



Can the Systemic Immune-Inflammatory Index Predict Nosocomial Infection in Term Newborns Who Underwent Congenital Cardiac Surgery?

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ABSTRACT

Objectives: In this study, we aimed to investigate the contribution of the early systemic immune-inflammatory index (SII) and acute-phase reactants in predicting nosocomial infections in term newborns who underwent congenital heart surgery.

Methods: This retrospective study was conducted in neonates who underwent cardiopulmonary bypass (CPB) surgery for congenital heart disease (CHD) between November 1, 2021, and December 1, 2022, and were followed in the pediatric cardiac intensive care unit. Demographic and clinical characteristics, as well as changes in the systemic inflammatory index (platelet count \times neutrophil/lymphocyte count) and acute-phase reactants during the preoperative period and the first 72 postoperative hours, were evaluated in patients with and without nosocomial infection. The results were statistically analyzed.

Results: This study included 160 neonatal patients. The median age was 10 days (IQR, 6–15 days), and the median weight was 3 kg (IQR, 2.8–3.2 kg). Eighty patients were male (50%). Fifty-five different nosocomial infections were identified in 44 patients (27.5%). Bloodstream infections were the most common (62%), followed by lower respiratory tract infections (23%) and wound infections (15%). Mortality due to nosocomial infections was 34%. SII and NLR values measured on postoperative days 2 and 3 were significantly higher in patients with nosocomial infections ($p<0.05$). An SII value >510 (72% specificity, 85% sensitivity) on postoperative day 2 and >730 (72% specificity, 80% sensitivity) on postoperative day 3 were highly predictive of nosocomial infection.

Conclusions: Nosocomial infections are an important cause of mortality and morbidity in neonates undergoing congenital heart surgery. An easy-to-use systemic inflammatory index measurement may help predict nosocomial infections.

Keywords: Congenital heart disease, newborn, nosocomial infections, systemic inflammatory index

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Introduction

Congenital heart disease (CHD) is a common disease with a general incidence of 4 to 10 per 1000 live births. In the neonatal period, 25% of cases require surgical and/or interventional palliative and corrective procedures due to the critical nature of the heart disease.^[1]

During the neonatal period, the immaturity of the immune system, the uncontrolled systemic inflammatory response

arising from cellular and humoral interactions during cardiopulmonary bypass, and the inherent complexity of the surgical procedure collectively predispose patients to a range of organ system complications. Infection represents one of the most significant complications observed following neonatal cardiac surgery, with some studies reporting an incidence ranging from 20% to 50%. Catheter-related bloodstream infections, ventilator-associated pneumonia (VAP), nosocomial pneumonia, urinary tract

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infections, and surgical site infections represent the primary sources of nosocomial infection in this population.^[2-4]

Early diagnosis and management of nosocomial infections in neonates may be hindered by the challenges of identifying clinical manifestations in the postoperative period and by the confounding physiological effects of cardiopulmonary bypass. Prompt diagnosis and timely initiation of appropriate antibiotic therapy are critical, as delays can significantly worsen outcomes. Since waiting for blood culture confirmation may be impractical, clinical judgment supported by laboratory parameters should guide early therapeutic decisions. In this specific period of life, the availability of biomarkers with high diagnostic accuracy, noninvasive and easily applicable attributes is also limited. Various tests such as complete blood count, C-reactive protein, procalcitonin, and neutrophil-lymphocyte ratio can be used for this purpose.^[5,6] Systemic immune-inflammation index (SII) has recently been used as a new index that comprehensively reflects the state of inflammation and immune balance in the body.^[7] SII can be obtained from a routine complete blood count.

In this study, we aimed to evaluate whether the systemic immune-inflammation index (SII) and acute-phase reactants, measured preoperatively and within the first 72 hours postoperatively, are predictive of nosocomial infections in neonates undergoing cardiac surgery in the pediatric cardiac intensive care unit.

