ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 68 . 16
PubMed - National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 5, Issue – 5, September - 2020, Page No. : 80 - 87

Diabetes mellitus- an orthodontic perception

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Citation this Article: Dr Mitali Brijmohan Sharma, Dr Suchita Tarvade (Daokar), Dr Amruta Chandak, "Diabetes mellitus- an orthodontic perception", IJMSIR- September - 2020, Vol – 5, Issue - 5, P. No. 80 – 87.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

With the popularization of adult orthodontics diabetes mellitus is a rising concern in orthodontic practice. The orthodontist needs to be aware of the types, causes, pathogenesis, risk factors and clinical manifestations and management of diabetes mellitus. The updated knowledge of the precautions and changes in the treatment planning and the applied mechanotherapy will lead to successful management of diabetic patients. This review aims to guide the orthodontist in the same.

Keywords: Diabetes mellitus; Orthodontic management; Orthodontic manifestations; Type 1 diabetes mellitus; Type 2 diabetes mellitus.

Introduction

Diabetes Mellitus is one of the most commonly encountered lifestyle diseases that is prevalent worldwide. It is estimated that its prevalence will increase from 6.4% in 2010 to 7.7% by 2030, and from 285 million in 2010 to 439 million adults by 2030.[1]Juvenile diabetes has always been an orthodontic concern, but, with the popularization of adult of orthodontics, Type 2 diabetes now requires the orthodontist's attention.

The Greek word 'Diabetes', coined by Araetus of Cappodocia (81-133AD) means 'to siphon or to pass through'. Later, the Latin word 'mellitus' was added by Thomas Willis (Britain, 1675) meaning 'honey sweet'.[2] The Egyptians thought Diabetes to be a polyuric syndrome(1550 BC). The Indian Vedic literature (5th and 6th century) described Type 2 diabetes and established its relation to sedentary lifestyle, heredity, obesity and diet.[2]

In the 18th century, due to the introduction of chemistry and endocrinology, a better understanding of the causes, symptoms, management and complications developed. Mathew Dobson (1776) confirmed the presence of sugar in diabetic urine, which Michael Chevreal (1815) demonstrated to be glucose. Dobson reported that diabetes is not just a kidney disorder, but, is a systemic disorder.[2]

According to contemporary literature, Diabetes mellitus is a group of metabolic disorders characteristic of hyperglycemia due to defective insulin secretion or insulin action or both.[3] Uncontrolled chronic hyperglycemia can inflict serious opthalmologic, nephrologic, neurologic and cardiovalscular complications. Thus, diabetes has been labelled- 'the silent killer' due to the severe involvement of vital organs.[4]

Diabetes also affects teeth and associated structures.[5] Orthodontic treatment of juvenile diabetes was always a part of the orthodontic practice. The newer concept that Orthodontic treatment can be commenced at any age as long as the periodontium is healthy, requires a thorough knowledge of diabetes and its implication in Orthodontics and this review aims for the same.

Classification

National Diabetes Data Group (NDDG) has classified diabetes (1979) based on pharmacologic therapy:[6]

1. Insulin-dependent diabetes mellitus (IDDM)
2. Non-insulin-dependent diabetes mellitus (NIDDM)[6]

The American Diabetes Association classification (1997) based on the pathogenesis along with modifications:

1. Type 1 Diabetes Mellitus (T1DM)/ autoimmune/ Juvenile/ Insulin dependent[7]

a. Antibody positive (type 1a)
b. Antibody negative (type 1b)
c. Latent Autoimmune Diabetes of Adults/ type 1.5
d. IPEX- Immune dysregulation, polyendocrinopathy, enteropathy, X-linked
e. MEA- Multiple endocrine abnormalities[7]

2. Type 2 Diabetes Mellitus (T2DM)/ non-autoimmune/ adult onset/ Non-insulin dependent[7]

a. Idiopathic hyperglycemia[8]
b. Obese
c. Non-obese
d. Ketosis-prone[7]

3. Gestational Diabetes Mellitus (GDM)[6]
4. Monogenic Diabetes[7]

a. Neonatal
b. MODY- like
c. Syndromic
d. Type 1
e. Genetic defects in insulin action
f. Congenital lipodystrophies

5. Secondary Diabetes Mellitus/ Other disease associated[7]

a. Cystic Fibrosis
b. Chronic Pancreatitis
c. Pancreatic neoplasia
d. Post Pancreatectomy
e. Hereditary Hemochromatosis
f. Endocrinopathies
g. HCV/HIV
h. Others

6. Drug associated[7]

a. Glucocorticoids
b. Atypical antipsychotics
c. Calcineurin inhibitors/mammalian target of rapamycin inhibitors
d. Human immunodeficiency virus
e. Thiazide diuretics
f. Hydroxymethylglutaryl-coa reductase inhibitors
g. Nicotinic acid
h. Diazoxide
i. Simvastatin and atorvastatin
j. β -adrenergic agonists

