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Extraoral Halitosis and Systemic Diseases: A Narrative Review

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ABSTRAK
Introduction: Halitosis is a common condition characterized by an unpleasant odor originating from the oral cavity or extra-oral sources. While most cases are associated with intraoral factors such as tongue coating, periodontal disease, and poor oral hygiene, approximately 10–20% of halitosis cases are linked to extra-oral or systemic conditions. Methods: This study was conducted as a narrative review to provide an overview of the current understanding of halitosis, including its etiology, pathophysiology, diagnosis, and management, with particular emphasis on associated systemic diseases. Relevant literature was identified through searches of electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. Result and Discussion: Extra-oral halitosis can be classified into non-blood-borne and blood-borne forms. Non-blood-borne halitosis arises from disorders of the respiratory tract, gastrointestinal system, and ear–nose–throat (ENT) region, whereas blood-borne halitosis results from volatile compounds entering the bloodstream and being exhaled through the lungs.Conclusion: Effective patient care often requires collaboration between dental practitioners and medical specialists. Recognizing halitosis as a potential manifestation of systemic disease enables earlier diagnosis, appropriate referral, and improved patient outcomes. Therefore, dental professionals play an important role in the multidisciplinary detection and management of halitosis associated with systemic disorders.

INTRODUCTION

Halitosis is a condition characterized by an unpleasant odor originating from oral cavity, and commonly referred to as mouth odor, oral malodor, or bad breath. [1,2] The term was derived from combination of the Latin *halitus* (breath) and Greek *osis* (pathological process).[3] Halitosis reported to affect between 20% and 50% of the global population and may affect a person's quality of life.[4] The unpleasant odor is primarily caused by odorous compounds called volatile sulfur compounds (VCS), which results from the activity of Gram-negative anaerobic bacteria on sulfur substrates.[1] Hydrogen sulfide, methyl mercaptan and, dimethyl sulfide, represent 90% of the VSCs in bad breath. [1] In most cases, halitosis are contributed by intra-oral factors such as poor oral hygiene, tongue coating, periodontal diseases, or dental caries.[3,5] The remaining are caused by extra-oral factors such as respiratory problems, gastrointestinal diseases, metabolic disorders, chronic medication, and other systemic involvements.[1,3,5] The oral–systemic link in halitosis is frequently underrecognized in routine dental practice, which might lead to misdiagnosis and suboptimal management. A comprehensive understanding of the relationship between systemic conditions and halitosis is essential for accurate diagnosis, appropriate referral, and effective interdisciplinary care.[3,4,6] This literature review aims to summarize current evidence on the association between halitosis and systemic diseases, highlighting underlying mechanisms and clinical implications.

METHODS

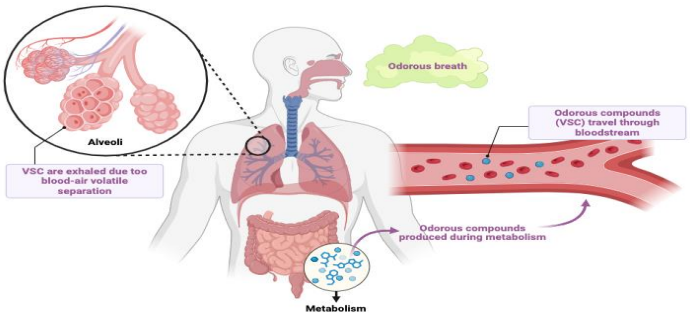
This study was conducted as a narrative review to provide an overview of the current understanding of halitosis, including its etiology, pathophysiology, diagnosis, and management, with particular emphasis on associated systemic diseases. Relevant literature was identified through searches of electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. The search strategy incorporated combinations of the following keywords: "halitosis", "oral malodor", "blood-borne halitosis", "extraoral halitosis", "volatile sulfur compounds", and "dimethyl sulfide". Articles were selected based on their relevance to the objectives of this review. Priority was given to original research articles, systematic reviews, meta-analyses and clinical studies. Articles not directly related to halitosis or lacking sufficient scientific evidence were excluded. The selected literature was reviewed and synthesized narratively.

