INTRODUCTION

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 ***(Tasali et al.,2008).***

These standards of care are intended to provide clinicians, patients and other interested individuals with the components of diabetes care, treatment goals, and tools to evaluate the quality of care***(Gaede et al.,2008).***

Type 1 diabetes is associated with a high risk for early atherosclerotic complications. Patients with type 1 diabetes have a fourfold (in men) to eightfold (in women) excess risk of coronary heart disease compared with that for the general population ***(Swerdlow et al.,1996).***

Microvascular disease affects the tissues resulting in ischaemia. This microvascular disease is both structural (thickened basement membrane, capillary wall fragility and thrombosis) and functional (vasomotor neuropathy with defective microcirculation and abnormal endothelial function. Ischaemia also results from macrovascular disease (atherosclerosis) brought about by the direct metabolic injury, changes in vascular reactivity and accumulating endothelial

injury. ***(Jeffcoate and Harding, 2003).*** Development of atherosclerotic lesions in healthy subjects begins upon childhood. In children with type 1 diabetes who had died an unnatural death, an asymptomatic increase in the intima-media thickness of the common carotid artery was found ***(Järvisalo et al.,2002).***

Known risk factors for vascular complications are long-standing diabetes, age, poor glycemic control, smoking, hypertension, obesity, and dyslipidemia ***(Soedamah-Muthu etal.,2004).***

Thus, there is an urgent need for prevention strategies to reduce these risk factors in childhood and adolescence. We recently showed that frequency of regular physical activity represents an important factor influencing glycohemoglobin and, in girls, BMI ***(Herbst et al.,2006).*** Earlier studies have shown that the frequency of regular physical activity affects hemoglobin A_{1c} levels, as well as body mass index in diabetic children.

AIM OF THE WORK

To study the impact of physical activity on:

-Glycemic control by doing HbA1c and by frequent self monitoring of blood glucose .

-Plasma lipids [HDL-c, cholesterol, triglycerides & LDL-c]

-Blood pressure .

-Weight, BMI and Waist circumference .

-Severity and frequency of hypoglycemia

DIABETES MELLITUS

Definition:

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, and blood vessels (***American Diabetes Association, 2007***).

Classification:

The vast majority of cases of diabetes fall into two broad etiopathogenetic categories.

One category, type 1 diabetes, the cause of which is an absolute deficiency of insulin secretion.

In the other, much more prevalent category, type 2 diabetes, the cause of which is a combination of resistance to insulin action and inadequate compensatory insulin secretory response (***ADA, 2007***).

The etiological classification recommended by the American diabetes association (ADA) and the World Health Organization (WHO) expert committee on the classification and the diagnosis is shown in table 1 with minor modification.

Type 1 diabetes is one of the most common childhood illnesses.The incidience of type 1 dibetes increasing rapidly worldwide, and it is also presenting an earlier age (***devendra et al.,2004***)

The goal of medical care for people with diabetes is to optimize glyceamic control and minimize complications.

Treatment that maintains blood glucose levels near normal in type 1 diabetes delays the onset and reduces the progression of microvascular complications. (***ADA, 2007***).

Table (1): Aetiological classification of DM

I. Type 1:

(β-cell destruction, usually leading to absolute insulin deficiency)
A. Autoimmune B. Idiopathic

III. Other Specific Types:

A) Genetic defects of B-cell function:

1.Mitochondrial DNA defect. 2. Wolfram's syndrome.
3. Maturity onset diabetes of the young (MODY).

a. Chromosome 20q (MODY-1). b. Chromosome 7q (MODY-2).
c. Chromosome 12q (MODY-3).

B) Genetic defects in insulin action:

1-Type A insulin resistance 2-Leprechaunism
3-Rabson-Mcndcnhall Syndrome 4-lipodystrophy.