7. Chemical induced diabetes[3]

a. Vacor (a rat poison)

b. Streptozotocin[7]

c. Intravenous pentamidine[7]

International statistical classification of diseases–9 and –10 coding based on the degree of control of diabetes[7]:

E08- Due to underlying condition

E09- Drug/ chemical induced

E10- T1DM

E11- T2DM

E13- Other

Etiology

More than 90% of T1DM patients have either HLA-DR3, HLA-DQ2 or HLA-DR4, HLA-DQ8 haplotypes of genes.[9] IDDM 2 on chromosome 11p5.5, a non-HLA gene, contributes to 10% of T1DM.[9-8] Congenital rubella syndrome[9] and early childhood diet may be influential. Physical inactivity, cigarette smoking, alcohol consumption have been linked to T2DM.[10] Obesity contributes to approximately 55% of cases of T2DM. [11] Aging[12] and a high fat diet[13] are also contributors. First degree relatives may genetically inherit T2DM[14] via genes like TCF7L2, PPARG, FTO, KCNJ11, NOTCH2, WFS1, CDKAL1, IGF2BP2, SLC30A8, JAZF1, and HHEX. [14] The environmental toxin, Bisphenol A is associated with T2DM. [15] Hypertension, hypercholesterolemia, hyperlipidemia, metabolic syndrome X or Reaven's Syndrome[16] cause or exacerbate T2DM. Other causes consist of acromegaly, Cushing's syndrome, thyrotoxicosis, etc. [17]

Major Risk Factors For Type 2 Diabetes: [18]

1. Family history of diabetes
2. Age above 35 years
3. Overweight (BMI ≥23), obese (BMI ≥25)
4. Higher upper body adiposity
5. Hypertension
6. Sedentary lifestyle
7. Gestational diabetes

Pathophysiology

The hallmarks of T1DM are polyuria, polyphagia, polydipsia, overt hyperglycemia and exogenous insulin replacement. [19] It is mainly immune-associated rather than immune- mediated destruction of insulin producing pancreatic β cells. [19-1,2] The CD4+, CD8+ T cells and macrophages infiltrating the islets cause autoimmune destruction β cells. [20] This leads to deficiency of insulin secretion, excess glucagon secretion[20] and uncontrolled lipolysis. There is decreased expression of the genes necessary for target tissues to respond normally to insulin. [20] Therefore, deranged lipid, protein and glucose metabolism are associated with T1DM due to problems in insulin, the only hormone that reduces blood glucose. [21]

The balance between insulin secretion and tissue sensitivity to insulin is deranged in T2DM as a consequence of impaired insulin secretion from pancreatic β-cells and impaired insulin action from increased insulin resistance. [22,23] When insulin secretion cannot compensate for insulin resistance, hyperglycemia results. [21]

Clinical features

Polyuria, Polydipsia, Polyphagia, sudden weight loss, lassitude, constipation, fatigue, cramps, blurred vision, candidiasis and other recurrent infections are the warning signs in both types of diabetes. [18,24] In type 2 diabetes, atherosclerosis of the large vessels[20] is seen amongst the other symptoms. Delayed wound healing, burning sensation or pain or numbness in feet, itching, reactive hypoglycemia, Acanthosis nigricans (indicator of insulin resistance) and impotence or erectile dysfunction may be associated. With chronic hyperglycemia, microvascular and macrovascular complications may arise.[20-25]

Diagnostic tests

An individual with more than two risk factor and aged 35 years and above should get screened for diabetes. The diagnostic tests include Oral glucose tolerance test, [25] random blood glucose (>150mg/dl is a warning), Fasting plasma glucose (Impaired- >110 - <125 mg/dl, diabetic- >126 mg/dl), Post-prandial glucose (Impaired- >140-<200 mg/dl, diabetic- >200 mg/dl) and Glycosylated Heamoglobin (HbA1c), the gold standard test[26] (prediabetic- 5.7 - 6.4%, diabetic- >6.5%).

Oral manifestations

Decreased polymorphonuclear leucocyte function causes frequent candidial, staphylococcal or streptococcal infections.[27,28] Defective collagen synthesis, degradation of collagenase and glycosylation of collagen at wound margins, all consequences of impaired protein metabolism, lead to delayed wound healing. [28] Other oral findings include glossopyrosis, xerostomia, dental caries, taste dysfunction and oral mucosal alterations like fissured tongue, geographic tongue, recurrent apthous stomatitis and oral lichen planus. [29]

The orthodontically significant findings in T1DM include the remarkable periodontal and alveolar bone destruction around the first molar and incisors, [27] which may clinically presents as mobility of these teeth. This destruction aggravates with age as an outcome of poor periodontal health in childhood. [27] Type 1 diabetics also show delayed craniofacial maturation. [30]

Increased prevalence of gingivitis and periodontitis, [31] loss of periodontal clinical attachment[32] and loss of alveolar bone[33] are the most important manifestations. Fulminant periodontitis may be attributed to Microangiopathies[34] and impaired neutrophil function. In a well-controlled diabetic patient, the periodontium is healthier than uncontrolled diabetics, but not as healthy as non-diabetic patients.

