CriticalCareNurse
The journal for high acuity, progressive, and critical care

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Crit Care Nurse 2012;32:52-59 doi: 10.4037/ccn2012340

Published online http://www.cconline.org

Critical Care Nurse is the official peer-reviewed clinical journal of the American Association of Critical-Care Nurses, published bi-monthly by The InnoVision Group 101 Columbia, Aliso Viejo, CA 92656. Telephone: (800) 899-1712, (949) 362-2050, ext. 532. Fax: (949) 362-2049.

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

Drug Reaction, Skin Care, Skin Loss

Karen L. Cooper, RN, MSN, CCRN, CNS, WOCN

Stevens-Johnson syndrome is a rare, potentially fatal drug reaction that causes necrosis of epidermal cells. Early recognition of the syndrome is essential to prevent complications. This article discusses identification, complications, and treatment of Stevens-Johnson syndrome. (*Critical Care Nurse.* 2012;32[4]:52-59)

Stevens-Johnson syndrome (SJS) is a rare, potentially fatal drug reaction that causes necrosis of epidermal cells. The syndrome occurs in approximately 2 to 3 patients per million per year.[1] Caring for patients with SJS is challenging because the syndrome is so rare, and most published accounts describe treatment based on only a few patients, or on patients' anecdotal accounts, and do not address all aspects of care. Although the appearance of a rash may lead to prompt recognition of the drug reaction, the specific drug causing the reaction may not be determined because of the number of medications generally ordered for patients in the critical care unit. SJS may not be diagnosed until the patient begins shedding layers of skin. Because the syndrome occurs infrequently, critical care nurses may not be knowledgeable about the comprehensive care needs for these patients. In this article, I provide information on the etiology and pathophysiology of SJS and the care of patients who have the syndrome.

doi: http://dx.doi.org/10.4037/ccn2012340

Diagnosis

The initial clinical features of SJS in intensive care unit (ICU) patients, who are receiving many medications, may be considered merely the signs and symptoms of an adverse drug reaction or drug allergy, and determining the medication involved is difficult. Increased stress in critically ill patients may lead clinicians to diagnose shingles (herpes zoster), because of the development of blisterlike lesions. However, SJS does not follow a dermatomal pattern. SJS may also initially be misdiagnosed as staphylococcal skin syndrome,[4] iatrogenic chemical burn,[4,5] mycoplasmal pneumonia, or a community-acquired pneumonia associated with epidermal lesions and mucositis.[6] Diagnostic testing for SJS includes allergy patch testing and skin biopsy.[7] Accurate diagnosis of SJS is essential for cessation of the causative agent and initiation of supportive treatment.

Would earlier diagnosis of SJS and discontinuation of the causative medications have changed PJ's outcome (see Case Report)? The answer is uncertain. However, bedside nurses are valuable in notifying physicians of signs and symptoms that require treatment. If nurses are aware of the potential for SJS, they may report findings to the physician by using an SBAR (Situation, Background, Assessment, Recommendation) approach and recommend interventions that will assist physicians in earlier diagnosis and prompt treatment of the syndrome. Early diagnosis and discontinuation of the medications causing SJS are extremely important to prevent progression of epidermal loss and complications.[1-3]

Etiology and Risk Factors

Current evidence[8] strongly suggests that SJS is due to a delayed hypersensitivity response to 1 or more medications. The syndrome has also been associated with malignant neoplasms, AIDS, systemic lupus erythematosus, and viral infections.[9,10] Patients at risk for SJS may have a genetic predisposition to skin reactions to specific medications.[10]

PJ was a 40-year-old woman with a brain abscess who had been discharged from the hospital 2 weeks before the current admission with a peripherally inserted central catheter for administration of intravenous meropenem and sulfamethoxazole plus trimethoprim. She was admitted to the intensive care unit (ICU) from the emergency department with a diagnosis of sepsis due to community-acquired pneumonia. Her chief reasons for coming to the emergency department were malaise and a new rash. PJ's history included systemic lupus erythematosus and steroid-induced diabetes controlled by diet and exercise.

