Received: 18.01.2026
Accepted: 30.01.2026
Published: 16.04.2026

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Neonatal infections caused by *Serratia marcescens*: a narrative review and implications for prevention in NICU settings

Zakażenia noworodków wywołane przez *Serratia marcescens*: przegląd narracyjny i znaczenie dla profilaktyki w warunkach oddziału intensywnej terapii noworodka

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https://doi.org/10.15557/PiMR.2026.0002

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Abstract

Serratia marcescens is a significant opportunistic pathogen responsible for recurrent outbreaks in neonatal intensive care units (NICU) worldwide. Its ability to persist in the hospital environment, develop antimicrobial resistance, and spread through diverse transmission routes makes prevention and control particularly challenging, especially among vulnerable neonatal populations. This narrative review summarises published literature from 2015 to 2025 on *S. marcescens* infections in neonates, with a focus on outbreak characteristics, transmission pathways, clinical manifestations, outcomes, and infection control strategies implemented in NICU settings. Thirty-eight outbreaks reported across five continents were analysed. Most outbreaks predominantly affected preterm and very low birth weight infants, with bloodstream infection and sepsis representing the most common clinical presentations. Reported mortality rates varied widely, ranging from approximately 7% to over 60%, reflecting differences in patient populations, outbreak severity, and healthcare resources. Environmental reservoirs, including sinks, medical equipment, and surrounding surfaces, were the most frequently implicated sources of transmission, followed by healthcare workers' hands and water-associated systems. Molecular typing methods, particularly pulsed-field gel electrophoresis and whole-genome sequencing, confirmed clonal transmission in a substantial proportion of outbreaks and proved essential for outbreak investigation and containment. Effective control measures included reinforcement of hand hygiene practices, thorough environmental decontamination, cohorting of colonised or infected neonates, and temporary reorganisation or closure of affected NICU areas. *S. marcescens* remains a persistent cause of neonatal outbreaks, underscoring the need for continuous surveillance, environmental monitoring, and standardised prevention strategies, particularly in resource-limited settings.

Keywords: infection control, outbreak, NICU, *Serratia marcescens*, neonatal infection

Streszczenie

Serratia marcescens (pałeczka krwawa) stanowi istotny patogen oportunistyczny, odpowiedzialny za nawracające ogniska epidemiczne na oddziałach intensywnej terapii noworodka (OITN) na całym świecie. Zdolność tej bakterii do przetrwania w środowisku szpitalnym, predyspozycja do rozwijania oporności na antybiotyki oraz wielokierunkowe drogi transmisji sprawiają, że zapobieganie i kontrola zakażeń stanowią wyzwanie, zwłaszcza w populacji noworodków szczególnie podatnych na zakażenia. Niniejszy przegląd narracyjny podsumowuje publikacje z lat 2015–2025 dotyczące zakażeń *S. marcescens* u noworodków, z uwzględnieniem charakterystyki ognisk epidemicznych, dróg transmisji, obrazu klinicznego, wyników leczenia oraz strategii kontroli zakażeń wdrożonych w warunkach OITN. Analizie poddano trzydzieści osiem ognisk epidemicznych zgłoszonych na pięciu kontynentach. Większość dotyczyła noworodków przedwcześnie urodzonych oraz z bardzo niską masą urodzeniową; najczęstszymi postaciami klinicznymi były zakażenia krwi i sepsa. Odnotowane wskaźniki śmiertelności były bardzo zróżnicowane (od około 7% do ponad 60%), co odzwierciedla różnice w populacjach pacjentów, nasileniu ognisk oraz zasobach opieki zdrowotnej. Do najczęściej wskazywanych źródeł transmisji należały rezerwuary środowiskowe, takie jak zlewy, sprzęt medyczny i sąsiadujące powierzchnie. W dalszej kolejności wymieniano ręce personelu

