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Prognostic value of kidney function on immediate and short term outcome after revascularization in patients with ST-Segment elevation myocardial infarction (STEMI)

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Abstract

One of the most dangerous heart problems, ST-segment elevation myocardial infarction (STEMI) is linked to a wide range of potentially fatal consequences (cardiac and non-cardiac). Renal hypoperfusion and increased renal vascular resistance leading to acute kidney damage are potential outcomes of a STEMI episode characterised by low cardiac output and/or raised right ventricular sided pressure (AKI). The kidney damage caused by AKI may be temporary and resolve on their own, or it can be persistent and lead to the patient developing chronic cardio-renal syndrome (CRS), which has both immediate and long-term consequences. Renal impairment has been linked to poor cardiovascular outcomes, particularly heart failure, suggesting that monitoring renal function should be one of the primary targets of cardiac monitoring programmes.

Keywords: Revascularization In Patients; elevation myocardial; infarction (STEMI)

1. Introduction

Globally, cardiovascular diseases (CVD) account for the greatest number of deaths and hospitalizations (1). Acute coronary syndrome (ACS) and chronic coronary syndrome (CCS) are two extremes of the spectrum of presentation of coronary artery disease (CAD), which is often regarded as the most significant subtype of cardiovascular disease (2).

One of the primary comorbidities connected to myocardial infarction (particularly STEMI) is abnormal kidney function or kidney damage; evidence indicated around 30% of individuals with STEMI have underlying renal impairment either of acute insult or chronic kidney disease (3). (4).

When acute kidney injury (AKI) occurs with a STEMI, we call this a case of cardio-renal syndrome type 1 (CRS-1) (5).

Beyond the concept of dual organ damage, the most pressing issues in CRS seem to involve the activation of pathological intercorrelated cardiorenal connections or connectors (6).

Major cardiorenal connectors, including sympathetic stimulation, RAAS activation, and enhancement of the inflammatory/immunological system, have deleterious and harmful effects on cardiac and renal functions, as well as multiple other organs and systems, thereby increasing the overall morbidity and mortality of patients (7).

Several studies have shown that renal damage is a predictor of both short- and long-term negative outcomes among p STEMI patients, as shown by the fact that the death rate in STEMI patients with kidney failure is double that of individuals with normal kidney function (8).

This research aims to determine whether or not kidney function is a useful prognostic indicator for ST-segment elevation myocardial infarction patients undergoing revascularization, both in the short and long terms (STEMI).

2. Subject and methods

After getting clearance from the cardiology department at Benha Faculty of Medicine and receiving informed consent from all participants, researchers from National Heart Institute in the United States performed the prospective, single-center study from September 2018 to February 2019. Patients admitted to the hospital with a STEMI attack were divided into two groups, one with normal kidney function/CrCl (the control group, n=76) and another with abnormal kidney function/CrCl (n=74).

All patients had a full set of demographic data gathered on them, as well as a clinical examination, laboratory investigations (cardiac troponin I, N terminal Pro-BNP), electrocardiogram (ECG), and echocardiographic evaluation (ejection fraction /EF and left ventricular end diastolic volume index /LVEDVI). All patients retested their kidney function/CrCl three months later, and based on the results, group 2 was split in half: subgroup 2A included those who had returned to normal kidney function, whereas subgroup 2B included those whose kidney function remained abnormal. Laboratory, electrocardiogram, and echocardiographic examinations were repeated at 3- and 6-month intervals for all patients.

Number crunching

Microsoft Excel 2016 and SPSS 26.0 (Statistical Package for the Social Sciences) were used to tabulate and analyse the data.

For numerical parametric data, we calculated the mean SD (standard deviation), as well as the minimum and maximum of the range; for numerical non-parametric data, we calculated the median and the first and third inter-quartile range; and for categorical data, we calculated the number and percentage.

Quantitative variables were subjected to inferential analysis using the independent t-test for parametric data and the Mann Whitney U for non-parametric data from two separate patients. The Kruskal-Wallis statistic: When comparing more than two groups with skewed data, it is used as a non-parametric alternative to ANOVA to account for the fact that ANOVA's assumptions have been broken.

The Chi-square test was used for inferential analysis of qualitative data for mobile patients. P-values less than 0.05 were considered statistically significant; higher values were not. As a statistical indicator, the p-value indicates the likelihood that observed findings in a research were due to chance alone (9).

3.Results

The demographic data of the study population are presented in Table-1. There was non-significant statistical differences between both groups regarding all demographic data and risk factors.

Table (1) Comparison between the studied groups regarding demographic data.

