Shock Index as a Predictor of Myocardial Damage and Clinical Outcome in ST-Elevation Myocardial Infarction

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Background: Data on the prognostic value of the shock index in patients with ST-elevation myocardial infarction (STEMI) are scarce. Furthermore, the relationship of the shock index with myocardial damage is unknown. The aim of this study was to evaluate the association of the shock index with markers of myocardial damage and clinical outcome in patients with STEMI.

Methods and Results: This multicenter study analyzed 791 patients. Patients were categorized in 2 groups according to the admission shock index (optimized cut-off=0.62). Infarct severity was determined by cardiac magnetic resonance (CMR) imaging. Patients with cardiogenic shock that were unable to undergo CMR acquisition were excluded. Major adverse cardiac events (MACE) were defined as a composite of death, reinfarction and congestive heart failure within 12 months. Patients with elevated admission shock index (n=321 [40.6%]) had a significantly larger area-at-risk (37.6 [27.8–50.4] % of left ventricular volume [LV] vs. 34.3 [24.5–46.0] % LV, P=0.02), larger infarct size (19.5 [10.7–28.0] % LV vs. 14.9 [7.7–22.3] % LV, P<0.001), lower myocardial salvage index (46.2 [27.9–64.5] vs. 53.5 [36.5–75.2], P<0.001), and a larger extent of microvascular obstruction (0.3 [0.0–2.2] % LV vs. 0.0 [0.0–1.4] % LV, P=0.01). An elevated shock index was associated with reduced MACE-free survival (P<0.001). Furthermore, the admission shock index was identified as an independent predictor of MACE (hazard ratio=2.92 [1.24–4.22], P<0.01).

Conclusions: STEMI patients with an elevated admission shock index had more pronounced myocardial and microvascular damage. Moreover, the shock index was independently associated with MACE at 12 months. (Circ J 2016; 80: 924–930)

Key Words: Cardiac magnetic resonance imaging; Myocardial infarction; Prognosis; Shock index

Early risk stratification is crucial for clinical decision-making and optimized therapy in patients with ST-elevation myocardial infarction (STEMI). Several risk scores, such as the Global Registry of Acute Coronary Events (GRACE)-risk score, the Thrombolysis in Myocardial Infarction (TIMI)-risk score or the Controlled Abciximab and Device Investigation to Lower Angioplasty Complications (CADILLAC)-risk score have been developed to identify STEMI patients at increased risk for adverse outcome. However, these risk scores need sophisticated calculation and are therefore not perfectly suited as bedside tools for fast risk stratification in daily clinical routine. In contrast, the shock index, which is defined as the ratio of heart rate and systolic blood pressure, might be a simple clinical tool that allows rapid risk stratification. The prognostic significance of the shock index was investigated in different populations of critically ill patients in the setting of trauma, surgery or sepsis. Nevertheless, there are only sparse data addressing the prognostic value of the shock index in patients with STEMI. Furthermore, the relationship of the shock index with myocardial damage has not been previously examined. Contrast-enhanced cardiac magnetic resonance (CMR) imaging emerged as a reference technique for comprehensive assessment of the jeopardized and infarcted myocardium following STEMI.
These CMR markers of infarct severity have been shown to be strongly related with adverse clinical outcomes in patients with STEMI.\(^{13-19}\) In the present study, we therefore sought to explore the relationship of the admission shock index with myocardial damage as visualized by CMR, and to determine the prognostic value of the shock index in a large multicenter cohort of STEMI patients.