medycznego oraz instalacje wodne. Metody typowania molekularnego, a zwłaszcza elektroforeza żelowa w polu pulsacyjnym oraz sekwencjonowanie całogenomowe, potwierdziły transmisję klonalną w znacznej części ognisk i okazały się kluczowe dla ich rozpoznania i opanowania. Skuteczne działania kontrolne obejmowały zwiększony nacisk na higienę rąk, dokładną dekontaminację środowiska, kohortację skolonizowanych lub zakażonych noworodków oraz czasową reorganizację lub zamknięcie obszarów OITN dotkniętych zakażeniem. *S. marcescens* pozostaje uporczywą przyczyną ognisk epidemicznych wśród noworodków, co podkreśla konieczność ciągłego nadzoru, monitorowania środowiska szpitalnego oraz wdrażenia standaryzowanych strategii prewencyjnych, zwłaszcza w warunkach ograniczonych zasobów.

Słowa kluczowe: kontrola zakażeń, ognisko zakażenia, OITN, *Serratia marcescens*, zakażenie noworodków

INTRODUCTION

Neonatal intensive care units (NICUs) are particularly vulnerable to nosocomial outbreaks due to their complex care environment, the frequent use of invasive procedures, and prolonged hospitalisation. *Serratia marcescens* has been identified as a recurrent cause of such outbreaks, demonstrating both environmental persistence and intrinsic or acquired resistance to commonly used antibiotics[1]. Its ability to cause prolonged and phenotypically diverse outbreaks in NICUs underscores its complex epidemiology and adaptability within hospital settings. Environmental survival, variable antimicrobial resistance, and multifocal transmission routes contribute to the pathogen's persistence. These factors make timely detection and effective containment especially challenging in neonatal care[2]. Infections caused by *S. marcescens* often arise from its remarkable ability to colonise hospital surfaces, medical equipment, and even disinfectant solutions. This pathogen is frequently associated with both sporadic and epidemic cases in neonatal populations, and it can result in severe clinical outcomes, such as bloodstream infections and meningitis[3]. Environmental reservoirs – particularly sink drains and other moist surfaces – play a crucial role in sustaining and spreading *S. marcescens* in NICUs. These reservoirs may act as unnoticed sources of colonisation, contributing to persistent transmission cycles despite standard infection control practices[4]. Less common vectors, such as contaminated donor human milk, have also been implicated in *S. marcescens* transmission, emphasising the potential for underrecognised reservoirs in routine neonatal care[5]. Moreover, outbreaks often necessitate enhanced infection control measures, as conventional protocols may be insufficient when environmental contamination is widespread or sources of colonisation remain unidentified[3]. The aim of this review was to summarise published reports on *S. marcescens* infections in NICUs between 2015 and 2025, with a focus on outbreak characteristics, transmission pathways, clinical outcomes, and infection control strategies.

MATERIALS AND METHODS

A narrative literature review was conducted to identify reports of *S. marcescens* infections and outbreaks in NICUs, with a focus on clinically relevant outbreak reports and surveillance studies. The literature search was performed using PubMed/MEDLINE, Scopus, and Web of Science databases. The search strategy combined the following keywords and Boolean operators: "*Serratia marcescens*" AND ("neonate" OR "newborn") AND ("NICU" OR "neonatal intensive care unit") AND ("outbreak" OR "infection" OR "colonisation"). The search was limited to articles published in English between January 2015 and December 2025. Reference lists of relevant articles were also manually screened to identify additional eligible publications. Eligible studies included publications reporting original data on colonisation or infection among neonates in NICUs, based on outbreak investigations, surveillance studies, or case series. Studies focusing on non-neonatal populations or lacking direct clinical relevance were excluded. From included studies, key epidemiological and clinical data were extracted, including geographic location, number of affected infants, clinical presentations, mortality, suspected or confirmed transmission sources, antimicrobial resistance patterns, diagnostic approaches, and infection control interventions. Because of heterogeneity in study design and reporting, findings were synthesised descriptively. A summary of the literature search and study selection process is presented in Tab. 1.

