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Oral Manifestations of Vitamin Deficiencies: A Periodontal Perspective and Clinician's Diagnostic Framework

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ABSTRACT

Nutritional deficiencies manifest early and characteristically in the oral cavity, with gingival and mucosal tissues serving as accessible diagnostic indicators of systemic micronutrient status. This short communication synthesises the clinically relevant oral and periodontal manifestations of key vitamin deficiencies — encompassing fat-soluble vitamins A, D, E, and K as well as the water-soluble B-complex and vitamin C — with an emphasis on radiographic correlates and a structured investigation framework applicable in periodontal practice. Fat-soluble deficiencies predominantly affect alveolar bone architecture and epithelial differentiation, while water-soluble deficiencies impair mucosal integrity, collagen synthesis, and haemostasis. A clinician-ready diagnostic table is presented to guide targeted laboratory investigation based on presenting oral signs. Early recognition of these patterns enables timely nutritional intervention and prevention of irreversible periodontal sequelae.

KEYWORDS: *gingival health; nutritional deficiency; oral manifestations; vitamin deficiency; periodontium; scurvy; glossitis*

INTRODUCTION

The oral cavity is a uniquely accessible environment where the systemic nutritional state may be assessed through mucosal appearance and radiographic bone quality.[1] The high epithelial turnover of gingival tissues renders them particularly sensitive to micronutrient depletion, often producing the earliest clinically detectable signs of systemic vitamin deficiency.[2] Despite this diagnostic value, nutritional aetiology is frequently overlooked in periodontal practice. A structured knowledge of vitamin-specific oral manifestations, paired with targeted laboratory investigation, allows the periodontist to identify and refer patients appropriately, and in certain cases, initiate life-saving nutritional therapy.

Vitamins are broadly classified into fat-soluble (A, D, E, K) — stored in the liver and adipose tissue, requiring bile salts for absorption — and water-soluble (B-complex, C) — not stored in the body and requiring daily dietary intake. Both groups carry distinct relevance to periodontal and maxillofacial health.[3] This communication presents a condensed, clinician-oriented synthesis of their oral manifestations and a diagnostic framework for use in outpatient periodontal settings.

FAT-SOLUBLE VITAMINS AND PERIODONTAL RELEVANCE

Vitamin A

Retinoic acid regulates gene expression in epithelial stem cells, preventing pathological keratinisation of non-keratinised mucosa. Deficiency causes squamous metaplasia of salivary gland ducts — replacing mucus-secreting epithelium with keratinising stratified squamous epithelium — leading to ductal obstruction, ascending sialadenitis, and xerostomia.[2] Clinically, patients present with dry, fissured tongue and increased susceptibility to cervical caries. Vitamin A and its synthetic analogues (retinoids) have been used adjunctively in the management of oral leukoplakia through induction of apoptosis in dysplastic cells and regulation of cell proliferation.[4]

Vitamin D

Calcitriol, the active form produced through sequential hepatic and renal hydroxylation of cutaneous cholecalciferol, promotes intestinal calcium absorption and skeletal mineralisation. The earliest radiographic periodontal sign of deficiency is loss of lamina dura, followed by indistinct periodontal ligament space and generalised reduction in alveolar bone density.[3] In children, rickets produces delayed tooth eruption, enamel hypoplasia, and widened pulp chambers ("thistle-tube" morphology). In adults, osteomalacia manifests as a "ground glass" radiographic pattern with loss of individual trabeculae. Renal osteodystrophy — driven by tertiary hyperparathyroidism — can produce brown tumours in the jaws, mimicking central giant cell granuloma.

Vitamins E and K

Vitamin E (alpha-tocopherol) protects cellular lipid membranes from peroxidation and has demonstrated utility in reducing oral mucositis during head-and-neck radiation therapy.[5] Vitamin K is indispensable for hepatic synthesis of clotting factors II, VII, IX, and X. Deficiency presents with spontaneous gingival bleeding, palatal petechiae and ecchymoses, and haemorrhagic bullae. Prothrombin time and INR must be assessed prior to any periodontal surgical procedure in patients with suspected deficiency.[3]

WATER-SOLUBLE VITAMINS AND PERIODONTAL RELEVANCE

B-Complex Vitamins

The B vitamins contribute to mucosal integrity, neural health, and haematopoiesis through distinct but complementary mechanisms. Vitamin B1 (thiamine) deficiency produces glossodynia and oral mucosal hyperaesthesia, frequently encountered in patients with chronic alcoholism or Wernicke-Korsakoff syndrome.[2] Vitamin B2 (riboflavin) deficiency classically presents as angular cheilitis, magenta tongue, and mucosal oedema — fissuring at the commissures is a reliable clinical sign. Vitamin B3 (niacin) deficiency (pellagra) produces the "4 D's" — dermatitis, diarrhoea, dementia, and death — with oral manifestations including scarlet glossitis that must be distinguished from erythematous candidiasis, atrophic lichen planus, and iron-deficiency anaemia (Plummer–Vinson syndrome).[4]

