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Association Between Rectal Colonization of Acinetobacter baumannii and Subsequent Invasive Infections in a Neonatal Intensive Care Unit

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Abstract

Objective: *Acinetobacter spp.* is a significant nosocomial pathogen in neonatal intensive care units due to its environmental persistence and multidrug resistance profile. Rectal colonization in neonates may precede invasive infections such as bloodstream infection and ventilator-associated pneumonia, serving as a potential marker for subsequent clinical deterioration. However, the factors associated with progression from rectal colonization to systemic infection in this vulnerable population remain insufficiently defined.
Materials and Methods: This retrospective cohort study was conducted between January 2018 and January 2023. Neonates with rectal *Acinetobacter spp.* colonization detected by surveillance cultures were included. Patients were classified according to the development of culture-confirmed bloodstream infection (BSI) and/or ventilator-associated pneumonia (VAP) during follow-up. The primary outcome was the development of invasive *Acinetobacter* infection after rectal colonization, and its association with recurrent colonization episodes was evaluated.
Results: A total of 73 neonates with rectal *Acinetobacter spp.* colonization were included. During follow-up, 10 patients (13.7%) developed BSI and 5 (6.8%) developed VAP caused by *Acinetobacter spp.* Birth weight, gestational age, prematurity, central venous catheter use, and duration of respiratory support were comparable between groups (all $p > 0.05$). However, neonates who developed invasive infection had a significantly higher number of positive rectal surveillance cultures. The overall in-hospital mortality rate was 16.4%, with a higher but not statistically significant mortality observed in patients with invasive infection (30% vs. 14%, $p = 0.14$).
Conclusion: Among neonates with rectal *Acinetobacter spp.* colonization, recurrent positivity in surveillance cultures appears to be associated with an increased risk of subsequent invasive infection, suggesting that persistence of colonization may serve as a practical clinical marker to identify high-risk patients who require intensified monitoring and infection control measures.

Keywords: Neonate; Bloodstream infection; Ventilator associated pneumonia; Rectal swab; Acinetobacter spp.

Rektal Acinetobacter baumannii Kolonizasyonu ile Yenidoğan Yoğun Bakım Ünitesinde İnvaziv Enfeksiyon Arasındaki İlişkinin Değerlendirilmesi

Özet

Amaç: *Acinetobacter spp.*, çevresel dirençliliği ve çoklu ilaç direnci profili nedeniyle yenidoğan yoğun bakım ünitelerinde önemli bir nozokomiyal patojendir. Yenidoğanlarda rektal kolonizasyon, kan dolaşımı enfeksiyonu ve ventilatör ilişkili pnömoni gibi invaziv enfeksiyonlardan önce görülebilir ve klinik kötüleşmenin potansiyel bir belirteci olabilir. Ancak bu hassas popülasyonda rektal kolonizasyondan sistemik enfeksiyona progresyonla ilişkili faktörler yeterince tanımlanmamıştır.
Gereç ve Yöntem: Bu retrospektif kohort çalışma Ocak 2018 – Ocak 2023 tarihleri arasında yürütüldü. Sürveyans kültürlerinde rektal *Acinetobacter spp.* kolonizasyonu saptanan yenidoğanlar çalışmaya dahil edildi. Hastalar takip sürecinde kültürle doğrulanmış kan dolaşımı enfeksiyonu (KDE) ve/veya ventilatör ilişkili pnömoni (VİP) gelişimine göre sınıflandırıldı. Birincil sonlanım noktası, rektal kolonizasyon sonrası invaziv Acinetobacter enfeksiyonu gelişimiydi ve bunun tekrarlayan kolonizasyon atakları ile ilişkisi değerlendirildi.
Bulgular: Rektal *Acinetobacter spp.* kolonizasyonu olan toplam 73 yenidoğan çalışmaya dahil edildi. İzlem süresince 10 hastada (%13,7) Acinetobacter kaynaklı KDE, 5 hastada (%6,8) ise VİP gelişti. Doğum ağırlığı, gebelik haftası, prematürite, santral venöz kateter kullanımı ve solunum desteği süresi açısından gruplar arasında anlamlı fark saptanmadı (tüm $p > 0,05$). Ancak invaziv enfeksiyon gelişen yenidoğanlarda pozitif rektal sürveyans kültürü sayısı anlamlı olarak daha yüksekti. Genel hastane içi mortalite oranı %16,4 olup, invaziv enfeksiyon gelişen hastalarda mortalite daha yüksek olmakla birlikte istatistiksel olarak anlamlı değildi (%30'a karşı %14, $p = 0,14$).
Sonuç: Rektal *Acinetobacter spp.* kolonizasyonu olan yenidoğanlarda sürveyans kültürlerinde tekrarlayan pozitiflik, sonraki invaziv enfeksiyon gelişme riski ile ilişkili görünmektedir. Kolonizasyonun persistan seyri, yoğun izlem ve enfeksiyon kontrol önlemlerinin artırılması gereken yüksek riskli hastaların belirlenmesinde pratik bir klinik belirteç olabilir.

