Management of denture stomatitis in removable dentures wearers

Indriani Oktaria
Department of Dentistry and Oral Diseases
Faculty of Medicine and Health Sciences, Atma Jaya Catholic University
Jakarta, Indonesia
Corresponding author: **Indriani Oktaria**, e-mail: **margaretha.indriani@atmajaya.ac.id**

ABSTRACT
Denture stomatitis (DS) is a pathological change in the oral mucosa that commonly occurs in denture users, which is characterized by an inflammatory reaction and erythema in the oral mucosa that is in contact with the denture. Clinical manifestations often include red lesions due to excessive occlusal trauma, frequently occurring in the palatal mucosa region. Loose and unstable prosthesis use can also be a predisposing factor for DS. This paper discusses cases of DS in patients with complete dentures (CD) and partial complex dentures. Chronic irritation due to heavy traumatic occlusion results in reduced stability of dentures. The fabrication of CD with proper vertical dimension setting and selection of smaller teeth elements can reduce progressive alveolar bone resorption. The use of topical gel medication like *Oxyfresh* can be an alternative in the clinical management of DS cases. Control of systemic conditions, especially in the presence of comorbidities, is crucial to minimize the risk of DS occurrence and expedite the healing process if it does occur. Maintaining oral hygiene and denture cleanliness are also key factors for the success of optimal denture care.
Keyword: denture stomatitis, occlusal trauma, denture stability, alveolar bone resorption, topical dental gel

INTRODUCTION

Oral mucosal lesions associated with the use of removable dentures are commonly occurs after insertion of dentures. The reaction can be acute or chronic reactions to microbial plaque on the denture, allergic reactions to denture base materials, or mechanical trauma from the denture. Acute reactions include traumatic ulcers, allergic reactions to denture base material, or acute infections. Chronic reactions are more common, such as denture stomatitis (DS) due to chronic infection or trauma, *angular cheilitis*, *denture irritation hyperplasia, flabby ridge*, and *oral carcinoma*. DS is one of the common issues encountered by denture wearers. In DS, pathological changes in oral mucosa occur over a period of time and are associated with the pressure exerted by the denture on the surrounding tissues. This is characterized by inflammatory reactions and erythema in the oral mucosa area, especially areas that are in contact with the denture. DS generally occurs in more than 50% of the denture-wearing population, and the incidence of DS is higher in partial denture wearers than in complete denture wearers.[1,2]

The DS is a multifactorial condition involving an interaction between local and systemic predisposing factors, without specific predilection for races and gender. The causes of DS include infection, trauma, and the host's defense response to oral cavity conditions and dentures. The immunological condition of denture wearers greatly influences the occurrence of DS. *Candida albicans* is the microorganism that causes DS. Poorly fitting dentures also increase mucosal trauma. Only a small percentage of patients complain of pain, itching, or burning sensation. Despite the high prevalence of DS, it is generally asymptomatic.[3,4]

Comprehensive and effective management of DS consists of maintaining denture hygiene, providing antifungal treatment, and correcting denture fabrication errors. To prevent or minimize the progression of lesions, patients need regular checkups with a dentist to examine the oral cavity and dentures. It is important for healthcare professionals to have knowledge of oral cavity and denture examination so that they can make accurate diagnoses and provide appropriate management.[1,5]

LITERATURE REVIEW

Denture stomatitis, also known as *chronic atrophic candidiasis* or *prosthetic stomatitis*, is characracterized by inflammation and redness of the oral mucosa beneath dentures (usually the palatal mucosa covered by an upper denture). DS is one of the common issues encountered by denture wearers and characterized by erythema, edema, often pinpoint petechiae (small red spots) on the palatal mucosa covered by the denture-bearing surface. The most common location for DS occurrence is the palatal mucosa, and it rarely occurs on the lower jaw due to protection by saliva flow. DS primarily occurs when dentures are worn throughout the night and not removed during sleep. It is more prevalent in women than in men. According to several studies in Switzerland, the prevalence of DS 11-67%.[3,4-6]

The DS is clinically classified into three types, namely type I, II, and III (Fig.1). Type I shows local-

