Abstract

Since beginning of night’s diabetes mellitus has been considered the main systemic disease that affects the periodontium. American Diabetes Association (ADA) has added periodontal disease as the 6th complication of diabetes. Diabetes attacks the periodontium and cooperate with periodontal microorganisms to induce rapid and severe attachment loss & alveolar bone resorption. Chronic hyperglycemia has negative pathological effects on periodontal tissues through many mechanisms as qualitative change of some subgingival pathogenic microorganisms, decreased salivation, disturbance of neutrophils functions, vascular changes, (AGE) accumulation in periodontal tissues, exaggerated production of prostaglandins and excessive generation of free radicals. Therefore, understanding of all these mechanisms is the first and the most important step in the management and better periodontal treatment outcome.

Key Words: Diabetes – Periodontitis – Relations – Mechanisms.

Material and Methods

Diabetes from the point of view of a periodontist:

Diabetes is a pathological condition of dysregulation of all body metabolism including carbohydrates, proteins, and fat. This condition results from a serious defect in this amazing hormone (insulin) either in its secretion or in its activity [4,5].

Types of diabetes mellitus:

The majority of diabetic population may have either type I or type II diabetes.

In type I: Autoimmune destruction of Beta cells of pancreas occurs in early life leading to lack of this hormone. It accounts for >10% of diabetics, most often occurs in children & young adults [6].

In type II: The defect is in its function, its influence on the cells receptors to open the gate for glucose. This type accounts for more than 90% and has an adult onset.

So, insulin is the key that opens the cell door to allow glucose to enter, without this key, the cell door will still be closed & glucose will accumulate everywhere in the body except in its right place inside the cell [7].

Diabetes family includes: Type I, type II gestational Diabetes, Diabetes due to specific disorders-and pre diabetes.

The normal blood sugar of human being lies within a narrow range of normal starting between 70 to less than 110 mg/dl. When we are fasting to less than 140 mg/dl. During the day, the body can accept this range, but if the fasting glucose level reaches 126 mg/dl or more and reaches during the
day 200 mg/dl or more here the troubles start to occur and diabetes is expected [8].

Diabetes complications: [2,9]

Complications are the major fear of patients with diabetes. Diabetic patients are 3 times more prone to heart disease & stroke, 25 times to blindness, 17 times to renal failure, 5 times to foot amputation due to diabetic neuropathy, finally 3 times more prone to severe periodontitis. So, diabetes attacks our body everywhere if not controlled.

Statistics about diabetes: [10-12]

Diabetes attacks people all over the world. Diabetes currently affects 246 million people worldwide and is expected to affect 380 millions by 2025.

- Every 10 seconds a person dies from diabetes-related causes.
- Every 10 seconds two people develop diabetes.
- In Egypt, the Egyptian diabetic population in 1994 was 4.3% of the total population while in 2006 this percentage increased to be 16% of the total population.
- In KSA, one Saudi in every five people older than 30 has diabetes mellitus. This means that every 5 persons attending your clinic one may be diabetic [13].

Diagnostic criteria for diabetes mellitus: [14]

1- Symptoms of diabetes plus casual plasma glucose \( \geq 200 \text{ mg/dl} \).
2- Fasting plasma glucose \( \geq 126 \text{ mg/dl} \) (no caloric intake for at least 8 hours (normal fasting glucose is 70-100 mg/dl)).
3- 2 hours postprandial glucose \( =/200 \text{ mg/dl during an oral glucose tolerance test} \).

Laboratory evaluation of diabetes control:

Glycated hemoglobin assay (HbA1c): [14]

1- 4% to 6% Normal
2- <7% Good diabetes control
3- 7% to 8% Moderate diabetes control
4- >8% Action suggested to diabetes control

Serum fructosamine (F.A): [15]

As with many lab values, the reference range is different from lab to lab, so all results must be interpreted within the context of the institution you are practising in.

As a guideline, each 75 umol change equals a change of approximately 60 mg/dl blood sugar or 2% HbA1c.

Evaluation of your patient's diabetic status is very important before starting any periodontal management:

- Blood glucose (Bl/gl) level gives an idea about the glycemic condition right now, any symptom of hyperglycemia (polyuria polyphagia, polydepsia, loss of weight, retarded wound healing, dry mouth .... etc ) is quite enough to measure the casual Bl/gl level, and if you find it 200 mg/dl or more so you may direct your patient to confirm his condition by doing laboratory tests for fasting and post prandial glucose level.
- Glycated Hemoglobin (GHB) on the otherhand tells the story of your patient diabetes in the last three months, it does not require fasting, and it is important especially if you are planning for periodontal surgery because it is quite related to tissue healing after surgery, it is not recommended to perform your surgery if Glycated Hemoglobin is more than 8%.
- Serum Fructosamine (F.A), measures the average hyperglycemic condition over a period of 1-4 weeks, as it is dependent on the half life of the albumin in plasma. It has been developed recently and-as in (GHB)-does not require fasting blood glucose. A study done by El-sayed, et al. [16] showed a positive relation between improvement of periodontal health and reduction of (F.A) in type II diabetic patients. They concluded that severe periodontitis exaggerates (F.A) values in diabetics.

