Submitted: 16 October, 2024 Accepted: 06 January, 2025 Published: 08 August, 2025 DOI:10.22514/sv.2025.109

Open Access
Signa Vitae

ORIGINAL RESEARCH

Ventilator-associated pneumonia in pediatric cardiac intensive care

Onur Ozalp[1,*], Erkut Ozturk[2]

[1]Department of Infectious Diseases and Clinical Microbiology, Sakura Hospital, Saglik Bilimleri University Basaksehir Cam, 34480 Istanbul, Turkey
[2]Department of Pediatric Cardiology, Sakura Hospital, Saglik Bilimleri University Basaksehir Cam, 34480 Istanbul, Turkey

***Correspondence**
onur.ozalp1@saglik.gov.tr
(Onur Ozalp)

Abstract

Background: This study aimed to evaluate the incidence of ventilator-associated pneumonia (VAP) and identify the risk factors contributing to its development in pediatric cardiac intensive care units. **Methods**: This retrospective study analyzed cases of patients <18 years old hospitalized in a pediatric cardiac intensive care unit between 01 January 2021, and 01 January 2024. Data patients diagnosed with VAP were matched (age and surgical procedure) with those of two control patients based. **Results**: During the study period, 1650 cardiac operations were performed. Among these, 520 cases requiring mechanical ventilation for more than 48 hours were included in the analysis. A total of 40 cases of VAP were identified and matched with 80 control cases. The incidence density of VAP was found to be 17.2 per 1000 ventilator days, and the median age of patients with VAP was two months. The isolated pathogens included *Klebsiella pneumoniae* (n = 14, 35%), *Stenotrophomonas maltophilia* (n = 7, 17.5%), *Pseudomonas aeruginosa* (n = 6, 15%), *Acinetobacter baumannii* (n = 6, 15%), *Staphylococcus aureus* (n = 4, 10%) and *Enterobacter* species (n = 3, 7.5%). The mortality rate in patients with VAP was 20% (8/40). Data analysis showed that independent risk factors for VAP included a RACHS-1 (Risk Adjustment for Congenital Heart Surgery) score of ≥4, the use of extracorporeal membrane oxygenation (ECMO), central venous catheterization lasting ≥14 days, mechanical ventilation dependency for ≥10 days, requirement for total parenteral nutrition, and delayed sternal closure of ≥2 days. **Conclusions**: VAP represents a significant cause of morbidity and mortality in pediatric cardiac intensive care units after congenital heart surgery. Gram-negative bacteria were identified as the predominant pathogens in this population.

Keywords

Congenital heart disease; Ventilator-associated pneumonia; Intensive care unit

1. Introduction

Congenital heart diseases (CHDs) represent a significant global public health concern, affecting populations in both developed and developing countries. Its prevalence is reported to be approximately 9 per 1000 live births, accounting for nearly one-third of all congenital anomalies [1].

CHDs are classified into various subtypes based on their pathophysiology, and the corresponding surgical treatment approaches range from corrective to palliative, depending on the specific condition. In recent years, the number of surgeries for CHDs has steadily increased, corresponding to the growing number of infants and children diagnosed with CHD. Consequently, this has also led to an increase in the incidence of postoperative complications in these patients [2].

Mechanical ventilation is commonly required after pediatric cardiac surgery, and its duration can be prolonged by factors such as malnutrition, immune system deficiencies, syndromic conditions, lung infections and extended cardiopulmonary bypass (CPB) times, all of which increase the risk of complications [3].

Ventilator-associated pneumonia (VAP) is among the most common complications in pediatric intensive care. In these patients, VAP is linked to adverse outcomes, such as extended hospital stays, prolonged mechanical ventilation and increased mortality rates [4]. However, data on VAP following pediatric heart surgery remains limited [4, 5]. This study aims to evaluate the incidence, causative factors, risk factors and outcomes of VAP in pediatric cardiac surgery patients admitted to our pediatric cardiac intensive care unit.

