RESEARCH ARTICLE

Clinical characteristics and in-hospital outcome in percutaneous coronary interventions with ST elevation myocardial infarction patients developing acute kidney injury

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Abstract

Objective: To find predictors, incidence and hospital mortality of acute kidney injury in ST elevation myocardial infarction patients undergoing percutaneous coronary interventions.

Methods: The retrospective cross-sectional study was conducted at Tabba Heart Institute Karachi, and comprised data from June 2013 to December 2017 of ST elevation myocardial infarction patients undergoing percutaneous coronary interventions during index admission. Acute kidney injury was defined as serum creatinine ≥0.3 mg/dl 48hrs after percutaneous coronary intervention, and was further graded into stages I-III and the need for haemodialysis. Predicted acute kidney injury risks were calculated using Mehran and National Cardiovascular Data Registry risk scores. Stata 14 was used for statistical analysis.

Results: Of the 2766 cases evaluated, the incidence of acute kidney injury was found in 543(19.6%) case. Diabetes, pre-percutaneous coronary intervention heart failure, ejection fraction <40%, post-percutaneous coronary intervention thrombolysis in myocardial infarction flow<3, glomerular filtration rate<60 ml/min and increased contrast volume were significant predictors of acute kidney injury. Hospital mortality was reported in 144(5.2%) cases.

Conclusion: Acute kidney injury is a serious complication in ST elevation myocardial infarction patients undergoing percutaneous coronary interventions and is related to adverse hospital outcomes. Pre-procedural risk scores may underestimate acute kidney injury in such patients.

Keywords: Acute kidney injury, ST elevation Myocardial infarction, Percutaneous coronary intervention, Mortality.

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Introduction

Acute kidney injury (AKI) is a recognised complication after myocardial infarction (MI).[1] Despite a declining trend, 16.1% patients in the National Cardiovascular Data Registry (NCDR) Acute Coronary Treatment and Interventions Outcomes Network (ACTION) registry develop AKI.[2,3] Percutaneous coronary intervention (PCI) itself increases risk of AKI due to the nephrotoxic effect of iodinated contrast media, potential for athero-embolism and haemodynamic changes. In Harmonising Outcomes with Revascularisation and Stents in Acute Myocardial Infarction (HORIZONS-AMI trial), 16% of acute STEMI patients undergoing PCI developed AKI.[4]

Multiple definitions have been proposed to identify patients as developing AKI. The most commonly used definition is the one by the acute kidney injury network (AKIN) and used by NCDR; an abrupt (within 48 hours) reduction in kidney function with an absolute increase in serum creatinine of ≥0.3 mg/dl.[5] Patho-physiology behind AKI in STEMI patients is multi-factorial. Use of iodinated contrast media during PCI is one of the factors,[6] while many other proposed mechanisms include haemodynamic changes, change in volume status and reduced left ventricular ejection fraction (LVEF), inflammatory response leading to increase renal oxidative stress, bleeding and use of multiple medication.[7] These factors may lead to alterations in renal blood flow or direct nephrotoxic effect leading to acute renal compromise.

Major predictors of AKI are volume of contrast used during PCI, age, pre-existing renal dysfunction, involvement of left anterior descending (LAD) artery, cardiogenic shock (CS) and heart failure (HF). AKI, compared to no AKI, is linked to a worse short- and long-term prognosis in STEMI patients with a very high 30-day (11.7% vs. 3.9%) and almost double (34% vs. 19.8%) 3-year risk of major adverse cardiac events (MACE).[4] Due to this unfavourable prognosis of AKI, it is crucial to be able to identify patients at a higher risk of developing AKI, and to take preventive measures such as intravenous hydration and judicious use of contrast. There are many suggested

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risk scores; the most notable of which is NCDR AKI risk prediction model based on pre-PCI parameters.[8] Another prediction model frequently utilised is the one described by Mehran et al.[9] These clinical risk scores have been validated in many studies and also correlate with MACE and mortality.

