Prevalence of increased intra-abdominal pressure and its impact on renal function in acute decompensated heart failure: A prospective pilot study

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ABSTRACT

Background: Intra-abdominal pressure (IAP) is a frequently overlooked aspect in clinical assessment that can have a significant impact on organ dysfunction in patients with acute decompensated heart failure (ADHF).

Aims: We aimed to investigate dynamics of IAP in patients with ADHF and its impact on diuretic response.

Methods: We conducted a prospective observational pilot study on a group consisting of 30 patients admitted for ADHF. In every individual IAP measurement, blood and urine samples were taken upon admission, on the second and third days of hospitalization.

Results: The study showed a high (63.3%) prevalence of intra-abdominal hypertension (IAH) defined as IAP ≥12 mm Hg upon admission, while only roughly 13% had signs of ascites. We observed poorer diuresis on the first day of hospitalization in the IAH group ($P = 0.03$). IAP was negatively correlated with urine output ($P = 0.01$) and positively correlated with urine osmolality ($P = 0.03$) on the first day of hospitalization. During follow-up, there was a significant decrease in IAP in patients with IAH upon admission who received standard decongestive therapy.

Conclusions: The study shows a high prevalence of IAH in patients admitted for ADHF, even in individuals who do not present symptoms of abdominal congestion. Established correlation between IAP, reduced diuresis, and increased urine osmolality, despite achieving target natriuresis, contributes novel insights into the understanding of pathomechanisms underlying diuretic resistance in ADHF.

Key words: cardiorenal syndrome, diuretic resistance, heart failure, intra-abdominal pressure

INTRODUCTION

Acute decompensated heart failure (ADHF) is a severe condition associated with high morbidity and mortality rates. While extensive research has focused on cardio-renal dysfunction, the role of intra-abdominal pressure (IAP) in the milieu of ADHF has received limited scientific attention. IAP represents a fundamental physiological parameter, frequently neglected by clinicians despite its significant implications for the homeostatic equilibrium of nearly every system within the human body. In 2004, the World Society of Abdominal Compartment Syndrome established definitions of elevated IAP, intra-abdominal hypertension (IAH), and abdominal compartment syndrome. According to the guidelines, IAH is defined as a rise above 12 mm Hg and can be divided into four grades, namely grade 1: 12–15 mm Hg, grade 2: 16–20 mm Hg, grade 3: 21–25 mm Hg, and grade 4: >25 mm Hg, while abdominal compartment syndrome is defined as sustained IAP >20 mm Hg accompanied by onset of organ dysfunction or failure [1].

Although there is research linking IAP with a deterioration of kidney function, which is one of the strongest predictors of adverse outcomes and frequently exacerbates the course of hospitalization for ADHF patients, in recent years only several small studies have

WHAT'S NEW?

The study provides novel insights into the intricate pathophysiological mechanisms governing diuretic resistance in this patient population. It demonstrates an exceptionally high prevalence of intra-abdominal hypertension despite the marginal presence of overt signs of ascites. The findings show significantly compromised diuresis in acute decompensated heart failure patients with intra-abdominal hypertension diagnosed upon admission. The study demonstrates a negative correlation between intra-abdominal pressure and urine output, even in patients achieving target natriuresis. The observational nature of the study highlights the effectiveness of the therapeutic approach proposed by the European Society of Cardiology guidelines. It underscores the clinical importance of intra-abdominal pressure monitoring as a profiling tool for acute decompensated heart failure patients and indicates potential areas for future research.

focused on this area in HF patients [2, 3]. Our prospective study was designed to explore a piece of *terra repromissionis* in the ADHF pathophysiology, explore IAP dynamics, and investigate whether increased IAP compromises the effectiveness of diuretic treatment.

METHODS

Study population

Thirty patients with symptomatic ADHF, class III or IV in the New York Heart Association classification, who presented with pulmonary, peripheral or mixed congestion, were prospectively enrolled in the study. All patients were admitted to the University Clinical Hospital in Wroclaw, Poland, and met the following inclusion criteria: 1) age ≥18; 2) AHF diagnosed according to the 2021 European Society of Cardiology (ESC) guidelines [4]; 3) informed consent to participate in the study. Exclusion criteria included: 1) acute coronary syndrome; 2) end-stage renal disease defined as a baseline estimated glomerular filtration rate (eGFR) ≤15 ml/min/1.73 m^2; 3) systemic infection; 4) abdominal or thoracic surgery within the last 90 days; 5) acute respiratory failure requiring mechanical ventilation; 6) inability to have the bladder catheterized. The flowchart diagram illustrating the selection of subjects is shown in Supplementary material, *Figure S1*. The study protocol was approved by the local ethics committee and was conducted in accordance with the Declaration of Helsinki. All patients provided written informed consent to participate in the study.

Approach to therapy

Patients were treated in accordance with the latest ESC guidelines for HF. The use of an armamentarium consisting of loop diuretics, vasoactive, and inotropic agents was left to the supervising physicians' discretion. Wherever feasible, evidence-based treatments were continued and optimized if patients had not received appropriate care before being admitted [5–7].

Study design and data collection

During the admission process, information on demographics, comorbidities, and previous therapies was collected. Trained cardiologists have conducted thorough physical examinations and taken manual IAP measurements in all patients upon admission and at 24-hour and 48-hour marks. To assess cardiac status, echocardiographic examinations were performed by an experienced cardiologist in every patient. Venous blood and urine samples were collected upon admission and at 24 and 48 hours. All samples underwent centrifugation and were subsequently frozen at –70° for further analysis.

