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Oral complications of diabetes mellitus and their underlying pathogenic mechanisms: a narrative review

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Abstract

Diabetes mellitus (DM) is a syndrome of abnormal carbohydrate metabolism causing tremendous mortality and morbidly worldwide. To date, there is no permanent curative treatment for diabetes and the patients have to rely on modification of their lifestyle and on the available timely medication. Understanding pathophysiology and complications of DM is crucial for clinicians and dental care providers in order to provide a proper management. Complications of DM are multisystemic with inevitable involvement of oral cavity. Diabetic patients have increased frequency of periodontitis, xerostomia, tooth loss, delay in wound healing, and impaired response to infection. Dental complications of DM exert medical, psychological, economical and national burden. This narrative review aims to outline the oral complications of DM and their underlying pathogenic mechanisms in the recent literature. The review concluded that; the commonest oral complications of DM are xerostomia, tooth decay, periodontal disease and gingivitis, oral candidiasis, altered taste sensation, oral mucosa alterations, and delayed wound healing. The important underlying pathogenic mechanisms include oxidative stress, alterations in salivary amylase protein, high level of sodium-glucose cotransporter 1 (SGLT1) protein, impaired neural structure and function, use of sodium glucose transporter 2 (SGLT2) inhibitor, the influence of the IL-23R gene polymorphism, and formation of advanced glycation end products (AGEP).

Keywords: *diabetes mellitus, oral complications, pathogenic mechanisms*

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Introduction

Diabetes mellitus (DM) is a syndrome of abnormal carbohydrate metabolism that is characterised by hyperglycaemia associated with a relative or absolute impairment of insulin secretion in addition to a variable degree of insulin resistance. It is one of the top 10 causes of death worldwide caused 4 million deaths in 2017 (Ismail *et al*., 2021). To date, there is no permanent curative treatment for diabetes and the patients have to rely on modification of their lifestyle and on the available timely medication.

Diabetes is one of the four major non-communicable diseases (NCD) causing devastating burden on health, economy, productivity, and on social life. According to the World Health Organisation's (WHO) latest statistics, DM caused death of 2 million people worldwide in 2019. Moreover, deaths due to DM increased by 3% in the period

between 2000 and 2019, in comparison to the other causes of NCD (WHO, 2023). Globally, around 463 million people live with diabetes (Ministry of Health Malaysia, 2020). In Malaysia, it is estimated that 3.9 million (18.3%) of the adult population had raised blood sugar in 2019 (Ministry of Health Malaysia, 2020). The incidence of type-1 DM alone, has been increasing globally since the 1950s, with an average annual increase of 3–4% over the past three decades (Akil *et al*., 2021). The prevalence of type-2 DM alone in Malaysia, was estimated to be 14.39%, which is considered among the highest figures in Asia. On the other hand, the prevalence of prediabetes was 11.62%, indicating the expected trend of diabetes in Malaysia will be increasing (Akhtar *et al*., 2022).

Diabetes mellitus has been classified into different subtypes with the most common one in adult population is called as type-2 DM representing more than 90% of overall cases of DM. On the other hand, the commonest type of DM affecting children is type 1 (Elsayed *et al*., 2023).

According to the American Diabetes Association (ADA), diabetes can be classified into four main categories (Figure 1) (Chiang *et al*., 2014; Elsayed *et al*., 2023)

