REVIEW ARTICLE

MOUTH AS A MIRROR OF SYSTEMIC DISEASES

Vishal Mehrotra, Parvathi Devi, Thimmarasa Venkappa Bhovi, Bhuvan Jyoti

Department of Oral Medicine & Radiology, Rama Dental College Hospital & Research Center, Kanpur, Uttar Pradesh, India

ABSTRACT

Oral clinicians are at pains to emphasize that mouth is a part of the body and not merely a gateway for delicacies. The oral cavity has an important anatomical location with a role in many critical physiologic processes, such as digestion, respiration, and speech. It is also unique for the presence of exposed hard tissue surrounded by mucosa. The oral cavity might well be thought as window to the body because oral manifestations accompany many systemic diseases. In many instances, oral involvement precedes the appearance of other symptoms or lesions at other locations. These oral manifestations must be properly recognized if the patient is to receive appropriate diagnosis and referral for treatment. This article is intended as a general overview of conditions that have oral manifestations but also involve other organ systems.

KEY WORDS: Oral cavity, Oral manifestations, Systemic diseases.

INTRODUCTION

The mouth is a mirror of health or disease, a sentinel or early warning system. As the gateway to the body, a constant barrage of invaders like bacteria, viruses, parasites, and fungi, challenges the mouth. Many systemic diseases have oral manifestations. These lesions develop on the oral mucosa, tongue, gingiva, dentition, periodontium, salivary glands, facial skeleton, extraoral skin and other related structures. These oral manifestations must be properly recognized if the patient is to receive appropriate diagnosis and referral for treatment.1 In a sizeable number of instances, the oral clues are the first and sometimes even the only evidence of a disturbed state e.g. the much described Koplik's spots in the buccal mucosa which precede the cutaneous eruption of measles. In other cases, the oral symptoms and/or signs may parallel the complaints and clues elsewhere in the body. Thus, the simultaneous development of an erosive lesion on the buccal mucosa near the angle of lips along with a butterfly rash on the face is presumptive of lupus erythematosus. Lastly, in still other situations, the oral reflections follow the evidence in other parts of the body, like in pemphigus, where the bullae may erupt on the skin days, weeks, or months before the oral ulcers can be demonstrated.2

Thus oral cavity is an important diagnostic area not just because it contains derivatives of all of the primary germinal layers, and includes tissues not demonstrable anywhere else in the body, but also because of its role played in diagnosing a number of systemic diseases just because of their oral manifestations.^{2,3}

This article is a brief overview of the oral manifestations of nutritional, hematologic, gastrointestinal, renal, cardiovascular, endocrine, metabolic and dermatologic disorders.

Nutritional Deficiencies

Due to rapid cell turnover of the oral mucosa, nutritional deficiencies may present first with oral manifestations. Changes may occur in the tongue papillae, mucosal color and integrity, and also in the oral sensations.⁴ (Table 1)

Hematological Disorders

The mouth may be the site of the earliest signs of blood dyscrasias. The manifestations may include hemorrhage, infections, and cellular infiltration of tissues. The acute leukemias tend to produce more obvious oral manifestations, characteristically the diffuse gingival hypertrophy. Gingival bleeding or accumulation of blood in tissues may occur secondary to thrombocytopenia. Pallor of the oral mucosa, loss of lingual papillae and nonspecific complaints including pain and burning sensation may occur secondary to anemia.⁵

Anaemia: Iron deficiency anemia is the most common hematologic disorder with characteristic oral manifestations including atrophic glossitis, mucosal pallor, and angular cheilitis. The oral manifestation of Plummer Vinson syndrome also includes dysphagia due to pharyngoesophageal ulcerations. Oral complications seen with sickle cell anemia include mandibular salmonella osteomyelitis that results in areas of osteoporosis and erosion followed

Table 1: Vitamin and other nutritional deficiencies and their oral manifestations.4

