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ORAL ULCERS : A REVIEW

Oral Medicine and Radiology

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ABSTRACT

As the saying goes face is index of mind so this is also true with oral cavity which is mirror of general health of the individual. The most common form of manifestation of any such systemic disorders is in the form of ulcer in the mouth .Ulcers are most common in the oral region with complaints that are usually redness, burning sensation frequently accompanied by pain and discomfort. There are several local and systemic conditions which manifests in the mouth as ulcer. Some can be identified thouroughly and some present as mimicking other ulcers posing delima and challenge to the clinician leading to misdiagnosing the condition. This article is an attempt to minimise such errors paving a way for the clinician to diagnose and treat them.

KEYWORDS

Diagnose, Oral, Ulcer

INTRODUCTION:

The prevalence of oral ulcer in worldwide is 4%, with aphthous ulcers being the most common, affecting as many as 25% of the population worldwide[1].

Ulcers. A defect or break in continuity of epithelium is called ulcer[2] Common causes of oral ulcers include physical or chemical injury, infectious neoplasia, and abnormalities of the immune system, particularly autoimmune disorders. The cause of most oral ulcers may be easily determined by history and clinical presentation, and many of these lesions can be satisfactorily treated[3].

Ulcers can be divided into two groups on the basis of their onset and duration of course; acute or short-term ulcers are those that persist no longer than 3 weeks and regress spontaneously without treatment. The other group of ulcers are the chronic or persistent ulcers that last for a longer duration of weeks to months and are usually painless.

They can also be classified as single or multiple depending on their presentation. The solitary lesions may result from a trauma, infection or it could be a carcinoma and can present as a single ulcerative lesion. Multiple lesions may be seen in viral infections or autoimmune diseases and can present with several ulcerations . Recurrent ulcers may present with a history of similar episodes along with intermittent healing[4].

Clinically the parts of an ulcer are the margin (junction between normal epithelium and wall of ulcer), the floor (exposed surface of ulcer), the edge (separates the margin and floor), and the base (ulcer rest seat)[5].

The size of the ulcers can vary from a few millimeters to centimeters and occasionally may present with fever and regional lymphadenopathy [6]

The current review article is to make an attempt to identify and diagnosis different ulcers by their clinical appearance which arise due to various local & systemic conditions thus paving a way for a clinician to treat accordingly.

These are the various kinds of ulcers manifesting in oral cavity depending on the local and systemic conditions.

Table 1:

1) Malignant ulcers
   a) Carcinoma in situ
   b) Ulcerating squamous cell carcinoma
   c) Basal cell carcinoma
   d) Malignant melanoma
   e) Mucoepidermoid carcinoma
2. Ulcers due to infection and inflammation
a) Viral
   i. Intraoral herpes simplex infection
   ii. Herpes zoster
   iii. Infectious mononucleosis
   iv. Hand-foot-and-mouth disease
   v. Herpangina
   vi. Primary HIV infection
b) Bacterial
   i. Acute necrotizing ulcerative gingivitis
   ii. Noma
   iii. Syphilitic ulcers
   iv. Gonorrhoeal ulcers
   v. Tuberculous ulcers
c) Fungal
   i. Histoplasmosis
   ii. Candidiasis
3) Ulcers due to allergy and immunological dysfunction
   a. Recurrent aphthous ulcers
   b. Behcet's syndrome
   c. Reiter's syndrome
   d. Erythema multiforme and Stevens–Johnson syndrome
   e. Lupus erythematosus
   f. Mucous membrane pemp higoid
   g. Erosive lichen planus
   h. Pemphigus vulgaris
   I. Wegener's granulomatosis
   j. Drug-related ulcers
4. Ulcers caused by hormonal changes
5. Metabolism, nutrition, and storage related ulcers
   a. Langerhans cell histiocytosis
   b. Vitamin C deficiency
   c. Glycogen storage disease
6. Traumatic ulcers
7. Iatrogenic ulcers
8. Idiopathic ulcers [7]

Traumatic Ulcers

The traumatic ulcer is by far the most common oral mucosal ulcer the source of which ranges from mechanical, chemical, electrical or thermal and may be accidental, self-inflicted or iatrogenic.

