Effects of periodontitis on diabetes



A growing body of research suggests that periodontitis may contribute to the development of new cases of type 2 diabetes and possibly gestational diabetes (a type of diabetes that develops during pregnancy in individuals who did not previously have diabetes).

Research has shown that even in systemically healthy individuals (i.e. those without diabetes), periodontitis is associated with moderate increases in blood glucose levels compared to those with little or no periodontal disease. This link suggests that periodontitis could influence diabetes onset and glycaemic control in otherwise healthy individuals.

In addition, research examining the impact of periodontitis on blood glucose control in individuals with diabetes has revealed a progressive worsening in glycaemic control among those with both diabetes and periodontitis. It also indicates that patients with diabetes and periodontitis experience more severe complications than those with diabetes but minimal or no periodontal disease.

Mechanisms by which periodontitis negatively affects glycaemic control and complications in patients with diabetes

Periodontal disease is a chronic inflammatory condition associated with a dysbiotic subgingival biofilm. This biofilm triggers an inflammatory response characterised by the release of host-derived mediators including: interleukin-1b, interleukin-6, tumour necrosis factor-alpha (TNF-a), matrix metalloproteinases (MMPs) – particularly MMPs -8, -9 and -13, t-regulatory cytokines, and chemokines

This inflammation leads to bone resorption, mediated by factors such as prostaglandin E2, interleukin-17, and receptor activation of nuclear factor kappa-B ligand (RANKL) and osteoprotegerin (OPG). Combined, these mediators, inflammatory cells and cells essential for bone formation and maintenance (osteoblasts and osteoclasts), contribute to the local destruction of both the soft and hard tissues supporting the teeth.

Research indicates that these inflammatory mediators are also elevated systemically in individuals with periodontitis, and more so in those with co-existing periodontitis and diabetes. This systemic inflammation likely promotes insulin resistance, resulting in hyperglycaemia and the progression of diabetic complications. This may then further activate pathways that lead to the formation of advanced glycation end-products (AGEs), which bind to receptors (RAGEs), heightening inflammation, oxidative stress, and apoptosis (cell death). Therefore, the systemic inflammation associated with periodontitis may negatively impact blood glucose control and increase the risk of complications, especially microvascular complications.

References

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