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Prevalence of Sepsis in Pediatric Populations

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Introduction

Sepsis and more specifically septic shock in the pediatric population is a diagnosis that is full of complexities. There are instances where a neutropenic oncology patient observes a better outcome than a previously healthy patient who is suffering from the same pathogen related sepsis. Sepsis is the 10th leading cause of death according to the Center for Disease Control and Prevention, and each year between 20,000 and 42,000 children are diagnosed with severe sepsis (Riley & Wheeler, 2012). Even with significant advances in medical treatment, sepsis is still associated with high morbidity and mortality rates. A retrospective study of patient outcomes across 26 countries found that pediatric sepsis mortality was 25%, was seemingly unaffected by age, and had only mild variations across developed countries. Of the survivors, 20% suffered from a form of moderate functional disability (Weiss et al., 2015). Despite the amount of clinical trials and research associated with pediatric sepsis its incidence continues to increase by close to 1.5% annually (Riley & Wheeler, 2012).

Signs and Symptoms

Those within the pediatric population who pose the greatest risk of developing sepsis include patients with indwelling vascular and urinary catheters, after recent surgery, and those that are immunocompromised, no matter the cause. Patients with sepsis generally exhibit characteristic signs and symptoms. The hallmark sign of sepsis is either hyperthermia or hypothermia associated with the onset of a new infection. The disease process is also associated with the symptoms of tachycardia, hyperglycemia without the incidence of diabetes, lethargy, warm skin, increased cardiac output, chills, and muscle weakness. Severe sepsis or septic shock is differentiated from sepsis by the presence of hypotension, decreased cardiac output, and other complications including liver failure, acute respiratory distress syndrome, encephalopathy, renal failure, disseminated intravascular coagulopathy, and multiple organ dysfunction syndrome (Duran-Bedolla, 2014).

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I. Cardiovascular
1. Systolic blood pressure, mm Hg
≤65 in infants
≤75 in children or
≤85 in adolescents
2. Heart rate, beats/min
<50 or >220 in infants
<40 or >200 in children
3. Continuous infusion of inotropic agents
4. Serum pH <7.20 (with a normal $Paco_2$)

II. Respiratory
1. Respiratory rate, breaths/min
>90 in infants or
>70 in children
2. Pao_2/Fio_2 <200 (in the absence of congenital heart disease)
3. Mechanical ventilation (>24 hrs in a postoperative patient)
4. $Paco_2$ >65 torr
5. Pao_2 <40 torr (in the absence of congenital heart disease)

III. Neurologic
1. Glasgow Coma Scale score of <5
2. Fixed and dilated pupils

IV. Hematologic
1. Hemoglobin, <5 g/dL
2. White blood cell count, <3000/mm³
3. Platelet count, <20,000/mm³
4. Prothrombin time, >20 secs or Activated partial thromboplastin time, >60 secs

V. Renal
1. Blood urea nitrogen, >100 mg/dL
2. Creatinine, >2.0 mg/dL (in the absence of preexisting renal disease)
3. Dialysis

VI. Gastrointestinal
1. Blood transfusions >20 mL/kg in 24 hrs because of hemorrhage

VII. Hepatic
1. Total bilirubin >5 mg/dL and aspartate aminotransferase or lactate dehydrogenase greater than twice normal (without evidence of hemolysis)

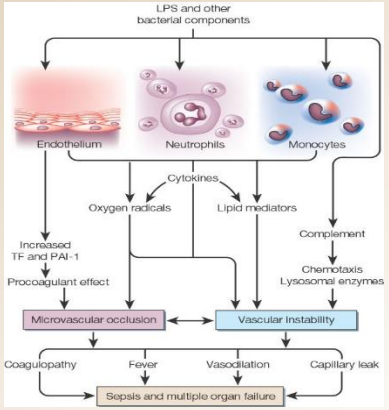
Source: PCCM © 2003 Lippincott Williams & Wilkins

This list details the clinical signs and symptoms associated with pediatric septic shock.

