Original Research

# Comparison of the Performance of the NCDR CathPCI Score and Mehran Score to Predict Acute Kidney Injury Post-Percutaneous Coronary Intervention

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## Abstract

### Background
A comparison of acute kindney injury (AKI) post-percutaneous coronary intervention (PCI) prediction models is lacking. In this study, we aim to compare the National Cardiovascular Data Registry (NCDR) CathPCI score to the Mehran score in acute coronary syndrome (ACS) vs non-ACS patients.

### Methods
We included patients who received PCI at our facility between July 2015 and December 2017. We excluded patients without a pre- and/or post-PCI serum creatinine, patients on dialysis at the time of PCI and patients with missing variables required to calculate the predictive scoring model. The primary outcome of this study was AKI post-PCI. Performance of the NCDR CathPCI score and the Mehran score were evaluated by comparing the area under the receiver-operating characteristic curve (AUROC) for both scores.

### Results
The analysis included 1,507 patients. In non-ACS patients, the Mehran score performed better than the NCDR CathPCI score with AUROC 0.75 and 0.68 respectively (p=0.014). When categorized into 4 risk groups, a Mehran score ≥ 2 had a sensitivity of 86% and a Mehran score of ≥ 3 had a specificity of 83% in non-ACS patients. In contrast, when the NCDR CathPCI score was categorized into risk groups, it was not able to predict the risk of AKI (p=0.78) with sensitivity of 0% for the intermediate and high risk group. In ACS patients, the NCDR CathPCI score was superior in predicting the risk for AKI with AUROC 0.79 versus 0.74 (p=.019).

### Conclusion
In predicting AKI post-PCI, the NCDR CathPCI score performed better in ACS populations, and the Mehran score performed better in the non-ACS population.

### Keywords
cardiac catheterization; contrast media/averse effects; acute kidney injury; coronary artery disease; myocardial infarction; percutaneous coronary intervention

## Introduction
Contrast-induced acute kidney injury (AKI) is a common complication in patients undergoing percutaneous coronary intervention (PCI) and is known to be associated with increased mortality rates, worsening of chronic kidney disease (CKD), prolonged hospital stays and significantly higher healthcare costs.[1-3] Even though the risk of contrast-induced AKI is relatively low in the general population, it is significantly augmented in patients undergoing diagnostic procedures for comorbid conditions such as coronary artery disease (CAD). A recent literature review revealed that of all the hospitalized

patients who develop AKI, 12% to 14% had a procedure involving radiographic contrast.[4,5]

Multiple prediction models have been developed to predict the risk of AKI after PCI. However, a direct comparison of the commonly used models is lacking. In addition, the differential performance of these scores in patients presenting with acute coronary syndrome (ACS) versus those with non-acute presentation has not been evaluated. Of all the post-PCI AKI prediction models, the NCDR CathPCI model has been validated by the largest research study to date.[6-17] It was also validated internationally and demonstrated good discrimination in the Japanese population.[18] Another well validated model is the one developed by Mehran et al. The Mehran score is one of the earliest derived scores and is the most widely used tool to predict contrast induced nephropathy (CIN) post-PCI.[19,20] Although more recent scores have been developed,[8-17] the Mehran score has superior clinical utility and usability. Recently, Abellas et al. validated the score in Europe and indicated good discrimination in five out of six subgroups (age >75 years, diabetes mellitus (DM), CKD, ST-elevation myocardial infarction (STEMI), Killip ≥2 and PCI), demonstrating that the Mehran score is applicable internationally and is still useful more than 10 years since it was established. Furthermore, the Mehran score was validated in predicting CIN in acute myocardial infarction (MI) patients.[20-22] (**Appendix A**)

In this study, we aim to compare the performance of the NCDR CathPCI and Mehran scoring systems in predicting AKI after PCI to determine which of the two models delivers a higher predictive value. We also sought to compare the performance of these scoring systems in patients who present with ACS versus non-ACS presentation.

# Methods

## Study Population

The NCDR CathPCI Registry is sponsored by the American College of Cardiology (ACC) and the Society for Cardiovascular Angiography and Intervention (SCAI) and has been previously described.[23,24] This registry contains data on patient demographics, clinical presentation, procedures, treatments, outcomes and mortality. It is from this registry where we obtained our data from. NCDR variable definitions can be found at the ACC NCDR web site (http://www.acc.org/ncdr/cathlab.htm).