Orthodontic treatment Considerations

From the moment a patient enters the out-patient department, the orthodontist needs to look out for the signs of risk factors associated with diabetes. Appropriate case history should be recorded which should reveal a history of diabetes and should rule out the associated sleep apnea. [35] Intraoral examination may reveal oral symptoms, reflecting diabetes. The patient has to be referred to a general physician and pathologist to ensure the patient's fitness and glycemic control. A medically well-managed diabetic patient can be treated without hesitation. If the HbA1c >9%, a good glycemic control should be obtained beforehand. [36]

An Orthopantamogram should be recorded at the commencement of orthodontic treatment and correlated with clinical findings to establish the periodontal status of the patient. Diabetic patients with periodontitis, should undergo prophylactic periodontal management.

Short, early morning appointments, approximately 1 hour after breakfast are suitable, because it ensures optimal blood glucose levels, avoiding hypoglycemic shock.[37] Patients with T1DM, whose main treatment is insulin, are more prone to hypoglycemic shock. It is characterized initially by weakness, confusion, paleness and excessive salivation followed by loss of consciousness. [28] Recognition of the signs and immediate administration of glucose rich beverage should be done. If there is loss of consciousness, intravenous 50% dextrose solution (50 ml) should be given. [34] If the appointments last longer, blood glucose should be monitored at the end of every hour. [37]

The orthodontist needs to cautiously follow strict sterilization protocol of all instruments and materials, as the diabetic patients are prone to infections, which

might lead to cardiovascular problems like bacterial endocarditis. [38]

Borderline cases should be treated with non-extraction approach to reduce trauma. When extraction is necessary appropriate protocol should be followed and sutures may be required to prevent oozing of blood due to delayed wound healing.

Fixed orthodontic appliances should be preferred over removable in diabetic patients. Due to greater alveolar bone resorption in diabetic patients, the center of resistance is shifted more apically[39] and the point of application of force becomes further away in removable appliances leading to excessive tipping. The lack of periodontal support will result in quicker movement, but, may lead to loss of vitality[39] of teeth and may aggravate the periodontitis. No influence of orthodontic appliances on glucose metabolism has been reported. [40]

During anchorage preparation for T1DM patient, undue stress onto the first molars has to be avoided as these teeth are periodontally involved.[27] Anchorage alternatives like second molars or orthodontic mini-screws may be employed. The reduced craniofacial development[30] should be considered while considering the timing of treatment in T1DM patients. In cases of bone loss, reinforced anchorage is necessary. [39] Tooth eruption may be normal or accelerated in diabetic patients[36] and therefore, while planning extrusive movements, lighter forces have to be applied.

There is evidence to suggest that diabetes affects bone remodeling and orthodontic tooth movement. [41] The antidiabetic drug 'Metformin' reverses the undesired effect of diabetes on tooth movement as shown in rats, [42] which makes it a drug of choice, when possible. No information was retrieved about antidiabetic drugs or insulin in relation to orthodontics. [39]

A diabetic patient's teeth are fragile and light physiologic forces safeguard against overloading of dentition. [43] Greater number of units of elastomeric chains, lighter elastics, segmental loop mechanics instead of frictional mechanics and round wires instead of rectangular wires, where possible, are some ways to reduce the force levels.

Invasive procedures like separator placement,[44] molar band placement and insertion of orthodontic mini-screws(TAD) require antibiotic prophylaxis. [35] Taking intraoral radiographs, making impressions, local anesthetic injection, bracket bonding,[36] replacement or removal of orthodontic appliances, changing wires and adjustments of appliances does not require antibiotic prophylaxis. [45] Steel ligature ties are preferable over elastic ties to reduce plaque retention. [39]

Maintenance of strict oral hygiene has to be emphasized. Instructions should include brushing teeth with specifically designed orthodontic toothbrushes as they are more convenient for patient use. [46] Adjunctive rinsing with chlorhexidine mouthwash may be advised 30 minutes after tooth-brushing. [28] Aggravation of symptoms like burning mouth, xerostomia and candidiasis may be expected. [36] The patients should be informed that treatment duration may be longer, as the rate orthodontic tooth movement is affected. [47] If there are signs of lapses in the glycemic control, the patient and the physician have to be informed. [28] The importance of physical activity, diet control and good long-term glycemic control have to be stressed.

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