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Original Article

Predictors of Acute Kidney Injury in Patients with Acute Decompensated Heart Failure in Emergency Departments

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Abstract

Background: Acute kidney injury (AKI) frequently occurs as part of the disease progression in individuals diagnosed with heart failure (HF) marked by the concurrent occurrence of acute cardiac and renal impairment, a condition termed as acute cardiorenal syndrome. The clinical significance of this condition and its management have recently garnered considerable attention. This research seeks to ascertain clinical determinants of AKI among individuals presenting with acute decompensated heart failure (ADHF) in the emergency setting.

Methods: This case-control research was implemented on 70 patients with acute ADHF. The participants were divided equally into two groups: Group I, consisting of patients with worsening (decompensated) ADHF, and Group II, consisting of patients with new-onset (de novo) ADHF.

Results: Estimated glomerular filtration rate **(**EGFR) was markedly reduced in Group I versus Group II ($P < 0.05$).

Creatinine and blood urea nitrogen (BUN) levels were markedly elevated in Group I versus Group II ($P < 0.05$). The incidence of AKI and exposure to nephrotoxic drugs were also markedly elevated in Group I than in Group II ($P < 0.05$). Troponin levels were markedly elevated in the AKI group compared to the non-AKI group ($P = 0.05$).

Conclusions: In comparison to de novo HF, ADHF correlates with reduced eGFR, higher serum creatinine, BUN, and higher AKI and offending drugs for renal injury. AKI in patients with ADHF was accompanied with elevated troponin

Keywords: Acute Kidney Injury, Acute Decompensated Heart Failure, Emergency Departments, Cardiorenal Syndrome

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Introduction:

Acute decompensated heart failure (ADHF) predominant factor in hospital admissions worldwide. It represents a growing worldwide health challenge, impacting more than 26 million people across the globe. (1)

Acute kidney injury (AKI) often occurs during the natural course of heart failure (HF). The clinical significance of the simultaneous occurrence of acute cardiac and renal dysfunction, termed acute cardiorenal syndrome (CRS), and its management has recently garnered considerable attention. (2)

Researchers have applied heterogeneous criteria in defining and classifying AKI, among these are the RIFLE criteria—Risk, Injury, Failure, Loss, and End-stage kidney disease—proposed by the Acute Dialysis Quality Initiative. (3) AKI can be categorized into five stages: risk of renal injury, renal impairment, renal failure, loss of renal function, and end-stage kidney disease. (4)

In recent years, researchers have reported that individuals with acute heart failure (AHF) often experience "congestion," which refers to clinical manifestations of extracellular fluid overload resulting in elevated cardiac filling pressures. (5) This provokes compensatory responses, such as the activation of the renin-angiotensin-aldosterone system, the stimulation of the sympathetic nervous system, and the participation of various local mediators. (6) Collectively, these compensatory mechanisms aim to preserve intravascular volume, with renal congestion now acknowledged as an integral aspect of systemic congestion. This phenomenon, resulting from factors such as decreased cardiac output, tubuloglomerular feedback, raised intra-abdominal pressure, and heightened venous pressure, is recognized as a major contributor to renal function decline in ADHF. (7)

A substantial proportion of patients experiencing acute cardiorenal syndrome receive treatment in the emergency department .These results offer valuable insights into the current status of individuals with ADHF who manifest AKI in the emergency setting. (8)

The present indicative framework for AKI primarily depends on renal function biomarkers such as serum creatinine and urine output, which have been utilized in clinical practice for more than five decades, are recognized for their limited sensitivity and delayed response in detecting kidney injury. [9] Early detection of kidney damage during the preclinical stage using new diagnostic methods is crucial for timely intervention in AKI. As the definition and classification of AKI have evolved over the years, established prognostic models also differ based on study population, geographic setting, sample size, and methodological approach .(10) However, although many researchers report a marked prevalence of AKI and significant influence on prognosis in individuals diagnosed with ADHF, Cumulative data from multiple investigations remain insufficient. (11, 12)

This investigation seeks to explore potential determinants for AKI in individuals admitted to the ED due to ADHF.

Patients and Methods:

This case-control investigation was applied to 70 participants aged 18 years and older, of both sexes. The study period extended from January 2024 to December 2024, upon receiving consent from the Institutional Ethical Committee of Sohag University, Sohag, Egypt. Informed written approval was acquired from all participants and their relatives.