Methods

This single-center retrospective study was conducted at Basaksehir Cam and Sakura City Hospital between November 2021 and December 2022. Medical records of 188 neonates who underwent cardiopulmonary bypass (CPB) surgery for congenital heart disease (CHD) were reviewed. Patients were excluded if they underwent surgery without CPB (n=16), had an infection within 48 hours of hospitalization (n=10), or had incomplete data (n=2). After applying these criteria, 160 cases were included in the final analysis and categorized into two groups: those with nosocomial infections (n=44) and those without (n=116). Following approval by the Basaksehir Cam and Sakura City Hospital Ethics Committee (Number: KAEK/17.12.2025.450), the study was conducted in strict accordance with the ethical principles outlined in the Declaration of Helsinki.

Neonates diagnosed with congenital heart disease by preoperative cardiac examination and echocardiography by a pediatric cardiologist and confirmed by cardiac CT or catheterization who underwent cardiac surgery during the neonatal period were included. Exclusion criteria were death within 48 hours postoperatively, infection within the first 48 hours of hospitalization (evidenced by fever, elevated C-reactive protein, white blood cell count, procalcitonin, or infectious lesions on physical examination or chest X-ray), surgery without cardiopulmonary bypass (CPB), transfer to another unit, prematurity, or incomplete clinical data (Fig. 1).

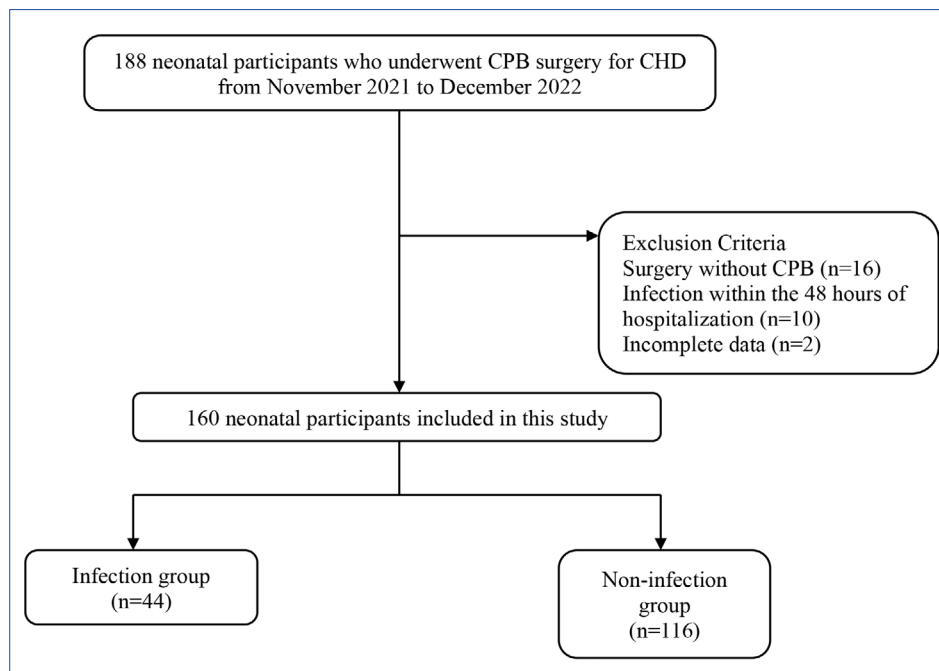


Figure 1. The flow chart of case inclusion study.

CPB: Cardiopulmonary bypass; CHD: Congenital heart disease.

All study data were obtained from the hospital's electronic medical record system. The onset of nosocomial infection occurred between 72 hours after surgery and the date of discharge from the intensive care unit. The study population was stratified into two groups: patients who developed nosocomial infections and those who did not. Nosocomial infections were defined in accordance with the criteria established by the Centers for Disease Control and Prevention (CDC) and the National Healthcare Safety Network (NHSN).^[8] The following infections were included: catheter-related bloodstream infection, clinical sepsis, nosocomial pneumonia, ventilator-associated pneumonia (VAP), urinary tract infections, and surgical site infection. Sepsis was defined in accordance with the criteria proposed by the International Sepsis Forum for infections in the intensive care unit setting.^[9]