2. Methods

2.1 Patients

This retrospective case-control study was conducted on patients under 18 years of age admitted to the pediatric cardiac intensive care unit (PCICU) between 01 January 2021, and 01 January 2024. The unit is a 39-bed level 3 cardiac center

managing and treating all forms of CHD, except for heart transplants. Patients requiring mechanical ventilation for 48 hours or longer were included in the study. Patients were divided into two groups: those who developed VAP and the control group. VAP was defined based on the Centers for Disease Control and Prevention (CDC) and National Nosocomial Infections Surveillance (NNIS) criteria [6]. For the diagnosis of VAP, X-ray images were evaluated for the development or progression of infiltrates, consolidation, cavitation or pneumatosis (in patients under one year of age). Clinical signs and symptoms were also assessed, based on whether the patients were infants or children, including worsening gas exchange, temperature instability, increased respiratory secretions or suctioning requirements, leukopenia or leukocytosis, apnea, tachypnea, wheezing, rales or rhonchi. The diagnosis was confirmed by identifying pathogenic bacteria in deep tracheal aspirate cultures.

Control patients who had undergone surgery for CHD during the same time frame ($\pm$30 days) were selected from the same PCICU and were matched with VAP cases based on age ($\pm$10%), weight ($\pm$10%) and similar types of surgery. Special attention was given to matching patients with single-ventricle physiology, ensuring comparable characteristics between groups. Patients with comorbidities unrelated to cardiovascular disease, which were absent in the VAP group, were excluded from the control group. For each VAP case, two matched control patients were included. The study was approved by the local ethics committee and conducted in accordance with the Declaration of Helsinki.

Anesthesia drugs were administered according to pre-planned protocols designed to facilitate early extubation. For children younger than six months, induction involved the administration of 0.1 mg/kg midazolam, 1 μg/kg fentanyl and 0.6 mg/kg rocuronium. Maintenance was achieved using 0.1 μg/kg/min remifentanil, 5 μg/kg/min rocuronium and a minimum alveolar concentration of 1–1.2 sevoflurane. For children older than six months, the same drugs were used for induction. Maintenance included 0.25 μg/kg/min remifentanil, 5 μg/kg/min rocuronium, a minimum alveolar concentration of 1–1.2 sevoflurane and 0.5 μg/kg/h dexmedetomidine. Neuromuscular blockade was reversed with sugammadex to ensure muscle function recovery. Following surgery, patients were transferred to the PCICU with a continuous dexmedetomidine infusion to support postoperative care.

The patients were given an initial dose of 0.5 μg/kg/min milrinone at the start of the operation, and the maintenance dose was determined by the anesthetist on a case-by-case basis, taking into account the patient's hemodynamic status, the need for catecholamines and volume management during the procedure.

2.2 Extubation criteria and routine care

The criteria for extubation included patients being awake without external stimulation, demonstrating spontaneous breathing efforts and meeting specific respiratory and hemodynamic parameters such as maintaining a positive end-expiratory pressure (PEEP) of 5 cmH_2O, an inspired oxygen fraction (FiO_2) $\leq$0.4, an oxygenation index (OI) and an arterial oxygen partial pressure to FiO_2 ratio (PaO_2/FiO_2) greater than 200. Additionally, patients were required to have a partial pressure of carbon dioxide ($PaCO_2$) $\leq$50 mmHg, a pH $\geq$7.25, stable hemodynamics, no evidence of systolic or diastolic dysfunction on echocardiography, a strong cough reflex, and proper swallowing function [7].

Daily chest X-rays were performed for all intubated patients to assess lung status and detect any complications. Gastric ulcer prevention was managed with pantoprazole, particularly in patients receiving prolonged corticosteroid treatment. Oral hygiene was ensured by cleaning the mouth with wet dental swabs three to four times daily. Perioperative antibiotic prophylaxis involved cefazolin administered for 72 hours. For empiric antibiotic therapy, Targocid and ceftazidime were prescribed for infants under two months of age, while cefepime was used for older children. In cases where infection was suspected, blood and urine cultures were obtained to guide further treatment. Endotracheal secretions were examined using standard tracheal aspiration with semi-quantitative and microscopic qualitative techniques. However, bronchoalveolar lavage was not performed.

2.3 Data collection

A data collection form comprising demographic information, cardiac diagnosis, echocardiographic findings, operation duration, use of CPB, type of surgery, cardiac surgery scores, extubation time, length of stay in the intensive care unit and hospital, mortality, re-intubation status, presence of a central venous catheter (CVC) for more than 48 hours, use of total parenteral nutrition (TPN) for over 48 hours, development of chylothorax and complications was completed for each case.

