HHS Public Access

Author manuscript

*Am J Emerg Med*. Author manuscript; available in PMC 2022 July 01.

Published in final edited form as:

*Am J Emerg Med*. 2021 July ; 45: 303–308. doi:10.1016/j.ajem.2020.08.074.

# The Prognostic Value of HEART score in Patients with Cocaine Associated Chest Pain: An Age-and-Sex Matched Cohort Study

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## Abstract

**Introduction:** HEART score is widely used to stratify patients with chest pain in the emergency department but has never been validated for cocaine-associated chest pain (CACP). We sought to evaluate the performance of HEART score in risk stratifying patients with CACP compared to an age- and sex-matched cohort with non-CACP.

**Methods:** The parent study was an observational cohort study that enrolled consecutive patients with chest pain. We identified patients with CACP and age/sex matched them to patients with non-CACP in 1:2 fashion. HEART score was calculated retrospectively from charts. The primary outcome was major adverse cardiac events (MACE) within 30 days of indexed encounter.

**Results:** We included 156 patients with CACP and 312 age-and sex-matched patients with non-CACP (n= 468, mean age 51±9, 22% females). There was no difference in rate of MACE between the groups (17.9% vs. 15.7%, p = 0.54). Compared to the non-CACP group, the HEART score had lower classification performance in those with CACP (AUC = 0.68 [0.56–0.80] vs. 0.84 [0.78–0.90], p = 0.022). In CACP group, Troponin score had the highest discriminatory value (AUC = 0.72 [0.60–0.85]) and Risk factors score had the lowest (AUC = 0.47 [0.34–0.59]). In patients deemed low-risk by the HEART score, those with CACP were more likely to experience MACE (14% vs. 4%, OR = 3.7 [1.3–10.7], p = 0.016).

**Conclusion:** In patients with CACP, HEART score performs poorly in stratifying risk and is not recommended as a rule out tool to identify those at low risk of MACE.

### Keywords

Cocaine; Chest Pain; HEART Score; risk stratification; triage

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Author Statement

**Ziad Faramand:** conceptualization, formal analysis, investigation, data curation, and writing original draft; **Christian Martin-Gill:** writing - review & editing, supervision, and project administration; **Stephanie O Frisch:** data curation, and writing – review & editing, **Clifton Callaway:** writing - review & editing, supervision, and project administration, **Salah Al-Zaiti:** conceptualization, methodology, formal analysis, writing Original draft, visualization, supervision, project administration, and funding acquisition

## INTRODUCTION

Cocaine is the second most used illicit substance in the US with an estimated 5.5 million users annually[1]. It has been reemerging as a major public health concern; it is one of the most common illicit drugs leading to Emergency Department (ED) visits, with more than 500,000 users presenting with cocaine-associated complaints each year[2]. Cardiovascular complaints are common among cocaine users seeking ED care, with chest pain being the single most frequent presenting symptom.[3,4] Of utmost concern, cocaine induces coronary vasospasm and perfusion mismatch, leading to myocardial injury and potential acute coronary syndrome (ACS)[3]. Thus, patients with cocaine-associated chest pain (CACP) need to be carefully evaluated during ED encounters.

The distinctive characteristics of patients with CACP makes it challenging to distinguish the likelihood of ACS from other benign etiologies. For instance, compared to general patients with chest pain, patients with CACP tend to be younger, and classical diagnostic tools, such as nature of presenting symptoms and ECG findings, provide poor predictive value for ACS in those with CACP.[4] This complicated clinical picture could be partly attributed to the fact that patients with CACP are more likely to have an underlying coronary vasoconstriction rather than an obstructive plaque.[5] Such diagnostic dilemma in patients with CACP leads to higher rates of admission, higher likelihood for invasive cardiac testing, and an increased length of stay.[5–7] However, patients with CACP have a similar rate of adverse cardiac events compared to general patients with chest pain,[8,9] which suggests that risk stratification tools need to be optimized in this specific population. Yet, TIMI score, a validated risk score for predicting adverse cardiac outcomes, has failed to provide value in patients with CACP.[10]