Variable		Group (1) (N=76)		Group (2) (N=74)		Test value	P-value	Sig.
		No.	%	No.	%			
Age groups	50-55 years	26	34.2%	22	29.7%	X2= 0.768	0.857	NS
	56-59 years	12	15.8%	12	16.2%			
	60-65 years	14	18.4%	12	16.2%			
	66-72 years	24	31.6%	28	37.8%			
Age (years)	Mean± SD	60.93± 6.81		62.85± 6.42		T=1.777	0.078	NS
	Median	61		64				
	Range	50- 71		50- 72				
Gender	Male	40	52.6%	37	50 %	X2= 0.025	0.874	NS
	Female	36	47.4%	37	50 %			
Risk factors	DM	18	23.7%	20	27%	X2= 0.080	0.777	NS
	Hypertension	16	21.1%	17	23%	X2= 0.008	0.931	NS
	COPD	3	3.9%	5	6.8%	X2= 0.162	0.688	NS
	Smoking	20	26.3%	22	29.7%	X2= 0.08	0.777	NS

P value < 0.05 is significant, p ≤ 0.01 is high statistically significant, SD: Standard deviation, T= Student T test, X2= Chi-Square test.

This table shows mean ± SD and range of the age and age groups among the two studied groups. No statistically significant differences were observed between the two groups as regards age and age groups (P>0.05). No statistically significant differences were observed between group (1) and group (2) as regards gender (P>0.05).

No statistically significant differences were observed between group (1) and group (2) as regards DM, hypertension, COPD or smoking (P>0.05).

Table 2 showed results between the studied groups regarding laboratory data on admission;

Table (2) Comparison between the studied groups as per laboratory data on admission.

		Group (1) (N=76)	Group (2) (N=74)	Test value	P-value	Sig.
Creatinine clearance (ml/min./1.73m2)	Mean± SD	78.99 ± 8.11	47.26 ± 6.92	**ZMWU** = 10.577	**<0.001**	HS
	Median (IQR)	80 (73 – 85.5)	48 (40 – 53)			
	Range	62 - 93	30 - 59			
Troponin I (ng/ml)	Mean± SD	2.16 ± 0.81	3.74 ± 1.36	**ZMWU** = 7.085	**<0.001**	HS
	Median (IQR)	2.1 (1.4 – 2.9)	3.75 (2.8 – 4.8)			

NT-ProBNP (pg/ml)	**Range**	0.9 – 4.5	1 – 7.3	**ZMWU** = 10.185	**<0.001**	HS
	Mean± SD	466.14 ± 241.59	2223.78 ± 1095.91			
	Median (IQR)	395 (282.5 – 652.5)	2000 (1400 – 2900)			
	Range	150 - 972	700 - 5100			

P ≤ 0.05 is considered statistically significant, p ≤ 0.01 is considered high statistically significant, SD: standard deviation, comparison between groups done by Mann-Whitney U test and Student T test

This table shows distribution of laboratory data on admission among the two studied groups. The results showed that group (2) had significantly lower creatinine clearance compared to group (1) (P<0.001). While, Troponin I and NT-ProBNP showed significant elevation in group (2) as compared to group (1) (P<0.001).

Table 3 showed results between the studied groups regarding echocardiographic data on admission;

Table (3) Comparison between the studied groups as per echocardiographic findings on admission.

		Group (1) (N=76)	**Group (2) (N=74)**	**Test value**	**P-value**	**Sig.**
LVEDVI (ml/m2)	**Mean ± SD**	65.45 ± 8.13	65.24 ± 7.99	**ZMWU** = 0.128	0.898	NS
	Median (IQR)	64.5 (59 – 71)	63 (60 – 72)			
	Range	49 - 82	49 - 82			
EF (%)	**Mean ± SD**	57.45 ± 7.71	39.78 ± 5.73	**ZMWU** = 9.8881	**<0.001**	HS
	Median (IQR)	58 (51.5 – 65)	39 (35– 43)			
	Range	40 - 72	29 - 53			

P ≤ 0.05 is considered statistically significant, p ≤ 0.01 is considered high statistically significant, SD: standard deviation, comparison between groups done by Mann-Whitney U test

This table shows Echo findings on admission among the two studied groups. The results showed that group (2) had significantly lower EF compared to group (1) (P<0.001). While, no significant differences were observed between two studied groups as regards LVEDVI (P>0.05).

Table 4 showed results between the studied groups regarding laboratory data after 3 months;

Table (4) Comparison between the studied groups as per laboratory data after 3 months.

