PROLACTIN LEVEL IN DIABETIC IMPOTENCE

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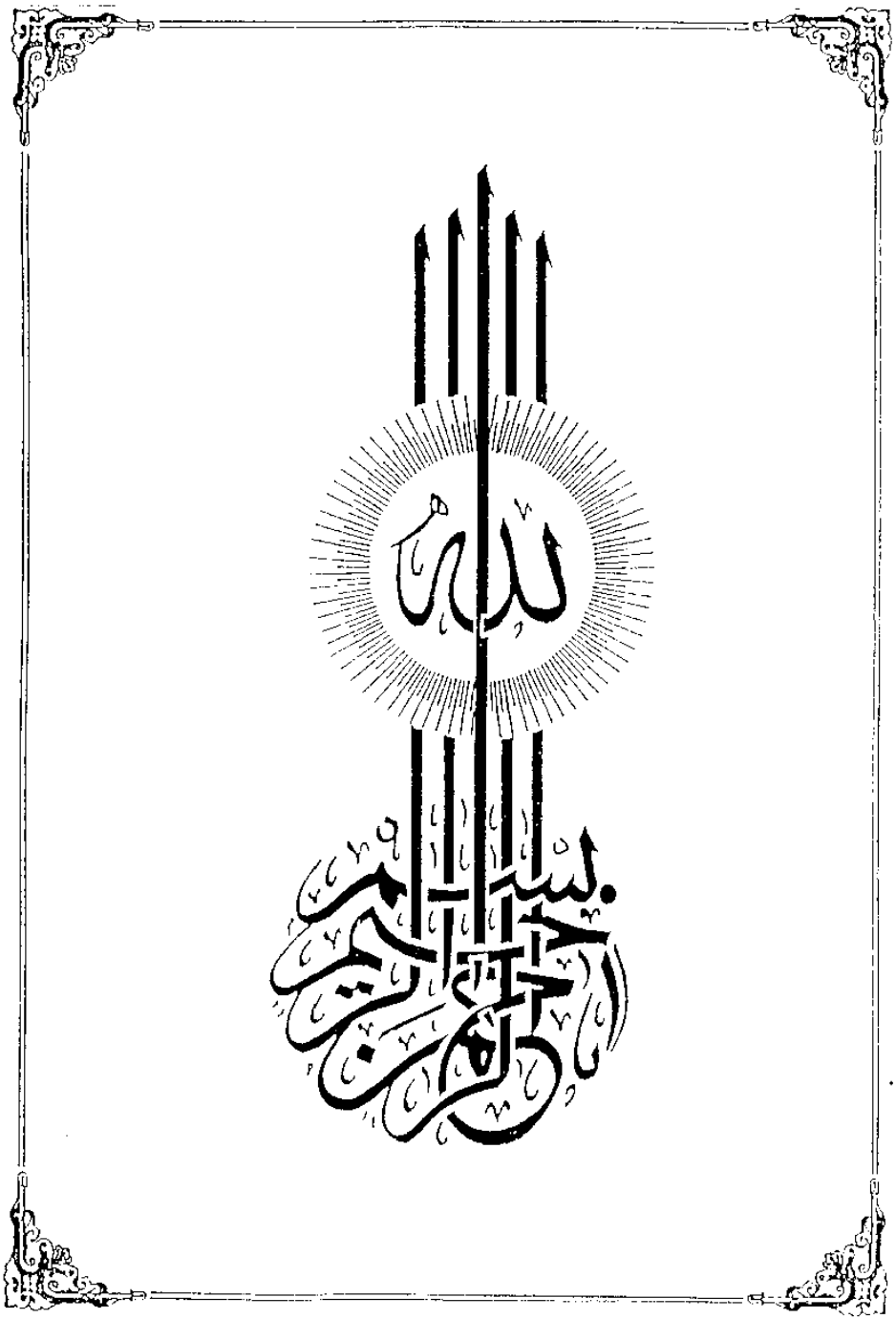

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(PART I)

(PART II)

(PART III)

(PART IV)

(PART V)

(PART VI)

DIABETES MELLITUS

PHYSIOLOGY OF CARBOHYDRATE METABOLISM

Dietary carbohydrates are for the most part polymors of hexoses , of which the most important are galactose , fructose , and glucose. Most of the monosaccharides occuring in the body are the D - isomers .(WILIAM F. GANONG , 1983) .

