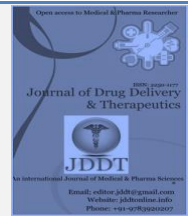


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Case Report

Management of Patients with Oral Lesions Associated Systemic Lupus Erythematosus (SLE) Nephritis: A Case Report

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Abstract

Introduction: Systemic lupus erythematosus (SLE) is a chronic autoimmune inflammatory disease involving multi-organ such as mucocutaneous, musculoskeletal, and blood vessels with varied extraoral and intraoral clinical manifestations that require treatment from many medical specialists including dentists. The purpose of writing a case report is to describe how to manage oral lesions in patients with SLE Nephritis.

Case report: A female 17 years old patient was referred to the oral medicine department of Dr. Hasan Sadikin General Hospital from the pediatrics department and was diagnosed with SLE nephritis due to complaints of ulcers on almost the entire oral mucosa. Extraoral examination revealed a moon face, malar rash, and erythematous rash on the palms of the hands. Intraoral examination revealed multiple ulcers, on labial mucosa, buccal mucosa, lateral right of the tongue, palate, and white plaque on the dorsum of the tongue. The therapy provided was non-pharmacological and pharmacological. Non-pharmacological therapies include oral hygiene instruction and communication, information, education. Pharmacological therapy is a solution containing sucralfate and prednisone which is gargled for one minute, then followed by nystatin oral suspension drug that discards after one minute, then followed by chlorhexidine digluconate 0,12% mouthwash, gargle for one minute, as an antiseptic (each drug is applied in 0,5 – 1 hours). The patient has received a systemic corticosteroid drug (methylprednisolone), but the treatment of oral lesions requires additional topical corticosteroids to accelerate the healing process because of its direct effect on the mucosa.

Conclusion: The management of oral lesions using topical corticosteroids in patients with SLE nephritis can help accelerate the healing process.

Keywords: Management, Oral Lesions, SLE nephritis

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune inflammatory disease that affects multiple organs such as mucocutaneous, musculoskeletal, and vascular, its etiology is unknown and involves connective tissue, as well as several organs such as the brain, lungs, heart, blood vessels, skin and kidneys.^{1,2} SLE is a type 3 hypersensitivity reaction in which antibody immune complexes precipitate and can cause further immune responses, and abnormal serum antibodies are a feature of the disease.² SLE can affect the organ system, especially the kidneys, so renal complications are quite often found in SLE patients and are called SLE nephritis, this can be proven histopathologically in some SLE patients with kidney biopsy and autopsy. Symptoms of SLE nephritis are proteinuria, hypertension, and renal impairment. SLE nephritis is the most serious complication and has a poor prognosis.³

The incidence rate of SLE varies between ethnic groups, geographical locations, genders, and age groups. Some studies report prevalence rates of SLE between 20-150 cases per 100,000 people in the general population, while incidence rates range from 0.9-3.1% among every 100,000 per year in Asian populations.^{2,4} Generally, SLE patients are diagnosed during adolescence, most commonly occurring in females compared to

males with a ratio of 9:1.2. The high prevalence in females may be related to hormonal and X chromosome influences. SLE mainly occurs in young women with the peak incidence rate occurring between the ages of 15 and 40 years. Childhood-onset SLE has a more aggressive disease course and has a much greater impact than adult-onset SLE.^{3,5}

The etiology of SLE is unknown, but genetic, hormonal, environmental, and immune-compromising factors have been identified as elements in the pathogenesis of SLE.⁶ Genetic factors and exposure to environmental stimuli in the form of ultraviolet light, dietary factors, certain infections and drugs, smoking, DNA demethylation, viruses, or infectious or endogenous elements similar to viruses exert an influence on the development of this disease.⁷ Specific gene products interacting with environmental stimuli are etiological factors that will result in deregulated immune responses. Various organ system damage will be caused by SLE, as a consequence of the formation and deposition of autoantibodies and immune complexes.⁸

SLE is a disease that can affect any organ system, so various clinical manifestations can be seen. One of them is lesions in the oral cavity which is the most frequent complication in SLE. The prevalence of mucosal involvement is found to be around 9-

45% of cases. According to the literature, women are more likely to have oral lesions with a female-to-male ratio of 2.7 times greater.⁶ According to the results of a study by Khatibi et al, out of 188 SLE patients, 102 (54%) patients had oral lesions.⁹ The usual oral manifestations are oral discoid lesions, honeycomb plaques, increased keratotic plaques, erythema, purpura, ptekie, irregularly shaped ulcers, and cheilitis.¹⁰

The purpose of writing this case report is to explain the role of dentists in manage the oral lesions in patients with SLE nephritis.

