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# Utilizing the $CHA_2DS_2$-VASc-HSF Score to Predict Subclinical Acute Kidney Injury from Contrast Agents: A Focus on Neutrophil Gelatinase-Associated Lipocalin Biomarker

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**Abstract**

**Background**: Contrast-Induced Acute Kidney Injury (CI-AKI) is a common complication in patients undergoing Percutaneous Coronary Intervention (PCI), with Subclinical Contrast-Induced Acute Kidney Injury (SCI-AKI) detectable via biomarkers like Neutrophil Gelatinase-Associated Lipocalin (NGAL). The $CHA_2DS_2$-VASc score, developed for stroke risk in atrial fibrillation, has been modified to $CHA_2DS_2$-VASc-HSF for assessing cardiovascular risk, but its role in predicting SCI-AKI remains unclear. **Objectives**: This study aimed to evaluate the $CHA_2DS_2$-VASc-HSF score as an indicator for SCI-AKI risk in PCI patients, using NGAL as a biomarker for early kidney injury. **Methods**: A cohort study was conducted at Dr. M. Djamil General Hospital, Padang, from May to December 2024. Thirty-four patients with stable and unstable angina undergoing PCI were included. NGAL levels were measured before and after PCI. Statistical analysis examined the relationship between $CHA_2DS_2$-VASc-HSF score and SCI-AKI. **Results**: Nine patients (26.5%) were diagnosed with SCI-AKI. NGAL levels showed a significant difference between SCI-AKI positive and negative groups ($p < 0.05$), but no significant association was found between $CHA_2DS_2$-VASc-HSF score and SCI-AKI ($p > 0.05$). **Discussion**: The $CHA_2DS_2$-VASc-HSF score is effective for cardiovascular risk assessment but not for SCI-AKI prediction. SCI-AKI was better predicted by NGAL, a sensitive biomarker of early kidney injury. This study highlights the importance of combining multiple risk factors for accurate SCI-AKI risk stratification in PCI patients. **Conclusion**: The $CHA_2DS_2$-VASc-HSF score is not a reliable predictor for SCI-AKI. NGAL remains a valuable biomarker for early detection, emphasizing the need for a multifactorial approach in managing renal complications post-PCI.

**Keywords**: Acute kidney injury, Neutrophil Gelatinase-Associated Lipocalin, Percutaneous coronary intervention, SCI-AKI

***Abstrak***

***Latar Belakang:*** *Cedera Ginjal Akut Akibat Kontras (CI-AKI) merupakan komplikasi umum pada pasien yang menjalani Intervensi Koroner Perkutan (PCI), dengan Cedera Ginjal Akut Akibat Kontras Subklinis (SCI-AKI) yang dapat dideteksi melalui biomarker seperti Neutrophil Gelatinase-Associated Lipocalin (NGAL). Skor $CHA_2DS_2$-VASc, yang dikembangkan untuk risiko stroke pada fibrilasi atrium, telah dimodifikasi menjadi $CHA_2DS_2$-VASc-HSF untuk menilai risiko kardiovaskular, tetapi perannya dalam memprediksi SCI-AKI masih belum jelas. Tujuan: Penelitian ini bertujuan untuk mengevaluasi skor $CHA_2DS_2$-VASc-HSF sebagai indikator risiko SCI-AKI pada pasien PCI, dengan menggunakan NGAL sebagai biomarker untuk cedera ginjal dini.* ***Metode:*** *Studi kohort dilakukan di Rumah Sakit Umum Dr. M. Djamil, Padang, dari Mei hingga Desember 2024. Sebanyak 34 pasien dengan angina stabil dan tidak stabil yang menjalani PCI diikutsertakan. Kadar NGAL diukur sebelum dan sesudah PCI. Analisis statistik mengkaji hubungan antara skor $CHA_2DS_2$-VASc-HSF dan SCI-AKI.* ***Hasil:*** *Sembilan pasien (26,5%) didiagnosis SCI-AKI. Kadar NGAL menunjukkan perbedaan yang signifikan antara kelompok SCI-AKI positif dan negatif ($p < 0,05$), tetapi tidak ditemukan hubungan yang signifikan antara skor $CHA_2DS_2$-VASc-HSF dan SCI-AKI ($p > 0,05$).* ***Pembahasan:*** *Skor $CHA_2DS_2$-VASc-HSF efektif untuk penilaian risiko kardiovaskular tetapi tidak untuk prediksi SCI-AKI. SCI-AKI diprediksi lebih baik*

*oleh NGAL, suatu biomarker sensitif untuk cedera ginjal dini. Studi ini menyoroti pentingnya menggabungkan berbagai faktor risiko untuk stratifikasi risiko SCI-AKI yang akurat pada pasien PCI.* ***Kesimpulan:*** *Skor $CHA_2DS_2$-VASc-HSF bukanlah prediktor yang andal untuk SCI-AKI. NGAL tetap menjadi biomarker yang berharga untuk deteksi dini, yang menekankan perlunya pendekatan multifaktorial dalam menangani komplikasi ginjal pasca-PCI.*