The HEART score is a commonly used tool to risk stratify patients presenting to the ED with chest pain.[11] The HEART score, an acronym of its components (**H**istory, **E**CG, **A**ge, **R**isk factors, and **T**roponin), has been previously validated in predicting major adverse cardiac events (MACE) in non-selected patients with chest pain. In fact, the HEART score is currently the most widely adopted decision tool to accurately identify patients with lowest rate of subsequent MACE who are eligible for early discharge.[12–15] However, HEART has never been validated in patients with CACP, which constitutes a missed opportunity to potentially address the common clinical conundrum in this population. Therefore, in this secondary analysis of a cohort study we sought to 1) evaluate the performance of the HEART score in patients with CACP compared to age- and sex-matched patients with non-CACP; and 2) investigate the incremental value of each component of the HEART score to identify potential room for improvement in triaging patients with CACP.

## METHODS

### Sample and Settings

Subjects for this sub-analysis were recruited from the ongoing EMPIRE study (*ECG Methods for the Prompt Identification of Coronary Events*). The methods of EMPIRE were described in detail elsewhere.[16] Essentially, EMPIRE is a prospective observational cohort study that enrolls consecutive, non-traumatic chest pain patients transported by EMS to one

of three University of Pittsburgh Medical Center-affiliated tertiary care hospitals (UPMC Presbyterian, Mercy, and Shadyside hospitals) in Pittsburgh, PA. The parent study enrolled around 2,400 patients between 2013 and 2018. Using this study population, we identified all patients evaluated for CACP and matched them based on sex and age to patients with non-cocaine associated chest pain in a 1:2 fashion. A 1:2 ratio has been shown to add power to detect a difference in observational studies[17]. The parent study enrolled consecutive eligible patients under a waiver of informed consent, there were no modifications to routine medical care, and the study was approved by the University of Pittsburgh Institutional Review Board.

## Data Collection

Independent reviewers manually abstracted the key in-hospital data elements from the electronic health records as recommended by the American College of Cardiology for measuring the management and outcomes of patients with ACS, including:[18] demographics, past medical history, home medications, clinical presentation and course of hospitalization, laboratory tests, imaging studies, cardiac catheterization, treatments, and in-hospital complications. To identify patients presenting with CACP, we reviewed the history of present illness in the electronic hearth records for patients who admitted using cocaine within 72 hours of presentation. In addition, we reviewed all urine toxicology screen results for the presence of cocaine and its metabolites. The urine toxicology screens were ordered based on the discretion of the treating physicians when deemed necessary. The choice of 72 hours as a cut off point for cocaine use was based on the ability for a urine toxicology screen to detect cocaine and its metabolites within <72 hours after administration.[19,20]

## Calculating HEART Score

The HEART score was calculated retrospectively by independent reviewers blinded to outcome data based on ED admission documentation. The HEART score calculation followed the original derivation method,[11] as previously described in our prior work.[15] In short, the calculation was based on the following components (Figure 1): (1) history of present illness, (2) electrocardiogram, (3) age, (4) risk factors (diabetes, smoking, hypercholesterolemia, family history of coronary artery disease, obesity, history of coronary revascularization, myocardial infarction, stroke or peripheral arterial disease), and (5) troponin assay. Each component of the risk score is assigned 0–2 points, and subsequently all components are summed up to a total score ranging from 0–10. A total score of 0–3 is considered low-risk, and subjects are eligible for early discharge without further evaluation; a total score of 4–6 is deemed intermediate-risk and requires further observation for evaluation or admission; a score of 7 or more is deemed high risk and requires an immediate intervention.

## Clinical Outcomes

The primary study outcome was major adverse cardiac events (MACE) within 30 days of indexed admission defined as a composite end point of one of the following conditions: (1) all-cause death, (2) confirmed ACS, (3) coronary revascularization, (4) postadmission subsequent pulmonary embolus, (5) cardiac arrest or fatal ventricular dysrhythmia, (6) cardiogenic shock, and (7) acute heart failure. ACS was defined as per the AHA / ACC Fourth Universal Definition as:[21] symptoms of ischemia with biomarkers,