For this study, we identified patients who received PCI at our institution between July 2015 and December 2017 (n=2,020). Excluded from the study were patients without a pre- and/or post-PCI serum creatinine (n=411, 20%), patients on dialysis at the time of PCI (n=35, 1.7%) and patients with missing variables required to calculate the predictive scoring models (n=67, 3.3%). The final cohort included 1,507 patients.

## Definitions

The primary outcome tracked in this study was AKI post-PCI using the change in creatinine from pre-procedure level to peak level after the procedure. AKI is defined using the Acute Kidney Injury Network (AKIN) definition as an absolute increase in serum creatinine ≥0.3 mg/dl and a ≥1.5-fold increase in serum creatinine from baseline.[25] Urine output was not collected in the NCDR CathPCI Registry and was not used as a measure for AKI in this study. Baseline glomerular filtration rate (GFR) was used to categorize patients as normal, mild CKD, moderate CKD, or severe CKD. GFR in mL/min per 1.73 m2 was calculated using the Modification of Diet in Renal Disease (MDRD) formula[26] as 175 x serum creatinine - 1.154 x age - 0.203 x 1.212 (if patient is black) x 0.742 (if patient is female). We used the World Health Organization (WHO) criteria for anemia: baseline hemoglobin <13 g/dl for males and <12 g/dl for females.[27] Hypotension in this study was defined as a systolic blood pressure less than 90 mm Hg for more than 30 minutes and/or cardiac index below 2.2 L/min/$m^2$ secondary to cardiac dysfunction and/or requirement of parenteral inotropes/vasopressors or mechanical support including intra-aortic balloon pump (IABP), extracorporeal circulation, ventricular assist devices to maintain blood pressure and cardiac index above the aforementioned levels. The timing of IABP (before PCI, during PCI or after PCI) and status (elective versus emergent) were not available and peri-procedure IABP was used instead.

### Calculations of NCDR CathPCI Score and the Mehran Score

The NCDR CathPCI score incorporates 11 variables: age, chronic kidney disease (CKD), prior cerebrovascular disease (CVD), prior heart failure (HF), CAD presentation (non-ACS and ACS [unstable angina/non-ST-segment elevation, ST-segment elevation]), diabetes mellitus (DM), hypertension (HTN), cardiac arrest on presentation, anemia, IABP and cardiogenic shock. The Mehran score incorporates eight variables: hypotension, IABP, HF, age >75 years, anemia, DM, contrast volume and renal dysfunction.[6] (**Tables 1 and 2**)

### Statistical Analysis

The baseline characteristics and outcomes were summarized by frequency tabulation and means with standard deviations as appropriate. A student's t-test and chi-square test were used to compare baseline characteristics between ACS and non-ACS patients. The discriminative ability of the scoring systems for predicting outcomes were evaluated by the receiver-operator characteristic curve analysis.

The area under the receiver-operating characteristic curve (AUROC) was calculated and compared for both scores using the DeLong test.[28] The CathPCI score was categorized to 3 risk categories (low risk <30 points, intermediate risk 30–37 and high risk >37 points) based on the original study by Tsai et al.[29] The Mehran score was categorized into 4 risk groups (low risk <6 points, moderate risk 6–10 points, high risk 11–15 points and very high risk >15) based on the original study by Mehran et al.[6] Estimates of sensitivity, specificity, positive and negative predictive values were calculated for each score. The comparison between risk groups for each score was performed using the chi square test. All statistical comparisons were two-tailed with value <0.05 considered statistically significant. The data analysis was performed by using STATA version 13.0 (StataCorp, College Station, TX).

## Results

Of the 1,507 patients included in the study, 1,020 (67.77%) presented with ACS (the ACS group); the rest did not present with ACS (the non-ACS group). A total of 70 (4.64%) patients developed AKI post-PCI, 63 (6.18 %) in the ACS group and only 7 patients (1.44%) in the non-ACS group. Baseline characteristics, treatments and outcomes are further outlined in **Table 1**.