Home medications included promethazine, meropenem, sulfamethoxazole plus trimethoprim, prednisone, paroxetin, phenytoin, metropolol, pantoprazole, and hydrocodone plus acetaminophen. Hospital medications ordered included all the prehospital medications plus potassium and magnesium supplements, vancomycin, gentamicin, flucanozole, insulin per protocol, and acetaminophen.

At the time of admission to the ICU, PJ had blisters, what appeared to be skin tears, and purple bumps over her neck, thorax, and abdomen. She weighed 9 kg (20 lb) less than she had at discharge, her lips were dry, and the mucous membranes in her mouth were reddened. These findings suggested that she might be dehydrated and require fluid replacement. She reported pain when her neck, chest, and abdomen were palpated.

Vital signs in the emergency department included oral temperature 37.6°C (97.9°F), heart rate 120/min, blood pressure 96/30 mm Hg, and respirations 24/min. Abnormal laboratory values included white blood cell count 18 000/μL, sodium 124 mEq/L, and potassium 2.5 mEq/L. Specimens for blood cultures, serum lactate level, and urinalysis with culture were obtained. A bolus of fluid was administered, and PJ was transferred to the ICU. In the ICU, a central catheter was inserted, and early goal-directed therapy was started for sepsis due to suspected community-acquired pneumonia. Within 30 minutes of ICU admission, PJ became unresponsive to verbal and noxious stimuli (score of 8 on the Glasgow Coma Scale). She was intubated, and mechanical ventilation was started.

On the day after admission, PJ's skin condition deteriorated. The nurse noted that more blisters had erupted and that the areas that originally appeared to be skin tears were now large areas of pink partial-thickness wounds on the neck, trunk, and abdomen. A wound care consultation was ordered to prescribe treatment. The wound nurse recommended petrolatum gauze applied to areas of skin loss and secured in place with tube gauze to avoid tape on the skin. The wound nurse did not know the cause of the wounds but recommended treatment on the basis of the partial-thickness wounds. The intensivist suggested that PJ might be having an adverse drug reaction because of the extension of blistering and progressive skin loss. The physician diagnosed Stevens-Johnson syndrome on the basis of the extensive, rapid epidermal loss and thought that any 1 of 3 medications—meropenem, phenytoin, and sulfamethoxazole plus trimethoprim—might have triggered the adverse drug reaction responsible for the epidermal loss. Administration of these 3 medications was discontinued, but PJ did not respond to treatment for septic shock. She was given comfort care and died 2 days later.

Ethnicity has also been identified as a risk factor.[10] The high rate of SJS associated with administration of carbamazepine and allopurinol in Asian patients may be due to HLA-B*1502.[10] Genetic testing may be prudent in Asian patients before carbamazepine therapy is started.[10]

Although as many as 100 to 200 medications may be implicated in SJS, the most frequently associated drugs include antibiotics, nonsteroidal anti-inflammatory drugs, and anticonvulsants[9] (Table 1). Because nonsteroidal anti-inflammatory drugs are over-the-counter medications, patients may not report the use of these medications as part of a health history. Nontraditional medications such as ginseng are also associated with SJS.[9] Nurses and physicians must be diligent in eliciting information from each patient about all medications the patient is taking, including over-the-counter medications and

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Table 1 Common medications associated with Stevens-Johnson syndrome

Allopurinol
Antibiotics (sulfonamides, β-lactams, tetracyclines, and quinolones)
Anticonvulsants
Antivirals
Carvedilol
Cimetidine
Clofibrate
Mefloquine (antimalarial)
Nonsteroidal anti-inflammatory drugs

dietary supplements such as herbal medications, in order to have an accurate list of medications, some of which may cause SJS.

