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Nursing care interventions for toxic shock syndrome: An updated review

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***Abstract*---Background**: Toxic Shock Syndrome (TSS) is a life-threatening condition characterized by rapid-onset fever, hypotension, widespread rash, and multi-organ dysfunction, most often caused by infections with *Staphylococcus aureus* and *Streptococcus pyogenes*. TSS was first described in 1978 and has been associated with both bacterial strains, presenting a severe risk of mortality if not treated promptly. Early diagnosis and treatment are vital in reducing the mortality rate, especially in pediatric patients. **Aim**: This review aims to explore the pathophysiology, clinical features, and management strategies for Toxic Shock Syndrome, focusing on nursing interventions and the importance of early recognition. **Methods**: This review examines the clinical presentation of TSS, including diagnostic criteria, the role of superantigens in the disease process, and the impact of prompt intervention. The article reviews available evidence on effective nursing care interventions, including fluid resuscitation, antibiotic therapy, and supportive care, to ensure positive outcomes. **Results**: The clinical manifestations of TSS are often characterized by a triad of high fever, hypotension, and multi-organ involvement. Antibiotic therapy, including beta-lactamase-resistant agents and clindamycin, is crucial for the management of TSS. Early diagnosis, aggressive fluid resuscitation, and source control significantly improve survival rates. Nursing care plays a pivotal role in managing the patient's hemodynamic status and ensuring the timely administration of therapies. **Conclusion**: TSS remains a critical condition requiring immediate medical attention. Timely nursing interventions, including monitoring vital signs, fluid management, and prompt antibiotic administration, are essential for improving patient outcomes. Healthcare providers must maintain a high clinical suspicion, especially in pediatric cases, to initiate treatment promptly and reduce the risk of mortality.

***Keywords*---**Toxic Shock Syndrome, Nursing Care, Superantigens, Antibiotic Therapy, Fluid Resuscitation, Pediatric Care.

Introduction

Toxic shock syndrome (TSS) is a severe acute condition marked by high fever, hypotension, widespread rash, and significant multi-organ dysfunction. The pathophysiology of the shock in TSS is toxin-mediated, typically linked to infections caused by *Staphylococcus aureus* and *Streptococcus pyogenes*. TSS was initially described in 1978 in children infected with *S. aureus* and later, in 1987, in association with invasive *S. pyogenes* infections. Over time, this syndrome has been reported across all age groups, with a higher prevalence in extreme age groups. Particularly, children under the age of two are notably vulnerable to TSS. Clusters of invasive *Group A Streptococcus* infections related to TSS have been

identified in various settings, including households, hospitals, and nursing homes. As a result, TSS is now classified as a notifiable disease in the UK, where screening and prophylactic antibiotic measures for close contacts are mandated. The progression of symptoms can occur rapidly, with a significant risk of mortality if not promptly addressed. Early recognition and intervention are crucial, as the overall mortality rate in pediatric cases ranges from 5% to 10%. A high clinical suspicion, coupled with prompt and aggressive treatment, is key to achieving favorable outcomes. While clinical differences exist between *S. aureus*-induced and *S. pyogenes*-induced TSS, the underlying pathophysiology is consistent, and therefore, the initial management strategies for both types are largely similar. Routine immunizations, such as those for meningococcus, pneumococcus, and *Haemophilus influenzae*, have led to a reduction in sepsis and shock cases in children from high-income countries. Despite this, TSS remains a leading cause of shock in children. This article aims to examine the mechanisms behind shock in TSS, highlight its clinical features, and explore the role of adjunctive treatments in managing the condition [1].

