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CHEMICAL INJURIES OF THE ORAL CAVITY IN DENTAL PRACTICE – A CASE REPORT AND REVIEW

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ABSTRACT:. Chemical injuries to the orofacial structures occur due to varied reasons. There is not much reported cases in the literature. This paper discusses a case of aspirin burn. The treatment of aspirin burns and discussion of various types of chemical injuries to oral cavity is discussed.

KEYWORDS: Chemical injuries, Aspirin, Burns, oral cavity

INTRODUCTION

Injuries to oral soft-tissues can occur due to accidental, iatrogenic, and factitious traumas presenting as burns, ulcerations, and gingival recession.[1] Chemical, thermal, and physical agents are the main causative agents for oral soft-tissues burns. Traumatic gingival lesions, whether chemical, physical, or thermal in nature, are among the most common in the oral cavity; however there are limited reports in literature on the diagnosis and management of such injuries.

Chemical injuries of the oral soft-tissues may occur due to variety of substances such as drugs and various agents, coming in contact with the oral cavity.[2-12] The severity and extent of lesions caused by chemical agents depends on the concentration, type and quantity of the substance, as well as on the time of contact with the oral soft-tissues. Severity of the lesion is directly proportional to concentration and amount time mucosa is exposed to the chemical. [13,14] The purpose of this review is to discuss the various causative agents, manifestations and management of chemical injuries of the oral cavity.

Case Report

A 55 year old male patient presented to the Department of oral and maxillofacial surgery at our centre with a chief complaint of painful ulcerated palate on the left side. Intraoral examination revealed an ulcerated left side of palate, with raised margins and covered by slough, which was highly tender on palpation **(Fig.1).** Patient gives history of mobility of the maxillary posterior teeth associate with severe pain and local application of aspirin (acetyl salicylic acid) in the region. Irrigation with normal saline was performed to remove the slough and any remnants of the irritant. Patient was put on oral analgesics and topical anesthetic (benzocaine gel) for pain relief, extractions performed for mobile teeth under local anesthesia and had an uneventful healing.

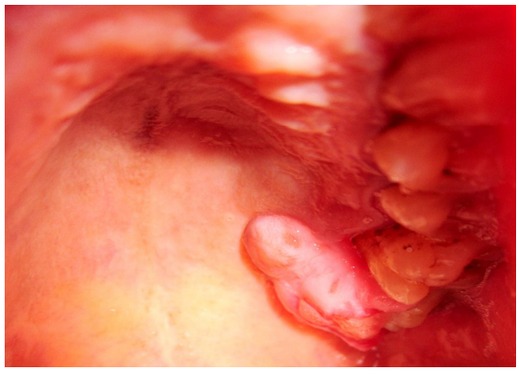

Fig.1. A patient with an Aspirin burn

Discussion

In the oral cavity, chemical substances may cause diffuse erosive lesions ranging from simple desquamation to complete obliteration of the oral mucosa with extension

past the basement membrane into the submucosa [5-9]. Acidic and alkaline substances and salts cause extensive damage to the oral mucous membranes, acting in different pathological mechanisms. Acidic substances cause a coagulation necrosis forming a scar limiting the penetration in to deeper layers. Alkaline substances cause damage by liquefaction necrosis resulting in ulcers and progressive lesions. Chemical injuries of the oral cavity can be divided as (a) non allergic reactions due to drugs and chemicals (b) allergic reactions. Locally non allergic reactions may be due to local use of aspirin, hydrogen peroxide, silver nitrate, phenol, sodium perborate, trichloroacetic acid and systemically administration of arsenic, bismuth, dilantin sodium, lead, mercury, silver, tetracycline and cancer therapeutic agents. Aspirin is used as a local obtundant giving rise to burning sensation of the mucosa, sloughing, separation of the epithelium, blanching and bleeding if area is traumatized. Aspirin burn heals on its own once the local irritating factor is removed. Hydrogen peroxide used in periodontitis and as root canal irrigant may cause epithelial necrosis when used in concentrations above 3%. Silver nitrate and phenol used as cavity sterilizing agents and also to treat apthous ulcers acts as a chemical cautery destroying the nerve endings resulting in wide spread mucosal damage and enhanced pain. Sodium perborate widely used in mouth washes and dentifrices to treat gingival disease may produce erythema of oral mucosa which may progress to sloughing of the tissue. Sodium hypochlorite used as a root canal irrigant is seen to cause non keratotic white lesions of oral mucosa when accidentally exposed. Usage of rubber dam during endodontic procedures reduces the risk of iatrogenic chemical burns.[15,16]. Calcium hydroxide causes a localized white slough, epithelial necrosis and erythema, while formocresol produces a characteristic white lesion and swelling. The white color of the lesions in chemical burns is attributed to the formation of a superficial pseudomembrane composed of a necrotic surface and inflammatory exudates. Systemically compounds like bismuth, arsenic, lead, silver and mercury used for treatment of pathological conditions and as occupational hazards cause acute or chronic poisoning both in organic and inorganic forms causing intense inflammation, increased salivation, metallic taste, ulcerative stomatitis, amalgam tattoo and pigmentation of oral mucous membranes. Several drugs including Dilantin sodium, tetracycline's and some chemotherapeutic agents used treatment of epilepsy, prophylaxis and malignancies may cause gingival hyperplasia, discoloration of teeth, mucosal ulceration and erosion. Allergic reactions of oral cavity may be (a) immediate or (b) delayed. Immediate reactions are seen due to presence of circulating antibodies in the serum e.g. angioneurotic edema, hay fever and serum sickness. Causative agents are not strictly antigenic in some cases; but however become antigenic on combination with tissues giving rise to a delayed reaction taking several hours to develop e.g. Contact allergy. Clinically immediate reactions may include swelling of the face involving lips, chin and eyes, feeling of tenseness or itching or pricking sensation, laryngeal edema, vomiting and abdominal pain. Delayed hypersensitivity reactions may manifest as dermatitis medicamentosa or Stomatitis medicamentosa, erythematic rashes, pruritis, Desquamative dermatitis, arthalgic fever, lymphadenopathy, multiple areas of erythema, areas of erosion or ulcerations and gingival ulceration mimicking acute necrotizing ulcerative gingivitis (ANUG).

CONCLUSION

The chemical burns of oral cavity can be treated with local care, including cleansing, rinsing with mouthwash and application of topical antibiotic ointment. On the initial examination care should be taken to remove any pieces of the caustic agent remaining in the mouth to prevent further burns. These burns are most often seen in small children secondary to accidental ingestion. Occasionally, mentally disturbed adults are victims. Homes with small children should either get rid of toxic chemicals or keep them locked up to prevent accidental injury. The early detection by the patient and the immediate institution of therapeutic measures will ensure a rapid cure preventing further damage to the oral soft tissues. Proper awareness, education and guidance are important prophylactic tools for preventing chemical injuries of oral cavity.

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