Vitamin & other nutritional deficiencies	Oral Manifestations
Vitamin A	Eruption rate is retarded, retarded alveolar bone formation, hyperplastic gingival epithelium followed by keratinization.
Vitamin D	Developmental anomalies of dentin and enamel, delayed eruption, and misalignment of the teeth in the jaws.
Vitamin D resistant rickets	There is often periapical involvement of grossly normal appearing deciduous or permanent teeth, followed by the development of multiple gingival fistulas. Radiographically there is abnormal alveolar bone pattern, cementum, and lamina dura around the teeth is absent or poorly defined.
Hypophosphatasia	There is loosening and premature loss of deciduous teeth, chiefly the incisors.
Vitamin K	Most common oral manifestation is gingival bleeding. Prothrombin levels below 35% result in bleeding after tooth brushing; and when below 20% result in spontaneous gingival hemorrhages.
Vitamin C deficiency (Scurvy)	There is inflammation of the interdental and marginal gingival followed by bleeding, ulceration, foul breaths due to fusospirochetal stomatitis. Hemorrhages into and swelling of the periodontal membranes occur, followed by loss of bone and loosening of the teeth, which eventually exfoliate.
Riboflavin deficiency	Initially glossitis involving the tip and/or the lateral margins of the tongue, followed later by complete atrophy of all papillae. The tongue has a magenta color. Pallor, involving oral mucosa followed by cheilosis, maceration and fissuring at the angles of the mouth.
Niacin deficiency	Oral mucosa becomes fiery red and painful. Glossitis, pain, redness and ulceration begin at the interdental papillae and spread rapidly.

by osteosclerosis. Anesthesia or paresthesia of the mandibular nerve, and asymptomatic pulpal necrosis may also occur. Associated dentofacial deformities are characterized radiographically by areas of decreased densities and coarse trabecular pattern most easily seen between the root apices of the teeth and the inferior border of the mandible.⁷

Leukemia: Oral complications of leukemia frequently include gingival hypertrophy, petechiae, ecchymosis, mucosal ulcers, and hemorrhage.⁸ Less frequently, mental nerve neuropathy, called "numb chin syndrome," may be the presenting complaint.⁹ Palatal ulcerations and necrosis may herald the presence of mucormycosis of the nasal cavity and the paranasal sinuses.¹⁰ Bacterial infections of the oral cavity, resulting in septicemia may occur. Treatment of leukemia with chemotherapeutic agents can result in reactivation of herpes simplex virus (HSV) leading to oral mucositis. The oral mucositis can also occur from chemotherapy without an HSV component, since thinning of the surface layer of mucosa and/or bone marrow sup-

pression allows for the opportunistic organisms to invade the mucosa.1

Multiple myeloma: It is a plasma cell dyscrasia in which there is overproduction of specific immunoglobulins.¹¹ When multiple myeloma involves the oral cavity, it is usually a late secondary manifestation of lesions in the jaws, most often the mandible. These lesions cause swelling of the jaws, pain, numbness, mobility of teeth, and pathological fractures.¹¹ Punched out lesions of the skull and jaw are characteristic radiographic findings.¹² Since multiple myeloma results in immunosuppression, a variety of infections may be present, including oral hairy leukoplakia and candidiasis.^{13,14} Amyloid deposits in the tongue can lead to macroglossia.¹⁵

Gastrointestinal Diseases

Crohn's disease: There is no direct time correlation between intestinal and oral lesions. Oral lesions have been documented to precede the intestinal lesions by years, and in some cases is the

only manifestation of the disease.¹ Clinically, these patients present with diffuse swelling of one or both lips with associated angular cheilitis, hyperplastic rigid mucosa and "cobble stoning" of the buccal mucosa.¹ The other manifestations include painful linear ulcerations in the buccal vestibule, painless localized swellings within the lips or face, tissue tags attached to the buccal mucosa, fissuring on the midline of the lower lip, gingival inflammation and cervical lymphadenopathy.¹