A recent study from Pakistan reported AKI incidence to be 20.7% in patients admitted with MI, and had association with higher risk of major bleeding and HF. But the impact of PCI, predictors of AKI and other prognostic outcomes such as mortality were not explored.[10] The current study was planned to describe the incidence of AKI in acute STEMI patients undergoing PCI, its predictors and effect of AKI on hospital mortality.

Patients and Methods

The retrospective cross-sectional study was conducted at Tabba Heart Institute, Karachi, which is a single-specialty cardiac care hospital. Data on all consecutive patients admitted with STEMI and undergoing PCI during index admission from June 2013 to December 2017 was obtained from the catheterisation (Cath) laboratory and acute coronary syndrome (ACS) database maintained according to NCDR, ACTION and Cath-PCI registry definitions. The two registries collect data on patient characteristics, clinical presentation, treatments and outcomes. There is a comprehensive data quality programme, including both data quality report specifications for data capture, transmission and an auditing programme. The complete definitions of all variables were prospectively defined by a committee of the American College of Cardiology (ACC).[11] The study questionnaire was designed in line with the registry questions that included data on all STEMI patients, their characteristics, clinical presentation such as Killip class (to predict risk of 30-day mortality), treatments, and outcomes. Patients undergoing multiple PCI procedures within the same admission, already on haemodialysis (HD), and missing initial or peak creatinine values were excluded.

Serum creatinine was measured at the time of admission and then daily thereafter. AKI was defined by AKIN criteria[5] (stage 1; $\geq$0.3 mg/dl absolute or 1.5 to 2-fold relative increase in serum creatinine; stage 2:>2-3-fold increase; stage 3: >3-fold or serum creatinine >4mg/dl with an acute increase of >0.5mg/dl). AKI requiring dialysis (AKI-D) was an in-hospital outcome identified using a pre-defined NCDR data element for acute or worsening renal failure necessitating new renal dialysis. Glomerular filtration rate (GFR) was calculated using Cockroft Gault equation [(140-age) $\times$ weight $\div$ S creatinine $\times$ 0.72 $\times$ (0.85 if female)].[12] STEMI or equivalents were characterised by the presence of both criteria: a. symptoms suggestive of acute coronary ischemia, and, b. ECG evidence of STEMI. New or presumed new ST-segment elevation or new left bundle branch block (LBBB) were measured with cut-off points: $\geq$0.2 mV in men or $\geq$0.15mV in women in leads V2-V3, and/or $\geq$0.1mV in other leads, or true posterior infarcts. LBBB refers to new or presumed new LBBB on the initial ECG. Detailed demographics, clinical presentation characteristics, in-hospital non-invasive and invasive evaluation, medical management, and revascularisation by PCI and in-hospital outcome were recorded. Approval was obtained from the institutional ethics review committee. On admission, patients were thoroughly explained with respect to storage of individual information and its use for research purposes while ensuring confidentiality of their identity and informed consent had been taken from all patients.

Mehran score for contrast induced nephropathy (CIN) related AKI was utilised to calculate predicted risk of AKI [9]. The score assigns points for 8 risk factors: hypotension, use of intra-aortic balloon pump (IABP), congestive heart failure (CHF), age >75 years, anaemia (haematocrit <39% for men and <36% for women), diabetes mellitus (DM, contrast volume, and serum creatinine or chronic kidney disease (CKD). Patients were stratified into low-risk ($\leq$5 points, AKI 7.5%), moderate-risk (6-10 points, AKI 14%), high-risk (11-15 points, AKI 26.1%), and very high-risk ($\geq$16 points, AKI 57.3%) groups. NCDR risk score for developing AKI or need of HD after PCI was also used to compare observed and predicted incidence rate, which scores on the basis of; age, HF history within two weeks, mild, moderate and severe stages of estimated GFR (eGFR), diabetes, history of HF and cardiovascular disease, STEMI/Non-STEMI (NSTEMI) at presentation, presence of CS and cardiac arrest, anaemia and use of IABP.[8]