### Performance of CathPCI Score and Mehran Score in the Non-ACS Group

In patients who did not present with ACS, the Mehran score performed better than the NCDR CathPCI score with AUROC 0.75 vs 0.68 respectively (p=0.014) (**Figures 1 and 2**). When categorized into 4 risk groups as described in the Methods section, the Mehran score ≥2 had a sensitivity of 86% and a Mehran score of ≥3 had a specificity of 83% in non-ACS patients. In contrast, when the NCDR CathPCI score was categorized into risk groups, as described in the Methods section, it was not able to predict the risk of AKI (p=0.78) with sensitivity of 0% for the intermediate and high risk group (i.e., none of the AKI patients were predicted based on the risk category). (**Tables 2 and 3**)

### Performance of CathPCI Score and Mehran Score in the ACS Group

In patients who presented with ACS, the NCDR CathPCI score was superior to the Mehran score in predicting the risk for AKI with AUROC 0.79 vs 0.74 (p=0.019). (**Figure 2**) When categorized into risk groups, both models were predictive of AKI risk (p<0.001 for both models). A Mehran score of ≥6 had 85% sensitivity and 48% specificity. (**Table 2**) A CathPCI score of ≥30 had a sensitivity of 52% and a specificity of 87%. (**Table 3**)

## Discussion

In this study, we compared the performance of the NCDR CathPCI score with the Mehran score in predicting AKI post-PCI in 2 different populations. The Mehran score was superior in predicting AKI in the non-ACS group, whereas the NCDR CathPCI score was superior in the ACS group.

This study is the first one to compare the CathPCI score and the Mehran score in the same population. The incidence of AKI in our population was 4.6%, which is lower than the incidence in the CathPCI (7.33%) and Mehran

**Table 1.** Patient characteristics in the ACS vs non-ACS groups.

| | Non-ACS (n=487) | ACS (n=1020) | p-Value |
|---|---|---|---|
| **DEMOGRAPHICS** | | | |
| **Age (years) – mean ± SD** | **71.59 ± 9.20** | **68.16 ± 11.78** | **<0.05** |
| Gender, male – no.% | 311 (63.86%) | 613 (59.86%) | 0.136 |
| White – no.% | 458 (94.05%) | 954 (93.16%) | 0.518 |
| Black – no.% | 29 (5.95%) | 62 (6.05%) | 0.939 |
| **HISTORY & MEDICAL CONDITIONS** | | | |
| **Smoker – no.%** | **78 (16.02%)** | **282 (27.54%)** | **<0.05** |
| **Hypertension – no.%** | **471 (96.71%)** | **926 (90.43%)** | **<0.05** |
| **Dyslipidemia – no.%** | **449 (92.20%)** | **853 (83.30%)** | **<0.05** |
| Prior Myocardial Infarction – no.% | 178 (36.55%) | 342 (33.40%) | 0.228 |
| **Prior Heart Failure – no.%** | **127 (26.08%)** | **209 (20.41%)** | **<0.05** |
| **Prior PCI – no.%** | **259 (53.18%)** | **476 (46.48%)** | **<0.05** |
| Prior CABG – no.% | 115 (23.61%) | 218 (21.29%) | 0.308 |
| **Cerebrovascular Disease – no.%** | **134 (27.52%)** | **212 (20.70%)** | **<0.05** |
| **Peripheral Artery Disease – no.%** | **161 (33.06%)** | **192 (18.75%)** | **<0.05** |
| Diabetes Mellitus – no.% | 219 (44.97%) | 443 (43.26%) | 0.532 |
| **CLINICAL EVALUATION** | | | |
| **Anginal Class within 2 weeks** | | | **<0.05** |
| **No symptoms – no.%** | **20 (4.11%)** | **1 (0.10%)** | |
| **CCSI – no.%** | **13 (2.67%)** | **0** | |
| **CCSII – no.%** | **75 (15.40%)** | **8 (0.78%)** | |
| **CCSIII – no.%** | **379 (77.82%)** | **281 (27.47%)** | |
| **CCS IV – no.%** | **0** | **733 (71.65%)** | |
| **Heart Failure within 2 weeks – no.%** | **35 (7.19%)** | **148 (14.45%)** | **<0.05** |
| **NYHA w/in 2 weeks – no.%** | | | **<0.05** |
| **Class I – no.%** | **7 (20.00%)** | **80 (54.05%)** | |
| **Class II – no.%** | **22 (62.86%)** | **26 (17.57%)** | |
| **Class III – no.%** | **6 (17.14%)** | **38 (25.68%)** | |
| **Class IV – no.%** | **0** | **4 (2.70%)** | |
| Cardiogenic Shock w/in 24 hours – no.% | 1 (0.21%) | 11 (1.07%) | 0.075 |
| Cardiac Arrest within 24 hours – no.% | 2 (0.41%) | 15 (1.46%) | 0.069 |
| **PCI PROCEDURE** | | | |
| **Diagnostic caths w/ PCI – no.%** | **283 (58.11%)** | **923 (90.14%)** | **<0.05** |
| Contrast Volume (ml) – mean ± SD | 166.69 ± 84.82 | 168.42 ± 84.79 | 0.645 |
| **IABP – no.%** | **0** | **15 (1.46%)** | **<0.05** |
| **PCI Status** | | | **<0.05** |

