

Dental Health of Osteopenia Diabetes Mellitus Male Patients

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Abstract

Objective: The aim of the study was to examine the oral health of osteopenia diabetes mellitus (DM) male, which includes condition of the teeth, the periodontal tissue, and the temporomandibular joint (TMJ). **Materials and Methods:** The basis of the absolute value and t-score, only 42 diabetic osteopenia male patients were included in this study. The examination used only male patients with DM osteopenia, aged between 30 and 70 years; they were divided into two groups according to the type of DM. The following clinical parameters used in this study determined the conditions of periodontal tissues involved: Carious of the teeth, gingivitis, periodontitis bleeding on probing, mobility of the teeth, and number of the teeth and the TMJ included TMJ tenderness, TMJ clicking, limitation in opening, drifting to one side, and statement of teeth like the presence of attrition. **Results:** The results of this study for the osteopenia DM patients found that the carious teeth and gingivitis occurred in all ages in both groups of DM. TMJ tender, clicking, limiting of mouth opening percentage increased with increasing the age in both groups of DM, the older age more suspected to, periodontitis, bleeding on probing, number of the teeth, and mobility of the teeth also attrition increased with increasing the age in both groups of DM. **Conclusion:** The oral health for the diabetic osteopenia male patients more effected, the percentage of carious teeth, gingivitis, attrition mobility of the teeth, bleeding on probing periodontitis, and number of the teeth were increased with increasing the age in both the groups of DM.

Keywords: Dental health, diabetes mellitus, male patients, osteopenia, temporomandibular joint

INTRODUCTION

Diabetes mellitus (DM) is one of the chronic metabolic diseases which result in disturbances in function of pancreatic beta cells,^[1] and defect in secretion of insulin or defect in action which causes hyperglycemia.^[2] It is considered as one of serious health problems, and it includes all type of gender and different ages.^[1] DM can be classified into kinds, Type 1 and Type 2 which called insulin-dependent that caused by the destruction of β -cell in the pancreas that leading to loss all insulin,^[3] the etiology may cause autoimmune reason, this type occur in male and female, its incidence in children and adolescents at any age. About 85%–90% of the affected patients contain human leukocyte antigens, or contain strong genetic predisposition which linked to environmental factors, such as coxsackie virus B4 and mumps virus act to destructive autoimmune process, and the pancreatic β -cells mostly selectively^[4,5] and other DM patients are idiopathic caused due to it have no indicator for autoimmunity reason.^[3]

The second kind is Type 2 which found in about 90%–95% from DM patients. It is described by peripheral resistance to insulin, which leads to progresses and increased formation

of glucose by the liver and dimension secretion of insulin leading to persistent hyperglycemia.^[3] This type DM usually occurs in more than 40-year-olds, in obese patients, and in others who don't make any practical physical exercise may effected. They usually used oral antidiabetic agents at the early years from disease also they may use insulin for the best control of hypoglycemia.^[4,5]

DM can cause many effects such as microangiopathy, arteriosclerosis, neuropathy, and nephropathy.^[6] However, modern studies for the reproductive system are not clear.^[7] Furthermore, it causes sexual dysfunction, for both male and female. It has been associated with vascular insufficiency, neuropathy, and psychological problems that include the pathogenesis of some phenomena, such as ejaculation, impotence disorders, and diminished libido, as well as to the decreased vaginal lubrication and orgasm

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dysfunctions.^[7-9] Dinulovic and Radonjic found the infertility in men to inappropriate synthesis of testosterone, which leads to molecular changes in the Leydig cells, secondary to DM.^[9]

