SYSTEMATIC REVIEW

Association Between Systemic Inflammation Immun Index and In-stent Restenosis, Stent Thrombosis, and No Flow Phenomenon After Stent Implantation Was Examined in Systematic Review and Meta-analysis

Hotmauli Siahaan[1], Hari Basuki Notobroto[2], Yudi Her Oktaviono[3], Paulus Parholong Siahaan[4], Lidia Jane Tandra[6], Rizky Novita Anjaswanti[5], Lim Poh Ying[7], Zawiah Binti Mansor[7]

[1] Faculty of Public Health, Universitas Airlangga, Mulyorejo, Surabaya, East Java, 60115, Indonesia
[2] Department of Epidemiology, Biostatistics, Population Studies and Health Promotion. Faculty of Public Health, Universitas Airlangga, 60286 East Java, Indonesia
[3] Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Airlangga, 60286 East Java, Indonesia
[4] Faculty of Medicine, Universitas Airlangga, 60286 East Java, Indonesia
[5] Faculty of Public Health, Universitas Airlangga , 60286 East Java, Indonesia
[6] Faculty of Medicine, Universitas Hang Tuah, Ahmad Yani Street number 1, Surabaya, East Java, 60244, Indonesia
[7] Department of Community Health, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor Darul Ehsan, Malaysia

ABSTRACT

Introduction: The systemic inflammatory index (SII) is a potential predictor for cardiovascular diseases. High SII may reflect a greater inflammatory response, which may worsen neointimal hyperplasia and increase the risk of restenosis. **Materials and methods:** This study employed a systematic review and meta-analysis design, comply the PRISMA 2020 guidelines. A search was conducted on several global scientific databases, such as PubMed, Science Direct, Google Scholar, Springer, and Proquest. **Results:** This study analyzed 10 studies with the total of 5.237 patients were included. The results revealed a significant association between a high SII value and intra-stent restenosis (OR= 4.94, 95% CI 4.23 – 5.77; p < 0.001). stent thrombosis (OR= 3.82, 95% CI 2.25 – 6.48; p < 0.001) no-reflow phenomenon (OR= 4.59, 95% CI 3.83 – 5.51; p < 0.001). **Conclusion:** There is a robust correlation between the SII value and intra-stent restenosis, stent thrombosis, and the no-reflow phenomenon incidents, particularly at elevated SII levels.
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Keywords: Intra-stent restenosis, No-reflow phenomenon, Systemic inflammatory index, Stent thrombosis

Corresponding Author:
Hotmauli Siahaan, PhD
Email: hotmauli.siahaan-2023@fkm.unair.ac.id
Tel : (+62)81331521186

INTRODUCTION

Percutaneous coronary intervention (PCI) is the primary approach for treating patients with acute coronary artery disease (CAD). In spite of advancements technology of stent and anti-platelet therapy, intra-stent restenosis (ISR) continues to be a significant concern. Previous studies have indicated the overall occurrence of ISR in majority of studies can be as high as 10% within five years after PCI (1,2). In-Stent Restenosis is the re-narrowing of more than 50% of the blood vessel after stenting. Studies have investigated risk factors for ISR following stent placement. An observational study done by Zhang et al. (2022), found various risk factors that might cause ISR, from the clinical aspect such as diabetes, hypertension, discontinuation of aspirin, and family history, and mechanical or anatomy factors such as calcified lesions, number of segmented stent, stent diameter, and stent length(3).

In the last few years, there has been a notable increase in interest in using the systemic immune-inflammatory index (SII) as a potential predictor for cardiovascular disease. SII, which works based on platelets, neutrophils, and lymphocytes, has demonstrated significant predictive value in heart failure, stroke, and coronary artery disease, highlighting its relevance in cardiovascular disease. (4,5). SII plays a pivotal role in the pathological process of atherosclerosis. Several inflammatory markers based on circulating blood cell counts, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune inflammation index (SII) have been related to the severity and prognosis of coronary artery disease (CAD) (8)

A study conducted by Jin et al. discovered a greater SII levels were linked to a higher risk of progression

cardiovascular disease over a 10-year follow-up period. Additionally, a cohort study revealed a relevant association between fluctuating SII levels and the risk of cardiovascular disease (6). Recent study also indicates that increased SII levels are independently associated with an elevated risk of intra-stent restenosis (ISR) following percutaneous coronary intervention (PCI) in patients with coronary artery disease and may function as a predictor for ISR (7).