Stata 14 was used for statistical analysis. Means and standard deviations were calculated for continuous variables and frequencies with percentages were calculated for categorical variables, such as gender, co-morbids i.e. diabetes, dyslipidaemia and hypertension (HTN), smoking history, EF, PCI indication and status and post-procedural complications. Chi-square or Fisher exact tests were used for categorical variables according to expected cell counts, and independent sample t-test for continuous variables depending upon variable characteristics. Incidence of AKI was reported as incidence ratio. To compare continuous variables, association across grades of AKI, one-way analysis of variance (ANOVA) or Kruskal Wallis test was used depending upon normality.

Hospital mortality was compared between AKI and non-AKI groups using chi square. Multivariable logistic regression was used to identify predictors of AKI and in-hospital mortality after PCI. Variables with p<0.25 in univariate analysis were considered significant for adjusting into multivariable model and p<0.05 was considered significant for final model.

Results

A total of 2766 patients had been admitted with STEMI and subsequently underwent PCI. Mean age was 56.3±11.3 years, and 2339(84.5%) of them were men. Diabetes was present in 1014 (36.7%) and 1422 (51.4%) had HTN. CS was present in 166 (5.9%) and 2418 (87.4%) had Killip class I/II on arrival. Moderate or severe renal dysfunction (GFR<60 ml/min) was present in 694(25%) patients (Table-1).

Overall, mean LVEF was 40.6%±8.9. A majority 2296 (83.1%) of the patients had 1 or 2 vessel disease. Post-PCI thrombolysis in myocardial infarction (TIMI) 3 flow was achieved in 2736(98.6%) patients. (Table-2). Mean hospital stay was 4±2.28 days.

Incidence of patients developing AKI after PCI was 543(19.6%) and new need for HD occurred in 13(0.46%). Incidence of AKI and new need for HD was higher compared to the predicted risk of AKI and need for HD based on Mehran risk score (mean risk score for CIN: 4.83±3.8; predicted risk of category <5 for AKI is 7.5% and for new HD is 0.04%) and NCDR risk (mean NCDR risk scores for AKI and AKI-D: 24.7±9.8 and 3.0±2.3; predicted risk: 6.7 to 9.2% for AKI and 0.09-0.15% for AKI-D). There was a significant trend in the presence of diabetes, hypertension, pre-PCICS and

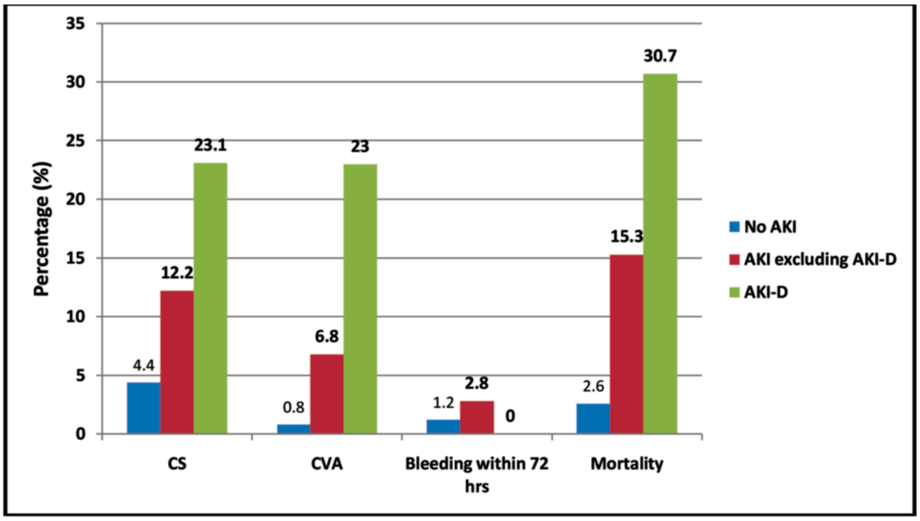

Figure: Distribution of post-Percutaneous coronary interventions (PCI) complications and mortality in ST elevation myocardial infarction (STEMI) patients (n=2766).