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ized inflammation or pinpoint hyperemia. Type I lesions are caused by occlusion of the salivary ducts by dentures. Type II is characterized by diffuse erythema. Type III is marked by non-neoplastic papillary hyperplasia, with varying levels of inflammation. The papillary hyperplasia is typically localized on the central hard palate, presenting as nodular or mossy in appearance. There is an association between simple localized inflammation (DS type I) with ill-fitting dentures, as well as related with irritation and trauma. Ill-fitting dentures increases the risk of DS. The more extensive form of DS manifests as granular inflammation, which is found to be associated with poor oral hygiene and Candida infection. After one year of use, the overall occurrence of DS reaches 64%, with the severity rate of type I being the same as type II. However, patients with conventional dentures are more prone to DS due to occlusal pressure. Type II lesions are confined to the mucosa that experiences trauma from denture use, and type III lesions have characteristics almost identical to type II but with extensive granular-like lesions on the mucosa (inflammatory papillary hyperplasia).[7,8]

Figure 1 Classification of DS (Source: Budtz-Jørgensen E. Oral mucosal lesions associated with the wearing of removable dentures. J Oral Pathol 1981;10(2):65–80).

The risk factors for DS are multifactorial due to the interaction of local and systemic predisposing factors. Local factors such as local trauma from ill-fitting dentures, hyposalivation which leads to an increased attachment of *C.albicans* to the oral mucosa, and this attachment also occurs on the denture. Poor oral hygiene and wearing dentures overnight, high-carbohydrate diet, smoking, and alcohol consumption are local factors that can increase the risk of DS. In addition to local factors, conditions such as advanced age, endocrine dysfunction, nutritional deficiencies (iron, folic acid, vitamin B12), neoplastic conditions, immunocompromised and immunosuppressed states, acute leukemia, agranulocytosis, as well as antibiotic therapy are systemic factors that can increase the occurrence of DS.[6,7]

Pathogenesis

The clinical manifestation of DS includes changes in soft tissue known as flabby mucosa. *C.albicans* is the causative agent and is considered the most pathogenic and invasive species. These changes are related to its dimorphic ability, alternating between forming hyphae and yeast as a prerequisite for biofilm formation. The hyphal form is more frequently found in DS patients, and *C.albicans* triggers a faster inflammatory response, characterized by the release of antigens, toxins, and irritants from the denture plaque, leading to the occurrence of DS.[4,8]

The formation of inflammatory lesions in the oral mucosa is a complex process that follows a five-phase model proposed by Sonis et al (Fig.2). These five phases occur sequentially, starting with initiation, signaling, amplification, ulceration, and healing. Initially, tissue injury occurs due to an underlying etiology, resulting in the death of basal epithelial cells and the formation of reactive oxygen species (ROS). The initiation phase occurs after the release of molecules generated by the damage to basal epithelial cells, submucosa, and endothelium. Following initiation by ROS and the innate immune response further damage cell membranes, stimulate macrophages, and activate several pro-inflammatory transcription factors. These factors express genes that lead to spikes in pro-inflammatory cytokines such as TNF-a, IL-6, IL-1, and Cox-2, as well as the expression of other genes that cause adhesion molecule expression and angiogenesis. After the signaling phase, an amplification phase occurs to strengthen the signal. In this phase, there is an upregulation of TNF-a regulation that reinforces the inflammatory response, damaging fibronectin, leading to increased macrophage activation. The inflammatory response causes direct apoptosis of basal and submucosal epithelium and progresses to the ulceration phase in the mucosa. In the final stage, the epithelium undergoes healing through proliferation, migration, and differentiation of the epithelium stimulated by the extracellular matrix. After the healing phase, the oral mucosa returns to normal, but patients are at risk of recurrence due to residual angiogenesis.[8-10]

The histopathological changes in DS are non-specific and vary according to the severity of the lesions. In DS, there are epithelial changes such as parakeratosis, lack of keratinization, epithelial atrophy, epithelial hyperplasia, and acanthosis, as well as chronic inflammation in the lamina propria layer (Fig.3). Based on electron microscopic studies of type II and III lesions, no keratohyalin granules were found in the superficial layer, but there was an increase in intracellular space in the stratum spinosum, along with infiltration of mononuclear cells in the epithelium.[10]