Type 1 and Type 2 diabetes, especially uncontrolled suspected to osteopenia and osteoporosis, that is, by created nonenzymatic glycosylation to different kind of bone proteins, that involving Type 1 collagen, give result-impaired bone quality.^[10] Moreover, other studies indicated to increase in the levels of pentosidine that lead to fractures.^[11] The presence of glucose acts as a source of energy for osteoclasts and leads to elevated avian osteoclast activity.^[12] Furthermore, indirect effect of DM on bone can be through the hypercalciuria secondary to the glycosuria and other contact with Vitamin D metabolism and parathyroid hormone,^[13] the Type 2 of DM in some studies is causes to diminished the Vitamin D levels if compared with healthy controls.^[14] The oral manifestation for the DM patients is, xerostomia, carious teeth, teeth lost, and periodontitis it also associated with pathological changes in the mouth which involve fungal infection, mucosal ulceration, burning mouth syndrome.^[15] The DM effect for male hormone by hypogonadism and lower testosterone which lead to low bone mineral density and risk to fractures, when compared to nondiabetic male.^[16]

The aim of this study was to examine the oral cavity for the diabetic mellitus osteopenia male patients and find the relation between male hormones for both type of DM and oral hygiene.

MATERIALS AND METHODS

The study was conducted in the dental clinic from May to July 2017; the study was conducted among 42 diabetic osteopenia male patients, on the basis of the absolute value of *t*-score, which excludes smoker, hypertensive, osteoporosis, rheumatoid arthritis, cardiac disease, respiratory disease, and thyroid disease patients.

The examined of the diabetic patients with osteopenia (reduced of bone mineral, it's $<1^\circ$ if compared with osteoporosis) male patients (fasting blood sugar about 150–225 mg per deciliter with duration more than 3 years), the age between 30 and 70 years, they were divided into two groups depend on type of DM as the following: Group 1: 17 osteopenia patients with Type 1 DM, Group 2: 25 osteopenia patients with Type 2 DM.

The patients were examined extraorally and intraorally using different clinical parameters by determining health of periodontal tissues by measurement the bleeding on probing, gum recession, gingivitis periodontitis mobility of teeth and also the presence of caries in the teeth and temporomandibular joint (TMJ) statement by asking the patient to open, close mouth to determine the clicking and limitation in opening (measure the opening about 3 fingers equal 40 mm), move it from side to another side for checking drifting also protrusion of mandible and palpation to record the presence of TMJ tender [Figures 1-4]. The diagnostic instruments used were dental probe, periodontal probe, tweezers, dental mirror, and dual X-ray absorptiometry (DXA) (if present).^[17] The bone

mineral density can be measured by standard protocol and densitometry spine with DXA operator (Osteosys, Korea).

RESULTS

Table 1 refers to osteopenia DM patients Type 1 and Type 2



Figure 1: Method for examination of temporomandibular joint for Type 1 diabetes mellitus in dental clinic



Figure 2: Multiple missing and gingival recession for osteopenia diabetes mellitus Type 2 patient in dental clinic



Figure 3: Multiple missing and attrition for osteopenia diabetes mellitus Type 2 patient in dental clinic

distribution of age and numbers. Figure 5 shows the distribution of the male patients with percentage of gingivitis, periodontitis, bleeding on probing, attrition, mobility of the teeth, and carious teeth for Group 1 DM. Figure 6 explains the distribution of the male patients with percentage of gingivitis, periodontitis, bleeding on probing, attrition, mobility of the teeth, and carious teeth for Group 2 DM.

Table 2 shows distribution of the DM patients with percentage of TMJ tender, TMJ clicking, limitation of opening, protrusion of mandible and drifting of the jaw to one side during opening for Group 1 DM. Table 3 shows distribution of the DM patients show with percentage of TMJ tender, TMJ clicking, limitation of opening, protrusion of mandible and drifting of the jaw to one side during opening for Group 2 DM. Figure 7

Table 1: Distribution number and age of the patients depend on the type of diabetes mellitus

Age	Group 1 (type one number: 17 patients)	Group 2 (type two number: 25 patients)
30-39	3	6
40-49	5	8
50-59	5	7
60-70	4	4



Figure 4: Bleeding on probing for osteopenia diabetes mellitus Type 2 patient in dental clinic

shows relation between number of teeth and patient's ages for osteopenia patient DM Group 1. Figure 8: shows relation between number of teeth and patient's ages for osteopenia patient DM Group 2.