**Methods**

**Study Population**

This multicenter CMR study was a predefined substudy of the AIDA STEMI trial (Abciximab Intracoronary vs. intravenously Drug Application in ST-Elevation Myocardial Infarction; NCT00712101). AIDA STEMI was a randomized, open-label, multicenter trial, comparing the effect of intracoronary vs. standard intravenous bolus application of abciximab in patients with STEMI.\(^{20}\) The study design and main results of AIDA STEMI, as well as of the CMR substudy, were published previously.\(^{16,20,22}\) Briefly, STEMI patients presenting <12h after symptom onset were enrolled. The CMR substudy included 795 patients according to the following exclusion criteria: (1) severe claustrophobia; (2) hemodynamic instability, which does not allow CMR acquisition; (3) pacemaker or internal cardioverter defibrillator; (4) metallic cerebral or intracranial implants; (5) known allergy to gadolinium; and (6) severe renal insufficiency. Importantly, the CMR substudy found no significant difference in CMR markers of myocardial damage between both groups.\(^{22}\) For the purpose of the current analysis, only patients with recorded admission heart rate and admission systolic blood pressure were eligible (n=791, 99.5%). Patient characteristics, including blood pressure and heart rate, were systematically assessed according to a predefined study protocol. The shock index was defined as the ratio of heart rate and systolic blood pressure. Patients were grouped into 2 categories according to the Youden index after application of a receiver operating characteristic (ROC) curve to assess the optimal cut-off value for the prediction of major adverse cardiac events (MACE): (1) normal admission shock index (<0.62); and (2) elevated admission shock index (≥0.62). For a secondary analysis, we dichotomized the patients based on previous literature:\(^{9}\) (1) normal admission shock index (<0.7); and (2) elevated admission shock index (≥0.7). The clinical end-point was the incidence of MACE, defined as a composite of all-cause death, non-fatal reinfarction and new congestive heart failure at 12 months after infarction. More detailed end-point definitions are reported elsewhere.\(^{20,22}\) The modified shock index was calculated as the ratio of heart rate and mean arterial pressure. The local ethics committee approved the study and patients were required to provide written informed consent.

**CMR Imaging**

Patients underwent CMR imaging on 1.5 Tesla or 3.0 Tesla...
baseline characteristics in Table 1 and admission shock index were investigated in a univariate Cox regression model. Variables with a P-value of <0.1 were incorporated in a stepwise multivariate Cox regression analysis. Outcome functions were estimated using Kaplan-Meier graphs, and shock index groups were compared using the log-rank test. ROC analysis was used to determine the predictive accuracy of the shock index. The shock index was dichotomized according to the optimized cut-off value (=0.62), which was calculated by ROC analysis using the following formula:

\[ \sqrt{1 - \text{Sensitivity}} + (1 - \text{Sensitivity})^2 \]

The incremental additive information of the shock index over Killip-class for the prediction of MACE was assessed with c-statistics, as previously described. 25 All statistical tests were 2-tailed, and a P-value <0.05 was considered statistically significant. Statistical analysis was performed with SPSS 22.0.0.1 (IBM; Armonk, NY, USA) and MedCalc Version 12.2.1 (Ostend, Belgium).

**Results**

**Baseline Patient Characteristics**

The admission shock index was available in 791 (99.5%) patients. Of these, 470 (59.4%) had a normal admission shock index. Scanners were available in 791 patients within 10 days after infarction. All scans were performed according to standardized sequences, as previously published. 20 Briefly, cine sequences were used for the measurement of left ventricular (LV) ejection fraction, T2-weighted imaging for assessment of the area-at-risk, and late enhancement imaging for determination of infarct size and microvascular obstruction. Images were evaluated with standard CMR post-processing software (cmr42; Circle Cardiovascular Imaging Inc, Calgary, Alberta, Canada). Readers blinded to study data performed the image evaluation at the CMR core laboratory (University of Leipzig - Heart Center, Leipzig, Germany). The core laboratory has proven excellent reproducibility and low inter- as well as intra-observer variability in STEMI patients. 23 The measurements of the area-at-risk, infarct size, and microvascular obstruction were expressed as the percentage of LV volume (%LV). 22 The myocardial salvage index was determined, as recently described. 22,24

**Statistical Analysis**

Continuous variables are shown as median plus interquartile range or frequencies and percentage. ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CABG, coronary artery bypass grafting; CAD, coronary artery disease; PPCI, primary percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction.