All pediatric patients received prophylactic antibiotics prior to surgery to reduce the risk of infection. Cefazolin was administered as the first-line agent, with clindamycin used as an alternative in patients with documented cephalosporin allergy. A single intravenous dose was given within 30–60 minutes before the surgical incision, and prophylaxis was continued for up to 72 hours postoperatively. CPB was conducted under moderate (core temperature 28°C) to mild (core temperature 32°C) hypothermia. Size-adapted bypass circuits and membrane oxygenators (FX05 oxygenators) were used. The total priming volume for the bypass circuit was 250 mL, consisting of packed red blood cells (PRBCs), fresh frozen plasma (FFP), Isolyte-S solution, mannitol, sodium bicarbonate, tranexamic acid, prednol, and antibiotic. PRBCs were transfused to maintain the hematocrit at 28–32% during CPB. Myocardial protection was achieved with cold intermittent blood cardioplegia (20 mL/kg), prepared during CPB by mixing buffered del Nido solution (Isolyte S) with whole blood from the arterial line at a ratio of 1:4. The cardioplegia solution was kept at 6°C before infusion. MUF was performed following weaning from CPB and 15–20 minutes before protamine administration, with a target hematocrit of 35–40%. Following termination of CPB, PRBC transfusion was initiated to reach a target hematocrit of 35–40% for non-cyanotic patients and 40–45% for cyanotic patients.

Surgical hand antisepsis procedures were performed by the practitioner, who wore a mask, cap, sterile gloves, and gown. A large sterile drape was applied to the field. Entry sites were swabbed twice with 10% povidone-iodine solution and allowed to dry before catheter insertion. All neonates received a 4 Fr, 5 cm double-lumen catheter, and the procedure was routinely performed under ultrasound guidance. Catheter site selection was based on the practitioner's preference. Intensive care dressings

for catheters were changed every two days or sooner if required. Catheter and dressing materials containing self-antiseptics were not used.

An infection control team consisting of physicians and nurses evaluated all patients daily. Surveillance included periodic tracheal aspiration, blood, and urine cultures. In cases of clinical sepsis, broad-spectrum antibiotics and pentoxifylline infusion (5mg/kg/h over 4 hours) were initiated. The antibiotic regimen was subsequently modified according to culture results and antibiogram findings.

Patient data were collected using a standardized form, including demographics, perinatal history, catheter and postoperative characteristics, surgery type, laboratory results, and isolated microorganisms. Peripheral venous blood samples were collected from all cases 24 hours before and on postoperative days 1, 2, and 3. The samples were sent to the laboratory for routine examination. White blood cell (neutrophil-lymphocyte) count (WBC), platelet count, albumin, C-reactive protein (CRP), and procalcitonin levels were determined. The systemic immune-inflammation index (SII) was calculated for each patient using the formula: $SII = (\text{neutrophil count} \times \text{platelet count}) / \text{lymphocyte count}$.

^[10] All calculated values were subsequently recorded and included in the statistical analyses.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were described as mean±SD or median (IQR) according to their distribution, and categorical data were presented as frequencies (%). t-tests or Mann-Whitney U tests were used to compare groups for continuous variables, and chi-squared tests or Fisher's exact tests were used for categorical data, as appropriate. Receiver operating characteristic (ROC) curve analysis and calculation of the area under the curve (AUC) were performed to determine the optimal cut-off value of SII for predicting nosocomial infection. The final model for estimation of SII parameters affecting nosocomial infection was expressed as an odds ratio with a 95% confidence interval. $p < 0.05$ was considered statistically significant.

Results

This study included 160 newborns. Nosocomial infections occurred in 44 patients (25.7%). Demographic characteristics of neonates with and without nosocomial infections are shown in Table 1.