Pathophysiology

The pathophysiology of epidermal breakdown in SJS is not completely understood, but metabolites from the medications most likely cause an immune reaction, resulting in keratinocytic apoptosis (ie, programmed cell death). Keratinocytes account for 90% of the cells in the epidermis, the outermost layer of the skin. The dermis is the layer below the epidermis. When keratinocytes die, they no longer provide adherence of the epidermis to the dermis, and the epidermal surface layers shed or slough off. The key characteristic of SJS is the separation and exfoliation of the epidermis from the dermis.[1-3]

Epidermal loss may also occur in the gastrointestinal tract, eye, genitourinary system, and tracheobronchial tree.[4,5] Loss of protective epidermal layers in the gastrointestinal tract may potentiate gastrointestinal bleeding, oral ulcerations (eg, mucositis), dysphagia, and diarrhea and prevent absorption of nutrients. Loss of epidermal layers in the tracheobronchial tree may result in interstitial edema and interference with the diffusion of oxygen and carbon dioxide, resulting in respiratory distress.[11] Separation of the epidermis in the eye causes ocular discomfort, photophobia, conjunctivitis and generalized inflammation of the eye lid and lacrimal glands, and possible loss of vision.[12]

Clinical Manifestations

Initial signs and symptoms of SJS are fever and myalgias followed by skin eruptions that blister and progress to necrosis or exfoliation of the epidermis.[1,7] The initial indications may mimic those of the flu. In patients at home, such signs and symptoms may lead to the continued use of nonsteroidal anti-inflammatory drugs, which may be the cause of the signs and symptoms and exacerbate the condition.

The descriptions of skin lesions associated with SJS include rash, macules, macules with a dark center, blisters, and erythema of the trunk or neck.[2,9] As the condition progresses, increased epidermal sloughing may result in considerable fluid loss and marked pain.[4] Ultimately, patients appear to shed layers of skin, or the skin may appear scalded, similar to the appearance of a burn.

Unlike allergic reactions to medications, which often occur within days, the initial skin eruptions in SJS occur 1 to 3 weeks after the start of therapy. Wheezing, shortness of breath, facial swelling, and itching are common in acute allergic reactions. In SJS, respiratory signs and symptoms are late indications and occur when epidermal layers in the pulmonary tree are affected (Table 2).

Severity of SJS

The terms SJS and toxic epidermal necrolysis (TEN) have been used interchangeably in the literature, but TEN is the most severe

Table 2 Comparison of drug reactions

Sign or symptom	Allergic reaction	Stevens-Johnson syndrome	Toxic epidermal necrolysis
Fever	Low grade	Possible	Yes
Skin eruption	Central rash, not painful	Macules or blisters progressing to detachment of epidermis. Rash may begin on face or trunk and spread to limbs or spread from trunk to neck and face. Macules may be painful.	Macules or blisters progressing to detachment of epidermis. Rash may begin on face or trunk and spread to limbs or spread from trunk to neck and face. Macules may be painful.
Shortness of breath, wheezing	Early	Late, due to lesions in respiratory tract	Late, due to lesions in respiratory tract
Swelling of face, tongue	Early	No	No
Runny nose, itchy eyes	Early	No	Late
Myalgia, joint pain	None	Yes	Yes

form of SJS and includes the loss of epidermal cells of the skin, mucous membranes, eye, and gastrointestinal system. In SJS, epidermal loss is 1% to 30% of the body surface area.[3] With TEN, epidermal loss is greater than 30% of the body surface area.[3,4]

Two classification systems have been described to assist with prediction of severity and mortality of patients with SJS and TEN.[3,5,6] With the Severe Cutaneous Adverse Reaction system, the type of skin lesion and the percentage of body surface epidermal loss are categorized.[5] In the Severity of Illness Score for Toxic Epidermal Necrolysis, 7 parameters are used to predict severity (Table 3). Each parameter is scored as 0 if absent and as 1 if present; the 7 scores are added for the final score. Patients with scores greater than 5 have an estimated mortality rate of 90% or greater.[3,6] Evidence to support the clinical application of the 2 classification systems is limited; however, knowledge of them may help critical care nurses recognize potential mortality, and the scores may present a clear case for immediate review of a patient's medications by a physician and the discontinuation of medications known to cause SJS.