***Kata kunci:*** *Cedera ginjal akut, Neutrophil Gelatinase-Associated Lipocalin, Intervensi koroner perkutan, SCI-AKI*

## INTRODUCTION

Acute Kidney Injury (AKI) is a condition characterized by a sudden decline in kidney function due to various factors such as sepsis, medications, hemodynamic disturbances, contrast media, and others, with diverse clinical manifestations. One form of kidney impairment associated with the use of contrast agents, known as Contrast-Induced Acute Kidney Injury (CI-AKI), can occur following Percutaneous Coronary Intervention (PCI). CI-AKI represents an acute kidney disorder that is generally reversible. It is characterized by a 25% increase in serum creatinine levels from baseline or an absolute increase in creatinine of 0.5 mg/dL (44 µmol/L) within 24–72 hours after contrast agent administration.[2,11,20]

Subclinical Contrast-Induced Acute Kidney Injury (SCI-AKI) is a diagnosis of subclinical AKI based on biomarkers without any detectable changes in filtration function. Patients with SCI-AKI do not exhibit classical clinical manifestations but show an increased detection of kidney injury biomarkers, one of which is Neutrophil Gelatinase-Associated Lipocalin (NGAL).[21] The $CHA_2DS_2$-VASc score is a scoring system that assesses the presence of Congestive Heart Failure (CHF)/left ventricular dysfunction, hypertension, age ≥75 years, diabetes mellitus, history of stroke, vascular disease, age 65–74 years, and female gender. Initially, the $CHA_2DS_2$-VASc score was developed for stroke risk stratification in patients with atrial fibrillation (AF). However, recent studies have utilized this score to predict the occurrence of CI-AKI, SCI-AKI, major adverse cardiovascular events (MACE) during hospitalization, coronary artery disease severity, and more. A modified version of this score, known as $CHA_2DS_2$-VASc-HSF, has been developed by adding Hyperlipidemia (H), Smoking (S), and Family History (F) as additional risk factors for CI-AKI.[12]

Recent studies have shown that the $CHA_2DS_2$-VASc score is associated with CI-AKI risk factors in elective patients undergoing Percutaneous Coronary Intervention (PCI). [12] Based on the PCI registry data at Dr. M. Djamil General Hospital, Padang, several cases of CI-AKI have been identified. However, an indicator to assess CI-AKI risk in PCI patients has not yet been established due to the limited number of previous studies in Indonesia. This gap in research serves as the background for this study, which aims to investigate the influence of the $CHA_2DS_2$-VASc-HSF score as a risk factor indicator for Subclinical Contrast-Induced Acute Kidney Injury (SCI-AKI) based on Neutrophil Gelatinase-Associated Lipocalin (NGAL) in patients undergoing elective PCI, particularly at Dr. M. Djamil General Hospital, Padang.

## I. METHOD

This study is a cohort study in which independent and dependent variables are observed simultaneously and followed over a specified period. The study was conducted at the Integrated Cardiac Service Unit of Dr. M. Djamil General Hospital, Padang, from May to December 2024. The study population included all patients with stable angina pectoris (SAP) and unstable angina pectoris (UAP) undergoing elective PCI in the hospital's cardiac catheterization laboratory. The research sample consisted of SAP and UAP patients who met the inclusion and exclusion criteria and underwent elective PCI from June to August 2024. The inclusion criteria are patients with stable and unstable angina pectoris undergoing elective PCI, patients with normal estimated glomerular filtration rate (eGFR), and who provided informed consent with exclusion criteria the patients with stable and unstable angina pectoris not undergoing PCI, and

patients with abnormal eGFR. Sampling was conducted consecutively, ensuring a balanced distribution of patients with $CHA_2DS_2$-VASc-HSF scores <2 and ≥2. Data collection was performed through medical records and laboratory data of patients undergoing elective PCI from June to August 2024. The level of NGAL were assessed by collecting venous blood samples (2 mL) twice—before and after PCI. NGAL analysis was performed using the ELISA method at the Biomedical Laboratory, Faculty of Medicine, Andalas University.