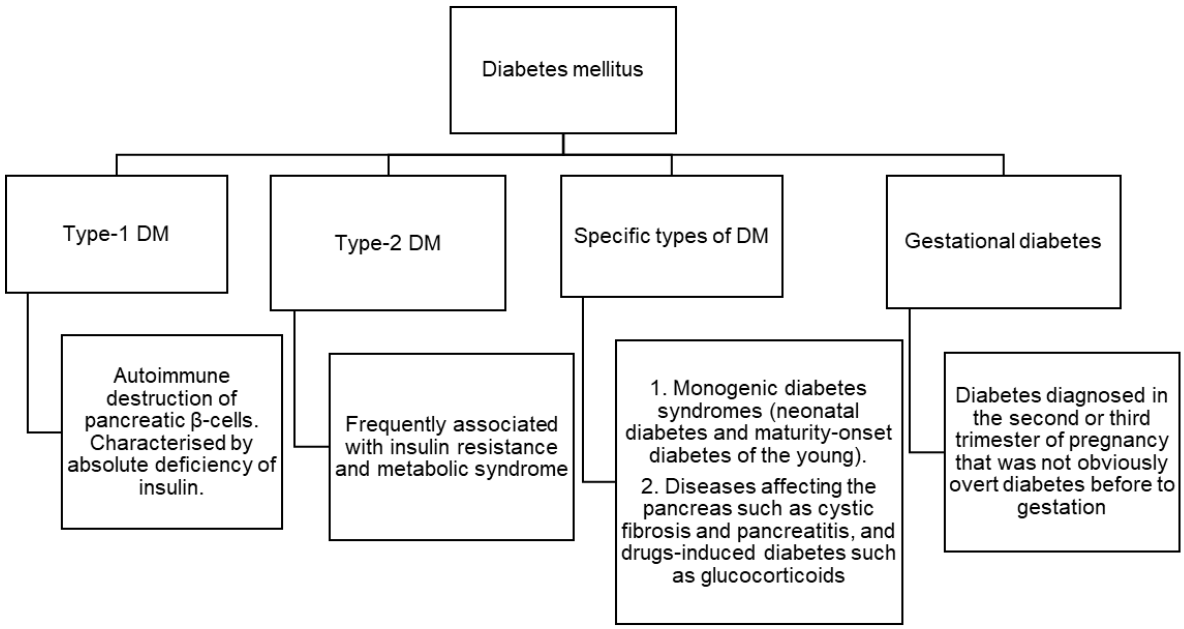

Figure 1. Main categories of diabetes mellitus.

Understanding pathophysiology and complications of each type of DM is crucial for clinicians including dental care providers in order to provide a proper management (Busa *et al*., 2022). However, it is important for stakeholders to consider the following fact during management of DM: "poor control of diabetes will end up with systemic complications regardless the type of DM".

In type-2 DM, the underlying pathogenesis of the onset of diabetes is attributed to a progressive defect in insulin secretion from β-cells on the background of insulin resistance owing to an interaction between environmental and genetic factors (Bellou *et al*., 2018). The pathogenic mechanisms are attributed to an interaction of multiple factors such as lifestyle, medical condition, hereditary, psychosocial and demographic risk factors such as high-level serum uric acid, sleep quality/quantity, smoking, depression, cardiovascular disease, dyslipidaemia, hypertension, aging, ethnicity, family history of diabetes, physical inactivity, and obesity (Ismail *et al*., 2021). However, the pathogenic mechanism behind type-1 DM is a multifactorial autoimmune

destruction of pancreatic β cells, resulting in a deficiency of insulin synthesis and secretion (Akil *et al*., 2021). Complications of DM are multisystemic, and manifested clinically as peripheral neuropathy, peripheral vascular disease, nephropathy, retinopathy, cardiac and cerebrovascular vasculopathy. However, complications of DM are not limited to these body systems (Tomic *et al*., 2022). Impact of diabetes on oral health represents another serious complication of diabetes. Patients with diabetes found to have a higher chance of developing oral mucosal lesions when compared to healthy individuals (Kataria Rinki *et al*., 2019). Diabetic patients have increased frequency of periodontitis, xerostomia, tooth loss, delay in wound healing, and impaired response to infection (Velasco-Ortega *et al*., 2016).

Dental complications of DM exert medical, psychological, economical and national burden. Dental professionals in particular and public health sector in general, are advised to be aware of the inter-relationship between diabetes and oral health. One of each ten diabetic patients found to be affected by periodontitis. Which represents a considerable percentage to alert the diabetes stakeholders (Genco & Borgnakke, 2020). Nazir *et al*. (2018), reported that more than 90% of diabetics have evidence of oral manifestations (Nazir *et al*., 2018). This review will highlight the important oral complications of diabetes and aims to emphasise the underlying pathogenic mechanisms for their development.

