

Stomatitis Venenata: A Case Report

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Citation this Article: Dr. Priyanka Mayer, Dr. Ranjani Shetty, Dr. Shivaprasad S, Dr. Shambulingappa P., Dr. Ashok L, “Stomatitis Venenata: A Case Report”, IJMSIR- July - 2020, Vol – 5, Issue - 4, P. No. 137 – 142.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Allergic contact stomatitis is a well-recognized entity, which may be easily overlooked by the clinician since its signs and symptoms are similar to various other oral lesions. The allergic contact stomatitis may present with clinical appearances that mimic classic oral vesiculobullous and ulcerative lesions. Differential diagnosis from specific mucosal diseases is important in the treatment of oral allergy. Precise history taking and the elimination of the causative agents will be necessary for an accurate diagnosis which can lead to prompt treatment bringing instant relief to the patient and also prevent further recurrences. Here, we present a case of a 37 year old male patient, who presented with multiple erythematous lesions in the oral cavity due to

change in the toothpaste, which was diagnosed and treated effectively.

Keywords: Allergic Contact Stomatitis, Oral Mucosa.

Introduction

Allergic Contact Stomatitis (ACS) is an immune-inflammatory disorder, caused by antigen- specific T cell mediated hypersensitivity immune reaction to an exogenous allergen or allergens that are in direct contact with oral mucosa.^[1]

Allergic Contact Stomatitis (Stomatitis Venenata) is a rare disorder, which most clinicians are not familiar with. A wide variety of substances are known to elicit adverse oral mucosal reactions. Flavoring agents, preservatives and dental materials are the most common causes of allergic/hypersensitivity reactions related to oral mucosa.^[2] The diagnosis of ACS must be

supported by history and clinical findings. Treatment includes the avoidance or removal of the antigens, and in persistent cases, use of topical, sublesion or systemic glucocorticosteroids.^[3,4]

Case Report

A 37 year old male patient reported to the department with a chief complaint of burning sensation in the mouth since past 1 week. The patient reported that the burning sensation was insidious in onset, gradually progressed to the current state, aggravated on eating spicy food and then relieved after sometime on drinking cold water. The Visual Analog Scale (VAS), score was found to be 6. In the history, he revealed that he had changed oral hygiene products, from Sensodyne Pro to Sensodyne Rapid relief toothpaste 10 days back. Immediately after the application of the new toothpaste, he started experiencing roughness and discomfort in the mouth for two days, after which he had burning sensation on eating normal food. Patient did not give a history of any such allergic episodes previously. On examination, there was presence of generalized erythema of the right and left buccal mucosa (Fig 1), hard palate (Fig-2) and left pterygomandibular raphae region. Also, A solitary, yellowish white pseudomembranous patch was present on the left retrocommissure area, right buccal mucosa and ventral surface of the tongue (Fig-3), irregular in shape, measuring approximately 1 x 2 cm in size, with yellowish overlying mucosa, and surrounding mucosa was erythematous. On Palpation, patch was scrapable, tender and did not bleed on provocation. Based on the above findings, a provisional diagnosis of Allergic Stomatitis Superadded with candida infection was considered.

Differential Diagnosis of Oral Lichenoid Contact Reactions and Erythema Multiforme was considered. However, because of involvement of palatal mucosa as well, which is never in contact with dental surfaces and neither was there any history of drug intake by the patient, helped us to rule out this possibility.

Erythema Multiforme is an acute, mucocutaneous condition which can manifest as a hypersensitivity reaction to infectious agents or medications. However, with no involvement of the skin, lips and labial mucosa in our case, we could rule out this condition.

Management

Patient was advised to discontinue the use of the new toothpaste. Cetirizine hydrochloride 5 mg tablet was prescribed once daily along with Chlorohexidine Mouthwash and Cool Ora Gargle (0.15% Benzylamine Hydrochloride), both to be taken undiluted in a swish and spit method 2-3 times daily. Patient was also prescribed Orasep OT ointment to be applied topically over the lesions, 3-4 times a day to alleviate the symptoms. During the follow-up visit, five days later (Fig- 4,5,6), most of the initial lesions had healed without any scarring, except for a solitary lesion was present on the left retrocommissure region which remained. These lesions were erythematous areas, 2 × 1 cm in size with whitish necrotic plaques. Patient was advised to continue the same medications for three more days, following which all the oral lesions healed completely (Fig-7 & 8).

