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Article

Acute Kidney Injury Secondary to Abdominal Compartment Syndrome: Biomarkers, Pressure Variability, and Clinical Outcomes

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Abstract: Abdominal compartment syndrome (ACS) is a severe clinical condition caused by intra-abdominal hypertension (IAH), often observed in surgical and trauma patients. However, ACS can also develop in non-surgical patients with massive ascites, leading to acute kidney injury (AKI) due to renal hypoperfusion. This study investigates the association between intra-abdominal pressure (IAP) changes, renal biomarkers, and mortality in patients with ACS-related AKI. A prospective cohort study was conducted on 24 hospitalized patients with ascites due to malignancy, cirrhosis, or heart failure. IAP was measured via the trans-vesical method on the 1st and 7th days of hospitalization. Serum and urinary biomarkers, including kidney injury molecule-1 (KIM-1), neutrophil gelatinase-associated lipocalin (NGAL), and interleukin-6 (IL-6), were assessed for their correlation with IAP changes. The primary outcome was in-hospital mortality, and secondary outcomes included AKI progression and the effect of paracentesis on IAP reduction. The overall in-hospital mortality rate was 50%. Patients who survived had significantly lower IAP on the 7th day compared to those who died (14.9 ± 3.5 mmHg vs. 20.2 ± 5.6 mmHg, $p = 0.01$). A 25% reduction in IAP was associated with improved kidney function and increased survival ($p < 0.001$). Urinary KIM-1 and serum NGAL levels showed a moderate correlation with IAP ($r = 0.55$, $p = 0.02$ and $r = 0.61$, $p = 0.018$, respectively), while IL-6 levels were significantly higher in non-survivors ($p = 0.03$). Paracentesis was associated with improved survival outcomes ($p = 0.04$). ACS is a critical but often overlooked cause of AKI in non-surgical patients with massive ascites. Lowering IAP significantly improves renal function and reduces mortality. Urinary KIM-1 and serum NGAL may serve as useful biomarkers for monitoring IAP changes. Early identification and management of IAH through timely interventions such as paracentesis and volume control strategies could improve patient outcomes.

Keywords: Acute kidney injury; abdominal compartment syndrome; intra-abdominal pressure; KIM-1; NGAL; mortality

1. Introduction

The abdominal cavity maintains a physiological pressure gradient due to the presence of serous fluid between the visceral and parietal layers of the peritoneum. This fluid, typically ranging from 5 to 20 mL, generates a normal intra-abdominal pressure (IAP) of approximately 5–7 mmHg under physiological conditions (1). This pressure plays a crucial role in maintaining organ function by facilitating the lubrication of abdominal organs and supporting electrolyte diffusion within the peritoneal cavity. However, when IAP exceeds 12 mmHg, a condition known as intra-abdominal hypertension (IAH) develops, which can have significant pathophysiological consequences (2).

IAH is classified into four grades based on IAP measurements: grade 1 (12–15 mmHg), grade 2 (16–20 mmHg), grade 3 (21–25 mmHg), and grade 4 (>25 mmHg). Grades 3 and 4 are typically associated with abdominal compartment syndrome (ACS), a critical condition characterized by progressive organ dysfunction due to sustained elevation of intra-abdominal pressure (3). ACS is commonly observed in postoperative and trauma patients; however, non-surgical conditions such as massive ascites secondary to cirrhosis, malignancies, or heart failure may also lead to ACS (4).

IAH can manifest in acute, subacute, or chronic forms, depending on the underlying etiology. Acute IAH typically develops within hours and is often linked to trauma, surgical interventions, or internal hemorrhage, where a rapid increase in intra-abdominal volume leads to a sudden elevation in IAP. In such cases, mortality rates can reach up to 60% (5). Conversely, subacute IAH is more frequently associated with conditions such as decompensated heart failure, advanced liver cirrhosis, or malignancy-related ascites, where IAP rises more gradually. Chronic IAH, on the other hand, is observed in conditions such as pregnancy and morbid obesity, where the risk of ACS is lower but persistent elevation in IAP may contribute to chronic organ dysfunction, including renal impairment (6).

