Article published online: 2021-11-01

Review Article

Repercussions of Diabetes Mellitus on the Oral Cavity

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Abstract

Oral health is a cardinal element of nutritional as well as systemic well-being and plays a substantial part in sustaining optimum general health condition. Various factors influence oral health including metabolic diseases such as endocrine (diabetes mellitus [DM]), hematological, gastrointestinal, cutaneous, and neurological diseases. The intent of this review is to accentuate the correlation between DM and oral disorders, like those upsetting oral mucosa and supporting tissues. A review of literature was performed using relevant key words ("Oral Manifestations of Diabetes" OR "Oral Complications of Diabetes" OR "Oral Impacts of Diabetes" OR "Oral Repercussions of Diabetes") in prominent journals pertaining to Endocrinology and Dentistry (Journal of Periodontology, Periodontology 2000, British Dental Journal, The Lancet Diabetes and Endocrinology, PloS ONE, and Nature Reviews Genetics). The most frequently witnessed diabetic manifestations in oral cavity include gingivitis and periodontitis leading to premature tooth loss, salivary dysfunctions, dental caries, delayed wound healing, bacterial and fungal infections, lichen planus, taste impairment, tongue abnormalities, neurosensory oral disorders, halitosis, and dry socket. In the end, I have comprehensively described the role of antidiabetic drugs in the management of DM and eventually leading to prevention of its oral complications. In this review, etiopathophysiology of each oral complication has been prudently analyzed to contemplate the establishment of a possible preventive and treatment approach.

Keywords: Dental caries, diabetes mellitus, dry socket, infection, periodontitis, xerostomia

Introduction

The terminology diabetes mellitus (DM) means a conglomerate of disorders involving unusual fat, protein, and carbohydrate digestion that eventually causes acute and chronic manifestations because of relative or absolute deficiency of insulin (a hormone that regulates blood glucose).[1] It includes three general classes of diabetes: type 1 (previously referred to as insulin-dependent or juvenile diabetes), that is due to an absolute insulin insufficiency; type 2 (formerly known as non-insulin dependent or adult-onset diabetes), which is the consequence of insulin resistance and a secretory flaw of insulin, and it can be gestational, a state of abnormal glucose resistance amid pregnancy. The two most common types of diabetes include type 1 and type 2.[2]

Literature Search Methodology

An electronic search was performed, from January 2010 to December 2018, to identify articles on the oral manifestations of DM on the oral cavity. Related articles published in the English language and prominent endocrinology as well as dentistry journals were included.

Access this article online

Quick Response Code:

Website: www.ejgd.org

DOI: 10.4103/ejgd.ejgd_28_19

- Journal of Prosthodontic Research
- Journal of Periodontology
- Periodontology 2000
- British Dental Journal
- Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology
- The Lancet Diabetes and Endocrinology
- Nature Reviews Genetics
- PloS ONE
- World Journal of Diabetes
- International Journal of Oral Sciences.

The keywords used for the search strategy are as follows:

- "Oral Manifestations of Diabetes" OR "Oral Complications of Diabetes" OR "Oral Impacts of Diabetes" OR "Oral Repercussions of Diabetes"

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How to cite this article: Ahmad P, Akhtar U, Chaudhry A, Rahid U, Saif S, Asif JA. Repercussions of diabetes mellitus on the oral cavity. Eur J Gen Dent 2019;8:55-62.

- "Diabetes and Oral Cavity" OR "Diabetes and Mouth" OR "Diabetes and Oral Health."

Diabetes Mellitus and its Oral Manifestations

Oral manifestations of DM include gingivitis and periodontitis leading to premature tooth loss,[3] poor oral wound healing,[4] salivary dysfunction such as xerostomia and sialosis,[5] dental caries,[6] bacterial and fungal infections,[7] halitosis,[8] tongue abnormalities such as fissured tongue, bald tongue, geographic tongue, and median rhomboid glossitis,[9] taste disturbance,[10] oral lichen planus and lichenoid drug reaction,[11] dry socket,[12] and neurosensory oral disorder like burning mouth syndrome.[13] Figure 1 represents oral manifestations of DM.

