

DENTINO
JURNAL KEDOKTERAN GIGI
VOL X. No 2. SEPTEMBER 2025

PHYSIOLOGICAL SOLUTION (NaCl 0,9%) AS ONE OF THERAPIES IN PATIENTS WITH ORAL LESIONS ASSOCIATED WITH STEVEN-JOHNSON SYNDROME:

I Nyoman Gede Juwita Putra¹⁾, Riani Setiadhi²⁾

¹⁾Department of Oral Medicine, Faculty of Dentistry, Mahasaraswati University, Denpasar, Bali-Indonesia

²⁾Department of Oral Medicine, Faculty of Dentistry, Padjadjaran University, Bandung-Indonesia

ABSTRACT

Background. Steven-Johnson Syndrome (SJS) is a hypersensitivity reaction characterized by acute blisters and necrosis of the skin and mucous membranes, triggered by drugs. This case occurs in 1 or 2: 1000,000 cases. **Objectives.** This case report aims to describe the role of NaCl 0,9% solution as one of the therapies in patients with oral lesions associated with SJS. A 40-year-old woman was consulted by the Internal Medicine Department with complaints of pain on lips approximately 3 days before, difficulty opening mouth and bleeding lips easily. Patients consumed Carbamazepin, Amitriptiline, Paracetamol, and Cefixime. Extraoral examination showed lips with erosive lesions accompanied by blackish sanguinolenta crusting, pain, tend to bleed. Intraoral examination showed white plaque on the dorsum of the tongue. The patient was diagnosed with oral lesions associated with SJS et causa Carbamazepin, and/or Amitriptiline and/or Cefixime, and/or Paracetamol. **Treatment.** Based on the diagnosis, the pharmacological management is intravenous dexamethasone 5mg by the Dermatology and Venereology division, while Oral Medicine treatment is compressing the lips with a NaCl 0,9% solution three times a day. After the 4th day of therapy, oral lesions were repaired. NaCl 0,9% solution as moist wound healing has the principle of moisturizing by keeping the condition of the wound base moist to facilitate cell migration at the base of the wound. The moist environment allows neutrophils and macrophages to migrate better so as to provide the best environment for the healing process. **Conclusion.** NaCl 0,9% has an important role in the oral lesions healing process in SJS.

Keywords: haling lesions, NaCl 0,9%, steven-johnson syndrome.

***Correspondence:** I Nyoman Gede Juwita Putra, Faculty of Dentistry, Mahasaraswati University, Denpasar, Bali-Indonesia, Email : juwita_putra@unmas.ac.id

INTRODUCTION

Allergic reactions are immune system responses that occur when normal body tissues experience injury or damage. These reactions are characterized by the involvement of Immunoglobulin E (IgE) antibodies. In addition to IgE, allergic reactions also involve lymphocytes and other immune cells that serve as protective components of the immune system. Immunoglobulin E binds to specific cells, including basophils in the bloodstream and mast cells in tissues. When IgE bound to these cells encounters an antigen, referred to as an allergen, it triggers the release of chemical mediators capable of damaging the surrounding tissue. Allergens, such as dust particles, pollen, certain foods, and drugs, act as antigens that stimulate immune responses.¹⁻³

Stevens-Johnson Syndrome (SJS) is classified as an allergic reaction characterized by inflammatory disorders of the skin and mucous membranes, leading to immune system activation and keratinocyte death. This condition is marked by acute blister formation accompanied by necrosis.⁴⁻⁷ Fever and malaise are the initial symptoms of this disorder, which may subside or persist when mucocutaneous lesions appear.^{2,4,5} The etiology of SJS is predominantly drug-induced, with agents such as antibiotics, anticonvulsants, nonsteroidal anti-inflammatory drugs (NSAIDs), and allopurinol accounting for approximately 50–80% of cases, although infections and malignancies may also trigger this disorder.^{5,8,9}

Statistically, the incidence of SJS in the United States ranges from 2.6 to 7.1 cases per million population. SJS can occur in both males and females but is more common in women, with a male-to-female ratio of 3:2 to 2:1. It affects all age groups; however, cases progressing to Toxic Epidermal Necrolysis (TEN) are more frequently observed in individuals over 60 years of age.^{2,8-10} The management of SJS includes discontinuation of suspected causative drugs,

supportive therapy, and specific therapeutic interventions.⁸ Supportive therapy constitutes the standard care provided due to fluid and electrolyte imbalance in patients, including involvement of the oral mucosa. Wound management in SJS patients typically involves dressing with petrolatum, silver nitrate, povidone-iodine, or hydrogel, as well as the use of physiological saline solution (0.9% NaCl).^{8,11,12}

Physiological saline solution (0.9% NaCl) is an isotonic saline fluid. Its concentration closely resembles that of body fluids, thereby preventing tissue irritation. The functions of 0.9% NaCl solution include cleansing, irrigation, and serving as a compress for wound management.¹²⁻¹⁴ This case report aims to describe the role of 0.9% NaCl solution as a therapeutic modality in patients with oral lesions associated with SJS.