Persistent ulcers not responding to local treatment might be a sign of malignant transformation of the ulcer .A cause-and-effect relationship helps in making a definitive diagnosis and in identifying the traumatizing agent. Healing usually occurs within 2 weeks of removal of the traumatic cause[4].

Traumatic ulcers on the dorsum of tongue may have close resemblance to ulcerations caused by specific infection, lymphoma, and proliferative reactive processes like lesion called Riga-Fede disease occurs in the sublingual area of infants and presence of natal or neonatal teeth as a source of trauma serves as a clue to diagnosis[6].

Traumatic ulcers mostly heal within ten days after the removal of injurious factors. A differential diagnosis is made from following factors: (i) the lesion's size, (ii) location, (iii) number, (iv) onset, (v) the age of the patient, (vi) association of other systems of the body and (vii) progression of the disease.

Regardless of the cause of traumatic ulcers, they are most often oval, shallow, and less than 1 cm in greatest diameter, A regular or irregular erythematous border encircles the yellow-white eschar covering the base of the ulcer. Generally, healing of the typical traumatic ulcer is completed in 10 to 14 days. Since traumatic ulcers are often small and minimally painful, treatment may not be required[2].

Iatrogenic Ulcers

Oral ulcers can occur due to procedure errors in dentistry from improper use of chemicals like formocresol during endodontic procedures or eugenol directly on the mucosa. These can be treated by local application of anaesthetic gel.

Idiopathic Ulcers

These are ulcers caused due to unknown reason which require proper investigations including serum and histopathological tests to arrive at specific diagnosis and treatment can be can be done according to it[8].

Ulcers due to Hormonal Changes:

Hormones induce physiological changes in almost all types of tissues in the body. Oral mucosa is like a mirror that reflects the health of an individual. Hormones play a major role in the life of a woman especially at the time of menses which evokes oral discomfort like slight burning recurrent oral ,ulcers and herpes labialis[9].

Oral ulcers seen in females during normal menstrual cycle and pregnancy occur due to the increase in salivary estrogen which causes desquamation of the oral mucosa[10].

Corticosteroids on prolonged use lower down inflammatory reaction of body tissue and become ulcerogenic[11]. These ulcers are self healing and regress after certain period of hormonal balance.

Ulcers Due To Immunological Dysfunction:

In contact stomatitis, ulcers appears due to Langerhans' cells bound to haptens in the mucosal epithelium leading to inflammatory reaction confined at the site of contact via destruction of epithelial cells resulting in ulceration[1].

Oral Allergy Syndrome (OAS) is a special contact allergy conditioned by specific amino acids in food allergens, usually fruits, and some proteins. It is usually manifested in the mouth, and is a allergic reaction mediated by IgE and IgM. symptoms include irritation or edemas of the oral mucosa, accompanied by itching and burning sensation of lips[12].

Ulcers Due To Infection And Inflammation.

Oral lesions of infectious mononucleosis include ulcers clinically similar to aphthous ulcers but are more persistent and resistant to treatment. These ulcers are painful and cause profound difficulty in eating and swallowing food[13].

The diagnosis of acute necrotizing ulcerative gingivitis (ANUG) can be made clinically identifying ulcers mostly confined to gingivae and interdental areas[14].

Some other infections like syphilis, tuberculosis, ulcers in the oral cavity simulate aphthous and traumatic ulcers , and these can be diagnosed based on the history and clinical features.[15]

Ulcers Due To Defect In Metabolism, Nutrition, And Storage:

Vitamin C

Ascorbic acid deficiency manifests as superficial lesions with bleeding gums and oral ulcers . This is due to the inadequate hydroxylation of procollagen which requires vitamin C, thus leading to decrease in antioxidant activity and aggrevation of ulcer formation in mouth[16].

Some of the glycogen storage diseases which is a rare group of genetic disorders involving defect in metabolism of glycogen also manifest as oral ulcer[7].

Ulcers Due To Virus:

The condition affected by VZV infection is acute with burning sensation and tenderness, due to appearance of clustered ulcers of 1–5mm in unilaterally on the hard palate or buccal gingivae. The ulcers tend to heal within 10–14 days. This entity is self-limiting[17].