Mycological and immunological studies support

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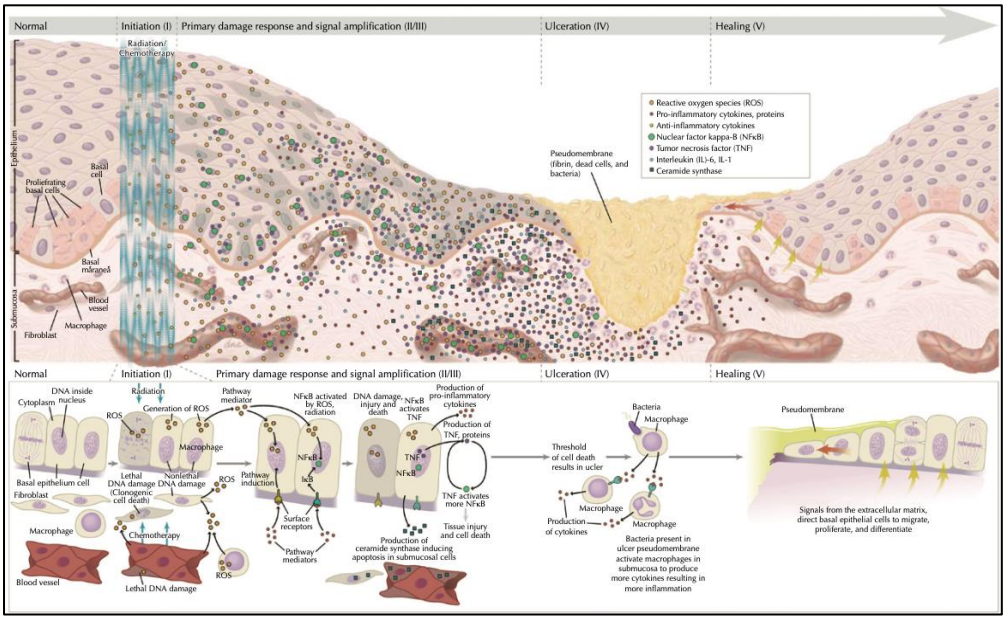
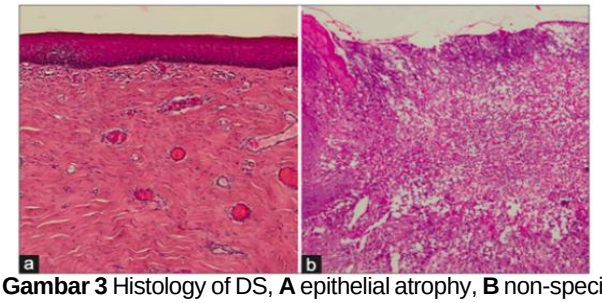


Gambar 2 Pathogenesis of the oral inflammation (Source: Glick M. Burket's oral medicine. Shelton, CT: People's Medical Publishing House; 2015).



Gambar 3 Histology of DS, **A** epithelial atrophy, **B** non-specific ulcer (Source: Budtz-Jørgensen E. Oral mucosal lesions associated with the wearing of removable dentures. J Oral Pathol 1981;10(2):65–80.

the association between infection and DS. Infection is primarily caused by the contamination of the denture surface by fungi, especially *Candida*. Poor denture hygiene is a significant risk factor for DS. On the surface of dentures with poor hygiene, there is biofilm attachment and accumulation of pathogenic plaque (Fig.4). In biofilm and plaque, there are bacteria and yeast that colonize the mucosa. Contaminants in biofilm and yeast cells also play a role in the development of DS. Studies have identified 82 bacterial phylotypes and 3 fungal species from denture swabs. Among the fungal species, *C. albicans* is the only species found in the denture biofilm of DS patients.[9,10]