DISCUSSION

From this study, the intraoral examination the [Figures 4 and 5] that refer to carious teeth in Group 1 of DM (33.3%, 50%, 60%, 60%) and the carious teeth in Group 2 of DM osteopenia patients (44.5%, 50%, 50%, 50%) the increase incidence of carious teeth refer to symptoms of diabetes such as dry mouth and physiological change in the salivary gland that causes salivary dysfunction such as hyposalivation and xerostomia Vernillo^[18] and also may be increase glucose in saliva that leads to activation of oral flora increase decay of the teeth, the result agree with Vernillo, Nauntofte *et al.*,^[18,19] who found the carious teeth effected with salivary dysfunction and the DM patients have high amount of glucose in gingival crevicular fluid, also the result accepted with Haddad,^[20] the oral cavity frequent complicated with DM by higher incidence of dental caries which related to increase enamel hypocalcification and hypoplasia. However, this study disagrees with Taylor *et al.*^[21] though there does not appear to be a direct correlation between DM and increased dental caries.

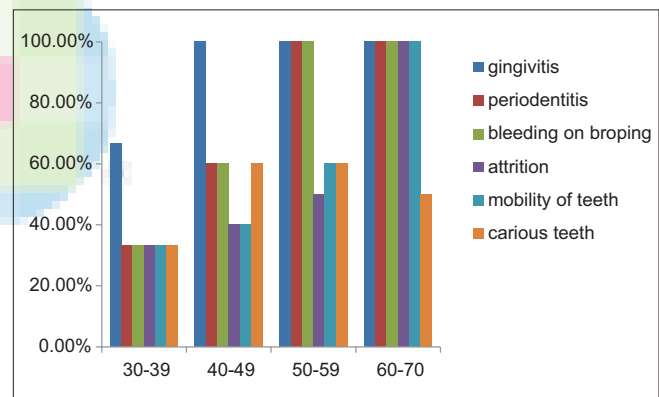


Figure 5: Distribution of the male patients with percentage of gingivitis, periodontitis, bleeding on probing, attrition, mobility of the teeth, and carious teeth for group one diabetes mellitus

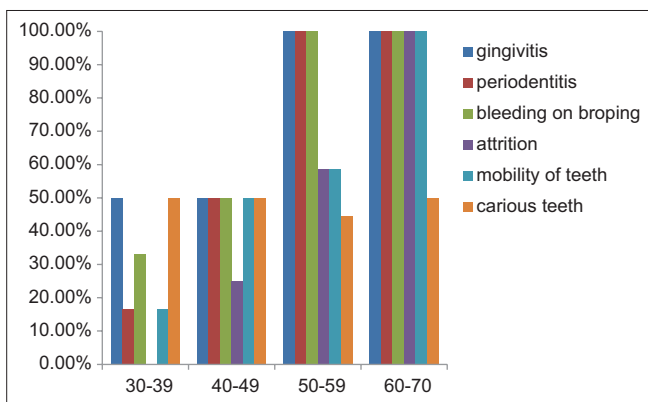


Figure 6: Distribution of the male patients with percentage of gingivitis, periodontitis, bleeding on probing, attrition, mobility of the teeth, and carious teeth for group two diabetes mellitus

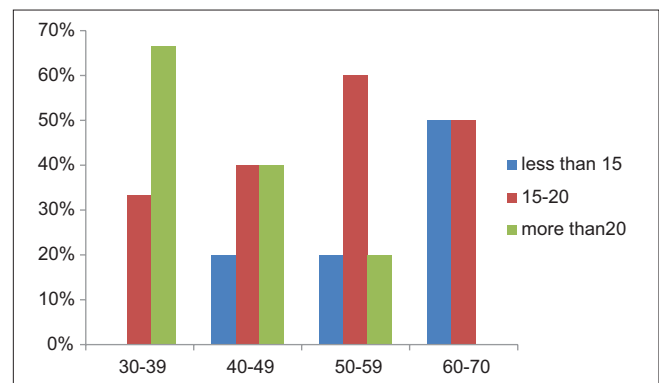


Figure 7: Relation between number of teeth present and patient's ages for osteopenia patient diabetes mellitus group one