Ulcerative colitis: Ulcerative colitis has been associated with destructive oral ulcerations resulting from immune-mediated vasculitis. 18 These are similar to aphthous ulcers, but appear less frequently than lesions of Crohn's disease. Pyostomatitis vegetans characterized by multiple painless intraepithelial micro abscesses connecting in linear or serpentine tracks mostly on the labial mucosa, soft palate and ventral tongue may be seen. Pyostomatitis gangrenosum is the extreme variant with large, long-lasting and destructive ulcers, which cause significant tissue scarring. 19

Celiac disease: This is a chronic intestinal disease caused by intolerance to gluten associated with poor digestion and absorption of the majority of nutrients and vitamins, which may affect both developing dentition and oral mucosa.²⁰ Enamel hypoplasia is the most common manifestation in untreated celiac children and adolescents.21 Incisors and permanent first molars are the most affected teeth, perhaps because the odontogenesis phase coincides with the active phase of the disease.22 Hypocalcaemia during enamel formation and other systemic factors such as malnutrition and vitamin D and A deficiency may result in enamel hypoplasia.23 The main oral signs associated with celiac disease are angular cheilitis, glossitis, depapillated tongue and dry mouth.20 Ulcers are the most common type of oral lesions presenting in a papular or erosive form, generally with an erythematous margin.24

Psychiatric Disorders

Anorexia & bulimia nervosa: The dentists and dental hygienists may be the first health care providers to assess the physical and oral effects of anorexia nervosa and bulimia nervosa. The oral manifestations include dental erosion, traumatized oral mucosal membranes and pharynx, dry mouth, dental caries, periodontal disease, and soft tissue lesions. More specifically, dental erosion involves lingual erosion on the palatal surfaces of the maxillary teeth with a smooth, glossy appearance and increased tooth sensitivity. Also known as

perimylolyis, this erosion is characterized by loss of enamel with rounded margins, a notched appearance of the incisal surfaces of the anterior teeth, amalgam restorations that appear as raised islands, and loss of contours on unrestored teeth. Self-induced vomiting may cause trauma to the soft palate and pharynx. Soft tissue lesions such as angular cheilitis, candidosis, glossitis, and oral mucosal ulceration may also occur, stemming from nutritional deficiencies.²⁶

Renal Disease

Uremic stomatitis has become relatively rare, seen mostly in cases of undiagnosed and untreated chronic renal failure. Painful plagues and crusts are distributed predominantly on the buccal mucosa, dorsum of the tongue, and floor of the mouth. This occurs when the intraoral concentration of urea exceeds 30 mmol/L. Hemorrhagic diathesis from inhibited platelet aggregation may also play a role due to local hemorrhage, resulting in decreased vitality and viability of the affected tissues, thus allowing bacterial infection. There are two predominant types of uremic stomatitis. In Type I, there is a generalized or localized erythema of the oral mucosa and a thick gray pseudomembranous exudate, which does not leave a bleeding or ulcerated base when removed. Additional findings may include pain, burning, xerostomia, halitosis, gingival bleeding, dysgeusia, or candidal infection. Type II leaves ulceration if the pseudomembranous film is removed. This type may indicate a more severe form of stomatitis, secondary infections, anemia or underlying systemic hematologic disturbances caused by renal failure.27

Cardiovascular Disorders

Medications used for management of hypertension have various oral manifestations. Diuretics cause oral dryness, adrenergic inhibitors cause oral dryness and ulcerations, calcium antagonists give rise to gingival overgrowth while ACE inhibitors may cause loss of taste and lichenoid reactions of the oral mucosa.²⁸

