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ORIGINAL ARTICLE

Heart and kidney crosstalk: risk factors, clinical features, and short-term outcomes associated with acute kidney injury in patients suffering acute non-ST elevation myocardial infarction

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ABSTRACT

Aim Acute kidney injury (AKI) presents a high mortality complication in patients with acute myocardial infarction (AMI). Yet, its correlation with non-ST elevation myocardial infarction (NSTEMI) remains neglected in the literature. This study aims to investigate the prevalence, risk factors, clinical features, and short-term outcomes associated with AKI development in patients with acute NSTEMI.

Methods A one-year prospective observational cohort study involved 170 consecutive patients hospitalized in the Intensive Care Department of the Internal Medicine Clinic at the University Clinical Centre Tuzla diagnosed with acute NSTEMI. Patients were subsequently categorized into AKI and non-AKI groups based on AKI development within 48 hours. Demographic characteristics, laboratory findings, and short-term clinical outcomes were compared between the groups.

Results Of 170 patients, 31 (18.2%) developed AKI within 48 hours of acute NSTEMI. Significant age differences, blood urea nitrogen (BUN), creatinine, estimated glomerular filtration rate (eGFR), blood glucose level (BGL), C-reactive protein (CRP), and high sensitivity (hs) troponin were observed, making patients with lower baseline kidney function, more extensive myocardial infarction, and a heavier systemic inflammatory response following acute NSTEMI more susceptible to AKI development. In the follow-up period, mortality rates were significantly higher in the AKI group, amounting to 35.5% compared to 10.1% in the non-AKI group. Additionally, mortality increased with the severity of AKI, reaching 100% in AKI stage 2.

Conclusion This study highlights demographic, clinical and laboratory findings in patients with acute NSTEMI, which contribute to AKI development. Early detection and tailored interventions are crucial in mitigating AKI-associated morbidity and mortality.

Keywords: cardiac biomarkers, intensive care unit, kidney failure, myocardial ischemia

INTRODUCTION

Acute Kidney Injury (AKI) is defined as a rapid decline in baseline kidney function, developing within hours or a few days, involving structural damage to the kidney and impairment of its function (1). This syndrome is diverse, often presenting with overlapping contributing factors that complicate its diagnosis and management (2). AKI incorporates various etiologies, including intrinsic kidney pathology that affects the glomerulus, interstitium, or tubule (acute onset glomerulonephritis, tubular necrosis, interstitial nephritis, and intratubular obstruction) and extrarenal pathology (prerenal and postrenal) (3). The prerenal form of AKI is caused by reduced blood flow to the kidney. This may be part of systemic hypoperfusion resulting from hypovolemia (hemorrhage, gastrointestinal fluid losses, severe burns), hypotension from reduced cardiac output (myocardial infarction, cardiac failure, pulmonary embolism), hypotension from systemic vasodilatation (sepsis, anaphylaxis), or selective hypoperfusion of the kidneys caused by renal vasoconstriction (NSAID's, iodinated contrast, hepatorenal syndrome) or vasodilation of efferent arteriole (ACE inhibitors, angiotensin receptor blockers) (4). Post renal etiology for AKI includes obstructive causes, most commonly prostatic enlargement in older men, pelvic masses in postmenopausal women, and calculi in younger patients. In the past, a widely used term to describe this type

