

## Review of Diabetes Mellitus effects on Peri-Implantitis

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### Abstract:

**Background:** Implant dentistry is one of the most suggested and practiced treatments for dental rehabilitation. High failure rates occur as a result of local and systemic risk factors. A chronic disease that causes many side effects is diabetes mellitus, with uncontrolled blood glucose. Diabetes being a relative contraindication for implant surgery is much debated. The prevalence of diagnosed diabetes increased to 10.5 % and most of those diabetic patients need for dental implants also has risen. We aimed to perform a systematic review to answer the subsequent questions: Does diabetes Mellitus is playing a major role in peri-implantitis.

**Materials and Methods:** Literature research is performed in sources of data like MEDLINE, online library journal PubMed NIH, NCBI, WILEY ONLINE LIBRARY, ORAL HEALTH GROUP, Scopus ETC. Electronic databases had been searched by the use of the subsequent key-words: periimplantitis, implants, diabetic and wound recovery diabetes-associated inflammation, glycosylated; hemoglobin A; hyperglycemia, gestational diabetes; etc. and Our research was carried out on articles published to date.

**Results:** After reviewing the titles and abstracts, many articles met the eligibility requirements. Diabetes Mellitus is a group of metabolic disorders characterized by chronic hyperglycemia resulting from defects within the secretion or action of insulin and perhaps DM type 1 or type 2 DM. It exerts an influence on the health of peri-implants, since it is responsible for increased inflammation, oxidative stress and cellular apoptosis leading to delays in healing. There are many factors associated with peri-implant changes, among which Diabetic Mellitus is one of the main significant risk factors.

**Key Word:** 'periimplantitis,' 'diabetic Mellitus', 'bone healing', 'inflammation', oral diseases with diabetes' and implant stability, implant survival rate, molecular analysis, hyperglycemia.

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### I. Introduction

The National Diabetes Statistics Report, a periodic publication of the Centers for Disease Control and Prevention (CDC), provides information on the prevalence and incidence of diabetes and prediabetes, risk factors for complications, acute and chronic infection.

These data can help focus efforts to prevent and control diabetes across the USA. It is an update to the 2017 National Diabetes Statistics Report and is intended for a scientific audience.[1]

### Diabetes

Total: 34.2 million people have diabetes (10.5% of the US population) Diagnosed: 26.9 million people, including 26.8 million adults Undiagnosed: 7.3 million people (21.4% are undiagnosed) **Prediabetes**

Total: 88 million people aged 18 years or older have prediabetes (34.5% of the adult US population) 65 years or older: 24.2 million people aged 65 years or older have prediabetes

Most studies in animals have demonstrated that DM adversely affects bone healing around implants. Some clinical studies have reported minor or no associations of DM with implant failures, while others have shown higher losses of dental implants and peri-implant diseases in diabetics than in non-diabetic subjects.[1]

The peri-implantitis is most accountable for the loss of dental implants, so we all know the DM interface within the disease process is essential to making protocols for prevention, treatment, and maintenance of peri-implant health. Furthermore, DM/hyperglycemia seems to be related to a high risk of peri-implantitis. However, this conclusion is predicated on a limited number of systematic reviews and first articles. the majority of systematic reviews presented are of weak quality.[1]