Definition: Streptococcal vs. Staphylococcal TSS

The Centers for Disease Control and Prevention (CDC) has defined case criteria for both *S. aureus* and *S. pyogenes* TSS, which serve as valuable tools for monitoring and surveillance. However, these criteria are less critical in the context of clinical management, as they are generally retrospective and often not applicable in the early stages of the disease. The CDC's classification includes a set of clinical and laboratory criteria for both staphylococcal and streptococcal TSS. For *Staphylococcus aureus* toxic shock syndrome, the diagnostic criteria include fever with a temperature exceeding 38.9°C, a diffuse erythematous rash, and desquamation that typically occurs one to two weeks after symptom onset, particularly affecting the palms and soles. Hypotension is characterized by systolic blood pressure falling below the 5th percentile for age. Multisystem involvement must affect at least three organs, with possible organ systems including gastrointestinal (vomiting or diarrhea), muscular (severe myalgia and elevated creatine kinase levels), mucous membranes (vaginal, oropharyngeal, or conjunctival), renal (elevated urea and creatinine), hepatic (elevated liver enzymes), hematologic (thrombocytopenia), and central nervous system (disorientation or altered consciousness). Laboratory findings include negative cultures for other pathogens and negative serology for diseases such as Rocky Mountain spotted fever, leptospirosis, and measles. A probable case of staphylococcal TSS requires meeting laboratory criteria and four out of the five clinical criteria, while a confirmed case meets all five clinical criteria, including desquamation, unless the patient dies before this occurs.

For *Streptococcus pyogenes* toxic shock syndrome, a confirmed diagnosis necessitates isolation of *Group A Streptococcus* from a sterile site such as blood, cerebrospinal fluid, or tissue biopsy, or from a non-sterile site like the vagina, throat, or sputum. Clinical features include hypotension (systolic blood pressure ≤90 mmHg in adults or less than the 5th percentile for age in children), and multi-organ involvement affecting at least two organ systems. Renal impairment is defined by creatinine levels greater than 2 mg/dL in adults or more than two times the normal limit for age in children, while coagulopathy is marked by

platelet counts below 100,000/mm³ or disseminated intravascular coagulation. Liver involvement is indicated by elevated liver enzymes or bilirubin levels. Other features include acute respiratory distress syndrome, erythematous macular rash, and soft tissue necrosis. A definitive case is diagnosed when both criteria for infection and clinical manifestations are met, while a probable case is defined if the infection is isolated from a non-sterile site without an alternative cause for the illness. Clinical suspicion of TSS in pediatric patients is paramount for timely diagnosis and intervention. Key indicators such as the erythematous rash, conjunctivitis, and early signs of multi-organ involvement, including acute kidney injury, are essential for early recognition and initiation of appropriate treatment [2].

Pathogenesis: Superantigens and their Role in Toxic Shock Syndrome

Superantigens (SAs) represent a class of nonglycosylated, low-molecular-weight exoproteins secreted by human-pathogenic *Staphylococcus aureus* and *Streptococcus pyogenes*. These proteins exhibit unusual resistance to heat, acid, and desiccation, allowing them to persist in diverse environmental conditions. *S. aureus* strains are capable of secreting up to 24 distinct serologically identifiable SAs, whereas group A streptococcal strains can produce up to 11 different SAs. Superantigens function by bypassing the standard antigen-processing pathways. Unlike typical antigens, which are processed within antigen-presenting cells (APCs) before presentation to T-cells, SAs directly bind to major histocompatibility complex (MHC) class II molecules. This interaction triggers an excessive activation of T-cells, culminating in the massive release of proinflammatory cytokines such as TNF-α, IL-6, interferon-γ, and additional interleukins. The subsequent cytokine storm induces systemic inflammation, which is a key contributor to the profound shock seen in patients with toxic shock syndrome (TSS). The superantigens most strongly associated with TSS include *Staphylococcal Toxic Shock Syndrome Toxin 1* (TSST-1) and *Streptococcal Pyrogenic Exotoxins* (SPEs). In the case of *S. pyogenes* infection, the M protein shed from its surface forms complexes with fibrinogen. These complexes then bind to integrins on polymorphonuclear leukocytes (PMNs), leading to their activation and subsequent damage to the endothelial lining, which further contributes to the pathophysiology of streptococcal TSS. An individual's immune response to superantigens is influenced by several factors, including the presence of antibodies to these toxins. While antibodies to superantigens are commonly found in adults (with more than 90% of adults displaying antibodies to TSST-1), their presence is less frequent in children. Studies have shown that at 12 months of age, only 47% of children exhibit antibodies to TSST-1, a figure that increases to 58% at age five and 70% by age ten. A deficiency in antibodies to SPEs correlates with an increased risk of invasive disease. Furthermore, certain MHC class II haplotypes, such as DRB1*1501/DQB1*0602, have been identified as influencing cytokine regulation. Individuals with these haplotypes exhibit a diminished cytokine response to superantigen stimulation, potentially reducing the likelihood of developing severe TSS [3].