Vitamin B6 (pyridoxine) participates in haem synthesis and amino acid metabolism; deficiency produces cheilosis, glossitis, and peripheral neuropathy affecting the trigeminal distribution. Folate (B9) is critical for epithelial cell regeneration; its deficiency produces

recurrent aphthous stomatitis and atrophic glossitis indistinguishable from B12 deficiency, and periconceptional deficiency is strongly implicated in orofacial clefting.[5] Vitamin B12 deficiency, through failure of intrinsic factor-mediated absorption, produces megaloblastic anaemia and Hunter's glossitis — complete atrophy of filiform and fungiform papillae yielding a smooth, erythematous tongue. Vitamins B12, B6, and folate are standard first-line investigations in Burning Mouth Syndrome; supplementation resolves symptoms even at borderline serum levels. Laboratory confirmation requires serum B12 (<200 pg/mL), serum methylmalonic acid (most sensitive early marker), serum homocysteine, and anti-intrinsic factor antibodies.[6]

Vitamin C

Ascorbic acid is obligatory for hydroxylation of proline and lysine during collagen biosynthesis; its deficiency (scurvy) disrupts periodontal ligament integrity, alveolar bone mineralisation, and capillary competence.[7] Scorbutic gingivitis is characterised by spongy, oedematous gingiva with bluish-red discolouration and massive haemorrhage on minimal provocation. Radiographically, generalised rarefaction of alveolar bone and impaired socket healing are observed. The recognised skeletal signs — Wimberger ring and Fraenkel's white line — may also be sought on jaw radiographs. Management combines oral prophylaxis, scaling, and ascorbic acid supplementation; clinical and radiographic resolution confirms the nutritional diagnosis.

DIAGNOSTIC FRAMEWORK FOR CLINICAL USE

When clinical suspicion is high but serum assays are borderline, a supervised therapeutic trial of the suspected vitamin is both diagnostic and curative — rapid resolution of oral pain confirms the nutritional aetiology.[4] Table 1 summarises a structured investigation protocol based on presenting oral sign.

Table 1: Periodontal oral signs and targeted laboratory investigation

Oral Sign / Presentation	Suspected Deficiency	First-line Laboratory Investigation
Angular cheilitis; magenta / fissured tongue	B2, B6, Iron	Serum riboflavin, pyridoxine; CBC with differential
Red, smooth (bald) tongue; burning mouth	B12, Folate (B9), B3	Serum B12, folate; MMA; homocysteine; Hb; peripheral smear
Spontaneous gingival bleeding; petechiae; haemorrhagic bullae	Vitamin C, Vitamin K	Serum ascorbic acid; PT / INR; platelet count
Recurrent aphthous stomatitis; atrophic glossitis	B12, B9, Iron	Serum B12, folate; CBC; anti-intrinsic factor antibodies
Dry, fissured tongue; xerostomia	Vitamin A	Serum retinol; liver function tests

Loss of lamina dura; reduced alveolar bone density	Vitamin D	Serum 25(OH)D; serum calcium, phosphorus; PTH; ALP
Desquamative / scorbutic gingivitis with peeling	Vitamin C ± B-complex	Serum ascorbic acid; serum B-complex; CBC

ALP: alkaline phosphatase; CBC: complete blood count; MMA: methylmalonic acid; PTH: parathyroid hormone; PT: prothrombin time; INR: international normalised ratio.

CONCLUSION

Vitamins are indispensable for periodontal and maxillofacial health. The gingiva and oral mucosa frequently display the earliest signs of systemic nutritional deficiency, and the periodontist is uniquely placed to detect, investigate, and initiate management of these conditions. Integrating the diagnostic framework presented here into routine periodontal assessment — correlating mucosal signs with targeted laboratory investigation and, where appropriate, supervised therapeutic trials — enables timely nutritional intervention and improved patient outcomes. Awareness of hyperavitaminosis is equally important: excess vitamin A causes cortical bone thickening and mucosal desquamation, while vitamin D toxicity produces hypercalcaemia and metastatic pulp calcifications.[3] A balanced, evidence-informed approach to nutritional assessment should form part of every comprehensive periodontal workup.

DECLARATIONS

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