Periodontitis

Periodontitis is the inflammation of gingiva as well as supporting tissues associated with the teeth and is primarily generated by periodontal bacteria.[14] It is remarkably the most prevailing oral disease related to diabetes.[15]

Pathophysiology of periodontitis

The definite mechanism involved in triggering the periodontal infections in diabetics is ambiguous; however, according to certain studies, the function of polymorphonuclear leukocytes is undermined as a consequence of conditions, for instance, flawed immune response, variation in structure of collagen, and advanced glycation end products (AGEs), ultimately promoting intrusion of bacteria into these tissues.[16]

The microbiota found in the dental plaque commences the inflammatory process. The toxins produced by bacteria induce gingival inflammatory process and eventually generate periodontal pocket. The pocket gets deeper and deeper as the condition is aggravated, and ultimately, the devastation of alveolar bone leads to periodontal detachment.[17]

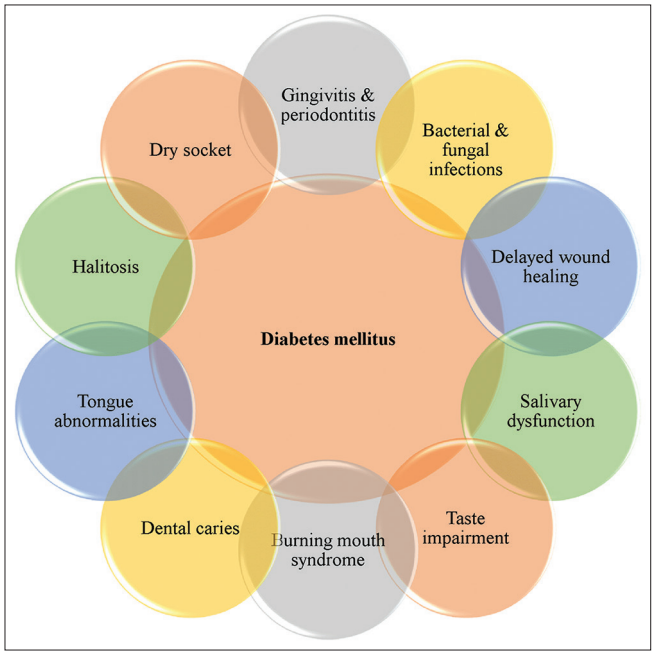

Figure 1: Oral complications of diabetes mellitus

Pathogenesis of effects of diabetes on periodontal disease

Subgingival microflora

Capnocytophaga species has been reported in higher proportion in diabetics as compared to nondiabetics. This higher proportion is related to the fact that subgingival microflora differ in quantity and quality in diabetics when compared with nondiabetics.[18]

Host immune cells

In diabetes, the operation of immune cells, i.e., macrophages, neutrophils, and monocytes is changed. This alteration in functions include impairment of neutrophil adherence, phagocytosis, and chemotaxis, which eventually might prevent killing of bacteria in the periodontal pocket and consequentially increase destruction of periodontium.[19]

Glucose level of gingival crevicular fluid

In diabetics, there is an escalation in the levels of gingival crevicular fluid glucose that is directly related to the decreased fibroblasts' wound-healing efficiency in periodontium.[20] The attachment of fibroblasts is necessary for proper wound healing and normal tissue turnover. This attachment of fibroblasts is inhibited in diabetes.[19]

Advanced glycation end products

Proteins merge with glucose molecules and go through glycation to generate AGEs in the circumstances of sustained hyperglycemia. These AGEs are directly involved in the formation of collagen and increased collagen cross-linking. This newly generated collagen is vulnerable to degradation by collagenase. In diabetics, collagenase is in its active form. All these factors induce alterations in collagen metabolism which ultimately influences normal wound-healing process. AGE-modified collagen may affect functional, cellular, and structural characteristics, which eventually leads to transformation in bone metabolism.[21]