CASES

A 40-year-old female patient was referred from the Department of Internal Medicine with the chief complaint of pain in the upper and lower lips for approximately three days, accompanied by difficulty opening the mouth and a tendency of the lips to bleed easily. The patient had been taking Carbamazepine and Amitriptyline, prescribed by a neurologist to manage sudden, sharp, stabbing pain in the right cheek. White patches resembling aphthous ulcers initially appeared on the lips, which subsequently developed into black crusts after several days. Extraoral examination revealed conjunctival hyperemia with discharge, erythematous macules and purpura involving almost the entire face, as well as erosive lesions with black sanguinolent crusts on the upper and lower lips, which tended to bleed easily. Intraoral examination could not be optimally performed due to limited mouth opening (Figure 1).

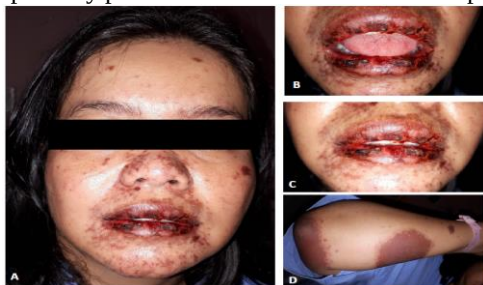


Figure 1. (A) Erythematous macules and purpura on the facial region. (B) Erosive lesions on the anterior region of the tongue. (C) Sanguinolent crusts accompanied by erosive lesions on the upper and lower lips. (D) Brownish macules on the right hand.

Laboratory examinations included a 14-parameter hematology panel, encompassing erythrocyte indices, leukocyte differential count, sodium (Na), potassium (K), and ionized calcium. Hematology results were within normal limits, except for an increase in segmented neutrophils, while band neutrophils, lymphocytes, and ionized calcium levels were decreased (Table 1).

MANAGEMENT AND TREATMENT

Based on the anamnesis, physical examination, and laboratory findings, the condition was diagnosed as oral lesions associated with Stevens-Johnson Syndrome with a suspected etiology of Carbamazepine, and/or Amitriptyline, and/or Paracetamol, and/or Cefixime, with a differential diagnosis of Erythema Multiforme (EM). The patient had previously been referred to the Department of Dermatology and Venereology and was treated with intravenous Dexamethasone 5 mg vials at a total dose of 15 mg/day. Meanwhile, management from the Department of Oral Medicine included lip compresses using gauze soaked in physiological saline solution (0.9% NaCl) for 20–30 minutes at least three times daily, as well as rinsing with 0.12% Chlorhexidine digluconate mouthwash three times daily.

On the fourth day of treatment, the sanguinolent crusts on the upper and lower lips showed significant improvement, with residual erosive lesions that were no longer painful. The patient was able to open her mouth, although not optimally. On the palate, white plaques were observed, which could be removed without leaving erythematous areas (Figure 2). Lip compresses with physiological saline solution (0.9% NaCl) and rinsing with 0.12% Chlorhexidine digluconate mouthwash three times daily were continued.

On the eighth day of treatment (Figure 3), the crusts on the upper and lower lips had further decreased, although the lips appeared relatively dry. Intraoral conditions showed significant improvement. Lip compresses with physiological saline solution (0.9% NaCl) and rinsing with 0.12% Chlorhexidine digluconate mouthrinse were continued.

Table 1. Laboratory Examination Results

Examinations	Result	Normal Value
Hematology 14 Parameters		
Hemoglobin	13.1	12.3-15.3 g/dL
Hematocrit	38.8	36.0-45.0%
Erythrocytes	4.71	4.2-5.5 juta/ μ L
Leukocytes	10.47	4.50-11.0 $\times 10^3/\mu$ L
Platelets	387	150.000-450.000/ μ L
Erythrocyte Index		
MCV	82.4	80-96 fL
MCH	27.6	27.5-33.2 pg
MCHC	33.8	33.4-35.5%
Leukocyte Type Count		
Basophils	0	0-1%
Eosinophils	0	0-4%
Rod Neutrophils	0 ^L	3-5%
Segmented Neutrophils	84 ^H	45-73%
Lymphocytes	13 ^L	18-44%
Monocytes	3	3-8%
Sodium	142	135-142mmol/L
Potassium	4.5	3.5-5.1mmol/L
Kalsium Ion	4.29 ^L	4.5-5.6mg/dL

Notes : L=low, H=high



Figure 2. (A) Reduction of erythematous macules and purpura on the face. (B) Decrease of sanguinolent crusts on the upper and lower lips. (C) Removable white plaque on the anterior tongue. (D) Removable white plaque on the hard palate.



