EFFECT OF EGG ON THE BLOOD GLUCAGON LEVEL IN NON INSULIN DEPENDENT DIABETICS

THESIS

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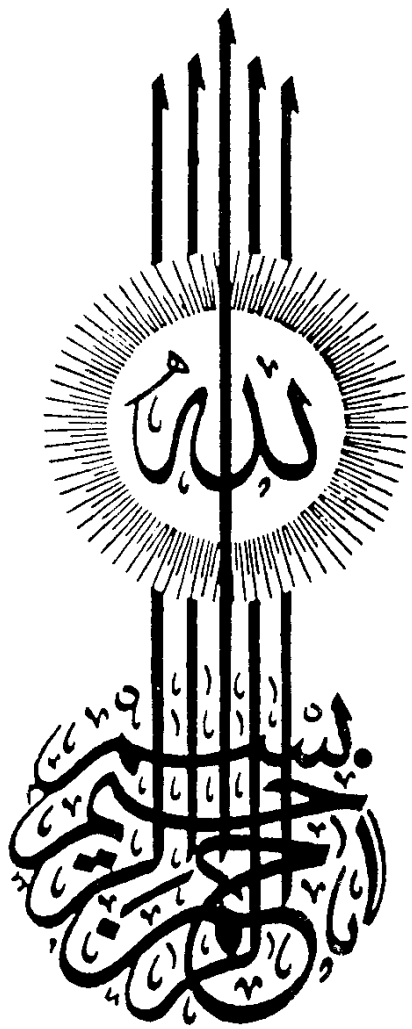