Receptors of advanced glycation end products

Receptors for AGEs (RAGEs) are activated by AGEs. These receptors are present on the surfaces of endothelial cells, macrophages/monocytes, smooth muscles, and neurons.[22] In hyperglycemia, there is an increased vascular permeability as a result of upsurge of AGE-RAGE interaction as well as hike in expression of RAGEs. On monocytes, AGE-RAGE interaction boosts cellular oxidant and triggers transcription factor nuclear factor-kappa B, leading to increased formation of pro-inflammatory cytokines, i.e., interleukin-1 β and tumor necrosis factor alpha (TNF-α). In a study conducted on diabetic animal models,[16] Llambés *et al.* established that blockade of RAGEs alleviates matrix metalloproteinase, TNF-α, and interleukin-6 levels in the gingival crevicular fluid. This reduces aggregation of AGEs in periodontal tissues which ultimately, in response to *Porphyromonas gingivalis,* lessens the alveolar bone loss.[19]

Pathogenesis of effects of periodontitis on diabetes

Individuals having periodontal infection and diabetes are at greater risk of developing uncontrolled glucose levels as compared to individuals with diabetes without periodontitis.[23] Improvement of glycemic control and reduction in requirements of insulin post periodontal treatment have been reported in clinical trials of diabetics. Improved results were evident when mechanical therapy was complemented with the use of antibiotics.[19]

Role of inflammatory mediators

The pouring of inflammatory mediators into the systemic circulation is suggested as a mechanism by which cytokine dysregulation related with diabetes could be amplified by periodontal infection.[24] There is an increased generation of these inflammatory mediators in the gingival tissues of diabetics with periodontitis as compared to diabetics without periodontitis.[25] These mediators are responsible for increasing inflammation and eventually worsen insulin resistance.[19] Figure 2 shows the role of C-reactive protein in DM.

Impacts of periodontal therapy on glycemic control

Majority of the studies have shown that periodontal therapy boosts glycemic control in diabetics. In the absence of periodontal therapy, glycemic control in diabetics was dropped from 17% to 6.8%.[25] A prospective research found that in diabetics having chronic periodontitis, there was a tremendous decrease in clinical attachment gains as well as probing depth when 1% alendronate was locally delivered into the periodontal pockets.[26]

Poor oral wound healing

In patients whose diabetes is under good control and who do not have complications from it, wound healing is generally fairly normal, especially in younger people. On the other hand, when diabetes is poorly controlled and when it is accompanied by chronic complications, wound healing can be significantly impaired.[27] The long-term complications of diabetes occur due to damage to the very small blood vessels nourishing the tissues and organs of the body.[28] The body's ability to heal a wound depends on a healthy blood supply which is needed to deliver nutrients on the cells that provide the protective defense against infection and those that cause inflammation.[29] In this sense, inflammation is beneficial in that it leads to the mopping up and removal of dead and damaged tissue, which paves the way for its replacement by new healthy healing skin and underlying tissue. Second, short-term high blood sugar paralyzes these blood and tissue defenses so that their infection-fighting and inflammatory actions are much weaker. Finally, insulin itself stimulates healing and regenerative actions in body tissues. If the diabetes is poorly controlled, this suggests that insulin is insufficient or infective, which can further impair healing.[4,27]

Salivary dysfunction

A gland comprises of specific kind of cells, wherein they create items which are utilized somewhere else in the body. A salivary gland is a gland that secretes saliva. In humans, parotid, sublingual, and submandibular glands are termed as major salivary glands which are in three pairs whereas oral cavity also harbors many minor salivary glands.[30]

Saliva is a complex natural fluid, which consists of approximately 99% of water and just 1% of organic and nonorganic materials.[31] These components are from inside the organ and shipped from the blood.[32] The compounds which form saliva are also available in blood.[33]

The major functions of saliva can be categorized into 5 classes: (1) antibacterial action, (2) preservation of tooth integrity, (3) clearance and buffer activity, (4) digestion and taste, and (5) protection and lubrication. People realize its significance when the salivary flow is alleviated. It leads to decrease of individual's quality of life.[34]

Xerostomia is an individual's subjective perception of oral dryness that usually, but not inevitably, is associated with reduced quantity of saliva. Normal range of resting whole saliva's flow rate fluctuates from 0.3 to 0.5 mL/min,

