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Assessment of Insulin Status in Periodontitis

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ABSTRACT

Periodontitis is the common chronic inflammatory disease. Diabetic patients are likely to develop chronic disease, which increases blood pressure and diabetic complications. 30 periodontitis patients and 30 healthy individuals from the OPD of Saveetha Dental College. Serum samples were analyzed for the parameters, FBS by GOD-POD Method using ERBA CHEM 5 plus analyzer. Insulin level was assessed by ELISA method using ROBONIK ELISA READER. Increase in FBS ($p<0.001$), Insulin ($P<0.001$) and Insulin Resistance IR ($p<0.005$) in periodontitis patients when compared with healthy controls. Our findings suggest that prolonged or profoundly affected periodontitis condition can develop a metabolic complication like diabetes, renal damage and other health problems.

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INTRODUCTION

Periodontal diseases are prevalent and can affect most of the worldwide population. Gingivitis, the mildest type of periodontal disease, is caused by the bacterial biofilm that accumulates on teeth adjacent to the gingiva (PihlstromBL *et al.,* 2005). The analysis of this disease is bacterial biofilm, which loads upon the teeth as dental plaque. In microorganisms, various factors such as environmental, systemic and genetics are responsible for periodontitis (Abhijit N Gaurav *et al.,* 2016). Periodontitis is a painless condition, so the subject looks for any professional assistance. If untreated it destroys tooth-supporting structures and which ends in the partial or complete loss of dentition (Armitage GC *et al.,* 2004). Insulin resistance (IR) is known as a constant and decreased level of inflammatory condition. It is correlated with altered glucose tolerance, hypertriglyceridemia and obesity. IR is showed by an increase in the levels of inflammatory cytokines like interleukin-1 and 6, tumour necrosis factor-α. These inflammatory cytokines play an essential role in the pathogenesis and progression of insulin resistance (Abhijit N. Gaurav *et al.,* 2012). In addition to IR increases inflammatory cytokines levels such as interleukin-1 and 6, tumour necrosis factor. These inflammatory cytokines play a crucial role in the progress of immunity and insulin resistance (Kelly M, Steele *et al.,* 1998). Clinical features of periodontitis include bleeding gums, interdental spacing, probing depth, awful oral breath and mobility of teeth in cutting edged cases (Chavarry NGM*et al.,* 2009). Cross-sectional found that the threat of periodontitis is approximately 3–4 times higher in people with diabetic patients than in non-diabetic subjects (Preshaw PM *et al.,* 2013). Periodontitis was found in 57.7% of T1DM patients and 15.7% in controls without diabetes (Popławska-Kita A *et al.,* 2014). Patients with T2DM were more prone to have a higher risk of severe forms of periodontitis when compared with non-diabetic subjects (Leite RS *et al.,* 2013). Diabetes and periodontitis are constant sicknesses that are biologically linked. Diabetes is one of the significant risk factors for periodontitis (Tsai C *et al.,* 2002).

METHODS AND MATERIALS

Patients were selected from those attending the outpatient department of Saveetha Dental College, and hospitals and divided into two groups as follows

Group I – Normal healthy individuals with normal BMI (19-24.9) – 20 individuals

Group II – Patients with Periodontitis – 20 individuals

Inclusion Criteria

Individuals with the age group of thirty-five to fifty-five.

Periodontitis Patients

Exclusion Criteria

Individuals with other systemic illness like diabetes mellitus, cardiovascular disease, Renal failure, Stroke, endocrine illness.Individuals with an acute illness like a fever.Immunocompromised individuals

Sample collection and Procedure

Informed consent was obtained from the patient before sample collection. 5ml of venous blood was collected and distributed in plain collection tubes and centrifuged in 3000rpm for 10mins. Then serum was separated and used to estimate the FBS by GOD-POD Method using ERBA CHEM 5 plus analyzer. Insulin level was assessed by ELISA method using ROBONIK ELISA READER.

Insulin resistance was calculated by using HOMA-IR calculation.

<1 indicates Optimal / No insulin resistance

>1.9 indicates Early Insulin Resistance

>2.9 indicates Significant Insulin Resistance

RESULTS AND DISCUSSION

Table 1: Data of Periodontitis patients

Parameters	Controls	Periodontitis patients	p-Value
FBS	86.35 ± 7.93	92.4 ± 7.12	<0.001
Insulin	3.48 ± 1.48	6.94 ± 1.43	<0.001
IR	0.76 ± 0.33	1.58 ± 0.34	<0.005

A dysregulated inflammatory response, involving immune response, neutrophil activity, and cytokine biology, is an essential pathologic mechanism linking diabetes and periodontitis (Jae Won Hong *et al.,* 2016). Periodontal infection can raise the systemic inflammation in turn, which may persuade the chronic state of insulin resistance, come up to the cycle of hyperglycemia and advanced glycation end product protein binding accumulation. Therefore, it can change the pathway of connective tissue degradation, destruction, and proliferation in diabetes (Demmer RT *et al.,* 2008). Patients with IGT (Impaired glucose tolerance) have normal blood glucose levels most of the time but become hyperglycemic after large glucose loads. On the other hand, patients with IFG (Impaired fasting glucose) have increased fasting blood glucose levels (Shoelson SE *et al.,* 2006).