### II. Material and Methods

#### Inclusion criteria:

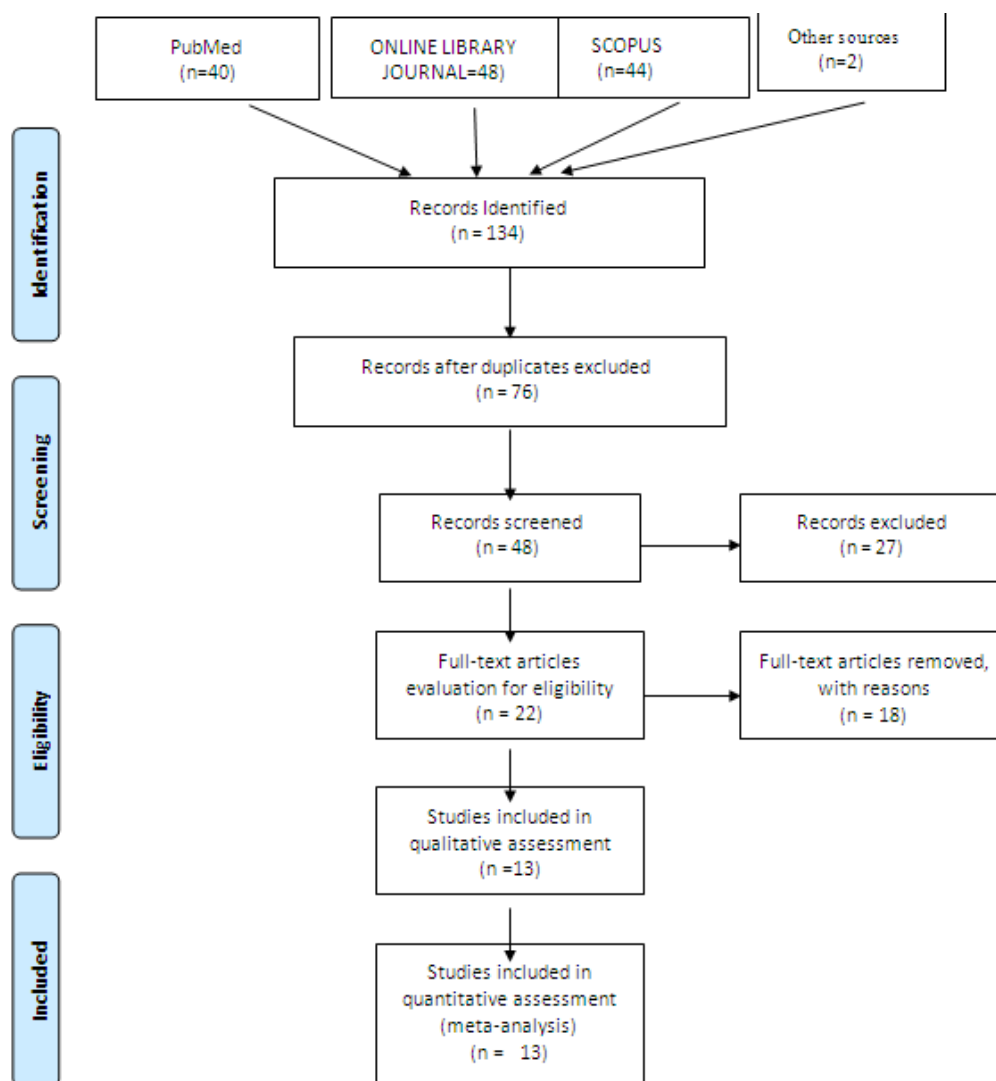
Systematic reviews, with or without meta-analyses, published in English from 2006 to 2020 and including studies, performed in humans, on the results of DM on early and late implant failures and complications.

**Exclusion criteria:**

Letters to the editor, personal opinions, book chapters, case reports, congress abstracts, studies with animals were excluded.

**Procedure methodology**

There are no pointers current to the topic of dental implants and diabetes mellitus. A total of 134 doubtlessly relevant titles and abstracts were found by way of the electronic search and additional assessment of reference lists. During the first screening, forty-eight publications had been excluded primarily based on the title and keywords. Additionally, 10 titles have been excluded primarily based on the abstract evaluation. 76 full-text articles had been thoroughly evaluated. A whole of fifty-eight papers had to be excluded at this stage because they did no longer fulfill the inclusion standards of the current systematic review. Thirteen articles went into qualitative evaluation (Fig. 1). Only research assessing diabetes as a risk factor causing the peri-implantitis was selected, irrespective of the guide date.



**III. Discussion**

Peri-implant diseases are inflammatory conditions affecting the soft and hard tissues around dental implants. Peri implant mucositis is a gingival inflammation, which occurs only around the soft tissues of the implant, with no evidence of bone loss. Generally, peri-implant mucositis could be a precursor to peri-implantitis. Evidence suggests that peri-implant mucositis is also successfully treated and is reversible if caught early. Peri-Implantitis is a destructive inflammatory process affecting the soft tissues and hard tissues surrounding the implant for retention is lost with time. [2]

A cross-sectional study was performed on 96 patients with 225 implants that were placed between 1998 and 2003. Peri implant mucositis occurred in 33% of implants and 48% of patients, while periimplantitis

occurred in 16% of implants and 26% of patients. 1 in 4 patients and 1 in 6 implants have a periimplantitis after 11 years. Evidence indicates that the periodontal and diabetic state of the patient is also useful in predicting implantation results[6].

Diabetes mellitus is a pro-inflammatory systemic condition and altered immune/microbiome reaction influences both catabolic and anabolic occasions of bone-healing that incorporate expanded osteoclast genesis and compromised osteoblast action, which can be clarified by the brokenness of affront receptor that's driven to the enactment of signals associated with osteoblast separation.

Literature has reported that up to 50% of dental implants could also be influenced by peri-implantitis, a bacteria- induced incessant provocative preparation, which advances osteoclast-mediated bone resorption and restrains bone arrangement, driving dynamic bone loss around implants. Current evidence focuses on an expanded hazard for the development of peri-implantitis DM (DM) conditions relative to the solid populace. Right now, there is no compelling treatment for peri-implantitis, and also the 50% predominance in DM, alongside its anticipated increment within the around the world populace, presents a significant concern in embed dentistry as hyperglycemic conditions are associated with the bone-healing disability; this might be through the brokenness of osteocalcin- induced glucose digestion system.