For patients with suspected infections, a more detailed review was conducted to evaluate endotracheal aspirates and clinical indicators of VAP, such as temperature fluctuations, purulent sputum and changes in ventilator settings. Microbiology, laboratory results and chest radiographs were also analyzed to confirm the presence of infection.

2.4 Definitions

Nosocomial infections were defined as infections that were not present or in the incubation phase at the time of admission to the PCICU and developed at least 48 hours after admission. VAP was classified as nosocomial pneumonia that occurred 48 hours or more after the initiation of mechanical ventilation in patients receiving ventilator support. Deteriorating gas exchange was characterized by increases in FiO_2, peak inspiratory pressure (PIP), and/or PEEP levels. For patients without cardiac shunting, PaO_2/FiO_2 ratios and alveolar-arteriolar oxygen (A-a) gradients were calculated.

Ventilator days were recorded to calculate the VAP density rate per 1000 ventilator days in the PCICU. The mechanical ventilation usage ratio was also evaluated based on the length of stay in the PCICU. The VAP incidence rate was determined using the formula: (Number of VAP cases/Total number of patients receiving mechanical ventilation) $\times$ 100 = VAP rate per 100 patients.

The VAP incidence density was calculated as follows: (Number of VAP cases/Number of ventilator days) $\times$ 1000 =

VAP rate per 1000 ventilator days [8].

RACHS-1(Risk adjustment for congenital heart surgery scores) scores were calculated as previously described [2].

Bacterial colonies were identified using matrix-assisted laser desorption/ionization-time-of-flight (MALDI-TOF) technology with the Microflex LT/SH Smart MS system (FlexControl, Bruker Daltonics GmbH & Co. KG, Bremen, HB, Germany) and the MALDI-Biotyper Compass IVD 4.2.90 database. Antibiotic susceptibility testing was conducted to determine the Minimum Inhibitory Concentration (MIC) using the Phoenix M50 automated microbiology system (BD Diagnostics, Becton, Dickinson and Company, Sparks, MD, USA), which integrates identification and susceptibility testing of bacterial isolates. The results were evaluated based on the clinical breakpoints set by the European Committee on Antimicrobial Susceptibility Testing (EUCAST).

2.5 Statistical analysis

Data were analyzed using SPSS Statistics version 21 (IBM Corp., Armonk, NY, USA). Demographic variables are presented as numbers, percentages and medians with interquartile ranges (IQR). Categorical variables were compared between the VAP and control groups using either the chi-square test or Fisher's exact test, as appropriate. Continuous variables were analyzed using the Mann-Whitney U test. To identify independent predictors of VAP, a multivariable logistic regression analysis was performed. A p-value of < 0.05 was considered statistically significant.

3. Results

During the study period, 1650 cardiac operations were performed. Of these, 520 cases requiring mechanical ventilation for more than 48 hours were included in the analysis. Among these, 120 patients were selected for the study, comprising 40 VAP cases and 80 matched controls. The median age of patients diagnosed with VAP was 2 months (IQR 1–3), and 50% were male.

The types of surgeries in patients with VAP were as follows: arch reconstruction (n = 8, 20%), arterial switch procedure (n = 6, 15%), atrioventricular septal defect (AVSD) repair (n = 5, 12.5%), ventricular septal defect (VSD) repair (n = 4, 10%), total anomalous pulmonary venous return (TAPVR) repair (n = 3, 7.5%), Norwood stage 1 (n = 3, 7.5%), Glenn procedure (n = 2, 5%), primary complete unifocalization (n = 2, 5%), total repair of tetralogy of Fallot (TOF) (n = 2, 5%) and other procedures (n = 5, 12.5%).

Among the entire cohort, nearly 8% of patients (40/520 × 100) developed VAP. The incidence density was 17.2 per 1000 ventilator days (40/2316 × 1000). Microorganisms were identified in the tracheal aspirate cultures of 40 out of 1650 patients (0.24%). The distribution of pathogens included *Klebsiella pneumonia* (n = 14, 35%), *Stenotrophomonas maltophilia* (n = 7, 17.5%), *Pseudomonas aeriginosa* (n = 6, 15%), *Acinetobacter Baumannii* (n = 6, 15%), *Staphylococcus Aureus* (n = 4, 10%) and *Enterobacter* (n = 3, 7.5%) (Fig. 1).