Case report

In this case report, a 17-year-old female was referred from the pediatric department to the oral medicine department of Hasan Sadikin Hospital, Bandung in October 2020. The patient

complained of pain especially when eating in almost the entire oral cavity four days before admission. The patient was diagnosed with SLE nephritis with hypertension and elevated liver enzyme and was admitted to the hospital to undergo chemotherapy cycle 15. On extraoral examination, the face appeared moon face, malar rash, dry lips, and ulcers. On intraoral examination, there were multiple ulcers, painful, shallow, yellowish white base, irregular shape, surrounded by erythema of varying size on the upper and lower labial mucosa, right and left buccal mucosa, palate, and floor of the mouth. On the gingiva, there was erythema on the cervicals of the maxillary and mandibular anterior teeth forming bands. On the dorsum of the tongue, there is a yellowish-white plaque that can be scraped off, leaving an erythema and sore area. (Figure 1).



Figure 1 (photo documentation of the Oral Medicine Department of Dr. Hasan Sadikin General Hospital) a. face looks moon face, b. ulcers on dry lips, c. upper and lower labials have erosive lesions and erythematous gingiva, d. ulcers on palate, e. a white plaque which when scraped leaves an erythematous area on dorsum of the tongue, f. ulcers on right lateral tongue, g. ulcers and erosive lesions on right buccal mucosa, h. ulcers and erosive lesions on left buccal mucosa.

Laboratory examination results obtained very high SGOT and SGPT values, lymphocytes, albumin, and low sodium. The management of lesions in the patient's oral cavity at the first visit was oral hygiene instruction and communication, information, education. Instructing patients to maintain oral health by brushing their teeth at least 2 times a day with a soft and fluffy toothbrush, cleaning the tongue with a gauze soaked in chlorhexidine digluconate 0.12% 3 times a day, rinsing the mouth with a mixture of prednisone and sucralfate solution for 1 minute, 2 times a day, pause 1 hour, gargle with chlorhexidine digluconate 0.12% 3 times a day, then after 1 hour of gargling, rinse with nystatin oral suspension 2 ml 4 times a day, the patient had received information about oral lesions in her mouth and understood how to use the medicine. Two weeks later, the patient came for control, complaints of pain on the

upper lip and inner cheek of the patient had decreased, but white plaque on the tongue was still present, (Figure 2). Routine use of drugs is carried out and is by the instructions given. Management at still continue, includes instructing of brushing teeth with a soft and fluffy toothbrush, scrubbing the tongue using a tongue scraper, gargling with dexamethasone twice a day, and taking folic acid. Two weeks later the patient returned for control, from the patient's history there was no pain in the patient's oral cavity, but an erosive lesion appeared on the right corner of the lip, the management at this time still continue includes instructing patients to keep brushing their teeth and cleaning their tongues, then for the lesion on the right lip corner they were given data in oral gel (Figure 3) After two weeks later the patient returned for control and the lesions in the oral cavity and lip corners had healed (Figure 4).

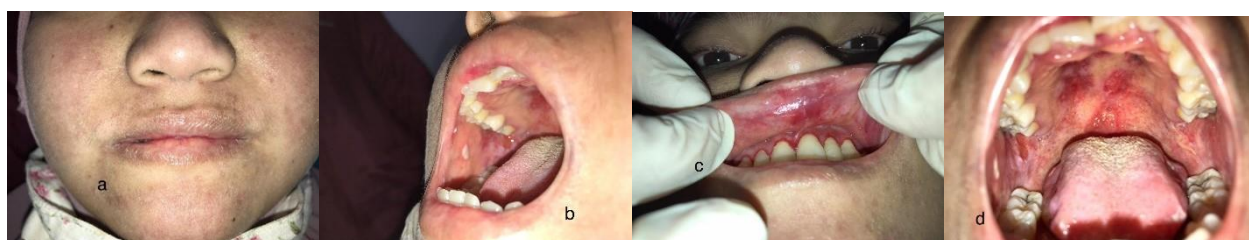


Figure 2. (photo documentation of the Oral Medicine Department of Dr. Hasan Sadikin General Hospital) a. the ulcer on the lip was healed, b. a new ulcer on the right buccal mucosa, c. an erosive lesion on the upper labial, d. an ulcer on the palate.



Figure 3 (photo documentation from the Oral Medicine Department of Dr. Hasan Sadikin General Hospital). There is cheilitis on the right lip corner.



Figure 4 (photo documentation of the Oral Medicine Department of Dr. Hasan Sadikin General Hospital). The lesion in the oral cavity is completely healed.