Intra-abdominal pressure measurement

In each patient, pressure within the abdominal cavity was measured using the transurethral method, which is the gold standard in noninvasive monitoring of IAP [8, 9]. In this technique, a Foley catheter is inserted into the urinary bladder and filled with 25 ml of saline, which ensures the transfer of hydrostatic pressure between the bladder wall and the pressure transducer (Figure 1). The transurethral method has been compared to direct measurements and validated in settings of ADHF [10]. The IAP was recorded upon admission, after 24 and 48 hours in accordance with the guidelines while patients maintained complete supine position to avoid muscle activity and at the end-expiration phase with the zero point oriented in the mid-axillary line at the level of the iliac crest. Commercially available Unometer Abdo-Pressure Kits were used to make measurements. The recorded values were expressed in mm Hg, and IAH was defined as IAP ≥12 mm Hg.

Renal assessment

Renal function was assessed on admission, on the second and third days of hospitalization. Plasma and spot urine samples taken 3 hours after initiation of intravenous diuretics were used to determine serum and urine osmolality, levels of creatinine, urea nitrogen, and electrolytes. During the process, the urine output was recorded.

The estimated glomerular filtration rate was calculated using the Modification of Diet in Renal Disease equation.

Statistical analysis

Statistical analysis was performed in Statistica 13.3 licensed to Wroclaw Medical University. Data visualization was performed in the said software and, additionally, with Python 3.10.7 (packages: numpy 1.21.4, pandas 1.4.4, seaborn 0.11.2).

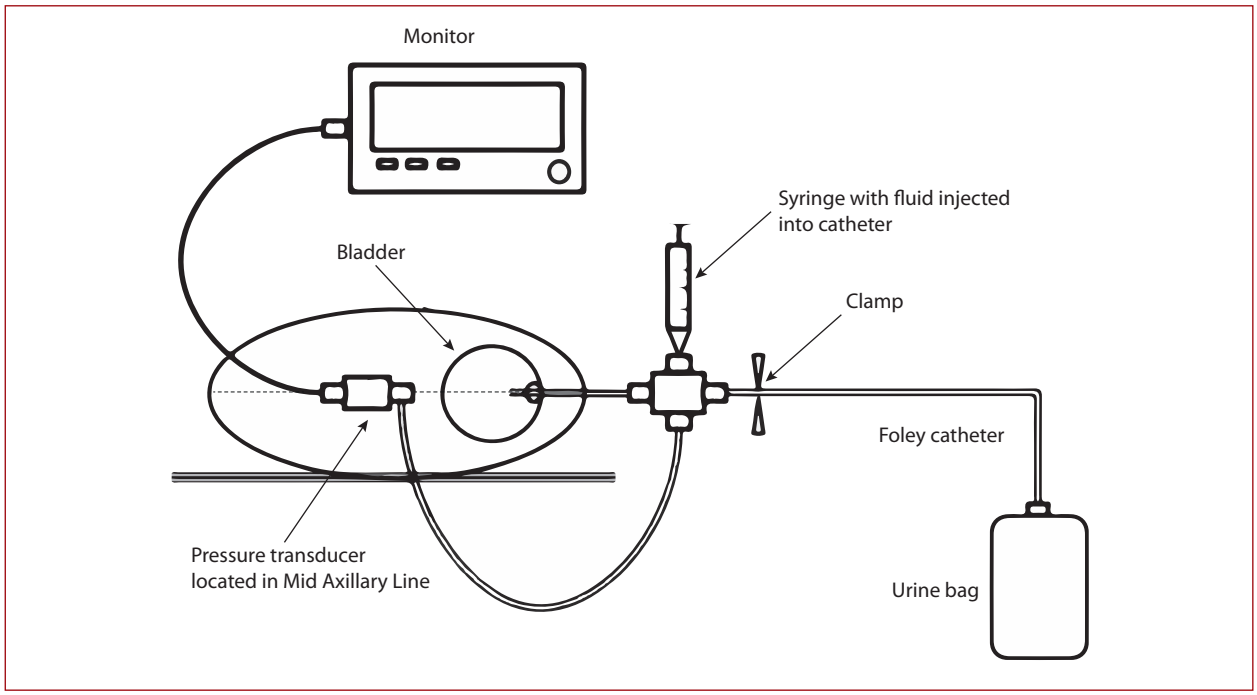

Figure 1. Intra-abdominal pressure measurement using the transurethral method

The frequentist convention was used for statistical inference ($\alpha = 0.05$). Pairwise, one-way comparisons between independent groups of patients were carried out with the use of the t-test (continuous variables; upon checking its pre-assumptions with Levene and Shapiro–Wilk tests) or Fisher's exact test (categorical variables). Cochran-Cox correction was used in the case of heteroscedasticity (observed based on the Levene test).

Multivariate comparisons of the time-dependent variable were performed with the use of Repeated Measures ANOVA (RM-ANOVA) models, containing sets of interactions with time (Supplementary material, *Table S1*). For this purpose, type VI sums of squares were analyzed. Upon observing significant results in these models, the interactions were further diagnosed with use of linear contrast analysis (Supplementary material, *Table S2*) based on the exploratory (Helmert) contrast-coding matrix. The sphericity pre-assumption of RM-ANOVA was checked with Mauchly's test and, subsequently, corrected by adjusting the degrees of freedom with Greenhouse-Geisser and Huynh-Feldt corrections.

Monotonic correlations were represented by Spearman r coefficients.

RESULTS

Baseline characteristics

A total of 30 patients met the inclusion criteria and were enrolled in the study. Patient characteristics in the overall cohort are listed in Table 1. The predominant sex was male, every patient presented signs of pulmonary congestion, 72.73% presented with peripheral edema, while 13.33% had signs of ascites in physical examination.