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of kidney dysfunction was acute renal failure (ARF), but it included only the most severe acute decline in kidney function, characterized by significant azotaemia and frequently accompanied by oliguria or anuria (5). This term was replaced with the term AKI considering new evidence suggesting that even slight injury or kidney dysfunction indicated by minor changes in serum creatinine levels (sCr) and/or urine output (UO) can serve as a predictor of serious clinical repercussions (6). Epidemiological data imply that even mild, reversible AKI carries significant clinical consequences, that include an amplified risk of mortality and developing or worsening pre-existing chronic kidney disease (CKD) (7). AKI and CKD are now recognized to have a bidirectional relationship. Existing CKD increases the risk of developing AKI. Conversely, exposure to AKI, especially if it occurs repeatedly, accelerates the progression of CKD compared to those who have not experienced AKI (8). AKI affects 30 - 60% of critically ill patients, and is associated with increased morbidity and mortality (9). Acute coronary syndrome (ACS) can be categorized into three subgroups: ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina (10). AKI may develop because of rapid hemodynamic changes caused by acute coronary syndrome (11). Reduced cardiac output and venous return subsequently lead to a decrease in the glomerular filtration rate (GFR). Additionally, there is a cascade of events unfolding in patients with AMI that exacerbate kidney damage. These patients experience acute activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS), which can result in vasoconstriction causing glomerular damage (12). In the setting of acute cardiomyocyte breakdown, activation of the systemic inflammatory response and oxidative stress also negatively impacts renal function by damaging renal tubular cells. This damage reduces the ability of the kidney to reabsorb sodium and water, which leads to decreased circulating blood volume and additionally impairs renal perfusion (13). Renal damage in patients with acute myocardial infarction (AMI) may be influenced by disruptions in the coagulation system. Systemic inflammation can trigger platelet activation, which may lead to microthrombosis in the glomeruli and subsequent nephron damage (14). Additionally, metabolic factors such as elevated glucose levels and acidosis can exacerbate renal impairment (15). The criteria for diagnosing AKI are outlined by standardized guidelines from the Kidney Disease: Improving Global Outcomes (KDIGO) group. According to these guidelines, AKI is defined by an increase in serum creatinine of at least 0.3 mg/dL (≥26.5 mmol/L) within 48 hours or a 1.5-fold increase from baseline within the past 7 days. Alternatively, AKI can be indicated by a urine output of less than 0.5 mL/kg/h for at least 6 hours (16). The development of AKI in acute NSTEMI imposes a great burden on short-term in-hospital mortality. Unfortunately, studies about this important issue were not previously conducted in our institution. Moreover, a comprehensive review of the existing literature did not find similar studies conducted in Bosnia and Herzegovina (B&H). We aimed to examine the prevalence, risk factors, clinical features, and in-hospital mortality associated with AKI in patients with acute NSTEMI.

PATIENTS AND METHODS

Patients and study design

In this prospective observational cohort study, we included 170 consecutive patients diagnosed with an acute NSTEMI admitted to the Intensive Care Department of the Internal Medicine Clinic at the University Clinical Centre Tuzla B&H, between January and December 2023. Inclusion criteria comprised newly diagnosed acute NSTEMI with normal or elevated baseline blood urea nitrogen (BUN) and creatinine level, and age >18. Exclusion criteria were acute STEMI, stable coronary artery disease, prior NSTEMI or STEMI, and patients with a history of end-stage renal disease (ESRD) requiring dialysis treatment.

All patients were evaluated and subsequently categorized into two groups: the AKI group (n=31) and the non-AKI group (n=139) based on AKI development within 48 hours from admission. The groups were compared according to demographic characteristics, laboratory findings, and short-term clinical outcomes over a three-month follow-up period.

The dataset comprised a range of demographic and medical history information. It included gender, age, height, weight, smoking status, history of hypertension, hyperlipoproteinemia, alcohol consumption, and family history of cardiovascular disease. Clinical data were collected from electronic medical records, anamnesis, and physical examination.

All patients provided an informed consent after explaining the study's purpose. An ethical approval was obtained from the Ethical Committee of the University Clinical Centre Tuzla.

Methods

The laboratory data were derived from the analysis of venous blood samples taken from all patients on admission and again after 48 hours. Those data included a full blood count of white blood count (WBC), red blood cell count (RBC), hemoglobin (Hb), hematocrit (HCT), platelet count (PLT), blood glucose level (BGL), blood urea nitrogen (BUN), creatinine, potassium (K), calcium (Ca), cholesterol, triglycerides, C-reactive protein (CRP), high sensitivity Troponin I (hsTroponin I), and ferritin. To estimate the glomerular filtration rate (eGFR), serum creatinine levels, initially recorded in μmol/L, were converted to mg/dL and then applied to the 2021 CKD-EPI Creatinine formula (17).

Acute NSTEMI was diagnosed using a combination of traditional symptoms of chest pain, characteristic electrocardiographic changes, serial increases in serum cardiac biomarkers, and wall motion abnormalities observed on an echocardiogram. According to KDIGO criteria, AKI is identified by a rise in SCr of more than 0.3 mg/dL within 48 hours and an increase of at least 1.5 times the baseline level within the first 7 days. The stages of AKI are classified as follows: Stage 1 involves a 1.5- to 1.9-fold increase over baseline or an SCr rise of at least 0.3 mg/dL; Stage 2 involves a 2.0- to 2.9-fold increase over baseline; Stage 3 includes a 3.0-fold increase over baseline, an SCr rise of at least 4 mg/dL, or the need for renal replacement therapy (16).