Anahtar kelimeler: Yenidoğan; kan dolaşımı enfeksiyonu; ventilatör ilişkili pnömoni; rektal sürüntü; Acinetobacter spp.

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INTRODUCTION

Acinetobacter spp., particularly *A. baumannii*, has emerged as a major nosocomial pathogen in neonatal intensive care units (NICUs) owing to its ability to persist on environmental surfaces for prolonged periods, spread clonally with ease, and rapidly acquire multidrug resistance (1). Carbapenem-resistant *A. baumannii* is categorized as a "critical priority" pathogen in the World Health Organization's updated List of Bacterial Priority Pathogens, underscoring the need for effective infection control strategies and early risk assessment in high-risk settings such as NICUs (2).

In NICUs, microbial colonization often precedes the onset of invasive infection and may reflect both nosocomial transmission within the unit and an increased individual risk for bloodstream infection (BSI) and pneumonia. Previous studies have demonstrated that rectal surveillance cultures can assist in identifying neonates colonized with resistant Gram-negative organisms who are at higher risk of developing invasive infections, and may also help guide empirical antimicrobial therapy and isolation precautions (3,4). In one neonatal intensive care cohort, gastrointestinal colonization was detected in 6.9% of newborns, of whom 56.9% were colonized with *A. baumannii* (5). Similarly, a study from a NICU in Morocco reported a 13.7% colonization rate with multidrug-resistant *A. baumannii* (6). In an Indian cohort, the prevalence of *A. baumannii* colonization among neonates was found to be 11%, with multidrug resistance observed in 60.7% and carbapenem resistance in 21.4% of isolates (7).

It is well recognized that a substantial proportion of neonates with rectal colonization may subsequently develop bloodstream infections. Several studies have highlighted the association between gastrointestinal carriage and later invasive infection, and in many instances, genotypic concordance between strains isolated from intestinal and bloodstream samples has been demonstrated (4,6). The high rates of colonization reported in NICUs, together with the considerable mortality associated with invasive *A. baumannii* infections, emphasize the importance of implementing strategies aimed at early detection and timely treatment.

Identifying the factors that predispose colonized neonates to systemic infection is therefore of critical importance. However, there remains limited evidence regarding the conditions under which rectal Acinetobacter spp. colonization progresses to invasive disease. The clinical and care-related determinants that contribute to the development of BSI or pneumonia are not yet fully understood, partly due to variations in patient characteristics, use of invasive devices, antibiotic exposure, and infection control practices across centers. Reported risk factors for Acinetobacter spp. infection in neonates include low birth weight, prolonged NICU stay, umbilical catheterization, central venous catheterization, need for ventilatory support, and prior antibiotic therapy (3,7). Some studies have further suggested that the requirement for respiratory support represents the most significant factor associated with the development of bloodstream infection among rectally colonized patients (3,5).

Rectal swab cultures are commonly employed in NICUs for surveillance purposes and outbreak detection. Although data directly examining the relationship between rectal *A. baumannii* colonization and ventilator-associated pneumonia (VAP) in neonates remain scarce, studies conducted in adult intensive care settings have shown that rectal colonization may serve as a strong predictor of *A. baumannii*-related VAP in mechanically ventilated patients (8).

The present study aims to determine the risk factors associated with the development of bloodstream

infection and ventilator-associated pneumonia in neonates with rectal Acinetobacter spp. colonization during NICU hospitalization. In addition, it seeks to highlight the clinical importance of routine surveillance through rectal colonization monitoring in this vulnerable population.

MATERIALS AND METHODS

Study Design and Ethical Approval

This single-center, retrospective observational cohort study was conducted to evaluate the risk factors associated with the development of bloodstream infection (BSI) and/or ventilator-associated pneumonia (VAP) during follow-up in neonates with rectal *Acinetobacter spp.* colonization in the neonatal intensive care unit (NICU).