Diabetic oral complications and their pathogenic mechanisms

A vast number of oral complications have been reported to be associated with diabetes mellitus, involving both soft as well as hard tissues of the oral cavity (Figure 2). The reported oral manifestations linked to DM include xerostomia, loose tooth and tooth decay, periodontal disease and gingivitis, oral candidiasis and mucormycosis, burning mouth syndrome, altered taste sensation, aspergillosis, oral lichen planus, geographic tongue and fissured tongue, delayed wound healing, and increased incidence of infection, salivary dysfunction, oral neurosensory disorders, impaired tooth eruption, and benign parotid hypertrophy (Rohani, 2019).

Xerostomia

Xerostomia is defined as the subjective feeling of a dry mouth due to dysfunctional involvement of the salivary glands (Kim, 2023). Saliva contributes to oral homeostasis through lubricating and protecting oral mucosa, in addition to its role in the early stages of digestion (Fouani *et al.,* 2021). Diabetes mellitus is a well-known cause of salivary glands dysfunction.

In diabetes, insulin deficiency is associated with hyperglycaemia, which consequently results in osmotic diuresis and loss of considerable fluid volume. These pathophysiological changes will result in a status of hyposalivation (Bhat *et al*., 2021). The underlying pathology in diabetic salivary dysfunction involves, recurrent microbial infection of salivary glands, oxidative stress, alterations in salivary amylase protein, high level of sodium-glucose cotransporter 1 (SGLT1) protein which plays a key role in xerostomia through salivary water reabsorption in salivary ducts and consequently reduces salivary flow, and reduction in nitric oxide which is required for salivary secretion (Fouani *et al*., 2021).

Oral mucosal alteration and taste dysfunction

Taste is transmitted by taste pathways from taste buds located in the mucous membrane of tongue, epiglottis, soft palate, and pharynx to the sensory cortex of the brain. Taste impairment (dysgeusia) is alteration of this normal gustatory function that may result in complete taste losses (ageusia), partial reductions (hypogeusia), or over-acuteness of the sense of taste (hypergeusia). On the other hand, taste impairments are not life-threatening conditions, but they can cause sufficient discomfort and lead to appetite

loss and changes in eating habits, with effects on general health (Risso *et al*., 2020). Approximately 3% of Type-1 DM and 5% of Type-2 DM have been found with ageusia or inability to detect tastes (Ahmad *et al*., 2019). The taste function is dependent on special sensory receptors located in the taste buds of the tongue and mediated by the chemosensory gustatory system (Sjöstrand *et al*., 2021). Furthermore, many diabetic patients showed numbness and tingling sensation of the tongue due to neuropathy (Kumari & Gnanasundaram, 2021). Neuropathy affects the nerves of the taste buds will results in an altered taste sensation. Diabetic neuropathy is a devastating chronic complication, and the most emphasized diabetic neuropathy is the peripheral symmetrical polyneuropathy (Smith *et al*., 2022). Diminished taste perception has been linked to the increased tendency toward consumption of beverages with a higher sweet taste intensity which could increase risk of obesity and its complications (Verhulst *et al*., 2019). Taste dysfunction has been reported to occur more frequently in patients with poorly controlled diabetes compared to health controls (Khan, 2018).