Metabolic and Endocrine Disorders

Oral manifestations may result from abnormal hormonal regulation. Manifestations of diabetes frequently occur in the oral cavity. These may include dry mouth, burning mouth syndrome, increased reactivity to local irritation of bacteria plaque, acute gingival inflammation, multiple periodontal abscess, delayed healing and secondary infection may be present following minor trauma and oral treatments. Sex hormone imbalance can result in marked reaction to local irritations of oral tissues. Changes resemble gingivitis and periodon-

titis, with marked inflammatory reaction to bacterial plaque present in the oral cavity. Also hyperplastic tissue responses are commonly seen, resulting in soft tissue growths on the gum tissue. Hypofunction of the adrenal cortex, resulting in Addison's disease, may present in accumulation of brownish melanotic pigment in a general fashion, or as blotches in the oral soft tissue.³

Diabetes mellitus: There are many oral manifestations of diabetes mellitus, some having been described as early as 1862.1 About a third of diabetic patients complain of xerostomia, which may be due to an overall diminished flow of saliva and an increased salivary glucose level. Concomitant diffuse, nontender, bilateral enlargement of the parotid glands, called diabetic sialadenoses, may be seen in these patients. Xerostomia, results in increase susceptibility to opportunistic infections like Candida albicans. Other oral manifestation includes Erythematous candidiasis presenting as central papillary atrophy of the dorsal tongue papillae, mucormycosis, benign migratory glossitis, altered taste and burning mouth syndrome.29 The increased glucose levels in the saliva and crevicular fluid in poorly controlled diabetes result in high incidence of dental caries. Poor healing, xerostomia with subsequent increased accumulation of plaque and food debris, higher susceptibility to infections, and pronounced hyperplasia of attached gingiva all contribute to the increased incidence of periodontal disease in diabetics.30

Hypoparathyroidism: If the hypoparathyroidism develops early in life, during odontogenesis or tooth development, a pitting enamel hypoplasia and failure of tooth eruption may occur. The presence of persistent oral candidiasis in a young patient may be a signal for the onset of endocrine-candidiasis syndrome.^{31,32}

Hyperparathyroidism: Radiographically loss of the lamina dura surrounding the roots of the teeth is an early manifestation of hyperparathyroidism, with alterations in the jaw trabecular pattern characteristically developing next. There is also a decrease in trabecular density, and blurring of the normal pattern resulting in a "ground glass" appearance on the radiograph. With persistent disease, "brown tumor" of hyperparathyroidism may develop. Radiographically, these lesions are unilocular or multilocular well-demarcated radiolucencies, which commonly affect the mandible, clavicle, ribs and pelvis. They may be solitary, but more often are multiple. The long-standing lesions may produce significant cortical expansion.33

Hypercortisolism: The patient may present with a variable degree of facial hirsutism. Pathological

fractures of the mandible, maxilla or alveolar bone may occur upon low impact trauma due to osteoporosis. Healing of fractures as well as healing of alveolar bone and soft tissues after dental extractions is also delayed.³⁴

Hypoadrenocorticism: Hypoadrenocorticism results from insufficient production of adrenal corticosteroid hormones caused by destruction of the adrenal cortex, a condition known as primary hypoadrenocorticism or addison's disease. Orofacial manifestations include "bronzing" or hyperpigmentation of skin, especially predominant in sun-exposed areas and over pressure points. These skin changes are often preceded by oral mucosal melanosis. The diffuse or patchy brown macular pigmentation most commonly occurs on the buccal mucosa, but can also occur on the floor of the mouth, ventral tongue, and other areas of the oral mucosa.³⁵

Dermatologic Diseases

In number of dermatologic diseases oral manifestations appear alone or prior to the general skin changes. The environment of the oral cavity is subject to significant local irritation and thus may present the most significant signs and symptoms of the condition.

