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Review Article

Common Causes of Local Halitosis: An Overview for Dental Clinicians

Dr. T. Maheswaran[1], Dr. G. Pamalai[2], Dr. K. Sivaguru[3]*, Dr. B. Roshan Arbaaz[4], Dr. P. Velmurugan[5], Dr. B. Adhithya[6]

[1]Professor, Department of Oral Pathology, Adhiparasakthi Dental College and Hospital, Tamil Nadu, India
[2]Assistant Professor, Department of Dental Surgery, Government Villupuram Medical College, Tamil Nadu, India
[3]Prosthodontist & Commandant, No. 1 Dental Unit Assam Rifles, Shillong, Meghalaya, India
[4]Department of Periodontics, Sri Venkateshwaraa Dental College, Pondicherry, India
[5]Assistant Professor, Department of Oral and Maxillofacial Surgery, Adhiparasakthi Dental College and Hospital, Tamil Nadu, India
[6]Assistant Professor, Department of Oral Pathology, Adhiparasakthi Dental College and Hospital, Tamil Nadu, India

***Corresponding Author:** Dr. K. Sivaguru
Prosthodontist & Commandant, No. 1 Dental Unit Assam Rifles, Shillong, Meghalaya, India

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Abstract: Local halitosis affects a substantial proportion of adults and is predominantly of intraoral origin. Volatile sulfur compounds, principally hydrogen sulfide and methyl mercaptan, are generated by anaerobic bacteria acting on sulfur-containing amino acids within periodontal pockets and tongue biofilms. Periodontal disease, tongue coating, reduced salivary flow, carious lesions, and prosthetic factors are the main local contributors. This review provides dental clinicians with a concise, evidence-based overview of the etiological factors and their underlying mechanisms.

Keywords: Halitosis, Volatile Sulfur Compounds, Periodontal Disease, Tongue Coating, Oral Microbiome, Xerostomia, Dental Caries.

INTRODUCTION

Halitosis, commonly termed oral malodor, affects an estimated 15–60% of the world's population and represents the third most frequent reason for dental consultation after caries and periodontal diseases [1]. This condition has significant social and psychological consequences, placing dental clinicians at the center of its identification and care [2]. Although systemic and psychogenic origins exist, approximately 90% of halitosis cases are intraoral [3]. Current research increasingly focuses on the microbial and biochemical mechanisms underlying local halitosis, with periodontal disease, tongue coating, dental caries, and xerostomia consistently identified as leading contributors [4]. This review provides a concise summary of the common causes of local halitosis for clinical dental practice.

Periodontal Disease

Periodontal disease is the primary local cause of halitosis, often in combination with tongue coating. A retrospective analysis of 547 patients attending a breath malodor clinic found that 90.7% of cases were intra-oral, with periodontitis and gingivitis identified as the dominant cause in 33.9% of subjects; combined periodontal disease and tongue coating accounted for a further 55.2% [3]. The anaerobic microenvironment of periodontal pockets strongly promotes the production of volatile sulfur compounds (VSCs), particularly by gram-negative periodontal pathogens that metabolize sulfur-containing substrates [1].

The principal VSCs in intraoral halitosis are hydrogen sulfide (H_2S) and methyl mercaptan (CH_3SH), with established clinical thresholds of 112 ppb and 26 ppb, respectively [5]. These gases are produced by anaerobic enzymatic degradation of sulfur-containing amino acids. Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, and Fusobacterium nucleatum convert L-cysteine to H_2S via desulfhydration and L-methionine to CH_3SH via lyase activity [2]. A prospective cohort study confirmed that subgingival counts of these species correlate significantly with elevated H_2S and CH_3SH concentrations measured using portable gas chromatography [6]. Metatranscriptomic profiling of tongue

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biofilms further demonstrated that cysteine-degrading enzyme genes are overexpressed in patients with halitosis, while halitosis-free individuals exhibit greater expression of L-cysteine synthesis and nitrate reduction pathways, the latter competitively limiting sulfate reduction and H_2S output [5]. Clinically, VSCs exert cytotoxic and pro-inflammatory effects on periodontal tissues, suggesting a bidirectional relationship between halitosis and tissue destruction [2].

Tongue Coating

Tongue coating is the second major local contributor to halitosis. It comprises oral bacteria, desquamated epithelial cells, blood cells, and food residues that accumulate on the papillated dorsal surface, creating an anaerobic milieu that supports VSC production [7]. While tongue coating alone is rarely the sole cause of halitosis — identified as such in only eight of 547 patients in one clinical series — it substantially amplifies malodor when superimposed on periodontal disease, and tongue coating load is approximately six times greater in periodontitis patients than in healthy controls [3-5].

The malodorous potential of the coating depends on its microbial composition. Metatranscriptomic analysis identified Prevotella, Fusobacterium, and Leptotrichia as predominant in subjects with elevated VSCs levels, whereas Streptococcus, Veillonella, and Rothia characterized halitosis-free microbiota [5]. Quantitative light-induced fluorescence imaging confirmed a strong correlation between bacterial activity and tongue coating distribution ($r = 0.937$), and the coating area correlated with H_2S levels ($r = 0.223$), supporting its utility as a clinical monitoring tool [8]. Mechanical tongue cleaning using a toothbrush or scraper significantly reduces coating extent and organic malodor, with no significant difference in efficacy between the two instruments [9]. For patients with a persistent bacterial burden, tongue brushing with povidone-iodine or hydrogen peroxide achieves a statistically significant additional reduction in colony counts on the tongue dorsum [10].

Other Local Factors

Several additional intraoral conditions sustain or intensify halitosis. Xerostomia impairs the mechanical cleansing and buffering of oral surfaces, promoting substrate accumulation and anaerobic overgrowth [11]. Deep carious lesions and necrotic pulpal exposure create protein-rich anaerobic niches that directly fuel VSC generation [1]. Poorly maintained removable prostheses present comparable conditions by harboring biofilms rich in gram-negative species [1]. Oral microbiome dysbiosis driven by smoking, poor hygiene, and dietary factors shifts the community composition toward proteolytic anaerobes, amplifying the VSC capacity [2]. Scientometric analysis has confirmed that dental caries, gingivitis, xerostomia, and smoking are consistently co-indexed with halitosis in a substantial body of peer-reviewed literature [4].

CONCLUSION

Local halitosis is a multifactorial condition whose management depends on the systematic identification of intraoral causes. Periodontal disease and tongue coating, frequently acting in concert, account for the majority of cases, while xerostomia, carious lesions, prosthetic factors, and microbial dysbiosis are important secondary contributors. A thorough clinical assessment encompassing periodontal status, tongue coating scores, salivary flow, and caries activity provides the foundation for targeted intervention and remains the cornerstone of effective halitosis management in dental practice.

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