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Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: A Guide for Nurses

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Abstract

Nurses are central to the care of patients with Stevens-Johnson syndrome and toxic epidermal necrolysis. Patients with these conditions present with nuanced symptoms and have complex nursing care needs. Although much of the exact pathophysiology of these diseases is not known, all nurses benefit from a fundamental understanding of the genesis of skin manifestations, associated pharmacology, and prognosis. The care of patients hospitalized with Stevens-Johnson syndrome and toxic epidermal necrolysis consists of wound care, infection prevention, comfort management, hydration and nutrition, psychosocial support, and the prevention of long-term complications. This article provides an overview of these diseases, including clinical diagnosis, history and physical assessment, related pharmacology, and nursing care priorities. A description of the current state of the science in clinical management for nurses at all levels is provided, with an emphasis on nursing's contribution to the best possible patient outcomes.

Keywords

burn care; nursing; skin disorders; Stevens-Johnson syndrome; toxic epidermal necrolysis

Stevens-Johnson syndrome (SJS) was discovered and named in 1922, and toxic epidermal necrolysis (TEN) was first described in 1956. Nearly a century later, the exact pathophysiology of these diseases is still not fully understood. Although both conditions have been broadly defined as a cutaneous hypersensitivity reaction resulting in skin sloughing,¹ they were not precisely differentiated until the late 20th century, with SJS considered to involve less than 10% of the body and TEN to involve more than 30% of the body. Although SJS and TEN are relatively rare, patients with this dermatologic emergency present several unique management and daily care challenges for nurses. Nursing care in SJS and TEN is central, as management focuses on wound healing, hydration, nutrition, infection prevention, comfort, and the prevention of long-term complications. Nurses, skilled in complex wound care and infection prevention, are in an ideal position to help patients

with SJS or TEN achieve good outcomes despite their challenging course. The purpose of this article is to describe the process of identifying and diagnosing SJS and TEN in patients of all ages, review the pathophysiology and common causes, describe nursing-specific clinical management, and consider the prognosis for SJS and TEN patients.

Pathophysiology

Immune Response

Most of the manifestations of SJS and TEN result from an aberrant reaction in the mucosa to a presenting agent, such as a drug or virus. Although most of the clinical sequelae involve the skin and mucous membranes, SJS and TEN can also cause multisystem organ failure, respiratory failure, and respiratory arrest.² To understand the pathophysiology, it should be remembered that cytotoxic T lymphocytes (CTLs) are associated with antigen-specific immune responses and play a role in recovery from viral illness and in allograft rejection. These T cells can destroy cells using 1 or more mechanisms, including those involving perforin and granzyme, Fas and Fas ligand (FasL), and tumor necrosis factor α (TNF- α). In SJS and TEN, when the tissue is histologically incompatible with a presenting drug or virus, it triggers the production of CD8⁺ CTLs, which travel to the skin and stimulate keratinocyte cell death (apoptosis).^{3,4} Keratinocytes are the skin cells that begin in the dermis and migrate upward to form squamous cells on the surface of the skin. The keratin protein they produce forms the strength of a person's skin, hair, and nails. When keratinocyte apoptosis is accelerated, skin lesions and breakdown can occur.

A hypersensitivity reaction occurs when the immune system overresponds to an allergen or antigen. Hypersensitivity reactions are classified as either immediate-response, which occur rapidly after exposure and are usually IgE mediated, or delayed-response, which occur more than 72 hours after exposure and are typically T cell mediated. Immediate reactions can manifest as respiratory and circulatory collapse as in anaphylaxis and may also include skin reactions such as urticaria and angioedema. In contrast, delayed reactions typically include maculopapular, bullous, or pustular drug eruptions on the skin.⁵ Approximately 10% to 15% of hospitalized patients and 6% of the general public experience an adverse drug reaction, of which hypersensitivity reactions account for about 15% to 20%; however, the true incidence of hypersensitivity reactions is largely unknown.¹ Stevens-Johnson syndrome and TEN are examples of rare but potentially fatal cutaneous delayed-response hypersensitivity reactions.

Etiology

Stevens-Johnson syndrome and TEN have an annual incidence of about 1 to 7 cases per million people per year,^{3,6,7} with SJS cases outnumbering TEN cases by nearly 3-fold.⁶ For unknown reasons, women are more likely than men to suffer from SJS and TEN,⁶ and rates are higher in pediatric patients younger than 10 years and adults older than 80 years. The incidence and severity also increase for immunocompromised patients, such as those who have undergone bone marrow transplantation or have had malignancies and those with HIV or chronic rheumatologic conditions. The annual incidence of TEN for those with HIV is approximately 1000-fold higher than in the general population, or approximately 1 person per 1000 people with HIV per year.⁸

Medication reactions are responsible for nearly 90% of SJS and TEN cases.⁶ The most common inducing drugs include sulfonamides, antiepileptics, antibiotics, nonsteroidal anti-inflammatory drugs, and other drugs such as allopurinol and chlormezanone.^{1,3,6,8} Other known causes include infections such as *Mycoplasma pneumonia* and herpes simplex and autoimmune diseases such as lupus erythematosus.⁸ Rarely, SJS and TEN have been attributed to vaccinations or contrast medium or considered a manifestation of acute graft-versus-host disease following bone marrow transplantation.⁹ Some cases have no known association with a drug or preceding illness.