Diagnosis

The diagnostic approach to DS involves taking a medical history and conducting a clinical examination (identifying signs and symptoms) with the aim of identifying the direct cause of DS (infection, trauma, or allergy), as well as predisposing factors such as oral hygiene, denture materials, denture use, and systemic diseases. The DS is most often asymptomatic or without symptoms, with only some patients complaining of pain, a burning sensation, or itching. DS is primarily diagnosed during clinical examinations as inflammation of the mucosa in contact with dentures, characterized by erythema and edema.[4,7]

Demographic factors such as age (elderly individuals), female gender, smoking, and comorbid conditions that affect the immune system could provide supportive data for the diagnosis of DS. Factors related to denture use, such as ill-fitting placements that worsen trauma and irritation of the oral mucosa, denture aging, the use of partial dentures, poor denture care and hygiene, and continuous denture wearing. Host vulnerability plays a role in DS, increasing with age and the patient's medical conditions, especially when taking certain medications as part of their treatment. Immunocompromised patients or those undergoing immunosuppressive therapy are more susceptible to *Candida* infections. One crucial aspect of DS management is oral hygiene. Over time, the denture surface deteriorates, leading to an accumulation polymicrobial complex and resulting in infections.[7,8]

Several supporting diagnostic alternatives can be conducted through mycological and immunological examinations, such as mycological examination, culture, and salivary counts.[4,9] The confirmation of a DS diagnosis related to *Candida* infection is based on the quantitative estimation of fungal growth on the oral mucosa and denture surface. Assessment can be done through culture or direct microscopy. Culture specimens and microscopic tests

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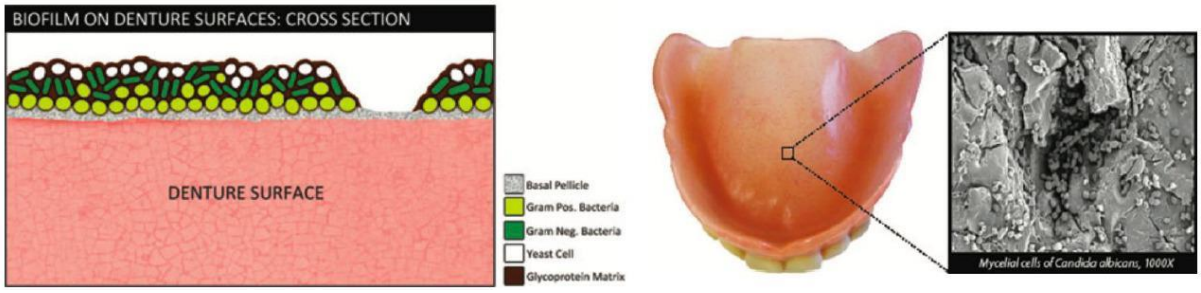

Gambar 4 Denture surface with poor hygiene (Source: Gendreau L, Loewy ZG. Epidemiology and etiology of denture stomatitis. J Am Coll Prosthodont 2011;20(4):251–60).

are taken by scraping the palatal mucosa or dentures. *Candida* infection is confirmed when dentures and mucosa are filled with fungi. Quantitative estimation through culture is obtained from a miniaturized culture test system, which serves as a cost-effective alternative to swab examination for candidiasis screening.[9]

Diagnosing DS due to allergies is established through immunological testing. Immunological testing is only relevant if infection or trauma has been ruled out, and if the clinical history and lesion appearance indicate an allergic reaction, such as a burning sensation, diffuse erythema, and edema of tissues in contact with the dentures. Positive results in delayed hypersensitivity skin tests and denture base materials indicate mechanical irritation or microbial contamination. Patients should be referred to a dermatologist for a skin test. Therefore, persistent DS may indicate systemic disease involvement and should be treated immediately.[4,8-10]

Management

The management of DS is a comprehensive approach, starting with the identification of local and systemic predisposing factors, eliminating errors in denture use, maintaining good oral hygiene and denture care, and adequate antifungal therapy. Education about dental and oral hygiene, as well as the care of dentures, is a key element in the success of DS therapy.[7-9] One study suggests that good oral hygiene and denture care, along with removing dentures before sleeping, are effective ways to manage DS. Additionally, the appropriate use of antifungal agents is also effective in most cases of DS.[10] Therefore, controlling underlying predisposing factors, maintaining oral and denture hygiene, and using appropriate antifungal treatments are considered effective for DS.[11]