The prevalence of IAH on admission was 63.3%. The population sample was assigned to two groups based on the prevalence of IAH according to definitions established by the World Society of Abdominal Compartment Syndrome, which defined physiological IAP below 12 mm Hg. Demographic, physical, laboratory, and echocardiographic variables were comparable between subjects with and without IAH. The mean (standard deviation [SD]) values of IAP in the groups were 14.92 (2.71) and 8.95 (1.13), respectively. There were no statistically significant differences in demographic variables, left ventricular ejection fraction, serum creatinine, and serum N-terminal pro B-type natriuretic peptide on admission. Obesity and hypertension were significantly more common in the IAH group. We showed a significant difference in baseline body weight and diuresis on the first day, while in laboratory tests, a significant disparity was found in white blood cell count, serum protein concentration, and urine osmolality.

The echocardiographic study showed no significant differences.

The more frequent prescription of thiazide diuretics on discharge in the IAH group was the only difference in pharmacological treatment. Comparison of baseline parameters between groups is shown in Table 2.

IAP dynamics in the cohort

The mean (SD) baseline IAP in the overall cohort was 12.73 (3.68) mm Hg. After 24 hours of standard ADHF treatment, the mean (SD) IAP improved to 10.87 (3.75)

Table 1. Patient characteristics upon admission

Demographic and clinical variables	
Age	72.5 (68–82)
Male	67.00%
Body mass, kg	91.74 (16.91)
IAP, mm Hg	12.73 (3.68)
Heart rate, bpm	91.20 (20.36)
Systolic BP, mm Hg	120.43 (20.72)
Diastolic BP, mm Hg	74.03 (13.30)
MAP, m mHg	89.59 (14.28)
RFG, mm Hg	64.12 (15.70)
In-hospital furosemide dose[a], mg	60 (60–100)
24 hours diuresis, ml	3089.20 (1175.20)
Pulmonary congestion, %	100.00
Peripheral edema, %	72.73
Ascites, %	13.33
Comorbidities	
Coronary arteries disease	54.50%
Hypertension	63.30%
Diabetes	63.30%
Atrial fibrillation	73.00%
Obesity	18.20%
Echocardiographic variables	
LVEF, %	37.50 (13.93)
LVEDD, mm	58.53 (9.17)
End-expiratory IVC, mm	26.53 (4.82)
TAPSE, mm	16 (14–17)
S', cm/s	8 (6.4–9.9)
Baseline laboratory parameters	
NT-proBNP, pg/ml	8778 (4714–16380)
Creatinine, mg/dl	1.25 (1.00–1.76)
eGFR, ml/min/1.73 m^2	56.23 (23.91)
Urea, mg/dl	62 (46–104)
Potassium, mmol/l	4.25 (3.80–4.90)
Sodium, mmol/l	137.87 (4.84)
Urinary osmolality, mOsm/kg	285.65 (247.40–345.20)
Urinary creatinine, mg/dl	19.80 (12.10–74.30)
Urinary potassium, mmol/l	22.15 (15.00–42.70)
Urinary sodium, mmol/l	85.55 (33.19)
Urinary chloride, mmol/l	98.07 (36.04)
Hemoglobin, g/dl	11.61 (1.90)
WBC, × 10^9/l	8.0 (6.5–10.3)
Platelets, × 10^9/l	226.30 (87.78)
Hematocrit, %	35.70 (32.20–39.10)
CRP, mg/dl	17.52 (5.30–36.56)
Plasma protein, g/dl	6.26 (0.75)
Plasma albumin, g/dl	3.38 (0.57)
ALT, IU/l	26 (14–35)
AST, IU/l	27 (21–36)
GGTP, IU/l	124.22 (86.33)
Total bilirubin, mg/dl	1.30 (0.90–1.80)

Quantitative variables are shown as mean values (standard deviations) or median values (1st – 3rd quartiles), depending on whether their distribution met the normality criterion.

[a]Or equivalent in torasemide

Abbreviations: ALT, alanine transaminase; AST, aspartate aminotransferase; BP, blood pressure; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; GGTP, gamma-glutamyltransferase; IAP, intra-abdominal pressure; IVC, inferior vena cava; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; NT-proBNP; N-terminal pro B-type natriuretic peptide; RFG, renal filtration gradient; TAPSE, tricuspid annular plane systolic excursion; WBC, white blood cells

(P <0.001). Over the next 24 hours, there was a further IAP decline to the mean (SD) IAP 9.86 (3.75) (P = 0.002), and the number of patients with IAH decreased by 42.1% compared to admission. The distribution of IAH grades over time and changes in IAP in individuals are presented in Figures 2 and 3. An increase in IAP after 48 hours was observed in 3 patients, of whom one died during hospitalization.

A multivariate analysis of variance showed an effect of time and IAH upon admission on the change in IAP within the first two days of hospitalization (P = 0.01). Contrast analysis indicated that the effect of IAH and time was statistically significant between both the first and the second, as well as the second and the third days of hospitalization. An increase in IAP was observed in the group without IAH on admission, with reduced left ventricular ejection fraction (LVEF) and poor diuresis. Patients with IAH on admission benefited from a decrease in IAP independently of diuresis and LVEF, but the greatest decrease was observed in patients with LVEF above 40% and urinary output >3000 ml (Figure 4).