Stent thrombosis (ST) is a critical complication that may arise after percutaneous coronary intervention (PCI), potentially resulting in acute myocardial infarction and sudden death. The occurrence rate is about 2.4%, and the mortality rate can reach as high as 34% (9). Stent thrombosis following coronary artery stenting presents a significant hazard, with fatality rates ranging from 5% to 45%. Factors such as smoking, diabetes, malignant tumors, and anemia have been identified as contributors to this condition, underscoring the need for specific prevention strategies. (10,11).

Previous attempts to predict stent thrombosis using white blood cells and C reactive protein have shown limited sensitivity and specificity. However, a study has found a correlation between the systemic inflammatory index, indicating its potential relevance to the progress of stent thrombosis. The systemic inflammatory index holds promise as an sign of inflammation severity and its connection to stent thrombosis, given the established link between stent thrombosis and inflammation (12,13).

In the last few years, there has been a notable increase in interest in using the systemic immune inflammatory index (SII) as a potential predictor for cardiovascular disease. Although previous studies suggest a correlation between SII and ISR, stent thrombosis, and the no-reflow phenomenon (NFP), a comprehensive meta-analysis is lacking.

MATERIALS AND METHODS

Study Design

This study utilized a systematic review and meta-analysis design, complying with PRISMA 2020 guidelines. PRISMA, it assists authors and researchers in producing high-quality systematic reviews and meta-analyses by providing a checklist that details the crucial elements to incorporate and comprehensively explain in such studies.

Eligibility Criteria

In this study, the inclusion criteria were: (1) Study design: Retrospective or prospective cohort studies; (2) Study population: patients undergoing implantion stent; (3) The main outcomes of the study consisted of intra-stent restenosis, stent thrombosis, and the no-reflow phenomenon. Meanwhile, the study has the following exclusion criteria: (1) Cross-sectional studies, reviews, case study; (2) Research that fail to provide outcome indicators for intra-stent restenosis, stent thrombosis, and the no-reflow phenomenon after SII grouping; (3) The research was not conducted on animals; (4) Did not use literature other than English. The study selection process was performed by three investigators (HS, RNA, LJT).

Data Extraction

The following data: age (years), sex (male), hypertension status, diabetes, smoking, and dyslipidemia of patients, were extracted from the included studies, representing low SII and high SII, respectively. In this section, the content is related to the extracted data, which consists of three extracted data, namely intra-stent restenosis, stent thrombosis, and the no-reflow phenomenon with the systemic inflammatory index. This compliance with the standard for meta-analysis.

Literature Search

The meta-analysis comprised an extensive literature search using MeSH terms (see Supplementary Table) of studies registered in the PROSPERO database (ID 583179), renowned for its systematic reviews and meta-analyses in human health. The study encompassed global scientific including PubMed, databases, Google Scholar, Science Direct, Proquest, and Springer. The searches continued until August 21, 2024, to identify publications comparing patients with increase and decrease systemic inflammatory index.

We utilized different search terms combined with logical operators, such as “In – Stent Restenosis” OR “stenosis intra stent” AND “thrombosis intra stent” OR “stent thrombosis” AND “Systemic inflammation index” OR “systemic immune inflammation index.” The conceptual framework outlining the literature search methodology is presented in Figure 1.