| Number of diseased vessels | Total study cohort (n=791) | Normal shock index (n=470) | Elevated shock index (n=321) | P value |
|----------------------------|---------------------------|---------------------------|-----------------------------|---------|
| 1, n (%)                   | 420 (53)                  | 257 (55)                  | 163 (51)                    | 0.40    |
| 2, n (%)                   | 224 (28)                  | 133 (28)                  | 91 (28)                     |         |
| 3, n (%)                   | 147 (19)                  | 80 (17)                   | 67 (21)                     |         |

| Infarct related artery     |                           |                           |                             |         |
|---------------------------|---------------------------|---------------------------|                             |         |
| Left anterior descending, n (%) | 345 (44)                  | 207 (44)                  | 137 (43)                    | 0.47    |
| Right coronary artery, n (%) | 343 (43)                  | 206 (44)                  | 138 (43)                    |         |
| Left circumflex, n (%)     | 96 (12)                   | 55 (12)                   | 41 (13)                     |         |
| Left main, n (%)           | 5 (1)                     | 1 (0)                     | 4 (1)                       |         |
| Bypass graft, n (%)        | 2 (0)                     | 1 (0)                     | 1 (0)                       |         |

| TIMI-risk score            |                           |                           |                             | <0.001  |
|----------------------------|---------------------------|---------------------------|                             |         |
| TIMI-flow before PPCI      |                           |                           |                             |         |
| TIMI-flow 0, n (%)         | 443 (56)                  | 251 (53)                  | 192 (60)                    | 0.03    |
| TIMI-flow 1, n (%)         | 103 (13)                  | 55 (12)                   | 48 (15)                     |         |
| TIMI-flow 2, n (%)         | 128 (16)                  | 85 (18)                   | 43 (13)                     |         |
| TIMI-flow 3, n (%)         | 117 (15)                  | 79 (17)                   | 38 (12)                     |         |

| TIMI-flow after PPCI       |                           |                           |                             | 0.05    |
|----------------------------|---------------------------|---------------------------|                             |         |
| TIMI-flow 0, n (%)         | 12 (2)                    | 6 (1)                     | 6 (2)                       |         |
| TIMI-flow 1, n (%)         | 19 (2)                    | 7 (2)                     | 12 (4)                      |         |
| TIMI-flow 2, n (%)         | 62 (8)                    | 31 (7)                    | 31 (10)                     |         |
| TIMI-flow 3, n (%)         | 697 (88)                  | 426 (91)                  | 271 (84)                    |         |

| Stent implanted, n (%)     | 775 (98)                  | 458 (97)                  | 317 (99)                    | 0.20    |
| Thrombectomy, n (%)        | 190 (24)                  | 107 (23)                  | 83 (26)                     | 0.32    |

| Concomitant medications   |                           |                           |                             |         |
|----------------------------|---------------------------|---------------------------|                             |         |
| Aspirin, n (%)             | 789 (100)                 | 468 (100)                 | 321 (100)                   | 1.0     |
| β-blockers, n (%)          | 755 (95)                  | 444 (95)                  | 311 (97)                    | 0.17    |
| ACE-I/ARB, n (%)           | 751 (95)                  | 443 (94)                  | 308 (96)                    | 0.41    |
| Statin, n (%)              | 750 (95)                  | 442 (94)                  | 308 (96)                    | 0.34    |
| Aldosterone antagonist, n (%) | 91 (12)                   | 50 (11)                   | 41 (13)                     | 0.37    |

Values are shown as median plus interquartile range or frequencies and percentage. ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CABG, coronary artery bypass grafting; CAD, coronary artery disease; PPCI, primary percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction.
Shock index (<0.62) and 321 (40.6%) had an increased admission shock index (≥0.62). Baseline characteristics of the entire study population as well as their association with the admission shock index are shown in Table 1. Patients with an elevated admission shock index had a higher Killip-class on admission (P=0.008) and a higher TIMI-risk score (P<0.001). Furthermore, male gender was less frequent (P=0.02) and body mass index was lower (P=0.03) in patients with an elevated shock index. Worse TIMI-flow before and after primary percutaneous coronary intervention (PPCI) was observed in the ≥0.62 shock index group (P=0.03 and P=0.05, respectively).