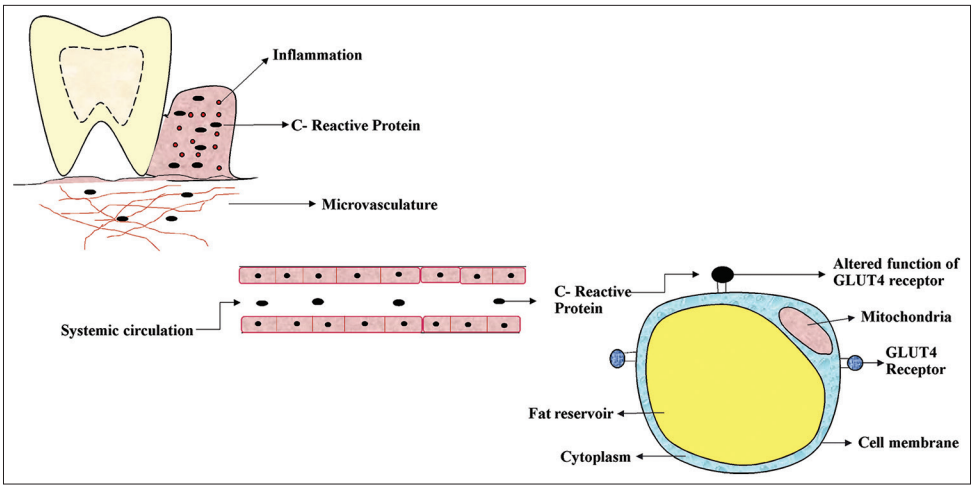

Figure 2: The ability of insulin to transport/infuse glucose into adipose cell via GLUT4 receptors is decreased which leads to increased blood glucose levels

whereas range of 0.10–0.01 mL/min is considered to be as hyposalivation.[35]

Individuals suffering from DM have been reported with the complaint of dry mouth or xerostomia and encounter salivary gland dysfunction. The reason remains unknown; however, it might be due to polyuria or changes in the salivary glands' basement membrane. In type 1 DM, xerostomia is most likely to be reliant on glucose control, while in type 2 DM, salivary secretion is likely to be specifically affected by autonomic neuropathy and xerogenic drugs. Xerostomic complaints might be because of thirst, a typical symptom of diabetes. Saliva may be valuable for diagnosing and/or monitoring systemic disorders, and in the future, it may be probable to assess glucose levels.[36]

Numerous epidemiologic studies have recommended that xerostomia is common among DM patients. Furthermore, there are studies which demonstrate that diabetics exhibited low rates of salivary flow as compared to nondiabetics.[5,37]

Sialosis

Sialosis or sialadenosis is a recurrent, nonneoplastic, noninflammatory growth or augmentation of salivary glands typically having an association with a basic systemic disorder.[38] It principally develops in the parotid gland.[39] Approximately 10%–25% of people suffering from long-standing DM may develop noninflammatory, nonneoplastic, and asymptomatic enlargement of salivary glands; however, a low relationship between DM and parotid enlargement was stated, and just 3% of 405 patients having type 1 DM were found having aforementioned complication.[40]

Both parotid glands are typically affected, in spite of the fact that the submandibular glands might also be involved. Histologically, this enlargement consists of acinar cells' enlargement and fatty invasion of the interstitium. Microangiopathy and autonomic neuropathy are the two most commonly occurring degenerative manifestations of DM. They are thought to play a role in development of structural changes in the salivary gland tissues, hence causing the hypofunction of the salivary gland by influencing the microcirculation of glandular tissue and autonomic innervation.[41]

Dental caries

The association between DM and dental caries is dubious.[42] According to some reports, the incidence of dental caries is higher in type 1 diabetes[43] whereas others propose escalated prevalence in type 2 diabetes.[44] It is interested to note that according to some studies, there is absolutely no relation between DM and dental caries.[28] In patients with type 1 DM, several risk factors have been associated with establishment of dental caries including elevated concentration of glucose in saliva, reduced salivary flow, decreased buffering effect of saliva, change in salivary biochemical characteristics, presence of dental plaque, cariogenic diet, and poor oral hygiene.[45]