We found that obesity in non-diabetic patients predisposed to an increase in IAP, while male patients with coronary artery disease (CAD) had higher baseline IAP and were better responders to the therapy. Among valvular diseases, we observed a significant impact of severe tricuspid regurgitation (TVR) on IAP dynamics. Patients with severe TVR were characterized by a higher baseline IAP and a steeper reduction in IAP (Figure 5).

Multivariate interaction analysis and contrast analysis are presented in Supplementary material.

The relationship between IAP and measures of kidney function

In the overall cohort, baseline IAP was negatively correlated with diuresis within the first 24 hours (P = 0.01) and positively correlated with baseline urine osmolality (P = 0.03). The full set of correlations is shown in Table 3. After 24 hours, IAP correlated only with urine potassium, and it was not correlated to renal or urine parameters after 48 hours following admission. Moreover, after its additional estimation, the body mass index upon admission turned out to be correlated with IAP (r = 0.54; P = 0.006; Supplementary material, *Figure S2*), but not with end-expiratory inferior vena cava diameter, as observed in some studies.

DISCUSSION

This study provides several interesting findings. Firstly, we observed an exceptional prevalence of IAH upon admission, which was diagnosed in over 63% of subjects enrolled in the study. This result was surprising, as only a few patients manifested pronounced symptoms of abdominal fluid overload. Thus, that prevalence clearly demonstrates the limitations of conventional physical examination, which lacks precision in assessing the actual pressure within the abdominal cavity. Given how prevalent IAH is and the rarity of overt symptoms such as ascites, transurethral IAP

Table 2. Comparison of baseline parameters between the groups with and without intra-abdominal hypertension upon admission

Parameter	IAP <12 mm Hg (n = 11)	IAP ≥12 mm Hg (n = 19)	*P*-value
Demographic and clinical variables			
Age	68.82 (15.45)	74.26 (8.31)	0.21
Male	63.30%	68.40%	0.54
Body mass, kg	80.61(13.31)	97.63 (15.86)	**0.01**
IAP, mm Hg	8.95 (1.13)	14.92 (2.71)	**<0.001**
Heart rate, bpm	92.91 (21.94)	90.21 (19.93)	0.73
Systolic BP, mm Hg	119.73 (24.25)	120.84 (19.09)	0.89
Diastolic BP, mm Hg	68.73 (9.83)	77.11 (14.29)	0.09
MAP, mm Hg	86.00 (12.55)	91.67 (15.12)	0.30
RFG, mm Hg	68.09 (12.91)	61.82 (17.01)	0.30
In-hospital furosemide dose[a], mg	66.36 (24.61)	79.47 (38.65)	0.32
24 hours diuresis, ml	3667.73 (953.71)	2754.26 (1181.88)	**0.03**
Pulmonary congestion, %	100%	94.74%	0.63
Peripheral edema, %	72.73%	84.21%	0.37
Ascites, %	0%	21.05%	
Comorbidities			
Coronary arteries disease	54.50%	68.40%	0.35
Hypertension	63.30%	94.70%	**0.047**
Diabetes	63.30%	52.60%	0.42
Atrial fibrillation	73.00%	68.00%	0.57
Obesity	18.20%	57.90%	**0.04**
Echocardiographic variables			
LVEF, %	36.91 (15.85)	37.84 (13.14)	0.86
LVEDD, mm	56.00 (8.32)	60.00 (9.53)	0.25
End-expiratory IVC, mm	24.70 (4.55)	27.87 (4.70)	0.09
TAPSE, mm	15.82 (3.84)	16.69 (5.29)	0.71
S', cm/s	8.84 (2.17)	8.67 (3.33)	0.52
Baseline laboratory parameters			
NT-proBNP, pg/ml	13738.83 (9232.72)	11394.86 (12023.04)	0.21
Creatinine, mg/dl	1.57 (0.64)	1.36 (0.46)	0.28
eGFR, ml/min/1.73 m^2	50.09 (19.20)	59.79 (26.08)	0.29
Urea, mg/dl	66.11 (33.47)	75.39 (38.55)	0.43
Potassium, mmol/l	4.48 (0.75)	4.31 (0.68)	0.52
Sodium, mmol/l	136.00 (5.78)	138.95 (3.98)	0.10
Urinary osmolality, mOsm/kg	256.75 (56.02)	373.76 (143.12)	**0.01**
Urinary creatinine, mg/dl	26.51 (25.89)	58.54 (57.35)	0.11
Urinary potassium, mmol/l	26.04 (17.81)	35.57 (27.01)	0.54
Urinary sodium, mmol/l	83.00 (33.07)	87.03 (34.07)	0.75
Urinary chloride, mmol/l	96.10 (26.67)	99.39 (42.01)	0.82
Hemoglobin, g/dl	11.86 (1.24)	11.47 (2.21)	0.59
WBC, × 10^9/l	10.25 (3.21)	7.84 (3.15)	**0.01**
Hematocrit, %	36.36 (3.88)	35.96 (6.72)	0.86
CRP, mg/dl	26.34 (5.3–45)	16.13 (3.85–32.97)	0.37
Plasma protein, g/dl	5.83 (0.58)	6.49 (0.75)	**0.03**
Plasma albumin, g/dl	3.14 (0.53)	3.52 (0.55)	0.09
ALT, IU/l	163.64 (318.81)	37.74 (50.81)	0.83
AST, IU/l	107.73 (187.76)	49.50 (65.83)	0.94
GGTP, IU/l	110.10 (111.32)	132.53 (70.31)	0.26
Total bilirubin, mg/dl	1.80 (1.16)	1.74 (1.75)	0.61
Treatment			
ACEI before admission	45.45%	42.11%	0.57
Sartan before admission	9.09%	0.00%	0.36
ARNI before admission	9.09%	10.53%	0.70
β-blocker before admission	90.91%	68.42%	0.17
MRA before admission	54.55%	47.37%	0.50
Furosemide before admission	36.36%	42.11%	0.53
Torasemide before admission	27.27%	31.58%	0.57
Thiazide diuretic before admission	18.18%	31.58%	0.36
SGLT2I before admission	45.45%	47.37%	0.61
Ivabradine before admission	9.09%	5.26%	0.60
Digoxin before admission	9.09%	5.26%	0.60

