ORIGINAL RESEARCH

Acute Kidney Injury Definition and In-Hospital Mortality in Patients Undergoing Primary Percutaneous Coronary Intervention for ST-Segment Elevation Myocardial Infarction

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***Background*—**Acute kidney injury (AKI) has been associated with increased mortality in ST-segment elevation myocardial infarction. We compared the mortality predictive accuracy of the 3 AKI definitions used most widely for patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention.

***Methods and Results*—**We included 3771 patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention at 2 Italian hospitals. AKI incidence was evaluated according to creatinine increases of ≥25% (AKI-25), ≥0.3 mg/dL (AKI-0.3), and ≥0.5 mg/dL (AKI-0.5). The primary end point was in-hospital mortality. Overall, 557 (15%), 522 (14%), and 270 (7%) patients developed AKI-25, AKI-0.3, and AKI-0.5, respectively ($P<0.01$). All AKI definitions independently predicted in-hospital mortality (adjusted odds ratio 4.9 [95% CI 3.1–7.8], 5.4 [95% CI 3.3–8.6], and 8.3 [95% CI 5.1–13.3], respectively; $P<0.01$ for all). At receiver operating characteristic analysis, the addition of each AKI definition to combined clinical predictors of mortality (age, sex, left ventricular ejection fraction, admission creatinine, creatine kinase-MB peak) found at stepwise analysis significantly improved mortality prognostication (area under the curve increased from 0.89 for clinical predictor combination alone to 0.92 for AKI-25, 0.92 for AKI-0.3, and 0.93 for AKI-0.5; $P<0.01$ for all). At reclassification analysis, AKI-0.5 added to clinical predictors, provided the highest score in mortality (net reclassification improvement +10% versus AKI-0.3 [$P=0.01$] and +8% versus AKI-25 [$P=0.05$]).

***Conclusions*—**Each AKI definition significantly improved the mortality prediction beyond major clinical variables. AKI-0.5 showed a mortality discrimination advantage, suggesting it should be the preferred definition in studies addressing ST-segment elevation myocardial infarction and focusing on short-term mortality. **(*J Am Heart Assoc*. 2016;5:e003522 doi: 10.1161/JAHA.116.003522)**

Key Words: acute kidney injury • serum creatinine concentration

Patients undergoing primary percutaneous coronary intervention (pPCI) for ST-segment elevation myocardial infarction (STEMI) are at high risk of acute kidney injury (AKI), a complication that is associated with a striking increase in mortality.[1–9] The AKI definitions that are most widely used are relative (≥25%) and absolute (≥0.5 mg/dL) increases in serum creatinine (sCr) concentration occurring in the first 48 to 72 hours after hospital admission.[10,11] Moreover, a lower threshold of absolute sCr increase (≥0.3 mg/dL) was proposed recently to define AKI in patients with both cardiovascular and noncardiovascular diseases.[12,13] Although these definitions were shown to be closely associated with increased in-hospital mortality in STEMI patients undergoing pPCI,[1–10] no study has prospectively compared them in terms of prognostic accuracy in a large cohort of patients; therefore, it is still unclear which definition may provide the best risk prognostication for STEMI patients. Notably, the relationship between sCr and glomerular filtration rate (GFR) is not linear.[14,15] In patients with normal baseline sCr, even a small absolute increase in sCr implies a significant GFR reduction. In contrast, in patients with high baseline sCr, a similar absolute increase of sCr is associated with a smaller reduction in GFR. When relative changes in sCr are

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considered, a similar percentage reduction in GFR is foreseeable, regardless of sCr baseline value.[16] Consequently, prognostic implications may differ according to the sCr threshold chosen to define AKI.

The aim of this study was to compare the performance of the most widely used AKI definitions in predicting in-hospital mortality in a large cohort of STEMI patients undergoing pPCI. Moreover, we investigated whether these definitions have any additional value for improving the predictive accuracy of the main clinical variables associated with mortality in this setting and which AKI definitions performed better.