Other virus-induced oral ulcers are seen in shingles that are caused by the reactivation of dormant varicella-zoster virus . Herpes zoster infections increases due to decrease in cell-mediated immunity [18]. These ulcers will frequently rupture resulting in the formation of crater-like ulcers and erosive areas. Shingles can mimic with the herpes simplex lesions, and can be differentiated by the distinctive pattern of the distribution of the lesion Within 10-14 days, the ulcers most heal and are self-limiting [19].

The most common lesions caused by EBV are infectious mononucleosis, nasopharyngeal carcinoma and Burkitt's lymphoma [20]. Ulcers caused by Epstein–Barr virus is typical and appears as small shallow ulcers[21].

Strains of Coxsackie A virus frequently causes hand foot and mouth disease . It is manifested as mouth ulcerations and vesicle and rashes involving the extremities [22 23]. After 1 to 2 days of infection, the oral ulcers appear and are typically restricted to the posterior part of hard and soft palate, buccal mucosa & tongue. Primary herpetic gingivostomatitis, recurrent aphthous stomatitis, erythema multiform, herpangina can be considered in the differential diagnosis of hand foot and mouth disease . It is a self-limiting and asymptomatic disease caused by coxsackie A virus.

Herpangina is typically related with soreness of throat, fever, blisters and ulcers involving the posterior part of the mouth especially palate and throat and this is differentiating feature from other lesion caused by other viral infections[24]. Acute necrotising ulcerative gingivitis identification can be done on clinical findings alone, as there are enough clinical signs to distinguish this disease from others. The most common symptoms are necrosis along with punched out ulceration, bleeding and are always limited to gingiva and interdental spaces [25]

Primary oral infection caused by Mycobacterium tuberculosis is uncommon. It characteristically presents as solitary, necrotic and ulcerative lesions with undermined edges most commonly affecting the tongue followed by gingivae, the floor of the mouth, palate, lips, and buccal mucosa[26].

Ulcers Due To Malignancy:

Malignant ulcers are usually painless early in their course due to destruction of peripheral sensory nerve endings and its invasion through penetration of connective tissue and vascular system[27] .

Ulcers in mucoepidermoid carcinoma (MEC) and SCC appear similar but can be differentiated on clinical as well as histopathological studies . If the lesion is painless, the suspicion of its being a traumatic ulcer, or a chancre, or ulcer secondary to systemic disease, or major aphthae is eliminated[28].

Ulcers Due To Systemic Disease :

Non-Hodgkin's lymphoma may manifest as a solitary area of necrotic ulcers typically affecting the gingiva,palate and fauces. This tumour often is associated with HIV disease.T-cell lymphoma tends to affect the upper anterior gingival and palate.[29]

Drug Therapy

A wide range of drugs can give rise to ulcers of the oral mucosa and the mechanisms by which drugs cause oral ulcers include drug-induced neutropenia and anaemia (e.g. cytotoxics), lichenoid disease (e.g.

sulphanylureas, b-blockers, gold, penicillamine), pemphigus (e.g. angiotensin-converting enzyme inhibitors), lupus disease and IgA dermatoses.[30]

Fungal Infections

While Candida species, usually Candida albicans, is the most common fungal infection of the mouth, this rarely gives rise to oral ulcers. Although chronic mucocutaneous candidosis (CMC) may occasionally give rise to ulcers of the dorsum of tongue. Systemic mycoses may also cause oral ulcers, typically in immunosuppressed hosts.In HIV disease, Aspergillus fumigatum may give rise to long-standing ulcers of the gingiva or oral mucosa . South American Blastomycosis may give rise to large areas of ulcers reminiscent of oral squamous cell carcinoma in both immunocompetent and immunosuppressed individuals[3132].

Uremic Stomatitis:

Uremic stomatitis is a rare oral mucosal disorder associated with a renal disorder due to increased levels of ammonia compounds and stomatitis may appear when the blood urea levels are higher than 300 mg/ml [33].

Affected tissue show ulceration with pseudomembrane formation multiple painful white keratotic lesions whereas the nonulcerative form may exhibit a erythematous patches characterized by red mucosa covered with a pseudomembrane. The tongue and the floor of the mouth are more frequently affected. Xerostomia, unriniferous breath, dysgeusia and burning sensation are common symptoms[34].