C) Diseases of the exocrine pancreas:

1-Pancreatitis, 2-Trauma 3-Pancreatectomy
4-Neoplasia 5-Cystic fibrosi 6-Hemochromatosis

D) Endocrinopathy:

1-Acromegaly, 2-Cushing syndrome. 3-Glucagonoma
4-Phaeochromocytoma 5-Hyperthyroidism 6-Somatostatinoma
7-Aldostcronoma.

E) Drug or chemical Induced:

1- Necotinic acid. 2- Glucocorticoids 3- Thyroid hormone.
4-B-adrenergic agonist 5-Thiazides. 6- Phenytoin
7-Pentamidine. 8- Diazoxide. 9- Vacor.
10-Interferon-a.

F) Infections:

1-Congenital rubella. 2-Mumps 3-Cytomegalovirus.
4- Adenovirus 5- Coxakie B virus.

G) Uncommon forms of immune mediated diabetes:

1- Anti-insulin receptor antibodies. 2- Stiff-man syndrome.

H) Other genetic syndromes sometimes associated with diabetes mellitus:

1- Down syndrome 2- Klienfelter's syndrome
3-Turner's syndrome 4-Prader-Wili syndrome.
5-Myotonic dystrophy 6-Laurance-Moon-Biedl syndrome
7-Friadreich's ataxia. 8- Huntington's chorea.
9-Porphyrea. 10-Others

IV. Gestational diabetes.

(ISPAD,2009)

Diagnostic criteria for diabetes in childhood and adolescence:

Diagnostic criteria for diabetes are based on blood glucose monitoring and the presence or absence of symptoms.

Three ways to diagnose diabetes are possible and each, in the absence of unequivocal hyperglycemia , must be confirmed , on a subsequent day,by any one of the three methods given in table 2.

Table (2): Criteria for the diagnosis of DM

Symptoms of diabetes plus casual plasma glucose concentration >200mg/dL. (Casual is defined as any time of day without regard to time since the last meal).
Or Fasting plasma glucose >126mg/dL. (Fasting is defined as no caloric intake for at least 8h),
Or 2-h postload glucose >200mg/dL during an oral glucose tolerance test (OGTT). The test should be performed as described by the World Health Organization (WHO), using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water or 1.75g/kg of body weight to a maximum of 75g.

(ISPAD,2009)

Type 1 diabetes mellitus

This condition is characterized by severe insulinopenia and dependence on exogenous insulin to prevent ketosis and to preserve life; It was therefore termed insulin dependant diabetes mellitus (IDDM), The onset occurs predominantly in childhood but it may come at any age. Hence, such terms as juvenile diabetes and brittle diabetes have been abandoned in favor of type 1 diabetes mellitus ***(Petrovsky et al., 2003).***

Incidence and prevalence:

Type 1 Diabetes is one of the most common chronic diseases in childhood. Its incidence varies drastically between populations and even within the same population, much of this variation is due to genetic defect. The incidence of type 1 diabetes is increasing in all population at a rate of approximately 3% per year, and the onset of the condition is occurring at younger ages. The increased incidence has been linear in recent decades ***(Petrovsky et al., 2003)***.

Pathophysiology of type 1 DM:

When 90% of functioning B-cell have been destroyed, loss of insulin secretion becomes clinically significant. With loss of the insulin which is the major anabolic hormone, a catabolic state develops, which is characterized by decreased glucose utilization and increased glucose production via gluconeogensis and glycogenosis, leading to hyperglycemia. In the state of insulin deficiency, levels of counter regulatory

hormones (i.e., glucagons, epinephrine, growth hormone and cortisol) are elevated fatty acids release and keto acids production follow***(ISPAD, 2009).***

Clinical manifestations:

Diabetes type 1 may be asymptomatic or may be presented by:

1) The classic presentation of diabetes in children is a history of polyuria, polydepsia, polyphagia and loss of weight (loss of body fat). The duration of these symptoms is often less than 1 month. Polyphagia occurs as a compensatory mechanism when calories are lost in urine (glucosuria) ***(Kuzuya et al., 2002).***

2) Enuresis: (due to polyuria) in previously toilet-trained child.