Fifty different nosocomial infections were identified. Thirty-one of these cases had only one infection, eleven had two infections, and two cases had three infections. Bloodstream infections were the most common in 62% (sepsis $n=22$,

Table 1. General characteristics of the newborns included in the study

Parameters	Nosocomial infection (+) n=44		Nosocomial infection (-) n=116		p
	n	%	n	%	
Operation age (day)	5 (3–9)		6 (4–8)		NS
Weight (gram)	2950 (2800–3100)		3100 (3000–3200)		NS
Male	21	48	59	51	NS
APGAR score					
1'	7 (6–8)		7 (6–8)		NS
5'	8 (7–9)		9 (8–10)		NS
Cesarean section	22	50	61	53	NS
Syndrome	11	25	5	4	0.01
STAT (≥ III)	40	92	105	90	NS
Cyanosis	25	58	70	60	NS
Single ventricle	13	30	41	35	NS
Urgent surgery	5	11	10	9	NS
Mechanical ventilation before surgery	11	25	28	24	NS
Duration of Cardiopulmonary bypass (minutes)	130 (110–150)		110 (100–120)		0.030
Aortic clamping time (minutes)	70 (60–80)		60 (50–70)		0.045
Temperature during surgery (°C)	36.2 (35.5–37)		36 (35.5–36.5)		NS
Delayed sternal closure	11	25	21	18	NS
Intensive Care stay (day)	10 (6–14)		7 (5–10)		0.003
Mortality	15	34	11	10	0.001

Data presented as n (%) or median (interquartile range). STAT: The Society of Thoracic Surgeons-European Association for Cardio-Thoracic Surgery.

catheter-associated bloodstream infection n=12), followed by lower respiratory tract infections in 23% (ventilator-associated pneumonia n=9, pneumonia n=4), and wound site infections in 15% (n=8).

Of the infectious agents detected, 55% were Gram-negative organisms, 30% were Gram-positive organisms, and 15% were fungal organisms. The most common pathogens detected in all three blood, lung, and wound swab cultures were Gram-negative organisms. The infections detected and their locations are shown in Table 2. The overall mortality was 16% (n=26/140). In 15 cases, the main cause of death was NI (15/44, 34%). The higher mortality in patients with nosocomial infections (p=0.01) was attributable to the infections.

The results of preoperative and postoperative 1st, 2nd, and 3rd day hemogram and acute-phase reactants in cases with and without nosocomial infections are shown in Table 3. According to these results, NLR and SII values were found to be significantly high in patients with nosocomial infections (p<0.05). Patients with nosocomial infections tended to show elevated NLR and SII values postoperatively, particularly on the 2nd and 3rd days (Fig. 2). Postoperative 2nd day SII c-index=0.80 (CI: 0.72–0.88, p=0.02), postoperative 3rd day NLR c-index=0.78 (CI: 0.66–0.88, p=0.04), and

postoperative 3rd day SII c-index=0.80 (CI: 0.72–0.88, p=0.001) could predict nosocomial infections.

The presence of congenital syndromes was identified as an independent risk factor associated with nosocomial infection (OR=1.8, 95% CI: 1.2–8). Cut-off points were postoperative 2nd day SII>510 (72% specificity, 85% sensitivity), postoperative 3rd day SII>730 (72% specificity, 80% sensitivity), and postoperative 3rd day NLR>3.8 (70% specificity, 70% sensitivity), which predicted nosocomial infection.

Discussion

This study evaluated the predictive value of the systemic immune-inflammation index (SII) and acute-phase reactants for the early detection of nosocomial infections following CPB in neonates undergoing cardiac surgery. The incidence of nosocomial infection in our cohort was 27.5%, aligning with previously reported rates.^[11,12] Importantly, we found that elevated SII levels on postoperative days 2 and 3 were significantly associated with the development of nosocomial infections, suggesting that SII may serve as an early, noninvasive, and readily accessible indicator of infection risk. To our knowledge, this study is among the few to explore and highlight the utility of SII in predicting nosocomial

Table 2. Distribution of microorganisms isolated in newborns with nosocomial infection

	Blood culture	Tracheal aspirate	Needle aspiration of wound	Total
Gram-negative	14	7	3	24
<i>Acinetobacter baumannii</i>	-	3	-	3
<i>Enterobacter cloacae</i>	1	1	-	2
<i>Escherichia coli</i>	2	-	-	2
<i>Klebsiella pneumoniae</i>	7	1	2	10
<i>Pseudomonas aeruginosa</i>	3	1	1	5
<i>Serratia marcescens</i>	1	1	-	2
Gram-positive	10	1	3	14
<i>Enterococcus faecium</i>	2	-	-	2
<i>Staphylococcus aureus</i>	3	1	1	5
<i>Staphylococcus epidermidis</i>	4	-	2	6
<i>Staphylococcus hominis</i>	1	-	-	1
Yeast	5	-	1	6
<i>Candida albicans</i>	4	-	1	5
<i>Candida parapsilosis</i>	1	-	-	1
Total	29	8	7	44

infections in neonates, contributing novel insights to a growing body of literature focused on inflammation-based biomarkers in pediatric cardiac intensive care.