PCI = percutaneous coronary intervention; CABG = coronary artery bypass grafting; CCS = Canadian Cardiovascular Society classification; NYHA = New York Heart Association Classification; IABP = intra-aortic balloon pump; LVEF = left ventricular ejection fraction; CKD = chronic kidney disease

**Table 1.** Con't.

| | Non-ACS (n=487) | ACS (n=1020) | p-Value |
|---|---|---|---|
| **Elective – no.%** | **470 (96.51%)** | **147 (14.36%)** | |
| **Urgent – no.%** | **15 (3.08%)** | **705 (68.85%)** | |
| **Emergent – no.%** | **2 (0.41%)** | **171 (16.70%)** | |
| **Salvage – no.%** | **0** | **1 (0.10%)** | |
| **Pre-PCI LVEF – mean ± SD** | **51.42 ± 12.43** | **47.81 ± 14.92** | **<0.05** |
| **PRE-PROCEDURE LABS** | | | |
| **Hemoglobin (g/dl) – mean ± SD** | **13.36 ± 1.65** | **13.02 ± 2.24** | **<0.05** |
| Creatinine (ng/ml) – mean ± SD | 1.02 ±0.29 | 1.05 ± 1.41 | <0.66 |
| **POST-PROCEDURE EVENT** | | | |
| **Cardiogenic shock – no.%** | **2 (0.41%)** | **18 (1.76%)** | **<0.05** |
| Heart Failure – no.% | 5 (1.03%) | 12 (1.17%) | 0.803 |
| Cerebrovascular Disease – no.% | 1 (0.21%) | 5 (0.49%) | 0.414 |
| Dialysis – no.% | 0 | 4 (0.39%) | 0.167 |
| **Bleed within 72hrs – no.%** | **3 (0.62%)** | **39 (3.81%)** | **<0.05** |
| CABG – no.% | 3 (0.62%) | 12 (1.17) | 0.308 |

PCI = percutaneous coronary intervention; CABG = coronary artery bypass grafting; CCS = Canadian Cardiovascular Society classification; NYHA = New York Heart Association Classification; IABP = intra-aortic balloon pump; LVEF = left ventricular ejection fraction; CKD = chronic kidney disease

**Table 2.** Incidence of AKI in Mehran score categories.

| | Mehran AKI Risk Score | | | | |
|---|---|---|---|---|---|
| | ≤ 5 (low) | 6-10 (moderate) | 11-16 (high) | ≥ 16 (very high) | p |
| **Non-ACS** | 1 (0.51%) | 3 (1.47%) | 1 (1.22%) | 2 (40.00%) | <0.001 |
| **ACS** | 9 (2.06%) | 23 (6.48%) | 24 (13.26%) | 7 (14.89%) | <0.001 |
| **Total** | 10 | 26 | 25 | 9 | |

AKI = acute kidney injury; ACS = acute coronary syndrome

**Table 3.** Incidence of AKI in CathPCI score categories.

| | CathPCI AKI Risk Score | | | |
|---|---|---|---|---|
| | < 30 (low) | 30-37 (intermediate) | ≥ 37 (high) | p |
| **Non-ACS** | 7 (1.49%) | 0 (0%) | 0 (0%) | 0.87 |
| **ACS** | 30 (3.46%) | 17 (19.77%) | 16 (24.24%) | <0.001 |
| **Total** | 37 | 17 | 16 | |