Discussion

Patient with SLE nephritis on intraoral examination had oral lesions in the oral cavity with the form of multiple ulcers on the right lateral tongue due to biting, on the buccal mucosa, labial mucosa, palate, and left lower lip. The ulcers that occurred in this patient, apart from being the result of her autoimmune disease, were also triggered by trauma and hormones. Traumatic ulcers in patients occur due to biting, causing wounds, and resulting in autoimmune-induced inflammation or deposition of immune complex tissues. Other factors have been implicated in the pathogenesis of SLE, but conclusive evidence is lacking. Factors involved include hormones, with an association between early menarche and SLE.¹¹ Hormonal imbalance expressed by an increased amount of salivary estrogen triggers physical changes such as increased sloughing of the oral mucosal epithelium leading to ulceration in the oral cavity.¹² In addition to multiple ulcers on the patient's tongue there is pseudomembranous candidiasis and on the right lip corner there is cheilitis angularis. This fungal infection occurs because the use of corticosteroids for a long time causes T cells to become inactive so that the direct fungal killing ability of IL17 and IL22 does not function.¹³ Extraoral examination of the patient revealed skin manifestations in the form of a reddish rash on both cheeks and palms. Oral lesions in SLE develop rapidly by 20-50% and are followed by skin manifestations, indicating a high prevalence of oral lesions, which is due to the patient's abnormal systemic condition.¹⁴ Oral ulcers were on of oral manifestations associated with SLE. The clinical features of oral lesions in SLE were described varied in different studies, including oral discoid lesions, red ulcers, chronic plaques, ulcerative plaques, lupus cheilitis, keratotic lesions, white keratotic plaques, red pebbly areas, honeycomb lesions, purpura lesions, and palatal erythema. In contrast to SLE lesions on the skin, there is no uniformity in classifying oral lesions.^{10,15}

The intraoral examination revealed palatal discoid erythematous ulcers. Oral ulcers in SLE patients have typical manifestations, namely palatal discoid erythematous.¹⁶ The

patient complained of pain in her palate, where erythematous lesions on the palate usually did not cause pain, but several studies explained that there was pain in 47% of patients, therefore a careful examination of the oral cavity in lupus patients must be carried out because there are significant asymptomatic oral lesions.^{17,18}

The management of oral lesions, in this case, was given a mixture of prednisone mixed with sucralfate given topically by gargling for 1 minute and then discarding, then a pause of 1 - 2 hours gargling with antimicrobial mouthwash for 1 minute, a pause of 1 hour followed by gargling, discarding antifungal drugs. Severity is related to the duration of corticosteroid use. Prednisone in combination with sucralfate is considered good and does not reduce the effect of prednisone.¹⁹ Sucralfate is not absorbed systemically so it is safe and does not cause negative effects.^{20,21} The aim is to combine prednisone and sucralfate so that the anti-inflammatory function on the oral mucosal tissue will stick longer due to the nature of sucralfate as a protective layer of the mucosal layer. Etiz et al's study reported positive results using sucralfate suspension in patients with stomatitis.²² Another study, Rattan et al. demonstrated the effectiveness of sucralfate suspension in the treatment of recurrent aphthous stomatitis (SAR), showing a reduction in healing period, pain duration, response time to first treatment, and duration of remission in patients taking sucralfate compared to placebo and antacids.²³ Chlorhexidine digluconate 0.12% was used as an antimicrobial in the case due to its anti-plaque role to help prevent periodontal disease, while Nystatin was used as a bactericidal measure to prevent secondary infection.^{2,24} The oral lesions gradually healed after 4 weeks of treatment, but lesions appeared on the corners of the mouth. After another 4 weeks of control, the oral lesions had completely healed. Chlorhexidine digluconate was used to disinfect the oral mucosa and clean traumatic wounds, such as on the right lateral of the patient's tongue. It is an antiseptic capable of eliminating bacteria by combining the mechanical action of an inert liquid and producing an active chemical antimicrobial effect without damaging the host tissue.²⁵

Conclusion

The management of oral manifestations in SLE nephritis is important because if oral lesions were not treated properly, they will reduced nutritional intake, leading to malnutrition and can affect the patient's systemic condition. Several case reports show the success of treatments performed by dentists for oral manifestations in SLE patients. Although the patient has received systemic corticosteroid drugs, topical administration of corticosteroids by dentists can help accelerate the healing process of oral lesions and can reduce pain in the patient's oral cavity so that the patient can eat well and peroral nutritional intake is not disturbed. ^{2,25,26,27}

Data Sharing: Statement data on the case clinical information informed consent form, and images are available for review from the corresponding author upon request.

Ethical Approval: No ethical approval is required for this case report.

Consent: Informed written consent was taken from the patient.

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Disclosure: The authors declare that there are no conflicts of interest in this work.

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