بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــسـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

GLYCEMIC CONTROL IN NON-INSULIN DEPENDENT DIABETIC PATIENTS UNDERGOING MAJOR SURGERIES

THESIS

Submitted In Partial Fulfillment For The Requirements Of The M. D. Degree In Anesthesiology

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بسم الله الرحمن الرحيم

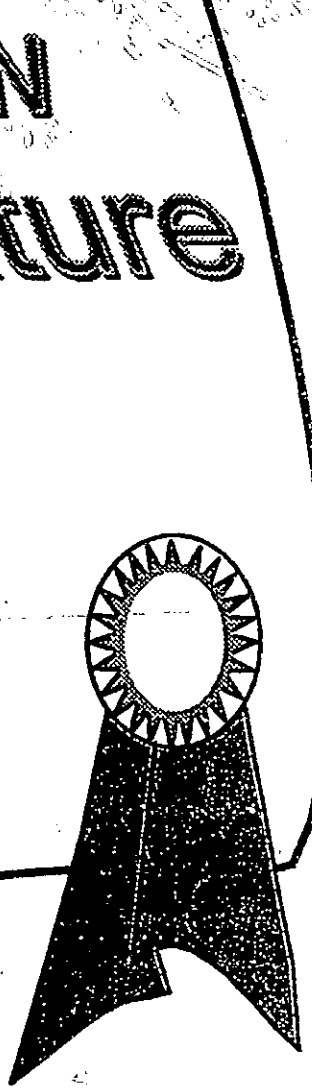
قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا

إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

"البقرة ٣٢"

Review Of Literature



Review of literature

Diabetes mellitus

1-Definition:

Different definitions have been made to DM and one of these definitions is somewhat satisfactory to understand the nature of this disease. So, diabetes mellitus is a syndrome characterized by chronic hyperglycemia and disturbances of carbohydrate, fat and protein metabolism associated with absolute or relative deficiencies in insulin secretion and/or insulin action. When fully expressed, diabetes is characterized by fasting hyperglycemia, but the disease can also be recognized during less overt stages and before fasting hyperglycemia appears, that, most usually by the presence of glucose intolerance. Diabetes mellitus may be suspected or recognized clinically by the presence of characteristic symptoms, such as excessive thirst, polyuria, pruritus, otherwise unexplained weight loss or one or more of the many complications associated with or attributable to the disease [1]

2-Classification:

Classification of patients with diabetes mellitus mandates adequate knowledge and awareness of pathophysiology that accompanies the diabetic state. The most widely accepted classification of D.M. was devised initially by the national diabetes data group (NDDG) in U.S.A. and then become the basis for the world health organization classification of diabetes. This classification permits a clinically useful characterization of D.M. even if the specific cause or etiology is not known. [1,2,3] The W.H.O. classification contains three major clinical classes:

I -/Diabetes mellitus.

II- Impaired glucose tolerance.

III- Gestational D.M.

The clinical class of D.M. is divided into four subgroups:

1-Insulin dependent diabetes mellitus (IDDM) or type I.

2-Non - insulin dependent D.M. (NIDDM) or type II.

3-Malnutrition - related D.M.

4-Secondary D.M.

I- Clinical DM:

1- Insulin-dependant DM (IDDM) or type (I) DM:

This type was called Juvenile or ketosis prone diabetes and is defined by the presence of classical symptoms of diabetes such as thirst, polyuria, wasting and or ketoacid and the necessity for insulin treatment, not only to control the hyperglycemia and symptoms but also to prevent the spontaneous occurrence of ketoacidosis.[4] In this type of D.M. there are marked deficits in insulin secretion that lead to profound deficits in insulin action leading to variety of metabolic consequences. The most common cause of I.D.D.M. is the autoimmune destruction of pancreatic β-cells, evidences of this autoimmune process include the presence of islet cell antibodies (I.C.A.), insulin auto-antibodies (I.A.A.) and antibodies to glumatic acid decarboxylase (G.A.D.) or 64 kilodalton protein.[5]

Autoimmune B cell destruction is common with certain HLA types but these types vary by racial and ethnic group. There is marked geographical variation in the incidence of type I diabetes with Northern

European countries like Finland and Sweden showing very high frequencies and oriental countries such as Japan, China and Korea showing a low incidence. Inherited susceptibility to type I diabetes depends on several genes at different loci. The strongest linkage is with the HLA genes lying within the MHC (major history compatibility complex) region on the short arm of chromosome 6 (now called type I diabetes 1'locus. Over 95% of Caucasian type I diabetic subjects carry HLA-DR3 and/or DR4 (class II antigens) compared to 50% in non-diabetic controls.[6]

The most likely environment agents involved in the etiology of type I diabetes are viruses and dietary components. Viruses may target the islet B cell and destroy them directly or may trigger an autoimmune reaction. In addition to viruses, a possible environmental determinant of type I diabetes is food. For example, exposure in women to nitrosamines in smoked meat eaten at the time of conception increase the occurrence of type I diabetes in their children. [7]

2-Non insulin dependant diabetes millets or type II DM:

NIDDM may present with classical symptoms but often is asymptotic. Despite the presence of hyperglycemia the concentration of ketone bodies in blood and urine is low and insulin treatment not necessary to maintain life or prevent spontaneous ketosis in those patients. It is the commonest form of the disease, comprising 85% to 90% of the diabetic population.[8] NIDDM develops in elderly and is the consequence of a deficiency in insulin action due to abnormalities at the cell surface or within the cell. A deficiency in insulin secretion is less

severe than that characterizing IDDM or combination of these processes. Most forms of NIDDM have positive family history. The genetic penetrance is unrelated to HLA genes and is associated with obesity in most patients but may develop in the absence of obesity, the extent to which the presence of obesity can be used to differentiate causes of NIDDM is debatable, nevertheless, the National Diabetes Data Group (NDDG) and W.H.O. recommended subdividing NIDDM into non obese and obese categories. (9)

About 80% of type II diabetic patients are obese. The risk of developing diabetes increases progressively in both men and women with the degree of overweight at least partially due to the decrease in insulin sensitivity as weight increases. The greatest risk of diabetes is associated with central or truncal obesity where fat is deposited subcutaneously and intra-abdominal sites. This type of obesity is more typical of men and is therefore known as android. Visceral fat (accumulation in central obesity) is more metabolically active than peripheral fat and release large quantity of non-esteroidal fatty acid (N.E.F.A.). N.E.F.A. has several metabolic actions that can cause insulin resistance, such as stimulating gluconeogenesis in the liver and inhibiting glucose uptake in muscle. (10)

The two main pathophysiological defects in type II diabetes are impaired insulin secretion and insulin resistance. The main B-cell abnormalities include a markedly reduced first - phase insulin secretion in the response to glucose and in established diabetes an attenuated second phase. Insulin pulsatility is also abnormal in type II diabetes, thereby reducing tissue insulin sensitivity. The processing of proinsulin to insulin

is impaired leading to over-secretion of proinsulin and its split products. Impaired meal-related insulin secretion in type II diabetes leads to exaggerated and prolonged post-prandial hyperglycemia, which is an important contributor to the overall loss of glycemic control. The realization of the importance of post-prandial glucose regulation and the evidence in some trials that post-prandial, but not fasting hyperglycemia is a risk factor for cardiovascular disease in type II diabetes has encouraged the development of treatments specifically aimed at reduction of post-prandial blood glucose levels.[11]

Insulin resistance is often associated with clustering of clinical and biochemical features known as metabolic syndrome (X) or the insulin resistance syndrome. This consists of glucose intolerance, truncal (central) obesity, hypertension, accelerated atherosclerosis, low serum high-density lipoprotein (HDL), cholesterol and high triglycerides and very low-density lipoprotein (VLDL) concentrations. This lipid abnormality with relatively normal serum total and Low density Lipoprotein (LDL) cholesterol concentration are characteristic of type II diabetes and known as dyslipidemia.[12]

3- Malnutrition related DM:

This type of diabetes includes two types of NIDDM that is quite rare but well described in developing tropical countries. The two forms are fibrocalculous pancreatic diabetes and protein deficient diabetes. [13]

4- Secondary DM:

The forth-major subclass of D.M. designated in the WHO and NDDG classification is that of other types of diabetes associated with certain conditions or syndromes. These other types of diabetes and glucose intolerance are recognized primarily by the presence of clinical features not present in the more typical forms of NIDDM or IDDM. From the therapeutic view each of these types can be classified as either insulin dependent or non-insulin dependant. [1,14] These types include diabetes with:

(1) Pancreatic disease e.g. chronic pancreatitis or hemochromatosis.

(2) Endocrine diseases and genetic syndromes such as acromegaly, pheochromocytoma, Cushing disease and cystic fibrosis.

(3) Drugs and chemicals induced forms of hyperglycemia.

(4) Abnormalities of insulin or its receptors.

(5) Inborn errors of metabolism as type I glycogen storage disease.

(6) Chromosome abnormalities as Down, Turner and klinefelter syndromes

(7) Muscular disorder as myotonic dystrophy and the polyglandular autoimmune syndromes. [1,14]

II-Impaired glucose intolerance.

The term applies to the finding of glucose levels that are higher than normal but lower than that diagnostic of DM. It produced neither symptoms nor complications associated with diabetes. About 25% eventually go on to develop type II diabetes. The patients classified as impaired glucose tolerance (IGT) if they have these findings.

(1) Fasting plasma glucose >115 mg /dl and <140 mg/dl.

(2) The 2-hour oral glucose tolerance test is between 140-200 mg/dl.

(3) Random blood glucose is 200 mg/dl or greater. [15]

III--Gestational diabetes.

This term categorizes increased glucose levels that are first detected during pregnancy. It occurs in about 2% of pregnancies and usually appears in the second or third trimester when antagonistic hormones peak. After delivery glucose tolerance usually returns to normal, within 5 to 10 years 30 to 40% develop type II diabetes. Vigorous treatment often with insulin is required to protect against fetal morbidity and mortality.[16]

Physiology of metabolic hormones:

Hormones play a key role in the regulation of metabolic pathways. A simplified approach is to consider insulin the prime anabolic hormone and epinephrine, glucagon, cortisol and growth hormone as the main catabolic or countregulatory hormones.

(1) Insulin.

Insulin is synthesized and secreted from the B-cells within the islets of langerhans in the pancreas. Islet cells interact with each other through direct contact and through their products e.g. glucagon stimulates insulin secretion and somatostatin inhibits insulin and glucagon secretion. Islet parasympathetic innervations from the vagus stimulates insulin