AKI = acute kidney injury; ACS = acute coronary syndrome

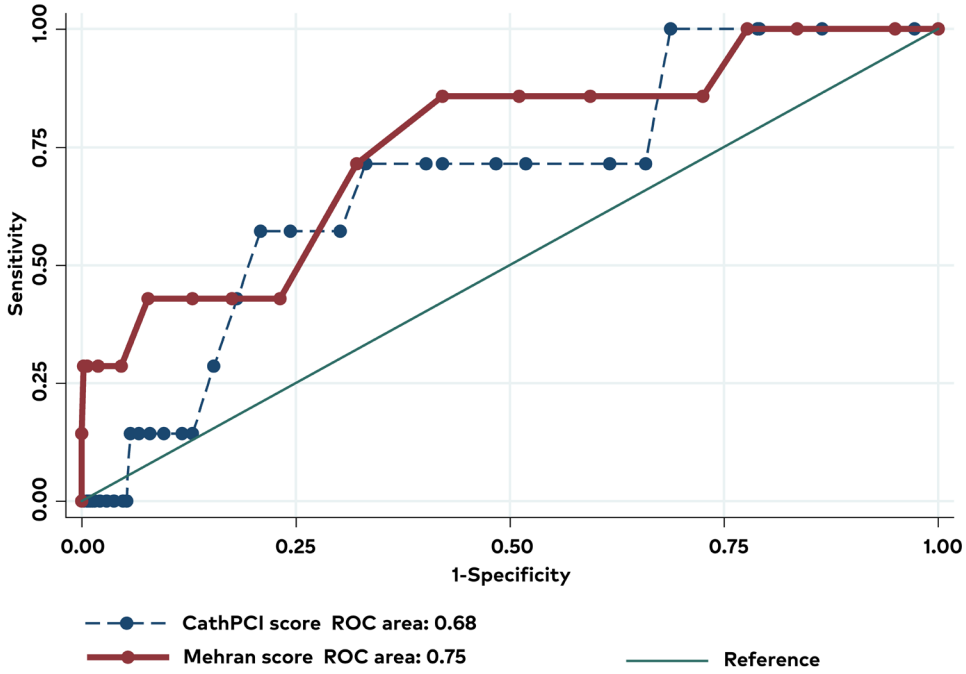


**Figure 1.** Comparison of the NCDR AKI model to the Mehran score in non-acute coronary syndrome group.

(13.1%) original studies, indicating that our population is probably a lower risk population than the ones included in the original studies by Tsai et al. and Mehran et al.[29] The difference in the definitions of AKI Network criteria and CIN likely contributes to the wide difference in the incidence of AKI between our study and Mehran's. We used the Acute Kidney Injury Network definition for AKI, which has been adopted by the broader medical community as a standard definition.[25]

Our study is also the first to evaluate the performance of both of these predictive models in ACS and non-ACS populations. In our study, the CathPCI score was superior to the Mehran score in ACS patients. However, the CathPCI did not perform as well in the non-ACS population. Several factors may contribute to this difference. First, the mechanism of AKI in patients with ACS is not limited to CIN. AKI in ACS patients is multifactorial and is currently believed to be a type of cardiorenal syndrome.[29] Thus, the CathPCI score, which relies on many factors that are related to hemodynamic stability, was superior to Mehran score in the prediction of AKI. On the other hand, the Mehran score was developed originally to predict CIN, which is likely the dominant etiology of AKI in non-ACS patients. Therefore, the Mehran score fared better in predicting outcomes in non-ACS patients. Secondly, most of the patients in the CathPCI derivation cohort[23] presented with ACS (71.1%); whereas, in the Mehran cohort, ACS represented only 35.7% of the population. This outcome may explain the superiority of the Mehran score in the non-ACS subgroup and the NCDR CathPCI score in the ACS subgroup. (**Figures 1 and 2**)

This study has obvious practical clinical implications. The identification of the best prediction model for different populations helps improve the accuracy of the prediction and, thus, identifies high risk patients who otherwise would have been undetected. For example, the CathPCI did not predict any of the patients with AKI in the non-ACS group.