electrocardiographic, nuclear or angiographic evidence of loss of viable myocardium. To adjudicate study outcomes, two independent reviewers were provided full access to patient index and discharge records, serial ECGs, results of cardiac diagnostic tests (e.g., imaging scans and catheterization laboratory reports), and other information pertinent to the course of hospitalization (e.g., interventions, procedures, and prescribed medications). All disagreements were resolved by a third reviewer. To ensure complete ascertainment of follow up data, we used Cerner and EPIC, the UPMC electronic health records of in-hospital and outpatient medical charts respectively, to identify all relevant subsequent medical visits within 30 days of the indexed admission. These electronic health records cover the entire UPMC healthcare network which means that we were able to access relevant follow up data even if the patient was readmitted to a different center.

### Statistical Analysis

For continuous type variables, measures of central tendency and dispersion were reported as mean ± SD for normally distributed variables and median [$25^{th}$ – $75^{th}$ IQR] for non-normally distributed variables. Categorical variables were described using frequencies and percentages and reported as n (%). Each outcome was treated as a dichotomous variable (yes/no) and HEART risk score was treated as a continuous variable in analyses. Chi-square statistics were used to compare the differences of categorical variables between groups. The classification performance to predict study outcome was evaluated using the area under the receiver-operator characteristic curve (AUC). The AUCs were compared using the Hanley and McNeil method.[22] All analyses were conducted using IBM SPSS Statistics version 26 (IBM, Corp., Armonk, NY).

## RESULTS

Our study screened 2400 chest pain patients, and subsequently included 156 (6.5%) patients with CACP and 312 age-and-sex matched patients with non-CACP. The mean age of the total study sample (n = 468) was 51 years and 22% of subjects were females. Table 1 describes the demographics and clinical characteristics of CACP patients and their non-CACP counterparts. Overall, patients with CACP were more likely to be Black and had more significant past medical history, notably, 44% of CACP patients had a history of coronary artery disease compared to 25% in non-CACP. These patients had a slightly higher HEART score compared to their counterparts (4.0±1.4 vs. 3.3±1.6),and were more likely to be risk stratified as intermediate risk (58% vs. 42%).

Overall, there was a total of 133 MACE occurring in 77 patients, including 28 out of the 156 patients with CACP (17.9%) and 49 out of the 312 patients with non-CACP (15.7%) (p = 0.54). There were also no differences between groups in term of final diagnosis of ACS (11% vs 12% respectively). However, the distribution of MACE across risk groups of the HEART score reflected a contrasting story (Figure 2). While only 4% of patients with non-CACP in the lowest risk group had an event, 14% of patients with CACP in the same lowest risk group had an event (OR = 3.7 [1.3–10.7], p = 0.016).

To further investigate the incremental value of each component of the HEART score in triaging patients, we compared the AUC of the total HEART score and each individual risk

component between those with non-CACP and CACP (Figure 3). Overall, the HEART score had a very good classification performance in predicting MACE in those with non-CACP, but performed poorly in those with CACP (AUC = 0.84 [0.78–0.90] vs 0.68 [0.56–0.80], p = 0.022). While coronary risk factors had the lowest discriminatory value in both groups, the ECG had the best incremental value in those with non-CACP, and troponin had the highest incremental value in those with CACP. In fact, troponin had a better classification performance than the total HEART score in this latter group (AUC of 0.72 vs. 0.68). To further examine these distributions, we explored the rate of MACE within each risk component of the HEART score (Figure 4). In non-CACP group, the rate of events increased correspondingly as the assigned risk points increased from 0 to 2 in all components of the HEART score. In contrast, such pattern was observed at lesser magnitude among patients with CACP. Troponin had the most conclusive increased risk of events corresponding to higher risk points. Age and risk factors reflected different but important patterns. While no events occurred in patients with CACP assigned an Age score of “2”, more than 40% of those assigned a Risk factors score of “0” had MACE events.

## DISCUSSION

In this study we sought to evaluate the performance of HEART score and its components in patients with CACP and compare that performance to age- and sex-matched patients with non-CACP. We found that while HEART score exhibits very good discriminatory accuracy in patients with non-CACP, it performs poorly in patients with CACP. Approximately 14% of those in the lowest risk group in the CACP group experienced MACE, suggesting the HEART score is an inappropriate rule out tool in this group. Examining the performance of each component of HEART in patients with CACP reveals that only Troponin score is commensurate to the increasing rate of MACE. Most of the components are not clinically useful, and potentially misleading in this group. Overall, these findings do not support the use of the HEART score as a risk stratification tool in patients with CACP.