Treatment

Treatment of SJS begins with identification and discontinuation of the medication causing the delayed hypersensitivity reaction. Nurses should elicit information from patients and patients' family members about the use of all medications—prescription, over-the-counter, and nontraditional—when obtaining a medication health history. A thorough medication history is essential because one of the challenges in SJS is determining which medication is causing the skin reaction.

Once the medication causing SJS has been discontinued, interventions are focused on supportive therapy, wound care, and and immunosuppression.[13]

Table 3 Severity of illness score for toxic epidermal necrolysis

Factor[a]
Age >40 years
Presence of malignant growth
Heart rate >120/min
Percentage of body surface epidermal detachment (>10%)
Serum glucose level (>250 mg/dL)
Urea nitrogen level (>28 mg/dL)
Bicarbonate level (<20 mEq/L)

[a] Each factor is scored as 0 or 1, and the factor scores are added for the final score (range, 0-7). Scores >5 are predictive of 90% mortality.
Conversion factors: To convert mg/dL to mmol/L, multiply by 0.0555 for glucose and by 0.357 for urea nitrogen.

Supportive Care

Supportive care is directed toward preventing infection, ensuring nutrition to promote wound healing, replacing fluid and electrolytes, controlling the environment, and providing effective pain management. If skin loss is extensive, the ambient temperature should be controlled to prevent shivering. If the room temperature cannot be controlled to optimize a patient's body temperature, a radiant warmer can be used. Placing the patient in protective isolation may be helpful to prevent transmission of hospital-acquired infections.[2] Intravenous fluid replacement should be based on vital signs suggestive of hypovolemia (tachycardia, decreased urinary output, and hypotension) and the results of serum electrolyte assays. Severe epidermal sloughing can result in marked insensible fluid loss that necessitates substantial fluid replacement.[5]

Adequate nutrition is a central element in effective wound healing. Enteral feedings should be started early after diagnosis of SJS, and a nutrition consultation should be considered. Parenteral nutrition may be necessary if the patient cannot tolerate enteral or oral feedings. Vitamin supplements such as vitamins A, B, C, D, and E and protein supplements may also be prescribed to assist with metabolic needs for tissue regeneration.[14]

Ocular complications such as conjunctivitis, conjunctival scarring, and corneal blisters and perforation occur in approximately 80% of patients with severe SJS or TEN.[12] Obtaining an ophthalmologic consultation[2,12] early in the care of the patient is essential to ensure appropriate interventions to minimize possible vision loss. Prevention of ocular complications may include taping the eyes shut, lubricating the eyes with artificial tears, or applying ointments, including corticosteroid ointment, that lubricate the eye and prevent inflammation or infection.[12]

If the patient has respiratory distress, endotracheal intubation may be necessary. Respiratory distress may be related to the primary problem, such as pneumonia requiring treatment with the medication that caused SJS, or it may be related to tracheobronchial edema or obstruction from epidermal lesions.[6,11] Interventions to prevent

pneumonia should be initiated per hospital protocols. Whether intubated or not, patients with SJS often experience oral mucosal sloughing, and good oral care that prevents irritation but enhances oral health is important. Oral care should be performed with a soft toothbrush or toothette to prevent mucosal irritation. Chlorhexidine-impregnated toothettes, suggested for patients receiving mechanical ventilation to prevent ventilator-associated pneumonia, may be contraindicated because the toothettes are often too drying for the oral mucosa.[15] An evidence-based regimen for the management of oral mucositis recommended by the Oncology Nursing Society consists of physiological saline and sodium bicarbonate oral rinses.[15]

Hygiene should be gentle, with care taken not to further denude the skin with excessive friction during bathing. The use of bath basins is discouraged because the basins may increase the risk of infection.[16] Evidence is lacking to suggest the use of specific skin emollients in the care of patients with SJS or TEN; however, good skin hygiene practices involve moisturizing the skin after bathing.