Beyond its direct effects on abdominal organs, IAH has systemic implications. Increased intra-abdominal pressure can elevate intrathoracic pressure, leading to reduced functional residual capacity of the lungs and impaired respiratory mechanics (7). Similarly, cardiovascular function may be compromised due to decreased venous return, reduced cardiac output, and inferior vena cava compression, predisposing patients to deep vein thrombosis (8). Furthermore, visceral hypoperfusion can lead to bacterial translocation and sepsis-like syndromes. Acute kidney injury (AKI) is one of the most severe consequences of IAH, resulting from decreased renal perfusion and filtration pressure. When left untreated, IAH-induced AKI may progress to multiple organ dysfunction syndrome (MODS), a critical condition associated with poor prognosis and high mortality (9).

This study aims to investigate the impact of intra-abdominal pressure changes on mortality in patients who develop ACS due to non-surgical medical conditions. In addition to monitoring IAP, we evaluate the prognostic significance of key serum and urinary biomarkers, including interleukin-6 (IL-6), superoxide dismutase (SOD), neutrophil gelatinase-associated lipocalin (NGAL), fibroblast growth factor-23 (FGF-23), and kidney injury molecule-1 (KIM-1), to explore their association with IAP fluctuations and patient outcomes. By elucidating the pathophysiological mechanisms linking ACS and AKI, our findings may contribute to the development of targeted therapeutic strategies for improving prognosis in high-risk patients.

2. Methods

2.1. Study Design and Setting

This study was conducted in a tertiary care training and research hospital. It was designed as a **single-center, prospective, observational cohort study** to evaluate the impact of IAP changes on mortality in patients who developed **AKI secondary to non-surgical medical conditions**. The study population included hospitalized patients in the internal medicine ward, who had **ascites due to malignancy, cirrhosis, or heart failure** and developed AKI. Ethical approval was obtained from the institutional review board (**Approval No: 2018.06.1.05.055**), and all participants provided written informed consent before enrollment.

2.2. Patient Selection, Inclusion and Exclusion Criteria

Patients were included based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria for AKI, which define AKI as an absolute increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours, an increase in serum creatinine to ≥ 1.5 times the baseline level within the last seven days, or a urine output of <0.5 mL/kg/hour for six consecutive hours. Exclusion criteria included patients with AKI caused by nephrotoxic agents, glomerulonephritis, vasculitis, or other identifiable renal

pathologies, as well as those with a history of prior abdominal surgery that could interfere with IAP measurements. After applying these criteria, only patients with an IAP ≥ 12 mmHg at admission were included in the final analysis.

2.3. Intra-Abdominal Pressure Measurement

IAP was measured **twice**, on **Day 1 and Day 7 of hospitalization**, using the **transvesical method with a Foley catheter** (8). The patient was placed in the **supine position**, and external abdominal tension was carefully avoided. **25 mL of isotonic saline** was instilled into the bladder, and the catheter was clamped at the intersection of the **spina iliaca and midaxillary line**. The pressure was then measured in **cmH₂O** once fluid fluctuations ceased. To convert values to **mmHg**, the following correction factor was applied:

$$\text{IAP (mmHg)} = \text{IAP (cmH}_2\text{O)} \times 0.74$$

2.4. Laboratory Measurements and Clinical Follow-Up

Routine laboratory parameters, including **serum creatinine, electrolytes, complete blood count (CBC), albumin, inflammatory markers** (C-reactive protein [CRP], procalcitonin), **uric acid, bicarbonate (HCO₃), parathyroid hormone (PTH), ferritin, lactate dehydrogenase (LDH), and transaminases**, were monitored daily.

Patients were initially treated with diuretics, with dosages adjusted according to **volume status and urine output**. Patients who were **unresponsive to diuretics** underwent **paracentesis and/or hemodialysis**. Data on **daily diuresis, total diuretic dosage, and frequency of hemodialysis or paracentesis** were systematically recorded.

2.5. Biomarker Analysis

To investigate potential prognostic biomarkers associated with IAP changes and mortality, the following markers were measured: Superoxide Dismutase (SOD), a marker of oxidative stress and ischemia/reperfusion injury, known to fluctuate during AKI; Fibroblast Growth Factor-23 (FGF-23), a phosphaturic hormone associated with chronic kidney disease (CKD) progression and potentially indicative of AKI-to-CKD transition; KIM-1, a proximal tubular injury marker that is highly sensitive to ischemia, hypoxia, and toxic injury, and is excreted in urine; NGAL, which is released from tubular cells in response to acute kidney inflammation and is detectable in both serum and urine; and IL-6, a pro-inflammatory cytokine elevated in septic and ischemic AKI, known to drive CRP synthesis.