Clinical Presentation

The clinical suspicion and prompt recognition of TSS, particularly in pediatric patients, is critical to ensuring favorable outcomes and survival. Routine vaccination against major pathogens implicated in sepsis, such as meningococcus and pneumococcus, has led to a decrease in sepsis-related shock, yet TSS remains one of the leading causes of shock in children. TSS is characterized by a triad of high fever, rapid-onset hypotension that does not respond to fluid resuscitation, acute renal failure, and multisystem involvement. While the clinical manifestations of *S. aureus*-associated TSS and *S. pyogenes*-associated TSS can overlap, distinguishing between the two can be challenging. The onset of *staphylococcal* TSS is typically abrupt, presenting with fever, chills, malaise, headache, myalgia, muscle tenderness, vomiting, and diarrhea. A diffuse erythematous maculopapular rash develops within the first 48 hours. Neurological manifestations, including confusion, agitation, and somnolence, are common, alongside conjunctival hyperemia. Desquamation of the skin typically occurs one week after the onset of the illness and is considered a late sign. Laboratory abnormalities include an increase in immature neutrophils, thrombocytopenia, anemia, and the potential development of disseminated intravascular coagulation. Renal and liver function tests may also reveal abnormalities. Although blood cultures are positive in only 5% of cases, cultures from the site of infection are more likely to yield a positive result and should be promptly obtained. Foreign body presence in the infection site is common, and TSS may arise following invasive infections such as pneumonia, osteomyelitis, pyogenic arthritis, and endocarditis. When TSS is associated with surgical wounds, the infection site may not exhibit typical signs of infection due to neutrophil suppression by TNF-α. *Streptococcal* TSS typically presents with the sudden onset of localized pain, followed by flu-like symptoms such as fever, chills, myalgia, nausea, vomiting, and diarrhea. Neurological symptoms, including confusion, combativeness, and in some cases, coma, may also be present. Soft tissue infections are common, with progression to necrotizing fasciitis or myositis in severely ill patients. Laboratory findings for streptococcal TSS closely mirror those seen in staphylococcal TSS; however, blood cultures are positive in 60% of cases. Younger children, particularly those with chickenpox, are at an increased risk of developing streptococcal TSS, often presenting them with bacteremia in the absence of an obvious source of infection, osteomyelitis, or central nervous system involvement [4].

Differential Diagnosis

The diagnosis of Toxic Shock Syndrome (TSS) can be challenging due to its clinical overlap with sepsis and septic shock of various etiologies. Sepsis from other causes typically does not present with characteristic dermatologic manifestations such as erythematous rashes and skin desquamation. Meningococcal sepsis is often presented with the acute onset of fever, nausea, vomiting, headache, confusion, and myalgia, along with a distinctive non-blanching purpuric rash. Identification of the causative organisms through blood and spinal fluid cultures or polymerase chain reaction (PCR) testing is essential for confirmation. Differentiating the early stages of Kawasaki Disease Shock Syndrome (KDSS) from TSS can also be challenging, as both conditions share

symptoms of fever, rash, and conjunctivitis in a shocked patient. Laboratory findings in KDSS commonly reveal anemia and thrombocytosis, with the latter helping to distinguish KDSS from TSS. KDSS typically affects younger children, with a mean age of three years. Echocardiography is a crucial diagnostic tool in KDSS, revealing cardiac involvement such as valvulitis and characteristic coronary lesions. More recently, the pediatric multisystem inflammatory syndrome temporally associated with COVID-19 (PIMS-TS) has been described, exhibiting clinical features similar to TSS, KDSS, and Kawasaki disease. Laboratory findings in PIMS-TS show increased inflammation, including elevated levels of C-reactive protein, ferritin, and D-dimers, further complicating the differential diagnosis [5].

Management

Early intervention using sepsis guidelines is essential, including aggressive yet controlled fluid resuscitation, early antibiotic therapy, and source control of the infection. Critical care support is necessary for patients with respiratory, cardiovascular, or renal failure.