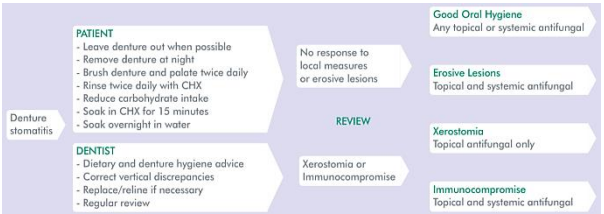

Figure 5 Protocol for the management of DS in primary care (Source: Elad S, Thierer T, Bitan M, Shapira MY, Meyerowitz C. A decision analysis: the dental management of patients prior to hematology cytotoxic therapy or hematopoietic stem cell transplantation. Oral Oncol 2008;44(1):37–42).

The maintaining oral cavity cleanliness can be achieved through dental health education (DHE), which involves brushing teeth in an up and down motion using a soft-bristle toothbrush. Avoiding local factors that trigger DS, such as smoking and wearing dentures overnight especially during sleep, is essential. According to The American College of Prosthodontists (ACP), dentures should be cleaned by brushing the prosthetic teeth outside the oral cavity at least once a day, using a specific non-abrasive toothpaste or hand soap. Ensure thorough cleanliness of the dentures, especially on surfaces that come into contact with the mucosa and gums. Afterward, dentures need to be disinfected with 0.5-2% (w/v) sodium hypochlorite solution. This solution has antimicrobial properties due to the dissociation of hydroxyl ion (OH-) and chloride ion (Cl-) in water, leading to the degradation of microbial cell walls. Disinfection with sodium hypochlorite should be done for ≤10 minutes to prevent surface damage to polymethyl methacrylate. Dentures should also be removed from the oral cavity when sleeping, should not be worn continuously 24 hours a day, and should be soaked in clean water when not in use.[10]

Improving denture fit is achieved by smoothing rough areas, and using tissue conditioner which can be applied to the denture base in areas affected by DS lesions. The objective is to reduce irritation and promote healing in the soft tissues surrounding the dentures. If there is significant contact, occlusal correction should be performed, such as selective grinding, by reshaping the occlusal surface of the teeth through smoothing to establish adequate contact between the upper and lower jaw teeth. The goal of selective grinding is to ensure that the dentures do not press on the palatal mu-

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cosa or other areas, and making the new dentures more comfortable to wear. Additionally, the addition of dental gel (Oxyfresh dental gel) can be done to expedite the healing process of mucosa affected by DS.[11]

Antifungal therapy as a treatment for DS is considered effective, when there is no improvement after eliminating predisposing factors and maintaining good oral and denture hygiene. Medication therapy is not recommended as a first-line treatment, because DS tends to recur if not accompanied by improved oral and denture hygiene. However, antifungal therapy is administered to immunocompromised patients. Commonly prescribed antifungals include *Nystatin, Miconazole, Amphotericin B, Fluconazole, Clotrimazole, Ketoconazole,* and *Chlorhexidine*. Topical antifungals are preferred over systemic because they have fewer side effects. Topical antifungals can be administered in various forms such as suspension, tablets, lozenges, creams, powders, and gels.[4,7,8] Typically, antifungal treatment is given for 14 days.

CASE

Case-1

The patient came to the dental clinic at Atma Jaya Hospital with a complaint of pain in the upper jaw gums when wearing dentures two months prior to admission. The pain was experienced after wearing dentures for two months. The pain was described as stabbing and does not radiate. The patient has not taken any medication for the current complaint. There was no noticeable swelling or redness in the gums. The patient had a history of heart disease for the past two years, but does not regularly take routine medications. General status examination showed stable hemodynamics, with blood pressure at 110/70 mmHg, heart rate 80 beats per minute, respiratory rate 20 breaths per minute, afebrile temperature, and overweight nutritional status (BMI: 23.6 kg/m2, Asia-Pacific criteria). No abnormalities are observed on extraoral examination. On intraoral examination, significant findings were found such as fissured tongue, edema and erythema on posterior mucosa of tooth 27, and gingival recession on teeth 31-33 and 41-45.