Regional differences in drug prescription patterns and demographic differences have an impact on the incidence of SJS and TEN.⁸ The diseases affect certain demographic groups more than others when specific medications are taken. People with Han Chinese and Taiwanese backgrounds have a higher incidence of a specific HLA allele (HLA-B1502), which, when presented with the antigen from carbamazepine, initiates the T cell-mediated immune response that is associated with SJS and TEN.⁴ The proposed relationships between different HLA allele types and drug reactions are presented in Table 1.³ People from Southeast Asia, Japan, Korea, and Europe may have 1 or more HLA alleles that have been found to interact with some medications. As a result of these HLA-specific drug reaction findings, the US Food and Drug Administration suggests genetic screening for patients whose ancestry has high rates of HLA-B1502 before prescribing carbamazepine or other similar medications with known interactions.³

Pathogenesis Pathways

Over the last 15 years, our understanding of the pathogenesis of SJS and TEN and similar delayed-response cutaneous reactions has evolved. These reactions have been confirmed to be an immune-mediated, severe hypersensitivity response that results in the release of mediators responsible for increased keratinocyte apoptosis. That apoptosis results in the classic presentation of visible blistering and sloughing of the skin and mucous membranes. Multiple theories have been developed on how the molecular compounds of certain medications are actually recognized by T cells. The hapten/prohapten theory hypothesizes that the drug or its metabolite reacts with a self-protein through covalent bonding to produce a haptenated product.⁴ That haptenated product is then transferred onto the cell surface, where it is recognized, stimulating the abnormal T-cell reaction. A second theory, the pharmacological interaction with immune receptors theory, suggests that there is a noncovalent, labile interaction of a specific drug with the major histocompatibility complex (MHC) receptor at the cell surface, resulting in MHC-dependent T-cell stimulation.⁴ This theory would explain why patients with certain demographic backgrounds are more likely than others to demonstrate this reaction to specific medications. The last theory is referred to as the altered repertoire model. In this model, drugs or their metabolites bind noncovalently to certain MHC molecules with extreme specificity, allowing a new repertoire of endogenous self-peptides to be bound and presented.⁴ This process fundamentally alters the MHC molecule, causing increased T-cell stimulation. Despite the questions that remain about precisely how T cells recognize certain drugs, the process results in a fundamental change in the cell structure, stimulating T cells to induce apoptosis of epithelial cells to produce the skin manifestations of the disease.

In addition to the various proposed methods of T-cell recognition, a number of potentially cytotoxic proteins and cytokines are associated with apoptosis and the development of SJS and TEN. These include Fas, FasL, granulysin, reactive oxygen species, TNF- α , TNF-related apoptosis-inducing ligand (TRAIL), and interferon gamma. Granulysin and Fas have been most recently recognized as the 2 key players in the pathogenesis of SJS and TEN.^{3,9} When released, both proteins trigger disseminated keratinocyte apoptosis. Specifically, granulysin and Fas attack the bonds between the epidermal and dermal layers, which causes separation and subsequent blistering and sloughing. Sloughing can involve not only the skin but also the eyes, mouth, respiratory and gastrointestinal tracts, and vaginal mucosa.¹⁰ Fas is a membrane-bound protein that causes apoptosis upon activation. Under normal conditions, FasL acts to bind Fas onto target cells. FasL is typically produced by CTLs and natural killer (NK) cells. Interestingly, histological studies of TEN skin suggest that keratinocytes, not lymphocytes or killer cells, produce the FasL in TEN pathogenesis, leading to their own destruction.³ FasL is present in the sera of TEN patients, which supports the role Fas plays in TEN pathogenesis. A common treatment for SJS and TEN patients is the administration of intravenous immunoglobulin (IVIG). It theoretically works by blocking the activity of keratinocyte-bound FasL or FasL-binding monoclonal antibodies and preventing further keratinocyte apoptosis. Despite these findings, no confirmative association has been established between FasL levels and disease severity.³

The most recent focus in the pathophysiology of SJS and TEN is on granulysin. Granulysin causes apoptosis through cell-mediated cytotoxicity without direct cell-to-cell contact. Similar to Fas, granulysin is secreted by CTLs and NK cells, which, in addition to NK T cells, have been found in high volumes in SJS and TEN blister fluid. The granulysin molecules found within SJS and TEN blister fluid are associated with high cytotoxic activity, and there is a correlation between granulysin levels and the severity of cutaneous involvement.³ In mouse models, the introduction of granulysin induces keratinocyte apoptosis, and the mouse displays symptoms very similar to those of SJS and TEN.⁴

Other cellular pathways believed to lead to apoptosis via Fas and FasL expression include the release of reactive oxygen species related to oxidative stress. The reactive oxygen species directly cause intracellular keratinocyte damage and apoptosis by damaging the intracellular membranes. Once damaged, the keratinocyte has increased FasL expression and produces TNF- α , an inflammatory cytokine that increases the expression of Fas and FasL, contributing to further apoptosis. Downey et al³ suggested that after the initial apoptotic cascade, cytokines such as interferon gamma and TRAIL are also released, potentiating additional keratinocyte damage.

Overall, keratinocyte damage and apoptosis are the key drivers of the clinical manifestations of SJS and TEN. Although the exact pathways are not yet completely known, they largely involve cytokines, oxidative stress, and the presence of cytotoxic proteins. The immune-mediated response that has been described is the basis of treating the disease with IVIG, corticosteroids, and cyclosporine, among other similar agents.