		Group (1) (N=73)	**Subgroup (2A) (N=30)**	**Subgroup (2B) (N=44)**	**Kruskal Wallis Test**	**P-value**	**P-value (between 2 groups)**
Creatinine clearance (ml/min./1.73m2)	**Mean ± SD**	78.51± 7.09	73.87 ± 5.51	42.64 ± 7.59	96.42	**<0.001**	P1-2A=0.107 P1-2B<**0.001** P2A-2B<**0.001**
	Median	79	74.5	43.5			
	(IQR)	(74 – 84)	(70 – 79)	(37.5 – 48.5)			
	Range	65 - 90	63 - 82	29 - 57			
Troponin I (ng/ml)	**Mean ± SD**	0.14± 0.15	0.17± 0.16	0.35 ± 0.14	42.85	**<0.001**	P1-2A=0.683 P1-2B<**0.001** P2A-2B<**0.001**
	Median	0.08	0.08	0.37			
	(IQR)	(0.02 – 0.23)	(0.04 – 0.31)	(0.25 – 0.45)			
	Range	0 – 0.6	0 – 0.43	0.1 – 0.67			
NT-ProBNP (pg/ml)	**Mean ± SD**	110.42 ± 45.85	113.93 ± 48.11	334.8 ± 135.17	76.28	**<0.001**	P1-2A=1.01 P1-2B<**0.001** P2A-2B<**0.001**
	Median	100	96	322			
	(IQR)	(76 – 135)	(80 – 143)	(207 – 436)			
	Range	45 - 225	43 - 214	125 – 610			

P ≤ 0.05 is considered statistically significant, p ≤ 0.01 is considered high statistically significant, SD: standard deviation, comparison between groups done by Kruskal Wallis Test

This table shows distribution of laboratory data after 3 months among the three studied groups. The results showed that group (2B) had significantly lower creatinine clearance compared to group (1) (P<0.001) and group (2A) (p<0.001). While, Troponin I and NT-ProBNP showed significant elevation in group (2B) as compared to group (1) (P<0.001) and group (2A) (p<0.001). There was no significant differenced noticed between group (1) and group (2A) as regards creatinine clearance, Troponin I and NT-ProBNP (p>0.05).

Table 5 showed results between the studied groups regarding echogardiographic findings after 3 months;

Table (5) Comparison between the studied groups as per echocardiographic findings after 3 months.

		Group (1) (N=73)	Subgroup (2A) (N=30)	Subgroup (2B) (N=44)	Kruskal Wallis Test	P-value	P-value (between 2 groups)
LVEDVI (ml/m2)	**Mean ± SD**	68.16 ± 7.83	69.71 ± 9.59	71.57 ± 7.85	4.765	0.092	P1-2A=0.147 P1-2B=0.051 P2A-2B=0.361
	Median (IQR)	69 (62 – 75)	70 (61 – 78)	73 (64.5 – 78)			
	Range	52 - 83	53 - 84	58 - 84			
EF (%)	**Mean± SD**	58.08 ± 6.86	42.67 ± 4.76	41.64 ± 3.81	99.71	**<0.001**	P1-2A<**0.001** P1-2B<**0.001** P2A-2B=0.579
	Median (IQR)	59 (52 – 63)	42.5 (39 – 47)	40.5 (39 – 45)			
	Range	42 - 71	35 - 52	35 - 50			

$P \leq 0.05$ is considered statistically significant, $p \leq 0.01$ is considered high statistically significant, SD: standard deviation, comparison between groups done by Kruskal Wallis Test

This table shows Echo findings after 3 months among the three studied groups. The results showed that EF was significantly decreased in both group (2A) and group (2B) compared to group (1) (P<0.001). While, no significant differences were observed between three studied groups as regards LVEDVI (P>0.05).

Table 6 showed results between the studied groups regarding laboratory data after 6 months;

Table (6) Comparison between the studied groups as per laboratory data after 6 months.

		Group (1) (N=73)	Subgroup (2A) (N=30)	Subgroup (2B) (N=44)	Kruskal Wallis Test	P-value	P-value (between 2 groups)
Creatinine clearance (ml/min./1.73m2)	**Mean ± SD**	79.44 ± 6.38	74.9 ± 8.51	43 ± 7.75	97.04	**<0.001**	P1-2A=0.072 P1-2B<**0.001** P2A-2B<**0.001**
	Median (IQR)	80 (74 – 84)	74.5 (68 – 78)	43.5 (38.5 – 48.5)			
	Range	65 - 91	62 - 97	28 - 56			
Troponin I (ng/ml)	**Mean ± SD**	0.11 ± 0.12	0.12 ± 0.11	0.41± 0.11	78.49	**<0.001**	P1-2A=0.504 P1-2B<**0.001** P2A-2B<**0.001**
	Median (IQR)	0.05 (0.01 – 0.19)	0.07 (0.04 – 0.17)	0.42 (0.31 – 0.48)			
	Range	0 – 0.4	0 – 0.37	0.22 – 0.66			
NT-ProBNP (pg/ml)	**Mean ± SD**	103.32 ± 35.16	103.77 ± 38.54	324.39 ± 121.19	83.49	**<0.001**	P1-2A=0.994 P1-2B<**0.001** P2A-2B<**0.001**
	Median (IQR)	100 (78 – 124)	98.5 (75 – 121)	316 (235.5 – 405)			
	Range	50 - 200	50 - 205	116 – 621			

$P \leq 0.05$ is considered statistically significant, $p \leq 0.01$ is considered high statistically significant, SD: standard deviation, comparison between groups done by Kruskal Wallis Test

This table shows distribution of laboratory data after 6 months among the three studied groups. The results showed that group (2B) had significantly lower creatinine clearance compared to group (1) (P<0.001) and group (2A) (p<0.001). While, Troponin I and NT-ProBNP showed significant elevation in group (2B) as compared to group (1) (P<0.001) and group (2A) (p<0.001). There was no significant differenced noticed between group (1) and group (2A) as regards creatinine clearance, Troponin I and NT-ProBNP (p>0.05).

