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Specific aspects for the management of diabetics in dentistry

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Summary.

Introduction. Diabetes mellitus is a very common metabolic disease and potentially fatal due to severe complications characterized by an absolute or partial absence of insulin. Nowadays it is increasing and it is not uncommon for diabetic patients to come to our office. It is very important for dentists as is quite common, to diagnose it due to its relationship with certain oral pathologies. Complications of Diabetes and therapy drugs interactions can lead to emergencies that dentist must manage.

Objetives. Goal of this project is to prepare the dentist to know all relevant data about the managing of diabetic patients.

Materials and methods. Thirty-two articles and two books taken from data webs highlighting Pubmed and Medline have been revised and most relevant information have been summarized and sintetised to do this bibliografic review about relevant aspects of diabetes related to dentistry field.

Discussion. The possible medication for diabetics and the possible interactions with dental treatment drugs prescription has been described. A relation has been proven between diabetes and oral pathologies, been periodontal pathologies the most important ones, follow by delay in healing, candida infections, xerostomia and many others. Special precautions for diabetics have been founded highlighting monitoring, short and morning appointments. Emergencies related with diabetes are dangerous, being hypoglycemia the

most common one presented at dental clinics. Hyperglycemic emergencies must not be underestimated as they are mortal as well.

Conclusion. Because of frequency and severity, the dentist must be fully capacitated to manage diabetic patient. Diagnose them, have knowledge of values, monitor glucose level to differentiate if controlled or not, take special precautions and manage emergencies in case they occur.

Resumen.

Introducción. La diabetes mellitus es una enfermedad de tipo metabólico muy común y potencialmente mortal debido a complicaciones graves caracterizada por una falta absoluta o relativa de insulina. Hoy en día está aumentando y no es raro que los pacientes diabéticos acudan a nuestra consulta. Es muy importante para los odontólogos ya que es común diagnosticarlo debido a su relación con determinadas patologías bucales. Sus complicaciones y la interacción con sus medicamentos terapéuticos pueden provocar emergencias que el dentista debe manejar.

Objetivos. El objetivo de este proyecto es preparar al dentista para conocer todos los datos relevantes sobre el manejo del paciente diabético.

Materiales y métodos. Se han revisado treinta y dos artículos y dos libros procedentes de webs de datos destacando Pubmed y Medline y se ha resumido y sintetizado la información más relevante para hacer esta revisión

bibliográfica sobre aspectos relevantes de la diabetes para el ambito de la odontología.

Discusión. Se ha descrito la posible medicación para diabéticos y sus posibles interacciones con la prescripción de medicamentos para el tratamiento dental. Se ha comprobado una relación entre la diabetes y las patologías bucales, siendo las patologías periodontales las más importantes, seguidas de retraso en la cicatrización, infecciones por cándida, xerostomía y muchas otras. Se han establecido precauciones especiales para diabéticos destacando la monitorización, citas cortas y matutinas. Las emergencias relacionadas con diabetes son peligrosas, siendo la hipoglucemia la más común que se presenta en las clínicas dentales. Las emergencias hiperglucémicas no deben subestimarse, ya que también son mortales.

Conclusión. Debido a la frecuencia y severidad, el dentista debe estar completamente capacitado para tratar a un paciente diabético. Diagnosticarlos, saber los valores, monitorear el nivel de glucosa para diferenciar si está controlado o no, tomar precauciones especiales y manejar las emergencias en caso de que ocurran.

Index by pages.

1. Introduction.

Diabetes Mellitus is a very frequent disease nowadays worldwide. International Diabetes Federation (IDF) has predicted that diabetic people could rise from 425 millions that were in 2017 to 629 millions in 2045 in the entire world.(1)

According to this same federation, by 2011, the 6% of the European population in ages from 20-80 years had Diabetes Mellitus (DM) which account for fifty-two millions of affected and this may increase by year 2030 to a 7% which is sixty-four millions. (2) It is more than a fact that it is necessary to give importance to glycaemic controls and healthy habits to prevent this disease and to avoid the development of complications.

DM is a metabolic disorder in which there is hyperglycemia, elevated content of glucose in the blood and its etiology depends mostly on genetic and environmental factors. This disease causes alterations in different organ systems of the body. In USA, it is the primary cause of end-stage renal disease, limb amputations, and blindness. Also it is a risk element for developing cardiovascular diseases (CVD) and can develop micro and macrovascular complications like retinopathy, nephropathy and neuropathy. (3)

Main types of DM are; type 1, which is autoimmune and is characterized by destruction of pancreatic β cells and total loss of insulin secretion; and type 2 caused by insulin resistance. (2) The high calories amount that characterizes the Occidental diet, combined with physical inactivity, has been related to a higher prevalence of obesity which can lead to insulin resistance (DM type 2). (3)

Among other types of Diabetes, we can find Gestational Diabetes Mellitus, which develops insulin resistance during pregnancy. It is present in about 1-14% of

pregnancies in USA, most of these women regain normal glucose tolerance after delivery, but these women will have a risk (35-60%) of suffering DM in the following ten to twenty years. (3) DM should not be confused with Diabetes Insipidus, characterized by a defect in antidiuretic hormone. (4)

In addition, the American Diabetes Association also recognizes an intermediate group that, although they are not considered diabetic, their glucose levels are too high compared to normal. It is called prediabetes and may be considered a risk factor for developing DM type 2 and for CVD. (4)

We must know how to differentiate the 2 main types and what is the cause of each one. Type 1 (total insulin deficiency) represents about 5-10% and is also called insulin-dependent diabetes or juvenile-onset diabetes. Within this there are two subgroups; Immune DM, total insulin deficiency because of autoimmune causes and destruction of beta cells from pancreas. This type has a great genetic predisposition, the affected are not usually overweight and the maximum incidence happens in puberty; Idiopathic DM, rare, the majority being Asian or African. DM Type 2 represents 90–95%, also called non-insulin-dependent diabetes, or adult-onset diabetes, these patients do not normally need to take insulin to live, they are usually obese and related to an older age. Complete etiology of Diabetes can be found in Annex 1. (5)

Severe hyperglycemia can cause polyuria (excessive urination), polydipsia (excessive thirst), polyphagia (excessive hunger), weight loss, blurred vision, prone to infections, life-threatening situations as ketoacidosis or non-nonketotic hyperosmolar syndrome, severe long term complications as blindness, foot ulcers and amputations, among others. Having DM is a risk factor for

cardiovascular pathologies, arteriosclerosis, or high blood pressure (hypertension). (5) Diabetes is a chronic disease and is not something to be taken lightly. It can complicate dental visit as are related with orthostatic hypotension, oral pathologies, emergencies at clinic as well as delay in healing. (4)

Among the risk factors for type 2 DM we can find; family history of this disease, obesity, sedentary lifestyle, ethnicity, gestational diabetes, hypertension, cholesterol, polycystic ovary and cardiovascular disease. (3)

Among the oral pathologies that can be related with DM are dental caries, ulcers, candidiasis, glossitis, xerostomia, lichen planus, leukoplakia, lichenoid reactions, eruption alterations, gingivitis and periodontitis. (4)

When making a correct diagnosis it is essential to know the standard values. First, know the concept of prediabetes, also called impaired glucose tolerance (IGT) or impaired fasting glucose (IFG) and their values are;

Impaired fasting glucose (IFG); fasting plasma glucose (FPG) values ≥100 mg/dl (5.6 mmol/l) and <126 mg/dl (7.0 mmol/l). Simpler, values of plasmatic glucose when patient has not eaten, are between 100-125 mg/dl or between 5.6-6.9 mmol/l. (5)

Impaired glucose tolerance (IGT): two hours postload glucose values in the oral glucose tolerance test (OGTT) of ≥140 mg/dl (7.8 mmol/l) and <200 mg/dl (11.1 mmol/l). Simpler, values 2 hours after eating are between 140-199 mg/dl or 7.8-11.1 mmol/dl. (5)

Normal values would be <100 mg/dl in FPG (fasting) and <140 mg/dl OGTT (after eating) and hyperglycemic values would be >126 mg/dl in FPG and >200 mg/dl in OGTT. (5)

With these data three categories have been arranged depending on whether we use the FPG or OGTT test; normal; prediabetes, impaired fasting glucose or impaired glucose tolerance; and provisional diagnosis of diabetes. They can be easily observed in Table 1 and Table 2. (5)

Table 1. Values in Fasting Plasma Glucose (FPG).

	mg/dl	mmol/l
Normal fasting glucose	<100 mg/dl	<5.6 mmol/l
IFG (impaired fasting glucose)	100 –125 mg/dl	5.6 – 6.9 mmol/ l
Provisional diagnosis of diabetes	≥126 mg/dl	≥7mmol/l

Table 1 showing values in FPG for patients with regular fasting glucose, impaired fasting glucose and tentative DM, represented in mg/dl and mmol/l.

Table 2. Values in the oral glucose tolerance test (OGTT).

	mg/dl	mmol/l
Normal glucose tolerance	<140 mg/dl	<7.8 mmol/l
IGT (impaired glucose tolerance)	140 –199 mg/dl	7.8 – 11 mmol/ l
Provisional diagnosis of diabetes	≥200mg/dl	≥11.1 mmol/l

Table 2 showing values in OGTT for patients with normal glucose, impaired glucose tolerance and tentative diabetes, represented in mg/dl and mmol/l.

When performing these tests we must take into account these three factors; Fasting means no caloric intake for at least 8h; diabetes apart from manifesting hyperglycemia can be accompanied by these typical symptoms; polyuria, polydipsia, polyphagia and unexplained weight loss; to carry out the after eating test (OGTT) according to the WHO (World Health Organization) it is necessary to do the test with 75g of glucose dissolved in water. (5)

There is another test to measure blood glucose; the glycated or glycosylated hemoglobin, HbA1c or A1c, which measures the average glucose level of a person in the last 2-3 months. It is a percentage and HbA1c range according to ADA is; non-diabetic between 4-5.6 %; prediabetes between 5.7-6.4% and ≥6.5% are considered diabetics. American Diabetes Association (ADA) recommends keeping HbA1c levels below 7% for diabetics, as well as diet plus exercise. HbA1c test is not recommended for pregnant women nor childhood diabetes. (6) For a better understanding there is a comparison chart between glycated hemoglobin and blood glucose levels in Table 3. (6). However, Ac1 test has some limitations such pathologies that affect the red blood cells as anemia, recent transfusion, some drugs and kidney disease. (7)

HbA1c offers advantages in the diagnosis of DM since it does not require fasting and it represents the average of glucose of the last two-three months. Its disadvantages are due to the variability in results observed in different ethnicity, age, gender groups and pathological processes that produce anemias. The ADA considers OGTT the gold standard for the diagnosis of DM. (8) The FPG is the test of choice to diagnose diabetes in children and non-pregnant adults. (9)

Table 3. Comparison between HbA1c and mg/dl glucose level.

HbA1c %	Mg/dl	mmol/l
5	97	5.4
6	126	7
7	155	8.6
8	184	10
9	212	11.8
10	241	13.4
11	268	14.9

Table 3 describe the different values comparing between two different common test, Hba1c and mg/dl.

Diabetes treatment is based on controlling the disease and the complications derived from it. For this, a series of recommendations are given such as glycemic controls, diet, physical exercise, quitting smoking, having other diseases controlled such as hypertension and cardiovascular diseases. (9)

Although diabetes recommendations, treatments and interactions will be further developed later, here is a small review of most common treatments.

For type 1 diabetes, the most common treatment is insulin therapy, which is usually subcutaneous administration and varies depending on the severity and time of onset, as well as the age and weight of the patient. Doses usually range from 0.5-1.5 IU/kg/day. For type 2 diabetes, the main therapeutic option is the use of oral hypoglycemic agents. There are different drugs with different functions, the ultimate goal of which will be to lower plasma glucose levels. These are the most common; thiazolidinediones, biguanides, meglitinides, sulfonylureas, α-glycosidase inhibitors, etc. (4)

2. Objectives.

Principal objective.

The purpose of this work is to review the parameters that a dentist must know to treat a diabetic patient, as have been previously discussed, DM is a fairly common disease that can be frequently found in dental clinic. Like many other diseases, Diabetes Mellitus has a series of pathologies and complications related or caused by the disease itself, and this project focuses on those that are related to dental field, the pathologies related to the oral cavity.

It is not surprising that dentists themselves suspect the existence of this disease when it is not yet diagnosed. For this, the following specific aims have been established to be developed in this work.

Specific objectives.

- Provide both dentists and other specialists useful information for when dealing with diabetic patients.
- Review all the theory related to DM, what DM is, what types do exist, the etiology, signs and symptoms, diagnosis and common treatments.
- Review typical diabetes related pathologies that can be found in the oral cavity so its diagnosis is possible in case it is not yet diagnosed and thus start an earlier possible treatment.
- Know glucose values to know when patients can or can not be treated in the dental office, if they are controlled or not. Assess the possibility of having a glucose test at clinic and know what kind of tests exist and what each one measures.

- Know the medication patients are taking and if there is any interaction with the treatment or medication that we are going to use while the treatment or prescribe after it; anesthesia, antibiotics, antifungals, analgesics, chlorhexidine, etcetera.

- Know better the emergencies that can happen at clinic related to diabetes, such as hyperglycemia and hypoglycemia, their cause, how to handle them and so ensure the safety of the diabetic patient.

- Communicate to diabetic patients a series of measures that they can adopt to prevent or improve their oral pathology, make patient conscious of the disease.

3. Methodology.

In order to carry out this end-of-degree project, a data collection has been realized, for which a review of several scientific articles and medical books written by different authors about dentistry, science and medicine fields has been analyzed. A search and reading of numerous documents have been done and thirty-four documents have been selected; observational, bibliographic reviews, clinical studies, where the most relevant information on the subject in question has been analyzed and collected. Specifically thirty-two articles and two books have been chosen, using inclusive criteria by year of publication, language, keywords, and relevance to the project.

A series of platforms have been set up to search for data, highlighting PubMed and Medline, this information collected is no more than 20 years old, is written in two languages, English and Spanish and from different countries.

Keywords have been used such as *Diabetes mellitus, oral pathology, treatment, hypoglycemic agents, insulin, diagnosis, complications, emergencies, management, periodontitis, caries,* etcetera.

The most relevant information has been collected regarding pathologies of the oral cavity in diabetic patients, drugs and treatments in these patients, tests, values, and emergencies. An order has been followed to review the articles, starting by looking for a theoretical basis in each one and then analyzing the most relevant data regarding the topics already mentioned, and then making a correct writing of the topic.

4. Discussion.

After the bibliographic review of the articles and books found, where the most relevant information for our clinical practice has been extracted, we are going to discuss the relevant part for the dental field. Once we have understood the theoretical basis of diabetes, we must know how to apply it in our clinical practice.

4.1. Diagnosis of the diabetic patient in clinic.

A first step to determine the oral health of our patient would be to carry out a complete survey as well as an anamnesis and medical history.

Also to provide a good education on oral hygiene, dental diseases and pathologies. It is vitally important that patients know hygiene techniques; times and technique of brushings, use of rinses and dental floss. (10)

Patients and relatives should participate in educational program, according to World Health Organization, the best way to provide health care is health education. (11)

It is also important that the patient knows the frequency with which he/she should visit the dentist (depending on his oral health) and that the dentist knows why this frequency may be reduced; lack of time, difficult access, fear of the dentist, socio-economic factors. (10)

In the case of diabetic patients, they should be aware of the relationship between diabetes and oral pathologies.

There is a problem associated with diabetic patients that would be solved if there were better communication between dentists and other specialists. A diabetic patient visits the dentist when it is urgent (not in all cases), the disease is advanced and requires treatment, they do not relate the oral symptoms to the

systemic disease. At the same time, the family doctor does not refer the diabetic patient to the dental clinic, he/she does not relate the oral pathologies with the systemic problem. All this slows down the diagnosis, and consequently the treatment. (10)

Few dentists measure patient's blood glucose and plan visits based on the severity of the diabetic condition. (12)

Values of tests for diagnosis of Diabetes.

1. Fasting plasma glucose: ≥126mg/dl ≥7mmol/dl)
2. Two-hour post eating plasma glucose: ≥200mg/dl ≥11.1mmol/dl)
3. Ac1 (glycated hemoglobin): ≥6.5 % (5)

Main types of diabetes.

- Type 1 DM. Young patient, autoimmune, Beta cell destruction.
- Type 2 DM. Adults, obesity. Insulin resistance.

Typical symptoms polyuria, polydipsia, polyphagia, weight loss, etc. (5)

Other types/causes are gestational, monogenic, pancreas disease, endocrinopathies, drug-induced, infections, genetc sindromes, etc. Extended list in annex 1. (13)

Monitoring is made by use of a glucometer. It is a devise where a strip with a blood drop is inserted. Lasts seconds in giving results. The training of the staff of the clinic in this devise is highly recommended.

Glycated hemoglobin (HbA1c) is used to control diabetes in long term. Hemoglobin is a protein bound to red blood cells and is responsible of the

transport of oxygen. When hemoglobin is wrapped by a lot of glucose, some of the glucose will stick to the hemoglobin. This process is known as glycosylation. HbA1c is the ratio of glycosylated hemoglobin to non-glycosylated hemoglobin and is a percentage. It gives the mean of the blood sugar level in the last two-three months. (14)

Testing for diabetes should be done in people over forty-five years and with obesity, usually three times per year. Also in younger people with obesity, inactivity, family history of diabetes, high baby delivery weight, hypertensives, vascular disease, among others. (9)

Gestational Diabetes Mellitus (GDM). It happens in pregnant women who have not had DM. It usually resolves after delivery. Many women develop type 2 DM later. GDM occurs due to beta cell dysfunction. Early diagnosis is important to avoid developing type 2 DM and fetal malformations. (13)

Insulin resistance related to metabolic and hormonal disturbances during pregnancy increases insulin requirements and can lead to hyperglycemia or glucose intolerance. (15)

Changes in lipid level during pregnancy are related to increased estrogen and insulin resistance. Low HDL and high triglycerides (TG) are observed. (16)

Pregnancy hormones block the action of insulin in the mother's body.

GDM is one of the most frequent complications of pregnancy and is increasing due to the increase in type 2 diabetes and obesity. The most frequent complication in the baby is macrosomia (big baby) and respiratory problems. Others are caesarean sections, fetal malformations, and preeclampsia (high blood pressure). (17)

Unlike the increase in the baby's weight, the following case can also occur, to treat gestational diabetes, the diet is modified and medications are perhaps used, this can alter the weight gain. Limited maternal weight gain is associated with low-weight newborns. (18)

4.2. Treatment and drugs.

Treatment of diabetes is based on the type of diabetes, the degree of severity of the disease, the level of hyperglycemia and the characteristics of the patient (age, weight, associated diseases, etc).

The range of treatments is wide, from lifestyle changes to oral antidiabetics and / or insulin.

We can order the treatment in the following steps if we talk about type two diabetic patients that we will see developed later; diet and exercise, monotherapy and combination therapy. (9)

A step prior to treatment is prevention. According to various studies, a benefit in oral health is proven in patients after an educational intervention such as training classes, workshops, talks, leaflets, group discussions, where oral hygiene techniques, oral pathologies, nutrition, exercise, healthy habits, monitoring blood sugar, and medications are taught, both to patients and their families, in order to have controlled diabetes and prevent or delay its complications. (11)

First step of treatment will be control risk factors, once a diabetes is correctly diagnosed.

A. Monitoring Diabetes.

Glycemic control by the patient (self monitoring). Also recommended in prediabetics. For type 1 patients and pregnant women who are taking insulin monitoring is recommended 3 or more times per day. Use of glucometers. (9)

Table 4. Recommendations values for diabetics adults (lipids included)

GLYCEMIC CONTROL	**VALUE**
A1C	<7.0%
PREPRANDIAL CAPILLARY PLASMA GLUCOSE	90-130 mg/dl
POSTPRANDIAL CAPILLARY PLASMA GLUCOSE	<180 mg/dl
BLOOD PRESSURE	<130/80 mmHg
LIPIDS	**VALUE**
LDL	<100 mg/dl
TRIGLYCERIDES	<150 mg/dl
HDL	>40 mg/dl

Table 4 showing recommendations for adults. Children, pregnant and elderly needs other values.

B. Diet and weight loss.

People with diabetes must follow a strict diet, preferably done by a doctor or dietitian. The amount (grams) and type of carbohydrates change blood glucose levels. Carbohydrates intake should be 45-65 % of total calories. Saturated fat is recommended to be <7% of total calories but the whole fat per day should be 25-35 % of total calories. Proteins intake must be 10-35% of total calories. Fiber foods, such as legumes, cereals, fruits, vegetables, etc are highly recommended. Reduce weight is advised for overweight (BMI 25–29.9 kg/m2) and obese (BMI >30.0 kg/m2) patients who already suffer, or can develop type II DM.

Diets for losing weight should be more or less 1.000–1.200 kcal per day for women and 1.200–1.600 for men but depends on the case.