The principal product of carbohydrate digestion and the principal circulating sugar is glucose . Once it enters the cells , glucose is normally phosphorylated to form glucose 6 - phosphate . The that catalyzes this reaction is hexokinase . In the liver , there is in addition an enzyme called glucokinase , which has greater specificity for glucose and which unlike hexokinase , is increased by insulin and decreased in starvation and diabetes mellitus . The glucose 6 - phosphate is either polymerized into glycogen or catabolised . The process of glycogen formation is called glycogenesis , and glycogen breakdown is called glycogenolysis .Glycogen the storage form of glucose , is present in most body tissues , but the major supplies are in the liver and skeletal muscle. (W. F. GANONG , 1983) .

DEFINITION OF DIABETES MELLITUS

Diabetes mellitus is the most common of the serious metabolic disorder of humans . The true frequency in the general population is difficult to ascertain but probably is somewhere around 1 % , The disease is characterised by a series of hormone - induced metabolic abnormalities , by long - term complications involving the eyes , kidneys , nerves , and blood vessels , and by a lesion of the basement membranes demonstrable by electron microscopy(DANIEL W.F. 1983) .

DIAGNOSIS OF DIABETES MELLITUS

The diagnosis of symptomatic diabetes is not difficult , when a patient presents with signs and symptoms attributable to an osmotic diuresis and is found to have hyperglycaemia. The problem arises with the asymptomatic patient who for one reason or another is considered to be a potential diabetic but has a normal fasting glucose concentration in plasma . Such patients are often given an oral glucose tolerance test , and , if abnormal values are found , diagnosed as having " chemical " diabetes(P.J. WATKINS , 1982) .

CRETERIA FOR DIAGNOSIS

Fasting blood glucose concentration greater than 8 mmol / 1 , and random blood glucose concentration greater than 11 mmol / 1 are clearly diagnostic of diabetes mellitus , and fasting values less than 6 mmol / 1 or random levels less than 8 mmol / 1 exclude diabetes mellitus . Glycosuria usually occures when blood glucose values are greater than 10 mmol/ 1 but this threshold varies considerably between individuals and increases with age(P. J. WATKINS,1982)

STAGES OF DIABETES MELLITUS

Diabetes mellitus passes through three generally recognized stages : pre - diabetes , chemical diabetes, and clinical or symptomatic diabetes (PAUL GH. &AHMED GH. , 1978).

PRE - DIABETES

This is the state of those individuals who could eventually develop diabetes , but in whom no abnormality of carbohydrate metabolism is detectable it covers life from birth to the first abnormal glucose tolerance test .

Clinically, candidates to this group are recruited amongst :

1) Healthy identical twins of diabetic persons .

2) Obese subjects with diabetes in blood relations .

3) Women with abnormal obstetrices , as large babies , repeated miscariages and stillbirths ,hydramnios, toxaemia of pregnancy , and faetal abnormalities .

4) Subjects with diabetes - like vascular or nervous manifestation as coronary artery disease , neuropathy , impotence , peripheral vascular disease.

Biochemically , although glucose tolerance tests are normal , the insulin response to glucose loads varies. Although the reality of the pre - diabetic state , and the importance of its early recognition are universally accepted , there is no agreement as to its characteristics (PAUL GH. & AHMED GH. , 1978).

CHEMICAL DIABETES

This is the stage when tests of glucose tolerance are abnormal although no symptoms have appeared (PAUL GH. & AHMED GH. , 1978) .