Table 2 (cd.). Comparison of baseline parameters between the groups with and without intra-abdominal hypertension upon admission

Parameter	IAP <12 mm Hg (n = 11)	IAP ≥12 mm Hg (n = 19)	*P*-value
Calcium blocker before admission	0.00%	31.58%	0.05
ACEI on discharge	66.67%	77.78%	0.61
Sartan on discharge	0.00%	0.00%	-
ARNI on discharge	22.22%	11.11%	0.45
β-blocker on discharge	100.00%	94.44%	0.40
MRA on discharge	66.67%	61.11%	0.32
Furosemide on discharge	44.44%	33.33%	0.63
Torasemide on discharge	66.67%	61.11%	0.32
Thiazide diuretic on discharge	0.00%	27.78%	**0.006**
SGLT2I on discharge	77.78%	94.44%	0.64
Ivabradine on discharge	0.00%	0.00%	-
Digoxin on discharge	11.11%	16.67%	0.54
Calcium blocker on discharge	11.11%	5.56%	0.34

Quantitative variables are shown as mean values (standard deviations) or median values (1st–3rd quartiles), depending on whether their distribution met the normality criterion.

[a]Or equivalent in torasemide

Abbreviations: ACEI, angiotensin-converting-enzyme inhibitors; ARNI, angiotensin receptor-neprilysin inhibitor; MRA, mineralocorticoid receptor antagonist; SGLT2I, sodium-glucose cotransporter-2 inhibitors; other — see Table 1

measurement may be a useful tool in identifying patients with ADHF. Recently, there has been an ongoing search for IAP surrogate markers, but their use in the intensive cardiac care unit setting does not seem to have an advantage over traditional bedside measurements, especially given the developing possibility of continuous IAP monitoring [11–13].

Secondly, the IAH group exhibited poorer diuresis and higher urine osmolality despite receiving similar doses of diuretics and comparable eGFR. Elevated IAP has been causally linked to multifaceted organ and systemic dysfunctions, including central nervous, cardiovascular, and pulmonary systems, gastrointestinal tract, and kidneys [14–16]. Previous research has also indicated the potential role of IAH as a contributing factor to renal dysfunction in the HF population, however, data on pathomechanisms underlying this condition are scarce [1, 2, 17]. In recent years, the classic paradigm of assessing renal function through GFR evaluation has been challenged. While eGFR and serum creatinine determinations are the traditional approach to assessing renal function, their utility is limited by high inertia and low sensitivity in capturing early renal damage [18–20]. In search of other determinants of kidney damage, the scholarly focus has increasingly turned to venous congestion, dysregulation of the renal lymphatic system, and subsequent renal tamponade caused by compression of renal structures as additional factors [21, 22]. We suggest that all of these factors can get exacerbated in the face of IAH, which may partially account for our results. In our study, we did not observe an association of IAP with traditionally defined renal function. Some of the studies published to date may be confusing, due to differences in the studied population (e.g., the spectrum of LVEF or discrepancies in the prevalence of chronic kidney disease), which results in disparate results regarding the effect of IAP on renal function and risk of worsening of renal function [23–25].

Thirdly, the current ESC guidelines recommend that spot urine sodium assessment should constitute an integral part of clinical monitoring in the milieu of ADHF, serving as an early indicator of diuretic response. Although we did not observe a discernible effect of IAP on natriuresis, IAP measurements correlated with urine output and urine osmolality during the first day of hospitalization. We believe that in this type of kidney dysfunction, the compromised function of renal tubules causes the residual capacity for water absorption to remain, even if sodium absorption deteriorates. Further, the effect of IAP on water and sodium handling in current ADHF patients varies due to the pharmacological blockade of the renin-angiotensin-aldosterone system, sodium-glucose transport protein 2 inhibitors, and the administration of loop diuretics, which are cornerstones of HF therapy, whereas the observed satisfactory natriuresis may indicate correct dosing [26, 27]. Moreover, the number of osmotically active compounds in the final urine that affects its volume goes well beyond sodium, and observed alterations in the final urine may be the result of heightened dietary salt intake, metabolic alkalosis, and dyselectrolytemia, overactivation of the sympathetic nervous system, or individualized patterns of fluid redistribution and intravascular filling which restrict aquaresis [28, 29]. As time progressed, the correlation between IAP, diuresis, and urine osmolality diminished, likely due to the predominantly good response to the treatment in the IAH group. To gain more comprehensive insights into the impact of elevated IAP on diuretic response, future studies should consider incorporating modern scientific techniques to phenotype patients, including machine learning, clustering, multi-marker testing, as well as chemical biomarkers specifically designed to detect early renal tubular damage [30–32].