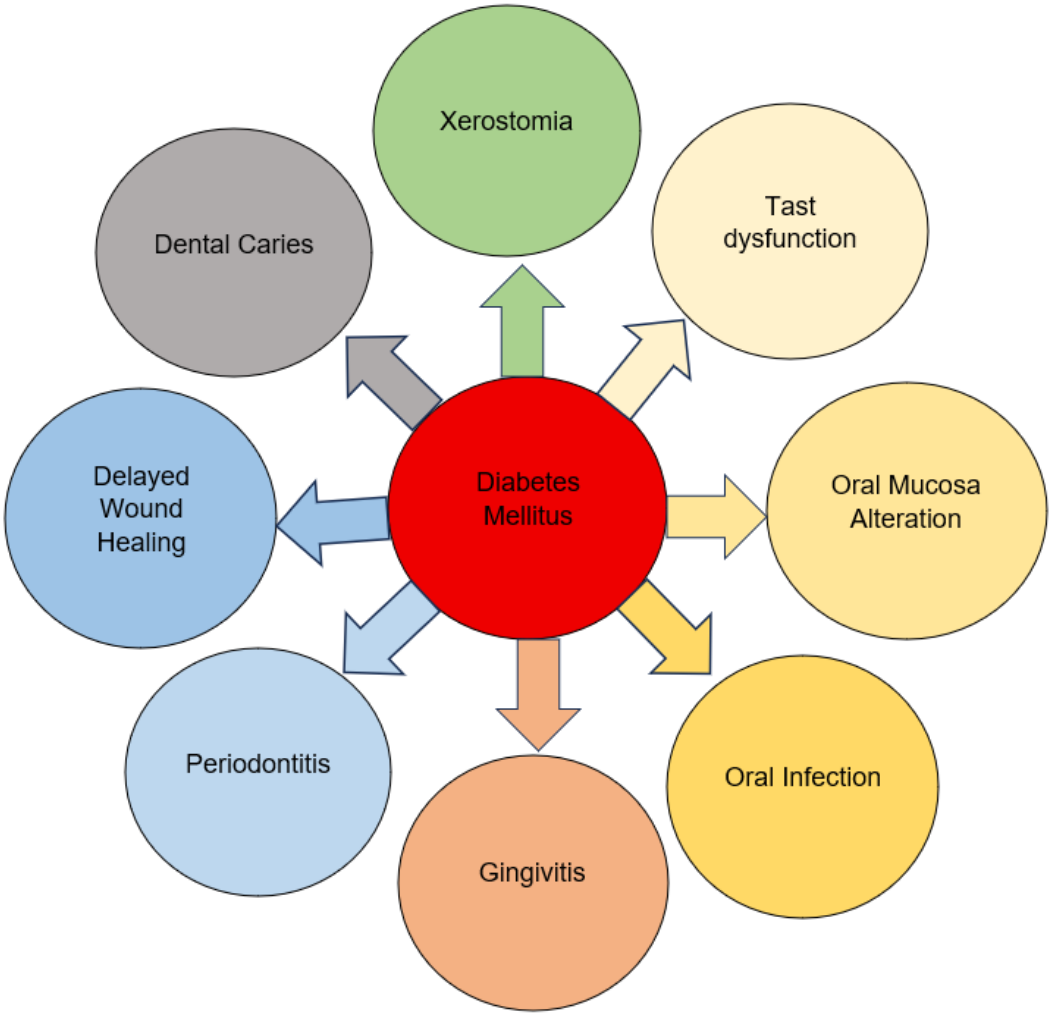

Figure 2. Common oral complications of diabetes mellitus.

Kushwaha *et al*. (2022) reported a significant alteration of taste sensation in association with elevated level of fasting blood glucose and HbA1c in patients with type-2 DM with regard to sense of sweet foods. This finding may be attributed to the

pathogenic effect of DM on the neural structure and perhaps on neural function. On the contrary, Nettore et al. (2024) showed that in type-1 DM the reduced taste modalities were sour, salt, and bitter, but the sweet modality was not affected. These interesting findings may indicate the difference in the natural history of taste impairment and pathogenesis in the common two types of DM.

Oral infections

The incidence of oral infection is high in diabetic patients owing to the impaired physiological defence mechanisms (Bhat *et al*., 2021). Pathogenesis of oral bacterial infection is initiated by bacterial irritation of the periodontal tissue; this will bring about a defensive reaction through recruitment of inflammatory cells and subsequent release of various inflammatory mediators to destroy the bacteria. This defence mechanism against bacterial infection will be accompanied by increase in vascularization, vascular permeability, in addition to increased number of inflammatory cells in gingiva cause swelling, redness, and bleeding (Gürsoy *et al*., 2024).