The HEART score has previously shown its ability to identify low-risk patients that are safe for early discharge from the ED. A recent meta-analysis reports a 2% rate of events in those deemed low-risk by the HEART score.[23] While our population of patient with non-CACP reflects a similar rate, an exceedingly high rate, up to 7 times higher (i.e., 14%), was observed in patients with CACP at the lowest risk group. This rate of misclassification suggests that the HEART score fails to safely identify patients with CACP eligible for early discharge. Similar to previous literature, patients with CACP in our study did not experience a considerably higher rate of events compared to their counterparts,[8,9,24] suggesting that it is unlikely that this misclassification rate stems from an excess risk of events in this group. A study examining the use of a similar risk stratification tool, TIMI score, reports similar results in CACP, with approximately half of the adverse events occurring in patients stratified as low-risk.[10]

The discriminatory accuracy of the HEART score is reported to be very good with an AUC of 0.83,[11] which is similar to our findings in patients with non-CACP. However, we are the first to report that the performance of the HEART score in CACP is poor. In fact, we show that the Troponin component solely outperforms total HEART score in CACP (Figure 3).

Interestingly, a previous study examining the performance of the TIMI score in this group also showed that the cardiac markers variable was the only risk score component associated with adverse outcomes.[10] Furthermore, a previous study reported that troponin is the strongest predictor for obstructive coronary artery disease in those with CACP.[7]

Our findings suggest that most components of the HEART score, except Troponin, could contribute to the misclassification observed in patients with CACP. For instance, ECG, usually a reliable predictor in typical chest pain, performed poorly in our study. Similar results have been previously reported, suggesting that ECG is a poor predictor of events in CACP.[3,25] Among the challenges in utilizing ECG to risk stratify CACP include the poor sensitivity and predictive value,[26] and the high prevalence of normal variants, such as early repolarization, which can lead to misinterpretation.[27] Likewise, utilizing history to predict events in CACP poses a challenge due to the similarity in character between non-ACS chest pain induced by cocaine to that of an ACS, and the commonly shared associated symptoms such as diaphoresis and shortness of breath.[26] This explains the poor performance of the history component in our cohort of patients with CACP. Moreover, age also seemed to be a poor predictor of events in patients with CACP. In fact, there were no events in those in the highest Age score group in our study. However, it is difficult to draw conclusions on this finding given that the majority of patients in our CACP group were young, with only 4% (n=6/156) older than 65 years.

The Risk factors component of the HEART score was overall the poorest predictor in patients with chest pain in our study, and specifically in those with CACP. More than 40% of patients with CACP with a Risk factors score of “0” had an event. This is not surprising since it has been previously shown that cardiac risk factors fail to provide substantial predictive value in the evaluation of typical acute chest pain.[28,29] Similarly, the components relevant to cardiac risk factors in TIMI score previously failed to provide any predictive value in the evaluation of CACP.[10]

The findings of our study provide an impetus to proceed with caution when evaluating patients with CACP. Considering that patients with CACP possess a different profile to non-selected patients with chest pain, it is not surprising that the predictors of adverse events can vastly differ. The American Heart Association guidelines suggest that patients with CACP be admitted for a 9–12 hours observation period.[3] This approach applies to patients with no cardiac biomarkers elevation, no ischemic ECG changes, and who are judged as low risk by clinical evaluation. The approach of serial troponin measurements has been validated in numerous previous studies,[24,30] with low rate of subsequent evets in those with normal serial troponin measurements despite observation periods as brief as 8 hours.[31,32] It is worth noting, however, that many of these studies recommending a brief observation practice have utilized urine toxicology screen to enroll patients with CACP.[24,30] While our study followed a similar approach in identifying patients, it is important to be mindful that the cardiac effects of cocaine peak during the first few hours after cocaine use[3], while urine toxicology screen remains positive for up to 72 hours[19,20]. This can overestimate the population of cocaine users and suggests that future work is needed to evaluate the temporal cardiac effects from the time of ingestion of cocaine up to 72 hours. Moreover, while subjects are often deemed as presenting with simple manifestations of cocaine use based on urine