Antibiotics

Antibiotic therapy is a critical component of TSS management. Empiric antibiotics should provide coverage for *S. aureus* and *Streptococcus pyogenes*. The first-line antibiotics should include beta-lactamase-resistant anti-staphylococcal agents such as flucloxacillin or first-generation cephalosporins. Given the rising prevalence of methicillin-resistant *S. aureus* (MRSA) as a community-acquired pathogen, it is crucial to consider the appropriate initial antibiotic regimen. Monotherapy is not recommended; a combination of clindamycin with other antibiotics has been shown to improve outcomes. Clindamycin is particularly effective in TSS due to several factors:

1. It retains antimicrobial activity regardless of inoculum size.
2. It inhibits protein synthesis independently of penicillin-binding proteins.
3. It disrupts the synthesis of antiphagocytic M proteins and bacterial toxins.
4. It exhibits a prolonged post-antibiotic effect compared to beta-lactams.

However, clindamycin should not be used as initial monotherapy, as 1–2% of *S. pyogenes* strains are resistant to it.

Intravenous Immunoglobulin (IVIG)

IVIG is routinely recommended as an adjunctive therapy for TSS, with potential benefits including neutralization of superantigens and enhancement of bacterial clearance. It may also inhibit T-cell activation by inactivating superantigens, thereby reducing cytokine release. Despite its widespread use, the evidence supporting IVIG's efficacy remains limited, with few studies and an absence of randomized controlled trials. A meta-analysis by Parks and colleagues suggests that adjunctive IVIG therapy in patients treated with clindamycin is associated with a statistically significant reduction in mortality. Most TSS treatment protocols now recommend a single dose of IVIG at 1–2 g/kg.

Other Treatments

Other treatment modalities, such as steroids and plasmapheresis, have not demonstrated any significant benefit in improving patient outcomes.

Nursing Interventions Plan for Toxic Shock Syndrome (TSS)

Toxic Shock Syndrome (TSS) is a rare but life-threatening condition caused by the release of superantigens from *Staphylococcus aureus* or *Streptococcus pyogenes*, which leads to a systemic inflammatory response, multi-organ dysfunction, and shock. Given the severity and rapid progression of TSS, timely and effective nursing interventions are critical to improving patient outcomes. A comprehensive nursing plan for managing TSS should focus on early detection, stabilization, infection control, monitoring, supportive care, and patient and family education. This plan involves a collaborative approach with other healthcare providers to ensure the best possible care.

Assessment and Early Recognition

Early identification of TSS is paramount for improving survival rates. Nurses play a critical role in recognizing the early signs and symptoms of TSS, which include high fever, hypotension unresponsive to fluid resuscitation, diffuse erythematous rash, multi-organ involvement, and desquamation of the skin. Nurses should perform frequent and comprehensive assessments of vital signs, including temperature, blood pressure, heart rate, respiratory rate, and oxygen saturation. Monitoring for signs of shock, such as tachycardia, hypoxia, and inadequate tissue perfusion, is essential. Additionally, nurses should conduct a thorough assessment of the patient's medical history, including potential sources of infection, recent surgeries, or presence of foreign bodies that could predispose the patient to TSS. It is essential to assess the patient's respiratory, cardiovascular, and renal status closely due to the possibility of multi-organ failure. Monitoring laboratory values, such as complete blood count (CBC), liver and kidney function tests, and coagulation studies, is also crucial for tracking the progression of the disease and evaluating the effectiveness of interventions.

Fluid Resuscitation and Hemodynamic Support

Fluid resuscitation is a cornerstone of TSS management, as hypovolemia and shock are common in the acute phase of the illness. Nurses should administer intravenous fluids promptly to restore circulatory volume and maintain blood pressure. Initial fluid therapy typically involves isotonic crystalloids, such as normal saline or lactated Ringer's solution. Close monitoring of fluid intake and output is required to prevent fluid overload, especially in patients with compromised renal function. Nurses should also assess signs of fluid retention, such as edema or pulmonary congestion, and adjust the fluid therapy accordingly. Nurses must frequently monitor hemodynamic parameters, including blood pressure, heart rate, and oxygenation status, to assess the effectiveness of fluid resuscitation and ensure adequate perfusion to vital organs. If hypotension persists despite fluid administration, vasopressors may be required to maintain perfusion, and nurses should assist with the administration and titration of these

medications based on the patient's clinical response. Continuous cardiovascular monitoring may be necessary in severe cases to guide therapy and detect changes in the patient's condition promptly.

