Editorial

Oral health and diabetes

Diabetes is a heterogeneous metabolic disorder characterized by insulin deficiency or insulin resistance or both leading to dysglycemia of various orders and its consequences. In 2021, approximately 537 million adults between age-group of 20–79 years are living with diabetes and are projected to rise to 643 million by 2030 and 783 million by 2045. Thus, one in 10 adults (10.5%) around the world are living with diabetes.[1] Since diabetes is a consequence of metabolic disease which principally regulates energy generation by glucose metabolism (glycolysis and Krebs cycle), insulin resistance and deficiency affect those cells whose turnover is high and having short cell cycle for few days to weeks like ectodermal cells (7 hrs–24 hrs), neutrophils (1-5 days), and epithelial cells of mucous membrane is 14–15 days. Stomodeum is created by invagination of ectoderm which forms primitive mouth it has all the metabolic memory of primitive ectodermal embryonic cells and responds equally to glycemic changes in diabetes. Historically, polydipsia is the first hyperosmolar symptom perceived by diabetics followed by dryness of mouth which has implications in decreasing the level of salivation and affecting the oral defense mechanism.

Saliva is a natural defense mechanism to fight against entry of bacteria through the oral route. Salivary glands are acinar gland which secretes isotonic salivary fluid which becomes hypotonic as it advances down below. Ptyalin, an enzyme present in saliva, converts starch into dextrin and maltose to begin the first stage of carbohydrate digestion and metabolism.[2] Saliva also contains epidermal growth factor (EGF) and transforming growth factor alpha (TGF alpha) and has GLP-1R which plays significant role in the pathogenesis of type 2DM. There are evidences that in animal model, the salivary gland has potential to secrete insulin substrate receptor 1 (IRS1) and insulin receptor 2 (IRS2) in murine animal model. High level of SGLT 1 protein has also been found in saliva and in xerostomia with diabetes.[3]

SALIVARY GLAND PROTEIN EXPRESSION IN DIABETES

Salivary amylase protein present in the oral cavity plays a significant role in the pathogenesis of diabetes. In both type 1DM and type 2DM, its level is increased. Saliva's secretion is regulated through parasympathetic nerve, and often, this could be a joint manifestation of diabetic autonomic neuropathy and metabolic consequences of hyperosmolar symptoms. Decreased muscarinic receptors activity also degenerates acinar cells and decreases salivary secretion and salivary protein which are protective to the oral mucous membrane. In recent studies, heat shock protein 60 (Hsp60) has been found to be elevated and is associated with severe periodontal mucous membrane damage among diabetics. Streptozotocin in experimental model of the offspring's of streotozolocin-induced (STZ) female rats' alteration in fetal salivary gland, protein expression has been demonstrated in several genes involved in differentiation and cellular apoptosis of epidermal growth factor (EGF), cytokeratin 5, CK7, AQP5, and B-cell lymphoma-2 (Bcl2).[4]

HEALING OF DENTAL EXTRACTION WOUND IN DIABETES

Healing of oral wounds, specially dental extraction wound, is greatly altered in hyperglycemia.

Soft tissue proliferation and remodeling are mildly influenced by hyperglycemia, but it is the quality of formed tissue which is altered in hyperglycemia. AGE causes poor cross-linking of collagen fibers causing decreased wound strength. Alteration in calcium metabolism is responsible for poor osteoblastic and remodeling activity of alveolar bone.

EFFECT OF INSULIN RESISTANCE ON PERIODONTIUM AND DENTAL IMPLANTS

Periodontitis is related to biofilm which could harbor various microbes and promote formation of plaque. This results in expression of various pro-inflammatory cytokines like interleukin (IL) 1 alpha (IL-1 beta), IL-6 tumour necrosis factor (TNF)-alpha, PGE-2, and matrix metalloproteinases (MMPs). Cytokines liberated by periodontal tissues, e.g., macrophages, osteoclast, fibroblasts, and mast cells, amplify disease destructive process and change in oral flora of microbes and its toxins with lipopolysaccharide (LPS) and present a low-grade chronic

inflammatory state which produces insulin resistance. A bidirectional relationship between periodontal disease and diabetes has been proposed where pro-inflammatory cytokines expressed over gingival mucosa could enter in systemic circulation and deteriorate pre-existing diabetes or in long-term slow persistent chronic inflammatory state increases acute phase reactant like hs-CRP expression which in turn produces insulin resistance and leads to clinical manifestation of type 2DM. Similar pathophysiological events are seen in periodontium around the implants. It has been found that hyperglycemia is single independent factor for high incidence of peri-implantitis in type 2DM patients. Since neutrophil function is impaired and pro-inflammatory cytokines' level is raised, this further causes progression of peri-implantitis into deeper tissue and consequent bone resorption followed by implant failure.[5] The bone repair mechanism is also altered due to accumulation of advanced glycosylation end products (AGE) and generation of free radicals. The osteoblast density is greatly reduced at the healing site. These cellular events have crucial effects over implant osseointegration and pull-out strength of dental implant.