RESULT AND DISCUSSIONS

Pathophysiology of Halitosis Related to Systemic Conditions

Extra-oral factors induce about 10-20% of all halitosis cases, which is associated by systemic disorders. The difference between intra-oral and extra-oral halitosis can be achieved by assessing both oral and nasal breath. [3,4,7] Patients with intra-oral halitosis exhibit bad breath originating only from the mouth, while nasal breath remains unaffected. Extra oral halitosis can be classified into non-blood-borne and extra-oral blood-borne halitosis.

Figure 1. Pathophysiology of Blood-Borne Halitosis



(This illustration was regenerated using BioRender)

The majority of patients have blood-borne halitosis, which is also caused by VSCs, in particular dimethyl sulfide (DMS). Volatile odorous compounds that was produced during metabolism are circulated by the blood and transported to the lung where an exchange of exhaled air occurs due to blood-air volatile separation in the alveoli (Fig.1).[6,7] Non–blood-borne halitosis arises from odorous compounds originating in the respiratory tract, oral or nasal cavities, and the gastrointestinal (GI) tract. It is commonly associated with anaerobic infections, which can create certain discharges and odors depending on the microorganisms or compounds involved. [8,9] Hydrogen sulfide (H_2S), methyl mercaptan (MM), and dimethyl sulfide (DMS) are the most reported VSC in halitosis and implicated as the contributor to the malodourous substances of flatus and feces. DMS is stable in blood and reported to be the biggest contributor of extra oral halitosis. [7,10]

Respiratory System

Respiratory tract diseases represent one of the most common extra-oral causes of halitosis, accounting for a substantial proportion of non-oral malodor cases. [11,12] Pathological processes affecting the nasal cavity, paranasal sinuses, and pharyngeal tissues may promote the accumulation of protein-rich secretions that undergo bacterial degradation and produce volatile sulfur compounds (VSCs), thereby contributing to unpleasant breath odor. [3,12] Among respiratory disorders, chronic sinusitis, tonsillitis with tonsillolith formation, and postnasal drip are the conditions most frequently associated with halitosis.

Chronic Sinusitis

Chronic sinusitis, also known as chronic rhinosinusitis (CRS), is characterized by persistent inflammation of the paranasal sinus mucosa lasting at least 12 weeks and is recognized as a significant extra-oral source of halitosis. [11,13] Patients with CRS commonly experience nasal obstruction, facial pressure, mucopurulent nasal discharge, and olfactory dysfunction, all of which may contribute to the development of oral malodor.

The pathogenesis of halitosis in CRS is primarily related to the retention of mucus within the sinus cavities, creating an environment favorable for bacterial colonization and biofilm formation. [12,14] Anaerobic microorganisms degrade proteins present in inflammatory exudates and mucus, resulting in the production of sulfur-containing compounds such as hydrogen sulfide, methyl mercaptan, and dimethyl sulfide. [3,12] These compounds may drain into the nasopharynx and subsequently be released during respiration, producing a characteristic foul odor. [3,14] Persistent inflammation also increases epithelial desquamation and leukocyte accumulation within sinus secretions, providing additional substrates for proteolytic bacterial metabolism. [3,14] Furthermore, reduced sinus ventilation and impaired mucociliary clearance facilitate the stagnation of secretions and prolong bacterial activity, thereby exacerbating malodor production. [13,14] Studies have reported that patients with chronic rhinosinusitis frequently complain of both oral and nasal malodor, highlighting the importance of

evaluating sinonasal pathology in individuals presenting with persistent halitosis of unclear origin. [11,12] Clinical improvement of sinus inflammation through medical or surgical management has been associated with a reduction in halitosis symptoms, supporting the causal relationship between CRS and oral malodor. [12,13] Endoscopic sinus surgery and targeted antimicrobial therapies may improve mucociliary function and decrease the bacterial burden responsible for VSC production.[13,14]

Tonsillitis and Tonsilloliths

Chronic tonsillitis is another well-recognized respiratory cause of halitosis and is frequently associated with recurrent throat infections and persistent inflammation of the palatine tonsils. [3,12] Repeated inflammatory episodes may alter the architecture of the tonsillar crypts, creating deep recesses that trap food debris, desquamated epithelial cells, and microorganisms.[1,12] The accumulation of organic material within these crypts promotes bacterial putrefaction and the generation of malodorous gases. [1,12] Anaerobic Gram-negative bacteria residing in tonsillar crypts metabolize sulfur-containing amino acids and produce VSCs similar to those detected in periodontal disease and tongue coating. [1,3] Consequently, patients with chronic tonsillitis often report persistent bad breath even in the presence of satisfactory oral hygiene practices.[1,12]