CLINICAL DIABETES

At this stage , the disease has already stamped its chemical and pathological print on the organism (PAUL GH. & AHMED GH. , 1978).

CLASSIFICATION OF DIABETES

Nearly all types of diabetes have hyperglycaemia in common , the causes vary as does treatment and long term outlook . Nearly all diabetics have " primary " diabetes , and most of the other syndromes and associations with diabetes is rare (P. J.WATKINS , 1982).

PRIMARY DIABETES MELLITUS

It is of two types:

1) Insulin - dependent diabetes (Type I) .

2) Non - insulin - dependent diabetes (Type II) .

DIABETES ASSOCIATED WITH OTHER CONDITIONS

All the following may be insulin - dependent or non - insulin - dependent diabetes :

1) Pancreatic diseases , pancreatectomy , Pacreatitis , haemochromatosis , carcinoma of pancreas .

2) Hormone - induced diabetes , Cushing's syndrome, phaeochromocytoma , glucagonoma.

3) After burns or other severe illness,usually temporary .

4) Drug - induced diabetes , corticosteriod drugs and A C T H , especially in large doses , diazoxide , thiazide diuretics and the oral contraceptive pill have a weak effect on carbohydrate tolerance .

5) Lipoatrophic diabetes , associated with complete abscence of fat from all tissues, subcutaneous and elsewhere .

6) Insulin receptor abnormalities , including conditions due to insulin - receptor antibodies .

7) Incorrectly synthesised proinsulin and insulin .

8) Genetic syndrome :

   a) Recessive inheritance , rare families developing insulin - dependent diabetes according to a recessive pattern . Other features are associated , namely diabetes insipidus , optic atrophy causing blindness and high - tone deafness(DI DM OA D syndrome)

   b) Other hereditary syndromes .(PETER J. WATKINS , 1982) .

INSULIN - DEPENDENT DIABETES (TYPE I) :

Insulin - dependent diabetes is due to damage to and eventual loss of the B cells of the pancreatic islets of Langerhans with resulting loss of insulin production . The agent that damages the islets is not known , in some cases it may be a virus , in others the damage may be due to autoimmunity . Islet - cell antibodies have been identified , though their precise role is not known (PETER J. WATKINS , 1982) .

GENETICS OF TYPE I DIABETES

Genetic influences are less marked in type I diabetes than in type II diabetes . Only 50 % of identical twins of type I diabetic patients will develop the disease . This also suggests that an environmental factor is required for induction of diabetes in these cases . In contrast , the identical twin of a type II diabetic will usually develop diabetes within a year of onset of the disease in the sibling . Type I diabetes is strongly associated with an increased frequency of certain HLA antigens. The predominant HLA antigens associated with type I diabetes vary in different racial groups (JOHN H.K. et al , 1983) .

Thus , HLA - B8 , -B15 , -B18 , -Cw3 , -Dw3 , -DR3 , and -DR4 occur with increased frequency on leucocytes of Caucasian diabetics , whereas only HLA -Dw3 , -DRw3 , and -DRw4 appear to be correlated with type I diabetes in Asians , Africans , and Latin - Americans . Circulating islet cell antibodies , virtually absent in nondiabetics , have been detected in as many as 85 % of type I diabetes tested in the first few weeks of onset of diabetes (JOHN H. K. et al , 1983)

SUBGROUPS OF TYPE I DIABETES

(1) Subgroup Ia , accounts for 80 % of type I diabetes . These patients have islet cell antibodies only transiently, at the onset of their disease , they seldom have any other associated autoimmune phenomena . The most common HLA types in this subgroup are B15 and DR4 . A viral infection appears to be mainly responsible for the B cell destruction in these patients .

(2) Subgroup Ib accounts for the remainder of type I diabetic patients . These patients have islet cell antibodies that tends to persist in high titers. (JOHN H. K. et al , 1983).