Table 2: Distribution of the diabetes mellitus patients in relation to temporomandibular joint tender, temporomandibular joint clicking, limitation of opening, protrusion of mandible and drifting of the jaw to one side during opening for Group 1 diabetes mellitus

Age	TMJ tender (%)	TMJ clicking (%)	Limiting of opening (%)	Protrusion of mandible (%)	Drifting of the jaw to one side during opening (%)
30-39	33.3	33.3	0	0	0
40-49	60	60	40	0	0
50-59	100	100	100	50	50
60-70	100	100	100	100	100

TMJ: Temporomandibular joint

Table 3: Distribution of the diabetes mellitus patients in relation to temporomandibular joint tender, temporomandibular joint clicking, limitation of opening, protrusion of mandible and drifting of the jaw to one side during opening for Group 2 diabetes mellitus

Age	TMJ tender (%)	TMJ clicking (%)	Limiting of opening (%)	Protrusion of mandible (%)	Drifting to one side during opening (%)
30-39	33.3	33.3	0	0	0
40-49	50	50	25	0	0
50-59	100	100	100	50	50
60-70	100	100	100	100	100

TMJ: Temporomandibular joint

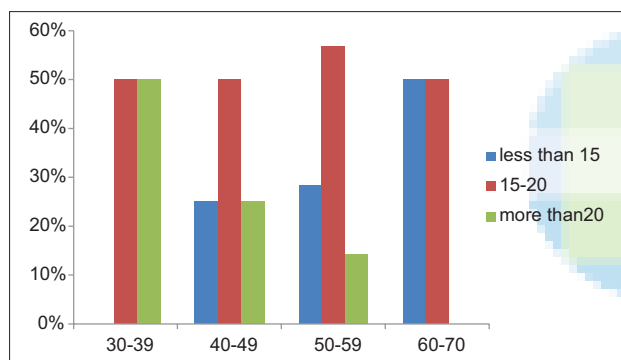


Figure 8: Relation between number of teeth and patient's ages for osteopenia patient diabetes mellitus group two

Figures 5 and 6 show the gingivitis in osteopenia patients with Group 1 (66.6%, 100%, 100%, 100%), and gingivitis in osteopenia patients with Group 2 (50%, 50%, 100%, 100%), the periodontitis in Group 1 is (33.3%, 60%, 100%, 100%), the periodontitis in Group 2 is (16.6%, 50%, 100%, 100%), the bleeding on probing in Group 1 equal (33.3%, 60%, 100%, 100%), the bleeding on probing in Group 2 equal (33.3%, 50%, 100%, 100%), and mobility of teeth in osteopenia patients Group 1 equal (33.3%, 40%, 60%, 100%) mobility of teeth in osteopenia patients Group 2 is (16.6%, 50%, 58.77%, 100%) from these result gingivitis highly incidence with DM and osteopenia and progressive to converted to periodontitis, these increased within arise the age in both groups of DM.

The physiological change for the gingiva for the DM patients occurs can explain by formation of gingivitis the reason represent as the DM autoimmunity disease the patients more susceptibility to incidence of gingivitis (inflammation of gum) by accumulation of plaque on the surface of the tooth, sulcus,

and gingiva that lead to stimulation pro-inflammatory cytokines and other chemical mediators, which induce an inflammatory response in the gum. The gingiva becomes edematous (fluid aggregation and cell infiltration). The polymorphonuclear cells, macrophages, monocyte, and lymphocytes are attracted to the gum by the chemotactic factors including microbial proteins and cytokine interleukin-8. The polymorphonuclear cells react to the gingival crevice became phagocytizing the bacteria which is useful in damping down the inflammation.