Tonsilloliths, commonly known as tonsil stones, are calcified aggregates formed from retained debris and bacterial biofilms within tonsillar crypts. [1,14] These structures contain microorganisms, epithelial cells, food particles, and mineral deposits that collectively serve as a source of foul-smelling compounds. [1,14] Tonsilloliths have been strongly associated with chronic halitosis and may produce a characteristic sulfurous odor that is difficult to eliminate through routine oral hygiene measures alone.[1] In addition to acting as reservoirs for odor-producing bacteria, tonsilloliths may sustain chronic local inflammation and further enhance bacterial proliferation within the tonsillar tissue. [1,14] Several studies have demonstrated that removal of tonsilloliths or treatment of chronic tonsillar disease can significantly improve halitosis symptoms1 In refractory cases, tonsillectomy may be considered when chronic tonsillitis or recurrent tonsillolith formation is identified as the primary source of malodor.[1]

Postnasal Drip

Postnasal drip refers to the excessive accumulation and drainage of nasal secretions from the posterior nasal cavity into the nasopharynx and oropharynx. [13,14] It is commonly associated with chronic rhinosinusitis, allergic rhinitis, upper respiratory infections, and other inflammatory disorders of the upper airway.[13] Postnasal secretions contain proteins, inflammatory cells, microorganisms, and cellular debris that may serve as substrates for bacterial degradation within the oropharynx. [3,14]As these secretions accumulate on the posterior tongue and pharyngeal surfaces, bacterial metabolism increases and promotes the production of VSCs and other odoriferous compounds. The continuous presence of mucus in the throat may also contribute to xerostomia-like symptoms by encouraging mouth breathing and disrupting normal salivary cleansing mechanisms. [3,8] Reduced salivary flow diminishes the natural clearance of microorganisms and odor-producing metabolites, thereby worsening halitosis. [9–11] Furthermore, chronic postnasal drip frequently leads to persistent throat clearing and mucus retention, conditions that further support microbial growth and malodor generation.[10,11] Evidence suggests that patients with chronic postnasal drip often report improvement in breath odor following successful management of the underlying sinonasal or allergic condition.

Gastrointestinal System

Gastrointestinal (GI) disorders have been suggested as potential extra-oral causes of halitosis, although it is considered less common than oral etiologies. [1,15] The gastrointestinal tract may influence breath odor through several mechanisms, including the retrograde movement of gastric fluids, alterations in the gastrointestinal microbiota, production of volatile metabolites, and systemic absorption of odorous compounds that are exhaled through the lungs. [3,15,16] While the direct relationship between gastrointestinal diseases and halitosis remains debatable, numbers of evidence suggests that certain GI conditions, particularly gastroesophageal reflux disease (GERD) and *Helicobacter pylori* infection may contribute to oral malodor in susceptible individuals.

Gastroesophageal Reflux Disease (GERD)

Gastroesophageal reflux disease (GERD) is a chronic digestive disorder characterized by the reflux of gastric contents into the esophagus due to dysfunction of the lower esophageal sphincter. [17] Common symptoms include heartburn, regurgitation, chest discomfort, and extra-esophageal manifestations affecting the upper respiratory tract and oral cavity. Several mechanisms have been proposed to explain the association between GERD and halitosis. Refluxed gastric contents may reach the oral cavity and pharynx, introducing acidic substances and volatile compounds that

contribute directly to unpleasant breath odor. [15–17] Repeated exposure of the oral environment to gastric acid may also alter salivary composition, reduce buffering capacity, and promote changes in the oral microbiota that favor the production of malodorous metabolites

In addition, GERD has been associated with symptoms of xerostomia and decreased salivary flow, both of which may impair the natural cleansing mechanisms of the oral cavity and facilitate bacterial accumulation. Reduced salivary clearance allows volatile sulfur compounds (VSCs) and other odoriferous substances to persist for longer periods, thereby increasing the perception of halitosis. [15,17,18] Furthermore, improvement of reflux symptoms through proton pump inhibitor therapy has been associated with a reduction in oral malodor in some patients, supporting a potential causal relationship.

