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Pediatric Sepsis: Early Recognition, Risk Stratification and Evidence-Based Management

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Abstract

Pediatric sepsis remains a time-dependent emergency with substantial mortality, morbidity and long-term functional consequences. Its early diagnosis is difficult because the first manifestations in children often overlap with common febrile illnesses, while hypotension may appear only after compensatory mechanisms have begun to fail. This narrative review discusses current evidence on early recognition, risk stratification and initial management of pediatric sepsis and septic shock, with emphasis on emergency departments, pediatric wards and pediatric intensive care units. The review addresses the transition from systemic inflammatory response syndrome-based concepts to organ dysfunction-centered definitions, including the Phoenix criteria and the Phoenix Sepsis Score. It also summarizes the clinical utility and limitations of pediatric severity scores, including the Pediatric Sequential Organ Failure Assessment score, Pediatric Logistic Organ Dysfunction-2, quick Pediatric Logistic Organ Dysfunction-2 and biomarker-based models such as the Pediatric Sepsis Biomarker Risk Model.

From a therapeutic perspective, the main principles are rapid clinical recognition, prompt antimicrobials when septic shock is suspected, individualized fluid resuscitation with frequent reassessment, early vasoactive support when shock persists, source control when feasible and continuous monitoring of perfusion, respiratory status, renal function, fluid balance and organ dysfunction. Current evidence supports structured sepsis pathways and bundled care, but important controversies remain regarding universal screening tools, optimal fluid volume and type, vasoactive selection, lactate thresholds and the best strategy to balance early antimicrobial therapy with stewardship. Therefore, pediatric sepsis care should combine validated definitions, bedside clinical judgment, institutional protocols and serial reassessment rather than relying on any single score, biomarker, or alert system.

Keywords: Pediatric sepsis, Septic shock, Early diagnosis, Risk stratification, Intensive care, Antimicrobial therapy, Fluid resuscitation, Phoenix criteria

Introduction

Pediatric sepsis is a complex clinical syndrome in which infection triggers a dysregulated host response that may progress to life-threatening organ dysfunction, shock and multiple organ failure. In children, the clinical spectrum ranges from suspected infection with early signs of physiologic deterioration to septic shock with cardiovascular failure and persistent tissue hypoperfusion. The Surviving Sepsis Campaign emphasizes that timely recognition and coordinated resuscitation are essential because pediatric

septic shock can deteriorate rapidly and because delays in antimicrobial therapy and hemodynamic support are associated with worse outcomes [1].

The most recent international pediatric sepsis consensus shifted the definition away from systemic inflammatory response syndrome alone and toward infection-associated organ dysfunction. The Phoenix criteria define pediatric sepsis as suspected or confirmed infection plus a Phoenix Sepsis Score of at least two points, reflecting dysfunction in respiratory, cardiovascular, coagulation and neurologic systems. Septic shock is defined as sepsis with at least one cardiovascular point in that score, which may reflect severe age-adjusted hypotension, lactate above 5 mmol/L, or vasoactive drug use [2,3].

This conceptual transition is clinically relevant. Fever, tachycardia, tachypnea, poor feeding, irritability, lethargy and malaise are common in many childhood infections and are not specific for sepsis. Conversely, hypotension is often a late sign in children because heart rate, systemic vascular resistance and other compensatory mechanisms may preserve blood pressure despite progressive circulatory failure. Therefore, early recognition depends on integrating age-adjusted vital signs, perfusion markers, mental status, respiratory effort, urine output, risk factors and repeated bedside reassessment [1,2].

The burden of pediatric sepsis remains substantial. The Sepsis Prevalence, Outcomes and Therapies Study reported that severe sepsis represented approximately 8.2% of pediatric intensive care unit admissions across 128 pediatric intensive care units in 26 countries, with hospital mortality near 25%. The impact is unevenly distributed, with higher mortality and delayed access to advanced support in low- and middle-income settings [4,5]. This review summarizes current evidence on early recognition, risk stratification and evidence-based management of pediatric sepsis and septic shock, translating the available literature into practical clinical principles for emergency, ward and intensive care settings.