Patients were excluded if they were receiving upkeep renal replacement therapy (RRT), such as hemodialysis or peritoneal dialysis; had a documented history of chronic kidney disease (CKD) or kidney transplantation; underwent surgery within one week before or after the AKI event; or presented with conditions such as drug toxicity, rheumatologic or autoimmune diseases, or acute infections. Additional exclusion criteria included recent diuretic use or exposure to radiopaque contrast media within the previous 15 days; use of aminoglycosides, metformin, or nonsteroidal anti-inflammatory drugs (NSAIDs) within the past 7 days; and the presence of thyroid dysfunction.

Participants were evenly categorized into two study groups: Group I: with worsening (decompensated) ADHF and Group II: with new (de novo) ADHF.

All patients underwent comprehensive history taking, clinical examination, and laboratory investigations [complete blood count (CBC), blood urea nitrogen (BUN), urine analysis, serum creatinine, estimated GFR (eGFR), lipid profile, serum electrolytes (Na^+, K^+, Cl^-, and Ca^{2+}),

microalbuminuria, creatine kinase MB (CK-MB) test, troponin test and arterial blood gas (ABG)], radiological investigation (chest X-Ray, echocardiogram and abdominal ultrasound) and electrocardiogram (ECG).

Estimated GFR (eGFR):

In accordance with relevant guidelines, chronic kidney disease (CKD) in this study, the estimated glomerular filtration rate (eGFR) is operationally defined as an under 60 mL/min/1.73 m², sustained for no less than three months. [13] We applied both serum creatinine and urine output criteria when the relevant data were available in the electronic medical records. For patients with missing urine output data, only the creatinine criteria were used. The eGFR was calculated using the CKD Epidemiology Collaboration (EPI) equation.(14)

$GFR = 141 \times \min(Scr/\kappa, 1)^{\alpha} \times \max(Scr/\kappa, 1)^{-1.209} \times 0.993^{Age} \times 1.018$ [if female] _ 1.159 [if black], where Scr is serum creatinine, κ is 0.7 for females and 0.9 for males, α is -0.329 for females and -0.411 for males, min indicates the minimum of Scr/κ or 1, and max indicates the maximum of Scr/κ or 1.

AKI was delineated according to KDIGO criteria as elevated serum creatinine of 26.5 µmol/L (0.3 mg/dL) or a 50% elevation from the preadmission baseline (calculated as the mean of at least three readings over the six months prior to admission) within seven days of admission.(15)

Sample Size Calculation:

Estimation of required sample size was performed using MedCalc Software Ltd version 20, with 90% power and a 5% significance level. The expected area under the curve (AUC) of the receiver operating characteristic (ROC) for the prediction model of any-stage AKD was set at a minimum of 0.726, with the null hypothesis AUC defined as 0.5, based on findings from a previous study. (16) To compensate for potential attrition, an additional eight participants were enrolled, bringing the total study sample to 70 patients.

Statistical analysis

Data analysis was performed using SPSS version 26 (IBM Corp., Chicago, IL, USA). The Shapiro–Wilk test and histogram visualization were employed to evaluate the parametric distribution characteristics of the data. Parametric continuous variables were presented as mean ± standard deviation (SD) and compared between two groups using the independent samples t-test; comparisons among three groups were conducted using one-way ANOVA with Tukey's post hoc test. Non-parametric data were expressed as median values and interquartile ranges (IQR). Comparisons between two groups were conducted using the Mann–Whitney U test, whereas the Kruskal–Wallis test was applied for comparisons across three groups, with subsequent pairwise Mann–Whitney U tests performed as appropriate. Categorical data were reported as frequencies and percentages and analysed via the Chi-square test or Fisher's exact test, based on expected cell counts. Statistical significance was set at a p-value less than 0.05.