Patient History and Disease Presentation

Stevens-Johnson syndrome and TEN are most often the result of an adverse drug reaction, although rarely they are triggered by an infection.^{4,8} Treatment is a dermatologic emergency, and initial management should include (1) cessation of the offending medication or agent and (2) referral of the patient to dermatology and acute wound care specialists, likely in a burn center or similar environment.¹⁰⁻¹² The first priority is early identification and cessation of the offending medication to halt the progression of further apoptosis.^{3,10,13} Drug pharmacokinetic knowledge is crucial to determine the effects of metabolites with long half-lives and frame expectations of disease progression and management based on each individual patient's history and exposure. The reaction consists of 3 phases with distinct symptoms (Figure 1). Illness commonly includes a prodromal phase of 1 to 3 days that precedes any visible skin manifestations. The prodromal phase is characterized by symptoms such as fever, malaise, eye pain, and upper respiratory symptoms such as coughing and sore throat.¹⁴ Full onset of SJS and TEN typically occurs between 4 and 28 days after the drug exposure, so it is important to ask the patient for a full medication history over the last 30 days, including all over-the-counter medications and complementary and alternative therapies.¹⁵ It may be easier to identify the causative agent in patients who do not take many medications than in those who take multiple medications or have recently changed regimens. It may be helpful to plot the patient's medications on a timeline chart similar to the one presented in Table 2 that includes the drug name, dose, date the drug was started and discontinued, and whether that drug is commonly associated with SJS or TEN.

Physical Assessment

Physical assessment should include a head-to-toe skin examination, including the inside of the mouth, the eyes and eyelids, and the genitalia. The earliest signs of disease are typically purpuric macules, usually appearing on the proximal arms and legs and on the face and torso. The lesions tend to spread out from those areas as the disease progresses. Involvement of the palms or soles of the feet is very common. A hallmark of SJS and TEN is the ability to easily tear the dermis with little force. Nikolsky sign is present when a small amount of lateral pressure on a lesion causes the detached epidermis to slide over the dermis and can confirm SJS or TEN as well as other skin disorders such as pemphigus.¹⁵ In areas where the epidermis becomes necrotic and separates from the underlying dermis, fluid-filled blisters form. Blister formation typically peaks 5 to 7 days after disease onset. Eventually, the blisters become fragile and break, resulting in large sheets of epidermal sloughing, leaving an exposed, weepy, painful, and infection-prone dermis.

Broadly, the severity of disease is indicated by how much skin is sloughing or detached in terms of the percentage of the patient's total body surface area (TBSA) that is involved. Calculation of the precise percentage of TBSA involved should use a diagram¹⁶ similar to the one presented in Figure 2 for accuracy. Note that each body area represents an overall percentage of the whole body, and that percentage is different for adults compared with children. Stevens-Johnson syndrome is considered less severe and is characterized by skin detachment on less than 10% of the TBSA and widespread purpuric macules.⁸ Overlap between SJS and TEN occurs when there is skin detachment on 10% to 30% of the TBSA,

plus widespread purpuric macules.^{8,17} Toxic epidermal necrolysis involves detachment of more than 30% of TBSA plus purpuric macules.^{8,17} Alternatively, TEN can occur without purpuric macules when more than 30% of TBSA is detached in large epidermal sheets.¹⁷

On initial presentation, it is helpful to complete a skin diagram drawing, such as the one pictured in Figure 3, that documents separately the areas of purpuric macules and skin detachment. This diagram will also help in the calculation of the percentage of TBSA involved. Using the Lund-Browder¹⁶ diagram (Figure 2) and visual assessment, note the percentage of detached skin (0%-100%) and the percentage of skin with macules present only. The percentage of detached skin, not the area with macules, is used to calculate severity and fluid replacement and to gauge disease progression.

Although no statistical correlation has been found between the percentage of TBSA involved and death, SJS and TEN patients who have involvement of other organ systems are at high risk of multiorgan dysfunction syndrome and sepsis. Sepsis is the leading cause of death in SJS and TEN patients.¹³ It is important to update the skin diagram frequently, noting if the macular areas or areas of detached skin change in appearance or size. If permitted per hospital policy, adding photographs to the patient's chart on a daily basis is helpful in determining the progression and diagnosis. Creamer et al¹⁵ suggest that SJS and TEN patients with more than 10% sloughing should, at minimum, be transferred to an intensive care unit owing to the risk of additional organ involvement. Despite the utility of classifying the disease according to the percentage of TBSA affected and the percentage of detached skin, a skin biopsy is necessary to confirm the diagnosis of SJS or TEN. It is also important to obtain bacterial swabs of the involved skin at various sites initially and on an ongoing basis to monitor for skin infection.¹⁵

It is important to inspect the mouth and tongue and to examine the airway clinically to determine airway and oral mucosal involvement. If the patient has a cough, dyspnea, or increased secretions or is coughing up blood, airway involvement should be immediately suspected and respiratory status monitored closely. A study of adults performed by de Prost et al² indicated that 1 in 4 SJS and TEN patients will require mechanical ventilation as a result of bronchial involvement. This proportion increases to 60% for patients with involvement of more than 30% of TBSA on admission. The mortality rate is nearly 60% to 70% for those requiring mechanical ventilation.² This marked increase in mortality necessitates an early and thorough initial evaluation of the airway and lungs. The threshold for intubation should be low in these patients and include consideration of the patient's ability to manage their secretions and the need for vasopressors upon intubation.

Bronchoscopy to look for bronchial epithelial lesions is often performed, as their presence is associated with increased infiltrates and a need for more antibiotics.² Bronchoscopy can also facilitate mechanical removal of sloughed bronchial epithelium, which can help prevent both obstruction and atelectasis.¹⁵

Laboratory tests that are helpful include complete blood count, erythrocyte sedimentation rate, C-reactive protein, bicarbonate, blood urea nitrogen and electrolyte panel, liver function tests, coagulation studies, and *Mycoplasma* serology (to rule out *Mycoplasma* infection).¹⁵ Trending these laboratory values can be useful in monitoring patient progression and allow

for early identification of complications, such as infection, electrolyte imbalances, or negative effects on the liver and kidneys. It is also good practice to obtain stable intravenous (IV) access through noninvolved skin to administer fluids and pain medications as needed.