The immune response started within Langerhans cells in the gingival tissue phagocyte bacterial to reach the regional lymph nodes, which help to stimulate lymphocytes to come to the gum and B-cells transform to plasma cells to formation antibody, or T-cells differentiate to form cell-mediated immune response. The antibodies will help the polymorphonuclear cells to phagocytises of bacterial pathogens in cervical area, the fibroblasts and collagen will lose to felicitated work of inflammatory cell by invasion of gingival to create physical room to the infiltration leukocytes which produce matrix-degrading enzymes that cause connective tissue destruction, and may layer of junctional epithelium has been broken down and lose of the contact to the tooth that physiology for gingivitis formation.^[16,22,23]

The gingivitis if untreated that may lead to form the periodontal pocket, this pocket will penetrated by anaerobic microorganisms and the colonization of the facultative which may extend to the apical region which bone is resorption that regarded room for defense cells. The production of tumor necrosis factor α , prostaglandin 2, and interleukin will increased bone resorption, also formed granulation tissue and lead to tooth loss,^[22,23] and another factor increased incidence of periodontitis, bleeding on probing and mobility of the

teeth is osteopenia, the osteopenia causes low systemic bone mineral density and low bone mineral density in oral cavity led to lose alveolar bone and periodontal disease progression Wactawski-Wende.^[24] the mechanism of loosening of the bone by stimulation of osteoclast by the production of cytokines, like interleukin-6.^[25]

The result of the study agrees with Yoshihara *et al.*,^[25] who thought in older patients with systemic bone mineral density predicted for progression periodontal disease. Furthermore, Yoshihara *et al.*^[26] agree with a study which improved that the systemic bone mineral density (osteopenia) in older age patients peri-causes factor for periodontal disease, and the result accepted from World Health Organization^[27] which thought the DM one of most systemic disease which causes periodontal disease and dental caries. Other researchers^[28,29] believed that the microvascular diabetic mellitus causes periodontal disease which gives scientific evidence explaining that the DM is a risk factor for gingivitis, mobility of teeth and periodontitis and that blood glucose control which accepted with the result.

The extraoral examination results in Tables 2 and 3 TMJ tenderness (33.3%, 60%, 100%, 100%) in Group 1 DM and (33.3%, 50%, 100%, 100%) in Group 2, TMJ clicking (33.3%, 60%, 100%, 100%) in Group 1, TMJ clicking (33.3%, 50%, 100%, 100%) in Group 2, limitation in opening (0%, 40%, 100%, 100%) in Group 1 and limitation in opening (25%, 100%, 100%) in Group 2, protrusion of mandible (0%, 50%, 100%) in Group 1, protrusion of mandible (50%, 100%) in Group 2, drifting to one side during opening (0%, 50%, 100%) in Type 1, drifting to one side during opening (0%, 50%, 100%) in Group 2.

The result can explain by indirect effect of DM on the TMJ by decreasing of bone mineral density, microarchitectural distraction of trabeculae, decrease of osteoblast activity and hypocalcaemia that causes osteopenia leading to pain in TMJ like other joint in the body also perforation of the disk causes TMJ clicking that occur in younger age in both group of the DM but the older ages the effect increased by resorption of the TMJ and alveolar process of the mandible leading limitation in opening, protrusion of mandible and drifting. There is no study agree or disagree with these results.

Figure 7 represents the relation between number of teeth to Group 1, and Figure 8 illustrates the relation between the number of teeth to Group 2. The osteopenia and DM not of direct cause but the incidence periodontal disease and dental caries (if untreated caries may progressive to form periapical lesion) that reach the patient loss his teeth, and from this study found the periodontal disease occur in the young and progressive with the age that lead to increasing the loosen of the teeth increased with age.

