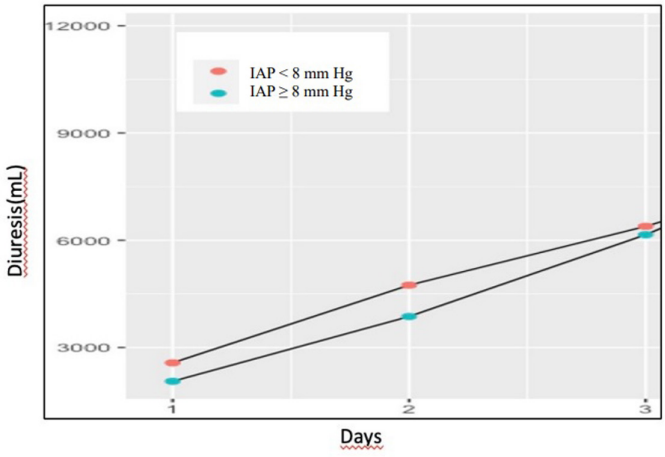

Figure 4. Comparison of diuresis between high and low intra-abdominal pressure (IAP) groups over time—red dots (0): represent the low IAP (<8 mm Hg) group. Blue Dots (1): Represent the high IAP (>8 mm Hg) group. This figure compares diuresis volumes over time between patients with high and low intra-abdominal pressures.

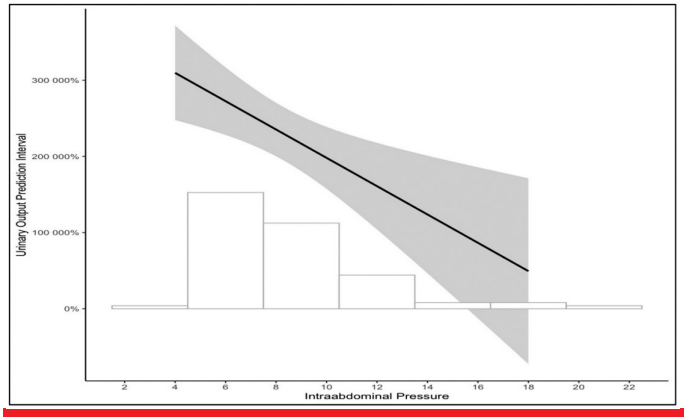

Figure 5. Multivariable regression analysis of urinary output and intra-abdominal pressure. This figure shows the results of a multivariable regression analysis examining the impact of intra-abdominal pressure on urinary output, adjusting for other variables.

60% of patients with ADHF had IAP ≥8 mm Hg, which correlated with worsened renal outcomes. These data underscore the importance of monitoring even sub-threshold elevations in IAP in ADHF to optimize decongestion and preserve renal function.

Elevated IAP is associated with reduced APP, calculated as MAP minus IAP. The APP is an established surrogate of splanchnic organ perfusion. In the study, APP was significantly lower in patients with elevated IAP, supporting the hypothesis that abdominal congestion may contribute to renal hypoperfusion. These findings align with the concept of renal filtration gradient (RFG = MAP − 2 × IAP) described by Łagosz et al,[4] which posits that elevated IAP compromises both abdominal and renal perfusion, ultimately reducing glomerular filtration pressure and accelerating renal dysfunction.

Renal venous Doppler ultrasonography (US) has emerged as a valuable tool for assessing renal congestion and predicting diuretic responsiveness in acute heart failure.[16] Unlike IAP, which reflects abdominal pressure indirectly, renal venous Doppler directly evaluates venous flow patterns and congestion at the renal level. While Doppler US offers detailed hemodynamic assessment, it is operator-dependent and requires technical expertise. In contrast, IAP measurement is simple, reproducible, and can be performed at the bedside with minimal resources. The findings suggest that IAP monitoring may serve as a practical alternative to Doppler US, particularly in resource-limited settings. However, future studies directly comparing these 2 modalities are warranted to clarify their complementary roles in guiding decongestive therapy.