Methods

This manuscript is a narrative, descriptive and analytic review based on the source article prepared in Portuguese and adapted into English for journal submission. The guiding question was formulated using the population, concept and context framework: neonates, infants, children and adolescents as the population; diagnostic criteria, screening tools, risk stratification and evidence-based interventions as the concept; and emergency departments, pediatric wards and pediatric intensive care units as the context. The literature scope included articles and guidelines addressing pediatric sepsis definitions, early recognition, clinical warning signs, electronic alerts, organ dysfunction scores, biomarkers, antimicrobial timing, bundled care, fluid resuscitation, vasoactive support and intensive care monitoring. Priority was given to international guidelines, consensus definitions, validation studies, multicenter observational cohorts, randomized trials and systematic reviews published in the last decade, while older landmark studies were retained when they remained relevant to hemodynamic support or fluid resuscitation.

Current definitions and diagnostic concepts

Historically, pediatric sepsis definitions relied heavily on systemic inflammatory response syndrome in the presence of suspected or confirmed infection. Although this framework increased awareness, it had limited specificity because children frequently meet inflammatory criteria during uncomplicated infections, trauma, postoperative states, or noninfectious inflammation. The newer pediatric consensus definitions seek to identify children with infection and potentially life-threatening organ dysfunction rather than children with inflammation alone [2,3].

The Phoenix Sepsis Score operationalizes this concept using four organ systems: respiratory, cardiovascular, coagulation and neurologic dysfunction. Respiratory dysfunction may be reflected by PaO2/FiO2 or SpO2/FiO2 abnormalities or need for invasive respiratory support. Cardiovascular dysfunction may include severe hypotension, high lactate, or vasoactive support. Neurologic dysfunction incorporates decreased Glasgow Coma Scale score and coagulation dysfunction incorporates platelet count, international normalized ratio, D-dimer and fibrinogen abnormalities. A total score of at least two identifies pediatric sepsis in children with suspected or confirmed infection [2,3].

The Phoenix framework is not intended to replace bedside suspicion at triage. Rather, it standardizes the identification of organ dysfunction once infection is suspected. This distinction matters because a screening tool should detect possible deterioration before full organ dysfunction is established, whereas a definition classifies severity and supports epidemiology, quality improvement and research. Consequently, Phoenix criteria should be integrated with clinical pathways, age-adjusted vital signs, perfusion assessment and serial examination rather than used as an isolated trigger [2,3].

Epidemiology and clinical impact

Pediatric sepsis is both a clinical and health-system problem. In the SPROUT point-prevalence study, severe sepsis accounted for a meaningful proportion of pediatric intensive care unit admissions and was associated with high mortality, particularly among children with septic shock. Respiratory infections were a leading source, followed by bloodstream and gastrointestinal infections, although the etiologic distribution varied according to age, comorbidities, local microbiology and healthcare-associated exposures [4].

Infants, children younger than five years, premature children, malnourished children, immunosuppressed patients, children with malignancy and those with chronic disease or technology dependence are at increased risk of severe outcomes. These groups may present with subtle manifestations, including reduced feeding, abnormal behavior, respiratory pattern changes, or general clinical worsening. A low threshold for reassessment and protocol activation is therefore appropriate when infection is suspected in a high-risk child [4,5].

Survivors may experience prolonged hospitalization, mechanical ventilation, vasoactive drug exposure, invasive monitoring, acute kidney injury, neurocognitive deficits, functional limitations, psychological symptoms and reduced quality of life after

discharge. These outcomes reinforce that pediatric sepsis care should not be limited to preventing early death; it should also aim to reduce organ injury, avoid iatrogenic complications such as fluid overload and unnecessary antimicrobial exposure and plan post-intensive care follow-up when indicated [1,5].

Pathophysiology

The pathophysiology of sepsis is multifactorial and dynamic. Pathogen-associated and damage-associated molecular patterns activate innate immune pathways, endothelial cells, leukocytes, complement, coagulation cascades and inflammatory mediators. The initial response may be strongly pro-inflammatory, but immune suppression and immune paralysis may also occur, especially in critically ill or immunocompromised children. The same patient may move through different immune phenotypes during the course of illness.