Sometimes surgery or drugs are recommended to achieve weight loss.

Eliminate or decrease alcohol intake. (9)

C. Physical activity.

If not contraindicated, exercise should be gradual and related to the patient's ability. It is recommended from thirty to forty-five minutes of medium exercise, three to five days per week. If better ability, harder exercise as 1 h per day walking or haft an hour per day jogging, for example. (9)

D. Other diseases or risk factors controlled.

Cardiovascular disease (CVD) is the main cause of mortality for diabetic patients. Hypertension, dyslipidemia, and coexisting of them are risk factors.

Hypertension. Blood pressure must be checked every time diabetic patients come to clinic. They should have less than 130/80 mmHg. Equal or more than 140/90 mmHg patient is considered hypertensive and must follow a drug therapy as calcium channel blockers, diuretics, beta-blockers, ACE inhibitors, among others and stablish healthy habits. Diabetes plus hypertension is a dangerous combination as increase the likelihood to develop cardiovascular problems and severe complications of diabetes, retinopathy and nephropathy.

Dyslipidemia. Goal is to reduce saturated fat and cholesterol intake. For individuals without cardiovascular disease (CVD), goal is LDL < 100 mg/dl and if CVD is present, LDL <70 mg/dl. Triglycerides <150 mg/dl and raise HDL cholesterol to >40 mg/dl in men and >50mg/dl in women. Use of statins and other

drugs to lower lipid level can be recommended (no use of statins in pregnant women).

Antiplatelet therapy. Aspirin therapy (about 100mg/day) is recommended as prevention in diabetics with history or high risk of developing CVD. (9)

E. Stop smoking.

It is recommended stop smoking not only for diabetics but for all kind on patients and not only for oral health but for general health. Cigar smoking is the most important modifiable responsible factor of premature death and has relation with macrovascular and microvascular complications of DM, as well as many other diseases. (9)

The first option treatment for type 2 diabetes (DM), is to control body weight, avoiding overweighting, through a healthy diet and physical exercise. Often and sadly, these measures are not enough to achieve good glycemic control and thus it is necessary to use oral antidiabetics, in monotherapy or combined with other antidiabetics, or with insulin. The oral antidiabetics number that we have nowadays is quite high, we are describing the most commonly used, as well as some adverse effects and some trade names (not all) and the most relevant interactions for dentistry, which can interfere with our treatment (analgesics, antibiotics, antifungals, etc). Firstly, oral antidiabetics can be classified regarding its mechanism of action (table 5). (19)

Table 5. Mechanism of action of different oral antidiabetics.

A. Increase sensitivity to insulin.	- Biguanides. - Glitazones (thiazolidinediones).
B. Increase the secretion / release of endogenous insulin at the pancreatic level.	- Sulfonylureas. - Fast acting secretagogues (glinides). - Inhibitors of dipeptidyl peptidase (DPP)-4 (gliptins).
C. Decrease the absorption of glucose in the digestive tract.	- Alpha-glucosidase inhibitors. - GLP-1 analogs (subcutaneous administration, not part of oral antidiabetics)
D. Increase glucose removal.	- Sodium-glucose transporter type 2 inhibitors (SGLT-2 inhibitors or gliflozins)

Table 5 showing the four main action mechanism of the most common oral antidiabetics.

1. Biguanides (metformin): overweight patients. It can be used alone or with other oral antidiabetics or insulin. Adults and children >10 years old. Metformin 850 mg/1000 mg. Common trade names *Dianben®, Metformin EFG (generic), Stada*. During or after meals and 1-3 daily intakes. Common side effects are gastrointestinal that usually disappear and can be decreased if the treatment is started in stages and with meals. They can also cause nausea and vomiting, metallic taste, weight loss or poor absorption of vitamin B12, rarely causing anemia. The most serious, although infrequent, is lactic acidosis, it is more common in patients with kidney failure or alcoholism. Due to its mechanism of action, it is unlikely to cause hypoglycaemia, unless it is combined with other antidiabetic or insulin. Metformin is contraindicated in case of renal failure, heart failure or recent myocardial infarction, respiratory or hepatic failure, ketoacidosis, diabetic precoma, alcoholism, and radiographic iodine contrast scans. (19) Mortality from lactic acidosis associated with metformin administration is estimated to be around 50-80%. (20)

2. Sulfonylureas: patients with normal weight or slightly overweight, alone or with other oral antidiabetic agent or with insulin, 30 min before or during meals.

Gliclazide 30; 60 mg *Diamicron®, Gliclazide EFG (Generic Pharmaceutical Equivalent).*

Glibenclamide 5 mg *Daonil®, Glucolon®.*

Glimepiride 2; 4 mg *Amaryl®, Roname®, Glimepiride EFG.*

Glipizide 5 mg *Minodiab®.*

Glisentide 5 mg *Staticum®.*

It can cause hypoglycemia, although the incidence is usually lower than with insulin. The most usual side effects are gastrointestinal and skin reactions. Sulfonylureas should not be given if type I diabetes, ketoacidosis, diabetic coma or precoma, hypersensitivity, kidney or liver failure. (19)

3. Glinides (meglitinides): fast-acting secretagogues: non-insulin-dependent DM in monotherapy or in association with metformin. In the case of nateglinide, it is only used in combination with metformin. Nateglinide 60 mg *Starlix®*, Repaglinide 0.5;1; 2 mg *Novonorm®, Prandin®*. Intake 30 min before meals Skip the dose if miss a meal. Three intakes. Adverse effects; weight gain and hypoglycemia with relative frequency, especially without eating. (19)

4. Alpha-glucosidase inhibitors: can be an adjuvant of metformin, sulfonylureas and insulin. At the beginning of meals, 3 intakes. Acarbose 50; 100 mg *Glucobay®*, *Acarbose EFG*. Miglitol 50; 100 mg *Diastabol®*, *Plumarol®*. Gastrointestinal adverse reactions. Contraindicated if hernias, intestinal ulcers, intestinal disorders, kidney or liver failure. (19)

5. Glitazones (thiazolidinediones): monotherapy when there is contraindication of metformin or can be given combined with metformin, sulfonylureas, or insulin, also is used in triple therapy with metformin and sulfonylureas. One intake at any time during the day. Pioglitazone 15; 30 mg *Actos®, Glustin®*, *Pioglitazone EFG*. Adverse effects are and fluid retention, weight gain, congestive heart failure, bladder cancer and bone fractures. Contraindicated if diabetic ketoacidosis, liver failure, heart failure or bladder tumor. (19)

6. DPP-4 inhibitors (gliptins): monotherapy or in double therapy (with sulfonylurea, metformin or thiazolidinedione) or in triple therapy (with metformin + sulfonylurea or metformin + thiazolidinedione) or with insulin, one intake before eating. Alogliptin 25 mg *Vipidia®*, Linagliptin 5 mg *Trajenta®*. Saxagliptina 2,5; 5 mg *Onglyza®*, Sitagliptina 25; 50; 100 mg *Januvia®*, *Ristabon®*, *Tesavel®*, Vildagliptina 50 mg *Galvus®*, *Jalra®*, *Xiliarx®*. Dermatological and gastrointestinal reactions. Contraindicated if type 1 DM, ketoacidosis, kidney or liver failure and pancreatitis. (19)

7. Gliflozins: inhibitors of cotransporter sodium-glucose type 2 (SGLT-2 inhibitors). Indicated in adults, alone or in combined with other oral antidiabetic agent and insulin. Canagliflozine 100; 300 mg *Invokana®*, Dapagliflozine 10 mg *Forxiga®*. One intake independent of food. Adverse reactions are urinary tract infections and postural hypotension. Contraindicated if DM type 1, ketoacidosis, kidney failure and bladder tumor. (19)

Special groups.

1. Pregnancy. Drug of choice for diabetes in pregnant women is insulin. Metformin and Acarbose are category B.

2. Lactation. Metformin, Acarbose, Miglitol and Glipizine risk very low.

3. Children. Metformin in children older than 10 years, or insulin.

4. Elderly. Glimepiride (sulfonylurea) is the best choice, due to its half-life is long, and administration is one dose, since old patients are generally polymedicated. It is not recommended to start treatment with gliflozins in people over 75 years, because there is an increase in adverse reactions. (19)

Relevant interactions for dentistry.

1. Biguanides:

- Tetracyclines: Increase the risk of metabolic acidosis.
- Corticosteroids: Decrease the effect of Metformin.
- Cimetidine (a drug to treat ulcers and reflux) can increase likelihood of lactic acidosis.
- Alcoholic drinks and furosemide: potency action of Metformin, which can cause lactic acidosis.

2. Sulfonylureas.

- Increases the risk of hypoglycemia (the combination of sulfonylureas and following drugs).

- Acetylsalicylic acid (NSAID).
- Oral anticoagulants.
- Chloramphenicol (antibiotic).
- Fluconazole and Miconazole (antifungals).

- Decrease the effect of sulfonylureas (blood glucose level increases):

  - Diuretics (thiazides, indapamide, chlorthalidone).
  - Estrogens.
  - Corticosteroids.

- Antibiotics interactions.

  - Erythromycin: potentiates liver toxicity.
  - Tetracyclines and sulfamides: Enhances their effect.
  - Cyclosporine: Risk of nephrotoxicity.

3. Thiazolidinediones (glitazones): pioglitazone.

   - Antifungals (ketoconazole): inhibit metabolism.

4. Inhibitors of α-glucosidases.

   - Corticosteroids: Risk of hyperglycemia.

5. Nateglinide and repaglinide (glinides)

   - Ketoconazole can increase effect.
   - Corticosteroids can decrease their effect.
   - NSAIDs can cause hypoglycemia. (19) (15)

Must be taken into account that some drugs also contain sugars in their composition: sucrose, fructose, lactose or even glucose and thus increasing hyperglycemia. In annex 2 there is a list with drugs that increase or decrease glucose level. (19)

In the following table is showed the order to choose the treatment.
Table 6. Steps of treatment for diabetics type 2.

1	- Diet, weight loss and physical exercise
2	- Monotherapy: Metformin (except if contraindication)
3	Doble therapy (one of the options) - Metformin + sulfonylurea - Metformin + glinide - Metformin + gliptin or pioglitazone
4	- Doble therapy + insulin - Triple therapy: metformin + sulfonylurea+ gliptin or pioglitazone If insulin intolerance, use of GLP1 analogue

Table 6 show the steps recommended to follow for treatment of diabetics. It is a progressive treatment, starting with the lower level treatment and if this fails, apply the next step. (19)

Insulin.

Mainly used for the treatment of type 1 DM (type 2 when necessary). (15)

Type 1 DM is a chronic autoimmune disease in which there is a destruction of beta cells, leading to an absolute insulin deficiency, so all patients require insulin treatment from day one of diagnosis. Insulin therapy in type 2 is frequently used as last option, when all other measures have failed. (21)

Plasmatic insulin (physiologic) inhibits the liver's production of glucose to prevent fasting hyperglycemia or basal. After eating, to avoid postprandial hyperglycemia, there is a rapid rise in insulin level, reaching the highest level in 30 minutes and returning to normal levels in 2-3 hours. Insulin suppresses the liberation of glucose from liver and kidneys, and stimulates its storage (glycogen form), in liver and skeletal muscle. There are several types of insulin;

1. Fast acting insulin (prandial).

Introduced subcutaneously before eating (boluses) to avoid postprandial hyperglycemia. Used to correct hyperglycemia and can be also administered

intramuscularly or intravenously. (21). Bolus or short-acting insulin generally injected just before snacks or meals. (14)

A. Regular human insulin. Subcutaneously, onset action at 30 minutes, maximum action at 2-4 hours and total duration 6-8 hours. Commercial name is *Actrapid*®.

B. Rapid-acting insulin analogues. Human insulin with modifications in the amino acid structure that reduce the absorption time. Onset action after 5-15 minutes, maximum action at 30min-2 hours and a total duration of 3-5 hours. Trade names are insulin glulisine or *Apidra*®, insulin aspart or *Novorapid*® and insulin lispro or *Humalog*®. (21)

2. Intermediate Acting Insulins and Intermediate and Rapid Acting Insulin Premixes.

Intermediate-acting insulins start at 1-2 hours, maximum action at 4-10 hours and total duration 10-18 hours. Trade names *Humulina NPH*® and *Humalog NPL.*

Intermediate and rapid acting premixed insulins consist of NPH or NPL insulin premixed with rapid-acting insulin.

Examples are *Novomix30*® (30% Aspart/70% NPH), *HumalogMix25*® (25% Lispro/75% NPL), *HumalogMix50*® (50% Lispro/50% NPL), *HumalogMix75*® (75% Lispro/25%NPL) and *NPL Mixtard30* ® (30% Regular/70% NPH). (21)

3. Long-acting or basal insulin.

Duration greater than 24 hours and lower risk of hypoglycemia. Some examples are Glargine *Lantus*® (20-24h), Detemir *Levemir*® (18-20h) and Degludec *Tresiba*® (> 42h). (21). Injected 1-2 times per day. (14)

Guidelines.

1. Basal-bolus insulin regimen (short and long duration).

Indications: type 1 DM; type 2 DM treated with basal insulin with good basal glucose but with elevated preprandial and/or postprandial blood glucose; situations in which marked insulinopenia (ketosis, spontaneous weight loss) and/or predominance of daytime hyperglycemia; hospitalized patient.

Pattern:

1. Previous basal insulin: maintain basal insulin and metformin/pioglitazone and add 10% of the basal dose before each meal as a rapid insulin analog.

2. No previous insulin: 0.3-0.5 IU / kg / day (type 2) and 0.5-0.7 IU / kg / day (type 1): 50% as basal insulin and 50% as prandial insulin, divided into the 3 meals. (21)

2. NPH / NPL or premixed insulin dose before breakfast and dinner.

Indications: patients with DM2 and insufficient control with basal insulin; suspected insulin deficit (ketosis, spontaneous weight loss) and/or predominance of daytime hyperglycemia; patients with DM1 in whom basal-bolus regimens are not indicated.

Initial guideline: 0.3-0.5 U / kg / day: 60% at breakfast and 40% at dinner. (21)

3. Basal insulin regimen with / without non-insulin hypoglycemic agents.

Indications: patients type 2 not stable with non-insulin agent intake.

Initial regimen: low doses (0.1-0.2 IU / Kg) before dinner (basal insulins) or at bedtime (NPH / NPL). (21)

Type 1 DM treatment options:

1. Subcutaneous insulin treatment

Insulin must mimic the body's physiological insulin release (continuous basal secretion that avoid hyperglycemia without eating) and mealtime insulin release (prevents postprandial hyperglycemia).

Basal / bolus insulin therapy is widely used and is divided in;

A. Subcutaneous injection of long-acting basal insulin (eliminate glucose creation between meals and at night)

B. Bolus injection insulin of rapid action at mealtime.

2. Insulin pump. Continuous subcutaneous infusions released at variable rates during the day. It allows very precise insulin amounts delivered, improved glycemic control and less long-term complications. However, they have high price and infection may develop as a permanent cannula is required and the likelihood of ketoacidosis is higher because they use just insulin of fast action.

3. Transplantation

Pancreas transplant alone or in combination with kidney transplantation. (22)

4.3. Oral pathologies related to diabetic patients and their treatment

As previously mentioned, there are pathologies that are related to diabetes and that the dentist must be able to diagnose and relate to this disease.

Diabetic patients have complications in wound healing, according to various theories this may be because they have worse vascularization, an alteration in collagen synthesis and a decrease in platelet activity. In addition, there is a decrease in the chemotaxis of polymorphonuclear neutrophils, which reduces their immune response and makes them more likely to suffer from infections and oral pathologies, especially if they are poorly controlled. (4)

1. Periodontal diseases. Having uncontrolled glucose for a long time is related to an increase in the incidence and progression of gingivitis, periodontitis, loss of alveolar bone and consequently loss of teeth. According to several studies, they are predictors of ischemic heart disease and diabetic nephropathy. The increased incidence can be due to alterations in the host's response, alterations in collagen metabolism and consequently a periodontal fibers change, alterations in the subgingival microflora, vascularity and crevicular fluid, and there is also a hereditary pattern. (22)

Scientific evidence supports that there is a bidirectional relationship between diabetes and periodontitis. (4)

The severity of diabetic gingivitis depends on the blood glucose level. If not controlled, soft, red, and sometimes bleeding swellings of the gums appear, often accompanied by dry mouth and changes in the smell of the breath. It is difficult to treat if glucose remains elevated due to the altered host's response. Successful treatment also depends on self-care of the patient and controlling glucose levels with diet and hypoglycemic drugs. Depending on the severity, the

treatment can be improving oral hygiene, prophylaxis, scaling and root planing or surgery. (23) Periodontal disease is suggested to be the 6th complication of Diabetes. (13)

2. Dental caries. Caries incidence should decrease because of the supposed lower sugars intakes, but is not the case, the incidence and aggressiveness increase in poorly controlled patients. (4)

Lactate levels in diabetics saliva are higher and in severe cases can be 5 times the normal level, which is a contributing factor to caries. (11)

The treatment of caries depends on its extension, remove caries and direct restoration (composite) or indirect (crown, inlay, etc.), endodontics if the pulp is affected and extraction if it cannot be restored.

3. Salivary gland dysfunction and xerostomia. Diabetic people commonly suffer dry mouth or xerostomia. This can be because glycosuria causes an osmotic diuresis. (22) More common in patients with type II and can sometimes be accompanied by swelling of the salivary glands (sialosis). (4) Sialodenosis is a painless, non-inflammatory swelling of the major salivary glands, caused by a systemic disease. Treatment attempts to control the systemic disease. (23) The treatment of xerostomia is; control systemic disease, suppress xerostomizing drugs, drink plenty of water, avoid irritants in the diet such as coffee, alcohol or smoking, treat stress and anxiety, salivary stimulants (sialogogues), more frequent meals, intake of lemonades or acidic drinks, chewing gum with xylitol, sialogogue drugs that directly stimulate salivary parenchyma such as anetoltrithione, pilocarpine and cevimilin, among others. (24) Due to the high

frequency of presentation in diabetics, the sensation of dry mouth can be a symptom that should alert the dentist to the possible existence of diabetes mellitus. (25)

4. Oral mucosa disease. Lichen planus, leukoplakia and lichenoid reactions incidence is increased by immunosuppression and by drugs for the treatment of diabetes. (4). Lichen planus treatment is corticosteroids and biopsy depending on the type. Lichenoid reaction remove irritant and leukoplakia remove irritant and biopsy if not removed or removal of the patch. (23)

5. Oral infections. Opportunistic infections as oral candidiasis occurs often in poorly controlled diabetic patients due to immunosuppression and salivary hypofunction. (22) Pseudomembranous candidiasis is an opportunistic infection caused by the excessive growth of the fungus C. albicans, it presents white, velvety, non-painful plaques, until they detach, leaving a red, or bleeding surface. It appears in the palate, tongue and jugal mucosa. Main cause is change in the normal oral microflora due to antibiotics, steroids or systemic alterations as diabetes. Topical antifungal medication, or systemic medication for two weeks, usually produces resolution. Candida cheilitis is also frequent in these patients. (23)

6. Buccal sensorial alterations. Burning mouth or tongue can happen in diabetic people perhaps due to xerostomia and candidiasis. Burning of the tongue is suggested to be because of diabetic neuropathy. (22)

Burning mouth syndrome or glosodinia treatment is to control the local or systemic factor causing it, also useful relaxant drugs as clonazepam (benzodiazepine) and paroxetine (antidepressant), topical lubricants and rinses. (26)

7. Glossitis: changes in the filiform papillae of the tongue accompanied by a burning sensation in the mouth. (4) Median rhomboid glossitis prevalence is higher in diabetic patients. Antimicrobial treatment depends on the cause of infection and good oral hygiene. (23)

8. Alterations in dental development: acceleration or slowing is possible, depending on the age of onset of diabetes. A higher incidence of cleft palate has been described in neonates of diabetic mothers with poor metabolic control. (4)

9. Oral ulcers: Diabetic patients develop ulcers in the mouth more easily and with more incidence. (4) Ulcers are usually painful, and often require drug treatment, topical or systemic, and mouthwashes. (23)

10. Tooth loss. Periodontal disease (destruction of periodontium) is related to tooth loss in diabetic patients. According to a study probing depth were greater in the first and second molars, and this coincides with the most frequently missing teeth**.** (27)

4.4. Complications and emergencies.

1. Macrovascular complications. Arteriosclerosis is the main risk at macrovascular level. (4) Arteriosclerosis lead to heart attacks, strokes and amputation. (14) Diabetic patients has 2-4 times more risk to die from heart disease and suffer a stroke. Stroke and heart pathologies are about 65% of the causes of death of diabetics. Amputations are 10 times higher in diabetics than normal. (22)

2. Microvascular complications.

2.1. Diabetic nephropathy. In fifteen years one third of diabetics type 1 have end-stage renal disease as kidneys can not filter correctly the blood. (4)

These patients have elevated protein urinary excretion and may need renal dialysis and transplantation. (22)

2.2. Diabetic retinopathy. Cataracts and glaucoma are common pathologies. (4)

Diabetes is the main etiology in new cases of blindness. (22)

2.3. Peripheral neuropathy. Symptoms are postural hypotension (autonomic neuropathy), pain and loss of sensation in feet and hands. It can increase appearance of wounds and ulcers as patient is not aware. (4). Carpal tunnel syndrome and slowed digestion. (22)

3. Diabetic emergencies.

Hyperglycemia usually can cause long-term complications while hypoglycemia cause immediately life threatening situation. (28)

Hypoglycemic crisis, Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) need urgent identification, diagnosis and treatment.