Methods

Study Population

The data analyzed in this study were obtained from prospectively enrolled STEMI patients who underwent pPCI at Centro Cardiologico Monzino in Milan, Italy, between January 1, 2002, and August 1, 2015, and at Policlinico San Matteo in Pavia, Italy, between January 1, 2005, and April 30, 2014. In general, patients underwent pPCI if they had typical chest pain that began within the previous 12 hours (24 hours for those with cardiogenic shock) and at least 1-mm ST-segment elevation in ≥2 contiguous leads or a new left bundle-branch block. We excluded patients in chronic peritoneal or hemodialysis treatment, those undergoing emergency cardiac surgery, and those who died during pPCI or before at least 2 consecutive sCr values were collected. The institutional review boards of both centers approved the study, and all patients gave written informed consent to the use of their clinical data for research purposes.

Study Design

According to the clinical protocols of the 2 hospitals, sCr values were measured in all patients by the Jaffe method at hospital admission (before pPCI) and each day for the following 72 hours. The total coefficients of variation for sCr determinations were not >3%.

The incidence of AKI was evaluated in the first 72 hours according to the following definitions: (1) a relative sCr increase ≥25% from hospital admission value (AKI-25); (2) an absolute sCr increase ≥0.3 mg/dL (AKI-0.3); (3) an absolute sCr increase ≥0.5 mg/dL (AKI-0.5).

Echocardiogram was performed in all patients within 24 hours of hospital admission, and left ventricular ejection fraction was calculated by the Simpson rule.[17]

The primary end point of the study was the occurrence of in-hospital mortality. A composite clinical end point of death and cardiogenic shock was considered as a secondary end point. Other in-hospital major adverse clinical events (atrial fibrillation, acute pulmonary edema, blood transfusions, ventricular fibrillation, and ventricular tachycardia) were also evaluated.

PCI Procedure

The pPCI procedure was performed by 24-hour on-call interventional teams according to standard clinical practice. Standard guide catheters (6F or 7F), guide wires, balloon catheters, and coronary stents were used with a radial or femoral approach. Periprocedural pharmacological therapy and poststenting antithrombotic treatment were given according to institutional protocols and guideline recommendations.

Statistical Analysis

Continuous variables are presented as mean±SD and were compared using the *t* test for independent samples. Variables not normally distributed are presented as median and interquartile ranges and were compared with the Wilcoxon rank sum test. Categorical data were compared using the chi-square or Fisher exact test, as appropriate.

A stepwise multivariable logistic model was developed to identify the independent predictors of the primary end point. Among the clinical variables found to be associated with AKI occurrence independent of the definition used (age, sex, weight, diabetes, hypertension, hyperlipidemia, smoking, creatine kinase-MB peak, left ventricular ejection fraction, admission sCr, GFR, anterior myocardial infarction location, prior myocardial infarction, prior coronary artery bypass grafting, and culprit lesion), the following remained independently associated with in-hospital mortality: age, sex, creatine kinase-MB peak, left ventricular ejection fraction, and admission sCr. Collinearity among the variables included in the model was tested by the variance inflation factor. The association between each AKI definition and in-hospital mortality was assessed by logistic regression; analyses were also adjusted for the combination of the above-mentioned clinical variables. Results are presented as odds ratios (ORs) with 95% CIs. Receiver operating characteristic curves were calculated, and the area under the receiver operating characteristic curve (AUC) with 95% CI was used to measure the ability of the model to predict in-hospital mortality. AUCs were compared as recommended by DeLong et al.[18]

Net reclassification improvement was used to identify the best definition of AKI for the prediction of primary and secondary end points when added to the combination of clinical variables selected at the stepwise analysis. All tests were 2-tailed, and $P<0.05$ was required for statistical significance. All analyses were performed using SAS version 9.4 (SAS Institute). Reclassification statistics were assessed with the SAS macros published by Cook and Ridker.[19]

Table 1. Clinical and Laboratory Characteristics of the Study Patients According to AKI Definition