In addition, we showed that the employed treatment led to a pronounced effect by reducing IAP in patients

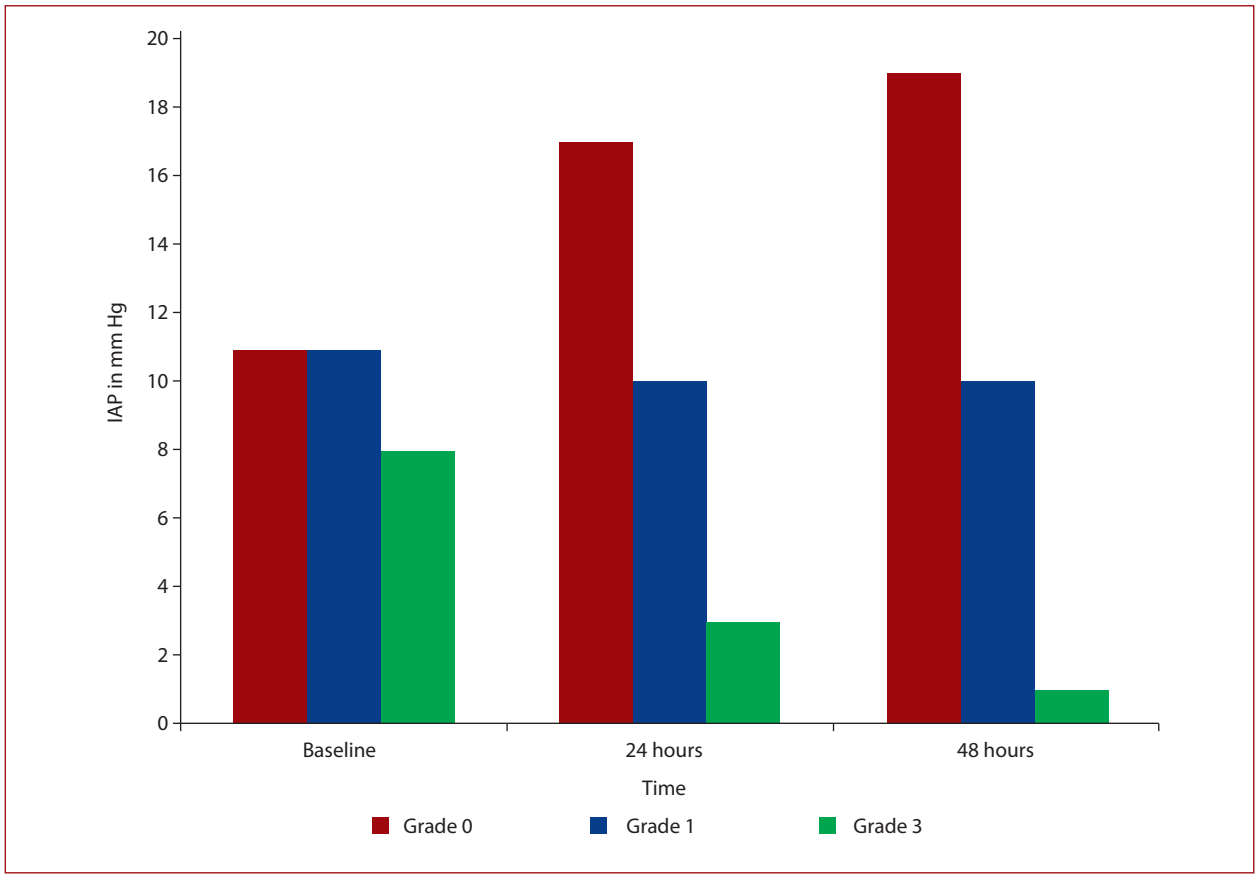

Figure 2. Change in the distribution of intra-abdominal pressure (IAP) grades over the time of the study

Grade 0 — IAP <12 mm Hg; Grade 1 — IAP between 12–15 mm Hg; Grade 2 — IAP between 16–20 mm Hg; Grade 3 — IAP between 21–25 mm Hg