Lichen planus: The oral manifestations of lichen planus may occur weeks or months before the appearance of the skin lesions. The oral lesions are characterized by presence of striae of Wickham, and the clinical presentation may range from radiating white striae to vesiculobullous, atrophic, or erosive form.⁴

Psoriasis: Characterized by angular cheilosis, fissured tongue and benign migratory glossitis. Lesions involve the lips, buccal mucosa, palate, gingiva and floor of the mouth and appear as gray or yellowish-white plaques; as silvery white, scaly lesions with an erythematous base; as multiple papular eruptions, which may be ulcerated; or as small, papillary, elevated lesions with a scaly surface.⁴

Erythema multiforme: Oral manifestations include hyperemic macules, papules or vesicles, which may become eroded or ulcerated and bleed freely. The tongue, palate, buccal mucosa and gingiva are commonly involved.⁴

Stevens Johnsons syndrome: Oral lesions may be extremely severe and so painful that mastication is impossible. Mucosal vesicles or bullae occur which rupture and leaves surfaces covered with a thick white or yellow exudate. The lips may exhibit ulcerations with bloody crusting and are painful.⁴

Acanthosis nigricans: the tongue and lips are most commonly involved. There are papillomatous growths involving the dorsum of tongue, lips and buccal mucosa. Gingival enlargement is also seen.⁴

Pemphigus: The bullae tend to rupture as soon as they form. The oral mucosa also exhib its Nikolsky's phenomenon and may be denuded by the peripheral enlargement of the erosions. Lesions are tender, bleed easily have ragged border and be covered by a white or blood tinged exudate. Extension onto the lips with the production of crusting may occur. There may be profuse salivation, and the stench is overwhelming.⁴

Cicatricial pemphigoid: Characterized by vesiculobullous lesions, which are relatively thick-walled and, may persist for 24 to 48 hours before rupturing and desquamating. When rupture they leave a raw, eroded, bleeding surface. The gingiva appears to be erythematous for weeks or even months after the original erosions have healed.⁴ Ocular involvement is also a characteristic of Cicatricial pemphigoid.²⁸

Epidermolysis bullosa dystrophica: Characterized by oral bullae which on rupturing result in pain and scar formation resulting in obliteration of sulci and restriction of the tongue. Dental defects consist of rudimentary teeth, congenitally absent teeth, hypoplastic teeth and crowns denuded of enamel.⁴

Connective Tissue Diseases

Sjogren's syndrome: It is characterized by dry mouth (xerostomia), keratoconjunctivitis sicca, and other collagen diseases-often rheumatoid arthritis. Signs and symptoms include difficulty in chewing and mastication, altered taste sensation, difficulty with speech, mastication and denture use, an increase in incidence of dental caries, especially around the cervical region of the teeth, ¹and burning sensation of the oral mucosa. Xerostomia can be associated with fissured tongue, depapillation and redness of the tongue, cheilitis, and candidiasis. Bacterial parotitis, which is usually accompanied by fever and purulent discharge from the gland, may also occur.³6

Scleroderma: Scleroderma is a chronic disease characterized by diffuse sclerosis of the skin, gastrointestinal tract, heart muscles, lungs, and kidney. The lips of a patient with scleroderma may appear to be pursed due to constriction of the mouth aperture, thus making it difficult to open the mouth. The oral mucosa appears pale and feels rigid. The tongue can lose mobility and

become smooth in appearance as the palatal rugae flatten.³⁷ Salivary hypofunction can also be present, although usually to a lesser degree than in Sjogren's syndrome. Radiographically, the periodontal ligament space is often thickened.¹

Lupus erythematosus: The oral lesions in the discoid form begin as erythematous areas, usually depressed, and typically with white spots or radiating white striae. Occasionally, superficial, painful ulceration may occur with crusting or bleeding. The central healing may result in depressed scarring. These lesions are most common on the buccal mucosa, palate, and tongue and vermilion border of mostly the lower lip. There is severe fissuring and atrophy of lingual papilla.4 The American Rheumatism Association Committee on Diagnostic and Therapeutic Criteria has defined the oral or nasopharyngeal ulceration as a major diagnostic manifestation of SLE.38 These ulcerations are generally painless and often involve the palate. Purpuric lesions such as ecchymoses and petechiae may also occur. In up to 30% of patients with SLE, salivary gland involvement may occur concomitantly, leading to secondary Sjogren's syndrome and severe xerostomia.39