The study was carried out in a tertiary-level NICU between January 2018 and January 2023. The study protocol was approved by the local Clinical Research Ethics Committee (Decision No: 369, Date: 03.03.2023). Written informed consent was obtained from the parents or legal guardians of all patients included in the study.

Study Population

Study Population and Definitions

Neonates hospitalized in the NICU during the study period who were found to have *Acinetobacter spp.* colonization in rectal swab cultures were included.

Patients were classified into two groups according to the development of invasive infection during follow-up:

- Group 1: Neonates who developed BSI and/or VAP
- Group 2: Neonates with rectal colonization only

Surveillance Protocol and Sampling Strategy

Rectal surveillance cultures were obtained according to the unit's standardized infection control protocol, with timing stratified by birth weight. In neonates with a birth weight ≥1500 g, rectal swab samples were collected at the time of NICU admission. In neonates with a birth weight <1500 g, sampling was initiated at the end of the first week of hospitalization to reduce early false-negative results and reflect colonization dynamics in very low birth weight infants.

For patients hospitalized longer than 7 days, rectal surveillance cultures were repeated at weekly intervals. All samples were collected under sterile conditions.

Inclusion and Exclusion Criteria

Inclusion criterion:

- Detection of *Acinetobacter spp.* growth in rectal swab culture

Exclusion criteria:

- Diagnosis of *Acinetobacter spp.*-related BSI or pneumonia prior to detection of rectal colonization
- Presence of primary or secondary immunodeficiency
- Death within the first 24 hours of NICU admission

Microbiological Analysis

Rectal swab samples were transported to the microbiology laboratory within 1 hour of collection. Microorganism identification was performed using standard microbiological methods and/or automated identification systems. Antimicrobial susceptibility testing was interpreted according to the criteria of the European Committee on Antimicrobial Susceptibility Testing (9).

Acinetobacter spp. isolates identified in rectal and blood cultures were further evaluated for carbapenem resistance. Carbapenem-resistant isolates were classified as CRAB (carbapenem-resistant *Acinetobacter spp.*).

Data Collection

Patient data were obtained retrospectively from medical records and the hospital information system.

Demographic and perinatal characteristics included sex, gestational age (weeks), birth weight (grams), mode of delivery (normal vaginal delivery or cesarean section), presence of multiple pregnancy, Apgar scores at 5th minutes, and the need for postnatal resuscitation.

Clinical follow-up data included age at NICU admission, total length of NICU stay, development of BSI and/or VAP, and in-hospital mortality.

Data related to intensive care practices were also recorded, including the presence and duration of invasive and non-invasive mechanical ventilation, duration of umbilical and non-umbilical central venous catheter use, duration of total parenteral nutrition (TPN), and timing of initiation of enteral feeding.

Outcome Definitions

The primary outcome of the study was defined as culture-confirmed *Acinetobacter spp.* bloodstream infection and/or VAP developing after the detection of rectal *Acinetobacter spp.* colonization.

Bloodstream infection was defined in accordance with established neonatal sepsis criteria as the isolation of *Acinetobacter baumannii* from at least one blood culture in the presence of clinical and/or laboratory signs of sepsis, including but not limited to temperature instability, apnea, bradycardia, feeding intolerance, hemodynamic instability, elevated inflammatory markers (e.g., C-reactive protein, procalcitonin), or abnormal leukocyte counts. Ventilator-associated pneumonia was defined based on international guidelines as pneumonia occurring ≥48 hours after initiation of invasive mechanical ventilation, characterized by new or progressive pulmonary infiltrates on chest radiography, accompanied by clinical signs such as increased respiratory secretions, worsening oxygenation, or ventilatory requirements, and supported by microbiological evidence of *Acinetobacter spp.* growth in tracheal aspirate cultures. These definitions were adapted from previously published neonatal and intensive care infection criteria to ensure standardized and clinically relevant outcome assessment

The association between the number of positive rectal swabs and the development of invasive infection was also evaluated.

Statistical Analysis

Statistical analyses were performed using SPSS (IBM SPSS Statistics, Version 26.0, IBM Corp., Armonk, NY, USA) software. The distribution of continuous variables was assessed using the Shapiro–Wilk test.

Normally distributed data were expressed as mean ± standard deviation, whereas non-normally distributed data were presented as median (minimum–maximum). Categorical variables were expressed as numbers and percentages.