Endothelial dysfunction is central to circulatory failure. Vasodilation, capillary leak, glycocalyx injury, altered microvascular flow and myocardial dysfunction reduce effective oxygen delivery. Fluid shifts from the intravascular space into the interstitium contribute to edema and relative hypovolemia, while mitochondrial and metabolic dysfunction may impair oxygen utilization even when macrocirculatory variables appear acceptable. These mechanisms explain why blood pressure alone cannot define tissue perfusion.

As shock progresses, compensatory tachycardia and vasoconstriction may become insufficient. Reduced preload, impaired contractility, low or high systemic vascular resistance, hypoxemia, anemia and altered microcirculation can interact to cause tissue hypoxia, lactate elevation, acute kidney injury, neurologic dysfunction, respiratory failure, coagulopathy and multiple organ dysfunction. Treatment must therefore be individualized and repeatedly reassessed rather than driven by a fixed volume or a single hemodynamic target [1,5].

Early recognition and clinical warning signs

Early recognition should be understood as the identification of a child with suspected infection who has signs of deterioration or increased risk of progression, not as the final confirmation of sepsis. Warning signs include persistent tachycardia, abnormal perfusion, prolonged capillary refill, weak pulses, cool extremities, mottled skin, altered mental status, reduced interaction with caregivers, respiratory distress, hypoxemia, increasing oxygen requirement, oliguria, hypotension, petechiae or purpura and rapid clinical worsening [1,5].

Because physiologic norms vary substantially by age, abnormal vital signs must be interpreted using age-adjusted thresholds. A heart rate that is concerning in an adolescent may be normal in an infant and normal blood pressure does not exclude shock. In compensated pediatric shock, tachycardia, capillary refill delay, narrow pulse pressure, cool extremities, altered behavior and decreasing urine output may precede hypotension. In distributive or vasoplegic physiology, warm extremities and bounding pulses may coexist with poor tissue oxygen delivery.

Serum lactate can assist initial assessment and follow-up because it may reflect tissue hypoperfusion, adrenergic stress, mitochondrial dysfunction, or impaired clearance. Elevated lactate supports concern for severe illness, but normal lactate does not exclude early sepsis or compensated shock. Similarly, C-reactive protein and procalcitonin may support diagnosis and monitoring but should not be used as stand-alone rule-in or rule-out tests. Biomarkers perform best when interpreted alongside history, examination, vital signs, cultures, organ function tests and the trajectory of illness [1,5].

Electronic sepsis alerts and structured screening tools may reduce missed recognition when they are embedded in clinical workflows. Vital sign-based electronic alerts, bedside clinician identification and electronic systems incorporating infection suspicion and clinical severity have been associated with improved recognition of severe pediatric sepsis in emergency settings. However, alert performance depends on implementation, thresholds, staff education, local resources and the ability of the service to respond promptly. Alerts should therefore augment, not replace, clinical judgment [6-8].

A practical recognition strategy has three levels. First, every child with suspected infection should undergo age-adjusted vital sign assessment and mental status evaluation. Second, clinicians should actively search for perfusion, respiratory, neurologic, urinary and skin warning signs. Third, a sepsis protocol should be activated when infection is associated with shock, organ dysfunction, high-risk comorbidities, or rapid deterioration. Recognition only improves outcomes when it is linked to timely action.

Risk stratification and severity scores

Risk stratification helps clinicians identify children at higher risk of deterioration, prioritize intensive monitoring, support transfer decisions and standardize comparison across studies. Scores should not replace clinical judgment, but they provide a common language for organ dysfunction and severity. The most relevant tools include the Pediatric Sequential Organ Failure Assessment score, Pediatric Logistic Organ Dysfunction-2, quick Pediatric Logistic Organ Dysfunction-2, the Phoenix Sepsis Score and biomarker-based models such as the Pediatric Sepsis Biomarker Risk Model.

The Pediatric Sequential Organ Failure Assessment score was adapted from the adult Sequential Organ Failure Assessment score to account for pediatric age-dependent physiology. It evaluates respiratory, coagulation, hepatic, cardiovascular, neurologic and renal dysfunction. In critically ill children, the maximum Pediatric Sequential Organ Failure Assessment score demonstrated strong discrimination for hospital mortality and performed comparably to established pediatric organ dysfunction scores [9]. In emergency department populations, however, a score of at least two was less sensitive as a screening trigger, showing that a score validated in intensive care may not function equally well at the front door of the hospital [10].