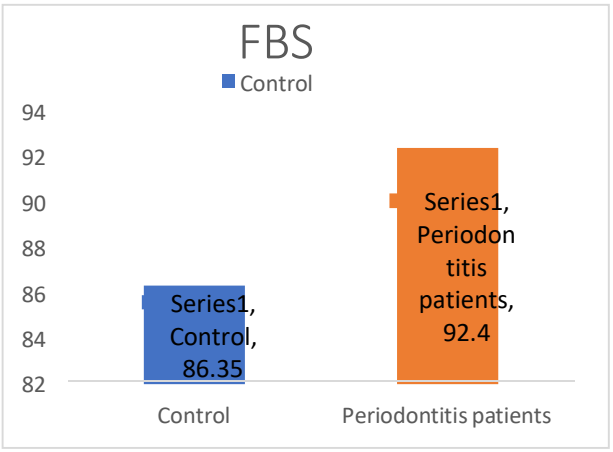

Figure 1: FBS on Periodontitis patients

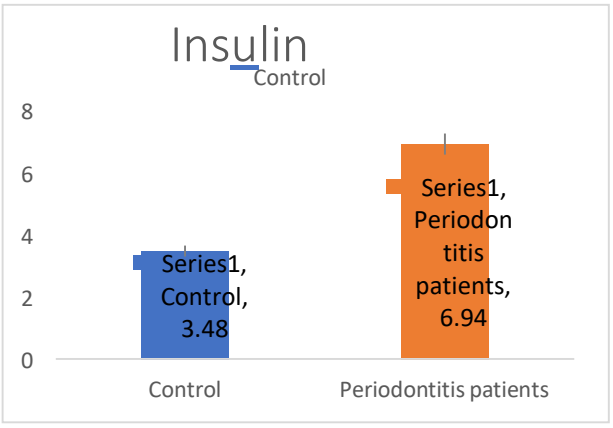

Figure 2: Insulin on Periodontitis patients

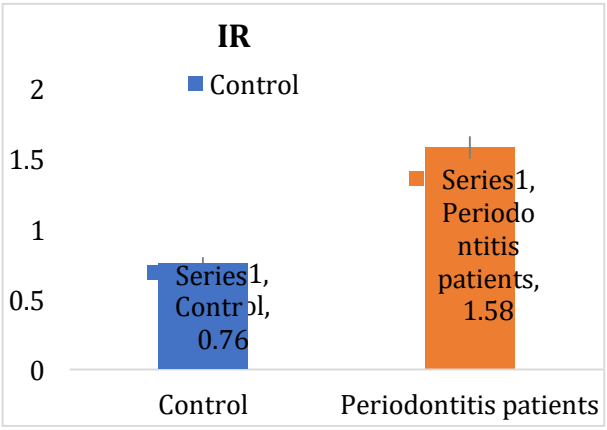

Figure 3: Insulin Resistance on Periodontitis patients

However, impaired glucose homeostasis results from chronic subclinical systemic inflammation, which is an early manifestation of type 2 diabetes. IL-6 and TNF-α are the primary inducers of acute-phase proteins and impair intracellular insulin signalling, potentially contributing to IR (Rotter V *et al.,* 2003). It has also been proposed that the link between periodontal infection and diabetes may be bidirectional. For example, an impaired immune response to dysbiotic subgingival biofilms among people with diabetes may contribute a severe inflammatory state and later both clinical periodontal disease as well as heightened insulin resistance and reduced glycemic control (Nidhi Arora *et al.,* 2014). This detection shows a relationship between hyperglycemia and periodontitis, with hyperglycemia increases the risk of periodontitis, and the periodontal disease affects the glycemic status (Preshaw PM *et al.,* 2012). They found a correlation of IL-21 with clinical attachment loss and borderline attention between plaque index and IL-21. As clinical attachment loss is the most valid indicator of periodontal tissue removal, this correlation indicates the finer way that IL-21 level increases with the severity of periodontitis (Archana Mootha *et al.,* 2016).

Glycemic status develops the prevalence and the extremity of periodontitis in diabetic participants. Many studies have clearly shown that inflammatory markers levels are higher in cases of periodontitis and increased levels of inflammatory markers causes' insulin resistance (Harshinee Chandrasekhar *et al.,* 2018). Current meta-analysis showed that individuals with type 2 diabetes and chronic periodontitis have significantly higher levels of IL-1β in gingival crevicular fluid (a fluid exudate that flows from the gingival margins) compared with systemically healthy individuals with chronic periodontitis (Atieh MA *et al.,* 2014). Evidence shows that insulin is associated with an increased prevalence and severity of periodontitis. It may raise the risk for worsening glycemic status and in insulin resistance patients as well as increases the risk for people with diabetes (Garcia D *et al.,* 2015).

CONCLUSION

In our studies, it shows that diabetes has a remarkable risk factor for immunity, while glycemic control is poor. Immunization and alveolar bone loss uncontrolled diabetes are considered as a severe factor of periodontitis. The causes of periodontal therapy on glycaemic control and systemic inflammation is not proven beyond doubt and need to be confirmed by clinical trials.

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