Ethics committee approval and informed consent were not required because this study is a narrative review of previously published literature.

RESULTS

Microbiological and epidemiological characteristics of *S. marcescens*

S. marcescens is a motile, facultatively anaerobic, Gram-negative bacillus of the Enterobacteriaceae family. It is commonly found in moist natural environments such as water, soil, plants, and animals, and is the species within

Step	Description
Records identified	Articles identified through database searches (PubMed, Scopus, Web of Science)
Screening	Titles and abstracts screened for relevance
Eligibility	Full-text articles assessed for eligibility
Included studies	Studies included in the narrative synthesis

Tab. 1. Summary of literature search and study selection process

 DOI: 10.15557/PiMR.2026.0002

the genus most frequently associated with human disease[3]. The organism can survive across a wide range of temperatures (5–40°C) and pH levels[5–9], facilitating its persistence in hospital environments. Considerable genetic variability has been described, with multiple clones exhibiting distinct phenotypic characteristics[3]. Several virulence mechanisms contribute to the ability of *S. marcescens* to persist and spread in NICUs. These include biofilm formation, adherence mediated by mannose-sensitive and mannose-resistant pili, and the production of extracellular enzymes such as lipases, chitinases, and chloroperoxidases. Lipopolysaccharides contribute to endotoxin activity, while pigment production (prodigiosin) has been inversely associated with virulence in some strains[3]. Antimicrobial resistance represents a major clinical concern. *S. marcescens* is intrinsically resistant to several antibiotic classes, including ampicillin and first-generation cephalosporins, and frequently acquires additional resistance mechanisms such as extended-spectrum β-lactamases and metallo-β-lactamases. Due to heterogeneous reporting across outbreaks, a consistent temporal trend in multidrug-resistant (MDR) or extensively drug-resistant (XDR) prevalence could not be established. However, several outbreaks have reported MDR *S. marcescens*, including carbapenemase-producing strains harbouring genes such as bla_{KPC} and bla_{NDM}[6–9]. Reduced susceptibility to disinfectants, including chlorhexidine and quaternary ammonium compounds, has also been reported, supporting the organism's capacity to persist in healthcare environments[3]. In NICUs, *S. marcescens* acts as an opportunistic pathogen capable of colonising and persisting in humid reservoirs such as sink drains and respiratory equipment[10]. Molecular epidemiological investigations, particularly those using pulsed-field gel electrophoresis, have demonstrated that many outbreaks involve clonal transmission. Spatially dispersed cases without direct patient-to-patient contact have been described, suggesting indirect transmission via healthcare workers or shared equipment[10]. Colonisation is especially common among preterm neonates due to frequent invasive procedures, mechanical ventilation, and antibiotic exposure. The gastrointestinal tract serves as the principal reservoir, and persistent colonisation increases the risk of subsequent invasive infection and may contribute to polyclonal transmission patterns[11]. Importantly, detection of *S. marcescens* in even a single neonate should be regarded as an epidemiological warning signal. In one outbreak, colonisation was identified through active screening prior to symptom onset, with genetically identical isolates recovered from all affected neonates[12]. Despite negative environmental cultures, case distribution and shared healthcare personnel indicated healthcare-associated transmission. The high transmissibility of *S. marcescens* and its potential to cause severe infections such as sepsis and meningitis underscore the need for immediate and comprehensive infection control measures once the pathogen is detected[12].