Figure 3. (A) Disappearance of erythematous macules on the face. (B) Reduction of crusts on the lips, with dry lips observed. (C) Disappearance of white plaque on the hard palate. (D) Resolution of erosive lesions on the tongue accompanied by disappearance of white plaque.

DISCUSSION

Stevens-Johnson Syndrome (SJS) is a rare mucocutaneous disorder that can be life-threatening. It is classified as a severe cutaneous adverse reaction involving the skin and mucous membranes, including the oral mucosa.^{6,7,9-11} This condition is characterized by keratinocyte apoptosis, resulting in widespread epidermal detachment involving less than 10% of the total body surface area.^{4,15} In adults, the occurrence of SJS reaches up to 60% and is frequently associated with drug use. Relevant factors contributing to the development of this condition include medications such as sulfonamides, phenobarbital, carbamazepine, and lamotrigine, as well as other drugs including valproic acid, nonsteroidal anti-inflammatory drugs (NSAIDs), and paracetamol.^{8,10,11} In addition to medications, infections also play a role in the development of SJS, including those caused by *Mycoplasma pneumoniae*, cytomegalovirus, herpes virus, and hepatitis A. Other predisposing factors include HIV infection—which increases the risk by up to 100-fold—malignancies, autoimmune disorders such as systemic lupus erythematosus, ongoing radiotherapy, vascular abnormalities, ultraviolet exposure, genetic susceptibility, and other immunological disorders.¹⁵⁻¹⁷ In this patient, the drugs suspected as potential triggers of SJS include carbamazepine, and/or amitriptyline, and/or paracetamol, and/or cefixime.

The pathogenesis of SJS remains unclear; however, genetic predisposition and immune mediators play crucial roles in its development. Several studies suggest that a cytotoxic reaction occurs, likely mediated by cells that suppress keratinocytes, leading to massive apoptosis. This reaction involves the activation of CD4+ and CD8+ T cells, which release mediators that trigger keratinocyte apoptosis. Elevated CD4+ T cell counts have been observed in the peripheral regions of SJS patients. Cytotoxic CD8+ T cells express $\alpha\beta$ receptors and induce cell death via perforin and granzyme B pathways rather than through Fas or TRAIL mechanisms. Consequently, the interaction between drugs and proteins is processed and presented by antigen-presenting cells (APCs) to naïve T cells, leading either to a tolerant immune response or an effector response, manifested as allergic reaction symptoms.^{7,15,17}

The general management of SJS involves discontinuing the suspected causative drugs, along with curative therapy, supportive care, and specific therapeutic interventions.^{8,16,18} The therapy administered by the Department of Dermatology and Venereology consisted of intravenous Dexamethasone 5 mg vials at a total daily dose of 15 mg. Meanwhile, the management provided by the Department of Oral Medicine included lip compresses using sterile gauze soaked in physiological saline solution (0.9% NaCl) for 20–30 minutes at least three times daily, along with rinsing using 0.12% Chlorhexidine digluconate mouthrinse three times per day.

Physiological saline solution (0.9% NaCl) is an isotonic solution that is physiologically compatible and harmless to the human body.^{13,19} The 0.9% NaCl solution has a composition and concentration closely resembling that of body fluids, making it non-irritating. It functions to protect granulation tissue from drying, maintain moisture around the wound, and support the healing process of lesions.^{13,20,21} Compresses using 0.9% NaCl solution can reduce edema due to its ability to draw fluid from the lesions through the process of osmosis. Additionally, 0.9% NaCl exhibits an anti-inflammatory response that helps alleviate pain and erythema in the affected areas.²²

The mechanism of 0.9% NaCl solution in the management of lip lesions in SJS patients involves its role in moist wound healing, based on the principle of maintaining optimal moisture.^{23,24} Maintaining moisture in the lesion facilitates cellular migration within the wound and accelerates the granulation process by up to 40% compared to dry conditions.^{19,23,24} Another function of 0.9% NaCl solution is to maintain lesion surfaces in a moist state, thereby enhancing epithelial tissue development and migration. A moist environment also optimizes the activity of neutrophils, macrophages, and fibroblasts. The underlying theory of moist wound healing includes accelerating fibrinolysis, promoting angiogenesis, reducing the risk of infection, enhancing the formation of growth factors, and facilitating the proliferation of active cells.^{19,25,26} In this patient, lip compresses using sterile gauze soaked in physiological saline solution (0.9% NaCl) were applied for 20–30 minutes at least three times daily over a period of eight days. By the eighth day of treatment, the blackish sanguinolent crusts on both the upper and lower lips had completely resolved.

As a moist wound healing agent, 0.9% NaCl solution works on the principle of maintaining optimal moisture in the wound bed. By keeping the lesion environment moist, 0.9% NaCl plays an important role as one of the therapeutic modalities in promoting the healing process of oral lesions in patients with Stevens-Johnson Syndrome (SJS).

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