Prognosis

Severe SJS and TEN lead to multiorgan system failure and ultimately death. Several factors are associated with higher mortality, including delayed transfer to a specialist unit, older age, greater percentage of TBSA involvement, sepsis, and granulocytopenia.¹⁵ Once laboratory results are obtained, they may be used along with the physical assessment and history findings to help predict mortality. A mortality prediction tool called the SCORTEN¹⁸ has been used historically, and more recently a tool called the ABCD-10¹⁹ has gained popularity. Both tools use clinical parameters known to predict mortality and sum those to predict mortality probability or odds. A higher score on either scoring system indicates a higher probability of mortality. Table 3 provides a comparison of the SCORTEN and the ABCD-10 according to the parameters measured and associated mortality odds. These tools are meant to provide anticipatory guidance rather than absolutely accurate predictions for each individual patient's presentation and disease progression.^{18,19}

Nursing Care Priorities

Treatment of SJS and TEN is largely nursing care focused and supportive, revolving around fluid replacement, comfort management, nutrition support for wound healing, infection prevention, and wound care of the affected areas. A summary of these recommendations and their rationale is provided in Table 4.

Care Location and the Patient Room

In the latest revision of the American Burn Association's burn center referral guidelines,²⁰ SJS and TEN patients are noted to benefit from the interdisciplinary nature of burn centers and their clinicians' experience in managing large wounds and the sequelae of extensive skin loss. Studies show that delay in transfer to a specialized center such as a burn center results in higher mortality rates for patients with more than 10% of TBSA detachment and TEN.¹⁰⁻¹² If transfer to a specialist center is not feasible, a multidisciplinary team approach is necessary and should include consultations with specialists in dermatology and/or plastic surgery, ophthalmology, critical care, respiratory therapy, physical therapy, and diet. Additional referrals should be considered on a case-by-case basis depending on disease progression and extent of organ involvement.

In addition to the hypermetabolic state caused by SJS and TEN, patients with sloughing skin lose additional albumin and protein via the fluid that forms inside blisters. Once the skin has opened or a blister has broken, the patient can become dehydrated from insensible losses. Patients with large or deep burn injuries are often cared for in patient rooms that are heated and humidified to lower energy consumption and reduce insensible fluid losses.²¹ Patients with SJS and TEN may also benefit from a heated and humidified room environment.⁶ In burn patients, raising the room temperature to 33 °C may decrease the resting energy expenditure to approximately 1.4 times rather than 2 times the normal resting expenditure.²¹

Patients with SJS and TEN who have 10% or more of the TBSA detached should be cared for in a private room with humidity control. The room temperature should be maintained at between 25 and 28 °C.¹⁵ A private room lowers the risk of bacterial cross-contamination between patients.

Airway Assessment and Management

After the initial airway assessment on admission, sloughing of the airway mucosa should continue to be monitored. If the patient is intubated, respiratory arrest can occur as a result of coughed bronchial slough suddenly occluding the endotracheal tube. For this reason, nontraumatic suctioning of the airway (ie, endotracheal suctioning to an appropriate depth, stopping when met with any resistance, with the use of a soft-tipped catheter) should be performed as needed to maintain tube patency, and supplies for emergent reintubation should be kept close to the patient. Equipment that allows for fiber-optic intubation may facilitate successful airway placement in these patients.

Most patients with SJS and TEN who have airway or oral mucosal involvement do not require intubation.²² Sloughing of the mucosa of the tongue, palate, and oropharynx is very common but does not typically compromise the airway. Patients with oral mucosal sloughing may have trouble chewing and swallowing food. To prevent choking, all foods should be soft, moist, and low in acidity.¹⁵ Symptoms such as hoarseness, coughing, coughing up blood, or shortness of breath signal progressive airway involvement and should be further investigated to determine the need for airway protection.²²

Daily Ongoing Skin Assessment

A thorough head-to-toe skin assessment should be performed daily for all patients with SJS and TEN. Nurses should assess for increased slough, worsening rash, signs of reepithelialization, and evidence of possible infection. The assessment should include documentation of the percentage of TBSA with papules and the percentage of TBSA containing detached skin. If a dressing change will be performed each day, full skin assessment should occur then. If dressings will remain intact, the appearance of the visible skin and any exudate or staining visible on the outside of intact dressings should be noted. Any changes from baseline should be discussed with the multidisciplinary care team, as they may alter the plan of care for the patient.

Baseline compromised skin integrity, the presence of medical devices such as urinary catheters, feeding tubes, and ventilator tubing, impaired nutritional status, and decreased mobility place SJS and TEN patients at increased risk for hospital-acquired pressure injuries. The use of specialty beds and/or equipment may be needed to reduce this risk.¹⁵ The patient should be examined at regular intervals for signs of pressure injury and a wound specialist consulted as early as possible to prevent further damage.

Prevention of Skin Separation

Prevention of further skin detachment should be a nursing clinical priority during the acute phase of illness. All available measures should be taken to reduce friction and shear, including use of specialty mattresses and maximal assistance with bed changes, turning, and

toileting or continence care. Medical devices should be used only if absolutely necessary, particularly those with an adhesive backing such as medical tapes or those that could cause shearing such as blood pressure cuffs.¹⁵ Unless the patient is unstable or there is an area of intact upper arm skin, the use of a blood pressure cuff over affected areas should be discouraged. The patient's identification bracelet may need to be attached to the patient's clothing or to their gauze dressings with a safety pin to minimize skin contact. The removal of wound dressings should be performed by staff members familiar with skin-altering disease processes and should be as gentle as possible.

Toileting may be a challenge for patients with perineal or lower-extremity skin involvement. In some cases, a fecal management system may be a good choice to keep the perineum clean and alleviate pain from sitting on a bedpan or toilet seat. If the patient is using a bedpan or toilet, padding the seat with soft nonadherent gauze may help prevent skin separation and promote comfort.