Clinical manifestations and outcomes in neonates

S. marcescens is associated with a broad spectrum of clinical presentations in neonates, ranging from asymptomatic colonisation to severe, life-threatening invasive infections. The most commonly reported infections in NICUs include late-onset sepsis, meningitis, pneumonia, urinary tract infections, and, less frequently, brain abscesses and conjunctivitis[12]. Sepsis remains the predominant clinical manifestation, particularly among preterm infants with indwelling central lines or prolonged mechanical ventilation. These infections are typically nosocomial and often present with nonspecific clinical signs such as temperature instability, apnoea, feeding intolerance, or respiratory distress. Mucosal barrier disruption may facilitate hematogenous spread, leading to central nervous system involvement, including meningitis and, in rare cases, cerebral abscess formation[12]. Disease severity is closely linked to host vulnerability. Extremely preterm neonates, infants with very low birth weight (VLBW), and those requiring invasive support such as central venous catheterisation or endotracheal intubation are at significantly increased risk of progression from colonisation to invasive infection[11]. Immunological immaturity and underdeveloped mucocutaneous barriers in this population further facilitate systemic dissemination. Mortality and morbidity associated with *S. marcescens* infections remain considerable. Reported outbreak-related mortality rates generally range from 10% to over 30%, depending on strain virulence, timeliness of diagnosis, and the adequacy of supportive and antimicrobial therapy[10,12]. Markedly higher mortality rates – exceeding 60% – have been described predominantly in low- and middle-income country (LMIC) settings, where diagnostic resources and access to second-line antimicrobials are limited. Survivors frequently experience prolonged hospitalisation and may develop long-term complications, including neurological sequelae following central nervous system involvement or bronchopulmonary dysplasia related to extended ventilatory support. A critical epidemiological feature of *S. marcescens* is its ability to silently colonise neonates before progression to invasive disease. In several outbreaks, colonisation was initially identified through surveillance cultures, with clinical infections developing days or weeks later in a subset of colonised infants[10,12]. In one documented NICU outbreak, two early cases presented with severe complications: one infant developed fatal sepsis with multiple brain abscesses, while another experienced seizures and long-term neurological sequelae following cerebral abscess rapture[13]. In a Moroccan NICU outbreak, eight preterm neonates developed nosocomial sepsis; 88% required mechanical ventilation, and overall mortality was 62.5%. Clinical manifestations included sepsis, pneumonia, and central nervous system involvement despite escalation of antimicrobial therapy[14]. During an outbreak

DOI: 10.15557/PiMR.2026.0002

in a Canadian NICU, 16 preterm neonates were affected, presenting with bloodstream infections, conjunctivitis, and necrotising enterocolitis, with a reported mortality rate of 12.5%. Whole-genome sequencing confirmed clonal transmission[15]. Rare but severe presentations have also been reported. Congenital brain abscess caused by *S. marcescens* has been described in a preterm neonate with suspected intrauterine infection; despite neurosurgical intervention and prolonged antimicrobial treatment, the infant remained at risk for long-term neurological sequelae[16]. A multicentre study from LMICs identified *S. marcescens* among the leading Gram-negative pathogens causing neonatal sepsis, frequently exhibiting resistance to third-generation cephalosporins and aminoglycosides, with high associated mortality[17]. In contrast, an outbreak in Switzerland involving 20 preterm neonates colonised with *S. marcescens* did not result in invasive infections and was successfully contained through a hospital-wide hand hygiene campaign, underscoring the role of person-to-person transmission[18]. Finally, a case-control study from Mexico involving 70 neonates with culture-confirmed *S. marcescens* sepsis reported a mortality rate of 32.9%. Most cases represented late-onset sepsis, with central venous catheter use, prior mechanical ventilation, and recent meropenem therapy identified as key risk factors, highlighting modifiable clinical practices relevant to prevention[19].

Transmission pathways and environmental reservoirs in NICUs

S. marcescens spreads in NICUs via multiple transmission routes, complicating outbreak detection and control. Outbreak investigations have demonstrated that the organism can persist in hospital environments and disseminate through both direct and indirect pathways[1,4,15,18]. Environmental reservoirs are particularly important in colonisation and infection, particularly on moist surfaces that are difficult to disinfect, including sink drains, ventilator circuits, infusion tubing, incubator components, and soap dispensers[1,4,15,20]. Several outbreaks have established a clear association between environmental contamination and neonatal colonisation or infection. In a Mexican NICU, *S. marcescens* was isolated from multiple environmental sites and care products, with genetic analyses confirming relatedness between environmental and clinical isolates[1]. Similarly, in a Canadian NICU outbreak, clonal transmission between environmental reservoirs and neonates was demonstrated, with colonisation preceding invasive infection despite routine cleaning measures[15]. Sink drains, in particular, serve as significant reservoirs due to biofilm formation, which facilitates prolonged bacterial survival and dissemination to surrounding surfaces and the hands of healthcare workers[4].