For intergroup comparisons, the Independent samples t-test or Mann–Whitney U test was used for continuous variables. For the comparison of categorical variables, the Pearson chi-square test, Yates' continuity correction chi-square test, or Fisher's exact test was applied as appropriate based on the expected cell frequencies.

A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 73 newborns with Acinetobacter spp. colonization detected in rectal swab cultures were included in the study. During follow-up, 10 patients (13.7%) developed bloodstream infection (BSI) caused by *Acinetobacter spp.*, while ventilator-associated pneumonia (VAP) was detected in 5 patients (6.8%) with *Acinetobacter spp.* growth in tracheal aspirate culture.

The median age of the patients' mothers was 26 years (range 16–39), and the median number of pregnancies was 2 (range 1–9). The mean birth weight of the newborns was 2255 ± 788 g, and the mean gestational age was 34.5 ± 4.27 weeks. Fifty-two percent of the patients were male, and 57.5% were delivered by cesarean section. The mortality rate in the study group was 16.4% (n=12). During the clinical course, VAP was observed in 9.6% of the total patient population. Acinetobacter spp. growth was detected in 13.7% of blood cultures and in 6.8% of tracheal aspirate cultures (Table 1).

Table 1. Demographic and clinical characteristics of patients

	Patient group (n=73)
Mother's age*	26 (16-39)
Number of Pregnancies*	2 (1-9)
Birth weight, g **	2255±788
Gestational age, weeks **	34.5±4.27
Gender, F, n (%)	38 (52)
C/S, n (%)	42 (57.5)
Mortality, n (%)	12 (16.4)
Ventilator-associated pneumonia, n(%)	7 (9.6)
Acinetobacter spp. in blood culture, n(%)	10 (13.7)
Acinetobacter spp. in tracheal aspirate culture, n (%)	5 (6.8)

*Median (minimum-maximum); **mean±SD

When comparing patients who developed bloodstream infection (Group 1) with those who did not (Group 2), birth weight (2388 ± 770 g vs. 2219 ± 796 g, p=0.408) and gestational age (35.2 ± 4 vs. 34.3 ± 4.2 weeks, p=0.43). The frequencies of intrauterine growth restriction, central venous catheter use, multiple congenital anomalies, birth asphyxia, and prematurity were similar between the two groups (all p>0.05). There was no significant difference in the 5-minute Apgar scores between patients with and without Acinetobacter bacteremia (median 6 [5–8] vs. 7 [5–9], p=0.61). (Table 2).

Table 2. Comparison of risk factors between patients with and without bloodstream infections

	Group 1 (n=10)	Group 2 (n=63)	p
Birth weight, g*	2388±770	2219±796	0.408**
Gestational age, weeks*	35.2±4	34.3±4.2	0.43**
Apgar score at 5 min.*	6 (5-8)	7 (5-9)	0.61**
Intrauterine growth restriction, n (%)	1 (10)	10 (15.9)	0.85***
Other central venous catheter, n (%)	4 (40)	15 (23.8)	0.606***
Multiple anomalies, n (%)	5 (50)	17 (27)	1.00***
Birth asphyxia, n (%)	3 (30)	7 (11)	0.107***
Prematurity, n (%)	5 (50)	40 (63)	0.47***

*mean±SD
**Independent samples t-test
***Fisher's exact test

No statistically significant differences were found between the BSI and non BSI groups in terms of the time to transition to full enteral feeding, invasive and non-invasive mechanical ventilation durations, and length of hospital stay (all p>0.05). Although the mortality rate was higher in the group with KDE development (30% vs. 14%), this difference was not statistically significant (p=0.14). There was no significant difference in the duration of total parenteral nutrition between patients with and without Acinetobacter bacteremia (median 12 [0–48] vs. 0 [0–95] days, p=0.12). Similarly, the duration of umbilical catheterization did not differ significantly between the two groups (median 4.5 [0–14] vs. 0 [0–17] days, p=0.46). There was no significant difference in length of hospital stay between patients with and without Acinetobacter bacteremia (median 31 (6–46) days vs. 29 (5–81) days, p=0.91). The need for postnatal resuscitation was higher in patients with Acinetobacter bacteremia compared to those without (40% vs. 15.8%); however, the difference did not reach statistical significance (p=0.11) (Table 3).