Underlying Pathophysiology

Sepsis and its associated symptoms are caused by a microorganism, and the body's dysregulation of compensatory mechanisms. There are 3 phases associated with sepsis after the microorganism has infiltrated the host body:

- The release of inflammatory mediators
- The lack of an appropriate anti-inflammatory response
- The subsequent immunoparalysis due to excessive inflammatory mediators present



This image demonstrates endothelial damage neutrophil activation, and monocytes that activate the complement cascade which leads to the associated symptoms of sepsis and multiple organ failure

After a microorganism has managed to invade a host, the body enters a proinflammatory phase. Gram negative and gram positive organisms cause the release of toxic substances into the body including endotoxins, exotoxins, lipoteichoic acids, and peptidoglycan. These toxic substances cause the release of macrophages, monocytes, platelets, polymorphonuclear leukocytes, and cytokines such as tumor necrosis factor α and interleukin 1, 6, 8, and 10. The tumor necrosis factor and interleukins cause endothelial cell and leukocyte adhesion, increase tissue coagulation factor levels causing coagulation, increase production of nitric oxide which results in vascular vasodilation, inhibit anticoagulation by decreasing thrombomodulin, and cause the activation of the complement cascade. These responses to tumor necrosis factor and interleukins result in inflammation. The cytokines present causes endothelial dysfunction which leads to capillary leak and eventually impaired perfusion. Because tissue and cell perfusion is inadequate, hypoxic cellular necrosis apoptosis occurs. Organ dysfunction and end organ failure results from cellular death (Sagy, Al-Qaqaa, & Kim, 2013).

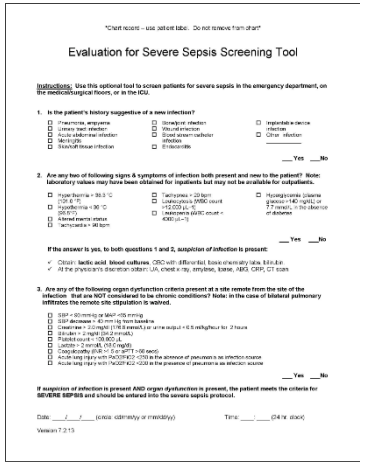
Next the body's negative feedback loop to inflammation causes an anti-inflammatory response. Anti-inflammatory mediators should inhibit T-lymphocyte function, immunoglobulins, tumor necrosis factor α, and inhibit activation of the coagulation cascade in a properly functioning inflammatory response. With sepsis, there is an increased proinflammatory response to mediators which the body tries to compensate with by causing an excessive anti-inflammatory response. Eventually the excessive anti-inflammatory response leads to a relative immunosuppression and proliferation of inflammation that ultimately may lead to multiple organ dysfunction syndrome.

Significance of Pathophysiology

The morbidity and mortality of sepsis is not from the disease process alone but is associated with the sequelae of organ dysfunction that sepsis may cause. During septic shock there is an inadequate oxygen perfusion to cells. This hypoxia causes an increase in reactive oxygen species and reactive nitrogen species. Reactive oxygen species can break polypeptide chains and change the charge of proteins which is associated with the misfolding or degradation of proteins. Reactive nitrogen species yield nitric oxide causing microvascular damage and vascular vasodilation which produces hypotension. Excessive amounts of nitric oxide can also cause cellular apoptosis which can progress to organ dysfunction. After prolonged organ dysfunction, death may occur depending on the patient and their specific inflammatory response. Sepsis has the capacity to cause fevers, tachycardia, and mild hypotension in some patients while other patients suffer the sequelae of irreversible organ damage or death (Duran-Bedolla, 2014).