Mortality in HHS is higher than in DKA. (29)

3. 1. Hypoglycemic crisis.

Hypoglycemia increases morbidity and mortality in diabetic patient (I and II DM). (28)

Diagnosis is made using Whipple's Triad:

- Hypoglycemic signs or symptoms
- Lower glucose value than normal (usually <70 mg/dl)
- Resolution of signs/symptoms + increase in plasma glucose. (28)

Risk factors: Old age, HbA1c <6.5%, renal disease, pregnancy, several diabetic medications, insulin therapy, previous history of hypoglycemia. (28)

Hypoglycemia counts from two to four per cent of deceased diabetic patients under insulin medication. (22)

Normal blood glucose level is 3.6-5.8 mmol/l (64.8-104.4 mg/dl)

Cognitive decline occurs at <3 mmol/l (54 mg/dl), although the threshold at which symptoms occur varies between individuals.

The most common trigger is an imbalance between insulin or oral hypoglycemic agents administered versus the necessary one.

Other precipitants are exertion, insufficient carbohydrate intake, excess insulin and alcohol. (22)

Symptoms are sweating, rapid heartbeat, hunger, tremors, irrational or violent behavior, irritability, alteration or total loss of consciousness, and seizures.

Recommended use of glucometers in clinics. Treatment will be chosen according to the level of consciousness and normally ends with the recovery of 90% of patients in twenty minutes. If she/he is conscious, oral glucose will be given (sugary food/drink, glucose gel) and if is unconscious, glucagon will be given;

Dose: Glucagon 1 mg, SC, IM or IV in adults and children <25 kg 0.5 mg of glucagon. (22). Alternatively, 50ml of dextrose 50% can be administered intravenously over 2-3 minutes. (30)

Glucagon normally takes 5-10 minutes to act. Check sugar with a glucometer after ten minutes to see if is increased to at least 5 mmol per liter or 90 mg per dl or more, and the patient's mental state improves. (22)

It is recommended, after glucagon, once patients are conscious and able to swallow, give a glucose food or drink. The patient can go home if he is recovered, he is not going to drive and is accompanied. (22) If situation worsens, call emergencies. (30)

3. 2. Hyperglycaemic crisis.

Decreased insulin level can cause DKA (Diabetic Ketoacidosis) and hyperosmolar non-ketotic hyperglycaemia (HONK) or hyperglycemic hyperosmolar state (HHS). Both can happen in type 1 and 2 DM

Decreased insulin level cause higher levels of blood glucose and lead to osmotic diuresis, sodium and water loss. This can cause shock and hypotension. (22)

Diabetic Ketoacidosis occurs because of absolute or almost absolute insulin deficiency and can develop as fast as hours or 1-2 days. HHS occurs when there is severe relative insulin deficiency and can take days to weeks to develop. Patients with HHS present plasma glucose level >30 mmol/L, serum osmolality >320 mOsm/Kg and pH>7.30. Patient with DKA, blood pH is <7.30 and will have concentrations of ketones of 5 mEq/L or greater. (30) Values are further extended in table 7.

When glucose levels in blood grow, the renal glucose threshold is overloaded, so urine changes into more watery, causing polyuria, polydipsia and dehydration. (31)

Mortality level are about 15% for HHS and 5% for DKA. Although they can both happen in the two types of DM, HHS happen more common in DM type 2 and DKA in type 1 DM. Main trigger for HHS is infection while for DKA is delay in diagnosis and infection. Other triggers include alcohol, myocardial infarction and drugs as corticosteroids and thiazines. (32)

Diabetic ketoacidosis (DKA).

Patient suffering this condition will have uncontrolled hyperglycemia, metabolic acidosis, and increased ketone concentration. (31)

Increased lipolysis produces non-esterified fatty acids, that are oxidized in the liver and convert into ketones.

Symptoms are polydipsia, polyuria, anorexia, vomiting, and abdominal pain. The most characteristic symptoms are deep and rapid breathing (Kussmaul's breath) and the acetone smell in the breath.

These conditions normally develop slowly and it is rare to occur abruptly in the dental practice.

If this condition is suspected, emergency calls should be made, and if the level of consciousness is lowered or there is a coma, in addition to calling the emergency, basic life support and supplemental oxygen must be administered. It happens more often in young people with diabetes type 1.

(22)

Hyperglycemic hyperosmolar state (HHS).

HHS causes altered mental status due to hyperosmolality, severe dehydration, and serious hyperglycemia without ketoacidosis or low. (31). HHS happens more common in type 2 DM. (33)

Despite of DKA and HHS share similar characteristics, and treatment is similar, it should focus differently. Patients with DKA need insulin to reverse ketoacidosis. Patients with HHS require fluid resuscitation first. (31)

Table 7. Diagnosis criteria DKA vs HHS.

	Mild DKA	Moderate DKA	Severe DKA	HSS
Plasma glucose (mmol/l)	13.9 (>250mg/dl)			33,3 (>600mg/dl)
Arterial pH	7.25-7.30	7.-7.24	<7	>7.30
Bicarbonate (mmol/l)	15-18	10-14	<10	>15
Anion gap mmol/l	>10-12			<12
Serum osmolarity mmol/kg	Variable			>320
Blood/urine acetoacetate (nitroprusside reaction)	Present			Not present or low
Urine/blood B-hydroxybutyrate (mmol/l)	>3			<3
Ketonemia/ketonuria	Present			Absent/mild
Conciousness	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

Table 7 shows difference of values between DKA and HHS. (29)

General treatment for DKA and HHS.

1. Check airway, breathing and circulation (A-B-C). Also asses patient position.

2. Obtain data of blood glucose, oxygen saturation, cardiac monitoring, heart and breath rate, blood pressure, temperature, electrocardiogram to seek arrhythmias and signs of hypo and hyperkalemia.

3. Call emergencies and start therapy. Some therapy can only be done in hospitals, treatment at clinic can be; give oxygen and hydratation (water). (31)

It is necessary to empathize that the following treatments are treatments carried out in hospitals that are beyond the skills of the dentist but it is interesting to know them.

Intravenous Fluid (saline 0,9% NaCl).

Critical patients with serious hyperglycemia (Ketoacidosis or hyperosmolar state) need urgently a bolus of normal saline.

DKA typical fluid deficit is 3–5L so patients need a fast bolus of 1L of saline and after one infusion of saline during hours at rate 500 ml/hour.

In case of HHS, the dehydration is worse, with fluid deficits of 10 liters or more. However, as they tend to be older and sicker, the resuscitation must be done carefully with a rapid bolus of 250 ml of saline repeated as needed and then a continuous infusion during hours (150–250 ml/hour). (31)

Insulin treatment.

First, a loading dose of 0.1 units/kg insulin (ten units maximum) and then an insulin regular infusion of 0.1 units/kg/h (ten units/hour max). Insulin can not be given until hypovolemia is over and serum potassium > 3.5 mEq/L.

Administering insulin to patients who have a serum potassium level < 3.5 mEq/L can cause life-threatening arrhythmias. (31) Intravenous insulin is administered

at hospitals to treat ketoacidosis and hyperglycemia. It is very important to know the potassium level of the patient before administering it because of the risk of hypokalemia (low potassium) and fatal arrhythmias.

Replacement of electrolytes.

- *Potassium (K)*. DKA or HHS patients suffer fast change in K level during resuscitation which can provoke dangerous arrhythmias. Death when starting resuscitation of DKA patients is normally due to hyperkalemia, however death after treatment already initiated is mostly caused by hypokalemia. Serum potassium must be verified each two hours in hyperglycemic crisis until patient is stable. (31)

If Serum K >5.0 mmol/l no more potassium is needed; if 4–5 mmol/l (add 20 mmol); if 3–4 mmol/l (add 40mmol); if <3 mmol/l add 10–20 mmol per hour until level >3 mmol/l. (29)

- *Bicarbonate.* DKA patients have hypophosphatemia during resuscitation that can cause respiratory depression, so if serum phosphate < 1.5 mg/dl it needs supplementation. (31)

Bicarbonate should be avoided for treating DKA or HSS. Only if DKA and severe acidosis (pH < 6.9). Bicarbonate can not be given if pH ≥7.0. (29)

Complications of the treatment can be; hypoglycemia due lowering blood sugar too fast (insulin); hypokalemia (lower K) because insulin and liquids used to treat DKA can cause that the level of potassium drop down (affecting heart, muscles and nerves); and cerebral edema due to too rapid adjustment of the blood sugar level (more common in children recently diagnosed with diabetes). (32)

Table 8. Summarized causes, signs and treatment for DM emergencies. (4)

Hypoglycemia	Hyperglycemia
Causes: excess insulin, exercise, alcohol or eating too little. Signs: Hot sweat, tachycardia, dilated pupils, anxiety, fear, aggressiveness, tingling in the mouth, headache, dysarthria, disorientation, unconsciousness.	Causes insulin defect, infections or myocardial infarction. Signs: Vomiting, hyperventilation, ketonuria, acetone breath, abdominal pain, low blood pressure, high heart rate, dry mouth and dry skin.
Treatment	
If the patient is conscious, administer 25 g glucose orally. If the patient is unconscious, administer glucagon 1 mg intramuscularly or dextrose 20 mg intravenously. Call EMS.	Rehydration of the patient and oxygen. Call EMS (emergencies).

4.5. Special precautions at diabetic patient.

When it comes to managing a diabetic patient, there are a series of measures that must be to adopt in the dental clinic:

1. Make sure that the patient has a good education about his illness, that knows well the medicines and the habits of life that must be taken. (4) Make emphasis on preventive measures. (22)

2. The well-controlled diabetic patient does not present any contraindication to be treated in the dental clinic, although it is advisable to take precautions. (4) Oral surgical procedures should be limited to when the blood sugar is less than 200 mg/dl and the patient is stable. (23) Well-controlled glucose value is needed for correct healing and prevent infection. (13)

3. The appointments should preferably be given at the morning to lower the risk of hypoglycemic crisis since patients can be more stable and tolerate treatments better. (4). Several short appointments would be better than one long visit. (22)

4. It is advisable to have a glucometer that allows to determine the capillary blood glucose instantly (hypo / hyperglycemia). (4)

5. It is essential to collect any systemic complication in the clinical history, especially kidney disease, for the management of antibiotic therapy. (4) We have to ask the patients if their DM is well controlled or not (more chance to suffer hypoglycemia), ask hypoglycemia symptoms if they have had one already and value of HbA1c. (14)

6. Monitor glycosylated hemoglobin; value HbAc1<7% indicates good control in the last 3 months, 7-9% regular control and >9% poor control. (4). For diabetic patient, the target is to achieve HbA1c values < 7% (healthy people range is 4-6%). (22)
A high HbA1c level would increase risk of infection and worsen the ability to heal. Any elective surgery should be postponed if patient has ketones (until resolution, usually with insulin). (14)

7. Plan visits in periods of no more than 3 months. (4). Patients with bad glucose control require more visits to dental clinic to control and treat their oral diseases. (22)

8. The use of antibiotics in diabetic people to prevent infection after surgery is a controversial point of view in last years. It would be cautious to give prophylactic covering to those that have inadequate glucose control (until more evidence will be available) but depends of doctor´s criteria. (22)

9. Oral infections need rigorous monitoring as systemic implication may be fast and may need hospitalization. Dental infections may cause DKA in patients with type 1 DM. (22)

10. Many drugs as NSAIDs, some antibiotics and antifungal can provoke interactions with oral hypoglycaemic agents and consequently strengthen their hypoglycaemia. (22)

11. Hypoglycemia is the main emergency that commonly face dentists when treating patients with DM. Dentists and staff must be trained to recognize and treat this kind of emergency. Patients that have more chance to develop hypoglycemia usually are the ones under insulin medication. Patients who take OHAs (oral antihyperglycemic agents) are less likely to suffer hypoglycemia than the ones taking insulin, but risk is higher when patients have hepatic or renal disorders. (13)

12. Dental office must prepare protocols for managing hypoglycemia. It is advised to have some snacks and glucose gels; these last ones are quite useful for uncooperative children or adults. Patients having an unusual behavior and

diaphoretic (sweating) should raise suspicion and glucose level must be tested. (13)

13. For patients under insulin therapy is recommended to bring their own glucometers. (13) Also to bring Insulin (pen or syringe), glucose or snack. (14)

14. When sedation or general anesthesia, glucose values have to be checked before the treatment and each hour when surgery is prolonged. (13) Conscious sedation for dental treatments is safe. Sedation doctors should use the ASA classification (American Society of Anesthesiologists). Well-controlled insulin-dependent diabetics (assuming there is no other diseases or pathologies) are ASA type II. (14) ASA II is controlled systemic disease. Uncontrolled diabetes with vascular complications would be ASA III (limiting but not disabling severe disease), even class IV if it affects more organs (disabling). Typically, ASA 3 or 4 patients should have an evaluation with an anesthesiologist if they require surgery. (34)

15. When glucose levels are high to the level that the patient has abnormal consciousness, delay the treatment. (13)

16. When the postoperative care instructions are given, emphasis must be placed on having good glycemic control during the healing phase. Also to inform the doctor of the patient to help them to control the glucose level. (13)

17. The loss of pain sensation related with diabetes usually affects the distal extremities. Joint flexibility can be affected on these patients, and therefore, in procedures that require immobility for a long period of time, can be advisable to take breaks so the patient can move their joints. The relationship of osteoporosis with type 1 diabetes is good established, so for this reason, dentists must be very vigilant in treatments that involve bones. Bone density is usually kept, but there is an increased risk of fractures attributed to drops in glucose due to hypoglycemia. (13)

18. Dentist can use some tests as the ADA (American Diabetes Association) DM risk calculator during the medical history, being able to detect people with possible undiagnosed Diabetes. (13)

19. If the patient has a tingling of the lips, it is prudent to make a glucose test previous to an ID block (inferior alveolar nerve block). If not, a block would eliminate the possibility of recognizing a coming hypoglycemia. (14)

5. Conclusions.

- Diabetes Mellitus is a very common metabolic disease worldwide, potentially fatal due to its complications and increasing due to obesity and unhealthy habits. As a consequence, a dentist will treat more and more diabetic patients. Special precautions must be taken, highlighting glucose monitoring, and short and early appointment.

- The clinical diagnosis of diabetic patients or the control of them in already diagnosed cases will be done mainly with a glucometer. The dentist must know the normal values, prediabetes and diabetes. A diabetic is diagnosed with these three main values; fasting glucose ≥ 126mg/dl, glucose after eating ≥200mg/dl and AC1 ≥6.5%.

- It is common for dentists to diagnose the disease due to the numerous oral pathologies related to diabetes in which periodontal disease stands out and will be the dentists the ones to diagnose and treat them.

- Since it is a very common disease, it is important that the dentist has full knowledge of the disease, as well as its causes, risk factors, types, treatments, pathologies, complications and emergencies.

- Diabetic patients, depending on type and severity, will be taking oral hypoglycaemic agents and insulin, these have side effects and complications that dentist must be able to diagnose and treat, highlighting hypoglycemia as the most common in the dental clinic, and being insulin and sulfonylureas the ones that

commonly cause it. Also, there are interactions of these diabetic drugs with other medications that dentists prescribe as painkillers, antibiotics and antifungals.

- It is also important to advise our patients about oral hygiene techniques since they are more prone to certain oral diseases. Additionally, teach healthy lifestyles to enrich their quality of life and avoid complications.

6. Responsibility.

In the social scope, diabetes is a disease on the rise due to unhealthy habits and obesity, at least type 2, this is something that can be changed by taking care of lifestyle. Every day there are more diseases that we cause ourselves with our actions such as smoking, not exercising and high calorie diet. Dentists like other doctors must emphasize that the patients take care of their health especially from an early age, focusing on prevention. Regarding the economic field, as frequency of disease grows, increases the costs of hospitals to cover doctors, medicines, surgeries and studies as well as the patient's own expenses, this project helps to review the knowledge about diabetes and how to advise and prevent its worsening, and its oral affectation, avoiding future health costs.

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8. Annexes.

8.1. Annex 1. Summarized etiologic classification of Diabetes Mellitus. (13)

- Type 1 DM. Immune or idiopathic.

- Type 2 DM.

- Gestational DM.

- Monogenic DM. Bizarre. β-cell function gene defects as MODY1, MODY 2 (maturity onset DM young) and mitochondrial ADN.

- Exocrine pancreas disease. Pancreatitis, neoplasia, cystic fibrosis and pancreatectomy.

- Endocrinopathies. Acromegaly, Cushing syndrome and Hyperthyroidism.

- Drug or chemical-induced DM. Thiazine, glucocorticoids, diazoxide, niacin, β-adrenergic agonists.

- Infections. Cytomegalovirus and Congenital rubella.

- Rare forms of immune-mediated DM as “Stiff-man” syndrome.

- Genetic syndromes associated with DM. Turner’s, Klinefelter’s, Wolfram’s and Down’s syndrome.

8.2. Annex 2. Medicines that increase or decrease glucose level. (19)

Drugs that lower blood glucose.

- AAS (Aspirin)
- Atorvastatin
- Bisoprolol
- Bupropion
- Captopril
- Cilazapril
- Cyproheptadine
- Clarithromycin
- Dexketoprofen
- Enalapril
- Haloperidol
- MAOI
- Imidapril
- Orlistat
- Propranolol

Drugs that increase blood glucose.

- Acetazolamide
- Adrenaline
- Tricyclic antidepressants
- Beta-agonists
- Lithium carbonate
- Thiazide diuretics
- Estrogens
- Furosemide
- Glucagon
- Glucocorticoids
- Heparin
- Indomethacin
- Morphine
- Nitrofurantoin
- Tacrolimus

8.3. Annex 3. Documents used.

1.

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ORIGINAL ARTICLE

Diabetes management and treatment approaches outside of North America and West Europe in 2006 and 2015

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Abstract

Aims The impact of introducing new classes of glucose-lowering medication (GLM) on diabetes management remains unclear, especially outside North America and Western Europe. Therefore, we aimed to analyse trends in glycaemic control and the usage of new and old GLMs in people with type 2 diabetes from 2006 to 2015.

Methods Summary data from clinical services from nine countries outside North America and Western Europe were collected and pooled for statistical analysis. Each site summarized individual-level data from out-patient medical records for 2006 and 2015. Data included: demographics; HbA1c and fasting plasma glucose levels; and the proportions of patients taking GLM as monotherapy, combination therapy and/or insulin.

Results Between 2006 and 2015, glycaemic control remained stable, although body mass index and duration of diabetes increased in most sites. The proportion of people on GLM increased, and the therapeutic regimens became more complex. There were increases in the use of insulin and triple therapy in most sites, while monotherapy, particularly in relation to sulphonylureas, decreased. Despite the introduction of new GLMs, such as DPP-4 inhibitors, insulin use increased over time.

Conclusions There was no clear evidence that the use of new classes of GLMs was associated with improvements in glycaemic control or reduced the reliance on insulin. These findings were consistent across a range of economic and geographic settings.

Keywords Diabetes mellitus · Glycated haemoglobin · Disease management · Therapeutics · Hypoglycaemic agents

Introduction

Based on the most recent International Diabetes Federation (IDF) report, the number of people with diabetes will increase from 425 million people in 2017 to 629 million by 2045 [1]. The prevalence of diabetes is increasing at a greater rate in some regions, such as Asia and the Middle East compared to the Western world [1, 2]. Furthermore, approximately 80% of people with diabetes reside in low- and middle-income countries (LMIC), representing a huge economic burden to these nations [3].

The importance of glycaemic control in preventing and delaying the progression of diabetes complications is well-established [4–6]. Despite considerable efforts undertaken in introducing new classes of glucose-lowering medications (GLM) and formulating guidelines for the use of these therapies to optimise glycaemic control [7], little is known about how this is actually put into practice in the different healthcare settings around the world and whether their introduction has led to significant improvement in glycaemic control.

A modest amount of information on the use of medications and the achievement of treatment targets is now

Managed by Massimo Federici.

Dianna J. Magliano and Jonathan E. Shaw have contributed equally on this paper.

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2.

Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2014 Mar; 158(1):117-123.