Statistical analysis

Descriptive statistics were employed to outline demographic and clinical features. Categorical data were displayed as frequencies

and percentages, whereas continuous data were described using means with standard deviations (SD) or medians with interquartile ranges (IQR), depending on their distribution. A comparative analysis between patients who developed AKI and those who did not was conducted using the χ^2 test (association between the presence of AKI and lethal outcome) or Fisher's exact test for categorical variables when the sample size was small or the expected frequencies were low, instead of the χ^2 test. T-test was used to compare the mean values of continuous variables between the two groups or Mann-Whitney U tests for continuous variables when the data did not meet the assumptions required for t-tests (such as normality). Differences between the analysed variables were considered significant if the $p<0.05$.

RESULTS

A total of 170 patients met the inclusion criteria. The median age was 69 (IQR= 61-77), with predominance of males, 103 (60.6%). Demographic and clinical characteristics of patients who developed AKI and those who did not develop AKI were compared and noteworthy differences between groups were observed in age stratification (Table 1).

In total, 31 (18.2%) patients developed AKI, and 139 (81.7%) did not. Laboratory findings upon admission and after 48 hours of hospitalization were obtained for all patients (Table 2).

Table 1. Demographicc characteristics of the patients with or without acute kidney injury (AKI)

Variable	No (%) of patients in the group			p
	Total	Non-AKI	AKI	
Age (based on median of 69) (years)				
<69	84 (49.4)	74 (53.2)	10 (32.3)	4.463
≥69	86 (50.6)	65 (46.8)	21 (67.7)	
Gender				
Male	103 (60.6)	86 (61.9)	17 (54.8)	0.525
Female	67 (39.4)	53 (38.1)	14 (45.2)	
Smoker				
Yes	85 (50.0)	72 (51.8)	13 (41.9)	0.986
No	85 (50.0)	67 (48.2)	18 (58.1)	
Arterial hypertension				
Yes	149 (87.6)	125 (89.9)	24 (77.4)	3.663
No	21 (12.4)	14 (10.1)	7 (22.6)	
Diabetes mellitus				
Yes	71 (41.8)	56 (40.3)	15 (48.4)	0.684
No	99 (58.2)	83 (59.7)	16 (51.6)	
Hyperlipoproteinemia				
Yes	124 (72.9)	104 (74.8)	20 (64.5)	1.364
No	46 (27.1)	35 (25.2)	11 (35.5)	
Alcohol use				
Yes	63 (37.1)	55 (39.6)	8 (25.8)	2.058
No	107 (62.9)	84 (60.4)	23 (74.2)	
Familial history of cardiovascular disease				
Yes	132 (77.6)	110 (79.1)	22 (71.0)	0.975
No	38 (22.4)	29 (20.9)	9 (29.0)	

AKI, acute kidney injury;

The markers of renal function, creatinine ($p<0.001$), BUN ($p<0.001$), eGFR ($p<0.001$), and inflammatory marker CRP ($p<0.001$) were significantly higher in the AKI group after 48 hours. Other parameters associated with inflammation, including WBC ($p=0.054$) and ferritin ($p=0.942$) levels, did not show a statistically significant difference after 48 hours. No significant difference was observed in parameters related to erythrocyte and thrombocyte count, hemoglobin levels, electrolyte status, lipid status, or blood glucose level between the groups. The median value of high-sensitive troponin I (hsTroponin I) in the non-AKI group was lower on admission and after 48 hours than the median values reported in the AKI group, but without statistical significance.

According to KDIGO criteria for AKI and AKI staging, out of 31 patients with AKI, 28 (16.4%) developed AKI stage 1, three (1.7%) developed AKI stage 2, and no patients with AKI stage 3. Of patients who developed AKI stage 1, eight (28.6%) patients had a fatal outcome in the follow-up period, and in patients who developed AKI stage 2 all had a fatal outcome in the follow-up period (Table 3). There was a statistically significant association between the presence of AKI and lethal outcome ($p<0.001$).

DISCUSSION

The results of this study showed that 18.2% of patients developed AKI within 48 hours of experiencing an acute NSTEMI. This prevalence underscores the importance of closely monitoring renal function in these patients. Persistent moderate-to-severe AKI is linked to a higher risk of death than transient moderate-to-severe AKI in patients after an AMI (18). Thus, there is a necessity for early diagnosis of these high-risk patients to improve their clinical outcomes.