The association between GERD and halitosis remains controversial because many affected individuals simultaneously present with oral conditions that may independently contribute to malodor. Consequently, GERD should be considered a potential contributing factor rather than a primary cause in most cases of halitosis. [1,16]

Helicobacter pylori Infection

Helicobacter pylori is a Gram-negative bacterium that is recognized as a major etiological factor in chronic gastritis, peptic ulcer disease, and gastric malignancy. Increasing evidence has suggested a possible association between *H. pylori* infection and halitosis. [15,16] Although the exact mechanism remains unclear, multiple pathways have been proposed. First, *H. pylori* may directly produce volatile sulfur compounds and other malodorous metabolites during its metabolic activity within the gastric environment. Second, chronic gastric inflammation associated with infection may alter gastrointestinal physiology and promote the release of odoriferous compounds that contribute to unpleasant breath.[7,15,16,18]

Another proposed mechanism involves the oral cavity as a potential reservoir for *H. pylori*. The microorganism has been detected in dental plaque, saliva, tongue coating, and periodontal pockets, suggesting that oral colonization may contribute indirectly to halitosis. [11,15] In such cases, oral malodor may result not only from gastric infection but also from local microbial interactions within the oral biofilm.

Mechanisms Linking Gastrointestinal Disorders to Oral Malodor

The relationship between gastrointestinal diseases and halitosis is multifactorial and involves both local and systemic pathways. One mechanism is the direct retrograde movement of volatile compounds from the stomach or esophagus into the oral cavity through regurgitation or reflux events. It is particularly relevant in patients with GERD and other disorders characterized by impaired gastroesophageal barrier function. A second mechanism involves microbial metabolism within the gastrointestinal tract. Certain microorganisms may contain sulfur-containing compounds, short-chain fatty acids, polyamines, and other volatile metabolites capable of contributing to malodor. Some of these compounds may reach the oral cavity directly through reflux or indirectly through systemic circulation and pulmonary excretion.

Inflammation is another important factor linking gastrointestinal disease and halitosis. Chronic inflammatory conditions of the GI tract may increase the production of odorous substances. Inflammatory mediators may also influence salivary gland function, oral microbial environment, and mucosal health, thereby creating favorable condition for malodor development. [7,15,16,18] At last, systemic absorption of volatile compounds generated within the gastrointestinal tract may contribute to blood-borne halitosis. After entering the circulation, these compounds can be transported to the lungs and released through alveolar gas exchange, resulting in detectable malodor in exhaled breath. This mechanism is particularly relevant when discussing extra-oral halitosis and highlights the complex interaction between gastrointestinal health and breath odor.

Endocrine and Metabolic Disorders

Endocrine and metabolic disorders represent important systemic causes of extra-oral halitosis because metabolic abnormalities may lead to the production and accumulation of volatile compounds that are subsequently released through exhaled air. [1,7,10] Unlike oral halitosis, which primarily originates from bacterial degradation of proteins within the oral cavity, metabolic halitosis results from alterations in biochemical pathways that results in characteristic breath odors.

Diabetes Mellitus

Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia resulting from impaired insulin secretion and/or insulin function. Poor glycemic control may lead to oral and systemic manifestations that contribute to halitosis. [20,21] One of the most characteristic breath odors associated

with diabetes is the fruity or acetone-like odor observed in diabetic ketoacidosis (DKA). During insulin deficiency, the body shifts from glucose metabolism to fatty acid oxidation as an alternative energy source. [21,22] This metabolic pathway results in the excessive production of ketone bodies, including acetoacetate, β-hydroxybutyrate, and acetone. Acetone diffuses into the bloodstream and is exhaled through the lungs, producing the fruity breath odor commonly referred to as ketone breath.

Diabetes may also contribute indirectly to halitosis through alterations in salivary gland function. Hyperglycemia is frequently associated with reduced salivary flow and xerostomia, particularly among individuals with poorly controlled disease. [20,21,23,24] Saliva plays a critical role in maintaining oral health by acting as mechanical cleansing, buffering acids, and limiting bacterial growth. [25] Reduced salivary secretion promotes bacterial accumulation on the tongue and oral mucosal surfaces, increasing the production of volatile sulfur compounds (VSCs)

Renal Failure (Uremic Fetor)

Chronic kidney disease (CKD) and advanced renal failure are frequently associated with a distinctive breath odor known as uremic fetor. Uremic fetor is considered a classical manifestation of severe renal dysfunction and is characterized by an ammonia-like or urine-like odor of the breath. [7,11,26] The pathogenesis of uremic fetor is primarily related to the accumulation of nitrogen-containing waste products resulting from impaired renal excretion. As kidney function declines, blood urea concentrations increase substantially. Urea diffuses into saliva, where it is metabolized by urease-producing oral bacteria into ammonia and carbon dioxide. The resulting elevation in oral ammonia levels contributes directly to the characteristic odor associated with renal failure.