The mortality rate associated with VAP was 20% (8/40).

Factors associated with the development of VAP in the PCICU were analyzed using univariate analysis (Table 1), and subsequent multivariable logistic regression analysis identified the following risk factors being independently associated with VAP (Table 2): a RACHS-1 score of ≥4 (OR (Odds Ratio): 2; 95% CI (Confidence Interval): 1.8–10; $p = 0.01$), use of ECMO (OR: 1.1; 95% CI: 1–5.6; $p = 0.005$), CVC usage ≥14 days (OR: 1.6; 95% CI: 1.2–6; $p = 0.02$), mechanical ventilation dependency for ≥10 days (OR: 3.2; 95% CI: 2–8; $p < 0.001$), requirement for TPN (OR: 0.8; 95% CI: 0.6–4; $p = 0.04$), and delayed sternal closure of ≥2 days (OR: 1.2; 95% CI: 1–3.5; $p < 0.001$).

4. Discussion

This study evaluated the incidence of VAP and its associated risk factors in pediatric patients undergoing congenital heart surgery in a PCICU over a three-year period. Our findings revealed that VAP remains a frequent complication in this population, with Gram-negative bacteria identified as the predominant pathogens. The independent risk factors for developing VAP included higher RACHS-1 scores, prolonged

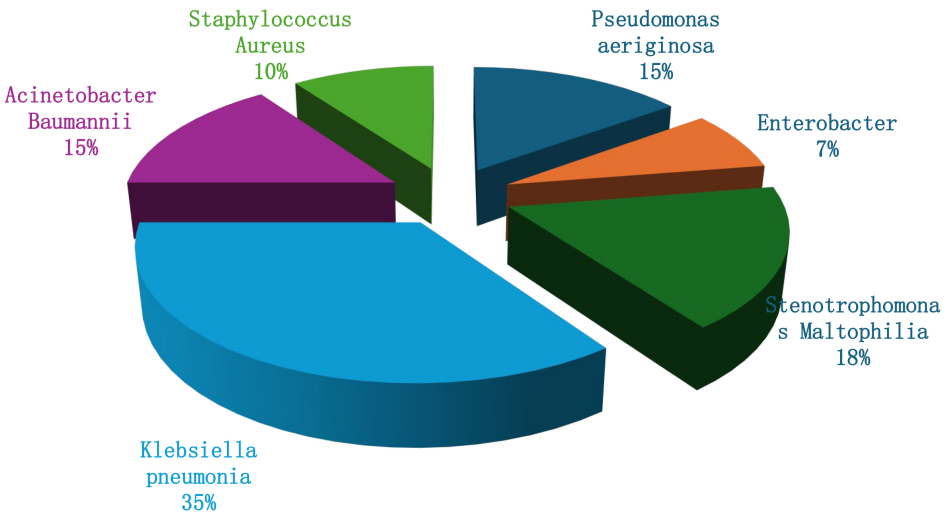

FIGURE 1. Distribution of microorganisms identified in the tracheal aspirate cultures.

TABLE 1. Demographic and clinical variables for patients with ventilator-associated pneumonia and control patients.