The probability of developing dental caries decreases when the individual is very well-controlled diabetic. On the other hand, consuming unrestricted sugar intake is at a greater risk of developing dental caries. It is a well-established fact that with the increasing age, the prevalence of type 2 DM also increases.[3] It is suggested that the caries arising in dental enamel decreases with the increase of age, whereas those carious lesions arising in cementum increase with age. According to a study, the carious lesions that occur in the radicular area of teeth are more commonly found in older diabetics.[28] In a study conducted by Alavi *et al*.,[46] the oral hygiene of type 1 diabetics was compared with a control group. The results showed that oral hygiene of the control group was better than diabetics, most probably because of hyposalivation. To obtain improved level of oral hygiene, it is advised to have regular checkups, consumption of fluoride, a tight and scheduled glycemic control, use of sealants, less ingestion of cariogenic diet, and consuming fluoride tap water for drinking purpose.

Correlation between diabetes, periodontitis, and dental decay

Normal functioning of saliva is imperative to preserve the soundness of oral soft tissues and teeth. Filtering of blood glucose is performed by salivary glands that would be transformed by neural or hormonal regulation. However, the basement membrane in salivary gland is changed by microvascular damage, consequently boosting leakage of glucose from ductal cells. This causes escalated salivary and crevicular glucose levels. This surge leads to declined fibroblastic activity causing intensification of plaque accumulation. The plaque metabolizes the salivary glucose to lactic acid as a result of which pH of saliva is decreased and an upsurge of acidophilic bacteria takes place. If the leakage of glucose into the saliva is uncontrolled and long termed, metabolic activity of oral microbiota is heightened. This alters the natural equilibrium of dental biofilm and leads to dental decay and periodontal complications.[47] The diagrammatic depiction is shown in Figure 3.

Bacterial and fungal infections of oral cavity

Bacterial infections

Diabetics are more prone to suffer from bacterial infections of the oral cavity.[7] Their impaired defense mechanism is the major contributing factor in acquiring these infections hence regarded as immunocompromised.[29] In poorly controlled diabetes, the bacterial infections are not confined to oral cavity; but, they can spread throughout the body and can recur as well. Many studies have proposed that diabetics are more susceptible to deep neck bacterial infections.[48] According to a prospective study, the region of oral cavity that is mostly involved in bacterial infection is submandibular space pursued by buccal space. *Streptococcus* species was found to be the main culprit in both the cases.[49] These bacteria form plaque by merging with the food-inducing gingivitis, halitosis, and dental caries. Furthermore, complicated oral bacterial infections may lead to mouth sores.[15] *Streptococcus mutans*, the causative agent of infective endocarditis, is found in mild oral infections.[50]

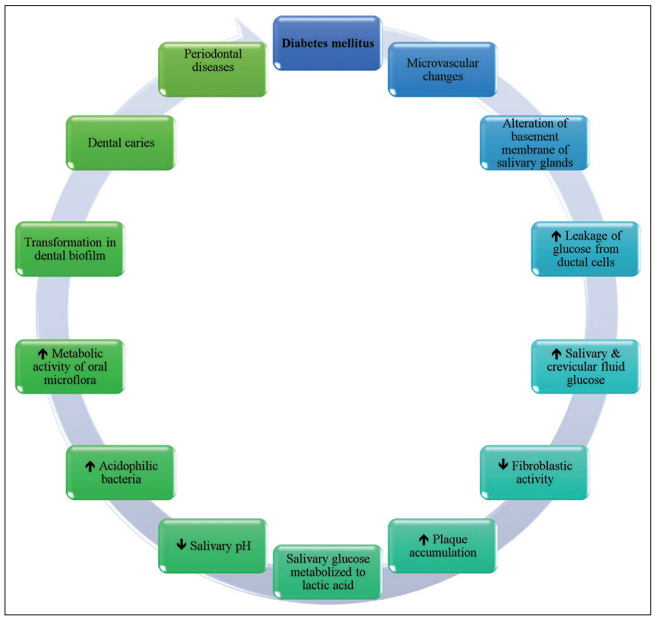

Figure 3: Depiction of the direct association between diabetes mellitus, caries, and periodontal diseases