One of the potential clinical studies examined 10 non-diabetic individuals (12 implants) and 20 persons with type 2 diabetes (30 implants). Glycated hemoglobin (HbA1c) levels range was from 4.7-12.6%. Resonance frequency analysis was accustomed to assess the results. In patients with HbA1c  $\geq$  10.1%, only 57.1% of the implants returned to or exceeded baseline stability levels after 16 wks, compared with 80% or more for every of the opposite HbA1c groups. The potential for alterations in bone metabolism associated with increased glucose levels is according to the longitudinal assessments of implant stabilization found during this study.[3]

Another prospective 3 years study was done on 67 patients divided into four different groups that supported their HB1AC levels. This study shows increased levels of marginal bone loss around implant areas in patients with high levels of HB1AC. Peri implant probing showed bleeding values of 0.62 within the group with HBA1C more than 10, compared to 0.43 in subjects with HB1AC less than 6. Peri implant probing depth values didn't show statistically significant differences between the groups.[4]

A cross-sectional study done on the Brazilian population indicates a relative risk of peri-implantitis in patients with diabetes. The study group consisted of 212 partially edentulous patients rehabilitated with Osseointegrated implants. Radiographic and clinical assessment of peri-implant status showed the prevalence of peri-implant mucositis and peri-implantitis by 64.6% and 8.9%. Diabetes was statistically linked with an increased risk of peri-implantitis.[5]

A prospective cohort study was administered on a sample of 117 edentulous patients, each of whom received two mandibular implants, for a total of 234 implants. The results indicated that hyperglycemia in patients with type 2 diabetes showed no link with altered implant survival one year after loading. However, alterations in early bone healing and implant stability were related to elevated HbA1c levels.[7] A retrospective study was performed on 215 implants placed in 40 patients at 2 clinical centers. The results of this study depict an implant survival rate of 85.6% after 6 years in subjects with diabetes, which is less than a standard healthy population.[8]

Another study done shows how proinflammatory gene expression at the implant site is impacted by blood glucose levels.[9][10]

Dental implants in patients with diabetes show an improved prognosis if HbA1c levels are below 8.[11] Extensive genomic data from sequencing studies and microarray shows global gene-expression patterns like peri-implantitis and DM 2. A survey of those reviews and studies propose some resemblance within the molecular mechanisms indicated in both diseases. These include reactive oxygen species-related genes, inflammatory cytokines (Interleukin-1 $\beta$ , Interleukin-6), and vascular endothelial protein.

Study of transcriptomic data from diabetes type 2 and peri-implantitis discloses shared molecular linkages. Genes like interleukin 6(IL-6), nuclear factor-kappa B subunit 1 (NFkB1), Phosphatidylinositol-4,5-Bisphosphate 3- Kinase Catalytic Subunit Gamma (PIK3CG) and interleukin 17 (IL-17) present themselves as foremost contenders for shared molecular linkages.[12]

Patients with DM are likely to develop peripheral vascular disease leading to an absence of oxygen and nutrients supply to the implant site. Microangiopathies and lack of pericytes are commonly found within the diabetic capillary bed. Deceased response to hypoxia, decreased production of pro-angiogenic factors and an impaired receptor function are a number of the foremost significant causes of impaired wound healing at the implant site.

Macrophages, a vital system cell shows modified functions in diabetic patients. In hyperglycemic patients, they fail to shift to pro reparative phenotype from proinflammatory one. Hence fails to stimulate tissue healing. Hyperglycemia not just causes a decrease in pro-angiogenic factors but also alters antiangiogenic factors and capillary maturation factors in wounds. Production of antiangiogenic factors like PEDF (Pigment

epithelium-derived factor) at wound sites in diabetic patients may also result in implant failure. Platelet-derived protein (PDGF) may be a maturation factor that's seen to be altered in diabetic wound healing. A decrease within the population of endothelial progenitor cells (EPCs) from bone marrow lowers vascularity in diabetic patients.

#### IV. Conclusion

In conclusion, one of the most cause of implant failure is peri-implantitis. And it can also cause permanent tissue and bone loss. peri-implantitis is often caused by a bacterial infection, during the implant process – but diabetic patients are more prone to this condition than others because of hyperglycemia, oxidative stress and cell apoptosis, micro/macrovacular disease, increased TNF IL-6, dysfunctional cellular activity, poor wound healing, bone-related cell impairment, decreased host immunity resistance.

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