Variables	VAP (n = 40)	Control (n = 80)	*p*
Background illness	11 (27.5)*	5 (6.2)	**0.010**
Age, mon	2 (1–3)	3 (2–5)	NS
Weight, kg	3.9 (3.5–5)	5 (4.1–6)	**0.030**
Birth weight, kg			
≥2.5	32 (80)	76 (95)	NS
<2.5	8 (20)	4 (5)	
Male	20 (50)	42 (52.5)	NS
Genetic syndrome	7 (17.5)	10 (12.5)	NS
Single ventricle physiology	16 (40)	34 (42.5)	NS
Cyanotic heart disease	23 (57.5)	60 (50)	NS
Duration of preoperative mechanical ventilation, d	2 (0–4)	1 (0–2)	NS
CPB use	36 (90)	75 (94)	NS
CPB time, min	85 (75–95)	75 (65–100)	NS
RACHS-1 ≥4	22 (55)	16 (20)	**<0.001**
Central venous catheter duration, d	21 (15–28)	7 (5–10)	**<0.001**
Duration of postoperative mechanical ventilation, d	15 (10–20)	4 (2–6)	**<0.001**
Transfusion	36 (90)	40 (50)	**0.002**
TPN	20 (50)	10 (12.5)	**<0.001**
Duration of TPN, d	10 (6–14)	2 (1–3)	**<0.001**
ECMO	5 (12.5)	–*	**<0.001**
Arrhythmias	4 (10)	9 (12.5)	NS
Acute kidney injury	6 (15)	8 (10)	NS
LCOS	12 (30)	20 (25)	NS
Chylothorax	8 (20)	2 (2.5)	**<0.001**
Peritoneal dialysis	7 (17.5)	6 (7.5)	**0.040**
Need for iNO	6 (15)	2 (2.5)	**0.020**
Delayed sternal closure ≥2 d	14 (35)	4 (5)	**<0.001**
Previous antibiotic use	4 (10)	10 (12.5)	NS
Use of corticosteroids	6 (15)	11 (13.7)	NS
Use of pantoprazole	36 (90)	64 (80)	NS
ICU stay, d	30 (24–36)	8 (6–10)	**<0.001**
Post-op hospital stay, d	42 (34–54)	15 (12–18)	**<0.001**
Tracheostomy	1 (2.5)	–*	NS
Mortality	8 (20)	2 (2.5)	**<0.001**

**Median (IQR), n (%).*
Values with $p < 0.05$, which are considered statistically significant, are written in bold.
Abbreviations: CPB: cardiopulmonary bypass; ECMO: extracorporeal membrane oxygenation; ICU: intensive care unit; LCOS: low cardiac output syndrome; NS: non-significant; RACHS-1: risk adjustment for congenital heart surgery; TPN: total parenteral nutrition; VAP: ventilator-associated pneumonia; –: none; iNO: Inhaled Nitric Oxide.*

TABLE 2. Multivariate logistic regression analysis of factors associated with the development of ventilator-associated pneumonia.

Variables	*p*	Odds Ratio	95% Confidence Interval
RACHS-1 score ≥4	**0.010**	2.0	1.8–10.0
ECMO	**0.005**	1.1	1.0–5.6
Central venous catheter duration ≥14 d	**0.020**	1.6	1.2–6.0
Mechanical ventilation dependency ≥10 d	**<0.001**	3.2	2.0–8.0
Total parenteral nutrition	**0.040**	0.8	0.6–4.0
Delayed sternal closure ≥2 d	**<0.001**	1.2	1.0–3.5

Abbreviations: ECMO: Extracorporeal Membrane Oxygenation; RACHS-1: Risk Adjustment for Congenital Heart Surgery. Values with $p < 0.05$, which are considered statistically significant, are written in bold.

mechanical ventilation, ECMO use, extended CVC use, delayed sternal closure and the need for TPN. Collectively, these results contribute to the limited body of research specifically addressing VAP in pediatric cardiac patients' post-surgery.

Postoperative factors, such as hemodynamic instability, pulmonary edema, cytokine release and capillary leakage syndrome, compromise the immune system, increasing the susceptibility of PCICU patients to infections like VAP. It is noteworthy that VAP is the second most common hospital-acquired infection in PCICUs, following bloodstream infections [4, 9].

Existing literature on VAP incidence in pediatric cardiac patients remains limited, and most of the current literature has focused on general pediatric intensive care units. For instance, Mohamed *et al.* [10] reported a VAP incidence of 2–6 episodes per 1000 ventilator days in a non-cardiac pediatric ICU (intensive care unit), while studies conducted in pediatric cardiac ICUs have reported higher rates. Shaath *et al.* [4] observed a VAP incidence of 9.6%, with a density of 29 episodes per 1000 ventilator days [4]. Similarly, Tang *et al.* [5] identified a 13% incidence, corresponding to 21.6 episodes per 1000 ventilator days. A Dutch study involving 125 patients reported a VAP incidence of 8.8%, equivalent to 17.1 episodes per 1000 ventilator days [11]. Our findings align with these studies, showing a VAP incidence of 7.7% and an incidence density of 17.2 episodes per 1000 ventilator days.