## II. RESULTS

**TABLE 1. CHARACTERISTIC OF RESEARCH SUBJECTS**

| Variable | n = 34, F (%) | Mean ± SD |
|---|---|---|
| Age (years), mean ± SD | | 61.47 ± 7.5 |
| Gender, n (%) | | |
| Men | 27 (79.4) | |
| **Blood Pressure (mmHg)** | | |
| Systolic | | 125 ± 14.3 |
| Diastolic | | 71.29 ± 9.2 |
| **Diagnosis** | | |
| *Stabe Angina pectoris* | 15 (44.1) | |
| *Unstable angina pectoris* | 19 ( 55.9) | |
| **CHA2DS2-VAS-HSF Score** | | |
| CHF | 8 (23.5) | |
| Hypertension | 15 (44.1) | |
| Age ≥ 75 yo | 1 (2.9) | |
| Diabetes Mellitus | 4 (11.8) | |
| Stroke History | 1 (2.9) | |
| Vascular disease (CAD,PAD) | 21 (61.8) | |
| Age 65-74 yo | 12 (35.3) | |
| Hyperlipidemia | 23 (67.6) | |
| Smoker | 23 (67.6) | |
| Family History | 1 (2.9) | |
| **NGAL Level** | | |
| NGAL pre | | 248.7 ±162.7 |
| NGAL pre for UAP | | 278.1767 ± 178.37 |
| NGAL pre for SAP | | 178.08 ± 89.16 |
| Ureum | | 28.91 ± 13.79 |
| Creatinine | | 1.0 ± 0.21 |
| Contrast volume | | 137 + 47 |

**TABLE 2. SCI-AKI ACCORDING TO CHA2DS2-VASC-HSF SCORE**

| CHA2DS2-VASc-HSF *score* | N | SCI-AKI (+) | | (-) | | *p-value* |
|---|---|---|---|---|---|---|
| | | n | % | n | % | |
| ≥4 | 23 | 6 | 26.1 | 17 | 73.9 | |
| <4 | 11 | 3 | 27.3 | 8 | 72.7 | 0.942[2] |

[2]Chi square test

**TABLE 3. SCI-AKI ACCORDING TO EGFR**

| eGFR | N | SCI-AKI (+) | | (-) | | *p-value* |
|---|---|---|---|---|---|---|
| | | n | % | n | % | |
| ≥60 | 31 | 8 | 25.8 | 23 | 74.2 | |
| <60 | 3 | 1 | 33.3 | 2 | 66.7 | 0.778[2] |

[2]Chi square test

**TABLE 4. SCI-AKI ACCORDING TO CONTRAST VOLUME**

| eGFR | N | SCI-AKI (+) | | (-) | | *p-value* |
|---|---|---|---|---|---|---|
| | | n | % | n | % | |
| ≥150 | 31 | 8 | 16.7 | 23 | 83.3 | |
| <150 | 3 | 1 | 31.8 | 2 | 68.2 | 0.339[2] |

[2]Chi square test

## III. DISCUSSION

The results of this study indicate that the majority of patients (55.9%) were diagnosed with unstable angina pectoris (UAP). Patients with UAP undergoing percutaneous coronary intervention (PCI) are at risk of developing renal dysfunction. A study by Reyes et al. reported that serum NGAL levels of 281 mg/dL, measured 6 hours post-PCI, could predict the need for hemodialysis. Additionally, higher NGAL levels are observed in patients with acute coronary syndrome (ACS) due to myocardial injury. Research by Kafkas et al. found that serum NGAL levels were significantly higher in patients with UAP (108.00 ng/mL), NSTEMI (166.49 ng/mL), and STEMI (178.63 ng/mL) compared to patients with stable angina pectoris (SAP) (79.23 ng/mL) (Kafkas, 2012). In this study, the mean pre-PCI NGAL level was 248.7 ± 162.7 ng/mL. Research by Merdler et al. reported an optimal pre-PCI NGAL threshold of 129 ng/mL, suggesting that elevated NGAL levels indicate acute kidney injury (AKI).

Furthermore, Makris et al. demonstrated that plasma NGAL levels in healthy adults range between 28.7 and 167.0 ng/mL.[21]

The mean $CHA_2DS_2$-VASc-HSF score in this study was 4.02 ± 1.40. A study by Al-Khaleq et al. (2024) comparing coronary artery disease (CAD) severity with the $CHA_2DS_2$-VASc-HSF scoring system found that a cut-off value >3 had a sensitivity of 89% and specificity of 59% for predicting severe CAD.[1] Additionally, cut-off values of 5.5 and 4.5 for the $CHA_2DS_2$-VASc-HSF score have been identified as independent predictors of in-hospital mortality and major adverse cardiovascular events (MACE) within 30 days. This modification aims to provide a more comprehensive risk assessment, allowing for better-tailored management strategies based on individual risk profiles. Furthermore, this score has been linked to poor in-hospital prognosis in patients with non-ST-segment elevation myocardial infarction (NSTEMI). However, to date, no specific studies have directly correlated the $CHA_2DS_2$-VASc-HSF score with the degree of stenosis in stable coronary artery disease (SCAD) populations.[1,3,12]