toxicology screen, those suffering from cocaine toxicity are often not identified, hence they are neither reported nor analyzed separately. Finally, while an observation strategy might seem overly prudent relative to the generally low rate of events in CACP,[5] our study provides further evidence supporting this practice in view of the diagnostic dilemma of CACP, especially considering that our data show that the HEART score fails to differentiate risk in the low vs. intermediate risk groups.

### Limitations

This study has some limitations to acknowledge. First the sample size of patients who are cocaine users is small (n=156). This represents 6.5% of 2400 patients with chest pain screened over a 5-year period of our study. Further research is needed to expand the sample size and test the validity in a larger population. It is worth noting, however, that difficulties to identify and recruit patients with CACP are common[33]. This is the principal reason for small samples commonly encountered in previous literature, [6,8,9,24] and contributes to the longstanding dearth of evidence regarding this population. Secondly, it is well known that long term cocaine use leads to widespread cardiovascular consequences[34], therefore we are unable to assess whether studying subjects with chronic cocaine use and its cardiovascular manifestations would yield similar results to our study. Thirdly, the HEART score was computed retrospectively, which is especially important for scoring the history component of the HEART score. To address this issue, we used a systematic coding scheme by a reviewer blinded to clinical outcomes and reviewed all available ED records for complete assessment. Using this systematic approach, we have previously demonstrated the adequacy of our HEART score computations given that they yield a prognostic performance similar to that reported in literature[15]. Finally, 30-day MACE was based on the review of the electronic health records (EHR) system of UPMC healthcare network, meaning we could have missed events that occurred outside of our catchment area, for example events occurring during travel of patients, or those who otherwise live in a different city and happened to be in town during their initial visit.

### Conclusion

In this study we sought to evaluate the performance of the HEART score and its components in predicting 30-day MACE in patients with CACP as compared to age- and sex-matched patients with non-CACP. We found that the HEART score performs poorly in patients with CACP, failing to distinguish risk of MACE in those in the low and intermediate risk groups. With the exception of the troponin score, this poor discriminatory value of the HEART score is largely attributed to the poor performance of the components involved in the score calculation, notably risk factors. These findings imply that the HEART score is an inappropriate rule out tool in patients with CACP, and an observation stay for repeated troponin assays seems to be an appropriate practice in these patients.

## Acknowledgments

**Funding Sources:** Supported by the National Institutes of Health (R01 HL-137761). The authors have no disclosures regarding interests in business or industry related to the planning, execution, and/or publication of this study.

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| Assigned Risk Points | | | |
|---|---|---|---|
| | 0 | 1 | 2 |
| **H**istory | Slightly suspicious | Moderately suspicious | Highly suspicious |
| **E**CG | Normal | Non-specific changes | Significant ST changes |
| **A**ge | <45 | 45—64 | ≥65 |
| **R**isk Factors | No known risk factors | 1—2 risk factors | CAD or ≥3 risk factors |
| **T**roponin | <99$^{th}$ percentile | 1—3 times > limit | >3 times > limit |

**Figure 1:**

Calculation of HEART score.

HEART score uses the criteria of **H**istory, **E**CG, **A**ge, **R**isk Factors, and **T**roponin to predict the risk of Major Adverse Cardiac Events (MACE) in patients with chest pain. The risk points are summed up for a total score ranging from 0-10. Based on the total score, the following estimated risk of MACE apply: 0-3 points = low risk; 4-6 points = intermediate risk; 7-10 points = high risk.

