UROGENITAL SURGERY IN DIABETIC PATIENTS

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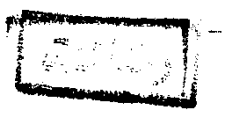

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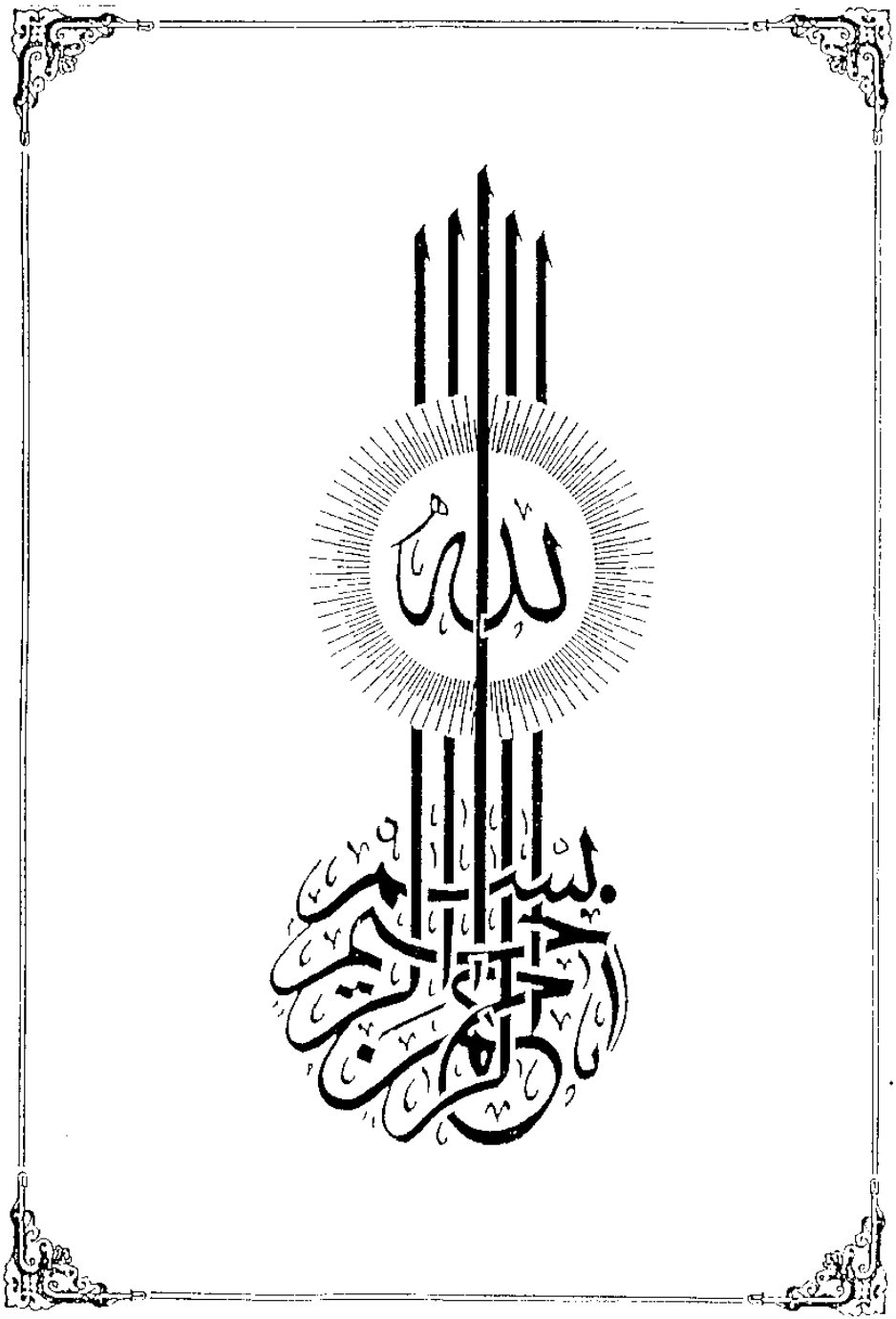

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INTRODUCTION

Diabetes mellitus is the most common endocrine disorder encountered in uro-genital surgery. The uro-genital complications in diabetic patients are not infrequent and they represent a special challenge problem to the urologists during total surgical care.