Severity of periodontal disease in adult patients with diabetes mellitus in relation to the type of diabetes

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Background. The purpose of this study was to evaluate associations between diabetes mellitus - related factors and periodontal parameters among adult patients with diabetes mellitus, with respect to type of diabetes.
Methods. Study participants were 179 randomly selected 18-62-year-aged patients with type 1 diabetes mellitus and 87 randomly selected 32-70-year-aged patients with type 2 diabetes. Metabolic control of diabetes was determined by the values of glycosylated haemoglobin (HbA1c). The periodontal status of all patients was evaluated by simplifying oral debris index (DI-S), probing pocket depth (PPD), gingival recession (GR), clinical attachment level (CAL), and bleeding on probing (BOP). Data analysis was performed with respect to patients' age, diabetes duration, metabolic control level, and diabetes type. Binary regression was used to test relationship of various parameters with CAL.
Results. All periodontal estimates were significantly higher among patients with type 2 diabetes. The periodontal disease was more severe in >45-year-aged participants and with DI-S>1. In patients with type 1 diabetes, the disease duration >12 years was negatively related to most periodontal parameters. No significant correlation between the periodontal estimates and HbA1c was observed in either group. The significant predictors of severe periodontal disease were type 2 diabetes mellitus (OR = 2.356), duration of disease (OR = 1.827), high BOP (OR = 3.343) and DI-S (OR = 2.958).
Conclusions. Severity of periodontal disease is related to diabetes type, being more pronounced in patients with type 2 diabetes patients than in patients with type 1 diabetes. Dental plaque seems to be the major contributing factor for all patients with progressive periodontitis.

Key words: diabetes mellitus type 1, diabetes mellitus type 2, periodontitis

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INTRODUCTION

Periodontitis and diabetes mellitus (DM) are prevalent chronic diseases that influence patients' health and quality of life. Periodontitis is defined as a chronic bacterial inflammatory process with the symptoms of gingival bleeding, clinical attachment loss, formation of periodontal pockets and gingival recessions leading to destruction of alveolar bone[1]. Although mild forms such as gingivitis or initial loss of periodontal attachment are very common in all age cohorts, advanced stages of periodontitis usually affect a relatively small proportion of the population[1,2]. It seems that there is no direct correlation between chronic gingival inflammation and further progression of periodontal breakdown, and lack of evidence on prognostic indicators of severe periodontal disease in those people who develop rapidly progressing periodontal symptoms (2). DM is a metabolic disorder characterized by hyperglycaemia, leading to impaired metabolism of glucose, lipids and proteins[3]. In 2011 DM affected 6% of the European population from 20 to 79 years, meaning 52.6 million people with DM[4]. According to the International Diabetes Federation this number may rise to 7.1%, or 64 million by 2030 (ref.[4]). Two types of DM differ with respect to physiopathology: type 1 DM (T1DM), an autoimmune disorder that leads to destruction of pancreatic β cells and thus total loss of insulin secretion; and type 2 DM (T2DM) that is linked to insulin resistance[5]. Chronic hyperglycaemia leads to long-term micro and macro vascular complications such as retinopathy, nephropathy, neuropathy and cardiovascular diseases, resulting in increased morbidity and premature mortality[6]. A large number of epidemiological studies have demonstrated that periodontitis is more prevalent and severe in patients with DM when compared to persons without DM (ref.[7]). Moreover, DM has been suggested as a significant risk factor for periodontal disease[8]. The mechanism of DM effect on the course of periodontal pathology is still not clear, but the severity of both diseases (DM and periodontitis) was shown to be dependent on various risk factors such as duration and metabolic control of DM as well as the patient's age, social behaviour, oral hygiene level and certain aggravating factors (e.g., smoking) (ref.[6,7]). The majority of research has focused on the relationship between periodontitis and DM of one particular type (either T1DM or T2DM) while the possible differences in the course of periodontal disease with respect to the different physiopathology of DM have not been studied. The aim of the

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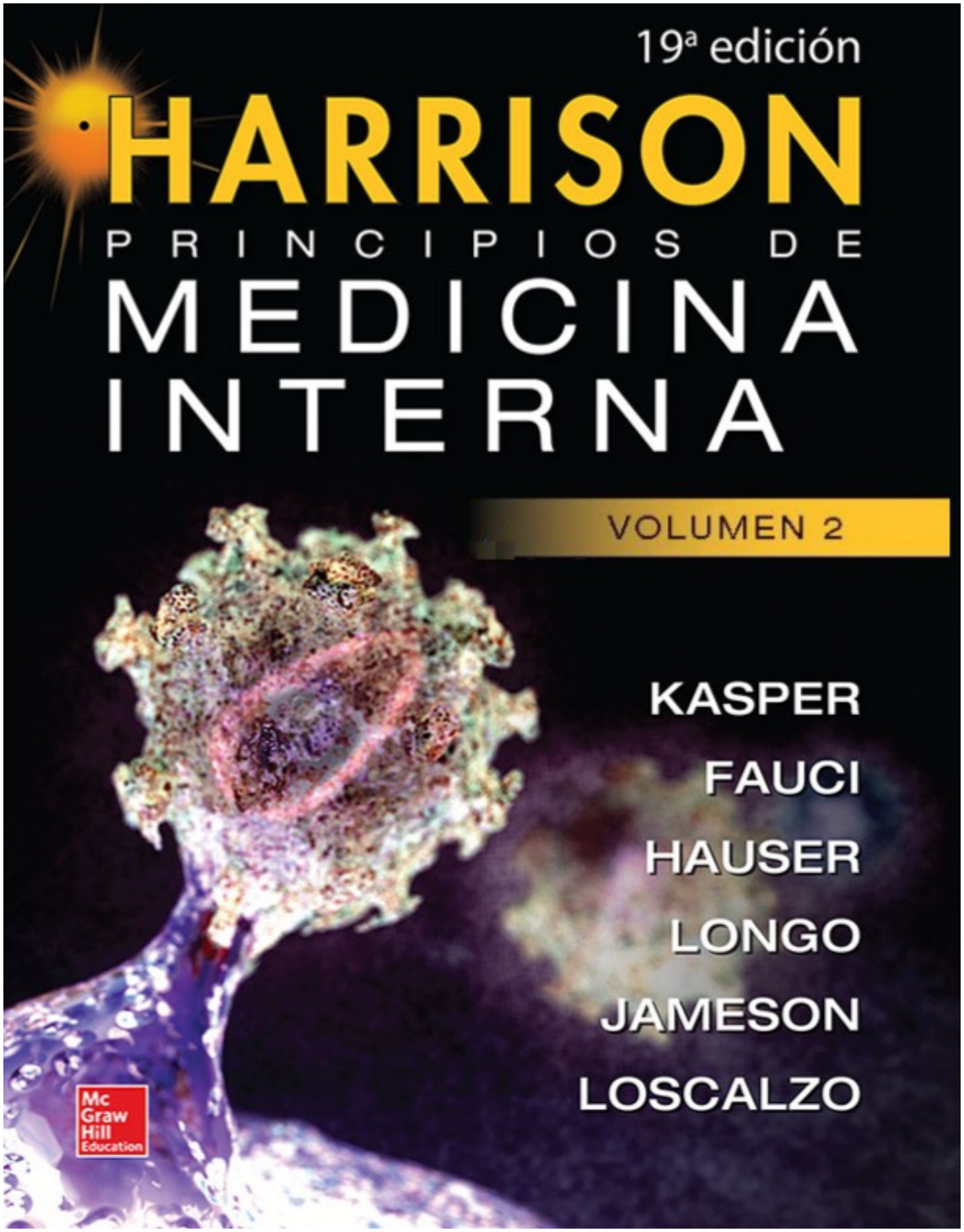
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4.

Diabetes mellitus: Su implicación en la patología oral y periodontal

Diabetes: oral and periodontal implications

Sanz-Sánchez I*, Bascones-Martínez A**

RESUMEN

Introducción: La diabetes es una enfermedad metabólica de alta prevalencia y con gran morbilidad, por lo que las medidas preventivas de las complicaciones derivadas y el control de la enfermedad son esenciales. Los objetivos son: a) diferenciar las rutas patogénicas que intervienen en la diabetes mellitus tipo 1 y 2; b) conocer cuáles son las complicaciones de la diabetes a nivel general y a nivel oral; c) hacer un repaso de la asociación entre la diabetes y la enfermedad periodontal.
Material, métodos y resultados: Para la realización de este trabajo se han analizado 36 artículos científicos y un libro de texto. Para la búsqueda se han empleado la base de datos MEDLINE y Cochrane.
Discusión: Existen dos rutas patogénicas que diferencian claramente la diabetes tipo 1 de la 2. Además, nos vamos a encontrar con un gran número de posibles complicaciones a largo plazo. Especial mención requiere la asociación existente entre la periodontitis y la diabetes.

Palabras clave: Diabetes, metabolic syndrom, insulin, autoimmunity, periodontitis, caries, oral disease.

SUMMARY

Introduction: Diabetes is a high prevalent metabolic disease with a high morbidity. That is why preventive measurements of the linked complications and the control of the disease are essential. The objectives are: a) differentiate the pathogenic ways that take place in diabetes mellitus type 1 and 2; b) to know which are the general and oral complications of diabetes; c) make a summary of the association between diabetes and periodontal disease.
Materials, methods and results: For the preparation of this work, we employed 36 scientific articles and one text book. The MEDLINE and Cochrane databases have been used to make the search.
Discussion: We can find two clearly different pathways that differentiate diabetes type 1 from type 2 have been analyced. We can also find lots of long term complications. The association between diabetes and periodontal disease requires a special mention.

Key words: Diabetes, metabolic syndrom, insulin, autoimmunity, periodontitis, caries, oral disease.

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5.

POSITION STATEMENT

Diagnosis and Classification of Diabetes Mellitus

AMERICAN DIABETES ASSOCIATION

DEFINITION AND DESCRIPTION OF DIABETES MELLITUS

— Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels.

Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the β-cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action. Impairment of insulin secretion and defects in insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycemia.

Symptoms of marked hyperglycemia include polyuria, polydipsia, weight loss, sometimes with polyphagia, and blurred vision. Impairment of growth and susceptibility to certain infections may also accompany chronic hyperglycemia. Acute, life-threatening consequences of uncontrolled diabetes are hyperglycemia with ketoacidosis or the nonketotic hyperosmolar syndrome.

Long-term complications of diabetes include retinopathy with potential loss of vision; nephropathy leading to renal failure; peripheral neuropathy with risk of foot ulcers, amputations, and Charcot joints; and autonomic neuropathy causing gastrointestinal, genitourinary, and cardiovascular symptoms and sexual dysfunction. Patients with diabetes have an increased incidence of atherosclerotic cardiovascular, peripheral arterial, and cerebrovascular disease. Hypertension and abnormalities of lipoprotein metabolism are often found in people with diabetes.

The vast majority of cases of diabetes fall into two broad etiopathogenetic categories (discussed in greater detail below). In one category, type 1 diabetes, the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers. In the other, much more prevalent category, type 2 diabetes, the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. In the latter category, a degree of hyperglycemia sufficient to cause pathologic and functional changes in various target tissues, but without clinical symptoms, may be present for a long period of time before diabetes is detected. During this asymptomatic period, it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load.

The degree of hyperglycemia (if any) may change over time, depending on the extent of the underlying disease process (Fig. 1). A disease process may be present but may not have progressed far enough to cause hyperglycemia. The same disease process can cause impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) without fulfilling the criteria for the diagnosis of diabetes. In some individuals with diabetes, adequate glycemic control can be achieved with weight reduction, exercise, and/or oral glucose-lowering agents. These individuals therefore do not require insulin. Other individuals who have some residual insulin secretion but require exogenous insulin for adequate glycemic control can survive without it. Individuals with extensive β-cell destruction and therefore no residual insulin secretion require insulin for survival. The severity of the metabolic abnormality can progress, regress, or stay the same. Thus, the degree of hyperglycemia reflects the severity of the underlying metabolic process and its treatment more than the nature of the process itself.

CLASSIFICATION OF DIABETES MELLITUS AND OTHER CATEGORIES OF GLUCOSE REGULATION

— Assigning a type of diabetes to an individual often depends on the circumstances present at the time of diagnosis, and many diabetic individuals do not easily fit into a single class. For example, a person with gestational diabetes mellitus (GDM) may continue to be hyperglycemic after delivery and may be determined to have, in fact, type 2 diabetes. Alternatively, a person who acquires diabetes because of large doses of exogenous steroids may become normoglycemic once the glucocorticoids are discontinued, but then may develop diabetes many years later after recurrent episodes of pancreatitis. Another example would be a person treated with thiazides who develops diabetes years later. Because thiazides in themselves seldom cause severe hyperglycemia, such individuals probably have type 2 diabetes that is exacerbated by the drug. Thus, for the clinician and patient, it is less important to label the particular type of diabetes than it is to understand the pathogenesis of the hyperglycemia and to treat it effectively.

Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)

Immune-mediated diabetes. This form of diabetes, which accounts for only 5–10% of those with diabetes, previously encompassed by the terms insulin-dependent diabetes, type I diabetes, or juvenile-onset diabetes, results from a

The information that follows is based largely on the reports of the Expert Committee on the Diagnosis and Classification of Diabetes (*Diabetes Care* 20:1183–1197, 1997, and *Diabetes Care* 26:3160–3167, 2003).

Abbreviations: FPG, fasting plasma glucose; GAD, glutamic acid decarboxylase; GCT, glucose challenge test; GDM, gestational diabetes mellitus; HNF, hepatocyte nuclear factor; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; MODY, maturity-onset diabetes of the young; WHO, World Health Organization.

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6.

Significance of HbA1c Test in Diagnosis and Prognosis of Diabetic Patients

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ABSTRACT: Diabetes is a global endemic with rapidly increasing prevalence in both developing and developed countries. The American Diabetes Association has recommended glycated hemoglobin (HbA1c) as a possible substitute to fasting blood glucose for diagnosis of diabetes. HbA1c is an important indicator of long-term glycemic control with the ability to reflect the cumulative glycemic history of the preceding two to three months. HbA1c not only provides a reliable measure of chronic hyperglycemia but also correlates well with the risk of long-term diabetes complications. Elevated HbA1c has also been regarded as an independent risk factor for coronary heart disease and stroke in subjects with or without diabetes. The valuable information provided by a single HbA1c test has rendered it as a reliable biomarker for the diagnosis and prognosis of diabetes. This review highlights the role of HbA1c in diagnosis and prognosis of diabetes patients.

KEYWORDS: diabetes, HbA1c, diagnosis, prognosis, blood test

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Introduction

Analysis of glycated hemoglobin (HbA1c) in blood provides evidence about an individual's average blood glucose levels during the previous two to three months, which is the predicted half-life of red blood cells (RBCs).[1] The HbA1c is now recommended as a standard of care (SOC) for testing and monitoring diabetes, specifically the type 2 diabetes.[2] Historically, HbA1c was first isolated by Huisman et al.[3] in 1958 and characterized by Bookchin and Gallop[4] in 1968, as a glycoprotein. The elevated levels of HbA1c in diabetic patients were reported by Rahbar et al.[5] in 1969. Bunn et al.[6] identified the pathway leading to the formation of HbA1c in 1975. Using the HbA1c as a biomarker for monitoring the levels of glucose among diabetic patients was first proposed by Koenig et al.[7] in 1976.

Proteins are frequently glycated during various enzymatic reactions when the conditions are physiologically favorable. However, in the case of hemoglobin, the glycation occurs by the nonenzymatic reaction between the glucose and the N-terminal end of the β-chain, which forms a Schiff base.[8,9] During the rearrangement, the Schiff base is converted into Amadori products, of which the best known is HbA1c (Fig. 1). In the primary step of glycated hemoglobin formation, hemoglobin and the blood glucose interact to form aldimine in a reversible reaction. In the secondary step, which is irreversible, aldimine is gradually converted into the stable ketoamine form.[10] The major sites of hemoglobin glycosylation, in the order of prevalence, are β-Val-1, β-Lys-66, and α-Lys-61. Normal adult hemoglobin consists predominantly of HbA ($\alpha_2\beta_2$), HbA2 ($\alpha_2\delta_2$), and HbF ($\alpha_2\gamma_2$) in the composition of 97%, 2.5%, and 0.5%, respectively. About 6% of total HbA is termed HbA1, which in turn is made up of HbA1a1, HbA1a2, HbA1b, and HbA1c fractions, defined by their electrophoretic and chromatographic properties. HbA1c is the most abundant of these fractions and in health comprises approximately 5% of the total HbA fraction. As mentioned above, glucose in the open chain format binds to the N-terminal to form an aldimine before undergoing an Amadori rearrangement to form a more stable ketoamine. This is a nonenzymatic process that occurs continuously *in vivo*. The formation of the glycated hemoglobin is a normal part of the physiologic function cycle. However, as the average plasma glucose increases, so does the amount of glycated hemoglobin in the plasma. This specific characteristic of the hemoglobin biomarker is utilized for estimating the average blood glucose levels over the previous two to three months.[11] In this review, we have described the current

7.

6. Glycemic Targets: *Standards of Medical Care in Diabetes—2019*

American Diabetes Association

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The American Diabetes Association (ADA) "Standards of Medical Care in Diabetes" includes ADA's current clinical practice recommendations and is intended to provide the components of diabetes care, general treatment goals and guidelines, and tools to evaluate quality of care. Members of the ADA Professional Practice Committee, a multidisciplinary expert committee, are responsible for updating the Standards of Care annually, or more frequently as warranted. For a detailed description of ADA standards, statements, and reports, as well as the evidence-grading system for ADA's clinical practice recommendations, please refer to the Standards of Care Introduction. Readers who wish to comment on the Standards of Care are invited to do so at professional.diabetes.org/SOC.

ASSESSMENT OF GLYCEMIC CONTROL

Glycemic management is primarily assessed with the A1C test, which was the measure studied in clinical trials demonstrating the benefits of improved glycemic control. Patient self-monitoring of blood glucose (SMBG) may help with self-management and medication adjustment, particularly in individuals taking insulin. Continuous glucose monitoring (CGM) also has an important role in assessing the effectiveness and safety of treatment in many patients with type 1 diabetes, and limited data suggest it may also be helpful in selected patients with type 2 diabetes, such as those on intensive insulin regimens (1).

A1C Testing

Recommendations

6.1 Perform the A1C test *at least* two times a year in patients who are meeting treatment goals (and who have stable glycemic control). **E**

6.2 Perform the A1C test quarterly in patients whose therapy has changed or who are not meeting glycemic goals. **E**

6.3 Point-of-care testing for A1C provides the opportunity for more timely treatment changes. **E**

A1C reflects average glycemia over approximately 3 months. The performance of the test is generally excellent for NGSP-certified assays (www.ngsp.org). The test is the major tool for assessing glycemic control and has strong predictive value for diabetes complications (1–3). Thus, A1C testing should be performed routinely in all patients with diabetes—at initial assessment and as part of continuing care. Measurement approximately every 3 months determines whether patients' glycemic targets have been reached and maintained. The frequency of A1C testing should depend on the clinical situation, the treatment regimen, and the clinician's judgment. The use of point-of-care A1C testing may provide an opportunity for more timely treatment changes during encounters between patients and providers. Patients with type 2

Suggested citation: American Diabetes Association. 6. Glycemic targets: Standards of Medical Care in Diabetes—2019. *Diabetes Care 2019; 42(Suppl. 1):S61–S70*

8.

La hemoglobina glucosilada A1c como prueba diagnóstica para diabetes mellitus en adolescentes con sobrepeso u obesidad

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Eulalia Garrido-Magaña,[a] Gigliola Margaretta Fiorentini-Fayad,[a]
Elisa Nishimura-Meguro[a]

Glycosylated hemoglobin A1c as a diagnostic test for diabetes mellitus in adolescents with overweight and obesity

Background: In 2009 it was introduced a new diagnostic criteria based on hemoglobin A1c (HbA1c) ≥ 6.5 % in the adult population; some studies suggest that the cutoff may be smaller in pediatric population. The objective was to determine the utility of HbA1c ≥ 6.5 % as a diagnostic test for DM in Mexican adolescents with overweight or obesity.
Methods: Full somatometry was performed. Also, Tanner stage, blood pressure, blood glucose, glucose tolerance curve (GTC) and HbA1c were analyzed. Specificity, sensitivity, positive and negative predictive values and ROC curve were calculated for the diagnosis of DM with HbA1c.
Results: 109 adolescents between 10 and 16 years referred for obesity or overweight plus comorbidities were studied; 58 % were females, the age was of 13 ± 1.74 years, the BMI percentile 95.3, and the HbA1c 5.73 ± 0.9 %. It was made a diagnosis of DM in 9 cases (8.3 %), prediabetes in 8 (7.3 %) and normal glucose tolerance in 92 (84.4 %). The HbA1c mean was 5.6 ± 0.04, 5.7 ± 0.4, and 5.6 ± 0.73 %, respectively. HbA1c ≥ 6.5 % had a sensitivity of 12.5 %, a specificity of 89.8 %, a PPV of 10.65 and a NPV of 14.28. The best cutoff point for diagnosing DM through ROC curve was 5.45 %, with a sensitivity of 62.5 %, a specificity of 57.1 %, PPV 2.53 and NPV 33.3.
Conclusions: The level of HbA1c ≥ 6.5 % had low sensitivity and specificity for the diagnosis of DM. A lower cutoff point is insufficient to use HbA1c as a diagnostic criterion. These results are consistent with the ones of other journals.