	AKI-25			AKI-0.3			AKI-0.5		
	No (n=3214)	Yes (n=557)	*P* Value	No (n=3249)	Yes (n=522)	*P* Value	No (n=3501)	Yes (n=270)	*P* Value
Age, y	62±12	69±12	<0.001	62±12	70±12	<0.001	63±12	72±11	<0.001
Men, n (%)	2557 (80)	393 (71)	<0.001	2569 (79)	381 (73)	0.002	2763 (79)	187 (69)	<0.001
Weight, kg	77±15	74±13	<0.001	77±14	74±13	<0.001	76±15	73±12	<0.001
Diabetes mellitus, n (%)	487 (15)	109 (20)	<0.001	485 (15)	111 (21)	<0.001	533 (15)	63 (23)	<0.001
Hypertension, n (%)	1636 (51)	341 (61)	<0.001	1634 (50)	343 (66)	0.002	1793 (51)	184 (68)	<0.001
Hyperlipidemia, n (%)	1366 (42)	181 (32)	<0.001	1379 (42)	168 (32)	<0.001	1467 (42)	80 (30)	<0.001
Smoking, n (%)	1988 (62)	264 (47)	<0.001	2026 (62)	226 (43)	<0.001	2146 (61)	106 (39)	<0.001
Anterior MI, n (%)	1470 (46)	316 (57)	<0.001	1486 (46)	300 (57)	<0.001	1619 (46)	167 (62)	<0.001
Index PCI vessel, n (%)			<0.001			<0.001			<0.001
LAD	1458 (45)	303 (54)		1473 (45)	288 (55)		1607 (46)	154 (57)	
RCA	1160 (36)	160 (29)		1171 (36)	149 (29)		1251 (36)	69 (26)	
LCx	541 (17)	78 (14)		550 (17)	69 (13)		586 (17)	33 (12)	
Bypass graft	36 (1)	4 (1)		36 (1)	4 (1)		37 (1)	3 (1)	
LM	19 (1)	12 (2)		19 (1)	12 (2)		20 (1)	11 (4)	
Prior MI, n (%)	446 (14)	93 (17)	0.08	443 (14)	96 (18)	0.004	475 (14)	64 (24)	<0.001
Prior CABG, n (%)	111 (3)	20 (4)	0.87	109 (3)	22 (4)	0.32	116 (3)	15 (6)	0.05
LVEF, %	48±10	41±12	<0.001	48±10	41±12	<0.001	48±10	38±12	<0.001
sCr, mg/dL	1 (0.9–1.1)	1 (0.8–1.3)	0.07*	1 (0.8–1.1)	1.1 (0.9–1.4)	<0.001*	1 (0.8–1.1)	1.3 (1.0–1.6)	<0.001*
sCr peak, mg/dL	1 (0.9–1.2)	1.5 (1.1–2.1)	<0.001*	1 (0.9–1.2)	1.6 (1.3–2.3)	<0.001*	1 (0.9–1.2)	2.2 (1.7–3.2)	<0.001*
eGFR, mL/min per 1.73 m^2	79±22	76±32	0.003	79±22	68±29	<0.001	80±23	58±25	<0.001
CK-MB peak, ng/mL	143 (59–278)	235 (103–409)	<0.001*	140 (58–270)	240 (107–420)	<0.001*	148 (61–283)	260 (107–444)	<0.001*

AKI indicates acute kidney injury; AKI-0.3, absolute serum creatinine increase ≥0.3 mg/dL; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; AKI-25, relative serum creatinine increase ≥25%; CABG, coronary artery bypass grafting; CK-MB, creatine kinase-MB isoenzyme; eGFR, estimated glomerular filtration rate (abbreviated MDRD equation); LAD, left anterior descending; LCx, left circumflex; LM, left main; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; RCA, right coronary artery; sCr, serum creatinine.
*By nonparametric Wilcoxon rank sum test.