Antifungal medication	Dosage and formulation	Treatment protocol
Fluconazole	50 mg oral tablet	Once daily; 14 days
Miconazole	2% Gel applied to denture intaglio	Three times daily; 14 days
Nystatin	215,000 IU powder to denture intaglio	Three times daily; 14 days
Nystatin	1,000,000 IU mouth rinse/denture soak	Twice daily; 28 days
Amphotericin B	10 mg lozenges	Four times daily; 14 days
Itraconazole	100 mg oral capsules	Twice daily; 14 days

Figure 6 Commonly Antifungals Medication for DS.McReynolds DE, Moorthy A, Moneley JO, Jabra-Rizk MA, Sultan AS. Denture stomatitis-An interdisciplinary clinical review. J Prosthodont. 2023;32(7):560–70.

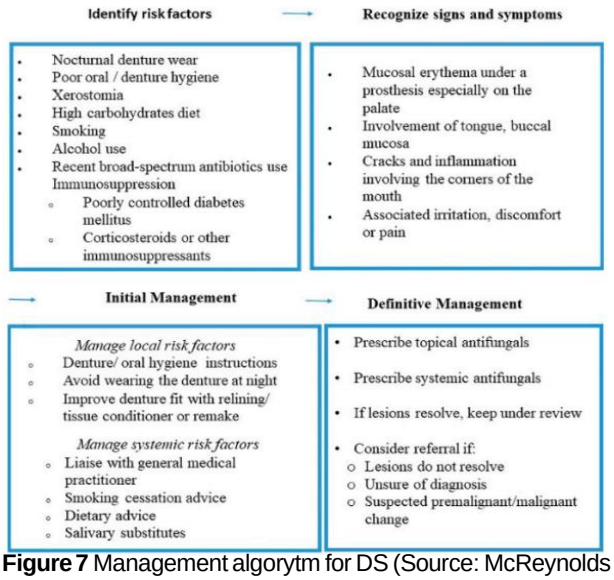

Figure 7 Management algorytm for DS (Source: McReynolds DE, Moorthy A, Moneley JO, Jabra-Rizk MA, Sultan AS. Denture stomatitis-An interdisciplinary clinical review. J Prosthodont 2023;32(7):560–70.

Figure 8 The patient's teeth in the first visit, using acrylic RPD

Figure 9 The condition of the oral cavity involves the significant loss of teeth in the upper and lower jaws

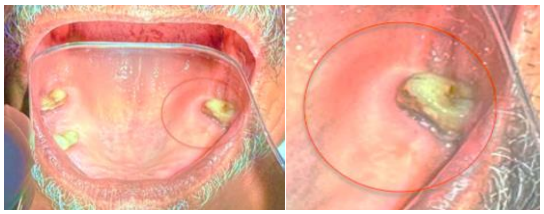

Figure 10 Clinical appearance of DS on the posterior teeth 27 of the left upper jaw.

Figure 11 Clinical appearance of mucositis around 2-3 weeks after selective grinding

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Figure 12 During follow-up

The denture appears unstable and fits loosely in the oral cavity, which could be one of the contributing factors to the occurrence of DS.

CASE II

Patient came in for a routine check-up for his dentures, which have been worn for more than 5 years. Currently, there are no complaints about the dentures, except for occasional erythema on the palate, but there was no pain. Extraoral examination appears normal with no asymmetry, and palpation around the lips and nose area is not painful and appears normal. Intraoral examination reveals poor oral hygiene, multiple missing teeth, and the presence of plaque and calculus accumulation, but without accompanying pain. There is a total tooth loss, only erythema is observed, without any pain.

Figure 13 Patient's intraoral and extraoral condition

Figure 14 Patient's old denture

Figure 15 Clinical appearance of mucositis during denture fabrication (left), after denture installation (right).

In this case, there is severe alveolar bone resorption, especially in the anterior upper jaw area, because the prosthesis is loose and heavy occlusal load. Furthermore, the patient's poor oral hygiene is evident from the dirty appearance of the old dentures and the presence of calculus on them. The clinical finding in this patient is mucosal erythema, mainly in the anterior and posterior left palatal areas. Mucosal erythema occurs due to excessive occlusal trauma and the ill-fitting old dentures, considering that some teeth have been extracted, leading to a change in denture design. This causes chronic irritation to the posterior left palatal mucosa.