Results and Discussion

How does diabetes attack our periodontium:

Diabetes worn us by exaggerated tissue response to microbial plaque severe gingival redness, gum bleeding, and repeated attacks of periodontal abscess. If we don’t respect the alarming signs, the damage rapidly becomes deeper with tissue detachment and sites with massive bony destruction will appear. Finally, it attacks the periodontium, so hardly leaving no tissues for the patient to use and for us to treat.

An important clinical study performed in pima Indians revealed that individuals with diabetes are twice as likely to exhibit periodontal attachment loss as non diabetics in different age groups.
Diabetes exaggerates gingival tissue response to infections [17]. To understand this statement we have to recognize the skin lesions of uncontrolled diabetic patient who was exposed to mosquito bite. Skin tissue reaction will show wide area of swelling, redness and degeneration of epidermal layer. This is exactly what happens in the gingival tissue in response to microbial toxins of the dental plaque which are severe swelling, redness and destructive responses around the neck of teeth. So, Diabetes creates a generalized condition of irritation over reacted inflammatory response all over the body through many mechanisms:

- **High glucose content:** In the gingival fluid in diabetic patient change the subgingival microflora by supplying bacteria with edogenous source of feeding leading to qualitative increase of some pathogenic microorganisms such as actinobacillus actinomycitum comitans and *p. gingivalis* [18].

- **Saliva:** Is the magic solution that cleans & flushes everything around periodontal tissue by its mechanical & immunological role and it has a major influence on plaque and calculus formation but chronic hyperglycemia creates a condition of severe tissue dehydration and of course decreased saliva and decrease all its functions [19]. So, decrease salivation allows accumulation of heavy microbial plaque and formation of huge masses of calculus deposition.

- **Disturbance of neutrophils functions:** Chronic hyperglycemia confuses and disturbs functions of neutrophils. This amazing inflammatory cell, is the main protective cell for the periodontium. In normal healthy condition it is a true killing machine against most of perio-pathogenic microorganism. Diabetes markedly reduces its adherence, directional mobility & phagocytosis ability, worsening of diabetes, exaggerating its catabolic activity resulting in excessive generation of its superoxides and collagenase enzymes and leading to more destruction of the surrounding periodontal tissues [20,21].

- **Vascular changes:** Comprise the main cause of all diabetic complications. Thickening of the of blood vessels (B.V) walls of diabetic gingiva called microangiopathy, the pathology here is similar to that occurs in the retinal and renal (B.V). Excessive unused blood sugar attacks blood lipoproteins specially Low density lipoproteins (LDL) forming a non enzymatic glycated complex called advanced glycation end product (AGE) which is considered the main player of diabetic complications. This (AGE) can penetrate the (B.V) walls then the blood monocytes follow the complexes and interact with it in the (B.V) walls with releasing of high amounts of inflammatory mediators leading to thickness of the wall on the expenses of the lumen till complete occlusion of these small vessels occur followed by its rupture, this is the main cause of gum bleeding in diabetics [16,22].

Based on the pathological similarity between diabetic periodontitis and diabetic retinopathy a study was performed to evaluate periodontal conditions in type I Diabetic patients with different grades of retinopathy [23]. The results demonstrated a significant increase in bleeding/plaque ratio (bl/pl ratio) in all groups with retinopathy even with minor retinal haemorrhage. So, Periodic periodontal examination may be of diagnostic value in early detection of diabetic retinopathy.

- **Advanced glycation end product:** (AGE) also accumulates in periodontal tissues with great affinity to collagen fibers forming an abnormal barrier against the integrity & the synthesis of these fibers. Also (AGE) attracts huge amount of neutrophils and macrophages-exactly as it occurs in the walls of blood vessels and the interactions resulting in excessive production of destructive mediators & enzymes. This is the main cause of excess fibrous breakdown and loss of clinical attachment [24].

- **Pathological changes in two hormones:** Progressive alveolar bone loss results from the severe disturbance in the following hormones: Reduction of circulating-systemically acting hormone (Insulin) and elevation of locally acting hormones which are called (Prostaglandins).

Insulin is one of the main anabolic hormones in the body so lack of it creates a condition of systemic osteoporosis and alveolar bone loss. on the otherhand, exaggerated production of prostaglandins, these potent inflammatory mediators due to over reacting inflammatory cells, induces osteoclastic activity and finally alveolar bone destruction. The amount of prostaglandins (PGE2) generated in the diabetic gingival tissue is more than 2 times in non diabetic gingiva [25].

- **Free radicals:**Excessive generation of free radicals (F.R) in the gingival tissues occurs due to generalized and localized causes. Free radicals molecules are lacking one or more of its electrons. Therefore, they are highly unstable and reactive always trying to steal these electrons from the surrounding cell membrane leading to cell damage. In diabetic status, chronic lack of intracellular
glucose directs cell mitochondria to use another source of fuel to produce energy (the FAT), but because fat is not so pure like glucose the results will be generation of large amount of toxic waste products as ketones and free radicals leading to generalized elevation of oxidative stress all over the body. The other source of (F.R) in the gingival tissues is the exaggerated production of neutrophils superoxides. High generation of these (F.R) will attack our precious periodontal cells (the fibroblasts) leading to its oxidative damage [26,27].

So, all these enemies cooperate with periodontal microorganisms to create periodontal profile of diabetes, therefore, if we want to manage and treat periodontal problems of diabetic patients, we have to understand the previously mentioned mechanisms to be able to fight all these enemies.

References