Wound Care

As the epithelium is sloughed off, an injury similar to a superficial partial-thickness burn occurs, leaving exposed, weepy, painful skin. Wound management is focused on preventing infection in open skin areas, limiting insensible fluid losses, and minimizing the patient's pain and discomfort. Despite the risk of infection, prophylactic antibiotic use is not recommended.^{6,10,15,23} Wound management can be done with very conservative daily wound care, surgically, or a combination of both. Research does not show evidence of one management method being superior to the other, but many burn centers use a conservative approach initially.⁶ The conservative approach typically involves leaving denuded skin intact to act as a biological barrier. If blisters are present, fluid should be aspirated or bullae partially unroofed with skin left in place. A variety of dressings can be used over the denuded skin, but typically a petroleum- or emollient-impregnated nonadherent gauze is chosen. Use of a nonadherent gauze prevents loss of the skin barrier, lessens insensible losses, and promotes reepithelialization.^{6,24} Antimicrobials such as bacitracin, silver sulfadiazine, or mafenide acetate should be used only on sloughed areas and where infection is present. These dressings are typically changed at least daily, which can be very painful for the patient. Advanced and longer-wear dressings such as silver-impregnated foams can be used, although no difference in healing time has been found with these dressings compared with nonadherent dressings.²³ They may facilitate less frequent dressing changes and thus be more comfortable for the patient and require less staff time.

When conservative measures fail and wound healing is delayed, infection occurs, or continued progressive sloughing is present, surgical intervention may be performed.^{6,15,23} Surgical intervention typically involves removal of denuded skin in the operating room followed by placement of a primary dressing (one that will remain in place for days or weeks) such as a biologic agent, a xenograft, or an allograft.⁶ Secondary dressings, or coverings for the primary dressing, vary depending on the choice of primary dressing and are likely to still include the use of nonadherent gauze. The benefit of surgical intervention is less frequent full dressing changes and the ability to use general anesthesia for pain management, but ultimate wound care choices should be based on patient condition. Either a

conservative or a surgical approach may need to change over time depending on disease progression and patient response.

Infection Prevention

As previously stated, the use of prophylactic antibiotics is not recommended; however, sepsis is the leading cause of death in SJS and TEN patients. Therefore, infection prevention is of paramount importance and falls largely on the shoulders of the care team. Wound infection prevention is achieved through careful and diligent daily wound care, including daily cleansing of areas surrounding denuded skin and gentle cleansing of open areas using irrigation. Cleansing is accomplished using warmed sterile water or saline solution or with a topical cleansing agent approved for use over sloughing areas, such as chlorhexidine solution.¹⁵ The use of sterile gloves during wound care is not required but may reduce risk in some patients. Wounds should be assessed during dressing changes, and bacterial swabs of several areas may be ordered to track bacteria presence and screen for wound sepsis.¹⁵ If a wound infection is present, the dressings in that area should be removed first and cultured and the wound redressed. Once the infected area is covered, the provider should remove their gloves, wash their hands, and open fresh supplies before beginning a dressing change of an uninfected area to prevent cross-contamination.

The risk of central catheter-associated bloodstream infection increases if the catheter is placed near or through areas of slough. In these instances, securing central catheter dressings can be challenging and the dressing may need to be changed more frequently than usual. Methods of central catheter dressing care in burn patients with similar challenges include painting gauze with povidone-iodine swabs, using an antimicrobial disc, using a semipermeable dressing with chlorhexidine, or some combination of these approaches.²⁵ In addition to maintenance of a central catheter dressing, the general principles of removing the catheter as soon as possible, handwashing, and the use of clean gloves and an alcohol swab of access ports before use are important.²⁶

Additional considerations may apply when determining nursing assignments to prevent cross-contamination of patients with infections such as methicillin-resistant *Staphylococcus aureus* or vancomycin-resistant *Enterococcus* with the SJS or TEN patient because of their compromised skin integrity.

Intake and Output

Precise fluid resuscitation using isotonic solutions is necessary and should not be overly aggressive. Fluid administration should cover the insensible fluid loss that results from sloughing and essential hydration in patients who are not taking food or fluids by mouth. For reference, fluid losses are approximately 2 to 3 L/d in adults with 50% of their skin sloughing or blistered.¹⁰ Isotonic crystalloid fluids should be individualized and titrated to maintain a urine output of 0.5 to 1.0 mL/kg/h in adults.^{6,10} Fluid needs can be calculated by referring to the percentage of TBSA obtained in the physical assessment. Only the detached skin areas are included in this estimation. For patients with more than 20% of TBSA detached, a rate of 2 mL/kg/%TBSA divided by 24 hours is a good starting point to maintain a urine output of 0.5 to 1 mL/kg/h.⁶ Daily assessment of new sloughing and/or healing

should be documented and used to determine the real-time percentage of TBSA involved and the ongoing fluid needs. Overresuscitation should be avoided to prevent associated complications such as pulmonary edema. During the acute phase, urine output, heart rate, blood pressure, lung sounds, and edema are appropriate indicators of fluid status.

Nutrition Support

The immunologic process that occurs in SJS and TEN patients is associated with increased catabolic demands resulting in a need for nearly double the usual number of calories.²⁷ In response to this increased need, enteral nutrition should be initiated early, with parenteral nutrition used only when the former is not an option. Early enteral nutrition is associated with improved outcomes and faster wound healing.^{3,10} Either by oral intake or through an enteral feeding tube, the goal is for the patient to receive about 20 to 25 kcal/kg/d in the acute phase of illness and 25 to 30 kcal/kg/d during the recovery phase to promote healing.²⁸ Close collaboration with a registered dietitian and the patient and their caregivers is key to achieving this goal.