Diabetic patients have higher incidence of fungal oral infections. A strong association has been shown between oral candidiasis and diabetes mellitus in comparison to non-diabetics (Wijesuriya *et al*., 2024). Susceptibility of diabetic patients to oral candidiasis may be attributed to administration of sodium glucose transporter 2 (SGLT2) inhibitor and the influence of the IL-23R gene polymorphism (Alawya & Catartika, 2024).

Gingivitis and periodontitis

Periodontal tissues are commonly affected in diabetes mellitus. Periodontitis is defined as a chronic inflammatory disease of root cementum, gingiva, periodontal ligament, and alveolar bone, that is initiated and sustained by an aberrant host immune response against resident bacterial biofilm on the teeth (Teeuw *et al*., 2017). Periodontitis is not a solely local oral disease, but it has been considered also as a manifestation of systemic diseases like diabetes mellitus (Mainas *et al*., 2023). Gingivitis in cases of DM develops due to dryness of the mouth and failure of the antibacterial function of saliva. In cases of DM, microbial induced gingivitis will not regress if the patient is subjected to dental management like scaling and polishing, it will improve only by therapeutic control of DM. Furthermore, untreated gingivitis in patients with uncontrolled DM eventually will end up with periodontitis (Kumari & Gnanasundaram, 2021).

Diabetic patients have a higher risk of approximately three to four times of developing periodontitis in comparison to the healthy population (Turner & Bouloux, 2023). Prevalence of periodontitis in diabetic patients in comparison to non-diabetics found to be 59.6% to 39% respectively (Rohani, 2019). Disturbed glycaemic control is the main leading cause for development and progression of periodontal disease. Although the pathophysiological relationship between diabetes and periodontal disease still under investigations, there are some pathogenic mechanisms indicated the link between the two diseases. Chronic hyperglycaemia is characterised by formation of advanced glycation end products (AGEP), that trigger and promote inflammatory response involving overproduction of cytokines, tumour necrosis factor alpha (TNF-α), and C-reactive protein (Turner & Bouloux, 2023). On the other hand, active periodontitis is characterized by impaired release of inflammatory mediators such as interleukins IL-1β and IL-6, TNF-α, prostaglandin E2 (PGE2), metalloproteinases, adipokines, and chemokines. It has been concluded that there is an interplay pathogenic mechanism (Figure 3) between diabetes and periodontal disease. Poor glycaemic control found to be associated with progression of periodontitis, and untreated periodontitis is associated with refractory control of diabetes (Santonocito *et al*., 2022).