2.6. Biomarker Sample Collection and Analysis

Blood and urine samples were collected **on Days 1 and 7**, coinciding with **IAP measurements**. Samples were stored at **-80°C** until analysis. Before testing, samples were **gradually thawed** (first to **-20°C**, then to **+4°C**). Biomarkers were measured using **commercial ELISA kits** with **Microplate Reader RT 2100C** and **Microplate Washer RT 2600C** devices.

2.7. Statistical Analysis

Data normality was assessed using the Shapiro–Wilk test. Continuous variables were reported as mean $\pm$ standard deviation (SD) for normally distributed data or as median [interquartile range (IQR)] for non-normally distributed data, while categorical variables were presented as counts and percentages. Comparative analyses were conducted using the Chi-square or Fisher's exact test for categorical variables, Student's t-test for normally distributed continuous variables, and the Mann–Whitney U test for non-normally distributed continuous variables. Correlation analyses were performed using Spearman's correlation test for non-parametric data and Pearson's correlation test for normally distributed data. Multivariate logistic regression analysis was used to identify

independent predictors of mortality and AKI progression. Statistical analyses were conducted using SPSS version 23.0 (SPSS Inc., USA), and a p-value <0.05 was considered statistically significant.

3. Results

A total of 24 patients were included in the study, with a mean age of **69.2 ± 9.0 years**, and a median hospital stay of **14.8 ± 7.1 days**. The primary underlying conditions were **congestive heart failure (n=12), cirrhosis (n=6), and malignancies (n=6; ovarian cancer (n=2), colon cancer (n=2), endometrial cancer (n=1), and gastric cancer (n=1))**. The overall in-hospital mortality rate among patients with IAH was **50%**.

IAP levels did not significantly differ between survivors and non-survivors (**22.2 ± 5.7 mmHg vs. 23.1 ± 4.1 mmHg, p=0.34**). However, at **Day 7**, patients who survived had significantly lower IAP compared to those who died (**14.9 ± 3.5 mmHg vs. 20.2 ± 5.6 mmHg, p=0.01**). A reduction of at least **25% in IAP** was significantly associated with **AKI recovery (p=0.01)** and overall survival (**p<0.001**). Logistic regression analysis demonstrated that changes in IAP were independently associated with mortality (**OR: 1.88, 95% CI: 1.19–2.95, p=0.006**). Additionally, **paracentesis was found to be significantly associated with improved survival outcomes (p=0.04)**, whereas the use of **diuretics and hemodialysis** did not differ between groups.

When serum electrolytes were analyzed, **higher phosphorus and uric acid levels** were observed in patients who died. Although hyponatremia was more prevalent among non-survivors, sodium levels did not reach statistical significance between the groups. **Inflammatory markers (CRP, procalcitonin) were significantly elevated**, while **serum albumin levels were lower** in non-survivors. Additionally, serum **HCO₃ levels were significantly reduced in non-survivors compared to survivors (15.8 ± 4.0 vs. 22.5 ± 8.4, p=0.02)**, indicating a possible metabolic acidosis in critically ill patients.

Among the studied **biomarkers, urinary KIM-1 and serum NGAL levels** showed a significant correlation with IAP levels (**r=0.55, p=0.02** and **r=0.61, p=0.018**, respectively). Furthermore, **IL-6 levels were significantly higher in non-survivors compared to survivors (11.4 ± 7.0 pg/mL vs. 5.6 ± 5.5 pg/mL, p=0.03)**. However, no statistically significant difference or correlation was found for **SOD or FGF-23** with IAP levels or clinical outcomes.

A detailed comparison of the groups and associated variables is presented in Table 1.

Table 1. Comparison of patients survivors and non-survivors in their hospital admissions.