Comfort

Pain management can be one of the most challenging aspects of caring for a patient with SJS or TEN. The epidermal sloughing results in exposure of dermal nerve endings, causing intense pain.^{3,6} Typically, this pain is worse when open skin is exposed to air, as occurs during dressing changes. No recommendations have been issued specific to the treatment of pain for SJS and TEN patients, and a typical multimodal approach should be used.¹⁵

Pain levels should be assessed using a valid pain assessment tool throughout hospitalization. It is important to capture the pain associated with dressing changes separate from baseline pain. For mild and background pain, use of nonopioid medications such as acetaminophen is recommended. Using nonsteroidal anti-inflammatory drugs is not recommended because of the potential for gastric and renal complications.¹⁵ For moderate to severe pain, short-acting opioids given orally or intravenously should be used.

Wound care can be particularly painful for patients; thus, frequent small doses of fast-acting IV medications such as fentanyl or morphine may be required. Use of IV anxiolytics in combination with analgesic medications may help reduce the patient's anxiety during wound care. Nursing assessment of respiratory, cardiac, and neurologic status should be performed more often when using these medications. Other pain management techniques include the use of topical analgesics such as lidocaine, particularly when placing devices such as nasogastric tubes or urinary catheters. Intubation and mechanical ventilation may be considered for poorly managed pain in the acute phase of illness.²² Nonpharmacological techniques to consider include distraction using music, positions of comfort, family support, and reassurance.

Eye Protection and Care

Of all the manifestations of SJS and TEN, ocular involvement and sequelae are among the most serious. Between 60% and 100% of adult patients have some type of eye involvement in the acute phase, and 20% to 79% of survivors suffer from long-term eye problems

including chronic inflammation, dryness, scarring, and blindness.²⁹ An ophthalmology consultation within the first 24 to 48 hours of admission is recommended and has been shown to mitigate long-term symptoms.²⁹ Acute ocular involvement can range from hyperemia to near total sloughing of the ocular surface. Ocular epithelial involvement can lead to improper distribution of tears, destruction of lubricating glands, an inability to properly blink, and decreased tear expression.^{29,30} During the acute phase of illness, saline flushes, lubricating ointments, occlusive dressings, and topical steroids may be used to protect the eyes, particularly if the eyelids cannot close completely.²⁹ If the eyelids are involved or there is a pseudo-membrane or conjunctival defect, amniotic membrane transplantation (AMT) should be considered.^{29,31} Amniotic membrane transplantation can occur either at the bedside with procedural sedation or in the operating room. The benefits of AMT include reduced inflammation, enhanced reepithelialization, and reduction of scarring, resulting in improved vision, fewer lid complications, and decreased corneal haze after recovery.^{30,32} To ensure the best patient outcome, AMT should be initiated within the first 7 to 10 days of disease onset.²⁹ Beyond that time frame, irreversible scarring of the eye can occur. Given the low risk associated with AMT placement and use, it is strongly recommended if any eyelid involvement is suspected.^{15,29}

Oral Care

Involvement of the mucosal lining, tongue, and lips can be painful and problematic. Pain associated with oral sloughing can prevent oral intake and further complicate issues related to nutrition for SJS and TEN patients. Oral intake should be encouraged with soft, bland, nonacidic food choices. Antiseptic and analgesic mouthwashes or sprays should be used before eating and throughout the day to reduce pain and bacterial colonization in the mouth.¹⁵ When the lips are affected, they can appear bloody, black and crusty. Application of plain white petroleum or paraffin ointment every 2 hours or more frequently can improve pain and complications with mouth closure.¹⁵ As with other affected areas, the mouth and surrounding areas should be assessed daily along with regular swallow assessments to evaluate for involvement of the esophagus.

Oral care of intubated patients is universally important to prevent pneumonia³³ and must be performed with care in SJS and TEN patients. The mouth of the intubated patient should be cleaned daily using a warm saline-soaked oral sponge, sweeping the soft tissue gently to prevent fibrotic scars.¹⁵ An antimicrobial mouthwash approved for intubated patients should be used at least twice daily according to institutional protocol.³³ It may be necessary to dilute 0.2% chlorhexidine mouthwash by up to 50% to reduce soreness.¹⁵ Fungal infection may develop in the mouth; if present, it should be treated with nystatin oral suspension as directed.¹⁵

Genitourinary Care

It is common for the epithelium of genitourinary areas to also slough and blister. In many cases, a urinary catheter should be placed soon after admission to monitor urine output closely and prevent adhesion formation in the urinary tract.⁶ Adhesion within the vulvovaginal tract can lead to long-term complications for female patients, particularly children, and may require manual lysis and a vaginal dilator.³ Consultation with a

gynecologist is strongly recommended for all female patients with a diagnosis of SJS or TEN.^{3,13} The most common complications include strictures and stenosis of the urethra, phimosis in males, vaginal scarring in females, and urinary and sexual dysfunction.¹⁵ To prevent these complications, application of plain ointments to the urogenital skin at regular intervals is indicated.¹⁵ If skin in this area is open, a nonadherent dressing may be applied to the area to prevent adhesions and promote comfort.