The Pediatric Logistic Organ Dysfunction-2 score is widely used in pediatric intensive care units to quantify organ dysfunction and estimate prognosis. In children with sepsis admitted to intensive care, day-one Pediatric Logistic Organ Dysfunction-2 showed high discrimination for hospital mortality. The quick Pediatric

Logistic Organ Dysfunction-2 score is faster and may help when data are limited, but it is less accurate than the complete score. These instruments are most useful for severity assessment and follow-up rather than as isolated early screening tools [11].

The Phoenix Sepsis Score has the advantage of being derived from a large international dataset and designed specifically to classify pediatric sepsis and septic shock. It aligns modern pediatric sepsis definitions with organ dysfunction and mortality risk. Nevertheless, its dependence on variables that may not be immediately available in all settings and its exclusion of some bedside perfusion findings, such as capillary refill time, mean it should be interpreted within the broader clinical picture.

Biomarker-based models such as the Pediatric Sepsis Biomarker Risk Model and its revised version combine inflammatory biomarkers with clinical variables to estimate baseline mortality risk in pediatric septic shock. PERSEVERE-II incorporated platelet count and has also been studied for predicting severe acute kidney injury and renal recovery. These models may refine prognostication and identify high-risk phenotypes, but their routine bedside use remains limited by availability, cost, turnaround time and the need for external validation in diverse settings [12,13].

Initial management and sepsis bundles

The initial management of pediatric sepsis should be simultaneous, structured and severity-driven. A bundle is not a rigid checklist applied identically to every child; it is a coordinated set of time-sensitive actions that typically includes recognition, vascular access, blood cultures when feasible, broad-spectrum antimicrobials, fluid resuscitation when shock is present, serial reassessment, vasoactive support when shock persists and source control when indicated [1,14].

In suspected septic shock, vascular access should be obtained promptly. If peripheral venous access cannot be achieved rapidly, intraosseous access is appropriate to avoid delaying antimicrobials, fluids, or vasoactive drugs. Initial monitoring should include heart rate, respiratory rate, oxygen saturation, blood pressure, temperature, mental status, capillary refill, pulse quality, extremity temperature, urine output, glucose and laboratory evaluation of organ dysfunction when feasible [1].

Quality improvement initiatives suggest that bundled care can improve process reliability and outcomes. The Improving Pediatric Sepsis Outcomes Collaborative reported improved adherence to recommended care processes and reductions in mortality across participating hospitals. Brazilian and other institutional experiences have also found that pediatric sepsis protocols can reduce mortality and organ dysfunction when they improve early recognition, first-hour bundle compliance and coordinated team response [14-16].

Bundles must be adapted to local resources. A hospital with immediate pediatric intensive care, ventilatory support, vasoactive medications and trained teams can pursue more aggressive hemodynamic resuscitation than a setting without those supports. In all settings, however, the central principle remains the same: suspected septic shock requires immediate evaluation, early antimicrobial therapy, restoration of perfusion, continuous reassessment and escalation of care when response is inadequate [1].

Antimicrobial therapy and source control

Early antimicrobial therapy is one of the most important interventions in pediatric sepsis. In children with suspected septic shock, empiric broad-spectrum antimicrobials should be administered as soon as possible, ideally within the first hour after recognition. In children with possible sepsis without shock, a brief diagnostic evaluation may be appropriate, but antimicrobial therapy should not be delayed when suspicion remains high or the child is deteriorating [1].

The association between delayed antimicrobial therapy and worse outcomes has been repeatedly observed. Delays have been linked to increased mortality and longer duration of organ dysfunction and recent multicenter data indicate that prolonged time to antibiotics in pediatric emergency departments is associated with increased mortality risk. These findings support a low threshold for treatment in children with suspected septic shock or high likelihood of bacterial infection [17-19].