Table 3. Comparison of clinical risk factors and outcomes between patients with and without bloodstream infection

	Group 1 (n=10)	Group 2 (n=63)	p
Duration of total parenteral nutrition*	12 (0-48)	0 (0-95)	0.12**
Duration of umbilical catheterization*	4.5 (0-14)	0 (0-17)	0.46**
Time to transition to full enteral nutrition, days*	9 (4-95)	10 (3-76)	0.94**
Duration of invasive mechanical ventilation support, days*	22 (0-27)	9 (0-72)	0.98**
Duration of non-invasive mechanical ventilation support, days*	2 (0-30)	3 (0-31)	0.44**
Length of hospital stay*	31 (6-46)	29 (5-81)	0.91**
Need for postnatal resuscitation, n (%)	4 (40)	10 (15.9)	0.11***
Mortality, n (%)	3 (30)	9 (14)	0.14***

*Median (Minimum-maximum)
**Mann–Whitney U test
*** Fisher's exact test

The number of rectal colonization recurrences was strongly associated with the development of infection. The median number of positive rectal swabs was 2 (1–7) in patients who developed BSI, compared to 1 (1–7) in those who did not, and this difference was statistically significant (p<0.01). Similarly, the number of positive rectal swabs was 5 (2–7) in patients who developed VAP, compared to 1 (1–7) in those who did not develop VAP, and this difference was significant (p=0.015) (Table 4).

Table 4. Comparison of rectal colonization recurrence rates between patients with and without bloodstream infections and ventilator-associated pneumonia

	Group 1 (n=10)	Group 2 (n=63)	p**
Bloodstream infection			
Number of positive rectal swabs*	2 (1-7)	1 (1-7)	<0.01
Tracheal aspirate culture*			
Number of positive rectal swabs*	5 (2-7)	1 (1-7)	0.015

*Median (Minimum-maximum); **Mann–Whitney U test

Multivariable analysis was not performed due to the limited sample size.

DISCUSSION

In this retrospective cohort study, we demonstrated that rectal *Acinetobacter spp.* colonization in the neonatal intensive care unit (NICU) is associated with the development of invasive infection, and that the likelihood of invasive infection increases as the duration of colonization becomes longer.

Rectal colonization is widely regarded as an early warning marker of resistant Gram-negative pathogens within the NICU environment, and previous studies have shown that colonization can be detected before the onset of invasive infection (10). However, because not every colonized patient subsequently develops infection, colonization alone is unlikely to be sufficient; additional factors such as bacterial burden, persistence of colonization, and care-related processes likely contribute to the progression to bloodstream infection. Adult intensive care studies have similarly reported that a higher colonization density is associated with an increased risk of infection (11).

Healthcare-associated infections caused by multidrug-resistant Gram-negative organisms remain a major source of morbidity and mortality in NICUs. Among these, bacteremia and ventilator-associated pneumonia (VAP) due to *Acinetobacter baumannii* are particularly associated with high mortality rates (12). In our cohort, 13.7% of neonates with rectal *Acinetobacter* spp. colonization developed bloodstream infection and 6.8% developed VAP, supporting the clinical relevance of colonization as a potential marker for invasive infection risk. The gastrointestinal tract and skin are recognized reservoirs for invasive infections caused by Gram-negative pathogens. Active surveillance studies in NICUs have demonstrated that colonized patients have a significantly higher risk of developing invasive infection (5,10,13). In the study by Çetin et al., invasive infection occurred in 4.5% of colonized neonates, suggesting that although colonization does not invariably lead to disease, it may represent a preliminary step in the infectious process (14). In our analysis, patients who developed invasive infection had a significantly greater number of positive rectal swabs, indicating that the persistence or cumulative burden of colonization may play a critical role in infection development.

Çetin et al. reported no significant differences in birth weight or prematurity between colonized infants who did and did not develop infection (14). Likewise, in our study, birth weight, gestational age, and several perinatal characteristics were comparable between neonates with and without bloodstream infection. These findings suggest that, within a colonized population, progression to invasive infection may be more closely related to colonization dynamics and unit-specific care practices than to baseline neonatal characteristics. Our results further imply that colonization may exert an effect on infection development that is independent of traditional neonatal risk factors.

Adult ICU studies have also highlighted that, beyond patient-related variables, factors such as invasive procedures and the presence of surgical patients significantly influence the risk of bloodstream infection (11). In our study, no significant differences were observed between groups in terms of total parenteral nutrition duration, umbilical catheterization, mechanical ventilation, or length of hospital stay. Consistent with this, antenatal and postnatal characteristics were not associated with bloodstream infection in our cohort, whereas indicators reflecting colonization burden such as the frequency of rectal positivity appeared more informative.