Effective management of pain is also important. Denuded, sloughing skin may cause pain, and patients should be given pain medication before wound care and bathing. Treatment of pain should be guided by local policies and physicians' orders based on patient-identified comfort levels and the use of valid pain scales. Nonsteroidal medications for relief of pain and myalgias should be avoided because of the risk that they might potentiate SJS. Opioid medications and acetaminophen-containing medications can be used without the risk of potentiating the syndrome. Around-the-clock schedules for administration of pain medications should be considered for patients with SJS to optimize the patients' comfort as wounds heal.

Physical therapy, range-of-motion exercises, and an activity treatment plan should be initiated for patients with SJS to prevent decreases in functional mobility. If a patient is not on bed rest, mobility should be encouraged. Evaluation of risk and prophylaxis for venothromboembolism should be explored to include pharmacological and nonpharmacological interventions (eg, compression stocking devices).

Wound Care

Treatment of skin lesions and epidermal loss should follow best practices to promote healing, prevent further damage of viable epidermal tissue, and prevent infection. The severity of tissue sloughing and the extensive wound care involved in the management of patients with SJS may require transfer of the patient to an ICU or a burn unit for care.[5,17]

Although no evidence-based guidelines for wound care for patients with SJS are available,[4,5] principles of wound management focus on preventing infection and further tissue damage.[5,18] Nonadhering dressings should be used for patients with SJS because the use of tape may further remove intact epidermal layers.[18,19] The goal with commonly used dressings includes protecting denuded areas, providing pain relief through the dressing covering, and minimizing disruption of healing tissues.[18-20] If petrolatum-impregnated gauze is used to cover denuded skin areas, the gauze should be changed each 12 to 24 hours to prevent drying of the wound surface. Burn dressings made of nonadherent fabric can also be used to cover the wounds. Frequent dressing changes are not recommended because they may cause unwanted debridement of tissue and may contribute to epidermal loss by harming fragile tissues.[5,18] Aggressive debridement of wounds is not recommended; however, all nonviable tissue should be gently removed during wound care treatments.[18] Papain-urea or collagenase enzymatic debridement agents can be used if wounds become necrotic.[5,18]

Treatment of partial-thickness skin loss should be based on the availability of products and the presence or absence of infection. Infection should be treated with both systemic (intravenous) and topical antibiotics. Both systemic and topical treatments should be based on the results of cultures of wound specimens. Prevention or treatment of infection may include the use of silver-impregnated, silver-gel, or silver-coated primary dressings (primary dressings come in direct contact with the wound surface).[18-20] The only contraindication to the use of silver dressings is known sensitivity to silver.[18] Saline should be avoided with the use of some silver-containing dressings because it deactivates the silver.

Biological or biosynthetic temporary skin replacements (eg, porcine xenografts) may also be used in the management of patients with SJS.[18,19] These materials provide protection and help control fluid loss through open wounds and are often left in place for several days. An outer protective antimicrobial dressing can

be applied over the temporary skin replacement and changed daily.[18,19]

Mattress surfaces for patients with surface skin loss include traditional mattress surfaces, powered pressure-redistribution surfaces, low-air-loss surfaces, and air-fluidized surfaces. Nurses should evaluate the capability of a patient's current mattress surface to contribute to healing of the skin condition. If the current surface does not optimize healing, a higher grade surface should be ordered. If high levels of moisture are preventing healing, a low-air-loss surface will aid in drying the skin surface. Air-fluidized beds may be helpful because they help keep the patient warm and minimize pressure.

Immunosuppressive Therapies

Immunosuppressive therapy has also been used in the treatment of SJS. Options may include the administration of anti-inflammatory drugs or immunosuppressants.[8-10] Currently, evidence is evolving in the management of SJS. Table 4 outlines treatment options that may be considered.

Prevention of Infection and Sepsis

The most severe complication associated with SJS is infection, which may lead to sepsis. The skin is the first line of defense against infection, and this barrier is compromised in SJS. The most frequent cause of death in patients with SJS is sepsis.[1,9] Although community-acquired pneumonia is a common cause for admission to the ICU, the antibiotic therapy used to treat the pneumonia may be the cause of SJS. Nurses should assess patients for early indications of infection and sepsis (Table 5). Changes in the assessment results should be reported to the prescribing health care provider so that treatment strategies can be implemented early. The challenge is to avoid adding medications to the patient's care regimen to treat the infection without potentiating the SJS drug-related hypersensitivity reaction.