Psychosocial Considerations

In addition to the physical complications associated with SJS and TEN, many patients experience psychological distress that must be addressed. Recovery from an illness as severe as SJS or TEN can have lasting implications for quality of life. Up to 65% of SJS and TEN survivors suffer from posttraumatic stress disorder–like symptoms including depression, anxiety, and flashbacks.⁷ Patients have reported difficulty sleeping, maintaining relationships, and being physically intimate with their partners. Providers and nurses should address these issues during the hospital stay and after discharge. Depression and anxiety assessment should be performed regularly during the hospitalization, and consultation with specialists in psychotherapy or psychiatry, if available, should be considered. Acute and primary care providers should encourage follow-up with primary care and mental health resources such as a psychotherapist or social worker upon discharge.⁷

Follow-up Care

Follow-up care after discharge should continue the multidisciplinary care provided during hospitalization, as SJS and TEN survivors may continue to experience multisystem symptoms. Any patient with eye involvement should follow up with an ophthalmologist upon discharge. Chronic ocular complications are common and due to physiological changes, including conjunctival keratinization, misdirected eyelashes, and impaired tear formation, resulting in repeated mechanical injury to the ocular surface.²⁹ Vision loss, eyelid malposition, and photophobia are also common and may be treated with eyelash removal, application of topical retinoic acid to the eyelid margin, or use of scleral contact lenses.^{29,30} Scleral contact lenses are large-diameter lenses that rest on the sclera and arch over the cornea. These lenses are typically used in cases of ocular issues such as corneal thinning or ectasia. When used in recovering SJS and TEN patients, they have been found to improve both comfort and vision.³⁰

Female patients with urogenital involvement should follow up with a gynecologist regularly after discharge, as vaginal complications are well documented. Potential healing sequelae include vaginal pain, stenosis, and formation of strictures between labia and within the vaginal canal that can continue to form for months after recovery from acute illness.³⁴ Preventing these complications is important to avoid problems with menstruation and fertility.

Scarring is often a concern among patients and families. Although the incidence of scarring is relatively low in SJS and TEN survivors, other cutaneous complications such as hypopigmentation or hyperpigmentation, nail dystrophies, and problems associated with dermatitis are common.^{8,34} Long-term oral and mucosal complications can also occur,

typically as a result of a lack of saliva production. The lack of saliva increases the risk of tooth decay and loss, scarring of the oral mucosa, and loss of the tongue papillae.^{8,34} As more studies have focused on the chronic complications of SJS and TEN, additional organ involvement lasting beyond the acute phase of illness has been discovered. Survivors with a history of respiratory tract involvement are more likely to be diagnosed with obstructive lung disease and formation of strictures in the esophagus and the trachea, which result in dysphagia, epiglottitis, and esophagitis.^{8,34}

Future Care Priorities

Future research on SJS and TEN will likely focus on preventing the disease by genetic evaluation before medication administration and obtaining a deeper understanding of the most effective pharmacological treatment. Although some genetic associations are known, these diseases often occur in individuals without known genetic risk. After disease onset, treatment is largely supportive, because no pharmacological therapy has been shown to be effective to date. In a recent systematic review of all available pharmacological treatments, the authors found major bias in the reporting of drug trial results.¹⁵ The bias was due to a low number of patients, variation in the timing and nature of drug administration, difference in patient illness severity among cohorts, and variations in the quality of the supportive care. Clinicians with experience managing these patients tend to favor some drug treatments, such as IVIG or cyclosporine, but to date no high-quality evidence of their effectiveness has emerged. One drug, thalidomide, has been associated with an increased risk of mortality, and its use is therefore discouraged.¹⁵ Nurses should be aware that the drug therapy evidence is likely limited by the rareness of SJS and TEN; therefore, administration of drug therapy to patients with active disease should follow institutional protocols.

Until there is a clear genetic explanation for why some patients react to certain drugs in this way, patients with this type of hypersensitivity reaction will continue to be encountered in hospital settings. Nurses are in a good position to help patients avoid the long-term complications of the disease, promote comfort, and improve survival by following these recommendations for daily care, surveillance, and the promotion of wound healing and recovery.

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Figure 1:
Symptom timeline.

Relative percentage of body surface areas (% BSA) affected by growth					
	0 yr	1 yr	5 yr	10 yr	15 yr
a— $\frac{1}{2}$ of head	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$
b— $\frac{1}{2}$ of 1 thigh	$2\frac{3}{4}$	$3\frac{1}{4}$	4	$4\frac{1}{4}$	$4\frac{1}{2}$
c— $\frac{1}{2}$ of lower leg	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{3}{4}$	3	$3\frac{1}{4}$

Figure 2:

Lund-Browder skin diagram for determining percentage of total body surface area involved.

A, Rule of nines. B, Lund-Browder diagram for estimating extent of burns. Reproduced from US Department of Health and Human Services, Radiation Emergency Medical Management. Burn triage and treatment of thermal injuries in a radiation emergency. <https://www.remm.nlm.gov/burns.htm> (public domain)

Relative percentage of body surface areas (% BSA) affected by growth

	0 yr	1 yr	5 yr	10 yr	15 yr
a— $\frac{1}{2}$ of head	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$
b— $\frac{1}{2}$ of 1 thigh	$2\frac{3}{4}$	$3\frac{1}{4}$	4	$4\frac{1}{4}$	$4\frac{1}{2}$
c— $\frac{1}{2}$ of lower leg	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{3}{4}$	3	$3\frac{1}{4}$

Figure 3:

Tracking disease progression using a skin diagram. A, Rule of nines. B, Lund-Browder diagram for estimating extent of burns. Areas outlined without gray indicate erythema only (about 40%); areas with gray have epidermal detachment present (about 20%). Only areas with epidermal detachment are used to calculate the percentage of total body surface area involved in Stevens-Johnson syndrome or toxic epidermal necrolysis. In this case, 20% is the percentage used for fluid replacement calculations and severity determination. Adapted from US Department of Health and Human Services, Radiation Emergency Medical Management. Burn triage and treatment of thermal injuries in a radiation emergency. <https://www.remm.nlm.gov/burns.htm> (public domain)

Table 1:**HLA Antigen–Related Drug Interactions^a**

HLA Type	Drug Interaction
HLA-B1502	Carbamazepine
	Phenytoin
HLA-A3101	Carbamazepine
HLA-B1511	Carbamazepine
HLA-B38	Sulfamethoxazole
	Lamotrigine
HLA-B5801	Allopurinol
HLA-B73	Oxicam nonsteroidal anti-inflammatory drugs

^aData were derived from Downey et al.³

Table 2:

Example Drug History Worksheet for Identifying Potential Causative Agents

Drug Name	Route	Daily Dose	Date Started	Date Ended	Taken Previously?	Known to Cause SJS/TEN? ^a	History of Previous Reactions to Drug?
Ibuprofen	Oral	1600 mg	4/1/20	4/8/20	Yes	Yes	No
Sulfamethoxazole/trimethoprim	Oral	2 Tablets	3/1/20	4/8/20	No	Yes	No

Abbreviations; SJS, Stevens-Johnson syndrome; TEN, toxic epidermal necrolysis.