Although data directly examining the progression from rectal *A. baumannii* colonization to VAP in neonates are limited, classical genotypic studies in intensive care settings have suggested a link between gastrointestinal colonization and subsequent respiratory tract infection (15). In our cohort, infants who developed VAP had a higher number of positive rectal swabs, raising the possibility that persistent or increasing colonization may elevate the risk of lower respiratory tract infection through mechanisms such as aspiration, cross-contamination, or endotracheal colonization. Notably, while traditional risk factors such as invasive procedures and nutritional support did not differ between groups, the frequency of positive rectal cultures remained the only variable significantly associated with infection. This supports the concept that colonization burden may be a more

relevant predictor of invasive disease than the mere presence of invasive interventions.

These findings underscore the importance of strict contact precautions and environmental cleaning measures in addition to standard VAP prevention bundles for colonized patients.

Carbapenem-resistant *A. baumannii* is classified among the critical priority pathogens due to its limited therapeutic options, making early detection and prevention strategies essential in intensive care settings (16). Our findings suggest that rectal surveillance cultures may not only identify colonization but also help predict infection risk, particularly in cases of repeated colonization. Previous studies have shown that active surveillance combined with reinforced infection control measures can reduce colonization and infection burdens in ICUs (17). Molecular epidemiological investigations further support the use of rectal screening as a tool for identifying patient-to-patient transmission (18). The association observed between repeated positive swabs and infection risk in our study indicates that high-risk colonized patients may benefit from intensified isolation precautions and closer clinical and microbiological monitoring.

Early identification of colonization also has implications for antimicrobial stewardship. Analysis of rectal surveillance data may inform empirical antibiotic strategies at the unit level and support individualized therapeutic decision-making (19). In patients with increasing colonization burden, antibiotic choices should be carefully evaluated in conjunction with clinical findings to ensure appropriate therapy while minimizing the development of resistance. Although the number of positive swabs provides a practical proxy for colonization burden, it may not precisely reflect actual bacterial density. Nevertheless, a key strength of our study lies in its ability to link colonization persistence within the same colonized cohort to meaningful clinical outcomes, highlighting a feasible and clinically applicable risk indicator for NICU practice. From a clinical perspective, these findings highlight that routine surveillance cultures should not only be used to detect colonization but also to monitor its persistence over time, as repeated positivity may identify a subgroup of neonates at particularly high risk for invasive infection.

CONCLUSION

This study has several limitations. Its retrospective, single-center design limits generalizability and raises the possibility of unmeasured confounding. Although rectal colonization was monitored through weekly surveillance cultures, colonization was not analyzed as a time-dependent variable; therefore, infants with longer hospital stays may have undergone more frequent sampling, introducing potential time-at-risk bias. The limited number of invasive infection events precluded multivariable analysis, and important variables such as antibiotic exposure and infection control practices could not be incorporated into the statistical models. Additionally, variation in the timing of surveillance cultures according to birth weight may have influenced the detection of colonization and introduced measurement bias.

In conclusion, our findings indicate that rectal *Acinetobacter* spp. colonization in NICU patients is associated with subsequent invasive infection, and that recurrent colonization episodes may serve as an important marker of infection risk. Early recognition of colonization and careful management of invasive interventions may contribute to reducing infection incidence and improving clinical outcomes in this vulnerable population.

Ethics Committee Approval

Ethical approval was obtained from the local Clinical Research Ethics Committee (Decision No: 369, Date: March 3, 2023). Written informed consent was obtained from the parents or legal guardians of all patients included in the study.

Author Contributions

Conception - Duygu Tunçel, Muhammed Asena, Leyla Şero, Nilüfer Okur; Design - Duygu Tunçel, Muhammed Asena, Leyla Şero, Nilüfer Okur; Supervision - Nilüfer Okur, Muhammed Asena; Data Collection and/or Processing - Duygu Tunçel, Leyla Şero; Analysis and/or Interpretation - Duygu Tunçel, Muhammed Asena, Nilüfer Okur; Literature Search - Duygu Tunçel, Leyla Şero; Writing - Duygu Tunçel, Muhammed Asena; Critical Review - Muhammed Asena, Nilüfer Okur.

Conflict of Interest

The authors declare that there is no conflict of interest in this study.

Financial Disclosure

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