Results:

Patient Demographics, comorbidities, vital signs, red blood cell (RBCs), white blood cell (WBCs), hemoglobin (Hb), platelets (PLT), triglyceride, albumin, CK-MB, troponin, sodium, potassium, partial pressure of carbon dioxide (pCO2), partial pressure of oxygen (pO2) and PaO2/ fraction of inspiratory oxygen concentration (FiO2) were insignificantly differed between groups. EGFR was markedly reduced in group I than in group II (P value <0.05). Creatinine and BUN were markedly elevated in group I than in group II (P value <0.05). **Table 1**

Table 1: Patient Demographics, Comorbid Conditions, Vital Parameters, and Laboratory Results in the Groups Studied

		Group I (n=35)	Group II (n=35)	P value
Demographic data				
Age (years)		42.71 ± 9.44	39.74 ± 12.64	0.269
Sex	**Male**	16 (45.71%)	21 (60%)	0.231
	Female	19 (54.29%)	14 (40%)	
Weight (kg)		79.43 ± 4.26	77.8 ± 3.55	0.087
Height (cm)		173.11 ± 2.29	171.8 ± 4.14	0.104
BMI (kg/m^2)		26.51 ± 1.31	26.39 ± 1.61	0.752
Comorbidities	**DM**	22 (62.86%)	17 (48.57%)	0.229
	HTN	30 (85.71%)	24 (68.57%)	0.153
	Dyslipidemia	6 (17.14%)	5 (14.29%)	1
	HF	11 (31.43%)	7 (20%)	0.274
	Vascular disease	9 (25.71%)	8 (22.86%)	0.78
	Liver disease	11 (31.43%)	9 (25.71%)	0.596
	Pulmonary disease	4 (11.43%)	5 (14.29%)	1
HR (beats/min)		96.89 ± 10.81	93.14 ± 9.82	0.134
MAP (mmHg)		96.17 ± 7.27	99.91 ± 9.2	0.063
Laboratory investigations				
RBCs (10^6/µL)		3.27 ± 0.17	3.37 ± 0.31	0.109
WBCs (10^3/µL)		7.06 ± 0.6	6.91 ± 1.65	0.618
Hb (g/l)		10.23 ± 1.26	10.77 ± 1.19	0.068
PLT (10^3/ul)		205.74 ± 36.4	207.89 ± 29.14	0.787
Triglyceride (mg/dl)		375.71 ± 76.76	373.2 ± 76.82	0.891
Creatinine (mmol/L)		193.29 ± 54.48	89.69 ± 14.28	**<0.001***
Albumin (g/l)		36.57 ± 1.84	37.34 ± 1.81	0.081
eGFR (mL/min/1.73 m2)		34.77 ± 9.34	64.34 ± 12.69	**<0.001***
BUN (mg/dL)		13.86 ± 3.58	8.57 ± 1.6	**<0.001***
CK-MB (IU/L)		26.97 ± 2.23	27.69 ± 1.73	0.139
Troponin (ng/mL)		0.34 ± 0.19	0.33 ± 0.19	0.827
Sodium (mmol/L)		142.97 ± 1.85	143.57 ± 1.56	0.147
Potassium (mg/dL)		4.69 ± 0.15	5.47 ± 6.81	0.505
pCO2 (mmHg)		40.23 ± 2.6	41.26 ± 2.54	0.099
pO2 (mmHg)		88.71 ± 6.36	89.69 ± 6.51	0.530
PaO2/FiO2 ratio (mmHg)		268.14 ± 22.14	261.71 ± 18.99	0.197

Data are shown as mean ± standard deviation (SD) or as frequency (%). The following abbreviations are used: BMI (body mass index), DM (diabetes mellitus), HTN (hypertension), HR (heart rate), MAP (mean arterial blood pressure), RBCs (red blood cells), WBCs (white blood cells), Hb (hemoglobin), PLT (platelets), eGFR (estimated glomerular filtration rate), BUN (blood urea nitrogen), CK (creatine kinase), pCO_2 (partial pressure of carbon dioxide), pO_2 (partial pressure of oxygen), and FiO_2 (fraction of inspired oxygen). A p-value less than 0.05 was considered statistically significant.

AKI and offending drugs for renal injury were markedly elevated in group I than in group II (P value<0.05). The main causes of AKI and RRT requirements where no meaningful difference was observed for both groups. **Table 2**

Table 2: AKI Characteristics, Causative Factors, Nephrotoxic Drug Use, and RRT Necessity in the Groups Studied

		Group I (n=35)	Group II (n=35)	P value
AKI		21 (60%)	11 (31.43%)	**0.016***
Main Causes of AKI	**Cardiorenal Syndrome**	8 (22.86%)	4 (11.43%)	0.164
	Hypovolemia	2 (5.71%)	8 (22.86%)	
	Obstructive Uropathy	4 (11.43%)	3 (8.57%)	
	Intrarenal Causes	21 (60%)	20 (57.14%)	
Offending drugs for renal injury		14 (40%)	6 (17.14%)	**0.034***
RRT requirement		6 (17.14%)	3 (8.57%)	0.477

Data are shown as frequency (%), *significant as p-value <0.05, AKI: Acute kidney disease. RRT: Renal replacement therapy.