Implications for Nursing Care

Though there has been a vast amount of advances with the medical field, sepsis is still a difficult disease to treat. It has been proven that early identification and disease specific intervention can reduce the morbidity and mortality of sepsis and even possibly prevent severe sepsis from becoming septic shock, or sepsis associated with hypotension. At the Methodist Medical Center of Illinois a sepsis initiative called "Think Sepsis" was created including sepsis specific training of those nursing staff that are often the first to encounter patients suffering from sepsis. This medical center also utilized a sepsis trigger tool that allowed for early recognition and documentation of sepsis. At this hospital prior to the completion of the sepsis initiative, in 2009 their mortality rate for patients was 19%. In 2011, two years after the initiative was started, their mortality rate had decreased to 11% (Hale, 2011). Nurses who are trained in the signs and symptoms of early sepsis can alert the medical team of their findings and potentially stop the progression of severe sepsis to septic shock.



*"Chart recent – use patient label. Do not remove from chart"

Evaluation for Severe Sepsis Screening Tool

Instructions: Use this optional tool to screen patients for severe sepsis in the emergency department, on the medical/surgical floors, or in the ICU.

1. Is the patient's history suggestive of a new infection?
Pneumonia, empyema / Bronchitis / Implantable device infection
Urinary tract infection / Wound infection
Acute abdominal infection / Blood stream catheter infection / Other infection
Skin/cellulitis / Endocarditis
Bowel/soft tissue infection
___Yes ___No

2. Are any two of following signs & symptoms of infection both present and new to the patient? Note: laboratory values may have been obtained for inpatients but may not be available for outpatients.
Hyperthermia > 38.3 °C / Tachypnea > 20 bpm / Hypoxemia (plasma glucose > 140 mg/dL) or new onset, in the absence of diabetes
Hypothermia < 36 °C / Leukocytosis (WBC count >12,000 µL-1) / Altered mental status / Tachycardia > 90 bpm / Leukopenia (WBC count < 4000 µL-1)
___Yes ___No

If the answer is yes, to both questions 1 and 2, suspicion of infection is present:
✓ Obtain lactic acid, blood cultures, CBC with differential, basic chemistry labs, bilirubin
✓ At the physician's discretion obtain: UA, chest x-ray, amylase, lipase, ABG, CPP, CT scan

3. Are any of the following organ dysfunction criteria present at a site remote from the site of the infection that are NOT considered to be chronic conditions? Note: in the case of bilateral pulmonary infiltrates the remote site stipulation is waived.
SBP < 90 mmHg or MAP <65 mmHg / SBP decrease > 40 mm Hg from baseline / Creatinine > 2 mg/dl (176.8 mmol/L) or urine output < 0.5 ml/kg/hour for 2 hours / INR > 1.5 or PTT > 60 seconds (aPTT) / Platelet count < 100,000 /µL / Lactate > 2 mmol/L (18.0 mg/dl) / Coagulopathy (INR > 1.5 or aPTT > 60 secs) / Acute lung injury with PaO2/FiO2 <250 in the absence of pneumonia as infection source / Acute lung injury with PaO2/FiO2 <250 in the presence of pneumonia as infection source
___Yes ___No

If suspicion of infection is present AND organ dysfunction is present, the patient meets the criteria for SEVERE SEPSIS and should be entered into the severe sepsis protocol.

Date: ___/___/___ (circle: calendar day or immediately) Time: ___:___ (24 hr. clock)

Version 7.2.13

The Center for Disease Control and Prevention recommends using the above trigger tool for early diagnosis and treatment of sepsis patients.

Conclusion

Sepsis is a complex disease originating from the infiltration of a microorganism into a host's body and the resulting improper inflammatory response and dysregulation of compensatory mechanisms such as the anti-inflammatory response. The ramifications of the body's inappropriate response to the microorganism induces cellular death thus leading to organ dysfunction and even death if severe. Despite many medical advances sepsis still maintains high morbidity and mortality rates especially for those children who are most at risk, including those that are hospitalized and immunosuppressed. According to the CDC, patients with sepsis still maintain higher risks of complications, receive longer treatment, and achieve higher healthcare costs (2014). Early recognition and utilization of sepsis specific interventions has been proven to reduce the mortality rate but little has been shown if it affects the morbidity rate for patients. Sepsis remains a complicated disease course and is entirely dependent on each person's inflammatory response and compensatory responses with its prevalence remaining and mortality rates remaining high in the pediatric population.

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