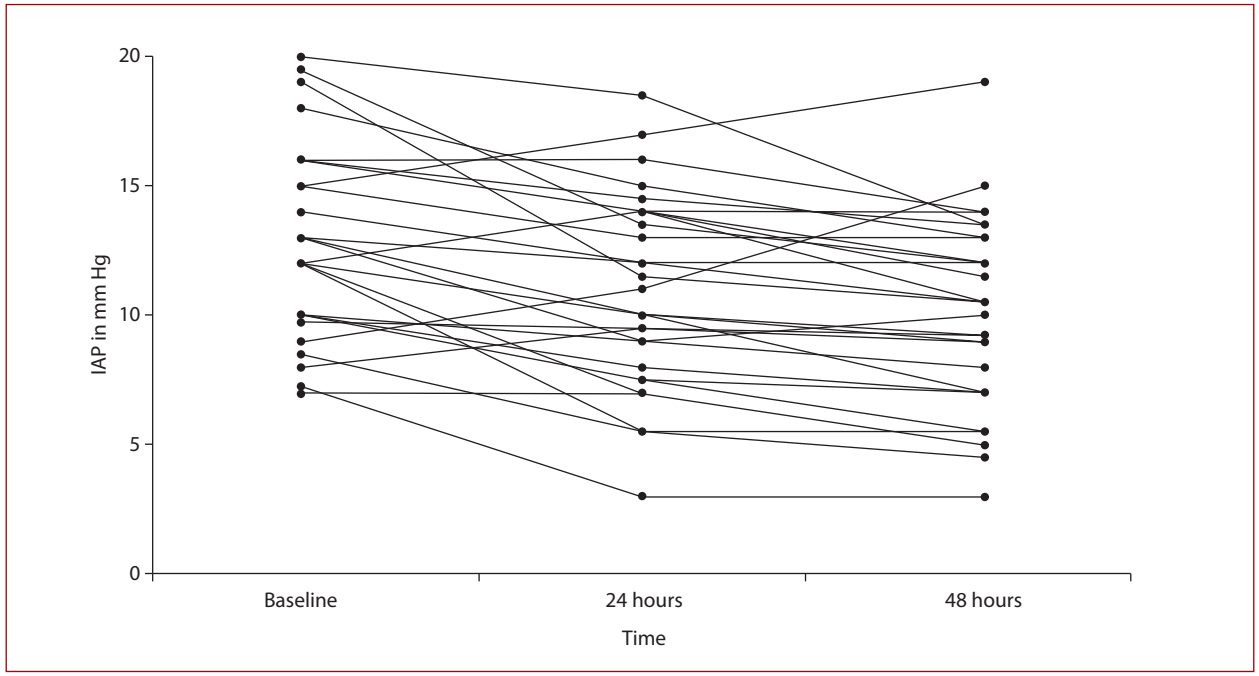

Figure 3. Changes in intra-abdominal pressure (IAP) in time

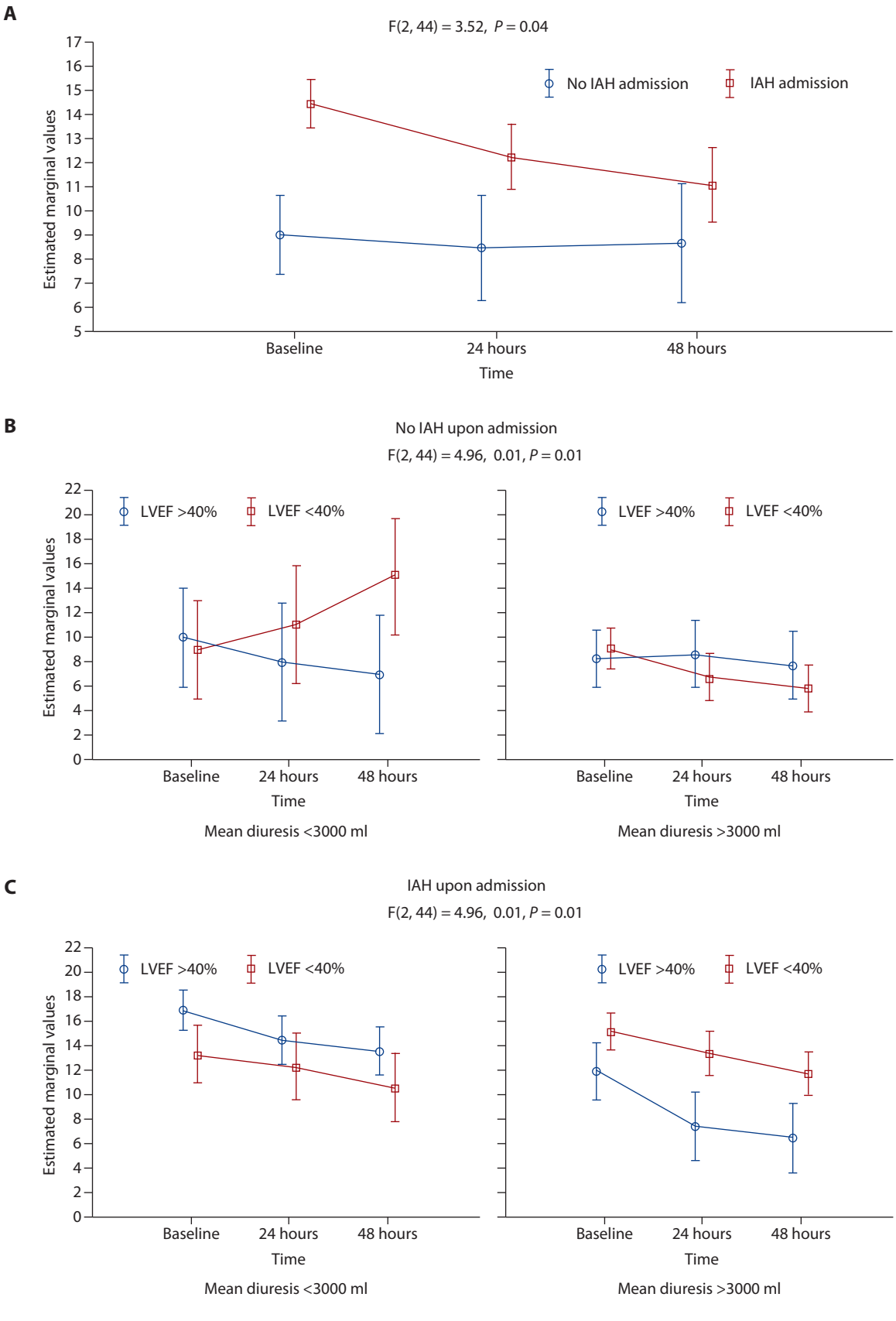

Figure 4. IAP values across different time points, in the context of occurrence of different clinical variables in tested the studied individuals according to RM-ANOVA analysis. **A.** With and without IAH upon admission. **B.** In the context of occurrence of variable diuresis and LVEF with no IAH upon admission. **C.** In the context of occurrence of variable diuresis and LVEF with IAH upon admission

Abbreviations: IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; IAP A, IAP on admission; IAP B, IAP at a 24-hour mark; IAP C, IAP at a 48-hour mark; LVEF, left ventricular ejection fraction