Discussion

The oral mucosa including the lips is constantly exposed to several noxious stimuli, irritants and allergens. However, oral contact pathologies are not frequently seen because the oral mucosa acts as a protective barrier and has relative resistance to irritating agents and allergens.^[6]

Allergic contact stomatitis (ACS) is a contact allergic reaction caused by different substances, which cause inflammation of the entire oral mucosa. It can also be called as Stomatitis Venenata^[6]

Oral lesions are less common than cutaneous manifestation, even though oral cavity is exposed to these materials more commonly than skin. The reasons maybe: (a) short duration of contact (b) dilution of the offending agent by the saliva, (c) less keratinization (less Langerhans cells) of oral mucosa as compared to skin making hapten binding difficult and (d) high vascularity of oral mucosa.^[7]

ACS occurs in patients who are genetically susceptible to it, but prior exposure to a particular sensitizer is necessary to generate an antigen-specific, T lymphocyte mediated, delayed hypersensitivity immune response after primary exposure. It takes 12 to 72 hours for ACS to develop after second or subsequent contact exposure.^[11]

An International Consensus of drug allergy has classified drug hypersensitivity reactions (DHRs) clinically as immediate DHRs which typically occur within 1–6 h after the last drug administration and non-immediate DHRs which can be delayed and may occur at any time as from 1 h from the initial drug administration. DHRs can be defined as allergic and non-allergic.^[8]

Allergens which can induce T cell-mediated hypersensitivity immune reactions are usually low-molecular weight substances, and an overt ACS will develop only after repeated exposure to subthreshold concentration of the allergen, because each exposure is insufficient to generate allergic signs and symptoms. It may therefore require weeks or months of repeated exposures before the allergic reaction will occur.^[5] In a susceptible subject, stimulation of receptors, singularly

or in combination by allergens or by allergen-induced danger signals, may trigger the production of pro-inflammatory cytokines and chemokines including tumor necrosis factor- α (TNF- α) and interleukin 1 β (IL-1 β), which play essential roles in the mobilization of dedicated antigen-presenting cells to the area of the mucosa in contact with the allergen.^[9] The non-specific inflammatory signals induced by the allergen penetrating the oral mucosa are essential for initiating the delayed T cell mediated hypersensitivity immune reaction.^[10]

In addition to this, it also appears that mast cells, in a histamine-dependent manner, have the capacity to mediate early innate immune-inflammatory responses to haptens and to promote T cell differentiation, thus possibly playing a role in the pathogenesis of contact hypersensitivity.^[11]

Clinically, ACS does not have a distinct age predilection but is more common in women than in men. Such lesions may manifest as intense erythema, vesicles, erosions, ulcers, shaggy hyperkeratosis, or a combination that may extend beyond the presumed zone of contact with the allergen, and it is commonly accompanied by pain, burning sensation, or itchiness. The clinical signs and symptoms usually resolve gradually after withdrawal of the causative allergen, if this can be identified.

An allergen is a usually harmless substance capable of triggering a response that starts in the immune system and results in an allergic reaction. The most potent allergens affecting the Oral Mucosa can be classified as ^[16]

- Dental Materials- Metals and Resin based materials.

➤ Flavouring agents- Cinnamic Aldehyde, Eugenol, Balsam of Peru, Methanol Essential Oil, Peppermint Essential Oil.

➤ Preservatives- Gallates, Benzoic Acid.

The clinical appearance depends on the nature and potency of the allergen, period of the exposure, concentration of the allergen, specific variant of the T cell-mediated response generated, and indeterminable degree of dysregulation of the genetically programmed immune-inflammatory response.^[1]

Microscopically, the epithelium of the affected oral mucosa may appear acanthotic and hyperkeratotic with elongated rete ridges; the superficial lamina propria is heavily infiltrated predominantly by lymphocytes, but also by plasma cells, histiocytes, or by eosinophils; and the papillary vessels are dilated, with a perivascular lymphohistiocytic infiltrate.^[12,13]

The treatment of allergic contact stomatitis involves eliminating the allergenic agent. It may be confirmed by the reappearance of inflammatory lesions on reintroduction of the agent. Complete disappearance of the lesions can take up to 2 weeks. Antihistamines, topical anesthetics and corticosteroids are the commonly used pharmacological agents. Use of antihistamine suspensions in a swish and swallow method provide the advantage of both local and systemic action.^[2] Patients experiencing more severe symptoms may need a topical corticosteroid in the form of a mouthwash, ointment, or gel to accelerate healing.^[14,15] Symptomatic Treatment with mouthwash can be given to reduce the pain and burning sensation while eating food. Also maintenance of oral hygiene is of prime importance to facilitate healing.