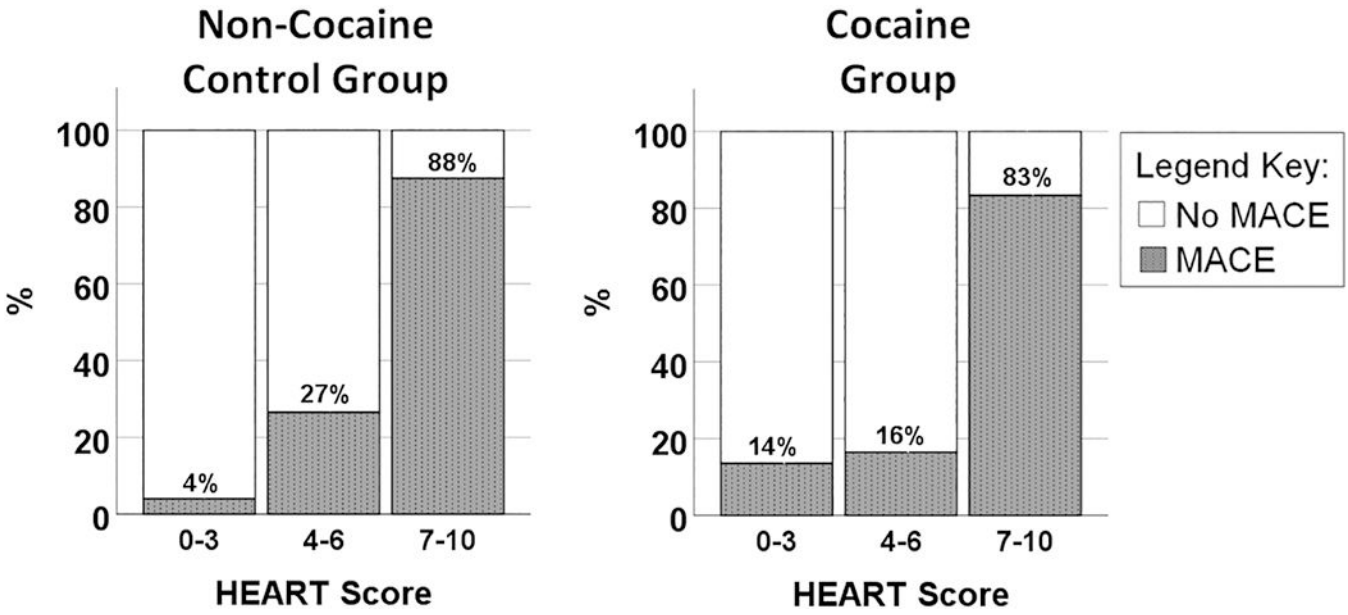


**Figure 2:**

Rate of MACE in each HEART score risk group (low, intermediate, and high).

This figure compares the rate of MACE in each HEART score risk group in Non-CACP vs CACP. In patients triaged as low-risk, patients with CACP were significantly more likely to experience MACE (14% vs. 4%, OR = 3.7 [1.3–10.7], p = 0.016).

Abbreviations: MACE: 30-day major adverse cardiac events; CACP: Cocaine Associated Chest Pain.