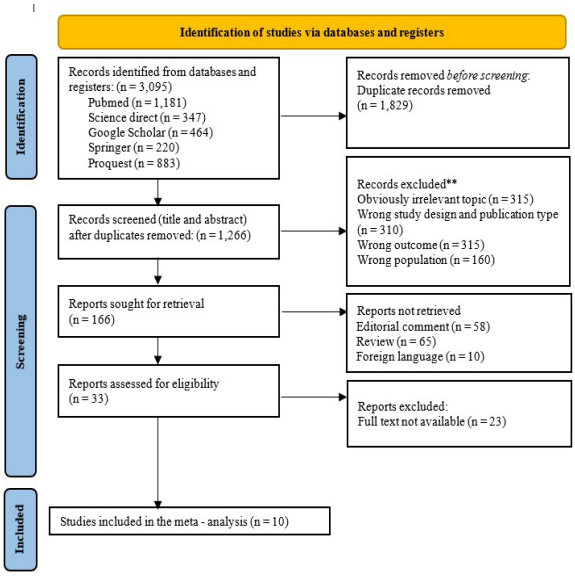

Figure 1: Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) Flow Diagram of Study Selection

Statistical and Sensitivity Analysis

The systematic review and meta-analysis process included extracting secondary data from various databases to address specific research queries. We employed the Review Manager 5.4 software to perform the statistical analyses. The Mantel-Haenszel method was utilized to ascertain dichotomous data, whereas the inverse variance method was used for continuous data like body mass index and stent length. Meanwhile, the DerSimonian and Laird random-effect model was used to evaluate heterogeneity. The data from the median and interquartile range were transformed into mean and standard deviation. Next, we established a sensitivity analysis with a leave-one-out approach to detect outliers and changes in heterogeneity (I2), with I2 values falling into the low (0-50%), moderate (50-75%), and substantial (76-100%) heterogeneity categories. Data analysis incorporated fixed and random effect models, with significance determined by a p-value of below 0.05. The meta-analysis entailed a thorough literature search.

RESULTS

Baseline Characteristics

There were 10 cohort studies on Systemic inflammatory index with intra-stent restenosis, stent thrombosis, and no-reflow phenomenon being analyzed, the total of 5.237 patients were included (14–22). Patients in the studies low Systemic inflammatory index had a mean age of 62.7 years, and 71,39% of patients were male 54.36% had hypertension, 36.51% had diabetes, 41.36% were active smokers or had a smoking history, and 36.99% had dyslipidaemia. In the high Systemic inflammatory index, the mean age was 61.58 years old, and 75% of patients were male, 56.10% of patients had hypertension, 41.31% had diabetes, 44.17% were active or former smokers, and 41% have dyslipidaemia. Supplementary Table I contains detail information on population characteristic.

Table I: Baseline Characteristics Low and High Systemic Immune Inflammation index

Studies	Mean Age (years)	Sex (Male)	HTN (%)	D (%)	SM (%)	DL (%)
LOW SYSTEMIC IMMUNE INFLAMMATION INDEX						
			IN – STENT RESTENOSIS			
Osken et al. 2024 [13]	55.8	67.8	46.8	29.6	44.4	35.0
Tian et al. 2023 [14]	67.2	68.8	55.8	34.4	46.1	29.2
Xie et al. 2024 [6]	64.99	80.0	25.14	32.0	53.18	6.86
Xu et al. 2024 [15]	61.98	68.7	65.9	48.4	24.2	N/A
			STENT THROMBOSIS			
Dhilion et al. 2024 [16]	57.25	86.3	N/A	N/A	N/A	N/A
Zheng et al. 2023 [17]	N/A	70.37	66.67	44.44	N/A	88.89
			NO – REFLOW PHENOMENOM			
Cakmak et al. 2024 [18]	62	73	67	44	43	N/A
Karauzum et al. 2022 [19]	58.1	53.3	41.3	21.7	42.3	25.0
Ozen et al. 2023 [20]	67	77.9	64	46	34	N/A
Wang et al. 2022 [21]	70.02	67.7	56.7	28.1	43.7	N/A
HIGH SYSTEMIC IMMUNE INFLAMMATION INDEX						
			IN – STENT RESTENOSIS			
Osken et al. 2024 [13]	56.1	72	50.6	29.6	44.4	36.9
Tian et al. 2023 [14]	68.5	78.7	63.2	40.0	56.1	36.8
Xie et al. 2024 [6]	66.25	71.84	11.49	30.46	44.12	2.30
Xu et al. 2024 [15]	61.27	64.4	81.5	55.6	22.2	N/A
			STENT THROMBOSIS			
Dhilion et al. 2024 [16]	57.25	86.3	N/A	N/A	N/A	N/A
Zheng et al. 2023 [17]	N/A	70.37	66.67	44.44	N/A	88.89
			NO – REFLOW PHENOMENOM			
Cakmak et al. 2024 [18]	55.5	88	57	47	42	N/A
Karauzum et al. 2022 [19]	55.7	74.3	43.8	36.2	61.0	35.2
Ozen et al. 2023 [20]	64	73.6	60	60	29	N/A
Wang et al. 2022 [21]	69.67	70.7	70.7	28.5	54.5	N/A