References

1. Feller L, Wood NH, Khammissa AG, Lemmer J. Allergic Contact Stomatitis: A review; Oral

Surgery, Oral Medicine, Oral Radiology and Oral Pathology: 123(5): 559-565.

2. Lokesh P, Rooban T, Elizabeth J, Umadevi K, Ranganathan K. Allergic Contact Stomatitis: A Case Report and Review of Literature. Indian Journal of Clinical Practice, Vol. 22, No. 9, February 2012: 458-462.
3. Fonacier L, Bernstein DI, Pacheko K et al. Contact Dermatitis: A practice parameter- update 2015. J Allergy Clin Immunol Pract. 2015; 3; S1- S39.
4. Siddiqi A, Payne AG, De Silva RK, Duncan WJ. Titanium allergy- could it affect implant integration? Clin Oral Implants Res 2011; 22: 673-680.
5. Kaplan DH, Igyarto BZ, Gaspari AA. Early immune events in the induction of allergic contact dermatitis. Nat Rev Immunol 2012; 12: 114-124.
6. Minciullo PL, Paolino G, Vacca M, Gangemi S, Nettis E. Unmet diagnostic needs in contact oral mucosal allergies. Clin Mol Allergy. 2016 Sep 1;14(1):10.
7. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology. 2nd ed. New Delhi: Elsevier India; 2005. p. 303-4.
8. Demoly P, Adkinson NF, Brockow K, Castells M, Chiriac AM, Greenberger PA, et al. International consensus on drug allergy. Allergy 2014;69:420-37.
9. Esser PR, Wolfle U, Durr C, et al. Contact sensitizers induce skin inflammation via ROS production and hyaluronic acid degradation. PLoS One. 2012;7:e41340.
10. Peiser M. Role of Th17 cells in skin inflammation of allergic contact dermatitis. Clin Dev Immunol. 2013;2013:261037.

11. Dudeck A, Dudeck J, Scholten J, et al. Mast cells are key promoters of contact allergy that mediate the adjuvant effects of haptens. *Immunity*. 2011;34:973-984.
12. Saint-Mezard P, Rosieres A, Krasteva M, et al. Allergic contact dermatitis. *Eur J Dermatol*. 2004;14:284-295.
13. Neville BW, Damm DD. Allergies and immunologic diseases. In: Falk K, ed. *Oral and Maxillofacial Pathology*. St Louis, Missouri: Elsevier; 2016:317-326.
14. Lawrence LM, Farquharson A, Brown RS, Vatanka HO. Oral tissue irritants in toothpaste: A case report. *J Clin Pediatr Dent* 2013;38:75-8.
15. Tremblay S, Avon SL. Contact allergy to cinnamon: Case report. *J Can Dent Assoc* 2008;74:445-61.
16. Minciullo PL, Paolino G, Vacca M, Gangemi S, Nettis E. Unmet diagnostic needs in contact oral mucosal allergies. *Clin Mol Allergy*. 2016 Sep 1;14(1):10.

Legends Figure



Figure 1



Figure 2



Figure 3

1st Follow-Up After 5 Days



Figure 4



Figure 5



Figure 6

Complete Healing of Lesions after 1 Week

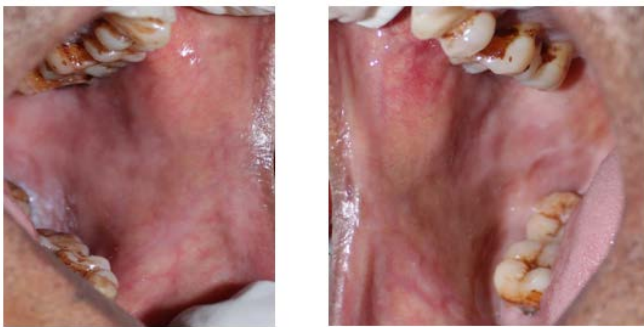


Figure 7

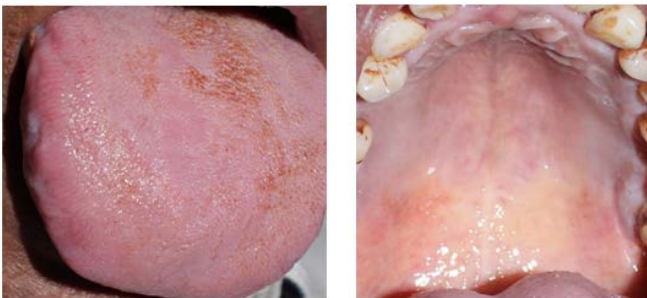


Figure 8