Shock Index and Clinical Outcome
Clinical follow up was available in 788 patients (99%). The cumulative 12-month MACE rate was 6.7% (n=53). Of these, 22 (2.8%) patients died. Patients with an elevated shock index (≥0.62, sensitivity 66%, specificity 61%, negative predictive value 96%) were at higher risk for MACE compared with patients with a normal shock index (10.9% vs. 3.8%, P<0.001) (Figure 1). The admission shock index was a significant predictor of MACE in ROC analysis (area under the curve [AUC]=0.67 [0.60–0.75]) (Figure 2). The shock index provided a higher predictive accuracy compared to systolic blood pressure (AUC=0.59 [0.50–0.68]) or heart rate (AUC=0.63 [0.55–0.70]) alone (P=0.04 and P=0.21, respectively). The inclusion of the shock index in addition to Killip-class resulted in an increase of c-statistics from 0.62 to 0.68 (P=0.04), thus demonstrating incremental prognostic value of the shock index over Killip-class. The comparison of TIMI-risk score (AUC=0.71 [0.64–0.79]) and shock index revealed no significant difference in the predictive accuracy between both risk tools (P=0.36). In multivariate Cox regression analysis, an admission shock index of ≥0.62 emerged as an independent predictor of MACE (hazard ratio=2.29 [1.24–4.22], P=0.008) (Table 2). Patients with a shock index of ≥0.62 also had a significantly higher rate of mortality (4.7% vs. 1.5%, P=0.008), re-infarctions (4.0% vs. 1.7%, P=0.04), and heart failure episodes (5.9% vs. 1.3%, P<0.001). Very similar results for the association between the shock index and clinical outcome were found when categorizing patients according to another cut-off value (shock index cut-off=0.7) reported in previous literature (data not shown). Moreover, the exclusion of all patients with cardiac arrhythmias on admission (6%) did not significantly change the association of the shock index with MACE (data not shown).

Shock Index and CMR Parameters
CMR imaging was performed at 3 days [IQR 2–4] after infarc-
A further retrospective}

**Discussion**

This is the first CMR study investigating the association of the admission shock index with markers of myocardial damage and MACE in a multicenter cohort of STEMI patients treated with PPCI. The main findings of our study are: (1) the admission shock index is significantly associated with CMR markers of myocardial injury after mechanical reperfusion therapy; and (2) the admission shock index is an independent predictor of MACE at 1 year following infarction. Together, our data support the use of this simple bedside risk tool for the estimation of infarct severity and clinical prognosis in STEMI patients.

**Shock Index and Clinical Outcome**

Cardiogenic shock occurs in approximately 5–15% of patients with acute STEMI. Despite improvements in treatment, it remains the leading cause of death in STEMI patients. Patients with a decrease in blood pressure and an increase in heart rate are at risk of developing cardiogenic shock. The shock index is an accurate and easily assessable risk marker at the time of initial evaluation, which combines the information of systolic blood pressure and heart rate. Indeed, several authors have suggested that the shock index may be a more reliable measure of the degree of hemodynamic stability than systolic blood pressure or heart rate alone.

**Table 2. Predictors of MACE in Univariate and Multivariate Cox Regression Analysis**

| Variable                        | Univariate |             | Multivariate |             |
|---------------------------------|------------|-------------|--------------|-------------|
|                                | HR (95% CI)| P value     | HR (95% CI)  | P value     |
| Age, years                      | 1.05 [1.03–1.08] | <0.001     | 1.05 [1.02–1.08] | <0.001     |
| Gender, female                  | 1.96 [1.13–3.42] | 0.02       | –            | NS          |
| Smoking status, yes             | 0.46 [0.24–0.88] | 0.02       | –            | NS          |
| Hypertension, yes               | 2.70 [1.27–5.73] | 0.01       | –            | NS          |
| Killip-class, I–IV              | 1.90 [1.46–2.48] | <0.001     | 1.61 [1.17–2.20] | 0.003      |
| Shock index, 1 ≥0.62            | 2.92 [1.65–5.15] | <0.001     | 2.29 [1.24–4.22] | 0.008      |

Stepwise Cox regression analysis for the prediction of MACE at 12 months after STEMI. Only significant variables in univariate analysis are shown. CI, confidence interval; HR, hazard ratio; MACE, major adverse cardiac events; NS, not significant; STEMI, ST-elevation myocardial infarction.