Table 4 Medical treatment options for Stevens-Johnson syndrome (SJS)

Treatment option	Effect
Discontinue medication believed to trigger hypersensitivity reaction	
Consider:	
Corticosteroids	Decrease inflammatory reaction, but may also delay wound healing
Immunoglobulin	May limit apoptosis
Acetylcysteine	May increase drug clearance of the medications causing SJS
Plasmapheresis	May increase drug clearance of the medications causing SJS

Table 5 Signs of sepsis

Fever >101.3°F (38.5°C) or <95°F (35°C)
Heart rate >90/min
Respiratory rate >20/min
Abnormal white blood cell count (>12 000 cells/μL, <4000 cells/μL, or >10% bands
Blood pressure decreased or less than the patient's normal pressure

Conclusion

SJS and TEN present several challenges. The initial challenge is early detection of the condition. Is the development of rash in a patient due to an adverse drug reaction or allergy or to SJS? After diagnosis of SJS, nurses may be instrumental in implementing appropriate supportive care and wound care (Table 6). Evidence-based practice related to the care of patients with SJS is evolving. Further research and clinical evidence to guide optimal care are needed to create appropriate holistic treatment guidelines. Raising awareness of SJS and critical care nurses' assessment of an abnormal blistering or a rash may increase early recognition of possible SJS. Early communication of the assessment findings to the prescribing health care provider may result in the early discontinuation of medications causing SJS and avert further complications. CCN

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To learn more about skin care in the critical care setting, read "Skin Integrity in Critically Ill and Injured Children" by Schindler et al in the *American Journal of Critical Care,* 2007;16: 568-574. Available at **www.ajcconline.org**.

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Table 6 Nursing priorities for care of patients with Stevens-Johnson syndrome

Care priority	Nursing management
Recognition of the syndrome[1,2,5]	Recognize indications of Stevens-Johnson syndrome and discontinue contributing medications
Prevention of infection[2,5,9]	Follow hospital guidelines on standard precautions Provide oral care with moisturizing agents (avoid chlorhexidine)
Pain relief[5]	Provide around-the-clock pain medication Premedicate patient for position changes, activity, and dressing changes
Promotion of wound healing and prevention of further epidermal loss[18-20]	Avoid tape Avoid frequent dressing changes Use nonadherent dressings Consider the use of silver-impregnated dressings to prevent infection Consider the use of specialized burn dressings Consider mattress surfaces that prevent skin breakdown and shear
Prevention of eye complications[10,12]	Obtain ophthalmology consultation Tape eyes shut Apply lubricant ointment or artificial tears
Control of the environment[8]	Consider transfer to a burn center Ensure that environmental controls can provide a warm environment

Acknowledgments

Grateful appreciation to Mary Beth Flynn Makic, RN, PhD, CNS, CCNS, CCRN, a research nurse scientist in critical care at the University of Colorado Hospital and an assistant professor adjoint at the University of Colorado, College of Nursing, Aurora, Colorado. She provided valuable assistance in writing this article. This article reflects the opinions of the author and does not reflect the opinions or endorsement of Sutter Hospitals.

Financial Disclosures

None reported.

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Facts

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Pain relief[3]	Provide around-the-clock pain medication Premedicate patient for position changes, activity, and dressing changes
Promotion of wound healing and prevention of further epidermal loss[5-7]	Avoid tape Avoid frequent dressing changes Use nonadherent dressings Consider the use of silver-impregnated dressings to prevent infection Consider the use of specialized burn dressings Consider mattress surfaces that prevent skin breakdown and shear
Prevention of eye complications[8,9]	Obtain ophthalmology consultation Tape eyes shut Apply lubricant ointment or artificial tears
Control of the environment[10]	Consider transfer to a burn center Ensure that environmental controls can provide a warm environment

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