Table 7 showed results between the studied groups regarding echogardiographic findings after 3 months;

Table (7) Comparison between the studied groups as per echocardiographic findings after 6 months.

		Group (1) (N=73)	Subgroup (2A) (N=30)	Subgroup (2B) (N=44)	Kruskal Wallis Test	P-value	P-value (between 2 groups)
LVEDVI (ml/m2)	**Mean ± SD**	66.51 ± 7.58	70 ± 9.06	82.75 ± 7.52	63.29	**<0.001**	P1-2A=0.359 P1-2B<**0.001** P2A-2B<**0.001**
	Median (IQR)	66 (60 – 73)	71 (63 – 77)	85 (78 – 88.5)			
	Range	52 - 83	53 - 84	63 - 94			
EF (%)	**Mean ± SD**	58.1 ± 6.42	54.53 ± 7.48	39.3 ± 3.76	89.41	**<0.001**	P1-2A=0.291 P1-2B<**0.001** P2A-2B<**0.001**
	Median (IQR)	58 (54 – 63)	55 (49 – 60)	39 (37 – 42)			
	Range	43 - 69	39 - 66	31 - 48			

$P \leq 0.05$ is considered statistically significant, $p \leq 0.01$ is considered high statistically significant, SD: standard deviation, comparison between groups done by Kruskal Wallis Test

This table shows echo findings after 6 months among the three studied groups. The results showed that group (2B) showed significant elevation in LVEDVI compared to group (1) (P<0.001) and group (2A) (P<0.001). Meanwhile, group (2B) showed significant decrease in EF compared to group (1) (P<0.001) and group (2A) (P<0.001).

4. Discussion

Acute STEMI accounts for nearly a third of all deaths in the globe due to cardiovascular causes (10). Heart attacks typically happen when a vulnerable atheromatous plaque bursts, blocking off blood flow to the heart. This causes a cascade of ischemic events, beginning in the subendocardial layer and progressing to the epicardial layer, and ultimately resulting in transmural necrosis and the death of the heart's cardiomyocytes (1)

When addressing a case of STEMI, it is important to remember that this condition is connected to a number of consequences that might impair the patient's overall mortality and prognosis (11). Clinical complications (such as acute heart failure or cardiogenic shock), electrical complications (such as potentially fatal arrhythmias), and mechanical complications (such as ventricular septal rupture or cardiac rupture) can all lead to sudden cardiac death if not treated promptly and effectively after a STEMI (12).

About 30 percent of patients with acute STEMI show indications of renal impairment, either from an acute insult or chronic kidney disease (13).

The mortality rate is doubled in STEMI patients with kidney injury compared to those with normal kidney function, and STEMI can cause a marked decline in kidney function with the possibility of dialysis. On the other hand, renal injury commonly affects STEMI prognosis by increasing the risk of serious arrhythmia, acute heart failure, and even mechanical ventilation (14).

Therefore, the purpose of our work was to identify the prognostic role of kidney function among ischemic patients experiencing a STEMI attack on short-term outcomes; our results showed that AKI was associated with in-hospital adverse outcomes among STEMI patients, such as serious arrhythmic events, acute pulmonary edoema, and mechanical ventilation, which was consistent with the findings of Ibrahim et al. 2021(15), Naves et al. 2016(16), Marenzi et al. 2013 (18). Unlike Marenzi et al. (2013) 17 and Venkatason et al. (2019 19), we found no association between AKI and an increased risk of mortality or cardiogenic shock. This was likely due to the exclusion of patients with associated STEMI mechanical complications, such as advanced age and multiple co-morbidities, from our study.

In agreement with Zamora et al. 2007 (20), Zhang et al. 2009 (21), McAlister et al. 2004 (22), and Hillege et al. 2003 (23), we found that AKI among ischemic patients was associated with combined clinical, laboratory, and echocardiographic evidence of worse events, most notably the development and/or worsening of heart failure documented by elevated levels of natriuretic peptides and worsening of ejection However, the VALIANT Echo research contradicted our findings by claiming that renal impairment was not connected with systolic dysfunction but was linked to increase in total heart wall thickness, which contributed to poor outcomes among those patients.

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