Systemic Immune Inflammation Index with In – Stent Restenosis

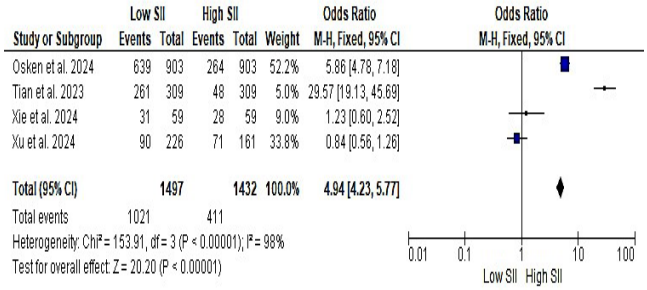

Figure 2: Forest plot for a high systemic immune inflammation index with in – stent restenosis

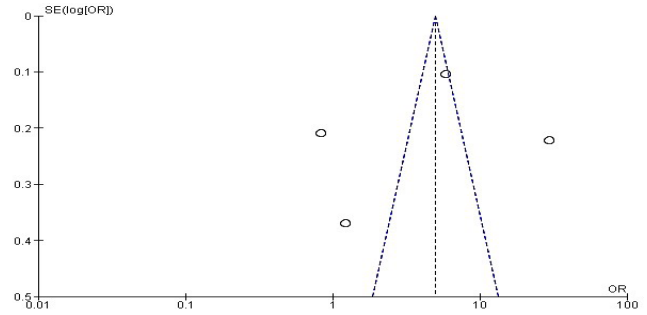

Figure 3: Funnel plot for a high systemic immune inflammation index with in – stent restenosis

Four studies indicated the relationship between systemic inflammatory index and intra-stent restenosis. The forest plot results above showed that patients in the increase systemic inflammatory index group had a greather risk of intra-stent restenosis compared to those in the low SII group (OR= 4.94 95%CI 4.23 – 5.77; p<0.001; I2 = 98%, Figure 2). These results show that high SII patients are 4.94 times more likely to develop ISR than low SII patients. The funnel plot image shows a symmetrical distribution of studies, where the distribution of studies is balanced on the left and right of the midline boundary. So it can be concluded that there is no publication bias that affects the meta-analysis of high systemic immune inflammation index with intra-stent restenosis (Figure 3).

Systemic Immune Inflammation Index with Stent Thrombosis

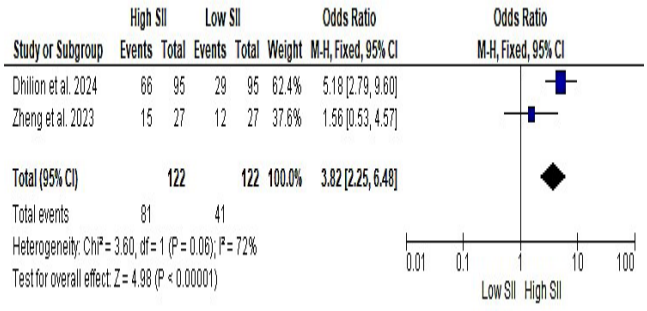

Figure 4: Forest plot for a high systemic immune inflammation index with stent thrombosis