Out of 34 study subjects, 9 subjects (26.5%) were diagnosed with subclinical contrast-induced acute kidney injury (SCI-AKI). The mean NGAL level in the SCI-AKI-negative group was -107.41 ± 135.32 ng/mL, while the SCI-AKI-positive group had a mean NGAL level of 174.50 ± 164.50 ng/mL. There was a statistically significant difference ($p < 0.05$) in NGAL levels between the two groups, highlighting the potential role of NGAL in identifying SCI-AKI. A study by Radias et al. on 21 patients undergoing PCI at Dr. M. Djamil General Hospital found a significant increase in plasma NGAL levels 6 hours post-contrast administration compared to baseline levels. This suggests that contrast media exposure during PCI is a major contributing factor to SCI-AKI.[8] Similarly, Filiopoulos (2013) reported that NGAL levels increased nearly tenfold at 6 hours post-contrast administration in the CI-AKI group, whereas no significant changes were observed in the non-CI-AKI group.[10] Baseline NGAL levels play a crucial role in interpreting changes, particularly in patients with eGFR <90 mL/min/1.73 m², as nephron mass affects the biological response to contrast-induced kidney injury. Petrova et al. demonstrated that serum creatinine levels increased at 24 hours post-coronary angiography and remained elevated for up to 48 hours. Additionally, plasma NGAL levels showed a rapid rise at 4 hours post-contrast administration, with a median value of 109.3 ng/mL, further increasing to 131.0 ng/mL at 24 hours. This indicates that NGAL is a sensitive biomarker for early kidney injury detection. Park et al. also found that NGAL elevation post-surgery could confirm the occurrence of SCI-AKI, reinforcing its role as an early indicator of subclinical renal injury.[21]

Among 23 subjects with a CHA2DS2-VASc-HSF score ≥4, 73.9% did not develop SCI-AKI. Similarly, in the group with a score <4, 72.7% did not develop SCI-AKI. The study found no statistically significant association ($p > 0.05$) between CHA2DS2-VASc-HSF score and SCI-AKI incidence. Although CHA2DS2-VASc-HSF is widely used to assess coronary artery disease severity and major adverse cardiovascular events (MACE) risk stratification, its role in predicting SCI-AKI remains unclear.[1,23] Some studies suggest that higher CHA2DS2-VASc-HSF scores are associated with risk factors such as hypertension, diabetes, and vascular disease, which contribute to kidney dysfunction. However, a direct correlation between high scores and SCI-AKI remains statistically insignificant ($p > 0.05$), likely due to the multifactorial nature of SCI-AKI. Other contributing factors include contrast volume, baseline renal function, and preventive strategies such as hydration and contrast agent selection.[1] These findings indicate that CHA2DS2-VASc-HSF may not

be a highly sensitive or specific predictor for SCI-AKI, though it remains valuable for overall cardiovascular risk assessment.

Most subjects with eGFR ≥60 mL/min/1.73 m² (74.2%) did not develop SCI-AKI, suggesting that preserved kidney function may offer protection against SCI-AKI. However, even among subjects with eGFR <60 mL/min/1.73 m², 66.7% did not develop SCI-AKI. The lack of a statistically significant association ($p > 0.05$) between eGFR and SCI-AKI suggests that eGFR alone may not be a strong standalone predictor for SCI-AKI. SCI-AKI is influenced by a combination of risk factors, including contrast volume, comorbidities (e.g., diabetes, hypertension), and preventive measures such as pre-procedural hydration. While low eGFR is a known risk factor for CI-AKI, several studies, including Andreucci et al., emphasize that SCI-AKI risk is highly dependent on interactions with other factors such as hydration status and contrast exposure.[1,23] Despite eGFR being a standard marker for kidney function, its predictive value for SCI-AKI remains limited unless additional risk factors are considered. This highlights the need for a comprehensive clinical approach that includes risk stratification and preventive interventions.[4-6,16,18-19]

Out of 10 subjects receiving ≥150 mL of contrast, 83.3% did not develop SCI-AKI. Similarly, 68.2% of subjects receiving <150 mL of contrast also did not develop SCI-AKI. No statistically significant relationship was found between contrast volume and SCI-AKI ($p > 0.05$). These findings align with previous research indicating that SCI-AKI risk is not solely determined by contrast volume but also by patient-specific factors such as baseline kidney function and hydration status.[1,8] Studies by Nijssen et al. suggest that while reducing contrast volume may lower SCI-AKI risk, it does not eliminate it entirely, particularly in patients with additional risk factors such as diabetes or hypertension.

Research by Solomon et al. further highlights that contrast volume only becomes a significant risk factor when combined with low eGFR, contrast agent type, and the absence of preventive strategies. Clinical interventions, such as intravenous hydration and the use of iso-osmolar contrast agents, remain crucial in mitigating SCI-AKI risk.[14,18-19,22]

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