**Table 3. CMR Imaging Findings**

| Variable                        | Univariate | Normal shock index | Elevated shock index | P value |
|---------------------------------|------------|--------------------|----------------------|---------|
| Area at risk, %LV               | 34.9 [25.4–47.4] | 34.3 [24.5–46.0] | 37.6 [27.8–50.4] | 0.02    |
| Infarct size, %LV               | 16.7 [8.4–24.9] | 14.9 [7.7–22.3] | 19.5 [10.7–28.0] | <0.001  |
| Presence of microvascular obstruction, n (%) | 400 (51) | 224 (48) | 176 (55) | 0.04    |
| Microvascular obstruction, %LV  | 0.0 [0.0–1.8] | 0.0 [0.0–1.4] | 0.3 [0.0–2.2] | 0.01    |
| Myocardial salvage index        | 51.0 [32.9–69.1] | 53.5 [36.5–75.2] | 46.2 [27.9–64.5] | <0.001  |
| LV ejection fraction, %         | 50.5 [43.3–57.6] | 51.8 [44.9–58.1] | 48.1 [40.4–56.9] | <0.001  |

CMR variables are shown as median plus interquartile range or frequencies and percentage. CMR, cardiac magnetic resonance; LV, left ventricular/ventricle.

**Shock Index vs. Modified Shock Index**

Patients with MACE showed a significantly higher modified shock index on admission as compared with patients without MACE (0.91 [0.79–1.08] vs. 0.78 [0.68–0.92], P<0.001). The modified shock index was a significant predictor of MACE in ROC analysis (AUC=0.67 [0.60–0.75]) (Figure 2). However, the accuracy for the prediction of MACE, death, re-infarction or heart failure did not differ significantly from that of the shock index (P>0.05) (Figure 2).

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**Shock Index and Clinical Outcome**

Cardiogenic shock occurs in approximately 5–15% of patients with acute STEMI. Despite improvements in treatment, it remains the leading cause of death in STEMI patients.6,27 Patients with a decrease in blood pressure and an increase in heart rate are at risk of developing cardiogenic shock. The shock index is an accurate and easily assessable risk marker at the time of initial evaluation, which combines the information of systolic blood pressure and heart rate. Indeed, several authors have suggested that the shock index may be a more reliable measure of the degree of hemodynamic stability than systolic blood pressure or heart rate alone.28,29 Accordingly, the shock index is proposed for risk stratification in patients with trauma, sepsis, pulmonary embolism or stroke.6,7,30,31 Although the concept of the shock index has been already described in 1967,32 only a few studies have addressed its prognostic significance in patients with STEMI.8-10 In addition, these studies were hampered by several shortcomings, including single-center cohorts, short follow-up periods and lacking data on other risk assessment tools (eg, Killip-class and/or TIMI-risk score). Bilkova et al retrospectively evaluated 644 patients treated with PPCI (92%) or rescue PCI (7%) and found that the admission shock index was independently associated with in-hospital mortality.9 A further retrospective
evaluation by Huang et al showed that the admission shock index may also be a useful predictor for short-term mortality and MACE. The only study investigating the prognostic significance of the shock index after a longer follow-up period (approximately 15 months) observed that patients with an elevated shock index were at a higher risk of death. Our multicenter study reflecting contemporary PCI practice complements and extends these previous findings by showing that the admission shock index is strongly related to the occurrence of MACE at 12 months after STEMI. The difference in MACE between groups was driven by a significantly higher rate of death, re-infarctions and heart failure events. The AUC for the shock index for the prediction of MACE was higher compared with the AUCs of systolic blood pressure or heart rate alone, highlighting the superior prognostic value of the shock index. Importantly, this study for the first time also demonstrates that the shock index offers incremental prognostic value for MACE in addition to the well-established Killip classification. Moreover, when comparing the shock index with the more sophisticated TIMI-risk score for STEMI we found that both risk tools were similar in their predictive accuracy. Considering this growing body of evidence and that the shock index is a very simple and objective index derived from 2 readily available vital signs, it should be used in daily routine to accurately risk stratify patients presenting with STEMI. Nevertheless, well-designed prospective studies are necessary to assess whether these patients can benefit from the early assessment of the shock index; for example, by higher triage priority or additional therapeutic measures.