Less common transmission pathways have also been reported. In a German NICU outbreak, raw donor human milk was identified as the source of infection using molecular typing methods, highlighting enteral feeding as a potential transmission route when handling or storage practices are inadequate[5]. The role of healthcare workers in transmission has been documented in multiple outbreaks, especially in settings with suboptimal hand hygiene or shared equipment. In Switzerland and Ecuador, outbreaks were effectively controlled by reinforcing hand hygiene and contact precautions, even when environmental sampling failed to identify a definitive source[18,21]. Molecular epidemiological approaches, particularly whole-genome sequencing, have enhanced understanding of *S. marcescens* transmission dynamics. Studies from Italy and Spain identified genetically related strains among neonates and environmental samples, supporting targeted infection control measures such as cohorting and enhanced surveillance[6,22]. In addition to environmental factors, device-associated practices – including central venous catheterisation, mechanical ventilation, and prior exposure to broad-spectrum antibiotics – have been identified as independent risk factors for *S. marcescens* sepsis, reflecting increased susceptibility to nosocomial transmission in highly invasive care settings[19].

In summary, *S. marcescens* transmission in NICUs is driven by environmental persistence, contaminated equipment or fluids, healthcare worker-mediated spread, and invasive medical devices. Effective prevention and control rely on environmental monitoring, strict adherence to hand hygiene, careful management of invasive procedures, and integration of molecular tools to support outbreak investigation and containment[20,23].

Outbreak reports: summary of recent literature (2015–2025)

Published outbreak investigations and surveillance reports from 2015 to 2025 indicate that *S. marcescens* remains a significant nosocomial threat in NICUs worldwide, with outbreaks reported across Latin America, Europe, the Middle East, Africa, and Asia. Most reports originated from tertiary NICUs and were based on retrospective cohort analyses, outbreak investigations, or case series. A limited number of review articles provided aggregated insights across multiple outbreaks[10,19]. The number of affected neonates per outbreak ranged from isolated cases to clusters involving more than 70 patients. Preterm infants – particularly those with birth weights below 1,500 g or requiring invasive support – were disproportionately affected. Clinical manifestations were predominantly characterised by late-onset sepsis, followed by meningitis, conjunctivitis, pneumonia, and urinary tract infections. Across reported outbreaks, sepsis was the most frequent presentation, while other clinical manifestations occurred less commonly[12–15].

Mortality rates varied widely, from approximately 7% to over 60%, with the highest case fatality reported in resource-limited settings or during outbreaks involving

 DOI: 10.15557/PiMR.2026.0002

multidrug-resistant strains[12,24–26]. Identified sources of infection included a range of environmental and procedural vectors. Based on published outbreak reports, environmental surfaces were most frequently implicated (63%), followed by healthcare workers' hands (37%), and water-related sources such as sinks and taps (24%). Less commonly, ventilator circuits and equipment, incubators, milk or formula, and infusion systems were identified as sources of transmission[1,13–15,26]. Advanced molecular typing methods, particularly pulsed-field gel electrophoresis (PFGE) and whole-genome sequencing (WGS), played a central role in confirming clonal transmission and elucidating outbreak dynamics. In a substantial proportion of outbreaks, PFGE or WGS demonstrated genetic relatedness between clinical and environmental isolates, supporting hypotheses of horizontal transmission and environmental persistence[19,22,27]. Several reports have documented clinically relevant resistance determinants, including carbapenemase-encoding genes, underscoring the increasing challenge posed by multidrug-resistant *S. marcescens* in neonatal care settings[10,27]. Outbreak control strategies were typically multifaceted and, in selected outbreaks, required not only cohorting and enhanced environmental decontamination but also discontinuation of contaminated products or replacement of implicated infrastructure[13,15,24,28]. Auditing and reinforcement of hand hygiene practices were consistently emphasised across outbreaks[14,15,29].