Keywords	Palabras clave
Glycosylated hemoglobin A	Hemoglobina A glucosilada
Child	Niño
Adolescent	Adolescente

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La incidencia y la prevalencia de la diabetes mellitus tipo 2 (DM2) se ha incrementado rápidamente en todo el mundo, en particular en los niños y adolescentes. Este incremento se ha atribuido en parte al aumento de sobrepeso y obesidad en esta población, tanto en Estados Unidos como en todas partes del mundo.[1,2,3]

En el 2006, de acuerdo con los resultados de la Encuesta Nacional de Salud y Nutrición (ENSANUT), uno de cada tres hombres o mujeres adolescentes tenía sobrepeso u obesidad, lo que representó alrededor de 5 757 400 adolescentes en el país.[4] La aparición temprana de DM2 implica el desarrollo prematuro de complicaciones crónicas tanto microvasculares como macrovasculares. Estas últimas conllevan a una mayor mortalidad por enfermedad cardiovascular, cerebrovascular y vascular periférica.[5,6]

La DM2 es usualmente asintomática y los factores de riesgo para desarrollarla incluyen sobrepeso u obesidad y la carga genética. Otros datos que potencian este riesgo por medio del incremento de la resistencia a la insulina son la obesidad central, la hipertensión arterial sistémica, las dislipidemias, el síndrome de ovario poliquístico y la pubertad. La Asociación Americana de Diabetes (ADA, por sus siglas en inglés) recomienda el escrutinio para diagnóstico de DM2 con glucosa plasmática en ayuno para los niños con índice de masa corporal (IMC) ≥ percentil 85 y quienes además tengan dos factores adicionales de riesgo, por ejemplo: historia familiar de DM en primer o segundo grado, pertenecer a una etnia de alto riesgo (indios americanos, afroamericanos, hispanos y asiáticos), datos de resistencia a la insulina o condiciones asociadas.[1,7]

El diagnóstico de DM se estableció con criterios basados en los niveles de glucosa plasmática en ayuno ≥ 126 mg/dL en dos ocasiones, glucosa plasmática ≥ 200 mg/dL en cualquier hora en presencia de síntomas y si se realizaba una curva de tolerancia oral a la glucosa (CTOG) y a las dos horas se determinaba una glucosa venosa ≥ 200 mg/dL, se confirmaba entonces este diagnóstico. En el 2009, un comité internacional de expertos, que incluyó representantes de la ADA, de la Federación Internacional de Diabetes (IDF, por sus siglas en inglés) y de la Asociación Europea para el Estudio de la Diabetes (EASD), recomendaron el uso de la hemoglobina glucosilada (HbA1c) para el diagnóstico de DM con un

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9.

POSITION STATEMENT

Standards of Medical Care in Diabetes–2006

AMERICAN DIABETES ASSOCIATION

CONTENTS

Originally approved 1988. Most recent review/revision, October 2005.

Abbreviations: ABI, ankle-brachial index; AMI, acute myocatdial infarction; ARB, angiotensin receptor blocker; CAD, coronary artery disease; CBG, capillary blood glucose; CHD, coronary heart disease; CHF, congestive heart failure; CKD, chronic kidney disease; CVD, cardiovascular disease; DCCB, dihydropyridine calcium channel blocker; DCCT, Diabetes Control and Complications Trial; DKA, diabetic ketoacidosis; DMMP, diabetes medical management plan; DPN, distal symmetric polyneuropathy; DPP, Diabetes Prevention Program; DRI, dietary reference intake; DRS, Diabetic Retinopathy Study; DSME, diabetes self-management education; DSMT, diabetes self-management training; ECG, electrocardiogram; ESRD, end-stage renal disease; ETDRS, Early Treatment Diabetic Retinopathy Study; FDA, Food and Drug Administration; FPG, fasting plasma glucose; GDM, gestational diabetes mellitus; GFR, glomerular filtration rate; HRC, high-risk characteristic; ICU, intensive care unit; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; MNT, medical nutrition therapy; NPDR, nonproliferative diabetic retinopathy; OGTT, oral glucose tolerance test; PAD, peripheral arterial disease; PDR, proliferative diabetic retinopathy; PPG, postprandial plasma glucose; RDA, recommended dietary allowance; SMBG, self-monitoring of blood glucose; TZD, thiazolidinedione; UKPDS, U.K. Prospective Diabetes Study.

Diabetes is a chronic illness that requires continuing medical care and patient self-management education to prevent acute complications and to reduce the risk of long-term complications. Diabetes care is complex and requires that many issues, beyond glycemic control, be addressed. A large body of evidence exists that supports a range of interventions to improve diabetes outcomes.

These standards of care are intended to provide clinicians, patients, researchers, payors, and other interested individuals with the components of diabetes care, treatment goals, and tools to evaluate the quality of care. While individual preferences, comorbidities, and other patient factors may require modification of goals, targets that are desirable for most patients with diabetes are provided. These standards are not intended to preclude more extensive evaluation and management of the patient by other specialists as needed. For more detailed information, refer to refs. 1–3.

The recommendations included are diagnostic and therapeutic actions that are known or believed to favorably affect health outcomes of patients with diabetes. A grading system (Table 1), developed by the American Diabetes Association (ADA) and modeled after existing methods, was utilized to clarify and codify the evidence that forms the basis for the recommendations. The level of evidence that supports each recommendation is listed after each recommendation using the letters A, B, C, or E.

I. CLASSIFICATION AND DIAGNOSIS

A. Classification

In 1997, the ADA issued new diagnostic and classification criteria (4); in 2003, modifications were made regarding the diagnosis of impaired fasting glucose (IFG) (5). The classification of diabetes includes four clinical classes:

- Type 1 diabetes (results from β-cell destruction, usually leading to absolute insulin deficiency).
- Type 2 diabetes (results from a progressive insulin secretory defect on the background of insulin resistance).
- Other specific types of diabetes due to other causes, e.g., genetic defects in

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Conocimiento, comportamiento y percepción sobre salud bucal en pacientes con diabetes tipo 2

Gabriela García-Morales,[a]
Denny Kennia Barrera-Lagunas[a]

Knowledge, behavior and perception of oral health in patients with type 2 diabetes

Introducción: la enfermedad periodontal se presenta de forma severa en los pacientes con diabetes, además ocasiona descontrol glucémico. El objetivo de este trabajo es determinar conocimiento, comportamiento y percepción sobre salud bucal en pacientes con diabetes tipo 2.
Métodos: estudio analítico transversal llevado a cabo en la Unidad de Medicina Familiar No. 26 con 376 pacientes con diabetes tipo 2. Se aplicó cuestionario de factores sociodemográficos, conocimiento, comportamiento y percepción de salud oral.
Resultados: 21% fue enviado al servicio dental por el médico familiar. 86% realiza cepillado dental de 2-3 veces al día; 57% percibió la relación diabetes y salud oral. En signos de enfermedad oral, predominaron sangrado gingival con 34% e inflamación con 30%. La razón principal de consulta dental fue por urgencia.
Conclusiones: la persona con diabetes no reconoce al estomatólogo como personaje principal en la atención integral de salud, aunado a ello el médico familiar no envía a estomatología para prevención. Es necesario elaborar estrategias educativas, enfocadas al médico familiar y paciente en cuanto a importancia de salud oral.

Background: Periodontal disease occurs severely in patients with diabetes, also causes high blood glucose. The objective of this article was to determine knowledge, behavior and perception of oral health in patients with type 2 diabetes.
Methods: Cross-sectional study performed at the Unidad de Medicina Familiar No. 26 in Acapulco, Guerrero, México; 376 patients with type 2 diabetes were included. Sociodemographic questionnaire, knowledge, behavior and perception of oral health was applied.
Results: 21% was sent to the dental service by the family doctor. 86% of the patients performed dental brushing 2-3 times a day; 57% perceived the relationship diabetes and oral health. In signs of oral disease they predominated with 34% gingival bleeding and inflammation with 30%. Main reason was for emergency dental surgery.
Conclusions: The patient suffering from diabetes does not recognize the dentist as the main character in the comprehensive healthcare; on the other hand, the family doctor does not send a prevention stomatology. It is necessary to develop educational strategies focused on the family doctor and patient in terms of importance of oral health.

Palabras clave
Salud bucal
Diabetes
Enfermedades periodontales

Keywords
Oral health
Diabetes
Periodontal diseases

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BMC Public Health

RESEARCH ARTICLE Open Access

A randomized controlled trial of an educational intervention to promote oral and dental health of patients with type 2 diabetes mellitus

Maryam Malekmahmoodi[1], Mohsen Shamsi[1*], Nasrin Roozbahani[1] and Rahmatollah Moradzadeh[2]

Abstract

Background: Diabetes is the most prevalent disease resulted from metabolic disorders. This study aimed to investigate the effect of training based on health belief model (HBM) on oral hygiene-related behaviors in patients with type 2 diabetes mellitus.

Methods: This study was conducted as an educational randomized controlled trial (single blind) on 120 patients with type 2 diabetes referring to a diabetes clinic selected through systematic sampling, who were assigned to two groups of control ($N = 60$) and intervention ($N = 60$). The data collection tool was a valid and reliable questionnaire based on HBM which was completed by both groups before the intervention. Then, the intervention group received 4 sessions of educational program based on HBM in 1 month, and the same questionnaire was completed again after 3 months and the data were analyzed through SPSS version 20 software with inferential statistics, t-test, paired t-tests, Chi square, Mann-Whitney test, and Wilcoxon test analysis.

Results: Three months after the intervention, awareness of the patients and perceived susceptibility, benefits, self-efficacy, internal cue to action, and performance in oral and dental hygiene-related behaviors had a significant increase in the intervention group ($p < 0.05$). So that the performance of oral and dental hygiene in the intervention group increased from 2.16 ± 0.71 to 3.25 ± 0.49 ($p = 0.001$) after the education.

Conclusion: Our results suggest that training patients with diabetes based on HBM as well as through active follow-up can enhance their skills in oral and dental hygiene-related behaviors. Controlling, monitoring and follow-up during the program are also recommended.

Trial registration: Iranian Registry of Clinical Trials, IRCT 2017050733847N1. Prospectively registered 14 June 2017, http://en.irct.ir/trial/26011

Keywords: Type 2 diabetes mellitus, Health belief model, Oral hygiene, Diabetes care

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12.

Original Paper

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Diabetes Mellitus and Periodontal Health: Dentists' Knowledge

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Key Words
Diabetes mellitus · Periodontal diseases · Knowledge of dentists

Abstract
Objectives: There is a strong body of evidence to support the relationship between periodontal diseases and diabetes mellitus. Unless dental practitioners are aware of this link, they cannot apply the information to their daily practice. The aim of the study was, therefore, to evaluate the knowledge of dental practitioners concerning the effect of diabetes on periodontal health. ***Subjects and Methods:*** This was a cross-sectional survey of randomly selected dental practitioners in Kuwait. Participants were asked about specific periodontal complications which they believed that patients diagnosed with diabetes were more susceptible to. ***Results:*** A total of 220 dental practitioners (133 general dental practitioners and 87 dental specialists) participated in the study. Less than 60% of all study participants reported that tooth loss due to periodontal reasons and periodontal abscess were frequent among diabetic patients. Dental specialists, especially periodontists, were significantly more aware of periodontal complications associated with diabetes. Factors significantly associated with having knowledge about the effect of diabetes on periodontal health in logistic regression analysis were dentists who were older and those who were specialists. ***Conclusions:*** The results of this study indicate that knowledge about the effects of diabetes on periodontal health among this sample of dental practitioners is generally low, and dentists may underestimate the outcomes of periodontal diseases in diabetic patients.

Introduction

A bidirectional relationship exists between diabetes mellitus and periodontal diseases [1, 2]. Patients diagnosed with diabetes are considered to be a high-risk group with greater susceptibility to severe forms of periodontal breakdown [3, 4]. Chronic periodontitis is a common periodontal disease among adults, a potentially progressive bacterial infection that may lead to tooth loss due to extensive destruction of periodontal attachment. In addition, inflammatory response plays a major role in the progression of periodontal diseases. Controlled clinical studies have shown that subjects diagnosed with diabetes have a greater prevalence of periodontal complications compared to healthy individuals,

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13.

Diabetes mellitus
Considerations for dentistry

Srividya Kidambi, MD; Shailendra B. Patel, BM, ChB, DPhil

ABSTRACT

Background. The connection between oral health and systemic health is bidirectional; systemic illnesses, especially metabolic disorders, affect oral health, and it appears that oral health may affect systemic health.
Methods. In this review, the authors outline the basic principles behind diabetes mellitus (DM) and provide some tips to help dentists manage the care of patients with DM better in general practice.
Results. DM negatively affects all microvasculature beds, and the soft tissues and bones supporting the teeth are susceptible. There is also strong evidence that the presence of periodontal disease is associated with increased cardiovascular morbidity in patients with DM.
Conclusions. DM is a chronic, systemic metabolic disorder in which the orosystemic connection is becoming more understood.
Clinical Implications. DM is a relatively common condition and, thus, is one that practicing dentists may encounter frequently.
Key Words. Diabetes; insulin; hypoglycemia; periodontal disease.
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JADA CE CONTINUING EDUCATION ARTICLE 2

Diabetes mellitus (DM) is a metabolic disorder characterized by impaired action, secretion of insulin or both, resulting in hyperglycemia. An estimated 20.8 million people in the United States (7 percent) have DM, and 1.5 million new cases were diagnosed in 2005.[1] Although the definition, the pathophysiological basis and much of management of DM is glucocentric, it is a true metabolic disorder, and a number of metabolic disturbances have been characterized.[2,3] In addition to experiencing well-known complications associated with DM such as premature cardiovascular disease, renal disease, retinopathy and neuropathy, about one-third of people with DM have severe periodontal disease. Attenuated immunity, which occurs as a result of hyperglycemia, and a variety of host factors associated with DM may be the pathophysiological basis for the increased prevalence and severity of periodontal disease.[4] In addition to altering the course of periodontal disease, the diabetic state influences treatment decisions. Osteoporosis increasingly is being associated with DM, which may affect the treatment of periodontal disease because of the involvement of mandib-

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14.

The diabetic patient and dental treatment: an update

L Wray[1]

VERIFIABLE CPD PAPER

PRACTICE

IN BRIEF

- Provides an update on the current management of Type 1 and Type 2 diabetes.
- Outlines the oral health implications of having diabetes.
- Advises on the safe dental treatment of the diabetic patient including under sedation.
- Discusses the possible effects of dental treatment on diabetic control.

This paper has been written to both refresh and update clinicians' knowledge of diabetes. Treatment for patients with diabetes continues to develop with the majority of Type 1 diabetics now using multiple daily injections and an increasing minority using insulin pumps. Blood glucose monitoring and patient education programmes have resulted in more patient involvement in controlling this condition. Type 2 diabetics have had improvement in care provision through the development of shorter acting sulphonylureas and the potential for GLP1 injections. The impact of diabetes on both oral health and quality of life is discussed. Practical suggestions are made regarding the dental treatment of diabetic patients using both local anaesthetic and under sedation. Diabetes continues to be a fickle master for those affected by this condition. The paper is written from the perspective of the 'expert patient'. It is hoped that a greater understanding of this chronic condition will improve both access to, and safety of, dental care for those patients with diabetes.

INTRODUCTION

As Martin Silink, past-president of the International Diabetes Foundation (2003-2006), expressed, 'diabetes is understood by few and ignored by many'.[1] With the current global epidemic of this condition it is important that clinicians should have background knowledge of diabetes and its implications for dental care so that barriers to treatment can be avoided.

WHAT IS DIABETES?

Diabetes is a condition where the body either fails to produce insulin (Type 1 diabetes) or the insulin that is produced is no longer as effective (Type 2 diabetes). A recent study in the United Kingdom reported that diabetes affects 4.3% of the UK population.[2] However, using recently introduced WHO criteria, there has been a drastic elevation in the prevalence of diabetes among the older British population (age range 60-79 years): approximately 8% of British men and 6% of British women are now known to have diabetes.[3] Of the remainder, approximately 6% of men and 5% of women are classified as having undiagnosed diabetes.

Insulin is a hormone produced in the beta cells of the Islets of Langerhans within the pancreas. Insulin is released directly into the bloodstream and is therefore part of the endocrine system. Insulin acts like a key which allows blood glucose to enter the cells around the body for use as an energy supply. Glucose is essential for the body to function properly. The brain is particularly affected by any reduction in blood glucose supply due to its lack of capacity for glucose storage.

There are two main types of diabetes:

Type 1 diabetes

Type 1 diabetes accounts for approximately 15% of all diabetics in the UK: it is usually juvenile onset, but can occur at any age. In Type 1 diabetes the beta cells in the pancreas undergo a chronic autoimmune destruction process which results in a long-term lack of endogenous insulin. Scientists and researchers remain unsure as to the exact cause of Type 1 diabetes but it is thought that a viral or other infection may trigger the autoimmune destruction.[4] The resultant lack of insulin must be replaced via injection, or via an insulin pump. This should be combined with knowledge of dietary carbohydrate (CHO) values such that injected insulin can be adjusted to carbohydrate consumed to avoid large fluctuations in blood glucose levels.

There are many other factors (apart from insulin and CHO) which can affect blood glucose levels. These are less easy to control or monitor and include: anti-insulin hormones, eg, adrenaline, growth hormone, cortisol and glycogen; exercise; and anxiety.

The above factors alter from day to day and even hour to hour thus making good blood glucose control a far from simple goal.

Type 2 diabetes

With Type 2 diabetes there are usually adequate levels (and sometimes even increased levels) of insulin but it is no longer as effective at the cellular level. Using the key analogy, it is as if the key is a bit rusty and it struggles to unlock the cell door to allow the blood glucose to enter. Blood sugar therefore becomes raised but as there is still some effective insulin the levels are not usually as high as with the Type 1 diabetes situation. Hence the undiagnosed Type 2 diabetic may misinterpret or even ignore their symptoms

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15.

Antidiabéticos orales y odontología

Oral antidiabetic agents and dentistry

González Montero C*, Manso Platero FJ**, López Alba AJ***

RESUMEN

La *diabetes mellitus* es un conjunto de trastornos metabólicos caracterizado por la existencia de hiperglucemia. Existen varios tipos de diabetes y por tanto distintos tratamientos farmacológicos. En esta revisión, se describen las interacciones de las insulinas y antidiabéticos orales en el tratamiento de la DM tipo 2, con los principales fármacos utilizados en odontología.

Palabras clave: Antidiabéticos orales, insulina, diabetes mellitus, odontología.

SUMMARY

Diabetes Mellitus is a metabolic disorder which is characterized by hyperglycemia. There are several types of diabetes, and therefore different pharmacological treatments. In this systematic review will focus on the interactions of insulins and oral antidiabetic agents in the treatment of type 2 Diabetes Mellitus, with the main drugs used in dentistry.

Key words: Oral antidiabetic agents, insulin, diabetes mellitus, dentistry.

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INTRODUCCIÓN

I. Concepto de diabetes

La Diabetes Mellitus (DM) comprende un grupo de trastornos metabólicos frecuentes que comparten el fenotipo de la hiperglucemia. Existen varios tipos diferentes de DM debido a una compleja interacción entre genética, factores ambientales y elecciones respecto a los estilos de vida. Dependiendo de la causa de la DM, los factores que contribuyen a la hiperglucemia pueden comprender diferentes combinaciones entre disminución de la secreción de insulina, disminución de la utilización de glucosa y/o un aumento de la producción endógena de glucosa. El trastorno de la regulación metabólica que acompaña a la DM provoca alteraciones fisiopatológicas secundarias en muchos sistemas orgánicos, y supone una pesada carga para el individuo que padece la enfermedad y para el sistema sanitario, por los cos-

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Nutrición Hospitalaria

Trabajo Original

Otros

Hyperlipidemia during gestational diabetes and its relation with maternal and offspring complications

Hiperlipidemia durante la diabetes gestacional, complicaciones maternas y para la descendencia

Aura D. Herrera-Martínez, Rafael Palomares Ortega, Rodrigo Bahamondes Opazo, Paloma Moreno-Moreno, M.ª José Molina Puerta and María A. Gálvez-Moreno

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Abstract

Introduction: lipid profile suffers adaptive changes during pregnancy due to estrogen stimulation and insulin resistance. Several relations have been suggested between maternal lipid profile, glucose tolerance, endothelial cell dysfunction and long-term cardiovascular risk; the effects of maternal lipid profile metabolism in fetal growth are also inconclusive. Since a regular evaluation and follow-up of lipid profile during pregnancy has not been established yet, we aimed to evaluate the incidence of dyslipidemia in patients with gestational diabetes (GDM) and analyze some putative relations with pregnancy, offspring complications and maternal metabolic syndrome parameters determined three and twelve months after delivery.