Infection Control and Antibiotic Administration

Nurses must ensure that appropriate antibiotics are administered promptly, as early antimicrobial therapy is crucial in controlling the infection. The standard antibiotic regimen for TSS includes coverage for both *Staphylococcus aureus* and *Streptococcus pyogenes*. First-line antibiotics, such as flucloxacillin or cefazolin, should be administered intravenously as soon as possible. In the case of suspected methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin or clindamycin should be included in the treatment regimen. Nurses should ensure that antibiotics are administered according to protocol and within the recommended timeframes. Monitoring for signs of antibiotic effectiveness, such as improvement in fever and vital signs, is essential. Nurses should also observe potential adverse reactions to antibiotics, such as allergic reactions or gastrointestinal disturbances, and report any issues to the healthcare team immediately. Additionally, if a foreign body is identified as the source of infection, the nurse should ensure prompt removal or appropriate management as directed by the physician.

Supportive Care and Monitoring of Organ Function

Supportive care is vital in managing the multi-system effects of TSS. Nurses should provide respiratory support, including supplemental oxygen or mechanical ventilation, if necessary, to address respiratory distress or hypoxia. Continuous monitoring of respiratory rate, oxygen saturation, and arterial blood gases (ABG) will help guide interventions and ensure adequate oxygenation. In addition, renal function should be closely monitored. Acute renal failure is a common complication of TSS due to inadequate perfusion, especially in the absence of effective fluid resuscitation. Nurses should assess urine output regularly and report any signs of oliguria or anuria, which may indicate renal dysfunction. In cases of severe renal impairment, nurses may assist with the initiation of dialysis if recommended by the healthcare team. The cardiovascular system should be monitored for signs of decompensation, and any changes in vital signs, such as hypotension or arrhythmias, should be promptly reported. Nurses may also assist with the administration of vasopressors, if necessary, to support blood pressure and improve organ perfusion.

Management of Cytokine Storm and Immune Modulation

The release of cytokines, such as TNF-α, IL-6, and interferon-γ, contributes significantly to the pathophysiology of TSS. Nurses should be vigilant in monitoring for signs of a cytokine storm, including fever, hypotension, and multi-organ dysfunction. As part of the immune modulation strategy, intravenous immunoglobulin (IVIG) is frequently used to neutralize superantigens and reduce cytokine release. Nurses should administer IVIG according to established protocols and monitor the patient for adverse reactions, such as allergic responses or infusion-related complications. Although the use of steroids is not

typically recommended in TSS, nurses should be familiar with any adjunctive treatments, such as plasmapheresis or immune-modulating therapies, that may be indicated in specific cases. Regular communication with the healthcare team is necessary to ensure that the most appropriate therapies are being implemented.

Patient and Family Education

Patient and family education is a vital component of nursing care, particularly for pediatric patients and their families. Nurses should explain the nature of TSS, the importance of early detection, and the potential complications. Education should also focus on the necessity of adhering to treatment regimens, including the administration of antibiotics and the potential need for supportive therapies such as IVIG. Additionally, nurses should provide guidance on the recognition of early signs and symptoms of TSS and educate families on when to seek immediate medical attention if they suspect a recurrence or worsening of symptoms. Family support is essential, as the diagnosis of TSS can be overwhelming and distressing. Nurses should offer emotional support and encourage families to be involved in the care process to reduce anxiety and promote coping. Nursing interventions for the management of TSS are multifaceted, focusing on early detection, infection control, hemodynamic stabilization, and supportive care. Nurses play a critical role in monitoring vital signs, administering medications, and providing both physical and emotional support to patients and families. Through careful assessment and prompt interventions, nurses can significantly improve patient outcomes and help manage the complexities associated with TSS. Collaboration with the broader healthcare team is essential to ensure the best possible care and reduce the risk of complications associated with this severe and rapidly progressing condition.