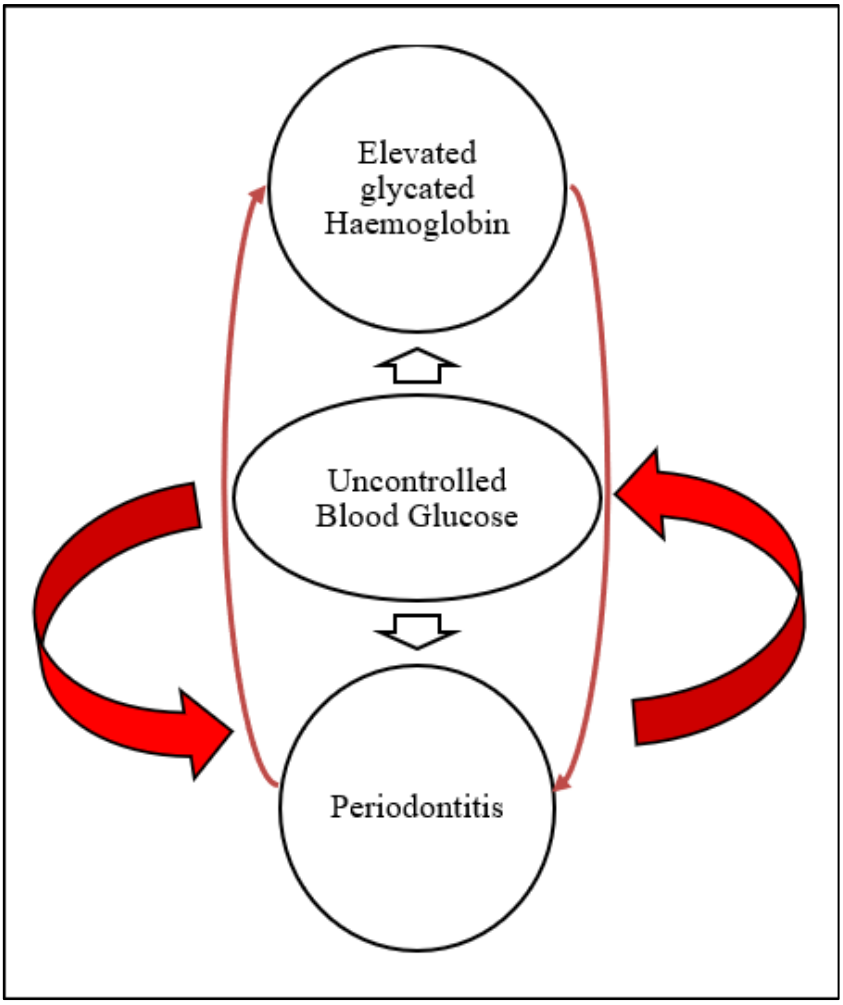

Figure 3. Proposed pathogenic interplay mechanism between glycaemic status and periodontitis.

Delayed wound healing

Healing is the body's response to injury to restore normal structure and function. Poor soft tissue regeneration and delayed osseous healing in patients with diabetes are well-known complications of oral surgery. The long-term complications of diabetes occur due to damage to the micro-vessels nourishing the tissues and organs of the body. The body's ability to heal a wound depends on a healthy blood supply which is needed to deliver nutrients to the cells that provide the protective defence against infection and those that cause inflammation. High blood sugar in the setting of DM is associated with paralysis of inflammatory cells and impaired tissue defences. Additionally, insufficient insulin level will further impair healing and regenerative functions in body tissues (Ahmad *et al.*, 2019). Elevated HbA1c above 6.5%, significantly increase the risk of infections after surgical dental interventions and increases the risk of delayed wound healing. For this reason, it is advisable to obtain better control of glycosylated haemoglobin figures. However, in the case when there is an ongoing periodontal pathological process which are suspected to contribute further to a poorer diabetic control, and at the same time there is a need for surgical intervention, the treatment should not be delayed in order to achieve better metabolic control of the disease. In these cases, post-surgical wound care should be maximized, and clinical considerations should be made on the convenience of using antibiotics in each specific case (González- Moles & Ramos-García, 2021).

Dental caries

The association between DM and dental caries has been investigated by a vast number of clinical studies. However, the pathogenic mechanisms remained complicated. The prevalence of dental caries in diabetic patients found to be not different between type-1 and type-2 DM (Bhat *et al*., 2021). Saliva is responsible for oral pH homeostasis (G. Dipalma, 2023), however, hyperglycaemia is accompanied by elevated glucose level in the saliva (Cui *et al*., 2022). Reduction of oral pH following glucose metabolism by oral microbiota will create a cariogenic environment causing demineralization of tooth enamel and dentin (Mbembela *et al*., 2023). Diabetes found to alter the overall function of salivary glands contributing not only to the development and progression of dental caries through creating acidic environment but by inducing a significant deterioration in the overall function of the salivary glands through deregulation of several key-salivary proteins such as: sodium-glucose cotransporter-1 (SGLT1), aquaporin 5 (AQP 5), and nitric oxide synthase (Fouani *et al*., 2021).

Conclusion

Oral complications of DM are a major concern for the patients, dental health care providers, and related stakeholders, exerting multifaceted burdens. Some oral complications associated with DM have a known pathogenic mechanism; however, many others need to be explored.

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