Our analysis revealed several findings regarding demographic and clinical characteristics associated with the development of AKI. Notably, older age was significantly associated with an increased risk of AKI. The median age of our patients was 69 years, with patients above the median age being more likely to develop AKI compared to younger patients. This aligns with previous research indicating that advanced age is a major risk factor for AKI in various clinical settings (19). In contrast, reportedly the survival outcomes of elderly patients with AKI are the same as those of younger patients with AKI, although the elderly required prolonged ICU stays and renal replacement therapy (RRT) (20); this difference may be attributed to sampling bias. Younger patients are less likely to be admitted to the ICU, and when they are, their condition tends to be more severe compared to elderly patients (21).

Our study found no significant differences between AKI and non-AKI groups in terms of gender distribution or comorbidities, such as arterial hypertension, diabetes, hyperlipoproteinemia, smoking history, or alcohol use. Previous research has suggested that individuals with AKI are more likely to have cardiovascular disease, diabetes, and Intensive Care Unit admission, but shows no significant differences in ethnicity, smoking status, or baseline measurements of body mass index, systolic, or diastolic blood pressure (22,23).

Laboratory parameters play a crucial role in diagnosing and monitoring AKI. In our study, significant differences were observed between patients with and without AKI. Our observation of a higher median value of hsTroponin I in the AKI group suggests a potential correlation between the severity of myocardial infarction and the likelihood of developing AKI. Patients with higher levels of hsTroponin I, indicating more severe myocardial necrosis, resulting in hemodynamic instability and

Table 2. Laboratory findings in the patients with or without acute kidney injury (AKI)

Variable	Groups		p
	Non-KI	AKI	
	Median (Q1 – Q2)		
WBC* (x10^9/L)	9.60 (7.70 - 11.30)	10.97 (7.70 - 14.81)	0.173
WBC†	8.68 (7.01 - 10.60)	10.20 (7.60 - 11.94)	0.054
RBC* (x10^12/L)	4.52 (4.08 - 4.91)	4.21 (3.67 - 4.76)	0.024
RBC†	4.52 (4.01 - 4.89)	4.02 (3.63 - 4.71)	0.004
Hb* (g/L)	138 (125 - 153)	127 (103 - 146)	0.007
Hb†	140 (123 - 151)	127 (102 - 139)	0.003
Hct* (L/L)	0.409 (0.373 - 0.446)	0.382 (0.307 - 0.417)	0.006
Hct†	0.405 (0.364 - 0.447)	0.372 (0.305 - 0.421)	0.003
PLT* (x10^9/L)	220 (179 - 267)	241 (205 - 295)	0.085
PLT†	223 (173 - 267)	251 (195 - 291)	0.176
Glucose* (mmol/L)	7.1 (5.9 - 10.8)	9.1 (7.2 - 14.8)	0.004
Glucose†	6.8 (5.6 - 8.7)	7.1 (6.1 - 9.3)	0.224
BUN* (mmol/L)	6.6 (5.2 - 9.9)	9.8 (7.7 - 14.7)	<0.001
BUN†	7.1 (5.6 - 10.3)	18.8 (10.1 - 25.1)	<0.001
SCr* (mmol/L)	91 (76 - 112)	131 (96 - 177)	<0.001
SCr†	91 (76 - 111)	194 (143 - 247)	<0.001
eGFR* (ml/min/1.73m2)	74.75 (50.43 - 92.32)	43.39 (28.19 - 70.99)	<0.001
eGFR†	75.15 (52.31 - 91.80)	30.58 (16.29 - 42.59)	<0.001
K* (mmol/L)	4.2 (3.9 - 4.5)	4.2 (3.9 - 4.8)	0.524
K†	4.2 (3.9 - 4.5)	4.1 (3.9 - 4.7)	0.969
Ca* (mmol/L)	2.30 (2.19 - 2.38)	2.26 (2.08 - 2.43)	0.736
Ca†	2.27 (2.16 - 2.39)	2.27 (2.09 - 2.32)	0.105
Triglyceride* (mmol/L)	1.98 (1.34 - 2.55)	1.47 (1.05 - 2.41)	0.051
Triglyceride†	1.99 (1.35 - 2.55)	1.50 (1.13 - 2.44)	0.157
Cholesterol* (mmol/L)	5.61 (4.58 - 7.00)	5.11 (4.13 - 6.72)	0.358
Cholesterol†	5.64 (4.54 - 6.83)	5.06 (4.08 - 6.44)	0.164
CRP* (mg/L)	6.0 (2.3 - 21.0)	21.2 (4.4 - 67.0)	0.013
CRP†	12.0 (4.2 - 57.7)	32.7 (19.0 - 92.8)	<0.001
hsTroponin I* (pg/mL)	1207.75 (337.70 - 8726.20)	3798.50 (1327.90 - 16282.30)	0.025
hsTroponin I†	4501.4 (978.4 - 17997.8)	10418.8 (2251.2 - 27321.2)	0.139
Ferritin* (µg/L)	149.20 (56.90 - 321.50)	113.50 (46.80 - 326.90)	0.741
Ferritin†	158.7 (66.3 - 325.4)	146.3 (47.4 - 469.3)	0.942