Regarding the causative pathogens, Gram-negative bacteria were the predominant isolates, consistent with findings from previous studies, such as Tan *et al.* [12], who reported that 86.1% of VAP-related pathogens were Gram-negative bacilli. Similarly, Sun *et al.* [13] identified Gram-negative bacteria in 78% of cases, while Roeleveld *et al.* [11] reported *Haemophilus influenzae*, *Moraxella catarrhalis*, *Staphylococcus aureus* and *Pseudomonas aeruginosa* as the most frequently observed organisms in their study. In contrast, Singh *et al.* [14] identified *Acinetobacter baumannii* (43%) and *Klebsiella pneumoniae* (23%) as the most common pathogens.

In our study, *S. maltophilia* was identified as a causative agent in 17.5% of VAP cases, making it the second most common isolate. Notably, infections caused by *S. maltophilia* have been increasing globally in recent years [15–17]. The unique characteristics of our patient population, including immunosuppression, open wounds, multiple interventions and the frequent use of medical devices and implants, likely contributed to their susceptibility to *S. maltophilia* infections.

Overall, 90% of the pathogens identified in our study were Gram-negative, with Klebsiella pneumoniae being the most frequently isolated bacterium.

Several factors have been identified in the literature as contributing to the risk of VAP following pediatric cardiac surgery. Prolonged CPB and the use of TPN have been associated with higher rates of VAP [4]. Additional factors such as lower albumin levels (<35 g/L), extended mechanical ventilation (≥7 days), multiple tracheal intubations (≥3), prolonged CPB (≥100 minutes) and extended aortic occlusion times (≥60 minutes) have also been highlighted as risk factors [13]. Antalová *et al.* [18] further reported that the duration of mechanical ventilation, enteral feeding and the presence of cardiovascular disease were independent predictors of VAP.

In our study, we identified several risk factors associated with the development of VAP, which included high RACHS-1 scores, prolonged ECMO use, CVC usage lasting 14 days or more, mechanical ventilation dependency for 10 days or longer, the need for TPN and delayed sternal closure of at least two days. These findings suggest that specific interventions, such as minimizing the duration of mechanical ventilation through fast-track procedures, ensuring timely sternal closure to reduce exposure, limiting the use of CVCs to the shortest duration necessary, and carefully monitoring the administration of TPN to prevent associated complications, may help reduce the incidence of VAP in pediatric intensive care units following cardiac surgery.

Mortality associated with VAP remains a significant concern [4, 11, 14], and conflicting results have been reported in current literature. For instance, Shaath *et al.* [4] reported a mortality rate of 11% among VAP cases, whereas Roeleveld *et al.* [11] observed no mortality in their cohort of 11 VAP patients. Comparatively, our present study identified a VAP-related mortality rate of 20%, which may be attributed to the severity of the patients' conditions and the presence of pre-existing comorbidities.

This study has several limitations, including a small sample size, restriction to a single center and retrospective design, all of which may limit the generalizability of the findings.

5. Conclusions

VAP remains a significant cause of morbidity and mortality in PCICUs following congenital heart surgery, with gram-

negative bacteria identified as the predominant pathogens in children with CHD who develop VAP after surgery. The key risk factors associated with an increased risk of VAP in these cases include high RACHS-1 scores, ECMO use, prolonged dependence on mechanical ventilation, extended CVC usage, delayed sternal closure and the need for TPN.

AVAILABILITY OF DATA AND MATERIALS

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

OO—performed the research study. OO and EO—designed the research study, analyzed the data, wrote the manuscript. Both authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Ethics Committee for Scientific Research at Başakşehir Çam and Sakura City Hospital (Approval No: 2024.340) and was conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from the parents of all participants included in the study.

ACKNOWLEDGMENT

The authors express gratitude to the Pediatric Cardiology and Cardiovascular Surgery team at Başakşehir Çam and Sakura City Hospital for their invaluable contributions.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] van der Linde D, Konings EE, Slager MA, Witsenburg M, Helbing WA, Takkenberg JJ, *et al*. Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis. Journal of the American College of Cardiology. 2011; 58: 2241–2247.

[2] Jacobs JP, Jacobs ML, Mavroudis C, Backer CL, Lacour-Gayet FG, Tchervenkov CI, *et al*. Nomenclature and databases for the surgical treatment of congenital cardiac disease: an updated primer and an analysis of opportunities for improvement. Cardiology in the Young. 2008; 18: 38–62.