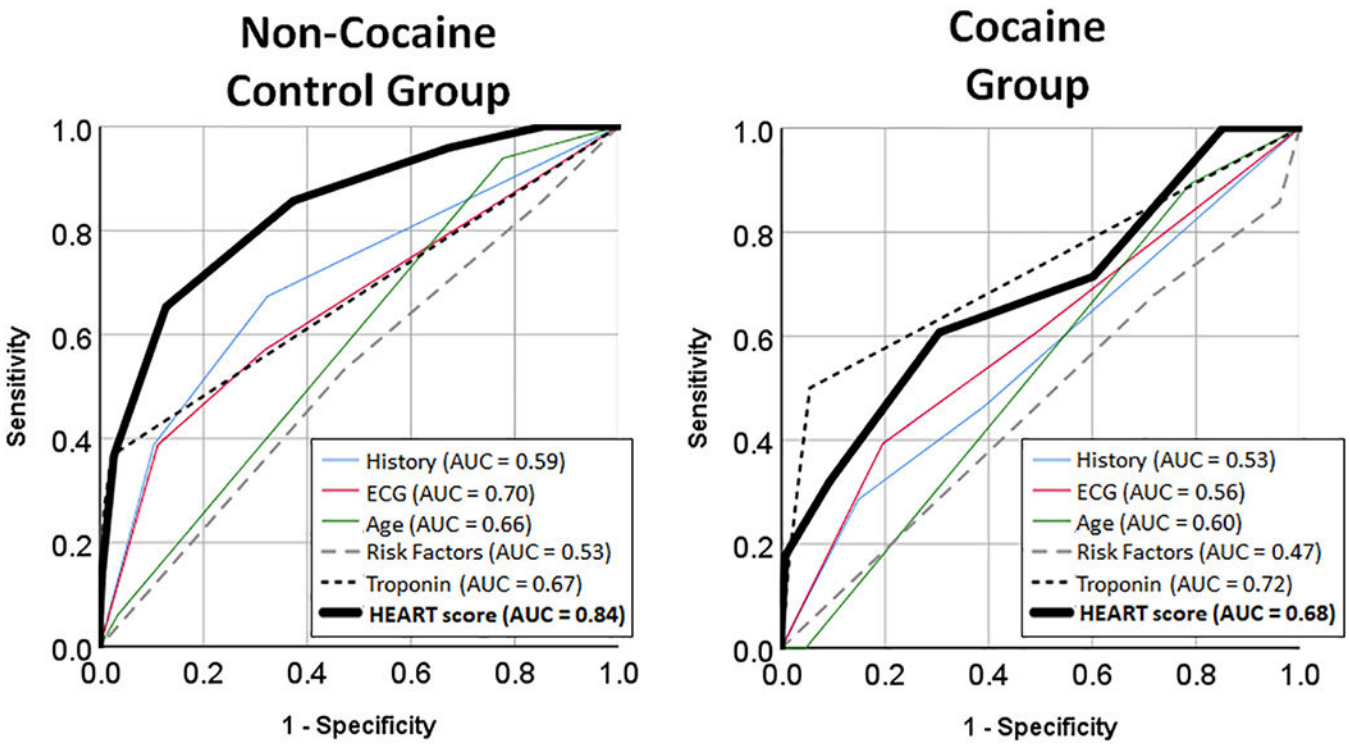


**Figure 3:**

Classification performance of HEART score and each of its component in predicting MACE. This figure shows the classification performance of HEART score and each of its components in predicting MACE. HEART score had a very good classification performance in those with non-CACP, but performed poorly in those with CACP (AUC = 0.84 [0.78–0.90] vs 0.68 [0.56–0.80], p = 0.022). In CACP group, Troponin score had the highest discriminatory value (AUC = 0.72 [0.60–0.85]) and Risk factors score had the lowest (AUC = 0.47 [0.34–0.59]).

Abbreviations: MACE: 30-day major adverse cardiac events; CACP: Cocaine Associated Chest Pain; AUC: Area under the receiver-operator characteristic curve.