Parameter	Survivors (n=12)	Non-survivors (n=12)	p-value
Age (years)	66.5 ± 9.3	72.1 ± 8.1	0.1
Male (n, %)	3 (25%)	3 (25%)	1.0
Malignancy (n, %)	4 (33.3%)	2 (16.6%)	0.34
Creatinine (mg/dL)	2.6 [2.0-4.57]	3.0 [1.7-4.7]	0.2
Sodium (mmol/L)	134.25 ± 2.9	131.3 ± 5.0	0.09
Potassium (mmol/L)	4.2 ± 1.2	4.4 ± 0.8	0.3
Phosphorus (mg/dL)	4.2 ± 1.1	5.4 ± 1.4	0.029
Calcium (mg/dL)	7.9 ± 1.6	8.1 ± 0.6	0.4
Uric acid (mg/dL)	8.3 ± 3.0	12.3 ± 5.4	0.03
Albumin (g/dL)	2.8 ± 0.4	2.4 ± 0.3	0.05
CRP (mg/L)	43.5 ± 65.8	128.9 ± 71.4	0.01
Procalcitonin (ng/mL)	0.6 ± 0.7	3.0 ± 1.7	0.001
Parathormon (pg/mL)	218.7 ± 198.4	164.5 ± 170.7	0.48

^d HCO ₃ (mEq/L)	22.5 ± 8.4	15.8 ± 4.9	0.02
^a ALT (U/L)	32.0 ± 9.5	139.9 ± 320.3	0.26
‡AST (U/L)	30.2 ± 36.7	177.8 ± 357.4	0.16
^b LDH (U/L)	363.5 ± 160.3	678.9 ± 599.6	0.09
Hemoglobin (g/dL)	10.1 ± 1.1	10.0 ± 1.6	0.86
Ferritin (ng/mL)	241.4 ± 217.5	503.1 ± 598.5	0.16
Daily diuresis (mL)	1366 ± 1117	1108 ± 631.6	0.49
Diuretic dose (mg)	211.6 ± 150.3	160.8 ± 81.6	0.26
Need for hemodialysis (n, %)	6 (50%)	5 (41.6%)	0.68
Paracentesis (n, %)	8 (66.6%)	3 (25%)	0.04
IAP on 1st day (mmHg)	23.1 ± 4.1	22.2 ± 5.7	0.34
25% decrease in IAP (n)	12 (100%)	1 (8.3%)	0.0001
IAP on 7th day (mmHg)	14.9 ± 3.5	20.2 ± 5.6	0.01
* Change in IAP* between 1st - 7th day (mmHg)	9 ± 3.9	1.08 ± 2.8	0.001
Follow-up time (days)	14.0 ± 4.6	12.1 ± 2.8	0.21
§IL-6 levels (pg/mL)	5.6 ± 5.5	11.4 ± 7.0	0.03

*IAP: Intraabdominal Pressure. §IL-6: Interleukin-6. ^aALT: Alanine Aminotransferase. ‡AST: Aspartate Aminotransferase. ^bLDH: Lactate Dehydrogenase. ^cCRP: C-reactive protein. ^dHCO₃: Serum Bicarbonate.

4. Discussion

IAH is a well-recognized pathological condition that can lead to ACS by reducing abdominal perfusion pressure and compromising organ function [13]. Early recognition and timely intervention to reduce IAP are crucial in preventing organ injury and improving clinical outcomes. In ACS, impaired renal venous return and reduced cardiac output contribute to the development of AKI, which may further exacerbate systemic hemodynamic instability. This study specifically focused on non-surgical patients with ACS-related AKI and employed **intravesical pressure measurements** as a surrogate for IAP monitoring, ensuring the exclusion of patients with prior abdominal surgeries to enhance measurement accuracy.

Previous studies have established a strong association between elevated IAP and impaired renal function [14–16]. Demarchi et al. reported that AKI developed when IAP exceeded **8 mmHg** in a cohort of 60 patients [17]. Additionally, increased IAP has been linked to higher mortality rates in critically ill patients, particularly in those undergoing surgery or suffering from sepsis, acute pancreatitis, or trauma [18,19]. In pediatric ICU settings, even a **1 mmHg increase in IAP** was shown to negatively affect prognosis and prolong ICU stay [18]. Similarly, a study of postoperative ICU patients demonstrated a significant association between elevated IAP and mortality, with rates increasing from **10-25% in grade 1 and 2 IAH to 75-90% in ACS cases** [20].