Demographic data, comorbidities, vital signs, RBCs, WBCs, Hb, PLT, triglyceride, serum creatinine, albumin, eGFR, BUN, CK-MB, sodium, potassium, pCO2, pO2, PaO2/FiO2, Contrast exposure, nephrotoxic drug use, and RRT necessity were comparable between both groups without significant differences.Troponin was markedly increased in the AKI group as opposed to the non-AKI group (P value =0.05). **Table 3**

Table 3: Demographics, Comorbidities, Vital Signs, Laboratory Findings, Offending Drugs, and RRT Needs of Group I

		AKI (n=21)	No AKI (n=14)	P value
Age (years)		45.05 ± 8.75	39.21 ± 9.66	0.073
Sex	**Male**	7 (33.33%)	9 (64.29%)	0.133
	Female	14 (66.67%)	5 (35.71%)	
Weight (kg)		79.81 ± 4.75	78.86 ± 3.48	0.525
Height (cm)		172.76 ± 2.23	173.64 ± 2.34	0.270
BMI (kg/m^2)		26.74 ± 1.38	26.16 ± 1.16	0.204
Comorbidities	**DM**	13 (61.9%)	9 (64.29%)	1
	HTN	19 (90.48%)	11 (78.57%)	0.369
	Dyslipidemia	3 (14.29%)	3 (21.43%)	0.664
	HF	8 (38.1%)	3 (21.43%)	0.460
	Vascular disease	7 (33.33%)	2 (14.29%)	0.262
	Liver disease	8 (38.1%)	3 (21.43%)	0.460
	Pulmonary disease	2 (9.52%)	2 (14.29%)	1
HR (beats/min)		98.95 ± 10.86	93.79 ± 10.33	0.169
MAP (mmHg)		95.19 ± 7.56	97.64 ± 6.81	0.336
Laboratory investigations				
RBCs ($10^6/\mu L$)		3.25 ± 0.18	3.3 ± 0.14	0.412
WBCs ($10^3/\mu L$)		6.97 ± 0.66	7.2 ± 0.48	0.266
Hb (g/l)		10.29 ± 1.45	10.14 ± 0.95	0.748
PLT (10^3/ul)		201.95 ± 36.8	211.43 ± 36.38	0.459
Triglyceride (mg/dl)		381.14 ± 85.88	367.57 ± 62.81	0.616
Creatinine (mmol/L)		186 ± 56.96	204.21 ± 50.56	0.340
Albumin (g/l)		36.48 ± 2.04	36.71 ± 1.54	0.713
eGFR (mL/min/1.73 m^2)		32.62 ± 7.89	38 ± 10.67	0.095
BUN (mg/dL)		13.86 ± 3.64	13.86 ± 3.63	1
CK-MB (IU/L)		26.76 ± 2.34	27.29 ± 2.09	0.504
Troponin (ng/mL)		0.39 ± 0.16	0.27 ± 0.21	**0.05***
Sodium (mmol/L)		143.14 ± 1.71	142.71 ± 2.09	0.511
Potassium (mg/dL)		4.7 ± 0.16	4.69 ± 0.14	0.786
pCO_2 (mmHg)		40.1 ± 2.76	40.43 ± 2.44	0.716
pO_2 (mmHg)		87.14 ± 6.07	91.07 ± 6.27	0.073
PaO_2/FiO_2 ratio (mmHg)		266.24 ± 22.69	271 ± 21.8	0.541
Offending drugs for renal injury		8 (38.1%)	6 (42.86%)	0.778
RRT requirement		4 (19.05%)	2 (14.29%)	0.714

Data are shown as mean ± SD or frequency (%), AKI: Acute kidney disease, BMI: Body mass index. DM: Diabetes mellitus, HTN: Hypertension, MAP: Mean arterial blood pressure. HR: Heart rate, *significant as p-value <0.05, RBCs: Red blood cell, WBCs: White blood cell, Hb: Hemoglobin, PLT: Platelets, eGFR: Estimated glomerular filtration rate, BUN: Blood urea nitrogen, CK: Creatine kinase. pCO2: Partial pressure of carbon dioxide, pO2: Partial pressure of oxygen, FiO2: Fraction of inspiratory oxygen concentration, RRT: Renal replacement therapy.

