FEATURES OF SEPSIS IN INFANTS AT THE PRESENT STAGE

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Abstract. Sepsis remains one of the leading causes of neonatal morbidity and mortality worldwide. Despite the advances in neonatal care, early diagnosis and treatment of sepsis in infants remain a significant challenge due to nonspecific clinical presentations and rapidly progressing disease. This review highlights the epidemiology, risk factors, clinical features, diagnostic approaches, and modern treatment strategies of neonatal sepsis in the current era of medicine.

Keywords: neonatal sepsis, infants, clinical features, diagnosis, immune response

Introduction. Neonatal sepsis is a systemic inflammatory response syndrome caused by infection, occurring in infants during the first 28 days of life. It is classified as early-onset sepsis (EOS) or late-onset sepsis (LOS) depending on the time of onset. The global incidence of neonatal sepsis remains high, particularly in low- and middle-income countries [4].

Epidemiology and Risk Factors. The incidence of neonatal sepsis varies by region, with reports ranging from 1 to 10 cases per 1000 live births [3]. Major risk factors include premature birth, low birth weight, maternal infections, prolonged rupture of membranes, and invasive procedures during delivery. In developed countries, group B Streptococcus and Escherichia coli are leading

causative agents, while in developing regions, Klebsiella, Pseudomonas, and other Gram-negative organisms are prevalent [3].

The etiology of neonatal sepsis is multifactorial and varies depending on the time of onset—early-onset sepsis (EOS) (≤72 hours of life) versus late-onset sepsis (LOS) (>72 hours). The underlying causes include vertical transmission from the mother, postnatal exposure to pathogens, immaturity of the neonatal immune system, and exposure to invasive medical procedures.

Bacterial Pathogens. Early-onset sepsis (EOS) is commonly associated with pathogens acquired during delivery. The most frequent causative organisms include: Group B Streptococcus (GBS) – the leading cause of EOS in developed countries. It colonizes the maternal genital tract and is transmitted to the infant during labor [3]. Escherichia coli – especially in preterm and very-low-birth-weight infants. It is often multidrug-resistant, complicating treatment [4]. Listeria monocytogenes – transmitted via contaminated food or maternal colonization; less common but associated with severe outcomes. Late-onset sepsis (LOS) is often due to environmental and nosocomial pathogens, including: Coagulase-negative staphylococci (CoNS) – particularly Staphylococcus epidermidis, associated with catheter-related bloodstream infections.Staphylococcus aureus – including methicillin-resistant S. Aureus (MRSA).Klebsiella spp., Pseudomonas aeruginosa, Enterobacter spp. – especially in neonatal intensive care units (NICUs), where antibiotic resistance is a growing concern [5; 2]. Fungal pathogens (e.g., Candida albicans) – more common in infants with prolonged hospital stays, antibiotic therapy, or central venous catheters. While less common, viral infections (e.g., herpes simplex virus, enteroviruses, cytomegalovirus) can cause sepsis-like syndromes in neonates and are associated with high mortality if untreated. Invasive fungal infections, primarily Candida species, contribute significantly to morbidity in premature infants, particularly those with central lines or on broad-spectrum antibiotics [2]. Beyond pathogens, the neonate's

immune system plays a critical role in susceptibility. Key contributing factors include: Immature innate and adaptive immunity – reduced neutrophil function, low complement levels, and impaired cytokine response increase vulnerability [5]. Disruption of skin and mucosal barriers – due to prematurity or medical interventions such as mechanical ventilation and central lines. Maternal factors – including chorioamnionitis, urinary tract infections, fever during labor, and colonization with pathogenic flora.

Clinical manifestations in infants are often subtle and nonspecific. Infants, especially preterm neonates, may present with subtle and non-specific symptoms: temperature instability (hypothermia or fever), lethargy, irritability, poor feeding, apnea, bradycardia, or desaturation episodes, respiratory distress or increased oxygen requirement, jaundice, abdominal distension, hepatosplenomegaly, hypotension, delayed capillary refill. These signs necessitate further laboratory testing and should prompt immediate evaluation [5; 6]. The diagnosis of neonatal sepsis is based on a combination of clinical signs, laboratory markers, and microbiological confirmation. However, the clinical presentation is often nonspecific, and reliance solely on symptoms may lead to both over- and under-diagnosis [3].

1. Laboratory Markers. Hematologic indicators: Complete Blood Count (CBC): Leukopenia (<5000 cells/μL) or leukocytosis (>25,000 cells/μL). Immature-to-total neutrophil ratio (I/T ratio) >0.2. Thrombocytopenia (<150,000/μL) [6].

Inflammatory markers: C-reactive protein (CRP): Elevated >10 mg/L. Procalcitonin (PCT): Increased >2 ng/mL is more specific than CRP in early diagnosis [8]. Interleukins (e.g., IL-6, IL-8): Elevated early in infection; limited by cost and availability. Serum lactate: May be elevated in severe cases, indicating tissue hypoperfusion.

Microbiological Confirmation. Blood culture is the gold standard and should be performed before starting antibiotics. A minimum of 1 mL of blood should be obtained to increase diagnostic yield [3]. Cerebrospinal fluid (CSF) analysis: Recommended if meningitis is suspected; cell count, glucose, protein, and culture should be performed. Urine culture: Especially in late-onset sepsis, obtained by catheterization or suprapubic aspiration. Surface cultures (e.g., skin, umbilicus) may be helpful in surveillance but have low specificity [6].

Empirical antibiotic therapy should begin immediately after sepsis is suspected. Current recommendations include ampicillin plus gentamicin for early-onset sepsis, and vancomycin plus third-generation cephalosporins for late-onset cases [3]. Treatment is tailored based on culture results. Timely and appropriate empirical antibiotic therapy significantly improves outcomes and reduces mortality.

1. Empirical Antimicrobial Therapy. Early-Onset Sepsis (EOS). Group B Streptococcus, Escherichia coli, Listeria monocytogenes Empirical regimen**:** Ampicillin (for GBS and Listeria), Gentamicin (for Gram-negative coverage); Alternative in penicillin allergy: Vancomycin or third-generation cephalosporins (with caution). Adjustments**:** If E. coli is confirmed and shows resistance, use cefotaxime instead of gentamicin [4].

Late-Onset Sepsis (LOS). Coagulase-negative staphylococci, Staphylococcus aureus *(including MRSA), Gram-negative bacilli (*Klebsiella spp., Pseudomonas aeruginosa*), and* Candida Empirical regimen**:** Vancomycin (for MRSA and CoNS), **c**efotaxime or ceftazidime (for Gram-negative coverage, including *Pseudomonas*); In case of suspected fungal sepsis**:** Amphotericin B deoxycholate (first-line), Fluconazole (alternative for hemodynamically stable neonates or prophylaxis). Targeted Therapy based on Culture Results. Supportive and Adjunctive Therapy: Fluid resuscitation - Crystalloids (e.g., normal saline) to

maintain perfusion. Inotropes**:** Dopamine or dobutamine for hypotension or shock. Oxygen and mechanical ventilation: For respiratory distress or apnea. Parenteral nutrition: Especially for very-low-birth-weight infants. Blood transfusion: If hemoglobin is <10 g/dL in critically ill neonates. Immunoglobulins: The use of intravenous immunoglobulins (IVIG) remains controversial; some studies show limited benefit.

Conclusion. Sepsis in infants remains a medical emergency with high mortality and long-term complications. Improved diagnostics, timely treatment, and research into novel therapeutic strategies are essential. A multidisciplinary approach and continued emphasis on prevention are key to improving outcomes.

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