In addition to ammonia production, patients with advanced kidney disease may experience xerostomia due to fluid restrictions, medication use, salivary gland dysfunction, and systemic dehydration. Reduced salivary flow promotes tongue coating accumulation and bacterial overgrowth, thereby increasing the production of malodorous compounds. Furthermore, uremic toxins may alter the composition of the oral microbiota and contribute to inflammatory changes within the oral cavity. [26–29] Several studies have demonstrated that volatile organic compounds (VOCs) associated with renal dysfunction can be detected in exhaled breath, highlighting the role of pulmonary elimination in systemic halitosis. Breath analysis technologies have identified elevated concentrations of ammonia and other nitrogen-containing compounds in patients with CKD, supporting the concept of blood-borne halitosis in renal disease.

Hepatic Disease (Fetor Hepaticus)

Liver disease may produce a distinctive form of extra-oral halitosis known as fetor hepaticus. Fetor hepaticus is most commonly observed in patients with advanced liver dysfunction, portal hypertension, or hepatic failure. [1,7,12] The development of fetor hepaticus is primarily associated with impaired hepatic detoxification and altered metabolism of volatile compounds. Under normal physiological conditions, the liver metabolizes numerous endogenous and exogenous substances before they enter the systemic circulation. In advanced liver disease, this detoxification capacity becomes compromised, allowing volatile compounds to accumulate in the bloodstream.

One of the principal compounds implicated in fetor hepaticus is dimethyl sulfide (DMS), a volatile sulfur compound that has been strongly associated with extra-oral halitosis. Elevated blood concentrations of DMS have been reported in patients with severe hepatic dysfunction and portosystemic shunting. Because DMS is highly volatile, it readily diffuses across the alveolar membrane and is released during exhalation, producing the characteristic odor of fetor hepaticus. [1,7,10,30]

Other sulfur-containing compounds, ketones, and aromatic metabolites may also contribute to the complex odor profile observed in liver disease. In addition, alterations in gut microbiota composition and increased intestinal permeability frequently associated with cirrhosis may increase the generation and systemic absorption of odoriferous metabolites. These compounds bypass normal hepatic metabolism through portosystemic shunts and enter the pulmonary circulation, where they are ultimately exhaled.[7,17,18,21,30] The presence of fetor hepaticus is considered a clinically significant sign because it may indicate advanced hepatic impairment and is often associated with severe complications such as hepatic encephalopathy. [1,30] Therefore, recognition of characteristic breath odors may provide valuable diagnostic information regarding underlying systemic disease.

Role of Xerostomia in Systemic-Related Halitosis

Xerostomia, commonly referred to as dry mouth, is one of the most important contributing factors to halitosis in patients with systemic diseases. [25] Although xerostomia itself is not a direct cause of malodor, the reduction in salivary quantity and quality creates an oral environment that

favors bacterial proliferation, tongue coating accumulation, and increased production of volatile sulfur compounds (VSCs). [9,31] Consequently, xerostomia serves as a critical link between systemic disorders and the development of oral malodor.

Causes of Xerostomia (Disease-Related and Medication-Induced)

Xerostomia may arise from a variety of systemic conditions that affect salivary gland function, fluid balance, or autonomic regulation. [25,32] Among endocrine disorders, diabetes mellitus is frequently associated with reduced salivary flow due to chronic hyperglycemia, dehydration, autonomic neuropathy, and microvascular alterations affecting the salivary glands. [23,25,33] Patients with poorly controlled diabetes often report persistent oral dryness, which may contribute significantly to halitosis. Autoimmune diseases, particularly Sjögren syndrome, are among the most common pathological causes of severe xerostomia. In Sjögren syndrome, lymphocytic infiltration and progressive destruction of salivary gland tissue result in marked salivary hypofunction and chronic oral dryness. Reduced salivary secretion promotes oral microbial imbalance and increases the risk of halitosis. [25,32–34] Chronic kidney disease and end-stage renal disease may also contribute to xerostomia through fluid restrictions, electrolyte disturbances, salivary gland dysfunction, and medication use. Similarly, patients with advanced liver disease may experience salivary alterations related to dehydration, nutritional deficiencies, and systemic metabolic disturbances.