Table-1: Baseline clinical characteristics and presentation of ST elevation myocardial infarction (STEMI) patients undergoing percutaneous coronary interventions (PCI) according to acute kidney injury (AKI) severity (n=2766).

Characteristics	Total (N=2766)	No AKI (n=2223)	AKI I (n=320)	AKI II (n=146)	AKI III (n=64)	AKI-D (n=13)	p-value
Age in years	56.3±11.3	55.3±11.1	60.6±11.5	60.8±10.9	61.2±10.9	57.5±8.8	<0.001
Male gender	2339 (84.5)	1901(85.5)	253 (79.0)	120 (82.2)	54(84.4)	11 (84.6	0.04
Diabetes mellitus	1014 (36.7)	769 (34.6)	129 (40.3)	71 (48.6)	37 (57.8)	8 (61.5)	<0.001
Dyslipidaemia	457 (16.5)	381 (17.1)	43 (13.4)	22 (15.0)	11 (17.2)	0 (0.0)	0.25
Hypertension	1422 (51.4)	1106(49.7)	179 (55.9)	89 (60.9)	41 (64.0)	7 (53.8)	0.005
Family history of premature CAD	598 (21.5)	499 (22.4)	66 (20.6)	27 (18.5)	5 (7.8)	1 (7.7)	0.02
Previous heart failure	21 (0.7)	19 (0.8)	2 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)	0.89
Smoking history	815 (29.4)	697 (31.3)	71 (22.2)	30 (20.5)	14 (21.8)	3 (23.0)	<0.001
Median GFR ml/min	80.1 (41.8)	81.4 (41.3)	68.4 (39.8)	84.5 (50.0)	89.7 (41.1)	77.6(15.5)	<0.001
Mean GFR ml/min	82.6±33.7	83.4±32.5	69.8±30.5	93.1±43.3	94.0±32.8	73.9±25.6	<0.001
GFR<60 ml/min	694 (25.0)	523 (23.5)	130 (40.6)	29 (19.8)	10 (15.6)	2 (15.4)	<0.001
Initial creatinine mg/dl	1.15±0.96	1.16±0.7	1.33±1.0	1.00±0.5	0.87±0.2	1.36±1.02	0.003
Killip class I/II	2418 (87.4)	2026(91.2)	259 (80.9)	94 (64.4)	35 (54.7)	4 (30.8)	<0.001
Pre PCI cardiogenic shock	166 (5.9)	82 (3.7)	30 (9.4)	31 (21.2)	16 (25.0)	7 (53.8)	<0.001
Systolic BP<100mm/Hg	207 (7.5)	174 (7.8)	22 (6.9)	10 (6.8)	1 (1.5)	0 (0.0)	0.29
Cardiac arrest at arrival	160 (5.7)	105 (4.7)	21 (6.5)	20 (13.7)	9 (14.0)	5 (38.5)	<0.001
Pre PCI heart failure	279 (10.0)	159 (7.1)	51 (15.9)	40 (27.4)	24 (37.5)	5 (38.4)	<0.001
Mean haematocrit %	42.1±5.5	42.3±5.3	41.8±6.1	41.5±6.0	40.4±6.6	37.1±7.3	0.004

CAD: Coronary artery disease; GFR: Glomerular filtration rate; BP: blood pressure.

Table-2: Procedural characteristics and post procedural outcomes in ST elevation myocardial infarction (STEMI) patients undergoing percutaneous coronary interventions (PCI) according to acute kidney injury (AKI).