Furthermore, multiple missing of the teeth is one of the factors responsible for protrusion of mandible and drifting the jaw to one side. The result accepted by Bartold *et al.*^[30] improved the loosen of the teeth related to diminish bone density in

the oral bone associated with systemic bone disease, also the study agreed with Tervonen and Karjalainen^[31] who found that the patients with uncontrolled diabetes may more subject to loss teeth in different sites in the mouth than did patients with good to moderate controlled DM and the periodontal included of patients with good to moderate controlled DM paralleled that of patients unaffected with diabetes and it accepted with Hugoson *et al.*^[32] who improved the patient's ages more than 40 years effected with Type 1 DM who give a significant increase in periodontal advanced position and also bone loss than unaffected DM subjects more than 40 years.

Figure 5 shows attrition of teeth (0%, 40%, 50%, 100%) for Group 1 and while Figure 6 shows attrition of teeth (0%, 25%, 58.77%, 100%) for Group 2, which found the attrition increased with increasing the ages can explain by many causes hypocalcaemia the teeth, the multiple missing of the teeth and drifting the jaw to one side that leads attrition teeth occur that means the osteopenia and DM not direct goal for attrition, there is no study agree or disagree with these results.

CONCLUSION

The study concluded that the DM and osteopenia have a direct effect for the oral hygiene for the male patients including increase percentage of gingivitis, periodontitis, bleed on probing mobility of the teeth, dental carious, and attrition with increase patient's ages in both types of DM. The study found change in the TMJ in patients male the percentage TMJ pain, TMJ clicking, drifting of the jaw to one side limitation of mouth opening, protrusion of the mandible, and number of the teeth increase with increase of patient's ages in both types of the DM. Another conclude the male hormone does not affect the oral health and the Type 1 and Type 2 DM for osteopenia patients has no significant effect for an oral cavity for both types another conclusion the male hormone has no direct effect on oral health.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Roghani M, Baluchnejadmojarad T. Survey the hypoglycemic and hypolipidemic effect of chronic oral administration of nigella sativa in diabetic rat. *J Med Fac Gullan Univ Med Sci* 2007;16:26-31.
2. Lalla RV, D'Ambrosio JA. Dental management considerations for the patient with diabetes mellitus. *J Am Dent Assoc* 2001;132:1425-32.