Tab. 2 summarises selected reported NICU outbreaks from 2015 to 2025, highlighting outbreak size, clinical presentation, mortality, antimicrobial resistance, and suspected or confirmed sources of transmission.

DISCUSSION

Wide variation in reported mortality rates across *S. marcescens* outbreaks was most consistently associated with the healthcare setting and baseline patient vulnerability. The highest mortality was predominantly reported in low- and middle-income neonatal units, where delayed outbreak recognition, limited diagnostic capacity, restricted access to second-line antimicrobials, and a high proportion of extremely preterm infants were common. Multidrug-resistant and carbapenem-resistant *S. marcescens* strains were present in several high-mortality outbreaks; however, resistance alone did not fully account for the observed differences. In contrast, outbreaks in well-resourced NICUs were generally associated with lower mortality, even when resistant strains were involved, reflecting earlier detection and more effective infection control measures. Although sepsis and bloodstream infections were the predominant clinical presentations, several reports also described less common manifestations, including conjunctivitis and persistent respiratory tract involvement or colonisation. These findings were often observed in the context of ongoing transmission and, in some cases, preceded invasive disease, suggesting their potential role as early clinical warning signs in NICU outbreaks[10,15,24,27]. In summary, *S. marcescens* outbreaks in NICUs are frequently driven by environmental reservoirs and horizontal transmission, often exacerbated by invasive procedures and compromised infection control. Early molecular identification of clonal patterns and strict environmental surveillance are critical for prompt containment. Given its ability to colonise and persist in hospital environments, *S. marcescens* warrants sustained vigilance and continuous adaptation of infection prevention strategies in neonatal care. Effective control of *S. marcescens* outbreaks in NICUs requires a multifaceted and proactive approach. Outbreak reports emphasise the importance of active surveillance and environmental cultures, including regular screening of high-risk neonates and systematic culturing of sinks, incubators, and equipment, which have enabled early containment of transmission chains[27]. Hand hygiene and equipment decontamination remain cornerstones of prevention. In several outbreaks, failures in hand hygiene adherence were directly implicated, whereas reinforcement campaigns and auditing proved decisive in halting further transmission[14]. Several reports have also highlighted the importance of environmental decontamination and ward reorganisation. In several outbreaks, removal or replacement of contaminated sinks, modification of water outlets, and temporary closure or reorganisation of affected NICU areas coincided with cessation of transmission. However, these measures were implemented alongside standard infection control interventions rather than as standalone actions. Deep cleaning of affected areas and replacement of contaminated infrastructure were required to eliminate persistent environmental reservoirs[27]. Although less common, contamination of donor human milk and infant formula has been reported as a source of transmission. These findings underscore the need for systematic microbiological testing of donated and stored milk prior to use in vulnerable neonates[19]. Earlier outbreaks were most commonly investigated using pulsed-field gel electrophoresis, which demonstrated clonal transmission but offered limited resolution[18]. In more recent outbreaks (2020–2025), whole-genome sequencing and other next-generation sequencing approaches have been increasingly applied[6,13]. High-resolution molecular typing enabled more precise discrimination between closely related strains, improved linkage of clinical and environmental isolates, and refined understanding of transmission dynamics in NICU settings. Taken together, these findings highlight that the most successful interventions combine active surveillance, strict hygiene, environmental decontamination, and molecular epidemiology, all implemented through a multidisciplinary team approach.