Patients and methods: two hundred and fifty patients with GDM were included. Full medical history, offspring characteristics, lipid profile and maternal variables of metabolic syndrome were evaluated during pregnancy and three- and twelve-months after delivery. The incidence of dyslipidemia during pregnancy was determined using two different classifications.

Results: lower plasma HDL and hypertriglyceridemia were the most current disorders; prematurity or birth weight were not correlated with dyslipidemia. During pregnancy, the lipid-related parameter that better predicted the risk of offspring macrosomia was triglycerides (TG). High TG three months after delivery were correlated to macrosomia and metabolic syndrome variables before and after pregnancy (three and twelve months).

Conclusions: TG during pregnancy is the parameter that best predicts the risk of macrosomia and is related to increased metabolic risk after delivery. The evaluation of lipid profile and other metabolic variables during pregnancy and after delivery is required to early diagnose cardiovascular risk factors, especially in high risk population.

Key words:

Lipid profile. Triglycerides. Gestational diabetes. Offspring complications.

Resumen

Introducción: los cambios del perfil lipídico durante el embarazo se relacionan probablemente con la estimulación estrogénica y la resistencia a la insulina. Diversas relaciones se han planteado entre el perfil lipídico gestacional, la intolerancia a la glucosa, la disfunción endotelial y el riesgo cardiovascular a largo plazo; sus efectos sobre el crecimiento fetal no son concluyentes. Dado que no existe un protocolo de diagnóstico y seguimiento de la dislipemia durante el embarazo, el objetivo del presente estudio fue evaluar la incidencia de dislipemia en pacientes con diabetes gestacional (DMG) y analizar su relación con complicaciones maternas, fetales y variables de síndrome metabólico 3 y 12 meses tras el parto.

Pacientes y métodos: fueron incluidos 250 pacientes con DMG. Se analizaron variables clínicas maternas y del recién nacido y se determinó el perfil lipídico durante el embarazo, 3 y 12 meses tras el parto. La incidencia de dislipemia se realizó utilizando dos clasificaciones diferentes.

Resultados: las alteraciones más frecuentes fueron bajos niveles de HDL y altos de triglicéridos (TG). La prematuridad o el peso al nacer no se relacionaron con la presencia de dislipemia. El parámetro lipídico que mejor predijo el riesgo de macrosomía fueron los TG. Altos niveles de TG tres meses después del parto se relacionaron con macrosomía y variables de síndrome metabólico pregestacional, así como 3 y 12 meses después del parto.

Conclusiones: los niveles de TG durante el embarazo representan el parámetro que mejor predice el riesgo de macrosomía y se relacionan con un mayor riesgo metabólico después del parto. La evaluación del perfil lipídico durante el embarazo y después del parto permite un diagnóstico precoz de factores de riesgo cardiovascular, especialmente en poblaciones de alto riesgo.

Palabras clave:

Perfil lipídico. Triglicéridos. Diabetes gestacional. Complicaciones fetales.

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17.

Artículo original
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Desenlace materno-fetal en pacientes con diagnóstico temprano o tardío de diabetes gestacional

Maternal and fetal outcome in early and late diagnosis of gestational diabetes.

Daniela Villota-Burbano,[1] Manuel Casillas-Barrera,[2] Martha Patricia Morales-Morales,[3] Madaí Farías-Barajas,[3] Carlos Mayagoitia-Miguel[1]

Resumen

OBJETIVO: Comparar el desenlace materno y fetal en pacientes con diabetes gestacional diagnosticada en la primera y segunda mitad del embarazo.

MATERIALES Y MÉTODOS: Estudio observacional, retrospectivo y analítico que incluyó a todas las pacientes con diabetes gestacional tratadas en el Hospital de la Mujer de la Ciudad de México entre los meses de marzo de 2014 y diciembre de 2017. Las pacientes se dividieron en, grupo 1: diagnosticadas entre las 13 y 20.6 semanas de embarazo y grupo 2: con diagnóstico posterior. Se identificaron y compararon las principales complicaciones maternas y fetales. El análisis incluyó medidas de tendencia central, t de Student y χ^2.

RESULTADOS: Se incluyeron 459 pacientes; 147 embarazadas en el grupo 1 y 312 en el grupo 2. La enfermedad hipertensiva del embarazo (32.7 *vs* 17.6% p 0.001) y el hiper e hipotiroidismo (7.5 *vs* 1.6% p 0.001) fueron más frecuentes en el grupo 1. El peso al nacimiento (3054 ± 718.4 *vs*. 3156 ± 555.7 g. p 0.04) la talla (48.9 ± 3.49 *vs* 49.2 ± 2.7 cm p 0.05) y el Capurro (37.9 ± 2.5 *vs* 38.2 ± 1.4 semanas p 0.01) fueron mayores en los neonatos hijos de madres del grupo 2.

CONCLUSIÓN: El desenlace materno y fetal de pacientes con diabetes gestacional se modifica de acuerdo con las semanas de embarazo al momento del diagnóstico; por esto debe insistirse en la detección oportuna de las complicaciones descritas con el propósito de reducir las repercusiones de la diabetes gestacional en la madre y su hijo.

PALABRAS CLAVE: Diabetes gestacional; embarazo; enfermedad hipertensiva; hipertiroidismo; hipotiroidismo; peso al nacimiento.

Abstract

OBJECTIVE: To compare the fetal maternal outcomes in patients with gestational diabetes detected in the first and second half of pregnancy.

MATERIALS AND METHODS: An observational, retrospective and analytical study was carried out including all patients with gestational diabetes treated at the Women's Hospital of Mexico City in the years 2014 to 2017. Were divided into two groups, group 1 inserted those patients diagnosed between 13 and 20.6 weeks of gestation and 2 with subsequent diagnosis, the main maternal and fetal complications were identified and compared. The results analysis included central trend measures, Student's t and square Chi.

RESULTS: 495 patients were included; Group 1 was composed of 147 pregnant women and group 2 of 312. Maternal complications such as hypertensive disease of pregnancy (32.7 vs 17.6% p 0.001), hyperthyroidism and hypothyroidism (7.5 vs 1.6% p 0.001) were more frequent at the group 1. The birth weight (3054 ± 718.4 vs. 3156 ± 555.7 g. p 0.04), height (48.9 ± 3.49 vs 49.2 ± 2.7 cm p 0.05) and Capurro rating (37.9 ± 2.5 vs 38.2 ± 1.4 weeks p 0.01) were greater in infants in Group 2.

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ARÁN

Nutrición Hospitalaria

Trabajo Original

Pediatría

Implication of gestational diabetes treatment on maternal weight gain and low neonatal weight: a large retrospective cohort study

Implicación del tratamiento de la diabetes gestacional en el aumento de peso materno y bajo peso neonatal: gran estudio de cohorte retrospectivo

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Abstract

Objective: the treatment for gestational diabetes is based on diet, and this may modify maternal weight gain. The limited maternal weight gain is related to newborns with small weight for their gestational age (SGA), and many studies have found an increase of SGA in women with gestational diabetes (GD), but the reason for this is not clear. The objective of this study is to evaluate the effects of gestational diabetes treatment on maternal weight gain and neonatal weight.

Methods: a retrospective cohort study of 1,765 patients with GD, according to the National Diabetes Data Group (NDDG) criteria. We assessed: pre-pregnancy BMI, total maternal weight gain (MWG), weight gain during the third trimester, gestational week of starting the treatment, and treatment modality (diet or diet plus insulin). Birth weight was adjusted by gestational age and gender: SGA (≤ 10th) and large for gestational age (LGA) (> 90th).

Results: the percentage of newborns with weight percentile ≤ 10 was 14.8%. The diet and the time of initiation of the treatment were related to maternal weight gain (MWG) in the third trimester. For every 1 kcal/kg of variation in the diet (increase or decrease), a MWG variation of 0.03 (0.001-0.06) kg occurred ($p < 0.01$). For each week before the beginning of treatment, the mother did not gain 0.13 ± [(-0.15) - (-0.11)] kg in the third trimester ($p < 0.01$). The SGA was related to the lowest MWG in total gestation: 7.0 (IQR 3.0-10.4) kg *vs.* 8.4 (IQR 5.0-11.6) kg ($p < 0.01$), and in the third trimester: 0.3 (IQR -0.9-1.5) kg *vs.* 0.9 (IQR -0.3-2.2) kg ($p < 0.01$).

Conclusion: the dietary treatment for gestational diabetes leads to a lower maternal weight gain and induces an impact on neonatal weight.

Key words:

Gestational diabetes. Birth weight. Pregnancy. Weight gain. Gestational age.

Resumen

Objetivo: el tratamiento para la diabetes gestacional se basa en la dieta y esto puede modificar el aumento de peso materno. Un aumento de peso materno limitado está relacionado con recién nacidos con bajo peso para su edad gestacional (SGA). Muchos estudios han encontrado un aumento de niños con bajo peso en mujeres con diabetes gestacional, pero la razón de esto no está clara. El objetivo de este estudio es evaluar los efectos del tratamiento de la diabetes gestacional sobre el aumento de peso materno y el peso neonatal.

Métodos: estudio de cohortes retrospectivo en 1765 pacientes con diabetes gestacional, según los criterios de los National Diabetes Data Groups (NDDG). Evaluamos: IMC antes del embarazo, aumento de peso materno total (MWG), aumento de peso durante el tercer trimestre, semana gestacional de inicio del tratamiento y modalidad de tratamiento (dieta o dieta más insulina). El peso al nacer se ajustó por edad gestacional y género: SGA (percentil de ≤ 10) y grande para la edad gestacional (LGA) (percentil de > 90).

Resultados: el porcentaje de recién nacidos con peso percentil de ≤ 10 fue del 14,8%. La dieta y el momento de inicio del tratamiento se relacionaron con el aumento de peso materno en el tercer trimestre. Por cada 1 kcal/kg de variación en la dieta (aumento o disminución) se produjo una variación de aumento del peso materno de 0,03 (0,001-0.06) kg ($p < 0,01$). Por cada semana antes de inicio del tratamiento, la madre dejó de ganar 0,13 ± [(- 0,15) - (- 0,11)] kg en el tercer trimestre ($p < 0,01$). El SGA se relacionó con un aumento de peso materno más bajo en el total de la gestación: 7,0 (IQR 3,0-10,4) kg *vs.* 8,4 (IQR 5,0-11,6) kg ($p < 0,01$), y en el tercer trimestre: 0,3 (IQR -0,9-1,5) kg *vs.* 0,9 (IQR -0,3-2,2) kg ($p < 0,01$).

Conclusión: el tratamiento dietético para la diabetes gestacional puede conducir a un menor aumento de peso materno y a su vez inducir un impacto en el peso neonatal.

Palabras clave:

Diabetes gestacional. Peso neonatal. Gestación. Ganancia de peso. Edad gestacional.

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19.

Farmacia Comunitaria

Curso básico sobre diabetes

Tema 4
Antidiabéticos orales

La principal forma de manejar la diabetes mellitus tipo 2 es a través del control del peso corporal, con una dieta saludable y la realización de actividad física. Cuando estas medidas no funcionan, es necesario recurrir a los antidiabéticos orales para conseguir un adecuado control glucémico.

ESTHER GANADO, ITXASO GARAY, LORENA VEGA
Farmacéuticas comunitarias en Bizkaia
Miembros del grupo Serantes del COFBi

Introducción

El tratamiento inicial para la diabetes mellitus (DM) tipo 2 es controlar el peso corporal, evitando el sobrepeso, mediante una alimentación saludable y la realización de ejercicio físico de forma moderada. Con frecuencia, estas medidas son insuficientes para conseguir un buen control glucémico y es necesario utilizar un tratamiento farmacológico con los conocidos antidiabéticos orales, que se pueden administrar tanto en monoterapia como en combinación con otros antidiabéticos, o con insulina para lograr el máximo beneficio terapéutico.

Según el mecanismo de acción, los antidiabéticos orales se clasifican, según su acción, en:

✓ Incrementan la sensibilidad a la insulina endógena:

– Biguanidas: actúan principalmente a nivel hepático, reduciendo la glucosa producida por el hígado. También presentan una acción a nivel muscular, aumentando la utilización y la captación de glucosa,

20.

Nutrición Hospitalaria

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Caso clínico

Acidosis láctica grave asociada a intoxicación por metformina

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Resumen

La metformina es una biguanida ampliamente utilizada en el tratamiento de la diabetes mellitus tipo II. Entre los efectos secundarios derivados de su empleo destaca por su baja frecuencia de presentación pero potencial gravedad la acidosis láctica. El diagnóstico de la misma se basa generalmente en la coexistencia de la acidosis láctica en un paciente en tratamiento con metformina con uno o mas factores de riesgo para la presentación de la misma. El desarrollo de acidosis láctica en relación con el tratamiento con metformina conlleva una mortalidad que oscila entre 50-80%.

(*Nutr Hosp.* 2007;22:124-125)

Palabras clave: *Metformina. Acidosis. Intoxicación.*

SEVERE LACTIC ACIDOSIS ASSOCIATED TO METFORMIN INTOXICATION

Abstract

Metformin is a biguanide extensively used in the treatment of type II diabetes mellitus. Between the nocive effects of the metformin emphasizes tha lactic acidosis because of its low frecuency but potential severity. The diagnosis of the poisoning due to metformin is based on the coexistence of lactic acidosis and one or more of the risk factors. The development of lactic acidosis in metformin poisoning is associated to a range of 50-80% of mortality.

(*Nutr Hosp.* 2007;22:124-125)

Key words: *Metformin. Acidosis. Poisoning.*

Introducción

Presentamos el caso de un paciente en tratamiento con metformina que presentó un cuadro de acidosis láctica grave que requirió de su ingreso en la Unidad de Cuidados Intensivos (UCI) de nuestro centro. El interés de este caso radica en que el diagnóstico de la intoxicación se realizó mediante determinación directa de los niveles de metformina en sangre, procedimiento raramente descrito en la bibliografía.

Caso clínico

Paciente varón de 72 años de edad entre cuyos antecedentes personales destacó haber sido diagnosticado de enfermedad pulmonar obstructiva crónica (EPOC), hipertensión arterial, diabetes mellitus tipo II e isquemia crónica en las extremidades inferiores. Se encontraba en tratamiento con metformina, acarbosa y glinclazida. Presentó un cuadro de 72 horas de evolución cuyo inicio se caracterizó por la existencia de un dolor abdominal difuso acompañado de diarrea y anorexia, con posterior deterioro del nivel de conciencia. Fue atendido en un hospital secundario en el que se objetivó la existencia de datos de fracaso renal agudo, importante acidosis metabólica e inestabilidad hemodinámica motivo por el cual se decidió su traslado a la UCI de nuestro hospital.

A su llegada a nuestra Unidad el paciente se encontraba consciente aunque con tendencia al sueño, taquicárdico (frecuencia cardíaca de 120 latidos por minuto en ritmo sinusal), hipotenso (tensión arterial 80/40 mmHg) y con signos indirectos de bajo gasto cardíaco en la exploración física. Entre las determinaciones analíticas realizadas a su ingreso destacó la existencia de una insuficiencia renal aguda con cifras de creatinina y urea de 6,6 mg/dl y 197 mg/dl respectivamente y de una acidosis láctica grave con pH: 6,8, déficit de bases -29 mmol/l, anión GAP de 41 y determinación de lactato de 176 mg/dl.

Ante la sospecha de que se tratase de una intoxicación aguda por metformina se tomó una muestra sanguínea para determinación de los niveles plasmáticos

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21.

Tratamiento de la diabetes mellitus (III). Insulinoterapia

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Palabras Clave:

- Diabetes
- Secreción fisiológica de insulina
- Preparados de insulina
- Pautas de insulina

Resumen

Introducción. Tras el descubrimiento de la insulina se han ido desarrollando nuevos preparados de insulina que, junto a las mejoras tecnológicas en la monitorización de la glucosa y el conocimiento de la secreción fisiológica de insulina, han permitido el diseño de pautas de insulinización ajustadas a las necesidades de los pacientes.

Preparados de insulina. Las insulinas de acción rápida o prandiales se administran antes de las comidas para evitar la hiperglucemia posprandial, las insulinas de acción intermedia combinan un componente basal y prandial, y los análogos de insulina de acción prolongada pretenden mimetizar la insulinemia basal endógena.

DM1. Hay una deficiencia absoluta de insulina, por lo que el objetivo del tratamiento es una administración de insulina lo más fisiológica posible, mediante pautas bolo-basal, combinando análogos de insulina lenta e insulina prandial, o mediante infusión subcutánea continua de insulina.

DM2. Caracterizada por una resistencia a la insulina, en su historia natural se produce una pérdida progresiva de la función beta pancreática, por lo que la mayoría de los pacientes acaban necesitando tratamiento con insulina. Esta se va introduciendo de manera secuencial y combinada con otros fármacos y medidas terapéuticas.

Keywords:

- Diabetes
- Physiological insulin secretion
- Insulin preparations
- Insulin regimens

Abstract

Treatment of diabetes mellitus (III). Insulin therapy

Introduction. After the discovery of insulin, new insulin preparations have been developed that, together with technological improvements in glucose monitoring and in the knowledge of the physiological secretion of insulin, have allowed to design insulinisation guidelines according to the patients' needs.

Insulin preparations. Rapid-acting insulin or prandials are administered before meals to avoid postprandial hyperglycaemia, intermediate-acting insulins combine basal and prandial components, and the analogous long-acting insulin preparations intend to mimick endogenous basal insulinemia.

DM1. There is an absolute insulin deficiency, therefore the treatment goal is to mimic, as closely as possible, physiological insulin secretion, by means of basal-bolus regimens, combining analogous long-acting and prandial insulin, or by means of continuous subcutaneous insulin infusion.

DM2. It is characterised by insulin resistance; its natural progression produces a gradual loss of the pancreatic beta-cell function, thus most patients end up requiring insulin treatment. It is introduced in a sequential manner and combined with other drugs and therapeutic measures.

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Journal of the Irish Dental Association

PEER-REVIEWED

Diabetes mellitus and its relevance to the practice of dentistry

Diabetes mellitus is a syndrome of abnormal carbohydrate, fat and protein metabolism that results in acute and chronic complications due to the absolute or relative lack of insulin. Globally, it is expected that the number of people with diabetes will increase, and as a result dental practitioners will encounter an increasing number of patients affected by this chronic condition, which may have implications for the provision of safe and appropriate dental treatment. This article aims to provide an overview of diabetes and to discuss aspects of the condition relevant to dentistry. The article also discusses the management of diabetic emergencies in a dental practice setting.

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Introduction

Diabetes mellitus is a syndrome of abnormal carbohydrate, fat and protein metabolism that results in acute and chronic complications, and is due to the absolute or relative lack of insulin. The disease is a significant global public health problem and is a major source of morbidity and mortality in the world today.

Globally, it is expected that the number of people with diabetes will increase from the current estimate of 150 million to 220 million by the year 2010 and to 300 million by the year 2025.[1] Consequently, dental practitioners will encounter an increasing number of patients with diabetes presenting for dental treatment in years to come.

This article aims to provide an overview of diabetes and to discuss aspects of the condition relevant to dentistry.

Prevalence

In 2005 the estimated population prevalence of type 1 and type 2 diabetes in adults was 4.7% in the Republic of Ireland. This equated to just over 141,000 adults. Taking into account population change and assuming the most realistic scenario that obesity rates will continue to rise in a linear fashion, the population prevalence of diabetes in adults in 2015 will be in the order of 5.6% (193,944 adults) in the Republic of Ireland.[2]

Classification of diabetes mellitus

There are three main categories of diabetes:

1. Type 1 (insulin-dependent diabetes mellitus), which results from an absolute insulin deficiency.
2. Type 2 (non-insulin-dependent diabetes mellitus), which is the result of insulin resistance and an insulin secretory defect.
3. Gestational diabetes presenting for the first time during pregnancy, which occurs in 2-5% of all pregnancies.

Impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) (**Table 1**) are intermediate conditions in the transition between normality and diabetes. Patients with these conditions are now referred to as having 'pre-diabetes' and are at high risk of progressing to type 2 diabetes mellitus, although this is not inevitable. IGT and IFG are associated with metabolic syndrome, which includes obesity (especially abdominal or visceral obesity), dyslipidaemia of the high triglyceride and/or low high-density lipoprotein (HDL) type, and hypertension.

Diagnosis

The criteria for diagnosis of diabetes include symptoms of hyperglycaemia (polyuria, polydipsia, unexplained weight loss, visual blurring, genital thrush, lethargy) associated with a raised random venous plasma glucose (≥11.1mmol/l) level, or a raised fasting plasma glucose level (≥7.0mmol/l) in the presence or absence of symptoms, with the test being performed on two separate occasions.