**Shock Index and Myocardial Damage**

Our study is the first that comprehensively evaluates the correlation between the shock index and CMR markers of myocardial damage in STEMI patients. We found that patients with a shock index of ≥0.62 on admission had significantly larger infarcts, more severe reperfusion injury and consequently more reduced LV ejection fraction. In recent years, it has been convincingly shown that CMR parameters of myocardial damage, primarily microvascular obstruction, but also infarct size and myocardial salvage index as well as LV ejection fraction, provide strong prognostic information post-STEMI. Accordingly, the ability of the shock index to immediately identify a subset of high-risk STEMI patients is further underlined by our CMR findings.

Animal and clinical studies by Rady et al showed that the shock index may be associated with parameters of cardiovascular function such as cardiac index, stroke volume, and LV stroke work. Furthermore, in patients with myocardial infarction, there is clear evidence of over-activity of the sympathetic nervous system, which plays a central role in regulating heart rate and blood pressure. This sympathetic hyper-activity was also related to the degree of LV dysfunction and clinical outcome. In the setting of acute myocardial infarction, heart rate and blood pressure may therefore reflect an integrated cardiovascular and neuroendocrine system as well as hemodynamic status. One might therefore speculate that the prognostic value of the shock index may be mediated by the sensitive recognition of hemodynamic variations. Our data further underline these findings and show, in contrast, that an elevated shock index also reflects the extent of myocardial necrosis and microvascular damage. Nevertheless, the detailed pathophysiological association between the shock index, cardiovascular function, myocardial damage and the mechanisms of associated sympathetic activation needs further evaluation in well-designed experimental studies.

**Shock Index vs. Modified Shock Index**

Some evidence suggests that the modified shock index (heart rate/mean arterial pressure) might be an even better predictor of outcome as compared to the shock index. However, to the best of our knowledge, only one small (n=160), retrospective study compared the shock index with the modified shock index for the short-term prediction of a combined clinical endpoint (all-cause mortality, life-threatening arrhythmias, cardiogenic shock, and heart failure) in patients after STEMI. The authors of this study found that both the shock index and the modified shock index were independent predictors of MACE, but the odds ratio of the modified shock index was numerically higher than that of the shock index. In our analysis, we observed no difference in the predictive value for MACE of the shock index and the modified shock index. Nevertheless, larger studies comparing both indexes are still needed to draw definitive conclusion for their use in STEMI patients.

**Study Limitations**

Our study has some important limitations. The sample size and event rate is relatively low and therefore further studies evaluating the shock index in STEMI patients are warranted. Patients with cardiac arrhythmias (eg, atrial fibrillation) complicating STEMI might have a higher risk of adverse events. Unfortunately, we were unable to characterize the influence of arrhythmias on shock index in detail due to the relatively low number of cases. In addition, the inclusion and exclusion criteria (eg, patients with cardiogenic shock complicating STEMI who were unable to undergo CMR acquisition) of this multicenter clinical trial, as well as the treatment of patients in specialized high-volume centers, might limit the generalization of study results. Although AIDA STEMI was designed to represent a “real-world” STEMI population, further confirmation remains desirable. Finally, information on pre-STEMI medications and prehospital treatment of STEMI patients that could affect heart rate and/or blood pressure was not available for the current analysis and therefore needs clarification in further studies.

**Conclusions**

An elevated shock index on admission is associated with more severe myocardial and microvascular damage at CMR, and independently predicts the occurrence of MACE at 1 year after mechanical reperfusion for acute STEMI. Consequently, this very simple and objective index can help to accurately identify high-risk STEMI patients in daily clinical routine.

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None.

**Conflicts of Interests**

None.

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