Results

A total of 3771 STEMI patients treated with pPCI were included in this study. Of them, 557 (15%) developed AKI-25, 522 (14%) developed AKI-0.3, and 270 (7%) developed AKI-0.5 (P<0.001 among the 3 definitions).

The clinical and laboratory characteristics of patients with or without AKI, according to the 3 different definitions, are listed in Table 1. Regardless of the definition used, patients with AKI were older and were more likely to have diabetes mellitus, larger infarct size, anterior STEMI, and worse renal function at hospital admission than patients without AKI.

Patients with AKI had a more complicated clinical course and longer hospitalization (Table 2). In-hospital mortality among our study participants was 4.1% (n=155). Figure 1 shows unadjusted and adjusted ORs for in-hospital mortality using the 3 AKI definitions.

For the entire population, sensitivity and specificity for in-hospital mortality were 55% (95% CI 48–63%) and 87% (95% CI 86–88%), respectively, for AKI-25; 58% (95% CI 50–66%) and 88% (95% CI 87–89%), respectively, for AKI-0.3; and 50% (95% CI 42–58%) and 95% (95% CI 94–96%), respectively, for AKI-0.5.

When the combination of independent predictors of mortality found at multivariable stepwise analysis was evaluated in terms of accuracy to predict in-hospital mortality in the entire population, the AUC was 0.89 (95% CI 0.87–0.92). Figure 2 shows the AUCs for in-hospital mortality prediction when each AKI definition was added to the combined clinical variables. The addition of AKI with any of the 3 definitions significantly improved in-hospital mortality prognostication (P<0.01 for all comparisons). When the AUCs of the AKI definitions in addition to clinical mortality predictors were compared, the AUC associated with AKI-0.5 was significantly higher than those of AKI-25 and AKI-0.3 (P<0.001 and

Table 2. In-Hospital Outcomes According to AKI Definition

	AKI-25			AKI-0.3			AKI-0.5		
	No (n=3214)	Yes (n=557)	*P* Value	No (n=3249)	Yes (n=522)	*P* Value	No (n=3501)	Yes (n=270)	*P* Value
Death, n (%)	69 (2)	86 (15)	<0.001	65 (2)	90 (17)	<0.001	77 (2)	78 (29)	<0.001
APE, n (%)	204 (6)	108 (19)	<0.001	200 (6)	112 (21)	<0.001	231 (7)	81 (30)	<0.001
CS, n (%)	226 (7)	133 (24)	0.008	221 (7)	138 (26)	0.0002	253 (7)	106 (39)	<0.001
AF, n (%)	302 (9)	113 (20)	<0.001	298 (9)	117 (22)	<0.001	338 (10)	77 (29)	<0.001
VT/VF, n (%)	337 (10)	85 (15)	0.001	336 (10)	86 (16)	<0.001	364 (10)	58 (21)	<0.001
Blood transfusions, n (%)	82 (3)	55 (10)	<0.001	84 (3)	53 (10)	<0.001	91 (3)	46 (17)	<0.001
CCU LOS, days	4 (3–5)	5 (4–8)	<0.001*	4 (3–5)	5 (4–8)	<0.001*	4 (3–5)	6 (4–10)	<0.001*
Secondary end point†, n (%)	242 (8)	148 (27)	<0.001	236 (7)	154 (29)	<0.001	271 (8)	119 (44)	<0.001

AF indicates atrial fibrillation; AKI, acute kidney injury; AKI-0.3, absolute serum creatinine increase ≥0.3 mg/dL; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; AKI-25, relative serum creatinine increase ≥25%; APE, acute pulmonary edema; CCU, coronary care unit; CS, cardiogenic shock; LOS, length of stay; VF, ventricular fibrillation; VT, ventricular tachycardia.
*By nonparametric Wilcoxon rank sum test.
†In-hospital death and cardiogenic shock requiring intra-aortic balloon pump.