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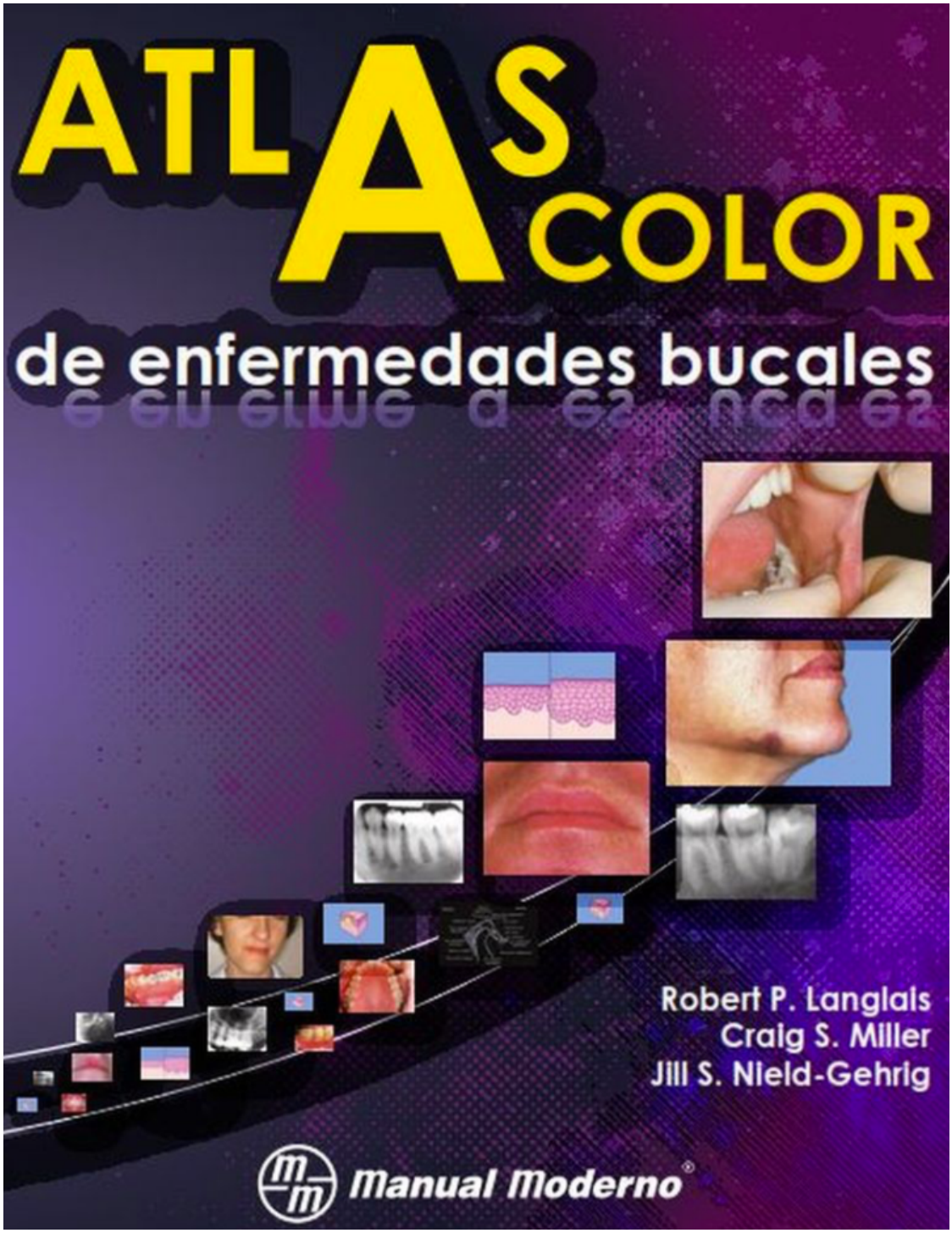
ATLAS COLOR
de enfermedades bucales
Robert P. Langlais
Craig S. Miller
Jill S. Nield-Gehrig
Manual Moderno

Tratamiento de la boca seca: puesta al día

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Silvestre-Donat FJ, Miralles-Jordá L, Martínez-Mihi V. Tratamiento de la boca seca: puesta al día. Med Oral 2004;9:273-9.

RESUMEN

La boca seca es una situación muy común en la clínica odontológica y las causas que producen esta alteración de la secreción de saliva pueden ser múltiples. Es especialmente frecuente en personas de edad avanzada y en pacientes que están tomando gran cantidad de fármacos. El tratamiento de esta situación deberá estar relacionado con la eliminación de la causa que la produce y cuando esto no es posible se basará en el estimulo de la secreción salival con determinados fármacos o en la sustitución de la misma con las llamadas salivas artificiales. En este articulo queremos sistematizar de forma sucesiva las actuaciones terapéuticas a seguir en este tipo de pacientes.

Palabras clave: *Boca seca, xerostomia, tratamiento.*

INTRODUCCION

Aunque muchos pacientes que acuden a nuestras consultas se quejan de boca seca, cuando exploramos la cavidad oral, en muchas ocasiones, no encontramos signos objetivos que justifiquen dicha situación. Mucho se ha escrito sobre el problema de los pacientes que padecen de sequedad de boca; sin embargo, los progresos en el tratamiento de esta situación no han sido demasiado importantes en los últimos años. Estos pacientes deberían ser bien estudiados por la relación existente entre la hiposialia y ciertos procesos sistémicos.

La sensación subjetiva de boca seca la conocemos como xerostomía pero cuando constatamos objetivamente mediante la sialometría una disminución en las tasas de flujo salival por debajo de 0,1-0,2 ml/min la saliva total de reposo y por debajo de 0,4-0,7 ml/min la saliva total estimulada hablamos de hiposialia o hiposecreción salival. Estas cantidades serían el equivalente a segregar de menos de 500 cc por día (1,2).

La persistencia en el tiempo de tasas bajas de secreción salival conllevan cambios en el medio bucal y condicionan la aparición de lesiones sobre los tejidos duros y blandos de la boca (3).

La hiposialia puede aparecer por múltiples causas que de alguna manera inhibirán el reflejo salival desde los receptores nerviosos periféricos pasando por las estructuras del sistema nervioso central, las vías vegetativas que inervan las glándulas salivales y llegando a afectar a las propias glándulas. Dentro de las causas más frecuentes que producen hiposialia están los fármacos llamados xerostomizantes, que bien a nivel central o periférico van a afectar las vías que inervan las glándulas (4). Entre los fármacos xerostomizantes se encuentran medicamentos tan frecuentes como analgesicos, antihipertensivos, anticolinergicos, antidepresivos, ansiolíticos, antipsicóticos, antihistamínicos y los derivados de los alcaloides opiáceos. También, ciertas situaciones psicopatológicas como el estrés o la ansiedad pueden producir xerostomía por su acción a nivel central (5).

Asimismo, el insuficiente aporte crónico de proteínas y la falta de aporte hídrico o su excesiva pérdida como en pacientes muy deshidratados pueden producir hiposiália, o en dietas muy restrictivas de sal, en pacientes que toman diuréticos de forma continuada y en diabéticos (6).

Aunque en los pacientes con boca seca las situaciones más importantes clínicamente se suelen ver cuando existe una destrucción aguda o crónica del parenquima salival. De forma especial existe dos modelos clínicos donde el grado de hiposecreción salival puede llegar a niveles más graves como en el síndrome de Sjögren de larga evolución y los pacientes a los que se les ha aplicado radioterapia por cáncer de cabeza o cuello a grandes dosis. Situaciones donde se produce hiposialia con menor severidad son los casos de inflamaciones o infecciones crónicas de las glándulas (4).

La boca seca junto a la sequedad en otras mucosas se puede presentar con gran frecuencia en la población general pero es en el estrato de mayores de 60 años donde aparecerá con mayor incidencia, especialmente debido a una mayor frecuencia de enfermedades sistémicas y a un mayor consumo de medicamentos (7).

Las primeras manifestaciones clínicas que aparecen en los pa-

Medicina Oral 2003;8:97-109

Problemas bucodentales en pacientes con diabetes mellitus (I): Indice de placa y caries dental.

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Arrieta-Blanco JJ, Bartolomé-Villar B, Jiménez -Martinez E, Saavedra-Vallejo P, Arrieta-Blanco FJ . Problemas bucodentales en pacientes con diabetes mellitus (I): Indice de placa y caries dental. Med Oral 2003;8:97-109.

RESUMEN

La diabetes mellitus es considerada, hoy día, como una de las enfermedades crónicas más frecuentes; por ello, es importante conocer cuales son sus alteraciones más relevantes a nivel bucal.

Objetivos: Estudiar los distintos signos y síntomas que presentan los pacientes diabéticos en la cavidad oral. Valorar el estado de higiene oral y la prevalencia de caries dental en una población diabética con respecto a una población control.

Diseño del estudio: Hemos realizado el trabajo sobre 70 pacientes diabéticos (30 varones y 40 mujeres) con edades comprendidas entre 11 y 81 años, y una población control de 74 pacientes no diabéticos (29 varones y 45 mujeres) con edades comprendidas entre 11 y 75 años. Dentro de la población diabética se valoró el tipo de diabetes, el grado de control de su enfermedad mediante la hemoglobina glicosilada, el tiempo de evolución de la diabetes y la existencia o no de complicaciones tardías. La higiene oral se midió mediante el índice placa de O´Leary. La prevalencia de caries se estudió mediante el índice CAOD.

Resultados: El estado de higiene oral fue significativamente peor en los pacientes diabéticos respecto a los controles a partir de los 56 años de edad. No hemos encontrado diferencias significativas en la prevalencia de caries ni en el índice CAOD, si bien, éste fue ligeramente más elevado en los pacientes diabéticos. El estudio de los pacientes diabéticos evidenció que sólo el tipo y la evolución de su enfermedad fueron parámetros significativos en relación al número de caries, mientras que para el índice de placa no se halló significación para ninguno de los parámetros analizados.

Conclusiones: En el presente estudio hemos observado un mayor número de ausencias dentarias en la población diabética con respecto a una población sana. No hemos encontrado diferencias en el número de caries, ausencias y obturaciones en función del control metabólico, tiempo de evolución y existencia o no de complicaciones tardías de la diabetes.

***Palabras Clave**: Diabetes mellitus, caries dental, índice de placa.*

INTRODUCCION

El estudio de las alteraciones del metabolismo, productoras de diversos síndromes y enfermedades (tal es el caso de la diabetes mellitus), ha despertado en

Glosodinia

Abordaje desde la farmacia

La boca ardiente, clásicamente definida como glosodinia, es un síntoma que refieren muchos pacientes, sobre todo mujeres. En un porcentaje importante de casos se descubren factores causales, pero en otros muchos no se detecta ninguna causa. En el presente artículo se abordan las principales características de este trastorno, haciendo especial hincapié en los consejos que el farmacéutico puede dar, tanto para su prevención como para su tratamiento.

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El síndrome de la boca ardiente consiste en un conjunto de síntomas intraorales que comprenden quemazón y/o dolor o adormecimiento, que afecta a personas con una mucosa oral clínicamente normal en las que se ha descartado previamente un problema médico o dental.

Si bien la lengua es la localización más usual, también pueden verse afectados los labios y el paladar, y en algunos casos, la garganta y el suelo de la boca.

Es habitual encontrar en la literatura médica diferentes términos para definir la sensación de boca ardiente; estos términos incluyen glosodinia, estomatopirosis, disestesia oral y estomatodinia. Actualmente la denominación de síndrome de la boca ardiente es la más aceptada, ya que hace referencia al síntoma principal que describen estos enfermos.

Epidemiología

La tasa de prevalencia de la glosodinia en la población general oscila entre el 2,6 y el 24% según los diferentes estudios, por lo que no se ha establecido una prevalencia fidedigna.

Este síndrome predomina en poblaciones con una media de más de 50 años y en la senectud. Todos los estudios muestran un predominio claro de casos en mujeres frente a varones: la proporción oscila entre 3:1 y 9:1. Hasta la fecha ha sido el grupo de mujeres menopáusicas, el que ha mostrado las tasas de prevalencia más elevadas. Por el momento, este síndrome no ha sido descrito en niños ni en adolescentes.

Etiopatogenia

Muchos investigadores han intentado explicar la causa del síndrome de la boca ardiente y de estos intentos científicos han brotado muchas teorías que han sido refutadas por otros, con lo que actualmente la duda persiste. Hoy día se admite que este síndrome tiene un origen multifactorial y que los factores pueden agruparse en locales, sistémicos y de origen psicógeno. Un hecho importante que cabe destacar es que, en muchas ocasiones, corregir o eliminar estos agentes presuntamente causales no determina la mejoría o desaparición de la sintomatología inicial. Seguidamente se analizan los factores que se acaban de comentar.

Factores locales

Se incluyen aquí distintos factores que tienen en común el hecho de que actúan irritando directamente la mucosa bucal, aunque su naturaleza puede ser diversa: física, química o biológica.

Los factores locales implicados son los siguientes:

Alteraciones de tipo mecánico. Se deben, básicamente, a prótesis mal ajustadas, especialmente si éstas son de tipo extraíble mucosoportadas; alteraciones de la dentición (malposiciones, piezas prominentes); ciertas disfunciones y hábitos anormales en los que se produce empuje lingual o tics que implican un frotamiento continuo sobre la prótesis o los dientes alterados.

Alteraciones de origen térmico o eléctrico. Entre las primeras cabe destacar el consumo de alimentos muy calientes y entre las segundas, el galvanismo bucal que produce pequeñas corrientes cuando en el interior de la cavidad bucal hay restauraciones de metales diferentes.

Alteraciones de tipo químico. Se deben a quemaduras en la mucosa por acción de ácidos o álcalis, bebidas alcohólicas o determinados fármacos de acción tópica.

Reacciones alérgicas locales. Por ejemplo, frente al polimetacrilato usado en la elaboración de dentaduras postizas y frente a ciertos aditivos alimentarios (ácido benzoico, canela, ácido sórbico).

Alteraciones de origen infeccioso. Candidiasis y fusoespiroquetas.

Biomédica 2012;32:52-9

ARTÍCULO ORIGINAL

Condición periodontal y pérdida dental en pacientes diabéticos del Hospital Universitario San Vicente de Paúl

Sandra Paola Ochoa, Carlos Andrés Ospina, Kelly Johana Colorado, Yenny Paola Montoya, Andrés Fernando Saldarriaga, Marisol Miranda Galvis, Natalia Muñoz Pino, María Eugenia Gómez, Fanny Lucía Yepes, Javier Enrique Botero

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Introducción. La diabetes es una enfermedad sistémica que afecta el metabolismo de la glucosa y se ha relacionado con el desarrollo de enfermedad periodontal.
Objetivo. El objetivo de este estudio fue determinar la condición periodontal y la pérdida dental de un grupo de pacientes diabéticos del Hospital Universitario San Vicente de Paúl.
Materiales y métodos. A 117 sujetos con diabetes de tipo 1 y 2 se les practicó un examen periodontal completo, determinación de valores de la hemoglobina A_1C (*glycosilated hemoglobin*) y radiografías periapicales, y se les indagó sobre hábitos de higiene oral e historia de diabetes. Los datos se analizaron de forma descriptiva y comparativa entre los parámetros clínicos, pérdida dental y tipo de diabetes.
Resultados. La prevalencia de gingivitis fue de 27,4 % y la de periodontitis de 72,6 %. La complicación sistémica más frecuente fue la hipertensión arterial (51,3 %). Los dientes más frecuentemente perdidos fueron los molares y, en promedio, los sujetos habían perdido siete dientes. El control de placa bacteriana fue pobre (55,4 %). No hubo diferencias en los parámetros clínicos entre pacientes diabéticos de tipo 1 y de tipo 2. El promedio de profundidad con sonda fue 2,62 mm, siendo los molares los dientes más afectados. El promedio de pérdida de inserción fue de 3,03 mm. Los dientes 17, 16, 27, 37 y 47 presentaron los valores más altos de pérdida de inserción.
Conclusiones. En conclusión, la condición periodontal en pacientes diabéticos fue mala, y presentaban periodontitis en la mayoría de los casos. Esto puede ser una causa importante de pérdida dental en sujetos diabéticos y requiere de especial atención por parte de los odontólogos y médicos.

Palabras clave: gingivitis, periodontitis, diabetes mellitus, glucemia, hemoglobina A_1C, pérdida de diente.

Periodontal condition and tooth loss in diabetic patients

Introduction. Diabetes is a systemic disease which affects the metabolism of glucose,and it has been associated with the development of periodontal disease.
Objective. The periodontal condition and tooth loss was evaluated in diabetic subjects.
Materials and methods. At the San Vicente de Paúl Hospital (Medellín, Colombia), 117 subjects with type 1 and 2 diabetes mellitus were examined. Patients underwent a comprehensive periodontal evaluation, glycosylated hemoglobin analysis, oral hygiene habits and history of diabetes. A descriptive and comparative analysis between the clinical parameters, tooth loss and type of diabetes was performed.
Results. The prevalence of gingivitis was 27.4% and periodontitis 72.6%. The most frequent systemic complication was hypertension (51.3%). The most frequently lost teeth were molars and in general, the subjects had lost an average of 7 teeth and had a poor plaque control (55.4%). No differences were seen in clinical parameters between type 1 and 2 diabetes patients. The mean probing depth was 2.6 mm. The first and second upper and lower molars showed the highest values of PD. The mean clinical attachment loss was 3.3 mm . Maxillary teeth 17, 16, and mandibular 37, 47 showed the highest values of clinical attachment loss.

Contribución de los autores:
El trabajo clínico y aporte de cada uno de los autores a la investigación y escritura del artículo fueron en igual proporción.
Sandra Paola Ochoa y Carlos Andrés Ospina se encargaron del examen clínico.
Kelly Johana Colorado, Yenny Paola Montoya y Andrés Fernando Saldarriaga se encargaron de la recolección de los datos clínicos y médicos.
Marisol Miranda Galvis, Natalia Muñoz Pino y María Eugenia Gómez se encargaron de la transcripción y de la base de datos.
Fanny Lucía Yepes y Javier Enrique Botero se encargaron de la coordinación del estudio y el análisis de los datos.

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PRACTICAL APPROACH

Practical Approaches to Diagnosing, Treating and Preventing Hypoglycemia in Diabetes

Kathryn Evans Kreider · Katherine Pereira · Blanca I. Padilla

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ABSTRACT

Hypoglycemia in individuals with diabetes can increase the risk of morbidity and all-cause mortality in this patient group, particularly in the context of cardiovascular impairment, and can significantly decrease the quality of life. Hypoglycemia can present one of the most difficult aspects of diabetes management from both a patient and healthcare provider perspective. Strategies used to reduce the risk of hypoglycemia include individualizing glucose targets, selecting the appropriate medication, modifying diet and lifestyle and applying diabetes technology. Using a patient-centered care approach, the provider should work in partnership with the patient and family to prevent hypoglycemia through evidence-based management of the disease and appropriate education.

Keywords: Cardiovascular Risk Reduction; Diabetes; Diabetes Technology; Hypoglycemia

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INTRODUCTION

Hypoglycemia is both a clinical and physiologic condition that is associated with increased morbidity and all-cause mortality in individuals with both type 1 (T1DM) and type 2 diabetes (T2DM) [1]. An increasing body of evidence suggests that hypoglycemia is harmful to patients with diabetes both immediately and over time, particularly in terms of cardiovascular health [2, 3]. While hyperglycemia can cause long-term complications, hypoglycemia can be imminently life threatening and significantly decrease the quality of life. Additionally, it is often difficult for patients to achieve the recommended glucose targets due to the fear of hypoglycemia or actual hypoglycemia. For these reasons, hypoglycemia can be one of the most difficult aspects of diabetes management from both the patient's and healthcare provider's perspective. However, using a patient-centered care approach and evidence-based practice, the provider can work in partnership with the patient to reduce the risk of hypoglycemia.

Compliance with Ethics Guidelines

This article is based on previously conducted studies and does not involve any new studies of human or animal subjects performed by any of the authors.

29.

Diabetic emergencies — ketoacidosis, hyperglycaemic hyperosmolar state and hypoglycaemia

Guillermo Umpierrez[1] and Mary Korytkowski[2]

Abstract | Diabetic ketoacidosis (DKA), hyperglycaemic hyperosmolar state (HHS) and hypoglycaemia are serious complications of diabetes mellitus that require prompt recognition, diagnosis and treatment. DKA and HHS are characterized by insulinopaenia and severe hyperglycaemia; clinically, these two conditions differ only by the degree of dehydration and the severity of metabolic acidosis. The overall mortality recorded among children and adults with DKA is <1%. Mortality among patients with HHS is ~10-fold higher than that associated with DKA. The prognosis and outcome of patients with DKA or HHS are determined by the severity of dehydration, the presence of comorbidities and age >60 years. The estimated annual cost of hospital treatment for patients experiencing hyperglycaemic crises in the USA exceeds US$2 billion. Hypoglycaemia is a frequent and serious adverse effect of antidiabetic therapy that is associated with both immediate and delayed adverse clinical outcomes, as well as increased economic costs. Inpatients who develop hypoglycaemia are likely to experience a long duration of hospital stay and increased mortality. This Review describes the clinical presentation, precipitating causes, diagnosis and acute management of these diabetic emergencies, including a discussion of practical strategies for their prevention.