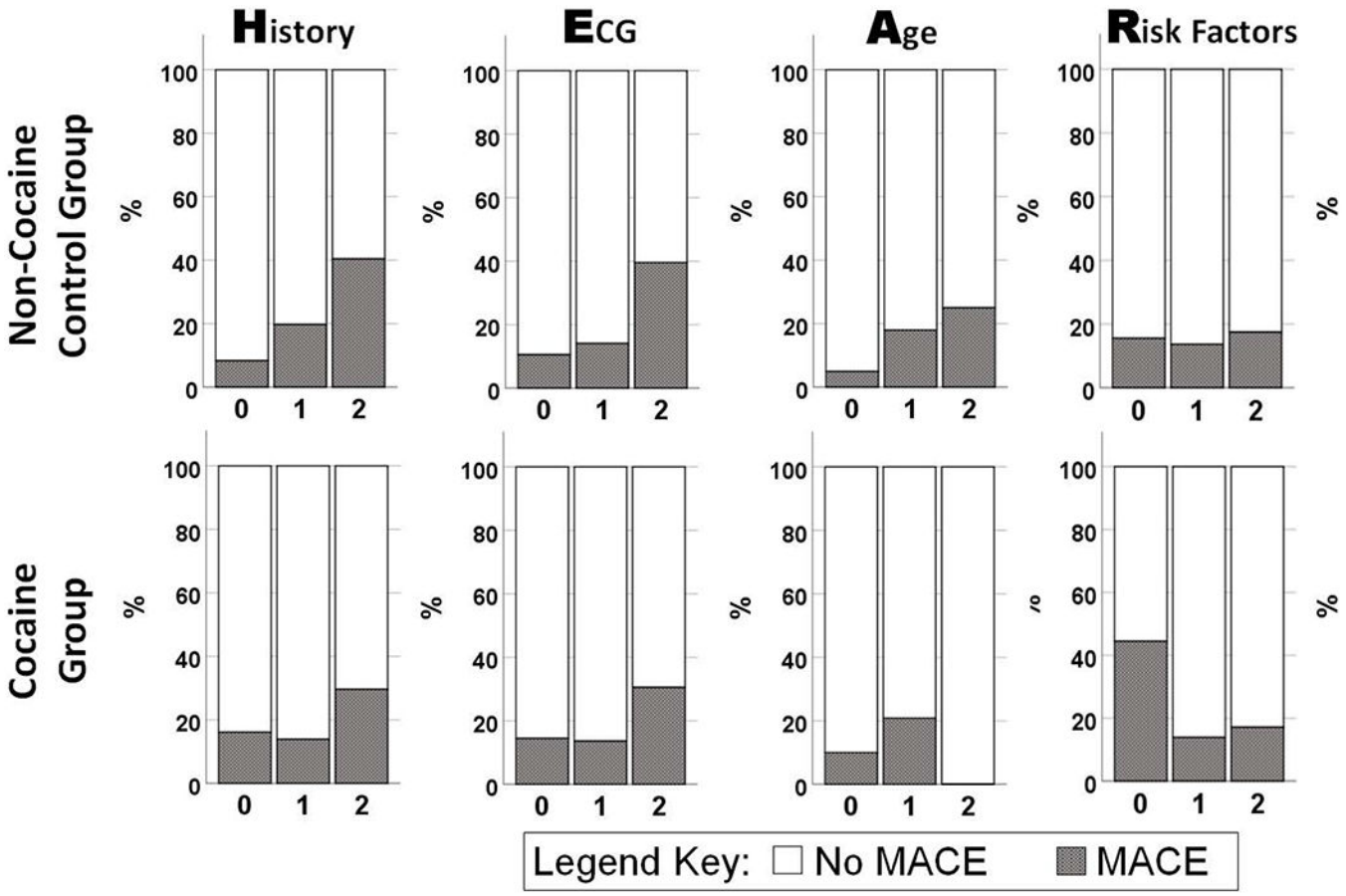


**Figure 4:**

Rate of MACE in each scored component of HEART score.

The rate of MACE within each component of HEART score shows that in non-CACP group, the rate of events increased correspondingly as the assigned risk points increased in all scored components. Such pattern was observed at a lesser magnitude among patients with CACP.

Abbreviations: MACE: 30-day major adverse cardiac events; CACP: Cocaine Associated Chest Pain.

## Table 1

Demographic and Clinical Characteristics of The Study Sample

| Clinical Characteristics | Non-Cocaine Associated Chest Pain (n=312) | Cocaine Associated Chest Pain (n=156) |
|---|---|---|
| **Demographic** | | |
| **Age (years)** | 51 ± 9 | 51 ± 9 |
| **Male Sex** | 244 (78%) | 122 (78%) |
| **Black Race** | 135 (44%) | 97 (62%) |
| **Past Medical History** | | |
| **Ever Smoked** | 191 (61%) | 124 (79%) |
| **High Cholesterol** | 91 (29%) | 53 (34%) |
| **Hypertension** | 183 (59%) | 117 (75%) |
| **Diabetes Mellitus** | 81 (26%) | 39 (25%) |
| **Known CAD** | 77 (25%) | 69 (44%) |
| **HEART Score (mean ± SD)** | | |
| **Total Score** | 3.3 ± 1.6 | 4.0 ± 1.4 |
| **Score 0–3** | 172 (55%) | 59 (38%) |
| **Score 4–6** | 132 (42%) | 91 (58%) |
| **Score 7–10** | 8 (3%) | 6 (4%) |

Values are mean ± SD; or n (%).

CAD: Coronary Artery Disease.