DIABETES AND MINOR ORAL SURGICAL PROCEDURES

The minor oral surgical procedures involving mucosa only can be performed safely in diabetics; however, precaution should be taken to keep checking the glycemic control till proper healing is achieved. For extensive procedures involving both the hard and soft tissues, pre-surgical control of glycemic level as well as over the healing period is mandatory. Experiments over animal models revealed that poor glycemic control not only alters proper healing but also can progress to chronic inflammation if proper control is not achieved. Necrosis and sepsis are late consequences of poor glycemic control.

ORAL DISEASES IN DIABETES WITH CARDIOVASCULAR RISK

Over many decades, clinically many observational studies have shown positive correlation between diabetes, periodontitis, and high cardiovascular mortality. The association of bacterial endocarditis and periodontal diseases has been found in several autopsies of landmark cardiovascular publication, but its association with coronary artery disease in diabetic patients is a recent observation. The real mechanism is yet to be established; however, bacteremia in a diabetic immunocompromised host has been often blamed as a causative factor. Bacteremia could also be the result of surgical and non-surgical dental procedures in a background of infective endocarditis. Periodontal organisms have potential to invade many organs including cardiovascular system. These periodontal organisms have been demonstrated in pericardial fluid of effusive pericarditis and in cardiac valve tissues of valvular heart disease. Periodontal pathogens can colonize the atherosclerotic plaque and invade arterial wall. Porphyromonas gingival which is a major pathogen in periodontal disease induces platelet aggregation and expression of cell adhesion molecules like vascular cell adhesion molecule 1 (VCAM), intracellular adhesion molecule 1 (ICAM), and p-selectin. In one of the recent publications, the prevalence of periodontitis with CVD was 54.3% and 30.9% with those having diabetes. Patients with DM and periodontal disease having cardiovascular disease tend to deteriorate much faster and develop complications early like heart failure, renal impairment, neuropathy, and cerebrovascular accidents.[6] To sum up, good oral health is important to prevent occurrence of diabetes and cardiovascular disorders, whereas in pre-existing diabetes and cardiovascular disorders, a good oral health helps to keep them in control.

Kamlakar Tripathi, Richik Tripathi[1]
Padma Shri Awardee 2022, Former Professor and Head, Department of Medicine, IMS, BHU, [1]Oral and Maxillofacial Surgery Unit, Faculty of Dental Sciences, IMS, BHU, Varanasi, Uttar Pradesh, India

Address for correspondence: Dr. Kamlakar Tripathi, Former Prof and Head, Department of Medicine, IMS, BHU, Varanasi, Uttar Pradesh, India.
E-mail: kamlakar.tripathi1951@gmail.com

Received: 01 November 2022,
Revised: 07 November 2022,
Accepted: 10 November 2022,
Published: 10 December 2022

REFERENCES

1. Diabetes -WHO. Available from: http://www.who.int. [2021 Nov 10].
2. Fouani M, Basset CA, Jurjus AR, Leone LG, Tomasello G, Leone A. Salivary gland proteins alterations in diabetic milieu. J Mol Histol 2021,52:893-904.
3. Sabino-Silva R, Okamoto MM, David Silva A, Mori RC, Freitas HS, Machado UF. Increased SGLT1 expression in salivary gland ductal cells correlates with hypersalivation in diabetic and hypertensive rats.

Diabetes Metab Syndr 2013;5:64.
4. Basset CA, Cappello F, Rappa F, Jurjus AR, Conway de Macario E, Macario AJ, *et al*. Chaperonin Hsp60 and Cancer Therapies. In: Asea A (a cura di), Heat Shock Proteins. Springer; 2020. p. 1-22. [10.1007/7515_2020_1].
5. Gurav AN. Periodontitis and Insulin resistance: Casual or causal relationship. Diabetes Mellitus J 2012;36:404-11.
6. Han SJ, Son YJ, Kim BH. Association between diabetes mellitus and oral health status in patients with cardiovascular disease: A nationwide population based study. Int J Environ Res Public Health 2021;18:4889.

Access this article online

Website: www.njms.in

DOI: 10.4103/njms.njms_197_22

Quick Response Code

How to cite this article: Tripathi K, Tripathi R. Oral health and diabetes. Natl J Maxillofac Surg 2022;13:319-21.