Cultures should be obtained before antimicrobials when this can be done without meaningful delay. Blood cultures, urine cultures, cerebrospinal fluid studies, respiratory samples, or other microbiologic tests should be selected according to suspected focus, age, comorbidities and clinical stability. Once microbiologic data and susceptibility results are available, therapy should be narrowed, optimized, or discontinued when appropriate. This antimicrobial stewardship step is essential because overtreatment contributes to resistance, adverse drug effects, microbiome disruption and diagnostic anchoring.

Source control must be considered early. Drainage of abscesses, removal of infected devices, surgical management of intra-abdominal or soft tissue infection and control of necrotic or purulent foci may be decisive when infection persists despite adequate antibiotics. Antibiotics alone are often insufficient when an uncontrolled focus continues to drive systemic inflammation. Timing should balance urgency with procedural risk and hemodynamic stability [1].

Fluid resuscitation, vasoactive agents and hemodynamic support

Fluid resuscitation remains central in pediatric septic shock, but contemporary practice is no longer equivalent to unrestricted rapid boluses. In settings with access to pediatric intensive care and the ability to provide ventilatory and vasoactive support, current guidelines suggest administering isotonic crystalloids in 10 to 20 mL/kg aliquots with reassessment after each bolus. Total fluid during the first hour may reach 40 to 60 mL/kg when shock persists and there are no signs of overload, but fluid should be reduced or stopped when perfusion improves or overload emerges [1].

Each bolus should be followed by reassessment of heart rate, blood pressure, pulse quality, capillary refill, extremity temperature, mental status, respiratory work, oxygenation,

hepatomegaly, lung findings and urine output. Signs such as new crackles, worsening respiratory distress, hepatomegaly, gallop rhythm, falling oxygen saturation, or progressive edema suggest fluid intolerance. Accumulating evidence links positive fluid balance and fluid overload with worse outcomes in critically ill children, including mortality, acute kidney injury and longer mechanical ventilation [20].

In resource-limited settings without intensive care, mechanical ventilation, or vasoactive support, rapid fluid boluses should be used more cautiously, especially in children without hypotension. The FEAST trial demonstrated increased mortality after fluid bolus therapy in African children with severe febrile illness and impaired perfusion, underscoring that the safety of aggressive fluid resuscitation depends on patient selection, disease context and available rescue therapies [21].

Vasoactive support should begin early when shock persists after initial fluid resuscitation or when additional fluids are unsafe. Waiting for central venous access should not delay vasoactive drugs; they may be started through a well-functioning peripheral intravenous or intraosseous line with careful site monitoring until central access is available. Epinephrine and norepinephrine are preferred initial agents, with selection guided by hemodynamic phenotype, local expertise and response. Epinephrine is often used when myocardial dysfunction and low cardiac output predominate, whereas norepinephrine may be favored in vasodilatory shock with low systemic vascular resistance [1,22].

Earlier pediatric hemodynamic trials supported goal-directed resuscitation using clinical targets and central venous oxygen saturation in selected settings, while later practice has emphasized individualized, multimodal assessment rather than fixed central venous pressure targets. Basic variables such as heart rate, blood pressure, perfusion pressure, urine output, lactate and central venous oxygen saturation may be combined with advanced measures such as cardiac output, cardiac index, systemic vascular resistance, bedside echocardiography and dynamic assessment of fluid responsiveness when available [23,24].

Hemodynamic support should be reassessed continuously. A child whose perfusion does not improve after epinephrine may require evaluation for vasodilation, myocardial dysfunction, tamponade, pneumothorax, ongoing bleeding, adrenal insufficiency, uncontrolled infection, or inadequate source control. Hydrocortisone may be considered in catecholamine-resistant shock or suspected adrenal insufficiency. Vasopressin may be considered in refractory vasodilatory shock, although evidence in children remains limited and local protocols should guide use [1,24].

Monitoring and intensive care support

Children with septic shock, persistent hemodynamic instability, respiratory failure, need for vasoactive drugs, progressive organ dysfunction, altered mental status, or high-risk comorbidities require intensive monitoring. The goal is to evaluate response to treatment, detect deterioration, avoid complications and adjust support in real time. Monitoring should integrate perfusion, oxygenation, ventilation, renal function, neurologic status, coagulation, glucose, lactate trend, inflammatory markers, medication response and cumulative fluid balance [1,24].