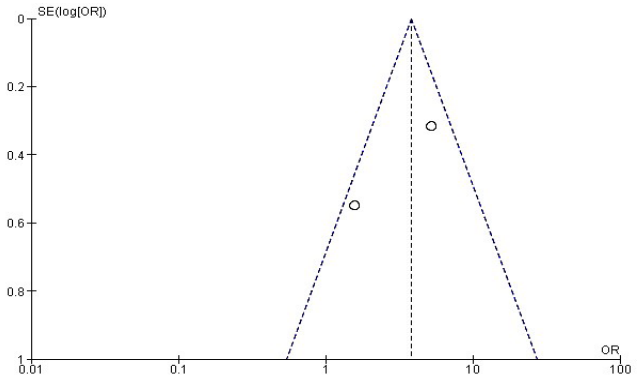

Figure 5: Funnel plot for a high systemic immune inflammation index and stent thrombosis

Two studies reported the correlation between systemic inflammatory index and stent thrombosis. The forest plot results above showed that patients in the high systemic inflamatory index group had a higher risk of stent thrombosis compared to those in the low systemic inflammatory index group (OR = 3.82 95%CI 2.25 - 6.48; p <0.001; I2 = 72%, Figure 4). These results show that patients with a high SII value have a 3.82 times higher risk of developing a stent thrombosis compared to patients with a low SII value. The funnel plot image shows a symmetrical distribution of studies, where the distribution of studies is balanced on the left and right of the midline boundary. So it can be concluded that there is no publication bias affecting the meta-analysis of high systemic immune inflammation index with stent thrombosis (Figureure 5).

Systemic Immune Inflammation Index with No Reflow Phenomenom

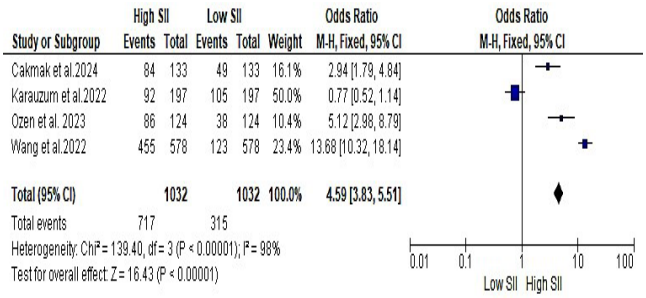

Figure 6: Forest plot for a high systemic immune inflammation index with no – reflow phenomenom

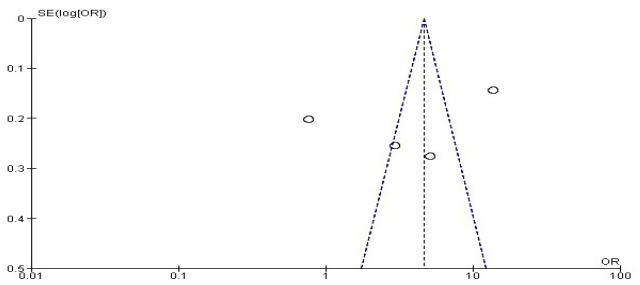

Figure 7: Funnel plot for a high systemic immune inflammation index and no-reflow phenomenom

Two studies reported the correlation between systemic inflammatory index and stent thrombosis. The forest plot results above showed that patients in the high systemic inflammatory index group had a higher risk of stent thrombosis compared to those in the low systemic immune inflammation index group (OR = 4.59 95%CI 3.83 – 5.51; p <0.001; I2 = 98%, Figureure 5). These results show that patients with high SII values are 4.59 times more likely to develop no-reflow compared to patients with low SII values. The funnel plot image shows a symmetrical distribution of studies, where the distribution of studies is balanced on the left and right of the midline boundary. So it can be concluded that there is no publication bias that affects the meta-analysis of high systemic immune inflammation index with no-reflow phenomenon (Figure 7).

DISCUSSION

Earlier research has highlighted that ongoing inflammation poses a considerable risk for multiple conditions, including cancer, diabetes, and atherosclerotic disease (23). The systemic inflammatory index (SII) is a novel biomarker that assesses systemic inflammation by analyzing the counts of platelets, neutrophils, and lymphocytes (24).