In clinical practice, physical examination alone is often insufficient to assess congestion, especially in complex cases such as obesity, advanced age, altered mental status,

Table 4. Regression Analysis Using Ordinary Least Square (OLS) and Proportional Odds Model

	Ordinary Least Square (OLS)			Proportional Odds Model		
Variables	**Coefficient CI**	**95% CI**	***P***	**Odds Ratio**	**95% CI**	***P***
IAP pressure from 6 to 9 mm Hg	−640	−1065; −215	.003	0.42	0.25-0.69	<.001
Creatinine from 1.06 to 1.94 mg/dL	−55	−435.4; 324.7	.77	0.99	0.65-1.51	.98
Inotropy usage (0/1)	−757	−2354; 830	.34	0.31	0.06-1.66	.17
Furosemide dose from 80 to 200 mg	360	−121; 842	.13	2.13	1.01-4.55	.04

All continuous regression variables coefficients and odds ratios represented as increase from 25th to 75th percentile values.
IAP, intra-abdominal pressure; OLS, ordinary least squares; OR, odds ratio.

Table 5. Model Comparison

Model	**R^2**	**Likelihood Ratio**
Ordinary least square	0.192	17.98
Proportional odds	0.242	13.87

R^2 values represent the proportion of variance explained by each model. The likelihood ratio indicates model fit compared to the null model.
OLS, ordinary least squares; R^2, coefficient of determination; PO, proportional odds model.

or mechanical ventilation. Therefore, objective and easily applicable tools are required. Both renal venous Doppler US and IAP monitoring address this need, but the findings emphasize that transvesical IAP measurement combines bedside applicability with strong predictive value. Zymliński et al[7] further confirmed the reliability of this technique by demonstrating a strong correlation ($r = 0.95$) with the gold-standard intraperitoneal measurement in acute heart failure patients.

While biomarkers such as NT-proBNP, sST2, and CA125 are commonly used to evaluate congestion, they are influenced by multiple confounding factors, including age, renal function, and body mass index. Notably, CA125 has been associated with peritoneal effusion, though its optimal cut-off values remain unclear. Rubio-Gracia et al[17] showed a positive, non-linear correlation between CA125 and IAP, with levels <17.1 U/mL potentially ruling out elevated IAP. Therefore, IAP measurement may provide a more direct and complementary approach, particularly when biomarker interpretation is limited.

Study Limitations

Several limitations of this study should be acknowledged. First, the study was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Second, diuretic therapy was individualized and not administered according to a standardized treatment protocol across all patients, making it difficult to determine whether changes in urine output were solely attributable to differences in IAP or influenced by therapeutic variability. Third, secondary congestion markers such as urinary sodium excretion and body weight changes were not assessed, which may have provided additional insight into volume status. Fourth, IAP was measured only at the time of hospital admission, and dynamic changes in response to treatment over the course of hospitalization were not evaluated. Lastly, due to limited statistical power, subgroup analyses—such as comparisons between HFpEF and HFrEF or de novo versus chronic ADHF—could not be performed with sufficient precision.

CONCLUSION

In summary, the findings highlight IAP as a clinically relevant and modifiable determinant of renal hypoperfusion, diuretic resistance, and persistent congestion in patients with ADHF. Early and accurate identification of elevated IAP through transvesical monitoring may facilitate risk stratification and enable the timely escalation of decongestive therapies. Importantly, the predictive value of IAP appears to be independent of conventional hemodynamic markers, underscoring its additive role in guiding patient management. Taken together, transvesical IAP measurement represents a simple, noninvasive, and cost-effective bedside technique with significant implications for personalized treatment approaches. Integrating IAP assessment into routine clinical workflows may improve therapeutic responsiveness, reduce the burden of renal complications, and ultimately enhance outcomes in patients with advanced heart failure.

Data Availability Statement: All relevant data is included in the manuscript or available from the corresponding author upon reasonable request.

Ethics Committee Approval: This study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Kartal Koşuyolu High Specialization Hospital Ethics Committee, Approval Number: 2023/09/694, Date: May 30, 2023.

Informed Consent: Written informed consent was obtained from all participants before any study-related procedures were performed.

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