Rheumatoid arthritis: The temporomandibular joint (TJM) is often involved in rheumatoid arthritis. This is usually characterized by erosions in the condyle leading to a decreased range of motion of the mandible with pain upon movement. Oral dryness and salivary gland swelling can also be found in patients with rheumatoid arthritis. These patients can also develop secondary Sjogren's syndrome. The limited jaw function may necessitate TMJ reconstruction once the active disease is controlled.⁴⁰

CONCLUSION

Often oral manifestations are the first sign or the most significant sign of systemic disease. Dentists must acquire familiarity with systemic conditions that can affect the oral cavity, so that appropriate referral can be made. Physicians need to be aware of significance of oral complaints, their relationship to local causes, and potentially to systemic diseases. Thus mouth presents a window for easy observation of signs and symptoms of many systemic diseases because of its easy accessibility for visual investigation, and examination by palpation.

REFERENCES

 Long RG, Hlousek L, Doyle JL. Oral manifestations of systemic diseases. Mt Sinai J Med 1998; 65: 309-15.

- Cheraskin E. Oral manifestations of systemic diseases. J Natl Med Assoc 1958: 50: 241-7.
- Epstein JB. The Mouth: Window on systemic disease. Can Fam Physician1980 26: 953-7.
- Shafer's Textbook of Oral Pathology. 4thed. Oral aspects of metabolic disease. In: WB.Saunders, 1993: p. 616-72.
- Blood Dyscrasias Caused by Hypersensitivity to Drugs. Aster R.H. Pichler WJ (ed): Drug Hypersensitivity. Basel, Karger, 2007: p. 306-20.
- McFarlane DB, Pinderton PH, Dagg JH, Goldberg A. Incidence of iron deficiency, with and without anaemia, in women in general practice. Br J Haematol 1967; 13: 790-6.
- Kelleher M, Bishop K, Briggs P. Oral complications associated with sickle cell anemia: a review and case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996; 82: 225-8.
- Lynch MA, Ship II. Initial oral manifestations of leukaemia. J Am Dent Assoc 1967; 75: 932-40.
- Hiraki A, Nakamure S, Abe K, et al. Numb chin syndrome as an initial symptom of acute lymphocytic leukemia: Report of three cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997; 83: 555-61.
- Jones AC, Bentsen TY, Freedman PD. Mucormycosis of the oral cavity. Oral Surg Oral Med Oral Pathol 1993; 75: 455-60.
- Lee S, Huang J, Chan C. Gingival mass as the primary manifestation of multiple myeloma: Report of two cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996; 82: 75-9.
- Witt C, Borges AC, Klein K, Neumann H. Radiographic manifestations of multiple myeloma in the mandible: A retrospective study of 77 patients. J Oral Maxillofac Surg 1997; 55:450-3.
- Blomgren J, Back H. Oral hairy leukoplakia in a patient with multiple myeloma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996; 82: 408-10.
- 14 Perusse R. Oral candidiasis and multiple myeloma: An unusual association. Oral Surg Oral Med Oral Pathol 1994; 78: 264-6.
- Reinish El, Raviv M, Srolovitz H, Gornitsky M. Tongue, primary amyloidosis, and multiple myeloma. Oral Surg Oral Med Oral Pathol 1994; 77: 121-5.
- Plauth M, Jenss H, Meyle J. Oral manifestations of Crohn's disease. An analysis of 79 cases. J Clin Gastroenterol 1991; 13: 29-37.