3) Insidious onset of lethargy, weakness, and weight loss (in spite of increased appetite) ***(Couper et al., 2007).***

4) Pyognic skin infection and monilial vaginitis (in teenage girls duo to chronic glucosuria) ***(Couper et al., 2007).***

5) Diabetic ketoacidosis (the clinical presentation of 20-40% of diabetic children) ***(ISPAD, 2009).***

Diagnostic difficulties at onset:

A. Young infants with hidden symptoms.

B. Hyperventilation misdiagnosed as pneumonia.

C. Abdominal pain or vomiting . misdiagnosed as abdominal "migraine" or appendicitis.

D. Enuresis or polyuria misdiagnosed as urinary infection.

E. Polydispsia misdiagnosed as habit or psychologic drinking. ***(ISPAD, 2009).***

Diagnosis of type 1 DM according to *(ISPAD, 2009)*:

1- The presence of classic symptoms of diabetes with causal plasma glucose concentration >200 mg/dl (11.1 mmol/L). Causal is defined as any time of the day without regard to time since last meal.

2- Fasting blood glucose (FBG)>126 mg/dl (7.0 mmol/L).

3- Tow hour post load (2hr PG) >.200 mg/dl (11.1 mmol/L) during oral glucose tolerance test (OGTT). The test should be performed as described by WHO (1985), using glucose load containing the equivalent of 1.75 gm/kg (maximum 75 gm) anhydrous glucose dissolved in water.

4- Impaired glucose tolerance (IGT): FBG <126 mg/dl and 2h PPG >140 mg/dl and <200 mg/dl.

5- Impaired fasting glucose (IFG): FBG>110 mg/dl and <126 mg/dl. Tables (2& 3) show diagnostic criteria for diagnosis of DM and IGT.

Table (3): Diagnostic criteria for IGT

New criteria	Criteria of the recent past
Fasting plasma glucose <126 mg/dl (7.0 mmol/I) Or 2hr plasma glucose during the oral glucose tolerance test <200 mg/dl (11.1 mmol/1) but > 140 me/dl	Fasting plasma glucose <140 mg/dl (7.8 mmol/1) Or 2hr plasma glucose during the oral glucose tolerance test > 140 mg/dl (7.8 mmol/I) but <200 mg/dl (11.1 mmol/l) Plus 0.5hr or 1hr or 1.5hr plasma glucose during the oral glucose-tolerance test >200 mg/dl.

(Expert Committee on the Diagnosis and classification of Diabetes Mellitus, 2001)

Table (4): Diagnostic criteria for DM

New criteria	Criteria of the recent past
Symptoms of diabetes plus a random plasma glucose >200 mg/dl (11.1 mmol/1) Or Fasting plasma >126 mg/dl(7.0 mmol/1) Or 2hr plasma glucose during the oral glucose tolerance test >200 mg/dl	Symptoms of diabetes plus a random plasma glucose >200 mg/dl (11.1 mmol/1) Or Fasting plasma glucose > 140 mg/dl(7.8 mmol/1) Or 2hr plasma glucose during the oral glucose Tolerance test >200 mg/dl

Symptoms include polyuria, polydepsia, and unexplained weight loss with glucosuria and ketonuria ***(ISPAD, 2009).***

Treatment of type-1- diabetes:

The principal aim of therapeutic management of children and adolescents with type 1 DM is to maintain them free from acute metabolic crisis of DKA and hypoglycemia and the common acute skin, genital and urinary infection, and to avoid long term micro vascular complications ***(ISPAD, 2009).***

(1)-Nutritional Management:

Diet and lifestyle modification are starting point and mainstay of the treatment of both type I and II DM ***(Pickup and Williams, 2003).***

Dietary management in type 1 diabetes should aim in the short-term to prevent hypoglycemia, and ultimately to reduce the risk of chronic diabetic complications and improve life expectancy ***(DCCT, 1993).***

Energy intake recommendations in *(ISPAD,2009)*

Total daily energy intake should be distributed as follows:

-Carbohydrate 50–55 %.