Traumatic Ulcerative Granuloma (eosinophilic Ulcer Of The Tongue)

Eosinophilic Ulcer is a chronic solitary ulcer of oral mucosa seen frequently in patients over 40 years of age but sometimes can be seen in children and young patients. The tongue is the most commonly involved site followed by the buccal mucosa, retromolar region, floor of the mouth and lips.

Similar ulcers can be seen on the ventral tongue in infants when the tongue is in frequent contact against newly erupted primary incisors, a condition known as Riga-Fede disease [35].

The tongue is also the common site of involvement in adults, which presents as an ulcer that may not be painful and persist for months anis usually accompanied by history of tauma. The ulcers are clean, punched out with surrounding erythema ranging from 0.5 cm to several centimeters in size. The surrounding tissue is usually indurated and in some of cases the lesion may also take a shape of mushroom and polyploidy mass[36].

Sustained Traumatic Ulcers (Decubitus Ulcer)

Long standing injury to the oral mucosa may lead to chronic ulcers and they are mostly seen on the tongue, lips, buccal mucosa and floor of the mouth . Traumatic ulcers heal within 7 to 10 days but some persist for weeks to months due to constant traumatic irritation [37]

Pemphigus And Pemphigoid

These lesions are a group of autoimmune, life threatening diseases that present with blisters and erosions of the skin and mucous membranes.

Pemphigus:

Pemphigus vulgaris is the most common form of pemphigus, accounting for over 80% of cases. The antibodies are targeted against DSG3 (a transmembrane glycoprotein adhesion molecule present on desmosomes). When the lesion is confined to the mucosa and targeted against DSG1 and DSG3, when there is involvement of both the skin and the mucosa.

The oral lesions may start as a bulla which breaks to form shallow ulcers. A thin layer of epithelium peels away leaving a denuded base referred as Nikolsky's sign . The lesions are mostly seen along the occlusal plane on the buccal mucosa. Palate and gingiva are also other sites of involvement[38].

These lesions can be misdiagnosed for herpes or candidiasis which can disguise the clinical appearance of pemphigus . It is also essential to distinguish these lesions from RAS. These pemphigus lesion may extend over weeks to months.

Pemphigoid:

Pemphigoid are broadly classified as mucous membrane and bullous pemphigoid. The auto antibodies are targeted against BP180 and BP230 seen at the basement membrane.

Oral lesions in bullous form occur in 30 to 50% of patients and are smaller which form slowly and are less painful than pemphigus. The gingiva is edematous, inflamed and shows desquamation with discrete vesicle formation . The lesions of Mucous membrane pemphigoid present as desquamative gingivitis and the gingiva appears bright red mimicking erosive lichen planus and pemphigus. The lesions may present as vesicles on the gingiva or other mucosal surfaces and progress slowly compared to pemphigus and are self limiting[39].

Management

Traumatic ulcers are treated initially by removing the etiological agent and observed for signs of remission. painful ulcers, the surface is treated with anaesthetic gel after meals and before bed time.

Mild cases of recurrent apthous ulcers are treated with protective emollient like Orabase, topical anesthetic gel for pain relief. In more severe cases, the use of a high potency topical steroid preparation is advised, such as hydrocortisone acetate, triamcinolone, fluocinlone, clobetasol cream beclomethasone spray placed directly on the lesion shortens healing time and reduces the size of the ulcers.

Recurrent herpetic lesions are self limiting and the use of topical antivirals reduces shedding, infectivity, pain, and the size and duration of lesions. The recommended systemic acyclovir for adults is 200mg for 5 times a day for 5 days and for children below 2 years 100 mg 5 times a day for 5 days. For children above 2 years dose similar to adults. Regarding the treatment of herpes zoster Systemic acyclovir 800 mg 5 times daily is recommended and also Prednisone 40-60 mg/daily for one week to prevent post herpetic neuralgia in old patient.(ACIVIR 200 mg, 400 mg, 500 mg DT.

In cases of fungal infections, the ulcers are treated with a combination of surgical debridement of the infected area with systemic amphotericin B. The underlying predisposing factor must be eliminated which may affect the outcome of the treatment.