Characteristics	Total (N=2766)	No AKI (n=2223)	AKI I (n=320)	AKI II (n=146)	AKI III (n=64)	AKI-D (n=13)	p-value
Median EF	40 (10)	40 (10)	35 (15)	35 (15)	35 (17.5)	25 (10)	<0.001
EF <40%	1029 (37.2)	720 (32.4)	184 (57.5)	73 (50.0)	42 (65.6)	10 (76.9)	<0.001
Access site							
Femoral	230 (8.4)	149 (6.7)	41 (12.8)	18 (12.3)	18 (28.2)	4 (30.7)	<0.001
Radial	2536 (91.6)	2074 (93.3)	279 (87.2)	128 (87.7)	46 (71.8)	9 (69.3)	
Coronary disease pattern							
Left main or 3VCAD	470 (16.9)	346 (15.6)	64 (20.0)	37 (25.4)	18 (28.2)	5 (38.5)	0.003
1 or 2 VCAD	2296 (83.1)	1877 (84.4)	256 (80.0)	109 (74.6)	46 (71.8)	8 (61.5)	
PCI indication							
Immediate PCI	1537 (55.6)	1192 (53.6)	204 (63.7)	93 (63.7)	41 (64.0)	7 (53.8)	<0.001
Unstable STEMI>12hrs	295 (10.7)	232 (10.4)	36 (11.2)	19 (13.1)	7 (10.9)	1 (7.7)	
Stable STEMI>12hrs	86 (3.2)	71 (3.2)	9 (2.8)	3 (2.0)	3 (4.7)	0 (0.0)	
Successful thrombolysis	699 (25.2)	625 (28.2)	46 (14.4)	21 (14.4)	6 (9.5)	1 (7.7)	
Failed thrombolysis	149 (5.3)	103 (4.6)	25 (7.9)	10 (6.8)	7 (10.9)	4 (30.8)	
PCI status							
Urgent	898 (32.5)	798 (35.9)	62 (19.4)	26 (17.8)	10 (15.6)	2 (15.4)	<0.001
Emergency	1868 (67.5)	1425 (64.1)	258 (80.6)	120 (82.2)	54 (84.4)	11 (84.6)	
Multi-vessel PCI	467 (16.8)	374 (16.8)	54 (16.8)	23 (15.7)	14 (21.8)	2 (15.3)	0.86
Post PCI TIMI 3flow	2736 (98.9)	2210 (99.4)	311 (91.2)	141 (96.6)	61 (95.3)	13(100.0)	<0.001
Contrast volume (ml)	130 (74)	130 (72)	140 (91)	120 (70)	140 (80)	114 (70)	0.02
IABP use	10 (0.3)	4 (0.2)	1 (0.3)	2 (1.4)	2 (3.1)	1 (7.7)	0.001
Post procedural complications							
MI	24 (0.9)	15 (0.7)	8 (2.5)	1 (0.7)	0 (0.0)	0(0.0)	0.01
CS	57 (2.0)	18 (0.8)	18 (5.6)	10 (12.5)	8 (12.5)	3 (23.1)	<0.001
HF	57 (2.0)	19 (0.9)	20 (6.2)	11 (7.5)	5 (7.8)	2 (15.4)	<0.001
Stroke	15 (0.5)	6 (0.3)	2 (0.6)	5 (3.4)	1 (1.5)	1 (7.7)	<0.001
Bleeding within 72hrs	41 (1.5)	26 (1.2)	6 (1.9)	5 (3.4)	4 (6.3)	0 (0.0)	0.03
In-hospital mortality	144 (5.2)	59 (2.6)	24 (7.5)	32 (21.9)	25 (39.0)	4 (30.7)	<0.001

EF: ejection fraction; SK: streptokinase; TIMI; Thrombolysis In Myocardial Infarction; IABP; intra-aortic balloon pump; MI; myocardial infarction; CS: cardiogenic shock; HF: heart failure; CAD: coronary artery disease.

Table-3: Multivariable model for predictors of acute kidney injury.