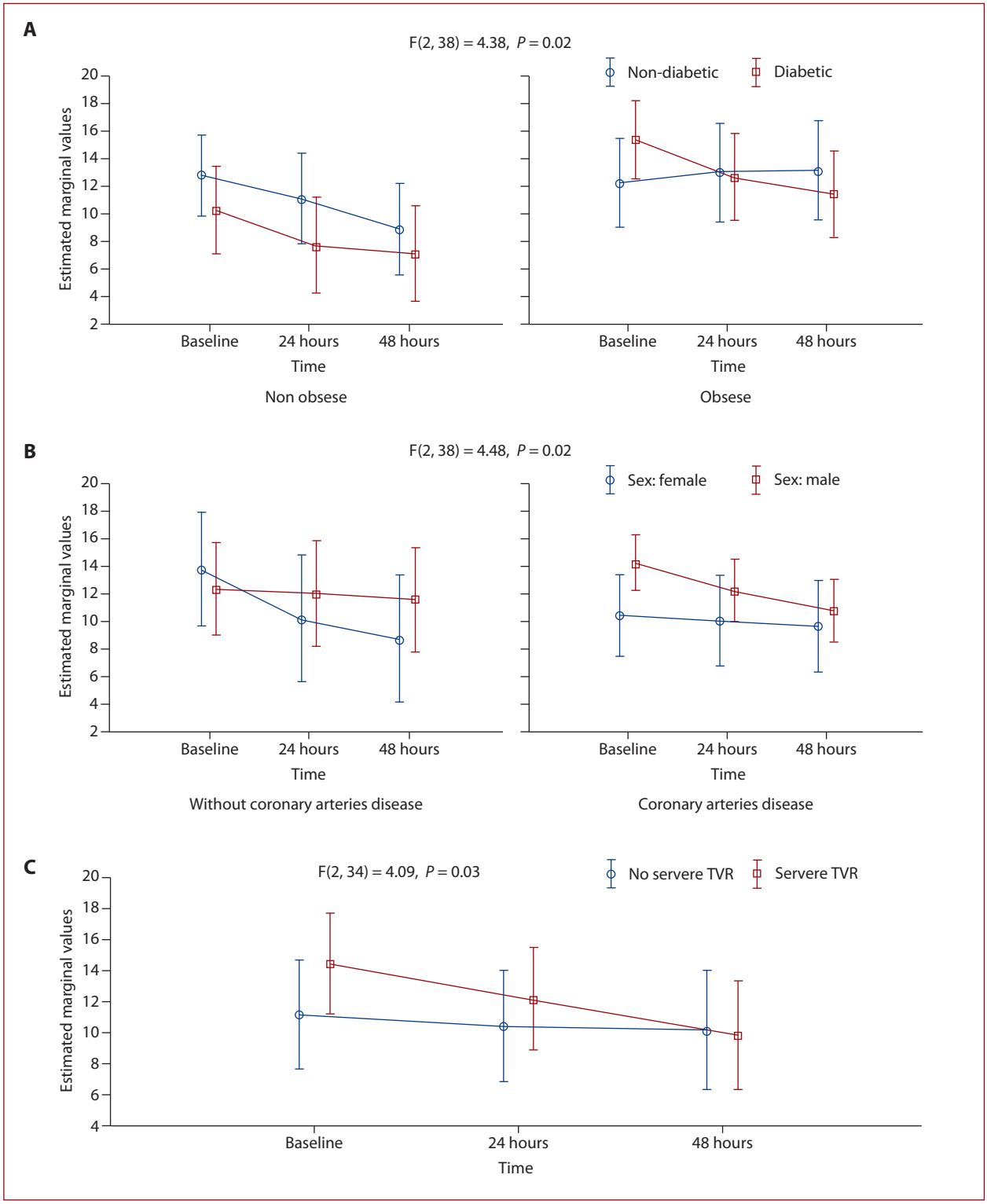

Figure 5. Intra-abdominal pressure values across different time points, in the context of occurrence of comorbidities in tested the studied individuals according to RM-ANOVA analysis. **A.** Obesity and diabetes. **B.** Coronary artery disease in different sexes. **C.** Severe tricuspid regurgitation (TVR)

Table 3. Spearman r coefficients with *P*-values, describing correlations between baseline (admission) intra-abdominal pressure and other variables

Variable pairs	r	*P*-value
IAP & diuresis	**–0.459**	**0.011**
IAP & creatinine	–0.223	0.236
IAP & uOsmolality	**0.420**	**0.033**
IAP & uCreatinine	0.241	0.246
IAP & uK	0.027	0.894
IAP & uNa	0.022	0.908
IAP & uCl	0.062	0.768

Significant (P <0.05) correlations are marked in bold

Abbreviations: Cl, chloride; K, potassium; Na, sodium; u, urine; other — see Table 1

admitted with IAH, whereas its impact was negligible in individuals without IAH. We suggest that in patients without IAH, loop diuretic therapy may be primarily targeting volume overload in the lungs and peripheral tissues, and as a result, the reduction in the abdominal fluid compartment might not be as prominent.

Finally, we demonstrated several distinctions between the groups, which align with the known risk factors for IAH development, as well as the impact of combined comorbidities on IAP dynamics. These observations, while promising, require further verification in a large cohort study.

CONCLUSIONS

The majority of patients admitted for ADHF were diagnosed with IAH despite the absence of overt signs of abdominal congestion. Patients with IAH were characterized by reduced urinary output compared to the group with IAP below 12 mm Hg. The study establishes the association of elevated IAP upon demonstrates with decreased diuresis, even with the presence of satisfactory spot urine sodium levels.

Limitations

The study poses several limitations. It was a single-center study, which ensures that the results cannot be applied with certainty to the general population. An obvious limitation of this pilot study is the size of the cohort. The small number of subjects increases the likelihood of insufficient statistical power to identify potential differences between groups and associations with IAP.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

Article information

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