Medication-induced xerostomia is particularly common among medically compromised patients. More than 500 medications have been associated with decreased salivary flow. Frequently implicated drug classes include antidepressants, antipsychotics, antihistamines, anticholinergics, antihypertensive agents, diuretics, and sedative medications. [32,35,36] Polypharmacy, which is common among older adults and patients with chronic diseases, further increases the risk of xerostomia and subsequent halitosis.. In addition, radiotherapy involving the head and neck region may cause irreversible salivary gland damage, resulting in severe xerostomia and persistent oral malodor. Although radiation-induced xerostomia is not a systemic disease, it represents an important medically related cause of salivary dysfunction and halitosis.

Salivary Dysfunction and Bacterial Overgrowth

Saliva plays a fundamental role in maintaining oral homeostasis through mechanical cleansing, lubrication, buffering of acids, antimicrobial activity, and regulation of the oral microbiome. Normal salivary flow continuously removes food debris, desquamated epithelial cells, and microorganisms from oral surfaces. When salivary secretion decreases, these protective functions become compromised. [23,25,32] Reduced salivary flow promotes the accumulation of bacterial biofilms on the tongue dorsum, periodontal tissues, and mucosal surfaces. The posterior dorsum of the tongue provides an ideal anaerobic environment for proteolytic bacteria capable of degrading sulfur-containing amino acids such as cysteine and methionine. This bacterial metabolism produces volatile sulfur compounds including hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), and dimethyl sulfide ($(CH_3)_2S$), which are the principal compounds responsible for halitosis.

In addition to increased VSC production, xerostomia enhances the retention of food debris and cellular remnants within the oral cavity. These organic substrates provide additional nutrients for proteolytic bacteria and further increase malodor production. Salivary hypofunction may also alter the composition of the oral microbiota, favoring species associated with periodontal disease and tongue coating formation. [1,12,25,32] Furthermore, saliva contains numerous antimicrobial proteins, including lysozyme, lactoferrin, histatins, and secretory immunoglobulin A (sIgA), which help regulate microbial growth. [25] Reduced secretion of these protective components may permit bacterial overgrowth and dysbiosis, thereby exacerbating oral malodor. The relationship between xerostomia and halitosis is therefore multifactorial, involving impaired mechanical cleansing, increased bacterial colonization, enhanced proteolytic activity, and disruption of normal microbial homeostasis.

CONCLUSION AND SUGGESTION

Halitosis is a common condition that is associated with intraoral causes; however, a significant proportion of cases may originate from extra-oral and systemic conditions. [3,7] This narrative review highlights the multifactorial nature of systemic-related halitosis and elaborates the contribution of systemic conditions to oral malodor through various mechanisms. [7,23,30] These mechanisms include the direct production of volatile compounds, systemic circulation and pulmonary excretion of odoriferous metabolites, chronic inflammation, microbial dysbiosis, and salivary gland dysfunction resulting in xerostomia. [15,17,30] Numerous systemic conditions and medications reduce salivary flow, impairing the protective functions of saliva and creating an environment that favours bacterial

overgrowth and VSC production. Therefore, the relationship between systemic diseases and halitosis is often indirect, involving interactions between systemic and oral alterations. Persistent or unexplained halitosis may present as early clinical manifestation of an underlying systemic disorder. Therefore, halitosis has potential value as a non-invasive clinical indicator of systemic health status.

For dental practitioners, a thorough understanding of systemic-related halitosis is essential for accurate diagnosis and effective patient management. Clinicians should provide comprehensive assessment that includes oral and periodontal conditions, salivary function, medical history, and medication history. When systemic involvement is suspected, proper referral is necessary to provid comprehensive patient care.

Successful management of systemic-related halitosis requires multidisciplinary collaboration among dentists, physicians, otolaryngologists, internist and other healthcare professionals. Early recognition, appropriate referral, and integrated treatment strategies can contribute to the improvement of halitosis and quality of life.

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