- Williams AJ, Wray D, Ferguson A. The clinical entity of orofacial Crohn's disease. Q J Med 1991; 79: 451-8.
- Beitman RG, Frost SS, Roth JLA. Oral manifestations of gastrointestinal disease. Dig Dis Sci 1981; 26: 741-7.
- Tyldesley WR. Mouth lesions as markers of gastrointestinal disease. Practitioner 1983; 227: 587-90.
- Catassi C, Rätsch IM, Fabiani E, Rossini M, Bordicchia F, Candela F, et al. Coeliac disease in the year 2000: exploring the iceberg. Lancet 1994; 343: 200-3.
- Rauen MS, Back JCV, Moreira EAM. Doença celíaca: sua relação com a saúde bucal. Rev Nutr 2005; 18: 271-6.
- Aguirre JM, Rodríguez R, Oribe D, Vitoria JC. Dental enamel defects in celiac patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.1997; 84: 646-50.
- Seow WK. Enamel hypoplasia in the primary dentition: a review.ASDC J Dent Child. 1991; 58: 441-52.
- Lähteenoja H, Toivanen A, Viander M, Mäki M, Irjala K, Räihä I, et al. Oral mucosal changes in coeliac patients on a gluten-free diet. Eur J Oral Sci 1998; 106: 899-906.
- DeBate RD, Tedesco LA. Kerschbaum WE. Knowledge of Oral and Physical Manifestations of Anorexia and Bulimia Nervosa Among Dentists and Dental Hygienists. J Dent Educ 2005; 69: 346-54.
- Clarke DC. Oral complications of anorexia nervosa and/or bulimia. J Oral Med 1985; 40: 134-8.
- Ross WF, Salisbury PL. Uremic stomatitis associated with undiagnosed renal failure. Gen Dent 1994; 9/10: 410-2.
- Greenberg MS, Glick M. Burket's oral medicine, diagnosis & treatment, 10th Ed. Diseases of the cardiovascular system. In: Canada: BC Decker Inc;2006: p. 368-9.
- 29. Rees TD. The diabetic dental patient. Dent Clin North Am 1994; 38: 447-63.
- Falk H, Hugoson A, Thorstensson H. Number of teeth, prevalence of caries and periapical lesions in insulin-dependent diabetics. Scand J Dent Res 1989; 97:198-206.
- Spiegel AM. Hypoparathyroidism. In: Wyngaarden JB, Smith LH Jr, Bennett JC, editors. Cecil's Textbook of Medicine. Philadelphia: W.B. Saunders; 1992: p. 1419-20.
- Walls AWG, Soames JV. Dental manifestations of autoimmune hypoparathyroidism.
 Oral Surg Oral Med Oral Path 1993; 75: 445-52.

- 33. Hayes CW, Conway WF. Hyperparathyroidism. Radiol Clin North Am 1991; 29: 85-96.
- 34. Gabrilove JL. Cushings syndrome. Compr Ther 1992; 18: 13-6.
- Davenport J, Kellerman C, Reiss D, Harrison L. Addison's disease. Am Fam Physician 1991;
 43: 1338-42.
- Atkinson JC, Fox PC. Sjogren's syndrome: Oral and dental considerations. J Am Dent Assoc 1993; 124: 74-86.
- Nagy G, Kovacs J, Zeger M, Czirjak L. Analysis of the oral manifestations for systemic sclerosis. Oral Surg Oral Med Oral Pathol 1994; 77: 141-6.
- Cohen AS, Canoso JJ. Criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 1972; 15: 540-3.

- Grennan DM, Ferguson M, Williamson J, et al. Sjogren's syndrome in SLE. N Z Med J 1977; 86: 374-9.
- Sullivan S, Fernandez L, MacFarlane IG, et al. Impairment of lachrymal and salivary secretion and cellular immune responses to salivary antigens in rheumatoid arthritis. Ann Rheum Dis 1978; 37: 164-7.

Corresponding author:

Dr. Vishal Mehrotra A-503, Twin Towers Lakhanpur, Kanpur-208024 India

E-mail: vishal4march@rediffmail.com