The bacteria flourish due to elevated salivary glucose levels of the diabetics. *Propionibacterium acnes, Fusobacterium nucleatum, Saccharomyces cerevisiae, Actinomyces israelii, Streptococcus sanguis, Peptostreptococcus prevotii, Prevotella intermedia, P. gingivalis, and Streptococcus intermedius* are the most commonly detected bacteria in oral infections of diabetics.[19]

Fungal infections

Diabetics are more susceptible to acquire abscesses and infections in the mouth which can eventually impair glycemic control. The likeliness to buccal infections including candidiasis is enhanced due to hyperglycemia, reduced salivary flow, and altered salivary composition. Oral and pharyngeal candidiasis is one of the most commonly occurring opportunistic infections caused by *Candida albicans* found in diabetics due to impaired immunological resistance.[51] Basically, this fungus colonizes the human body normally in warm and moist areas, i.e., rectum, diaper region of children, mouth, and vagina; but, it becomes harmful and can cause infections when its amount is increased in the body. Most common risk factors for candidiasis include immunologically compromised states (DM, cancers, or HIV/AIDS), wearing of dentures at night without any proper oral as well as prosthesis hygiene, prolonged consumption of broad-spectrum antibiotics like tetracycline, and hyposalivation (reduced quality and quantity of saliva). Oral lesions that are associated with candidiasis are median rhomboid glossitis, angular cheilitis, denture-induced stomatitis, atrophic glossitis, and acute pseudomembranous candidiasis (oral thrush).[52]

The probability of being colonized by the yeast is higher in diabetics who smoke cigarettes.[51] The chances of having oral candidiasis in diabetics are significantly less in dentate patients as compared to them who use dentures. A level of hemoglobin A1c >12% is a potent predictor oral infections caused by fungi, regardless of denture usage.[28] In other words, since the immune system is impaired, and ultimately reducing chemotaxis and phagocytosis, the risk of acquiring other kinds of infections is increased as well. These situations lead to alleviated aggression of polymorphonuclear cells against bacterial attack and consequently heighten the tendency of gaining infections not only in the oral cavity but also in the whole body.[53]

Halitosis

Halitosis (also known as fetor oris, oral malodor, or bad breath) should never be ignored. Halitosis with a typical ketonic smell is one of the earliest symptoms of diabetes. Another vital signal is the odor of volatile sulfide compounds that signifies the existence of periodontal disease and/or tongue's coat. Currently, oral odors are thoroughly being investigated for diagnosis purposes. There is a specific smell in the breath of type 1 diabetics that can be indirectly utilized to check the levels of glucose in blood. This smell is because of oxidative stress that is a result of elevated levels of methyl nitrate and fatty acids in bloodstream.[8]

Tongue abnormalities

After periodontal tissues, the mucosa of the tongue is the most commonly affected region of oral cavity. Normally, tongue has a textured light pink-colored appearance due to equal distribution of fungiform and filiform papilla on the dorsal surface of the tongue. Its lateral and ventral surfaces are smooth, papilla-free, dark pinkish in color, and infrequently having prominent veins. Fissuring of tongue is a usual repercussion of type 1 DM on the oral cavity.[9]

Fissured tongue is characterized by interruption of smooth textured dorsal surface of tongue with one or more fissures which are principally adjusted along the length of the tongue. Fissuring develops as a consequence of both reduced quantity and quality of saliva.[54]

Bald tongue is also frequently found in diabetics. In this condition, the papillae of tongue are atrophied completely or in patches.[54] Focal sites of atrophy may signify candida infection.[55]

Another abnormality of tongue associated with DM is geographic tongue also known as benign migratory glossitis.[56] As the name illustrates, it is a benign condition characterized by an area of erythema (redness), with atrophied filiform papillae, fenced by a white, serpiginous, hyperkeratotic border.[57] The patient may experience discomfort, itching, and burning sensation of mucosa.[55]