[3] Elward AM, Warren DK, Fraser VJ. Ventilator-associated pneumonia in pediatric intensive care unit patients: risk factors and outcomes. Pediatrics. 2002; 109: 758–764.

[4] Shaath GA, Jijeh A, Faruqui F, Bullard L, Mehmood A, Kabbani MS. Ventilator-associated pneumonia in children after cardiac surgery. Pediatric Cardiology. 2014; 35: 627–631.

[5] Tang CW, Liu PY, Huang YF, Pan JY, Lee SS, Hsieh KS, *et al*. Ventilator-associated pneumonia after pediatric cardiac surgery in southern Taiwan. Journal of Microbiology, Immunology and Infection. 2009; 42: 413–419.

[6] Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. American Journal of Infection Control. 2008; 36: 309–332.

[7] Özalp Ş, Dilek Özcanoğlu H, Öztürk E, Sağlam S, Kahraman İA, Tan ZB, *et al*. Assessment of the factors that affect fast-track or early extubation following pediatric cardiac surgery. Turkish Journal of Thoracic and Cardiovascular Surgery. 2023; 31: 1–7.

[8] Galal YS, Youssef MR, Ibrahiem SK. Ventilator-associated pneumonia: incidence, risk factors and outcome in paediatric intensive care units at Cairo university hospital. Journal of Clinical and Diagnostic Research. 2016; 10: SC06–SC11.

[9] Tablan OC, Anderson LJ, Besser R, Bridges C, Hajjeh R; CDC; Healthcare Infection Control Practices Advisory Committee. Guidelines for preventing health-care—associated pneumonia, 2003: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR Recommendations and Reports. 2004; 53: 1–36.

[10] Mohamed HT, Farhan Alenezi WA Sr, Alanzi MAA, Saleh Alsuqub FI, Salem Alhazmi SA, Mohammed Alhazmi OM. Prevalence of ventilator-associated pneumonia in children admitted to pediatric intensive care units in the middle east: a systematic review. Cureus. 2023; 15: e51230.

[11] Roeleveld PP, Guijt D, Kuijper EJ, Hazekamp MG, de Wilde RB, de Jonge E. Ventilator-associated pneumonia in children after cardiac surgery in The Netherlands. Intensive Care Medicine. 2011; 37: 1656–1663.

[12] Tan L, Sun X, Zhu X, Zhang Z, Li J, Shu Q. Epidemiology of nosocomial pneumonia in infants after cardiac surgery. Chest. 2004; 125: 410–417.

[13] Sun YL, Fu YQ, Ma HT, Liu CJ, Xu F. Pathogens and risk factors for ventilator-associated pneumonia in children with congenial heart disease after surgery. Chinese Journal of Contemporary Pediatrics. 2015; 17: 1204–1209. (In Chinese)

[14] Singh D, Sahu MK, Singh SP, Singh U, Shiv C, Shivam P. Outcome of ventilator-associated pneumonia in children post cardiac surgery: a prospective observational study. Journal of Cardiac Critical Care TSS. 2022; 6: 033–039.

[15] Banar M, Sattari-Maraji A, Bayatinejad G, Ebrahimi E, Jabalameli L, Beigverdi R, *et al*. Global prevalence and antibiotic resistance in clinical isolates of Stenotrophomonas maltophilia: a systematic review and meta-analysis. Frontiers in Medicine. 2023; 10: 1163439.

[16] Mojica MF, Humphries R, Lipuma JJ, Mathers AJ, Rao GG, Shelburne SA, *et al*. Clinical challenges treating Stenotrophomonas maltophilia infections: an update. JAC-Antimicrobial Resistance. 2022; 4: dlac040.

[17] Carbonell N, Oltra MR, Clari MÁ. Stenotrophomonas maltophilia: the landscape in critically Ill patients and optimising management approaches. Antibiotics. 2024; 13: 577.

[18] Antalová N, Klučka J, Říhová M, Poláčková S, Pokorná A, Štourač P. Ventilator-associated pneumonia prevention in pediatric patients: narrative review. Children. 2022; 9: 1540.

How to cite this article: Onur Ozalp, Erkut Ozturk. Ventilator-associated pneumonia in pediatric cardiac intensive care. Signa Vitae. 2025; 21(8): 24-29. doi: 10.22514/sv.2025.109.