Conclusion

Toxic Shock Syndrome (TSS) is a severe, life-threatening condition requiring rapid intervention to prevent complications and reduce mortality rates. The pathophysiology of TSS involves the release of superantigens by *Staphylococcus aureus* and *Streptococcus pyogenes*, triggering a systemic inflammatory response that can lead to multi-organ failure. While clinical features of TSS can vary, the hallmark signs include fever, hypotension, rash, and multi-organ involvement, with rapid deterioration observed without early intervention. For effective management of TSS, nursing interventions are critical in ensuring the early recognition of symptoms and the initiation of appropriate therapies. Nursing care must focus on continuous monitoring of vital signs, especially blood pressure and temperature, to detect any signs of shock early. Early identification of multi-organ involvement is crucial, as this can guide the adjustment of treatment plans and the prioritization of organ-specific support. In the management of TSS, aggressive fluid resuscitation is a cornerstone intervention, as it helps to stabilize blood pressure and improve tissue perfusion. Fluid management, coupled with the prompt administration of antibiotics, plays a vital role in controlling the infection. A combination of beta-lactamase-resistant antibiotics, such as flucloxacillin or cephalosporins, along with clindamycin, is recommended due to its ability to suppress bacterial toxin production and enhance the effectiveness of antibiotic therapy. Moreover, nursing staff must be well-versed in the importance of source

control—identifying and managing the infection source, whether it is a surgical wound, foreign body, or soft tissue infection. TSS can present with subtle or delayed signs, and healthcare providers must maintain a high level of suspicion, especially in high-risk pediatric populations. In conclusion, nursing care plays an essential role in the successful management of TSS. Timely interventions, including monitoring, fluid resuscitation, and antibiotic administration, are essential to improving outcomes. Nurses must remain vigilant for early signs of this critical syndrome to ensure the best possible patient prognosis.

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التدخلات التمريضية لمتلازمة الصدمة السمية - مراجعة محدثة

الملخص:

الخلفية :متلازمة الصدمة السمية (TSS) هي حالة تهدد الحياة وتتميز بحمى مفاجئة، وهبوط في ضغط الدم، وطفح جلدي واسع النطاق، وعجز متعدد الأعضاء، وغالبًا ما تكون ناتجة عن التهابات بكتيرية ناتجة عن *المكورات العنقودية الذهبية* و *المكورات العقدية المقيحة* .تم وصف TSSلأول مرة في عام 1978 وقد ارتبطت هاتان السلالتان البكتيريتان بحالة شديدة من خطر الوفاة إذا لم يتم علاجها بسرعة. يعد التشخيص المبكر والعلاج أمرًا بالغ الأهمية لتقليل معدل الوفيات، خاصة لدى المرضى الأطفال.

الهدف :تهدف هذه المراجعة إلى استكشاف الفيزيولوجيا المرضية، والسمات السريرية، واستراتيجيات الإدارة لمتلازمة الصدمة السمية، مع التركيز على التدخلات التمريضية وأهمية التعرف المبكر .الطرق :تستعرض هذه المراجعة العرض السريري لTSS ، بما في ذلك المعايير التشخيصية، ودور السوبرانتجينات في عملية المرض، وتأثير التدخل السريع. كما تستعرض المقالة الأدلة المتاحة حول التدخلات التمريضية الفعالة، بما في ذلك الإنعاش بالسوائل، والعلاج بالمضادات الحيوية، والرعاية الداعمة، لضمان النتائج الإيجابية.

النتائج :غالبًا ما تتميز الأعراض السريرية لـ TSS بثالوث من الحمى الشديدة، وهبوط ضغط الدم، واشتراك الأعضاء المتعددة. يعتبر العلاج بالمضادات الحيوية، بما في ذلك العوامل المقاومة للبيتا لاكتاماز وكليندامايسين، أمرًا حاسمًا في إدارة .TSS كما أن التشخيص المبكر، والإنعاش السائل العدواني، والسيطرة على المصدر تحسن بشكل كبير معدلات البقاء على قيد الحياة. تلعب الرعاية التمريضية دورًا محوريًا في إدارة حالة المريض الديناميكية الدموية وضمان الإعطاء في الوقت المناسب للعلاجات .

الاستنتاج :لا تزال TSS حالة حاسمة تتطلب اهتمامًا طبيًا فوريًا. التدخلات التمريضية في الوقت المناسب، بما في ذلك مراقبة العلامات الحيوية، وإدارة السوائل، والإعطاء السريع للمضادات الحيوية، أمر أساسي لتحسين نتائج المرضى. يجب على مقدمي الرعاية الصحية الحفاظ على شكوك سريرية عالية، خاصة في الحالات الأطفال، لبدء العلاج على الفور وتقليل خطر الوفاة .

الكلمات المفتاحية: متلازمة الصدمة السمية، الرعاية التمريضية، السوبرانتجينات، العلاج بالمضادات الحيوية، الإنعاش بالسوائل، الرعاية للأطفال.