Nosocomial infection is a serious morbidity that can lead to fatal complications after cardiac surgery for congenital heart disease and is even more critical in neonates. The incidence of nosocomial infection in patients with CHD has been reported at different rates. Yu et al.^[13] reported a nosocomial infection rate of 10.8% for all patient groups and 32.9%, 15.4%, and 5.2% for neonates, infants, and children, respectively. In another study, the rate of nosocomial infection in neonates was 25.3%, which was five times higher than in other age groups.^[14] Our incidence of nosocomial infection was 27.5%, which was consistent with the literature.

The most common postoperative complications in neonates with CHD requiring surgery are infectious complications. Bloodstream, lower respiratory tract, wound site, and urinary tract infections are major sites of postoperative infection. Pasquali et al.^[15] reported sepsis, wound infection, and pneumonia as the most common sites of nosocomial infection, accounting for 51%, 35%, and 10% of patients, respectively. Similarly, we identified bloodstream infections as the most common type of nosocomial infection.

Mortality was higher in patients with nosocomial infections. Magliola et al.^[16] and Garcia et al.^[3] reported 14% and 17.8% mortality rates, respectively. In our study, the mortality rate was significantly higher in patients with nosocomial infections (34%) compared with those without infections (10%). This elevated mortality may

be partly attributable to the complexity of underlying pathologies, as suggested by previous studies.

Systemic inflammatory response syndrome (SIRS), which can negatively impact neonatal mortality and morbidity, can be triggered by surgical trauma or CPB during cardiac surgery or in the early postoperative period. Contact of CPB equipment with blood cells initiates the inflammatory cascade with the release of cytokines and activation of the complement and coagulation systems. In this complex process, hypothermia and ischemia-reperfusion injury further impair tissue oxygenation, resulting in hemodynamic deterioration. Several biomarkers are used to diagnose and manage this process. However, none of them is easily obtainable, reproducible, or sufficiently useful in clinical practice.^[17,18] CRP is one of the most reliable parameters for the detection of nosocomial sepsis, with its level increasing significantly during infection, especially in Gram-negative bacterial sepsis.^[19] Some of the readily available parameters, such as platelet/lymphocyte ratio and neutrophil/lymphocyte ratio (NLR), calculated from routine complete blood count, have been studied as potential indicators of infection. These ratios have been suggested to be potentially more sensitive biomarkers of inflammation than the absolute levels of individual blood cell components.^[20]

Nowadays, NLR is accepted as a more valuable marker of inflammation than lymphocytopenia or neutrophilia alone, especially for the detection of bacterial infections.^[21,22] NLR and SII have been used to predict disease activity, prognosis, and survival in diseases with systemic inflammation, particularly in many clinical cancer scenarios, hepatocellular