CONCLUSIONS

Outbreaks of *S. marcescens* continue to occur in NICUs, primarily affecting preterm infants and neonates requiring

DOI: 10.15557/PiMR.2026.0002

Authors (year)	Country/centre	Article type	Number of cases	Clinical manifestation	Outcome/ mortality	Resistance/ therapy	Source of infection
Rodríguez-Villodres et al. (2023)	Spain – University Hospital Virgen del Rocío, Seville	Outbreak report and implementation of qPCR surveillance	35 (4 sepsis; remainder colonised)	Sepsis in 4; mostly asymptomatic colonisation	No deaths reported	Not reported	Environmental contamination (sinks, incubators; multiclonal)
Howard-Jones et al. (2022)	Australia, Sydney (NICU)	Outbreak report + WGS	10 (2 infections, 8 colonised)	Sepsis, brain abscess, seizures; others colonised only	1 death; 1 neurological sequela	Susceptible to cefepime, meropenem, gentamicin	Likely contact transmission; WGS showed two clusters
Daoudi et al. (2018)	Morocco, Marrakesh	Outbreak report	8 (all sepsis)	Sepsis; most ventilated	5/8 deaths (63%)	Sensitive to imipenem/ amikacin; colistin-resistant	Likely environmental/ HCW transmission
Montagnani et al. (2015)	Italy, Florence	Outbreak report + typing	25 (14 infections, 16 colonised)	Sepsis (1), conjunctivitis (2), others colonised	No deaths reported	Resistant to colistin and amoxicillin-clavulanate; some gentamicin/ ceftazidime resistance	Likely HCW hands (external consultants)
Böhne et al. (2021)	Germany, Hannover (NICU)	Outbreak cluster	4 preterm infants (2 twin pairs)	Colonisation; 1 sepsis	No deaths reported	Susceptible to cephalosporins, fluoroquinolones, carbapenems	Likely HCW hands
Martineau et al. (2018)	Canada – NICU, CHU de Québec–Université Laval	Outbreak report + genomic investigation (WGS and metagenomics)	15 neonates	Sepsis, conjunctivitis, respiratory infection, asymptomatic colonisation	1/15 death (sepsis-related)	High-level colistin resistance; one carbapenem-resistant isolate; therapy not specified	Tap drain biofilm (environmental reservoir, WGS-confirmed)
Čičová et al. (2024)	Slovakia, Trenčín	Case report/ Outbreak description	3 neonates	Sepsis, meningitis/ meningoencephalitis, brain abscess, colonisation	No deaths reported; severe neurological sequelae in one case	MDR strains; resistant to colistin and several β-lactams; susceptible to meropenem and aminoglycosides; dual antibiotic therapy used	Likely sink biofilm/ HCW hands (environmental swabs negative)
Moradigaravand et al. (2016)	United Kingdom and Ireland – multiple hospitals including neonatal and adult ICUs	Genomic surveillance/ outbreak and transmission analysis	13 neonates	Sepsis and colonisation (details limited)	No deaths reported	MDR; ESBL ($bla_{CTX\text{-}M\text{-}15}$); resistance to cephalosporins, fluoroquinolones, aminoglycosides; therapy not specified	Nosocomial transmission confirmed
Escribano et al. (2019)	Spain, Madrid	Prospective observational study during an outbreak	10 neonates	Asymptomatic colonisation; 1 sepsis	No deaths reported	Not reported	Likely horizontal transmission in NICU
Tahiri et al. (2023)	Morocco, Marrakech	Outbreak report/ retrospective case series	30 neonates	Sepsis (all cases); meningitis; respiratory infections; few jaundice, conjunctivitis, UTIs	21/30 deaths (70%)	Multidrug-resistant; resistance to most cephalosporins and colistin; meropenem + amikacin commonly used	Likely HCWs/ invasive devices
Guel-Gomez et al. (2023)	Mexico, Sinaloa	Outbreak report/ retrospective case series	15 neonates	Sepsis (most cases); respiratory distress; jaundice; GI symptoms	1 death	Strain mostly susceptible; ampicillin, amikacin, cefotaxime, occasionally meropenem	Likely cross-transmission via devices/HCWs