3. Mealey BL, Ocampo GL. Diabetes mellitus and periodontal disease. *Periodontol* 2000;44:127-53.
4. Sousa A, Rodrigues E, Oliveira A, Vinha E, Medina J. Controle metabólico nos doentes diabéticos: O que nos separa das recomendações atuais? *Rev Port De Diab* 2006;1:11-3.
5. Cruz S. Tratamento não farmacológico na diabetes tipo. *Rev Port De Clín Geral* 2005;21:587-95.
6. Camilleri M. Clinical practice. Diabetic gastroparesis. *N Engl J Med* 2007;356:820-9.
7. Zarzycki W, Zieniewicz M. Reproductive disturbances in type 1 diabetic women. *Neuro Endocrinol Lett* 2005;26:733-8.
8. De Berardis G, Pellegrini F, Franciosi M, Belfiglio M, Di Nardo B, Greenfield S, *et al*. Clinical and psychological predictors of incidence of self-reported erectile dysfunction in patients with type 2 diabetes. *J Urol* 2007;177:252-7.
9. Dinulovic D, Radonjic G. Diabetes mellitus/male infertility. *Arch Androl* 1990;25:277-93.
10. Vashishth D, Gibson GJ, Khoury JI, Schaffler MB, Kimura J, Fyhrie DP, *et al*. Influence of nonenzymatic glycation on biomechanical properties of cortical bone. *Bone* 2001;28:195-201.
11. Schwartz AV, Garnero P, Hillier TA, Sellmeyer DE, Strotmeyer ES, Feingold KR, *et al*. Pentosidine and increased fracture risk in older adults with type 2 diabetes. *J Clin Endocrinol Metab* 2009;94:2380-6.
12. Williams JP, Blair HC, McDonald JM, McKenna MA, Jordan SE, Williford J, *et al*. Regulation of osteoclastic bone resorption by glucose. *Biochem Biophys Res Commun* 1997;235:646-51.
13. Okazaki R, Totsuka Y, Hamano K, Ajima M, Miura M, Hirota Y, *et al*. Metabolic improvement of poorly controlled noninsulin-dependent diabetes mellitus decreases bone turnover. *J Clin Endocrinol Metab* 1997;82:2915-20.
14. Scragg R, Holdaway I, Singh V, Metcalf P, Baker J, Dryson E, *et al*. Serum 25-hydroxyvitamin D3 levels decreased in impaired glucose tolerance and diabetes mellitus. *Diabetes Res Clin Pract* 1995;27:181-8.
15. Alyasiry AM. Oral hygiene for the diabetes mellitus and osteoporosis patients. dentistry college, dentistry department, Babylon university. *Res J Pharm Biol Chem Sci Iraq* 2017;8:783.
16. Asano M, Fukui M, Hosoda H, Shiraishi E, Harusato I, Kadono M, *et al*. Bone stiffness in men with type 2 diabetes mellitus. *Metabolism* 2008;57:1691-5.
17. Al-Yasiry A, Al-Jammali Z, Al-Rubbaie S, Abbas A. Dental health in osteoporotic women". 1 dentistry college and medicine college, AQ5 dentistry department, Babylon university. *Res J Pharm Tech* 2015;8.
18. Vernillo AT. Dental considerations for the treatment of patients with diabetes mellitus. *J Am Dent Assoc* 2003;134 Spec No: 24S-33S.
19. Nauntofte B, Tenevuo JO, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd E, eds. *Dental Caries. The disease and its clinical management*. Oxford. Blackwell Munksgard; 2003. p. 16.
20. Haddad A. Distúrbios Endócrino-Metabólicos. In: *Pacientes Portadores De Necessidades Especiais Manual de Odontologia e Saúde Oral*. São Paulo: Pancast; 2000. p. 157-64.
21. Taylor GW, Manz MC, Borgnakke WS. Diabetes, periodontal diseases, dental caries, and tooth loss: A review of the literature. *Compend Contin Educ Dent* 2004;25:179-84, 186-8, 190.
22. Kinane F, Berglundh T, Lindhe J. Host-Parasite Interactions in Periodontal Disease. In: Lindhe J, Karring T, Lang NP. editors. *Clinical periodontology and implant dentistry*. 2003. p. 150-76.
23. Nisengard R, Haake S, Newman M, Miyasaki K. Microbial interactions with the host in periodontal diseases, In: Carranza's clinical periodontology, Newman, Takei, Klokkevold and Carranza, 2006. p. 233-40.
24. Wactawski-Wende J. Periodontal diseases and osteoporosis: Association and mechanisms. *Ann Periodontol* 2001;6:197-208.
25. Mundy GR. Cellular and molecular regulation of bone turnover. *Bone* 1999;24:35S-38S.
26. Yoshihara A, Seida Y, Hanada N, Miyazaki H. A longitudinal study of the relationship between periodontal disease and bone mineral density in community-dwelling older adults. *J Clin Periodontol* 2004;31:680-4.
27. World Health Organization. Continuous Improvement of Oral Health in the 21st Century-the Approach of the WHO Global Oral Health Programme. Geneva: WHO; 2003.
28. Lamster IB, Lalla E. Periodontal disease and diabetes mellitus: Discussion, conclusions, and recommendations. *Ann Periodontol* 2001;6:146-9.
29. Newman M, Carranza F. *Periodontia Clinica*. 11th ed. Rio de Janeiro: Editora Guanabara Koogan; 2012.
30. Bartold PM, Cantley MD, Haynes DR. Mechanisms and control of pathologic bone loss in periodontitis. *Periodontol* 2000;53:55-69.
31. Tervonen T, Karjalainen K. Periodontal disease related to diabetic status. A pilot study of the response to periodontal therapy in type 1 diabetes. *J Clin Periodontol* 1997;24:505-10.
32. Hugoson A, Thorstensson H, Falk H, Kuylenstierna J. Periodontal conditions in insulin-dependent diabetics. *J Clin Periodontol* 1989;16:215-23.