-Moderate sucrose intake (up to 10% total energy).

-Fat 30–35%

<10% saturated fat + trans fatty acids.

<10% polyunsaturated fat.

>10% monounsaturated fat (up to 20 % total energy).

-n-3 fatty acids (cis configuration): 0.15 g /day.

-Protein 10–15%.

Foods that are naturally rich in dietary antioxidants and micronutrients including B-carotene, vitamin E and C, should be encouraged.

WHO is recommending 400 gm/day of fruits and vegetables for diabetics. Antioxidants may help to reduce free radical-mediated tissue damage and prevent atherosclerosis in diabetics ***(Brekke et al., 2007).***

(2) -Exercise:

Regular exercise (>20 minutes /day) in people with type 1 diabetes has been shown to reduce total cholesterol, low density lipoprotein(LDL) cholesterol, and triglycerides, in addition to marked improvement in live expectancy ***(Moy et al.,2000)***. Patients should be encouraged to exercise regularly. Educate the patients about the effects of exercise on the blood glucose level.

If patients are planning to participate in vigorous exercise for more than 30 minutes, they either decrease the insulin by (10-20% or have an extra snak to prevent hypoglycemia that may develope. These patients must maintain their hydration status during exercise ***(Aneela et al., 2002)***.

(3) -Insulin:

Human insulin is manufactured by recombinant DNA (r DNA) technology (biosynthesis) or by enzymatic conversion of porcine insulin (semi synthesis) ***(Thim et al., 1986)***, Insulin -preparations in current clinical practice are presented in table (5).

Table (5): Types of insulin preparations and suggested action profile

Insulin type	**Onset of action(h)**	**Peak of action(h)**	**Duration of action (h)**
Rapid-acting analogs short acting	0.15-0.35	1-3	3-5
Regular/soluble	0.5-1	2-4	5-8
Intermediate acting			
Semi-lent	1-2	4-10	8-16
Isophane NPH	2-4	4-12	12-24
IZS lent type	3-4	6-15	18-24
Long acting			
Ultralente type	4-8	12-24	20-30
Analog (glargine)	2-4	None	16-24

NPH, neutral protamine Hagedorn insulin; IZS, insulin zinc suspension ***(ISPAD, 2009).***

Insulin galargin is a recombinant human insulin analogue produced by DNA technology using a non pathogenic strain of Escherichia coli ***(Phillip et al., 2000).*** And has a peakless action profile and last approximately 24 hours, so taken once daily ***(Owens and Griffiths, 2002).***

Insulin glargine provides the opportunity to achieve target blood glucose level, more effective and safely compared with NPH ***(Fci et al., 2003, Albright et al., 2004).*** The action profile of insulin glargine suggests that it may replace insulin in a more normal physiological way than NPH insulin, thus it should decrease the risk of hypoglycemia without compromising glycemic control ***(Albright et al., 2004).***

Dose of insulin:

Children with long standing diabetes and no insulin reserve require about 0.7 U/Kg/day If pre pubertal, 1.0 U/Kg/day at mid pubertal, and 1.2U/Kg/day by the end of puberty.

Most children with new-onset diabetes have some residual B-cell function (honey moon period), which reduces exogenous insulin needs ***(Sperling, 2004).***

Monitoring of Type 1 Diabetes Mellitus:

The DCCT provided clear evidence in adult and adolescents that:

• Better metabolic control is associated with fewer and delayed micro vascular complications ***(DCCT, 2001)***.

• Optimal glycemic control could only be assessed and maintained by frequent and accurate monitoring ***(ISPAD, 2009).***