Vesiculo bullous lesions like pemphigus and pemphigoid are treated with topical and systemic corticosteroids depending on the severity of symptoms or site and extent of involvement. Combinations of topical and systemic steroids are preferred for pemphigus involving only the oral mucosa. Most importantly if any ulcer is not showing signs of healing for 2 weeks need biopsy to rule out malignancy.

CONCLUSION

The present article has presented an overview of the common clinical presentations of oral ulceration. Most of these ulcers if examined properly, the dentist can be first person who can help in diagnosing patients underlying systemic condition and alerting patient to avoid further complications that can be caused by that particular disease. So skillful examination and effective treatment must be done.

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REFERENCES:

1) Patil S, Reddy SN, Maheshwari S, Khandelwal S, Shruthi D, Doni B.Prevalence of recurrent aphthous ulceration in the Indian Population. J Clin Exp Dent. 2014;6(1):e36-40
2) Greenberg MS, Glick M, Ship JA. Burket's Oral Medicine. 2008; BC Decker, Hamilton, Canada.
3) Marjorie A. Woods,Abdel R. Mohammad, James E. TurnerHarry H. Mincer. Oral ulcerations. Quintessence Int *1990;2i:14l-15L*
4) Wood NK, Goaz PW, editors. Differential Diagnosis of Oral and Maxillofacial Lesions. 5th ed. St. Louis, Missouri: Mosby; 1997
5) Das S, editor. Concise Textbook of Surgery. 3rd ed. Calcutta: Type Setters; 2001
6) Sivapathasundharam B, Sundararaman P and Kannan K. Oral Ulcers - A Review. J Dent & Oral Disord. 2018; 4(4): 1098
7) Khwaja T, Tayaar SA. Review of oral ulcers: A diagnostic dilemma. J Med Radiol Pathol Surg 2016;3:20-24.
8) Ariyawardana Anura.Traumatic Oral Mucosal Lesions: A Mini Review and Clinical Update.2014;OHDM; 13(2) :1-6
9) Usha Balan, Nitin Gonsalves, Maji Jose, KL Girish.Symptomatic Changes of Oral Mucosa during Normal Hormonal Turnover in Healthy Young Menstruating Women. Journal of Contemporary Dental Practice 2012;13(2):178-181
10) Lakshmanan Suresh, Lida Radfar.Pregnancy and lactation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97:672-82)
11) Guslandi M. Steroid ulcers: Any news? World J Gastrointest Pharmacol Ther 2013;4:39-40.
12) Ausukua M, Dublin I, Echebarria MA, Aguirre JM. Oral Allergy Syndrome (OAS). General and stomatological aspects. Med Oral Patol Oral Cir Bucal 2009;14:e568-72.
13) Luzuriaga K, Sullivan JL. Infectious mononucleosis. N Engl J Med 2010;362:1993-2000.