MANAGEMENT

For case-I, combination therapy was carried out using topical ointments which led to better healing in addition to adjusting the rough denture surface. Case-II, on the other hand, involved the fabrication of a new denture with an extended posterior wing base area to reduce occlusal load. In addition, a tooth-arrangement technique was also performed, whereby the artificial teeth were positioned over the alveolar lingir to reduce progressive alveolar resorption.[5,7] In addition, a new denture is also recommended to improve DS.

DISCUSSION

The DS often does not cause the initial lesions to be painful as reported by patients. Some references state that DS occurs in denture wearers, especially in complete dentures, due to plaque accumulation, trauma from poorly fitting dentures, microbes on the denture surface, and poor oral hygiene. Trauma from dentures can be eliminated by observing the dentures using *pressure indicating paste* (PIP), which is indicated by areas where the PIP is removed from the dentures, signifying excessive trauma. The best management of DS involves improving the denture by smoothing the denture base surface with a stone bur and adjusting it for balanced occlusion and articulation.[2,3]

Education on denture use is highly necessary, such as maintaining denture hygiene, especially by removing them at night and cleaning them after meals, and providing denture cleaner, that are effective therapies to reduce plaque and microorganism accumulation. In addition, antifungal medications should be administered, and improvements to the dentures should be made, such as application of tissue conditioner and allowing the dentures to rest. Tissue conditioner can be applied to the denture base in areas affected by DS lesions. The use of tissue conditioner should be continued for one week and re-evaluated. Furthermore, it is necessary to observe improvements in denture occlusion and articulation. If there is excessive contact, occlusal correction and adjustments in chewing movements may be required. Selective grinding and polishing of the dentures are performed to prevent them from pressing on the palatal mucosa and other areas. If necessary, dental gel can be added

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to expedite the healing process and recovery. If left untreated, DS can cause pain and palatal inflammatory papillary hyperplasia, resulting in ill-fitting dentures in the future. Correcting loose dentures through adjustments and smoothing the denture surface can alleviate discomfort for denture wearers. Reducing the load on denture-bearing areas is also essential.[8,10]

Microwave irradiation has become a fast, economical, and effective method that works on *Candida* by exposing them to radiation using 2450 MHz waves, 3540 watts, for 8 minutes. This will change the energy, causing distortion in the dentures. Photodynamic therapy is also an alternative treatment for DS. Photosensitizing agents and exposure to specific wavelengths, along with binding to free radicals, result in cell membrane lysis and protein inactivation. Advising patients to limit unhealthy habits such as smoking and choosing a diet with restricted carbohydrates can reduce oral candidiasis.[8,9]

It is summarized that denture stomatitis is an inflammation of the oral mucosa, which contacts with dentures. The causes of DS are multifactorial, primarily due to chronic trauma from denture use, *Candida* infection, and poor oral and denture hygiene. DS is generally asymptomatic but can lead to complaints such as pain, a burning sensation, or itching. The diagnostic approach for DS involves recognizing the signs and symptoms of DS, as well as predisposing factors, to determine the direct cause of DS, which can be infection, trauma, or allergy. Diagnosis confirmation is done based on suspected causes, such as culture or microscopic examination of scrapings from the palatal mucosa or dentures, or immunological tests.[6]

Effective management of DS depends on the patient's compliance in maintaining oral cavity and denture hygiene. The DHE are the cornerstone of successful treatment of DS, involving mechanical cleaning of dentures using a soft-bristle toothbrush and non-abrasive soap or special denture paste, soaking dentures in a disinfectant solution, removing dentures from the oral cavity at night before sleep, and control to a prosthodontist about denture-related trauma and remove microbial deposits. The administration of antifungal agents improves healing in most DS cases, especially in immunocompromised patients, so DS therapy is based on the condition of intraoral lesions, oral and denture hygiene, and the patient's systemic condition.

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