Diabetic ketoacidosis (DKA), hyperglycaemic hyperosmolar state (HHS) and hypoglycaemia are frequent and serious complications arising among patients with type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM). In the USA, ~145,000 cases of DKA occur each year[1,2]. The rate of hospitalization for HHS is lower, accounting for <1% of all diabetes-related admissions[3,4]. The frequency of emergency room visits for hypoglycaemia is similar to that reported for severe hyperglycaemia[1,5]. Among hospitalized individuals, hypoglycaemia is a frequent complication of ongoing treatment for hyperglycaemia, with a reported incidence of 5–28% in intensive care unit (ICU) trials (depending on the intensity of glycaemic control)[6], and 1–33% in non-ICU trials using subcutaneous insulin therapy[7,8].

DKA, HHS and hypoglycaemia are associated with substantial morbidity and mortality, as well as high healthcare costs. DKA is the leading cause of mortality among children and young adults with T1DM, accounting for ~50% of all deaths in this population[9]. The overall DKA mortality recorded in the USA is <1%[1,2], but a higher rate is reported among patients aged >60 years and individuals with concomitant life-threatening illnesses[1,2,9,10]. Death occurs in 5–16% of patients with HHS[4,11], a rate that is ~10-fold higher than that reported for DKA[4,12]. Similarly, hypoglycaemia is associated with twofold to threefold increased mortality, particularly as age increases and among patients who have a history of severe hypoglycaemic episodes[13]. Several studies have reported that mortality in hyperglycaemic states is not caused by metabolic disarray but rather reflects the precipitating factor[14,15]. In the case of hypoglycaemia, in-hospital mortality is reported as being more frequent among patients with spontaneous hypoglycaemia than among those with insulin-induced or iatrogenic hypoglycaemia; however, these claims have been disputed[16–19]. Treatment of diabetic emergencies represents a substantial economic burden. For example, in the USA, the average cost of managing DKA is US$17,500 per patient, which represents a total annual hospital cost of $2.4 billion[1]. Similarly, hypoglycaemia is associated with immediate and delayed adverse clinical outcomes, as well as an increase in economic costs[20–22].

This Review describes the clinical presentation, precipitating causes, diagnosis and acute management of DKA, HHS and hypoglycaemia, including a discussion of practical approaches to prevent the onset of these diabetic emergencies.

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30.

Review Article

Medical emergencies in Dentistry: Practical tips in Implementation

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KEYWORDS

Medical emergencies; Dentistry; Dental clinic; Management

ABSTRACT

In a rapidly aging society, many patients will have multiple medical co-morbidities and on polypharmacy. Dental patients rarely have medical emergencies during their treatment and it is because of this rarity, that the odd emergency that presents can overwhelm the dentist's ability to cope despite their theoretical knowledge. The authors discuss how to adapt the clinic facilities for managing an emergency and provide an overview of management of common emergencies that dentists may encounter.

INTRODUCTION

It is not uncommon to see a dentist practising in a solo or small group dental office. Many of these offices are not within the vicinity of a hospital. In the event of an unexpected medical emergency, the dental practitioner will be the first line of care of the patient. These emergencies are not common, and it may be precisely why it can be problematic.

In a survey of Australian Dentists, it is estimated that 1 in 7 dentists will experience a patient collapse requiring resuscitation in their practice lifetime [1]. In the Great Britain, an emergency event was reported, on average, for every 4.5 practice years in England and Wales, and 3.6 years in Scotland [2]. Similarly, it was reported by New Zealand's dentists that only 2 medical emergencies occurred per 10,000 patients treated under local anaesthesia [3].

A review of present literature suggests that there is a wide range of medical emergencies which may occur in a dental clinic setting (Table 1). While broadly described as medical emergencies, some conditions require prompt pharmacologic intervention, resuscitation or activation of emergency services as compared to others.

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Vasovagal syncope is the most common medical emergency described in many cross-sectional studies. However, it is typically not life threatening and usually does not have negative sequalae. The management is mostly supportive in nature. In comparison, other emergencies such as acute myocardial infarction, anaphylaxis or foreign body ingestion require intervention as soon as possible to ensure the survival of the patient.

The low frequency of the emergency events in a dental office may result in dental schools putting less focus of it in their syllabus and also means that dental practitioners may not be well-prepared and current with management of emergencies. More than half the respondents in a New Zealand survey expressed dissatisfaction with the training they had received for medical emergencies as undergraduate students, and 14.1% continued to feel inadequately prepared for an emergency in practice [3]. While 75% of the respondents in the survey conducted in the Great Britain received relevant training as undergraduates, only 30% considered themselves 'well' or 'fairly well' prepared to manage emergencies. This is also reflected in surveys conducted in India, Nigeria, Saudi Arabia and Poland [10-13].

Theoretical knowledge derived from lectures, tutorials and exams may not adequately prepare the dental practitioner. Simulation training with hands on involvement may better facilitate memory

Diabetes in the Emergency Department: Acute Care of Diabetes Patients

Candace D. McNaughton, MD, Wesley H. Self, MD, and Corey Slovis, MD

Diabetes is a common condition, afflicting > 20% of the American population over the age of 60 years.[1] Patients with diabetes, particularly those with lower socioeconomic status or limited access to primary care, frequently seek care in hospital emergency departments.[2–6] This article will review the most common and immediately life-threatening diabetes-related complications seen in emergency departments: diabetic ketoacidosis (DKA), hyperglycemic hyperosmolar state (HHS), and hypoglycemia. It will also address the evaluation of patients with hyperglycemia and no previous diagnosis of diabetes.

Hyperglycemic Crisis: DKA and HHS

DKA is responsible for > 110,000 hospitalizations annually in the United States, with mortality ranging from 2 to 10%.[7–9] HHS is much less common but confers a much greater mortality.[10] In both diseases, mortality is largely related to underlying comorbidities such as sepsis.[11]

Clinical presentation

DKA and HHS are characterized by absolute or relative insulin deficiency, which prevents the body from metabolizing carbohydrates and results in severe hyperglycemia. As blood glucose levels rise, the renal glucose threshold is overwhelmed, and urine becomes more dilute, leading to polyuria, dehydration, and polydipsia.

Patients with DKA classically present with the triad of uncontrolled hyperglycemia, metabolic acidosis, and increased total body ketone concentration. On the other hand, HHS is defined by altered mental status caused by hyperosmolality, profound dehydration, and severe hyperglycemia without significant ketoacidosis (Table 1).[9,12]

Initial evaluation

Patients with severe hyperglycemia should immediately undergo assessment and stabilization of their airway and hemodynamic status. Naloxone, to reverse potential opiate overdose, should be considered for all patients with altered mentation. Thiamine, for acute treatment of Wernicke's encephalopathy, should be considered in all patients with signs of malnutrition. In cases requiring intubation, the paralytic succinylcholine should not be used if hyperkalemia is suspected; it may acutely further elevate potassium.

Immediate assessment also includes placing patients on a cardiac monitor and oxygen as well as obtaining vital signs, a fingerstick glucose, intravenous (IV) access, and an electrocardiogram to evaluate for arrhythmias and signs of hyper- and hypokalemia.

IN BRIEF

This article reviews the most common and immediately life-threatening diabetes-related conditions seen in hospital emergency departments: diabetic ketoacidosis, hyperglycemic hyperosmolar state, and hypoglycemia. It also addresses the evaluation of patients with hyperglycemia and no previous diagnosis of diabetes.

The differential diagnosis for hyperglycemic crisis includes the "Five I's": infection, infarction, infant (pregnancy), indiscretion (including cocaine ingestion), and insulin lack (nonadherence or inappropriate dosing). In addition to clinical history and physical examination, diagnostic tests should include a venous blood gas,[13–15] complete blood count, basic metabolic panel, and urinalysis; a urine pregnancy test must be sent for all women with childbearing potential.

Critically ill patients should undergo additional testing as clinically indicated, including a complete metabolic panel, serum osmolality, phosphate, lactate, and cardiac markers for older patients. A urine drug screen, blood alcohol level, and aspirin and acetaminophen levels should be sent for any patient with unexplained DKA and all patients with HHS. Evaluation for infection or injury should be guided by history and the physical examination. Effective serum osmolality should be calculated (Table 1). The corrected serum sodium is estimated by decreasing measured serum sodium by 1.6 mEq/L for every 100 mg/dl increase in blood glucose over a baseline of 100 mg/dl[16,17]; for every 100 mg/dl increment increase in

POSITION STATEMENT

Hyperglycemic Crises in Diabetes

AMERICAN DIABETES ASSOCIATION

Ketoacidosis and hyperosmolar hyperglycemia are the two most serious acute metabolic complications of diabetes, even if managed properly. These disorders can occur in both type 1 and type 2 diabetes. The mortality rate in patients with diabetic ketoacidosis (DKA) is <5% in experienced centers, whereas the mortality rate of patients with hyperosmolar hyperglycemic state (HHS) still remains high at ~15%. The prognosis of both conditions is substantially worsened at the extremes of age and in the presence of coma and hypotension (1–10).

This position statement will outline precipitating factors and recommendations for the diagnosis, treatment, and prevention of DKA and HHS. It is based on a previous technical review (11), which should be consulted for further information.

PATHOGENESIS — Although the pathogenesis of DKA is better understood than that of HHS, the basic underlying mechanism for both disorders is a reduction in the net effective action of circulating insulin coupled with a concomitant elevation of counterregulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. These hormonal alterations in DKA and HHS lead to increased hepatic and renal glucose production and impaired glucose utilization in peripheral tissues, which result in hyperglycemia and parallel changes in osmolality of the extracellular space (12,13). The combination of insulin deficiency and increased counterregulatory hormones in DKA also leads to the release of free fatty acids into the circulation from adipose tissue (lipolysis) and to unrestrained hepatic fatty acid oxidation to ketone bodies (β-hydroxybutyrate [β-OHB] and acetoacetate), with resulting ketonemia and metabolic acidosis. On the other hand, HHS may be caused by plasma insulin concentrations that are inadequate to facilitate glucose utilization by insulin-sensitive tissues but adequate (as determined by residual C-peptide) to prevent lipolysis and subsequent ketogenesis, although the evidence for this is weak (14). Both DKA and HHS are associated with glycosuria, leading to osmotic diuresis, with loss of water, sodium, potassium, and other electrolytes (3,15–20). The laboratory and clinical characteristics of DKA and HHS are summarized in Tables 1 and 2. As can be seen, DKA and HHS differ in magnitude of dehydration and degree of ketosis (and acidosis).

PRECIPITATING FACTORS — The most common precipitating factor in the development of DKA or HHS is infection. Other precipitating factors include cerebrovascular accident, alcohol abuse, pancreatitis, myocardial infarction, trauma, and drugs. In addition, new-onset type 1 diabetes or discontinuation of or inadequate insulin in established type 1 diabetes commonly leads to the development of DKA. Elderly individuals with new-onset diabetes (particularly residents of chronic care facilities) or individuals with known diabetes who become hyperglycemic and are unaware of it or are unable to take fluids when necessary are at risk for HHS (6).

Drugs that affect carbohydrate metabolism, such as corticosteroids, thiazides, and sympathomimetic agents (e.g., dobutamine and terbutaline), may precipitate the development of HHS or DKA. In young patients with type 1 diabetes, psychological problems complicated by eating disorders may be a contributing factor in 20% of recurrent ketoacidosis. Factors that may lead to insulin omission in younger patients include fear of weight gain with improved metabolic control, fear of hypoglycemia, rebellion from authority, and stress of chronic disease (13).

DIAGNOSIS

History and physical examination

The process of HHS usually evolves over several days to weeks, whereas the evolution of the acute DKA episode in type 1 diabetes or even in type 2 diabetes tends to be much shorter. Although the symptoms of poorly controlled diabetes may be present for several days, the metabolic alterations typical of ketoacidosis usually evolve within a short time frame (typically <24 h). Occasionally, the entire symptomatic presentation may evolve or develop more acutely, and the patient may present in DKA with no prior clues or symptoms. For both DKA and HHS, the classical clinical picture includes a history of polyuria, polydipsia, polyphagia, weight loss, vomiting, abdominal pain (only in DKA), dehydration, weakness, clouding of sensoria, and finally coma. Physical findings may include poor skin turgor, Kussmaul respirations (in DKA), tachycardia, hypotension, alteration in mental status, shock, and ultimately coma (more frequent in HHS). Up to 25% of DKA patients have emesis, which may be coffee-ground in appearance and guaiac positive. Endoscopy has related this finding to the presence of hemorrhagic gastritis. Mental status can vary from full alertness to profound lethargy or coma, with the latter more frequent in HHS. Although infection is a common precipitating factor for both DKA and HHS, patients can be normothermic or even hypothermic primarily because of peripheral vasodilation. Hypothermia, if present, is a poor prognostic sign (21). Caution needs to be taken with patients who complain of abdominal pain on presentation, because the symptoms could be either a result or an indication of a precipitating cause (particularly in younger

The recommendations in this paper are based on the evidence reviewed in the following publication: Management of hyperglycemic crises in patients with diabetes (Technical Review). *Diabetes Care* 24:131–153, 2001.

The initial draft of this position statement was prepared by Abbas E. Kitabchi, PhD, MD; Guillermo E. Umpierrez, MD; Mary Beth Murphy, RN, MS, CDE, MBA; Eugene J. Barrett, MD, PhD; Robert A. Kreisberg, MD; John I. Malone, MD; and Barry M. Wall, MD. The paper was peer-reviewed, modified, and approved by the Professional Practice Committee and the Executive Committee, October 2000. Revised 2001.

Abbreviations: β-OHB, β-hydroxybutyric acid; AKA, alcoholic ketoacidosis; DKA, diabetic ketoacidosis; HHS, hyperosmolar hyperglycemic state.

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ORIGINAL ARTICLE

Multicentre analysis of hyperglycaemic hyperosmolar state and diabetic ketoacidosis in type 1 and type 2 diabetes

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Abstract

Aims To compare diabetes patients with hyperglycaemic hyperosmolar state (HHS), diabetic ketoacidosis (DKA), and patients without decompensation (ND).

Methods In total, 500,973 patients with type 1 or type 2 diabetes of all ages registered in the diabetes patient follow-up (DPV) were included. Analysis was stratified by age (≤/> 20 years) and by manifestation/follow-up. Patients were categorized into three groups: HHS or DKA—during follow-up according to the most recent episode—or ND.

Results At onset of diabetes, HHS criteria were met by 345 (68.4% T1D) and DKA by 9824 (97.6% T1D) patients. DKA patients had a lower BMI(-SDS) in both diabetes types compared to ND. HbA1c was higher in HHS/DKA. During follow-up, HHS occurred in 1451 (42.2% T1D) and DKA in 8389 patients (76.7% T1D). In paediatric T1D, HHS/DKA was associated with younger age, depression, and dyslipidemia. Pump usage was less frequent in DKA patients. In adult T1D/T2D subjects, metabolic control was worse in patients with HHS/DKA. HHS and DKA were also associated with excessive alcohol intake, dementia, stroke, chronic kidney disease, and depression.

Conclusions HHS/DKA occurred mostly in T1D and younger patients. However, both also occurred in T2D, which is of great importance in the treatment of diabetes. Better education programmes are necessary to prevent decompensation and comorbidities.

Keywords Hyperglycaemic hyperosmolar state · Diabetic ketoacidosis · Acute complication · Metabolic decompensation · Multicentre registry

Introduction

Hyperglycaemic hyperosmolar state (HHS) and diabetic ketoacidosis (DKA) are life-threatening events for diabetes patients. According to the ISPAD guidelines [1], criteria for HHS include (1) plasma glucose concentration > 33.3 mmol/l, (2) arterial pH ≥ 7.3, (3) serum bicarbonate ≥ 15 mmol/l, (4) serum osmolality > 320 mOsm/kg, (5) decreased consciousness or seizures, (6) absent or mild ketonuria, (7) absent to mild ketonemia; criteria for DKA are (1) blood glucose concentration > 11 mmol/l, (2) pH < 7.3 and/or bicarbonate < 15 mmol/l, (3) ketonemia or moderate to large ketonuria.

HHS is found more frequently in type 2 diabetes (T2D) and occurs in 2% of adolescents at manifestation [2]. However, HHS can also be present in type 1 diabetes (T1D). A common symptom of T1D manifestation is polydipsia, which leads to an increased ingestion of (high sugar) beverages. The high sugar content increases blood glucose and serum osmolality, promoting an HHS, in spite of T1D pathophysiology [3]. Polydipsia may go unnoticed first, so that an HHS develops relatively slow. However, untreated HHS leads to death [1]. Mortality ranges from 5 to 20%, which is about 10 times higher compared with DKA, due to higher age or delayed diagnosis [4–6]. Previous studies reported higher occurrence of HHS in females and older patients (60 + years) and at diabetes onset [7–9]. The most common

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¿Qué entendemos por la Clasificación ASA-PS?

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What do we understand by ASA-PS Classification?

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Introducción

Una paciente de 70 años es referida al gastroenterólogo para una colonoscopia por hematoquecia ocasional. Es hipertensa, fumadora de toda la vida y obesa con índice masa corporal de 35. ¿Cómo afectan estos factores su clasificación ASA y cómo puede afectar esto el post-operatorio?

Esta pregunta muestra la complejidad de la evaluación del riesgo de esta paciente, que ha tratado de ser objetivado con diversos instrumentos clínicos.

El objetivo de la evaluación pre-operatoria es identificar riesgos potenciales del paciente enfrentado a un tipo de procedimiento particular, y de optimizar los resultados y la calidad de la atención médica. La información del estado del paciente nos permite además, optimizar alguna condición deficitaria, y/o programar una técnica anestésico-quirúrgica ajustada a la condición del paciente. Además, es una instancia que permite hacer un registro del paciente e informarle acerca de los riesgos y beneficios del proceso.

En 1940-41 la Asociación Americana de Anestesiología (ASA) forma un comité de tres médicos (M Saklad, E Rovenstine, I Taylor,) para estudiar, examinar, experimentar y diseñar un sistema para la colección y tabulación de datos estadísticos en anestesia, que pudiesen ser aplicables bajo cualquier circunstancia.

Proponen entonces una clasificación centrada en el estado físico de los pacientes, que pudiera asociarse a riesgos potenciales. Desde entonces la Clasificación ASA de estado físico (ASA-PS) es ***uno*** de los componentes del riesgo perioperatorio.

Comenta Saklad, ya en ese entonces, que la clasificación ASA-PS sirve, pero junto con:

- Tipo de cirugía planificada.
- La habilidad y experiencia del cirujano en el procedimiento contemplado.
- La calidad de los cuidados post-operatorios.
- La experiencia del anestesista en circunstancias similares.

En 1963, y luego en 1980, se hacen pequeños ajustes a la clasificación. En 1980, a la ASA-PS se le agrega la clase 6 para incorporar a los donantes con muerte cerebral antes de entrar a pabellón de cirugía.

Clasificación ASA

La Clasificación actualmente se comprime al formato siguiente[1,2] (Tabla 1).

Se agrega "E" a la clasificación si la cirugía es de emergencia.

Esta clasificación a pesar de intentar dar un carácter objetivo, no es siempre interpretada de igual forma por los anestesiólogos. En un estudio finlandés donde se compararon Hospitales Universitarios *versus* otros, se observó una importante variabilidad en la interpretación de la escala por los anestesiólogos.[4] En la Figura 1, se observa que 10 casos clínicos diferentes se interpretaron con un valor ASA-PS, muy diferente. En el caso 2 y 4 se llegan a dar 4 valores diferentes.

Tabla 1. American Society of Anesthesiologists Physical Status Classification[3]

ASA1	Paciente sano, sin enfermedad orgánica, bioquímica o psiquiátrica
ASA 2	Paciente con enfermedad sistémica moderada, por ej. asma moderada o hipertensión arterial bien controlada. Sin impacto en la actividad diaria. Poca probabilidad de impacto por cirugía o anestesia
ASA 3	Enfermedad sistémica significativa o grave que limita la actividad diaria normal, por ej. falla renal o diálisis o insuficiencia cardíaca congestiva clase 2. Probable impacto con anestesia y cirugía
ASA 4	Enfermedad grave que requiere apoyo constante o terapia intensiva, por ej., infarto agudo al miocardio, falla respiratoria que requiere ventilación mecánica. Seria limitación de la actividad diaria. Impacto mayor por anestesia y cirugía
ASA 5	Paciente moribundo, con riesgo de muerte en las siguientes 24 h, aun sin cirugía
ASA 6	Muerte cerebral donante de órgano