Many patients may have pre-existing diabetes, which may go out of control during or after urogenital surgery, or they may have clinically unrecognized diabetes, which becomes evident only after surgery, or they may complain of frequent urinary tract infection or impotence as a first presenting feature, in others, renal failure without an obvious etiology may be the presenting picture. Thus, it is very important to identify these patients with the appropriate urine analysis and blood sugar studies to get early diagnosis and careful management of these fragile patients.

Statistical informations revealed that about 30% of maturity onset diabetics have diabetic nephropathy while those who develop their diabetes before the age of 15 years, 60% of them will develop renal failure despite insulin therapy. Renal papillary necrosis is also a frequent disease in diabetic patients being detected in

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up to 7.2% of autopsied diabetics while it is only 0.3% in non diabetics.

Abut 83% of diabetic patients with neuropathy may develop neurogenic bladder dysfunction. Impotence is also a common symptom in diabetic patients.

In the following study we are going to deal with all these complications in details and to point out their proper management.

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CHAPTER (1)

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BIOCHEMICAL AND PATHOLOGICAL CHANGES IN DIABETIC PATIENTS

Diabetes mellitus is not a disease in the classic sense. It has no distinct pathogenesis, etiology, clinical picture or definitive curative therapy. Clinical diabetes mellitus represents a syndrome with disordered metabolism and inappropriate hyperglycemia due to either an absolute deficiency of insulin secretion or a reduction in its biological effectiveness or both (Williams, R.H. 1981).

* Classification and Pathogenesis of Diabetes Mellitus:

Diabetes mellitus is classified into 2 major types in which the age of onset is no longer a criterion (Karam J.H., 1983).

[A]Type-I: Insulin-dependent diabetes mellitus [IDDM]:

This severe form is associated with ketosis in the untreated state. It occurs most commonly in juveniles but also occasionally in adults, especially the non obese and those who are eldery when hyperglycemia first appears. It is a catabolic disorder in which circulating insulin is absent, plasma glucagon is elevated, and the pancreatic B-cells fail to respond to all insulinogenic stimuli. Exogenous insulin is therefore required to

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reverse the catabolic state, prevent ketosis, reduce the hyperglucagonemia, and bring the elevated blood surgar level down.

Certain HLA antigens-B_8, B_{w15}, Dw_3 and Dw_4 - are strongly associated with the development of type-1 diabetes. In addition, circulating islet cell antibodies have been detected in as many as 85% of patients.

Because of these immune characteristics, type-I is felt to result from an infectious or toxic environmental insult to pancreatic B-cells of genetically predisposed persons.

[B] Type - II: Non-insulin-dependent diabetes mellitus [NIDDM]:

This represents a heterogenous group comprising milder forms of diabetes that occur predominantly in adults but occasionally in juveniles. Circulating endogenous insulin is sufficient to prevent ketoacidosis but is often either subnormal or relatively inadequate in the face of increased needs due to tissue insensitivity.

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Type-II diabetes is defined in essentially negative terms: It is a non-ketotic form of diabetes that is not linked to HLA markers on the sixth chromosome; it has no islet cell antibodies; and it is not dependent on exogenous insulin therapy to sustain life.

An element of tissue insensitivity to insulin has been noted in most NIDDM patients irrespective of weight.

Two subgroups of patients with type-II diabetes are currently distinguished by the absence or presence of obesity:-

1. Nonobese NIDDM patient:

The hyperglycemia in this subgroup of patients often responds to oral hypoglycemic agents or, at time, to dietary therapy alone. Occasionally, insulin therapy is required to achieve satisfactory glycemic control even though it is not needed to prevent ketoacidosis.

2. Obese NIDDM patients:

The primary problem is a "target organ" disorder resulting in ineffective insulin action that can secondarily influence pancreatic B-cell function. Hyperplasia of pancreatic B-cells is often present and probably accounts for the fasting hyperinsulinism and exaggerated insulin responses to glucose.