Tab. 2. Selected reported outbreaks of S. marcescens *in NICUs (2015–2025)*

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Henares et al. (2025)	Spain, Barcelona	Outbreak investigation, prospective study	16 neonates	Sepsis (5); colonisation at multiple sites	No deaths reported	Resistant to early β-lactams; mostly susceptible to later-generation cephalosporins and carbapenems; vancomycin + meropenem used	WGS showed patient-to-patient transmission (three clusters)
Lona-Reyes et al. (2025)	Mexico, Guadalajara	Case-control study	70 neonates	Sepsis (≈99%); some CNS involvement	23/70 deaths (32.9%)	High susceptibility to ceftriaxone, cefepime, amikacin, meropenem; no molecular typing	Likely nosocomial (invasive devices)
Yeo et al. (2020)	Singapore	Outbreak investigation	11 neonates	1 bacteraemia; 5 conjunctivitis; others colonised	No deaths reported	Ceftazidime for BSI; topical aminoglycosides for conjunctivitis; no treatment for colonisation	Likely environmental/ HCW transmission
Bechmann et al. (2023)	Germany, Magdeburg	Outbreak investigation	17 neonates	Mainly colonisation; 1 meningitis + sepsis (fatal), 1 sepsis	1 death	Susceptible strains; meropenem used empirically	Raw donor milk (confirmed by PFGE/WGS)
Toorop et al. (2024)	Netherlands, Leiden	Outbreak investigation	20 neonates	Mostly colonisation; sepsis and conjunctivitis in a few cases	No deaths reported	Non-MDR strains; aac(6′)-Ic gene detected; temporary empiric meropenem use	Likely HCW transmission
Merla et al. (2024)	Italy, Pavia	Outbreak investigation (short report)	8 neonates	Conjunctivitis; asymptomatic colonisation in two cases	No deaths reported	Wild-type isolates; only one isolate with AmpC (*blaCMY-70*)	Likely patient-to-patient transmission
Soria et al. (2016)	Ecuador, Guayaquil	Outbreak investigation	21 neonates	Respiratory-origin sepsis; conjunctivitis; septic arthritis; surgical site infection	3/9 deaths	Resistant to 3rd/4th gen cephalosporins, cotrimoxazole, gentamicin; susceptible to amikacin and carbapenem	Likely HCW transmission
Zingg et al. (2017)	Switzerland, Geneva	Outbreak investigation	20 neonates	Only colonisation (rectal/nasal swabs)	No deaths	Not MDR; genotyping showed clonal strains (identical by fingerprinting, closely related by WGS)	Likely cross-transmission via HCWs' hands; one suspected maternal amnion infection syndrome as possible origin

BSI – bloodstream infection; **HCW** – healthcare worker; **MDR** – multidrug-resistant; **PFGE** – pulsed-field gel electrophoresis; **WGS** – whole-genome sequencing.

Tab. 2. Selected reported outbreaks of S. marcescens *in NICUs (2015–2025) (cont.)*

invasive medical support. Sepsis is the most frequently reported clinical manifestation, while mortality varies considerably across settings, depending on antimicrobial resistance patterns, availability of diagnostic and therapeutic resources, and timeliness of outbreak recognition. Evidence from the reviewed studies indicates that transmission most often involves environmental reservoirs, healthcare workers' hands, and water-associated sources. Effective outbreak control depends on early identification of colonisation, consistent adherence to infection prevention measures, and integration of molecular typing methods into surveillance practices. Ongoing epidemiological monitoring and structured prevention strategies are essential in all neonatal units, with particular emphasis on settings with limited resources, to support timely outbreak containment and improve clinical outcomes.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study; final approval of manuscript: SM. Collection, recording and/or compilation of data; analysis and interpretation of data; writing of manuscript; critical review of manuscript: SM, IM.

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