To the best of our knowledge, no previous study has systematically investigated ACS secondary to malignancy, heart failure, or liver cirrhosis, nor assessed the impact of IAP changes on renal function and survival in this patient population. The majority of existing literature has focused on **surgical ICU patients** who developed ACS as a result of sepsis and/or trauma. In contrast, the present

study included **only non-ICU patients**, all of whom had **massive ascites** and were managed with diuretic therapy, hemodialysis/hemofiltration, and/or paracentesis. The observed **in-hospital mortality rate of 50%** in our study is consistent with previously reported mortality rates ranging between **40% and 100%** in ACS patients.

Our findings highlight the clinical significance of **reducing IAP** in improving renal function and overall survival. A **≥25% reduction in IAP** was independently associated with **improved kidney function and reduced mortality**, suggesting that IAP modulation should be a key therapeutic target in ACS management. Given that ACS can precipitate AKI in patients with massive ascites, **paracentesis** emerges as a reasonable intervention to rapidly alleviate intra-abdominal pressure. Additionally, **diuretic therapy and ultrafiltration via hemodialysis** may further enhance renal function and reduce mortality risk in these patients. To optimize patient outcomes, clinicians should implement **early and effective volume control strategies** to prevent progression to multi-organ dysfunction.

For patients in whom **abdominal hypertension is suspected**, **regular IAP monitoring** can guide therapeutic decisions. Although dependent on operator expertise, **the trans-vesical method remains a simple and practical approach** for IAP measurement. However, **biomarkers may serve as alternative non-invasive monitoring tools**. In this study, **urinary KIM-1 and serum NGAL** emerged as promising **biomarkers correlated with IAP**, suggesting their potential role in assessing treatment response. Additionally, **serum IL-6 levels were significantly higher in non-survivors**, indicating a possible prognostic value in ACS patients. IL-6 levels may therefore help **identify high-risk patients who require more aggressive intervention strategies**. Furthermore, routine inflammatory markers such as **CRP** may provide prognostic insights, while elevated **uric acid levels and decreased HCO₃** were associated with poorer outcomes, highlighting the need for closer monitoring in these patients.

Despite its strengths, this study has certain limitations. Firstly, the **small sample size** may have limited statistical power, as the study focused specifically on **patients with ACS**, rather than including those with milder forms of **grade 1 or 2 IAH**. Future studies with larger cohorts may help refine these findings by incorporating a **broader range of IAP levels**. Secondly, although malignancy is an independent risk factor for mortality, **the proportion of malignancy cases did not significantly differ between survivors and non-survivors**, suggesting that IAP changes were a more critical determinant of outcomes in this study. Thirdly, due to the limited sample size, **subgroup analyses** for different **ACS etiologies (heart failure, cirrhosis, and malignancy)** could not be performed, necessitating further research to explore potential differences in disease progression and response to treatment.

In conclusion, our findings underscore the importance of **early IAP monitoring and intervention** in patients with ACS-related AKI. Lowering IAP through **paracentesis, diuretics, and hemodialysis** can improve renal function and overall survival. Moreover, **biomarkers such as KIM-1, NGAL, and IL-6** may serve as valuable tools for risk stratification and treatment optimization. Further **prospective studies with larger cohorts** are warranted to validate these findings and refine management strategies for this high-risk patient population.

5. Conclusions

ACS is a complex clinical condition characterized by visceral, renal, and cardiopulmonary complications resulting from sustained IAH. While ACS has been extensively studied in surgical, trauma, and critically ill patients—where it is associated with high morbidity and mortality—its occurrence in non-surgical settings, particularly in patients with massive ascites due to **congestive heart failure, liver cirrhosis, or malignancy**, remains underrecognized. The present study highlights the significant role of **ACS in the development of AKI** in these patients, emphasizing the importance of **early detection and intervention** to mitigate adverse outcomes.

Our findings demonstrate that **reducing IAP is associated with improved renal function and decreased mortality**. These results support the clinical utility of **serial IAP measurements** as a guiding tool for both **diagnostic and therapeutic decision-making** in high-risk patients.

Furthermore, biomarkers such as **urinary KIM-1 and serum NGAL** emerged as potential indicators of **IAP fluctuations**, suggesting their role in **non-invasive monitoring and risk stratification**.

Given the substantial mortality associated with ACS in this population, **early recognition and timely intervention** remain critical. Future research should focus on **prospective studies with larger cohorts** to further refine management strategies and establish optimal treatment protocols for **ACS-related AKI in non-surgical patients**.

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