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Type	Ketosis	Islet cell Antibodies	HLA Association	Treatment
(1) Insulin-dependent (IDDM)	Present	Present at onset	Positive	Insulin (mixtures of rapid and intermediate acting, at least twice daily)+diet.
(2) Non-insulin-dependent (NIDDM) (a) Nonobese	Absent	Absent	Negative	1) Eucaloric diet alone. 2) Diet+insulin or sulfonylureas
(b) Obese				1) Weight reduction. 2) Hypocaloric diet+ sulfonylureas or insulin for symptomatic control only

Table (1): Clinical classification of idiopathic diabetes mellitus syndromes. (Karam J.H., 1983).

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The following table shows the differences in clinical features of both types of diabetes mellitus.

No	Symptoms	Type-I diabetes (IDDM)	Type-II diabetes (NIDDM)
1	Polyuria and thirst.	++	+
2	Weakness or fatigue.	++	+
3	Polyphagia with weight loss.	++	-
4	Recurrent blurred vision.	+	++
5	Vulvovaginitis or pruritus.	+	++
6	Peripheral neuropathy.	+	++
7	Nocturnal enuresis.	++	-
8	Often asymptomatic.	-	++

Table (2): Clinical features of diabetes mellitus (Karam J.H., 1983).

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* **Biochemical and metabolic disturbances of diabetes mellitus:**

In order to understand the biochemical and metabolic disturbances of diabetes mellitus as a result of insulin deficiency, it is essential to know the main pathways of carbohydrate metabolism and their interrelationships with those of fat and protein

- **Carbohydrate metabolism:**

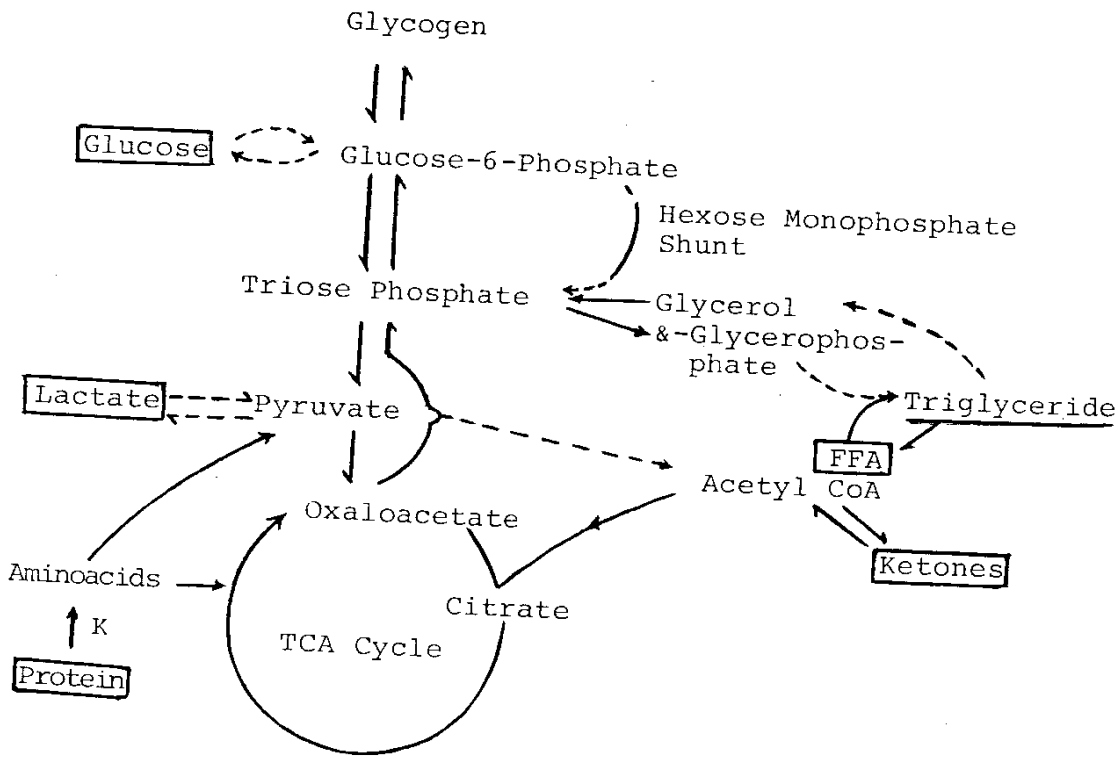

Fig. (1): Carbohydrate metabolism and interrelationships with proteins and fat (Catt K.J. 1971).

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