Variables	Univariate				Multivariable			
	Odds ratio	p-value	95% CI		Odds ratio	p-value	95% CI	
			Lower	Upper			Lower	Upper
Diabetes mellitus	1.54	<0.001	1.25	1.86	1.46	0.001	1.20	1.79
Pre PCI heart failure	3.71	<0.001	2.85	4.83	2.84	<0.001	2.17	3.74
EF<40%	3.13	<0.001	2.56	3.82	2.49	<0.001	2.05	3.49
Post PCI TIMI flow <3	4.66	<0.001	2.26	9.61	4.18	0.002	1.92	9.10
GFR<60 ml/min	1.13	0.002	1.04	1.22	1.40	<0.001	1.12	1.74
Contrast volume (ml)	1.00	0.03	1.000	1.002	1.00	0.02	1.000	1.003

CI: Confidence interval; GFR: Glomerular filtration rate; PCI: Percutaneous coronary interventions; TIMI: Thrombolysis In Myocardial Infarction.

HF, cardiac arrest at arrival and decreasing haematocrit % across AKI grades (p<0.05 each).

EF <40%, left main or multi-vessel disease (MVD) and emergency PCI status showed a statistically significant worsening distribution across AKI grades (p<0.05). Femoral access site for PCI was more common among higher grades of AKI (p<0,05). Occurrences of post-PCI complications i.e. CS, HF, stroke and major bleeding were also higher with worsening AKI grades (p<0.001 each). Mortality was significantly higher in patients developing AKI compared to non-AKI and with an increasing pattern across AKI grades (p<0.001). On multivariable adjustment, diabetes, pre-PCI HF, EF<40%, post-PCI TIMI flow <3, GFR<60ml/min and unit increase in contrast volume in ml were found to be significant predictors of AKI (Table-3).

Table-4: Multivariable model for predictors of mortality.

Variables	Univariate				Multivariable			
	Odds ratio	p-value	95% CI		Odds ratio	p-value	95% CI	
			Lower	Upper			Lower	Upper
Acute kidney injury	6.80	<0.001	4.81	9.63	5.01	<0.001	3.36	7.47
Diabetes Mellitus	1.96	<0.001	1.40	2.75	1.59	0.02	1.08	2.36
Cardiac arrest at arrival	17.8	<0.001	12.12	26.25	8.52	<0.001	5.24	13.84
Post PCI TIMI flow <3	15.12	<0.001	7.19	31.79	7.25	<0.001	3.02	17.37
Pre PCI cardiogenic shock	10.90	<0.001	6.16	19.28	5.13	<0.001	3.20	8.24

CI: Confidence interval; PCI: Percutaneous coronary interventions; TIMI: Thrombolysis In Myocardial Infarction.

Overall in-hospital mortality was 144 (5.2%) patients. Mortality was significantly higher in AKI groups compared to non-AKI (85[15.6%] vs. 59[2.6%]). Mortality was even higher in AKI-D subgroup 04(30.7%). Also, the deceased patients were more diabetic, had worse GFR, had more frequent cardiac arrest and had more HF (p<0.05 each). Deceased patients also had higher predisposition to left main or MVD, more likely to go for emergency PCI, and less likely to have post-procedure TIMI 3 flow (p<0.05). Other complications, including bleeding, CS, stroke and HF, were also higher among AKI and AKI-D compared to non-AKI (Figure).

AKI remained a strong independent predictor of mortality in multivariable adjustment. Other independent predictors of mortality were diabetes, cardiac arrest at arrival, post-PCI TIMI flow<3 and post PCI-CS (Table-4).

Discussion

The findings showed that around 20% of STEMI PCIs were complicated by AKI. Major predictors of AKI were pre-PCIHF, EF<40%, post-PCI TIMI flow <3, diabetes and baseline GFR <60ml/min and increased volume of contrast. AKI is related to adverse in-hospital outcomes in terms of post-PCIHF, shock and bleeding complications. AKI is an independent predictor of hospital mortality with around five times odds of hospital death in AKI compared to non-AKI. Patients with worse baseline clinical and procedural parameters tend to develop higher grades of AKI, and also severity of AKI correlates with worse adverse hospital events, including mortality.