Age, sex, weight, height, BMI, comorbidities, vital signs, laboratory investigations, nephrotoxic drugs and RRT requirement were insignificantly different between groups. **Table 4**

Table 4: Demographics, Comorbidities, Vital Signs, Laboratory Findings, Offending Drugs, and RRT Needs of Group II

		AKI (n=11)	No AKI (n=24)	P value
Age (years)		38 ± 14.1	40.54 ± 12.14	0.588
Sex	**Male**	6 (54.55%)	15 (62.5%)	0.655
	Female	5 (45.45%)	9 (37.5%)	
BMI (kg/m^2)		25.9 ± 1.26	26.62 ± 1.72	0.224
Comorbidities	**DM**	8 (72.73%)	9 (37.5%)	0.075
	HTN	9 (81.82%)	15 (62.5%)	0.435
	Dyslipidemia	1 (9.09%)	4 (16.67%)	1
	HF	2 (18.18%)	5 (20.83%)	1
	Vascular disease	2 (18.18%)	6 (25%)	1
	Liver disease	2 (18.18%)	7 (29.17%)	0.685
	Pulmonary disease	2 (18.18%)	7 (29.17%)	0.639
HR (beats/min)		91.45 ± 10.27	93.92 ± 9.74	0.499
MAP (mmHg)		102.09 ± 7.57	98.92 ± 9.84	0.351
Laboratory investigations				
RBCs (10^6/µL)		3.39 ± 0.38	3.36 ± 0.28	0.779
WBCs (10^3/µL)		7.08 ± 1.92	6.83 ± 1.54	0.685
Hb (g/l)		10.91 ± 1.38	10.71 ± 1.12	0.650
PLT (10^3/ul)		211 ± 31.45	206.46 ± 28.61	0.675
Triglyceride (mg/dl)		375.45 ± 72.72	372.25 ± 80.17	0.911
Creatinine (mmol/L)		88 ± 15.49	90.46 ± 13.97	0.643
Albumin (g/l)		37.73 ± 1.79	37.17 ± 1.83	0.404
eGFR (mL/min/1.73 m^2)		61.36 ± 12.57	65.71 ± 12.77	0.355
BUN (mg/dL)		8.91 ± 1.45	8.42 ± 1.67	0.405
CK-MB (IU/L)		27.91 ± 1.64	27.58 ± 1.79	0.612
Troponin (ng/mL)		0.34 ± 0.19	0.33 ± 0.19	0.953
Sodium (mmol/L)		144.09 ± 1.51	143.33 ± 1.55	0.186
Potassium (mg/dL)		4.32 ± 0.18	5.99 ± 8.23	0.508
pCO_2 (mmHg)		40.73 ± 2.53	41.5 ± 2.55	0.411
pO_2 (mmHg)		88 ± 6.81	90.46 ± 6.36	0.306
PaO_2/FiO_2 ratio (mmHg)		255.36 ± 18.87	264.63 ± 18.72	0.184
Offending drugs for renal injury		2 (18.18%)	4 (16.67%)	1
RRT requirement		0 (0%)	3 (12.5%)	0.536

Data are shown as mean ± SD or frequency (%), *Significant as P value<0.05, BMI: Body mass index. DM: Diabetes mellitus, HTN: Hypertension, MAP: Mean arterial blood pressure, HR: Heart rate, RBCs: Red blood cell, WBCs: White blood cell, Hb: Hemoglobin, PLT: Platelets, eGFR: Estimated glomerular filtration rate, BUN: Blood urea nitrogen, CK: Creatine kinase. pCO2: Partial pressure of carbon dioxide, pO2: Partial pressure of oxygen, FiO2: Fraction of inspiratory oxygen concentration, RRT: Renal replacement therapy.