Table 3. Changes in hemogram parameters in nosocomial infection

Variable	Nosocomial infection (+)	Nosocomial infection (-)	p
Preop Hemoglobin	13 (11–14)	13.3 (11.5–14.5)	NS
Preop Hematocrit	41(36–45)	43 (39–46)	NS
Preop Platelet	294 (170–450)	269 (160–350)	NS
Preop Neutrophil	6250 (4000–12000)	5900 (3700–9100)	NS
Preop Lymphocyte	3200 (2250–5750)	3500 (2950–6400)	NS
Preop NLR	2.1 (0.9–2.5)	1.8 (1.2–2)	NS
Preop CRP	0.1 (0.1–3)	0.1 (0.1–3)	NS
Preop Procalcitonin	0.08 (0.05–0.1)	0.10 (0.05–0.5)	NS
Preop SII	620 (500–750)	590 (480–600)	NS
1 st day Hemoglobin	13.4 (10–14)	13.5 (10–14)	NS
1 st day Hematocrit	41 (36–44)	40 (36–42)	NS
1 st day Platelet	183 (80–250)	200 (150–270)	NS
1 st day Neutrophil	7200 (5400–9300)	7000 (5100–10900)	NS
1 st day Lymphocyte	1800 (1500–3100)	1950 (1600–3450)	NS
1 st day NLR	4.5 (3–6)	3.9 (3.4–5.5)	NS
1 st day CRP	25 (10–50)	15 (8–25)	NS
1 st day Procalcitonin	0.12 (0.05–0.20)	0.10 (0.05–0.20)	NS
1 st day SII	733 (510–920)	670 (550–800)	NS
2 nd day Hemoglobin	12.6 (10.4–13)	12.4 (10–13.6)	NS
2 nd day Hematocrit	38 (34–40)	37 (35–41)	NS
2 nd day Platelet	170 (140–230)	150 (100–210)	NS
2 nd day Neutrophil	7200 (5000–9200)	7000 (4900–8800)	NS
2 nd day Lymphocyte	1400 (1000–2400)	1700 (1300–2100)	NS
2 nd day NLR	4.6 (4–5)	3.8 (3.5–5)	0.008
2 nd day CRP	25 (10–50)	15 (8–25)	NS
2 nd day Procalcitonin	0.12 (0.05–0.20)	0.10 (0.05–0.20)	NS
2 nd day SII	780 (630–930)	570 (440–700)	<0.001
3 rd day Hemoglobin	11.8 (10–12)	11.9 (10.5–12.1)	NS
3 rd day Hematocrit	35 (33–38)	36(34–39)	NS
3 rd day Platelet	160 (120–270)	150 (130–250)	NS
3 rd day Neutrophil	8100 (6000–10000)	7250 (5900–8500)	NS
3 rd day Lymphocyte	1900 (1550–2300)	1850 (1500–2000)	NS
3 rd day NLR	4.2 (3–5.4)	2.5 (1.8–3.2)	0.003
3 rd day CRP	35 (25–50)	30 (20–40)	NS
3 rd day Procalcitonin	4 (2–8)	3 (2–6)	NS
3 rd day SII	800 (660–940)	550 (450–650)	<0.001

Data presented as median (interquartile range). CRP: C Reactive protein; NLR: Neutrophil lymphocyte ratio; SII: Systemic immune inflammation index.

carcinoma, breast and colorectal cancer, and bacterial and bloodstream infections.^[7,21–24] SII is an innovative inflammatory biomarker that integrates neutrophil, lymphocyte, and platelet counts to reflect the body's overall inflammatory status. It can easily be obtained through a noninvasive and widely accessible full blood count. NLR and SII have also been recognized as inflammatory markers of neonatal sepsis due to inflammation-induced changes in neutrophil, platelet, and lymphocyte counts.^[7,21–24]

The SII has been investigated for its diagnostic and prognostic potential in various immune and cardiovascular conditions, particularly those involving ischemia.^[23,25,26] Its utility is largely attributed to the involvement of neutrophils and platelets in the pathogenesis of endothelial dysfunction and atherosclerosis, where these cells contribute synergistically to vascular injury and systemic inflammation.^[27] In a study by Aydogan et al.,^[7] SII values were significantly elevated in CHD