In our research exhibits a strong correlation between the systemic inflammatory index and intra-stent restenosis. The research determined that the SII is capable of accurately forecasting the incidence of ISR. This finding aligns with Faysal Saylik's study "Systemic Immune-Inflammation Index Predicts Major Cardiovascular Adverse Events in Patients with ST-Segment Elevated Myocardial Infarction". The study showed that a increase SII was independently associated with adverse major cardiovascular events in patients with acute coronary syndrome following Primary PCI, such as nonfatal MI, cardiac death, nonfatal stroke, and hospitalization for heart failure (25).

Furthermore, a study by Mustafa Candemir investigated the correlation between the Systemic Inflammatory Index (SII) and the severeness of coronary artery disease. The research found that the systemic inflammatory index (SII) is a contributing factor to atherosclerosis and may provide a more accurate prediction of coronary artery lesion severity compared to ratios such as NLR, PLR, and MLR (26). Additionally, a study review conducted by Huang et al. explore the relationship between the systemic inflammatory index (SII) and clinical outcomes in patients who have experienced a stroke. The study revealed that the post-stroke inflammatory response is crucial for immune-regulatory treatment. It was recommended that patients who have experienced a stroke with increased SII levels should be conscientinously monitored, as this could lead to a potential approach for managing brain damage following a stroke (27).

Silvia Lee investigated the associations of PLR, NLR, and LMR with TVR following elective infrainguinal angioplasty with stent implantation for symptomatic PAD. High PLR and NLR were linked to an increased risk of TVR over two years urak et al. also in his study found a correlation between NLR and intra-stent restenosis following percutaneous coronary intervention (PCI) with bare metal stent implantation In their study, an NLR >2.73 predicted TVR with a sensitivity of 80% and a specificity of 75%. Therefore, their cut-off was almost simillar to the threshold for high NLR obtained in our study. Since all patients in both studies received bare metal stents, it remains to be established if NLR can predict TVR after drug-eluting stent implantation (30).

Silvia Lee explored the relationships between PLR, NLR, and LMR concerning TVR after elective infrainguinal angioplasty with stent insertion for symptomatic PAD. Elevated levels of PLR and NLR were associated with a heightened risk of TVR over a two-year period (28). Turak et al. also found a relationship between NLR and intra-stent restenosis after percutaneous coronary intervention (PCI) (29). In their research, an NLR greater than 2.73 was shown to predict TVR with 80% sensitivity and 75% specificity. Consequently, their cut-off was quite similar to the threshold for elevated NLR determined in our study.

This study found that a high systemic inflammatory index value is significantly correlated with stent thrombosis. Another study shows that SII is not correlated with thrombosis but with hemorrhage. Wang et al. found that the inflammatory index value can forecast results in individuals suffering from intracerebral hemorrhage, where a higher level of systemic inflammatory and immune index was independently associated associated with an increased likelihood of infections and a poor prognosis over the next three months. (31).

Ayca et al. investigated other inflammation markers related to stent thrombosis. They discovered that a higher neutrophil-to-lymphocyte ratio (NLR) serves as an inflammatory marker that can forecast stent thrombosis and is linked to increased mortality rates in STEMI patients (32). Our study shows that a high systemic immune inflammation index value significantly correlates with the no-reflow phenomenon. Ilzen et al. obtained comparable findings, revealing that the SII It is an independent predictor that can be calculated easily from a standard blood test to forecast the development of no-reflow, which is a complication after PCI in patients with saphenous vein interventions (33).

There are several reasons for high heterogeneity. First, variations in the patient population, such as differences in age, comorbidities (diabetes, hypertension), or type of coronary artery disease, i.e., acute coronary syndrome or chronic heart disease. Second, variations in high and low SII cut-offs, where there are differences

across studies. Future research needs meta-regression to evaluate factors contributing to heterogeneity (34).

Limitation

The robustness of the research is constrained by the limited number of available studies. Consequently, additional research is needed to enhance the reliability and precision of the research outcomes.

CONCLUSION

There is a strong association between the systemic inflammatory index (SII) value and the occurrence of in-stent restenosis, stent thrombosis, and the no-reflow phenomenon, especially at increased levels of SII. This research indicates that elevated SII values can serve as predictors for ISR, stent thrombosis, and the no-reflow phenomenon.

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