^aDrugs known to cause SJS and TEN include allopurinol, carbamazepine, lamotrigine, nevirapine, nonsteroidal anti-inflammatory drugs, phenobarbital, phenytoin, sulfur antibiotics, and sulfasalazine.³

Table 3:

Comparison of the SCORTEN¹⁸ and ABCD-10¹⁹ Prognosis Tools

Parameter	SCORTEN	ABCD-10
Age, y	> 40 (1 point)	> 50 (1 point)
Heart rate, BPM	> 120 (1 point)	<i>Not included</i>
Body surface detached, %	TBSA detached > 10 (1 point)	TBSA detached > 10 (1 point)
Bicarbonate level, mmol/L	< 20 (1 point)	< 20 (1 point)
Urea level, mmol/L	> 10 (1 point)	<i>Not included</i>
Active cancer	1 point	2 points
Glucose level, mmol/L	Serum > 14 (1 point)	<i>Not included</i>
Dialysis	<i>Not included</i>	Before admission (3 points)
Total score and interpretation ^a		
Probability of mortality:		
Predicted mortality odds:		
1 = 0-0.1	0 = 2.3	
2 = 0.1-0.2	1 = 5.4	
3 = 0.2-0.4	2 = 12.3	
4 = 0.4-0.6	3 = 25.5	
5 = 0.6	4 = 45.7	
	5 = 67.4	
	6 = 83.6	

Abbreviations: BPM, beats per minute; TBSA, total body surface area.

^aScores range from 1 to 7 for SCORTEN and from 0 to 8 for ABCD-10.

Summary of Nursing Care Priorities

Table 4:

Nursing Care Focus Area	Recommended Interventions	Rationale
Patient location and room	Transfer to specialized center for > 10% total body surface area	Reduced mortality and improved patient outcomes
	Facilitate team collaboration	Ensures patient-centered care
	Arrange for a private room	Decreased infection risk
	Place in a heated and humidified room	Lowered energy expenditure and less fluid loss
Airway	Frequent assessment focusing on symptoms of airway involvement and compromise	Prevent respiratory distress and failure
	Use of fiber-optic intubation	Reduce bronchial sloughing and bleeding
	Nontraumatic and regular endotracheal tube suctioning	Prevent occlusion of endotracheal tube
		Facilitate successful intubation
Skin	Daily head-to-toe assessments and areas of rash and slough	Establish disease progression and healing
	Implement pressure injury prevention measures	Prevent additional skin breakdown
	Protect against skin friction and shear	Reduce damage to affected areas
Wound care	Leave denuded skin in place for a conservative approach	Acts as a biologic infection barrier
		Reduces insensible fluid loss
	Surgical debridement with biologic covering if a conservative approach fails	Requires less frequent dressing changes
		Improves pain management
		Allows for daily skin assessment
Infection prevention		Improves re-epithelialization
	Daily wound care using nonadherent gauze to cover sloughed areas	Reduces fluid loss
	Use of prophylactic antibiotics is not recommended	Antibiotics are only recommended for a known infection
	Diligent daily wound care and cleansing of surrounding skin is the best prevention techniques	Daily wound care and cleansing will decrease risks of sepsis
	Central line catheter care and management may need to be tailored	Impaired skin integrity places patients with Stevens-Johnson syndrome/toxic epidermal necrolysis at higher risk of central line-associated blood stream infection
Intake and output	Use isotonic crystalloids to maintain urine output of 0.5-1.0 mL/kg/h in adults	Reduces risk of fluid overload and renal complications
	Conduct daily cardiopulmonary, renal, and skin assessments to determine fluid status and needs	Fluid requirements will change as disease progresses
	Initiate early enteral feeding via nasogastric tube	Improves wound healing and overall patient outcomes
Pain and comfort	Use a multimodal approach	Pain can be better managed when a variety of analgesics are used

Nursing Care Focus Area	Recommended Interventions	Rationale
Specialty areas	NSAIDs are not recommended	NSAIDs increase risk for renal and gut complications
	Analgesic and anxiolytics are recommended for wound cares	A combination of medications can improve a patient's perception of wound care
	Eyes	Enhances re-epithelialization, reduces scarring and reduces improves vision
	Initiate an ophthalmology consultation within the first 24–48 h	
	Amniotic membrane transplantation placement for any eyelid involvement	
	Mouth	Encourages oral intake, reduces pain and bacterial colonization
	Soft, bland food choices	
	Antiseptic and analgesic mouth sprays	Prevents fibrotic scars
	Frequent application of white petroleum ointment	Prevents ventilator-associated pneumonia
	Thorough oral care for intubated patients	
Genitourinary	Prevents adhesions in urinary and vulvovaginal tract	
Urinary catheter placement		
Manual lysis or vaginal dilator use		
Gynecology consultation for all females		
Psychosocial considerations	Depression and anxiety assessments during hospitalization and after discharge	Addresses and manages posttraumatic stress disorder–like symptoms
	Psychotherapy consultation	
Abbreviations: NSAIDs, Nonsteroidal anti-inflammatory drugs.		