P=0.03, respectively), without significant difference between AKI-25 and AKI-0.3 (*P*=0.26). No difference in the AUC was observed for the combination of AKI-25 or AKI-0.5 versus AKI-0.5 alone when both were added to the clinical model (*P*=0.69). At reclassification analysis, AKI-0.5 plus clinical predictors provided the highest score in predicting mortality (Table 3).

When the combination of independent predictors of mortality was evaluated in terms of accuracy to predict the secondary end point in the entire population, the AUC was 0.84 (95% CI 0.81–0.86). At reclassification analysis, the combination of AKI-0.5 and clinical predictors again provided the highest score (Table 3).

Discussion

The main finding of this study was that the AKI-0.5 definition performed better for in-hospital mortality prognostication in STEMI patients treated with pPCI than AKI-25 and AKI-0.3,

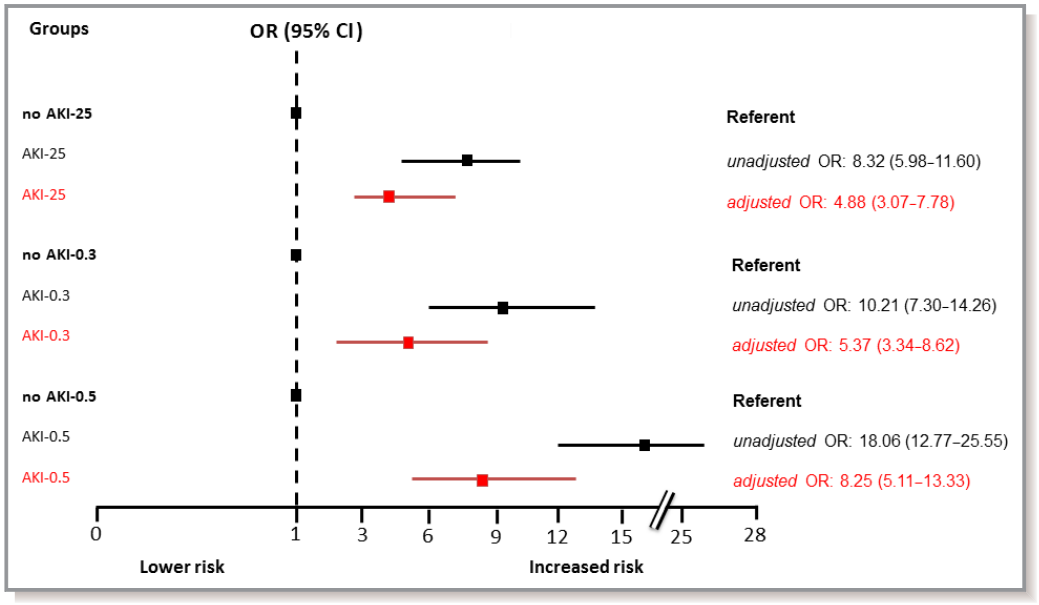

Figure 1. In-hospital mortality ORs and 95% CIs according to the 3 acute kidney injury definitions (AKI-25, AKI-0.3, and AKI-0.5). Reported ORs are unadjusted and adjusted for age, sex, left ventricular ejection fraction, creatine kinase-MB peak, and admission serum creatinine. AKI-0.3 indicates absolute serum creatinine increase ≥0.3 mg/dL; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; AKI-25, relative serum creatinine increase ≥25%; OR, odds ratio.