14) Newman MG, Takei HH, Carranza FA. Carranza's Clinical Periodontology. 9th ed. Philadelphia, PA: W. B. Saunders Company; 2002.
15) Rajendran R, Sivapathasundharam B, editors. Shafer's Textbook of Oral Pathology. 6th ed. Noida, India: Elsevier; 2009.
16) MacKay D, Miller AL. Nutritional support for wound healing. Altern Med Rev 2003;8:359-77.
17) Hamed Mortazavi,Yaser Safi, Maryam Baharvand and Somayeh Rahmani1Diagnostic Features of Common Oral Ulcerative Lesions:An Updated Decision Tree.International Journal of Dentistry 2016:1-14 https://doi.org/10.1155/2016/7278925 PMid:27781066 PMCid:PMC5066016
18) Cunningham AL, Breuer J, Dwyer DE, Gronow DW, Helme RD, Litt JC, Levin MJ, MacIntyre CR. The prevention and management of herpes zoster. Med J Aust. 2008; 188(3):171-176.
19) McCullough MJ, Savage NW. Oral viral infections and the therapeutic use of antiviral agents in dentistry. Aust Dent J. 2005; 50:S31-S35
20) Wu YM, Yan J, Ojcius DM, Chen LL, Gu ZY, Pan JP. Correlation between infections with different genotypes of human cytomegalovirus and Epstein-Barr virus in subgingival samples and periodontal status of patients. J Clin Microbiol. 2007; 45(11):3665-70.
21) Szczepański T, de Vaan GA, Beishuizen A, Bogman J, Jansen MW, van Wering ER, van Dongen JJ. Acute lymphoblastic leukemia followed by a clonally-unrelated EBV-positive non-Hodgkin lymphoma and a clonally-related myelomonocytic leukemia cutis. Pediatr Blood Cancer. 2004; 42(4):343-349.
22) Lott JP, Liu K, Landry ML, Nix WA, Oberste MS, Bolognia J, King B. Atypical hand-foot-and-mouth disease associated with coxsackievirus A6 infection. J Am Acad Dermatol. 2013; 69(5):736-741.
23) Österback R, Vuorinen T, Linna M, Susi P, Hyypiä T, Waris M. Coxsackievirus A6 and hand, foot, and mouth disease, Finland. Emerg Infect Dis. 2009; 15(9):1485-8.
24) Li W, Gao HH, Zhang Q, Liu YJ, Tao R, Cheng YP, Shu Q, Shang SQ. Large outbreak of herpangina in children caused by enterovirus in summer of 2015 in Hangzhou, China. Sci Rep. 2016; 6:35388.
25) Leão JC, Gomes VB, Porter S. Ulcerative lesions of the mouth: an update for the general medical practitioner. Clinics. 2007; 62:769-780
26) Ajay GN, Laxmikanth C, Prashanth SK. Tuberculous ulcer of tongue with oral complications of oral antituberculosis therapy. Indian J Dent. Res. 2006; 17(2):87
27) Anil Ghom,Savita Ghom;Keratotic & Non Keratotic Lesions; Textbook of Oral Medicine; Second Edition: Jaypee Brothers Medical Publishers (P) Ltd 2010;P no
28) Gnepp DR, editor. Diagnostic Surgical Pathology of the Head and Neck. 2nd ed. Philadelphia, PA: Elsevier; 2009
29) S. R. Porter* & J. C. Leao Oral Ulcers and its Relevance To Systemic Disorders Aliment Pharmacol Ther 2005; 21: 295–306.
30) Scully C, Bagan JV. Adverse drug reactions in the orofacial region. Crit Rev Oral Biol Med 2004; 15: 221–39.
31) Anil Ghom,Savita Ghom;Fungal Disease, Textbook of Oral Medicine; Second Edition: Jaypee Brothers Medical Publishers (P) Ltd 2010;P no 775-783.
32) Moraru RA, Grossman ME. Palatal necrosis in an AIDS patient: a case of mucormycosis. Cutis 2000; 66: 15–8.
33) Sudarshan R, Annigeri RG, Mamatha GP, Vijayabala GS. Uremic stomatitis. Contemporary Clinical Dentistry. 2012; 3: 113-115
34) Yuan Q, Xiong QC, Gupta M, López-Pintor RM, Chen XL, Seriwatanachai D, et al. Dental implant treatment for renal failure patients on dialysis: a clinical guideline. International Journal of Oral Science. 2017; 9: 125-132.
35) Van der Meij EH, de Vries TW, Eggink HF, de Visscher JG. Traumatic lingual ulceration in a newborn: Riga-Fede disease. Ital J Pediatr. 2012; 38: 20.
36) Chavan SS, Reddy P. Traumatic ulcerative eosinophillic granuloma with stromal eosinophilia of tongue. South Asian Journal of Cancer. 2013; 2:144.
37) Ariyawardana Anura. Traumatic Oral Mucosal Lesions: A Mini Review and Clinical Update. OHDM 2014; 13:1-6
38) Rai Arpita, Arora Monica, Naikmasur Venkatesh, Sattur Atul, Malhotra Varun. Oral Pemphigus Vulgaris: Case Report Ethiop.J Health Sci 2015;25;(4) :367-72
39) Hasan S. Desquamative gingivitis - A clinical sign in mucous membrane pemphigoid: Report of a case and review of literature. J Pharma Bioallied Sci. 2014; 6:122-126.