*Values measured on admission, †values measured after 48 hours
Q1, 25th percentile; Q2, 75th percentile; WBC, white blood cell count; RBC, red blood cell count; Hb, hemoglobin; Hct, hematocrit; PLT, platelet count; BUN, blood urea nitrogen; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; K, potassium; Ca, calcium; CRP, C - reactive protein; hsTroponin I, high sensitivity Troponin I

Table 3. Survival rates of patients with and without acute kidney injury (AKI) during the follow-up period

Outcome (No of patients)	No (%) of patients in the group			
	Non-AKI	AKI Stage 1	AKI Stage 2	AKI Stage 3
Total (n=170)	139 (81.7)	28 (16.4)	3 (1.7)	0
Survived (n=145)	125 (89.9)	20 (71.4)	0	0
Died (n=25)	14 (10.1)	8 (28.6)	3 (100)	0

renal hypoperfusion, could be more likely to develop AKI. Similar data were reported by a retrospective study in China on 6014 patients that found an amplified risk of AKI in patients with increased troponin values (24). Elevated CRP level indicates a heightened inflammatory response (25). The severity of myocardial infarction is not only a hemodynamic problem, but also more extensive myocardial damage is accompanied by a more pronounced inflammatory cascade. This systemic inflammation can exacerbate renal injury and contribute to the development of AKI (26). This proinflammatory setting with increased hs-CRP level measured at hospital admission in patients with AMI was independently associated with AKI risk, its severity, and in-hospital clinical outcomes (27). Our results showed BUN and creatinine levels measured upon admission and after 48 hours were significantly lower in the non-AKI group than those in the AKI group. This implies pre-existing kidney dysfunction in AKI patients, increasing their vulnerability to additional kidney damage from hemodynamic changes and the inflammatory response associated with myocardial infarction (28). Many available studies imply that pre-existing renal dysfunction was the primary risk factor for developing AKI (29,30). Older age, more extensive myocardial infarction, heavier inflammatory response, and lower baseline kidney function predispose the development of AKI, and these findings highlight the multifactorial nature of AKI and the potential utility of these parameters in risk prediction and early detection (31). Furthermore, our analysis of AKI staging according to KDIGO criteria revealed that the mortality rate increased dramatically with the higher AKI class. This high mortality was reported in previous surveys (32) and underscores the importance of prompt treatment.

Treatment strategies for AKI involve stabilizing hemodynamics, maintaining adequate mean arterial pressure (MAP) to optimize tissue perfusion, fluid resuscitation, avoiding nephrotoxic agents in therapy, adjusting antimicrobial doses, and

managing complications such as volume overload (33). The gold standard of AKI prevention includes identifying at-risk patients and implementing preventive strategies to avoid AKI development or reduce its severity (34). AKI survivors need to be educated upon discharge about managing risk factors and the necessity for long-term nephrologist follow-up due to the increased risk of developing chronic kidney disease and end-stage renal disease (ESRD) (35).

Despite the valuable insights gained from our study, several limitations should be acknowledged. Our institutional practices, patient demographics, and resources could have influenced this single-center study. Furthermore, the small sample size may have affected the statistical power of our analysis, preventing us from performing multivariate analysis to examine the associations between various factors and the development of AKI. Future research efforts should focus on validating our findings in larger, prospective cohorts.

In conclusion, our study provides important insights into the demographic characteristics, risk factors, and laboratory findings associated with the AKI development in patients with acute NSTEMI. These results can help identify high-risk patients, enabling targeted and early interventions to reduce the occurrence and degree of AKI and relieve the mortality burden.

AUTHOR CONTRIBUTIONS

Conceptualization, M.B. and E.B.; Methodology, M.B.; Writing – review & editing, M.B.; Supervision, S.H. and E.B.; Data curation, L.R.T. and A.B.; Writing – original draft preparation, L.R.T. and A.B.; Software, N.A.J., A.R., L.F., A.J.E. and D.L.; Visualization, N.A.J., A.R., L.F., A.J.E., D.L. and K.L.; Investigation, K.L. All authors have read and agreed to the published version of the manuscript.

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