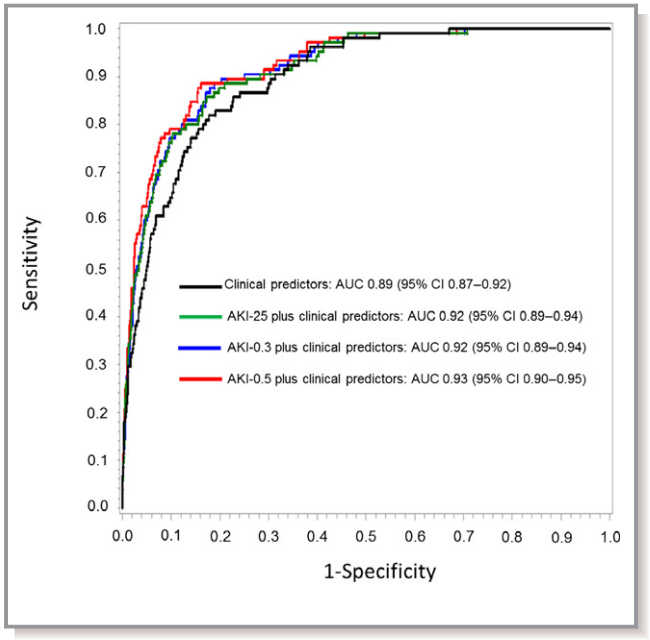

Figure 2. Receiving operating characteristic curves and corresponding AUCs with 95% CIs for in-hospital mortality prediction in the study patients, using a combination of prognostic clinical variables alone and each acute kidney injury definition added to clinical variables. The clinical variables that independently predicted in-hospital mortality at stepwise analysis were age, sex, left ventricular ejection fraction, creatine kinase-MB peak, and admission serum creatinine. AKI-0.3 indicates absolute serum creatinine increase ≥0.3 mg/dL; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; AKI-25, relative serum creatinine increase ≥25%; AUC, area under the curve.

even when added to clinical variables known to have critical impact on prognosis.

Patients treated with pPCI represent a population at higher risk for AKI for which renal prophylactic therapy, particularly hydration, is unfeasible.[1–9] To date, several AKI definitions based on absolute and/or relative sCr increase have been shown to be useful in predicting clinical outcome in STEMI[1–9]; however, no study prospectively compared them in terms of prognostic accuracy. To our knowledge, only 2 studies investigated the incidence and associated mortality of 2 AKI definitions in patients with STEMI undergoing pPCI.[2,20] Nonetheless, the Risk, Injury, Failure, Loss, and End-stage kidney disease (RIFLE) classification was 1 of the 2 definitions used in both studies. RIFLE takes into account a ≥50% increase in sCr, a criterion that has been investigated less extensively in this specific clinical setting.[16] Moreover, because of a retrospective design,[2] a small patient population,[20] and lack of mortality discrimination assessment,[20] no definite conclusion can be drawn about which AKI definition should be preferred for mortality risk prediction in STEMI patients.

In this study, we included a large population of STEMI patients undergoing pPCI at 2 Italian centers, and we prospectively compared the prognostic role of the AKI definitions that are most widely used.[10,13] In agreement with previous studies, we found that AKI incidence ranged between 7% and 15% and was associated with 7- to 15-fold higher in-hospital mortality based on the definition used, along with a more complicated clinical course and a longer stay in the coronary care unit.[7–9,16] Notably, all 3 AKI definitions were strongly associated with poor prognosis in our study population. Regardless of the definition used, it is important to know whether AKI occurrence provides prognostic information beyond that carried by well-known independent clinical predictors. The combination of variables identified by our stepwise analysis accurately predicted mortality, with an AUC of 0.89. This finding is not surprising because all of those variables, either alone or in combination, were strongly associated with patient outcome in this clinical setting.[7,21–23] Nevertheless, the addition of each AKI definition to those

Table 3. Reclassification Analysis Comparisons in the Prediction of Primary and Secondary End Points of the 3 AKI Definitions Added to the Clinical Variables* Associated With In-Hospital Mortality

Model	NRI %	95% CI	*P* Value
Primary end point			
AKI-0.5 plus independent predictors of mortality vs AKI-25 plus independent predictors of mortality	8.1	0.3–16.4	0.05
AKI-0.5 plus independent predictors of mortality vs AKI-0.3 plus independent predictors of mortality	10.0	2.0–18.0	0.01
AKI-25 plus independent predictors of mortality vs AKI-0.3 plus independent predictors of mortality	1.9	−6.0 to 2.6	0.41
Secondary end point			
AKI-0.5 plus independent predictors of mortality vs AKI-25 plus independent predictors of mortality	28.3	20.2–36.5	<0.001
AKI-0.5 plus independent predictors of mortality vs AKI-0.3 plus independent predictors of mortality	29.5	21.3–37.7	<0.001
AKI-25 plus independent predictors of mortality* vs AKI-0.3 plus independent predictors of mortality	28.5	20.3–36.7	<0.001