Respiratory support ranges from supplemental oxygen to noninvasive ventilation and invasive mechanical ventilation. Intubation may be required for severe respiratory distress, hypoxemia, impaired consciousness, inability to protect the airway, or shock with excessive work of breathing. Mechanical ventilation can reduce oxygen consumption and support gas exchange, but it also introduces risks, including ventilator-induced lung injury, hemodynamic compromise from positive pressure, infection and prolonged sedation. Pediatric ventilatory management should follow lung-protective principles and account for the underlying disease, lung compliance, oxygenation, carbon dioxide targets and hemodynamic effects.

Renal monitoring is essential because acute kidney injury is common in pediatric septic shock and may be worsened by hypoperfusion, nephrotoxic drugs, inflammation and fluid overload. Urine output, creatinine, electrolyte abnormalities, acid-base status and cumulative fluid balance should be followed closely. When fluid overload is severe or progressive despite restriction and diuretics, renal replacement therapy may be considered, particularly when it coexists with refractory metabolic abnormalities or organ dysfunction [10,20].

Biomarkers can contribute to monitoring but should not dominate decision-making. Lactate clearance may suggest improving perfusion but is not a universal target. Procalcitonin may help in bacterial infection assessment and antimicrobial de-escalation in selected contexts, but false positives and false negatives occur. Biomarker-based prognostic models may improve risk stratification in research or specialized centers, yet routine decisions should remain grounded in the full clinical trajectory.

Controversies and evidence gaps

Several important uncertainties remain. First, no universal screening tool reliably identifies all children with early sepsis across emergency departments, wards, intensive care units and resource-limited settings. Electronic alerts improve sensitivity in some systems but may produce alert fatigue or perform poorly when data quality, thresholds, or clinical response pathways are inadequate.

Second, the Phoenix criteria improve standardization of pediatric sepsis definitions, but they are not an early triage tool and do not fully capture all bedside signs of compensated shock. Their performance also depends on the availability of laboratory data, oxygenation measures and vasoactive information. The criteria are a major step forward for diagnosis, research, epidemiology and benchmarking, but they must be paired with clinical suspicion and institutional protocols [2,3].

Third, the optimal fluid strategy continues to be debated. Guidelines recommend individualized aliquots and reassessment, but the best balance between under-resuscitation and fluid overload is context-dependent. The type of crystalloid, total volume, timing of vasoactive initiation and the role of dynamic fluid responsiveness assessment require further pediatric trials, especially in low-resource environments and in children with comorbidities [1,20,21].

Fourth, vasoactive selection remains incompletely resolved. Epinephrine and norepinephrine are both recommended as reasonable first-line drugs and the best choice may vary according to myocardial function, vasoplegia, age, prior comorbidities and response to treatment. More pediatric data are needed to define phenotype-specific first-line strategies and escalation algorithms [1,22,24].

Finally, antimicrobial stewardship creates a persistent tension. Delayed antimicrobials are harmful in septic shock, but indiscriminate broad-spectrum therapy exposes many children without bacterial sepsis to unnecessary treatment. Future research should clarify how biomarkers, host-response signatures, rapid diagnostics and structured clinical pathways can shorten time to treatment for high-risk patients while reducing unnecessary antibiotic exposure in low-risk patients [14-16].

Conclusion

Pediatric sepsis is a time-dependent emergency in which early recognition, structured risk stratification and coordinated initial management can alter the clinical trajectory. Modern definitions centered on organ dysfunction, especially the Phoenix criteria, improve standardization, but they do not eliminate the need for bedside clinical suspicion. Children may deteriorate before hypotension appears, so clinicians must value perfusion abnormalities, mental status changes, respiratory distress, oliguria, high-risk comorbidities and repeated reassessment. Effective care requires more than completing isolated tasks within a time window. It depends on a system that connects screening, communication, vascular access, cultures without therapeutic delay, early antimicrobials when septic shock is suspected, individualized fluid resuscitation, early vasoactive support, source control, intensive monitoring and continuous adjustment to response. Remaining gaps in screening accuracy, fluid strategy, vasoactive selection, biomarker use and antimicrobial stewardship justify further pediatric research, but current evidence already supports institutional protocols that combine standardized pathways with careful clinical judgment.

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