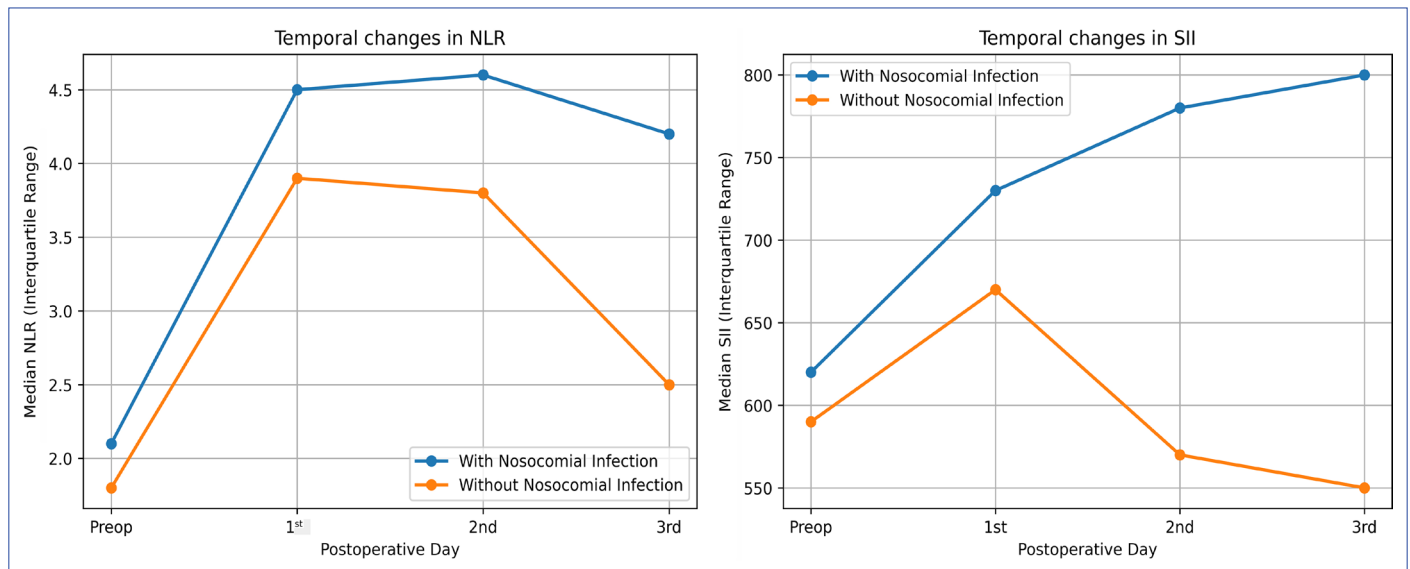


Figure 2. Changes in inflammatory markers over time.

patients with sepsis, with a proposed diagnostic cut-off value of 517. Additionally, a meta-analysis demonstrated a significant association between elevated SII and the risk of postoperative atrial fibrillation following cardiac surgery, reinforcing the role of SII as a predictive marker in cardiovascular surgical outcomes.^[28]

Despite the growing evidence supporting its use in cardiovascular and oncologic settings, studies focusing on the association between SII and infectious outcomes, particularly in pediatric populations, remain limited.^[26] Elevated SII levels have been associated with increased mortality in adult sepsis patients.^[29] However, the strength and consistency of this relationship are still under debate. Research on the role of SII in pediatric infectious diseases is especially scarce. Walian et al.^[30] reported that SII may serve as a predictor of adverse outcomes in children undergoing CPB surgery for acyanotic CHD. In contrast, Li et al.^[31] found a negative correlation between SII levels and early nosocomial infections in pediatric patients undergoing CPB for CHD.

In our study, both NLR and SII values were significantly elevated on postoperative days 2 and 3 among neonates who developed nosocomial infections. These findings support the potential of SII as an early indicator of postoperative infection in this vulnerable population and contribute to the limited body of literature addressing its use in neonatal and pediatric infectious outcomes.

This study has certain limitations. It is a retrospective analysis with a limited sample size from a single center, which, in our case, is a tertiary institution specialized in complex congenital cardiopathies in a specific age group of patients, neonates. Moreover, the underlying pathologies

in this population exhibited considerable heterogeneity. As the complexity of pathologies could significantly affect the outcomes, we were unable to demonstrate cut-off values for specific pathologies. Multicenter, prospective studies with a larger sample size are needed to validate cut-off values for using SII as a biomarker in clinical settings.

Conclusion

In conclusion, nosocomial infections cause significant mortality and morbidity in neonates undergoing congenital heart surgery. The SII, being a straightforward, simple-to-quantify, and reproducible inflammatory marker, may facilitate the early identification of postoperative nosocomial infections.

Disclosures

Ethics Committee Approval: The study was approved by the Basaksehir Cam and Sakura City Hospital Ethics Committee (no: KAEK/17.12.2025.450, date: 23/12/2025).

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