Discussion

ADHF is a rapidly progressing condition marked by fluid overload and pulmonary congestion, with symptoms like edema, raised jugular venous pressure, breathlessness, and weight gain, often requiring urgent treatment and hospitalization .[17, 18] De Novo AHF arises suddenly due to increased intracardiac pressures or myocardial dysfunction, commonly caused by cardiac ischemia. Its management prioritizes stabilizing hemodynamics and restoring heart function through reperfusion .[19, 20]

In the current study, sodium, potassium, pCO_2, pO_2, PaO_2/ (FiO_2), RBCs, WBCs, Hb, PLT, triglyceride, albumin, CK-MB, and troponin didn't differ between both participants. eGFR was reduced in worsening ADHF participants than in de novo ADHF patients. Creatinine and BUN were elevated in worsening ADHF participants than in de novo ADHF ones.

Supporting our findings, Galas et al. [21] found that eGFR was lower in ADHF than in de novo HF. Serum creatinine was elevated in ADHF than

in de novo HF. Troponin didn't differ between both patients.

Also, Nawrocka-Millward et al. [22] found that sodium, WBCs, Hb, PLT, and troponin didn't differ among the de novo AHF and ADHF patients. However, creatinine and blood urea didn't differ among the de novo AHF and ADHF patients. This difference may be due to variations in sample sizes, and we included both genders with variants while they included male predominance.

In our study, AKI and offending drugs for renal injury were elevated in worsening ADHF participants than in de-novo AHF participants. There were no differences regarding the main causes of AKI, contrast exposure, and RRT requirement between both patients.

AKI represents a key adverse outcome of acute HF and cardiogenic shock (CS). ADHF patients often have pre-existing kidney damage due to chronic HF, which makes them more susceptible to AKI. In contrast, de novo HF patients tend to have relatively normal kidney function before their acute presentation.[16] It was reported that AKI is common in patients with ADHF .[23]

A prospective study reported that AKI serves as a marker for poor outcomes in patients with ADHF complicated by CRS. [24]

Supporting our results, Bottiroli et al. .[25] reported that was diagnosed in 70 patients (79.5%). AKI is a prevalent and early manifestation of ADHF-CRS. Significant contributors to AKI pathogenesis include venous congestion and severe reductions in perfusion.

Also, a study reported that offending drugs for renal injury were elevated in ADHF participants compared to de novo AHF participants. [21, 22, 26]

In the current study, in worsening ADHF patients, troponin was elevated in AKI patients than without AKI patients. RBCs, WBCs, Hb, PLT, triglyceride, serum creatinine, albumin, eGFR, BUN, CK-MB, sodium, potassium, pCO2, pO2, and PaO2/FiO2 didn't differ among AKI patients and no AKI patients. The use of contrast agents, prescription of renally harmful drugs, and initiation of RRT weren't different between both patients.

In de novo AHF participants, RBCs, WBCs, Hb, PLT, triglyceride, serum creatinine, albumin, eGFR, BUN, CK-MB, sodium, potassium, pCO2, pO2, and PaO2/FiO2 didn't differ between both patients. Participants did not differ markedly with respect to contrast exposure, nephrotoxic drug use, or the necessity for RRT.

A retrospective multicenter observational study by Ge et al. .[27] found that troponin was more in the AKI than in the no AKI, and pO_2 wasn't different among the no AKI and the AKI participants. However, Hb, serum creatinine, albumin, eGFR, and BUN differed between the AKI and no AKI. However, sodium, pCO_2, and PaO_2/FiO_2 were reduced in the AKI than in non-AKI. This difference may be due to different sample sizes, and we included both genders with variants while they included male predominance.

This is supported by Hata et al. [28] found that serum creatinine, BUN, and offending drugs for renal injury weren't different among the non-AKI and the AKI.

In contrast with our findings, Lee et al. [5] reported that Hb, serum creatinine, eGFR, BUN, sodium, potassium, and offending drugs differed between AKI patients and non-AKI participants. This difference may be attributed to the larger sample sizes.

Limitations included single-center study may yield findings that differ from those observed in other settings. Additionally, a small sample size may limit the statistical power, potentially leading to insignificant results. We didn't evaluate other biomarkers such as NT pro brain natriuretic peptide. The study lacked a healthy control group.

Conclusions:

Our findings revealed that in comparison to de novo HF, ADHF is accompanied with lower eGFR, higher serum creatinine, BUN, and higher AKI and offending drugs for renal injury. AKI in patients with ADHF was correlated with elevated troponin, while there weren't differences except for height between those with AKI and no-AKI in patients with HF.

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Conflict of Interest: Nil

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