## Study Limitations

Firstly, this study is retrospective. However, the data was collected prospectively by trained staff according to predefined criteria. The results were based on data from a single institution with a relatively small sample size. In addition, the registry lacks blood pressure measurements, and hypotension was considered when patients developed cardiogenic shock, as defined in the Methods section, within 24 hours of presentation and/or IABP was required for

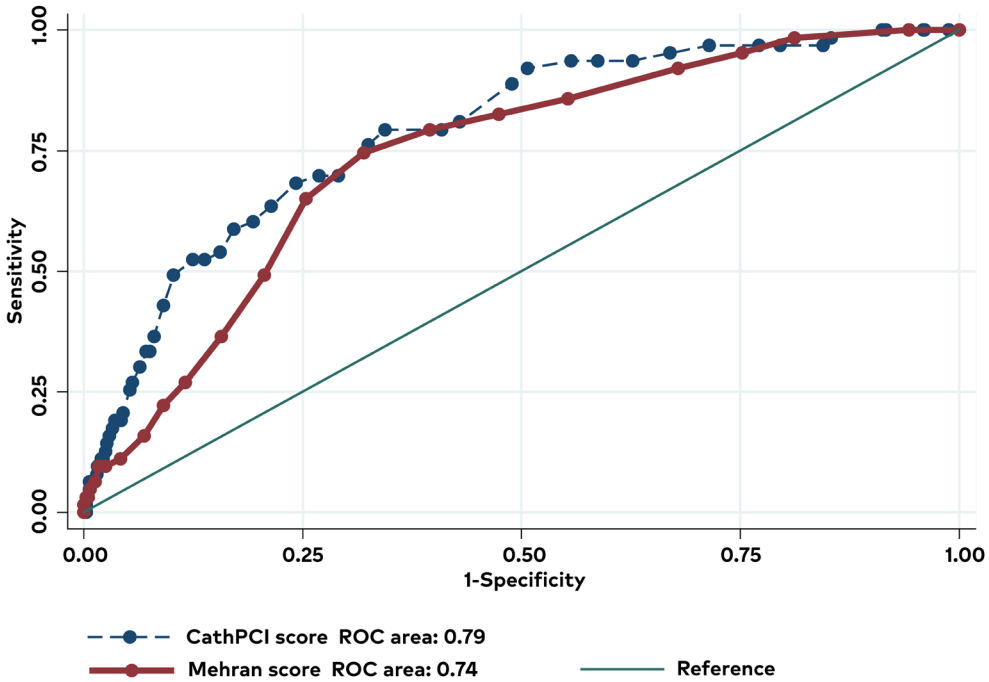


**Figure 2.** Comparison of the NCDR AKI model to the Mehran score in acute coronary syndrome group.

blood pressure support. Nevertheless, we believe these slight variations did not affect the validity of the calculation of the scores.

## Conclusion

NCDR CathPCI score performed better in the ACS population, and the Mehran score performed better in the non-ACS population in predicting AKI post-PCI. Given the aforementioned results, we suggest using the NCDR CathPCI score to predict AKI in the ACS patients, and the Mehran score for non-ACS patients. We can better predict AKI post-PCI using the appropriate score for the specific clinical presentation.

### Conflicts of Interest

The authors declare they have no conflicts of interest.

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# Appendix A

## Predictive Scoring Systems for Predicting Acute Kidney Injury

**Table A1.** Mehran Score for calculating risk of AKI.

| Risk Factors | Integer Score |
|---|---|
| Hypotension | 5 |
| IABP | 5 |
| CHF | 5 |
| Age > 75 years | 4 |
| Anemia | 3 |
| Diabetes | 3 |
| Contrast media volume | 1 for each 100 $cc^3$ |
| Serum creatinine > 1.5 mg/dl<br>OR<br>eGFR < 60 ml/min/1.73 $m^3$ | 4<br>2 for 40–60<br>4 for 20–40<br>6 for <20 |

**Table A2.** CathPCI AKI score for calculating risk of AKI.

| Risk Factors | Integer Score |
|---|---|
| Age (years) | |
| <50 | 0 |
| 50 – 59 | 2 |
| 60 – 69 | 4 |
| 70 – 79 | 5 |
| 80 – 89 | 8 |
| >90 | 10 |
| Priors weeks HF | 11 |
| Severe GFR (<30) | 18 |
| Moderate GFR (30–45) | 8 |
| Mild GFR (45–60) | 3 |
| Diabetes | 7 |
| Prior HF | 4 |
| Prior CVD | 4 |
| NSTEMI/UA | 6 |
| STEMI | 15 |
| Prior cardiogenic shock | 16 |