In the current data, predicted AKI risk, according to both NCDR and Mehran risk scores, was lower than the actual incidence of AKI. There might be several explanations to it. One might be that Mehran risk score is relatively simpler and may not encompass the potential of additional risk of renal complications in STEMI[9] related to haemodynamic instability leading to renal and cardiac blood flow disturbance, inflammatory mechanisms and relative lack of use of preventive strategies. In most STEMI patients undergoing emergent procedures, there is no ample time for intravenous (IV) hydration and quite a few patients may even be ineligible for IV fluids. Also, baseline GFR and coronary anatomy is unknown leading to less control over volume of contrast. Although the NCDR AKI risk score is more comprehensive, it is based on predictors which include only pre-procedural parameters; the logic behind it related to the fact that predictive ability is utilised before taking the patient for PCI. In general, AKI risk is around 7% in all-cause PCI according to NCDR data findings.[13] However, STEMI patients may have significant procedural and post-procedural aspects that possibly lead to adverse outcomes higher than in routine PCI. STEMI have more post-PCI <3 TIMI flow, presence of CS or HF leading to higher AKI and AKI-D.[14] More than two-thirds of patients in NCDR model development cohort were without MI, thus STEMI was overall under-represented.[8] This suggests potential of development of separate risk prediction scores for STEMI that include post-procedural parameters as well.

In a study related to our population, HORIZONS-AMI trial reported AKI incidence in STEMI of 16.1%[4] which was slightly lower than our findings. Reasons for this disparity might be related to more patients with underlying CKD (23 vs. 16%), CS (6% vs. <1%) and HF (10% vs. 3%) in our data compared to the trial patients.[4] It is seen that with CS complicating STEMI, the incidence of AKI in STEMI may become as high as >50%.[15] Major predictors of AKI in the current study are also similar to the trial data. Although contrast volume is considered the major contributor to AKI and was found to be a significant predictor in HORIZONS-AMI as well as our data, the weak odds ratio (OR) points to the fact that there are other factors at play too.[16] A recent study comprising more than 3000 STEMI patients compared primary PCI and a thrombolytic-only control group for AKI development showed equal incidence of AKI both in primary PCI and control STEMI (10.3 vs. 12.1%), suggesting alternative mechanisms are also at play. Major predictors related to underlying renal dysfunction, age, diabetes, anterior infarction, LV systolic dysfunction and diuretics use plus haemodynamic instability and use of IABP.[17] Thus, many a time labelling all post-PCI AKI in STEMI as contrast-induced may not be justifiable.

AKI is one of the significant predictors of post-PCI adverse events, hospital mortality and late outcomes in terms of progression to CKD, haemodialysis and mortality.[18,19] Our study also demonstrated five times higher risk of death in the presence of AKI even after adjusting for additional worse prognostic factors. Multiple studies have shown variable results with short-term mortality ranging from three times than non-AKI to even 50%[10] in the presence of concomitant CS.[1,15,20] Another important finding is gradation of risk of adverse outcomes across stages of AKI as defined by NCDR risk score in our study that correlates with prior NCDR risk score validation studies both in US and in Japan.[13,21]

There is no clear consensus on preventive strategies despite numerous trials on agents like N-acetyl cysteine, soda bicarbonate, statins and renal ischaemic preconditioning.[22-25] The only two proven strategies are judicious use of contrast and adequate volume status to maintain renal perfusion, and they remain valid.[26]

To our knowledge, this is the first comprehensive study from the region on incidence of AKI in STEMI using current definitions for AKI and application of prediction scores. Limitations include retrospective single-centre data with no long-term outcomes.

Conclusion

A significant number of STEMI patients suffered complications in the presence of AKI. Predictors included the use of IV contrast, but a patient's clinical and haemodynamic status also had a central role. While there is no current consensus on targeted therapies for prevention of AKI in STEMI, the need is to ensure maintenance of haemodynamics and judicious contrast use. Devising separate risk scores for high-risk STEMI population should be considered.

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