AKI indicates acute kidney injury, AKI-0.3, absolute serum creatinine increase ≥0.3 mg/dL; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; AKI-25, relative serum creatinine increase ≥25%; NRI, net reclassification improvement.
*The following clinical variables, found at multivariate stepwise analysis, were considered: age, sex, left ventricular ejection fraction, admission serum creatinine, creatine kinase-MB peak.

variables further improved mortality prognostication. Moreover, when the 3 AUCs were compared, AKI-0.5 was associated with the highest improvement in mortality prediction accuracy. At reclassification analysis, AKI-0.5 provided the highest score in mortality risk, confirming its prognostic superiority over the other 2 AKI definitions. In our study, the net reclassification improvement of AKI-0.5 added to clinical predictors of mortality was >8% and >28% for primary and secondary end points, respectively, compared with both AKI-0.25 and AKI-0.3.

Our findings agree with previous studies that investigated the prognostic impact of absolute (≥0.5 mg/dL) and relative (≥25% mg/dL) sCr increases among patients undergoing elective or emergency PCI.[24–26] Notably, all of these studies showed that an absolute increase of ≥0.5 mg/dL was a better predictor of short- and long-term outcomes than a ≥25% increase. This result further supports the notion that AKI-0.5 should be the preferred definition for mortality prediction in this setting, despite its lower expected sensitivity.

The reason why our study and previous ones suggested that AKI-0.5 is a better indicator of mortality risk cannot be inferred from our findings; however, we can suggest the following hypotheses. Considering the nonlinear relationship between sCr and GFR, an identical relative sCr increase is associated with the same GFR percentage reduction independent of the baseline sCr value.[16] Conversely, an identical absolute sCr increase is associated with a progressively lower percentage reduction of GFR as baseline sCr value increases. Consequently, an absolute cutoff takes into account both the baseline number of functioning nephrons at hospital admission and the loss of nephrons due to AKI, and it appears to be associated more strongly with mortality compared with a variable reflecting the acute GFR decline only (Figure 3). Indeed, the use of the AKI-0.5 definition is associated with a significant GFR reduction in healthy kidneys and with a small GFR decrease in vulnerable and already compromised kidneys. In both cases, the diagnosis of AKI using the AKI-0.5 definition translates into a striking mortality increase, suggesting a relatively constant association of AKI-0.5 on mortality regardless of baseline sCr. Overall, the AKI-0.5 definition for mortality prediction seems generalizable to STEMI patients with varying degrees of baseline renal function because it provides the highest score even when adjusted for admission sCr.