One of the most significant oral manifestations of DM involving tongue is median rhomboid glossitis. It is basically a subgroup of erythematous candidiasis that is characterized by atrophic and erythematous lesion on the dorsal surface of tongue right in front of circumvallate papillae. Typically, the lesion is oval or rhomboid shaped and is present in the midline of dorsum

of the tongue. The lesion is usually depapillated, symmetrical, and well demarcated, having a smooth shiny surface. In some instances, an approximating erythematous lesion is developed on the palate due to frequent touching of the tongue with palate. Such a lesion is referred to as a kissing lesion.[58]

Taste disturbance

Taste is a pivotal element of oral health that is influenced adversely in diabetics. Several studies show that the ability to distinguish and perceive different tastes is diminished in type 1 as well as type 2 DM.[10,59] One study revealed that >33% of adults suffering from diabetes experience hypogeusia or reduced ability to taste things, which could eventually cause obesity and hyperphagia.

Approximately 3% of type 1 diabetics and 5% of type 2 diabetics have been found with ageusia or inability to detect tastes.[59] In newly diagnosed type 2 diabetics, impairment of taste for salt and glucose is partially reversed after resolution of hyperglycemia. This resolution of hyperglycemia with the help of oral hypoglycemic agnets and diet clearly suggests an association between taste perception and blood glucose concentration.[60] Furthermore, diabetics without peripheral and/or autonomic neuropathy have also been seen with the problem of taste impairment.[61]

Oral lichen planus and lichenoid drug reactions

Oral lichen planus (OLP) is a dermal disorder that is characterized by production of lesions in the oral cavity. White oral mucosal areas that do not wipe off is considered as a sign of OLP.[56] OLP is reported to be more frequently common in type 1 diabetics compared to type 2 diabetics.[11] The main reason is that type 1 diabetes is regarded as an autoimmune disorder, and OLP is known to have fundamental autoimmune mechanism.[29]

When DM is correlated with hypertension and OLP, it is termed as Grinspan syndrome. This interesting association was found by Grinspan in 1963.[40] Basically, it is a triad of OLP, hypertension, and DM. There is no direct role of DM and hypertension in etiology of OLP. However, related mucosal alterations known as lichenoid drug reactions develop as an adverse effect to medications that diabetics are usually prescribed. These medications are antihypertensive and antihyperglycemic.[62] The chief symptoms associated with lichen planus or lichenoid reactions include pain or discomfort, burning sensation of oral mucosa, and increased sensitivity to acidic foods.[55] They are linked with an escalated probability for dysplastic or malignant transformation. However, it is mandatory to refer such patients to a specialized dental surgeon for proper evaluation.[16]

Dry socket

Dry socket or alveolar osteitis is a postoperative complication that happens due to dislodgment of blood clot formed after extraction. It frequently occurs after extraction of mandibular teeth because of alleviated blood supply to the mandible. The main reason for reduced blood supply is atherosclerosis resulting from longstanding diabetes.[12] Hence, the use of epinephrine in local anesthetics is discouraged because it further lessens the blood supply to the target site, thereby escalating the probability of dry socket.[63]

Neurosensory oral disorder

Burning mouth syndrome (BMS) is seen in small percentage of diabetics.[13] It is an orofacial neurosensory disorder having an unknown etiology featured by burning sensation that is bilateral in nature and without the presence of any laboratory and clinical findings.[6] It is characterized by a burning sensation that initiates in tongue and slowly disseminates throughout the whole oral cavity; feeling of pain, tingling, or paresthesia can also be perceived palate, throat, lips, or gingiva. Mostly, it becomes almost impossible to identify one single cause. Systemic conditions that are related to burning mouth are thyroid dysfunctions, Sjögren's syndrome, DM, anxiety and stress, deficiencies of minerals such as zinc and iron, scarcity of vitamin B complex, and some infectious conditions as a result of candidiasis.[28] Long-lasting oral dysesthesias may also be experienced by diabetics that have devastating implications on the maintenance of oral hygiene. Peripheral neuropathies also have repercussions on oral cavity. It may hinder the patient to use equipment for maintaining proper oral hygiene. Neuropathies such as retinopathy could be a cause of blindness which ultimately have a direct impact on daily prosthesis as well as oral hygiene. Altered coordination, speed, and strength of cranial nerve musculature could cause dysphagia as well.[27] Figure 4 depicts the possible association between DM and BMS.

Future Prospects

Given the magnitude of the problem, oral repercussions of DM are grave concern for the public health all over the world.

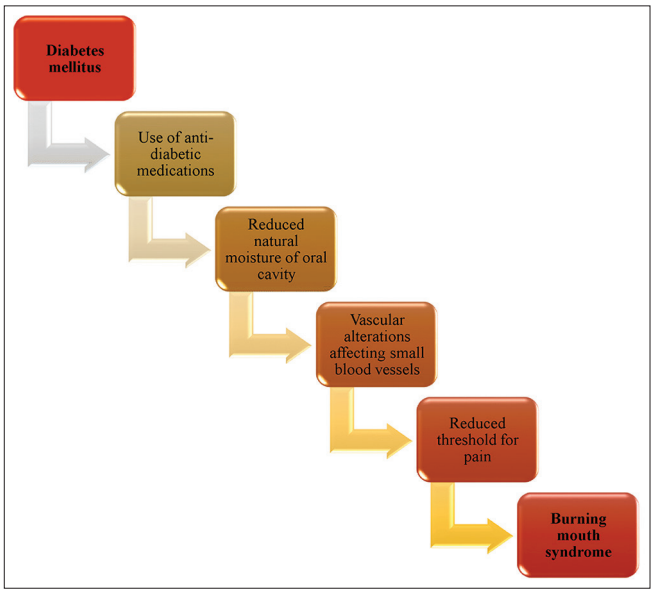

Figure 4: The possible association between diabetes mellitus and burning mouth syndrome

Their effect on individuals as well as communities due to suffering and pain, alleviated quality of life, and inability to function properly they inflict, is substantial. An extensive and exhaustive research is being done on this very topic of concern worldwide as some influential breakthroughs have been made but there is a dire need to transform these researches into practical and clinical application.

The major shortcoming related to this issue is that most of the diabetics are unaware of the fact that diabetes can also have negative impacts on their oral cavity which in turn can affect their glycemic control. Hence, basically, it is a vicious cycle. DM and oral disorders are interrelated and either of them could have grave consequences on the body. Here, we should spread more awareness among diabetics that the role of a dentist is equally as important as that of a physician. Health care providers associated with medicine and dentistry should coordinate with each other to combat this fatal duo.

Diabetics have a peculiar odor in their breath that can be helpful in detecting blood glucose levels. This seems quite promising since in this manner, diabetics could have a modernistic approach to evaluate their blood glucose levels in the near future. Moreover, diabetics have elevated levels of ketone in their breath when compared with control patients, even in normoglycemic conditions. This could be utilized to indirectly assess levels of glucose in blood, through breath ketone levels. Even very low levels of ketone bodies present in the breath can also be detected by a device and could be utilized when the diagnosis of DM is suspicious. These contemporary noninvasive techniques that can assess glucose levels of blood through ketonic constituent of the breath will be optimistically available for diagnosis and control of DM as well as curbing the menace of its oral manifestations.

Conclusion

DM and oral disorders go hand in hand. Diabetes affect almost every age group, and undoubtedly, its prevalence has been hiked due to, but not limited to, lifestyle alterations, increased life expectancy, etc. Oral cavity is one of the major sites of the human body where diabetes can unleash devastation. Oral complications of DM are almost inevitable, but their incidence and severity can be lessened by regularly visiting physician and dentist. The role of a dentist should never be underestimated or ignored in such instances as he/she plays a crucial role in preventing as well as management of oral disorders associated with DM. As the literature suggests, the risk of developing oral manifestations is greater in diabetics who have poor glycemic control as compared to those who have well-controlled blood glucose levels. Prevention and management of oral disorders, especially periodontitis and gingivitis, in diabetics is pivotal because they can have negative effect on glycemic control.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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