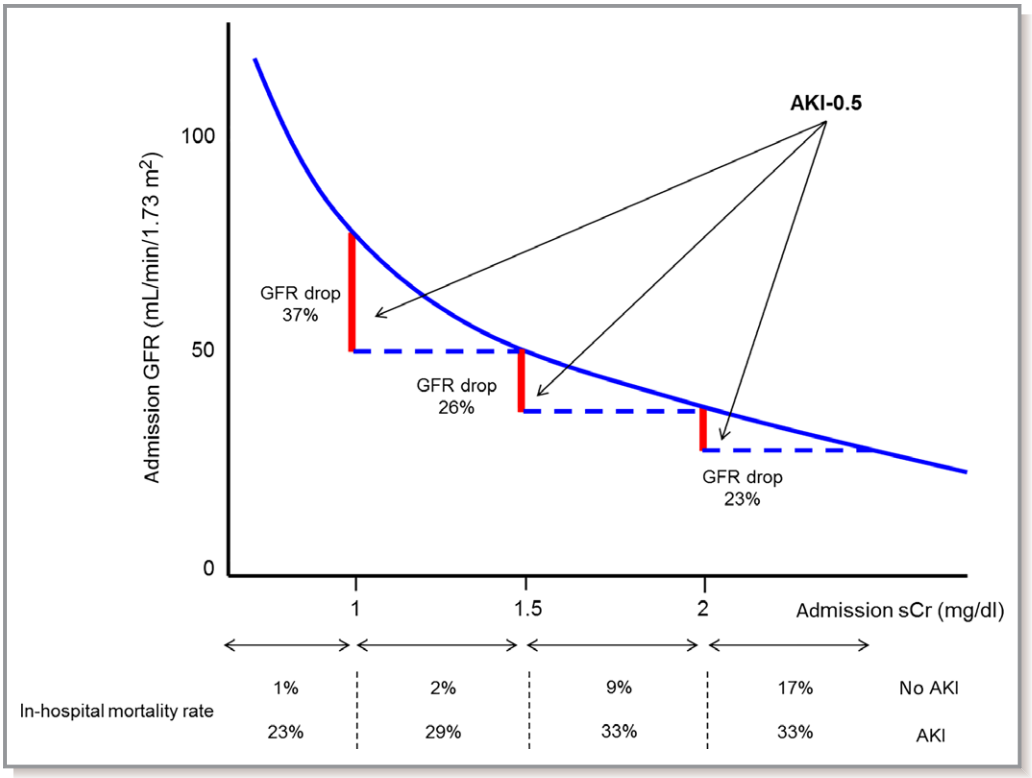

Figure 3. Schematic representation of the nonlinear relationship between sCr and GFR (blue line) measured at admission. When the AKI-0.5 definition was used, a progressively smaller GFR reduction (red line) was observed in parallel with the increase in sCr measured at hospital admission. The mortality rates observed in our study are reported according to admission sCr (allocated in 4 groups) and to AKI occurrence. GFR drop was calculated by considering the mean age of our population (63 years). AKI indicates acute kidney injury; AKI-0.5, absolute serum creatinine increase ≥0.5 mg/dL; GFR, glomerular filtration rate; sCr, serum creatinine.

Some clinical implications can be inferred from our study. First, because of the important prognostic relevance of AKI, its occurrence should always be looked for and diagnosed after pPCI, independent of the definition used. Second, the use of the AKI-0.5 definition allows better prediction of mortality risk in this setting and, after the first 48 to 72 hours, identification of very high-risk patients in whom additional therapeutic strategies should be applied. Finally, the potential impact of novel preventive and therapeutic approaches should be evaluated and compared among studies, also taking into account the incidence of AKI by using a standardized AKI-0.5 definition among the well-established hard end points. Nevertheless, our findings warrant prospective validation in larger studies.

A number of potential limitations to this study must be addressed. First, exclusion of patients who died before having 2 consecutive sCr determinations reduced the overall mortality rate in our population. Second, sCr measured at hospital admission cannot be considered a true baseline value in STEMI patients because an increase may have already occurred before hospital admission. Third, we considered only the AKI definitions that are used most frequently, thus we cannot exclude the possibility that different absolute and/or relative thresholds of sCr value may better assess mortality in these patients. Fourth, we evaluated in-hospital outcomes only; however, previous studies assessing the long-term outcomes of STEMI patients found that the greatest mortality difference between those with and without AKI[1–3] was observed during hospitalization. This was shown to be true even when the criterion of 0.5 mg/dL sCr increase was considered.[27] Although increasing evidence suggests a detrimental role for AKI in chronic kidney disease development and progression,[16] we focused on the early STEMI phase.

Conclusions

Regardless of definition, AKI development has an incremental predictive value beyond well-known clinical variables associated with in-hospital mortality in STEMI patients undergoing pPCI. A mortality prognostication advantage is provided by the AKI-0.5 definition, suggesting that this definition should be preferred in clinical and interventional studies addressing STEMI patients and focusing on short-term mortality.

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Disclosures

None.

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