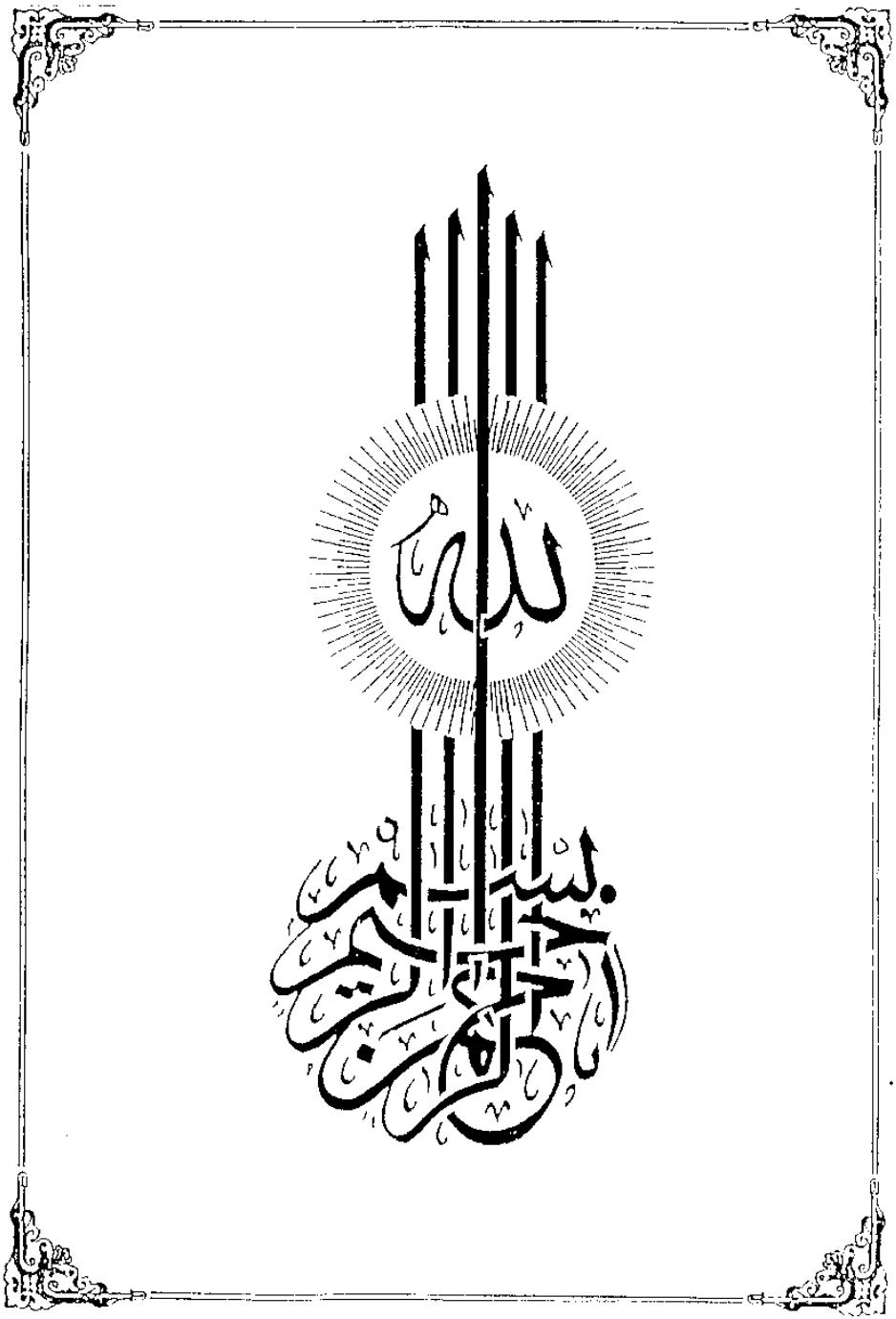

بسم الله الرحمن الرحيم

سنريهم آياتنا في الآفاق وفي أنفسهم حتى يتبين لهم أنه الحق

صدق الله العظيم

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INTRODUCTION

AIM OF THE WORK

INTRODUCTION AND AIM OF WORK

Diabetes Mellitus has been classified as "Insulin dependent " and "Non- insulin dependent" by the national diabetes data group 1976.

Treating diabetes by manipulation of the diet is the oldest form of therapy, being practiced by the Egyptians in 3500 B.C."christakis and Miridjanian 1970!" Through subsequent years and centuries a number of dietary recommendations have been made for treating diabetes. Many of these are based on faulty or incomplete knowledge of the pathophysiology of diabetes.

Recent studies have demonstrated striking abnormalities in the alpha cells function of diabetic subject. In diabetic patients glucagon is not suppressed by carbohydrates and rises normally after protein despite hyperglycemia. Inappropriate hyperglucagonaemia appears to be a common feature of human diabetes and exaggerates the metabolic consequence of insulin lack and influence diabetic control " Muller et al., 1970 ".

The aim of work: Is to evaluate the effect of egg "as a test meal" on the plasma glucose and glucagon levels in patients with type II "Non Insulin Dependent, Diabetes Mellitus" .

REVIEW OF LITERATURE

DIABETES MELLITUS

Definitions :

Diabetes mellitus was defined as a clinical syndrome, characterized primarily by chronic hyperglycaemia and glucosuria. It is caused by a heterogenous group of disorders which have in common either a deficiency or diminished effectiveness of endogenous insulin resulting in a distrubance of carbohydrate, protein and lipid metabolism. " Seth, 1981 ".

Harold E, Leboritz,"1984" also defined diabetes mellitus as a clinical syndrome characterized by inappropriately elevated fasting and/ or postprandial blood glucose and the development of longterm microvascular, macrovascular, and neuropathic changes.

Diagnosis :

Diabetes mellitus occurs either because of lack of insulin or because of the presence of factors that oppose the action of insulin. Diabetes can be diagnosed by measuring blood glucose. Fasting blood glucose concentration greater than 8 m mol/L and random blood glucose concentration greater than 11 m mol/L are clearly diagnostic of diabetes; and fasting values less than 6 m mol/L or random levels less than 8 m mol/L exclude diabetes." Watkins;1982" .

Classifications :

Form the clinical point of view diabetes can be classified into five types :

1- Clinical diabetic :

Is a patient who has diabetes with clinical manifestations (symptoms or complications).

2- Subclinical (or chemical) diabetic :

Is a person whose glucose tolerance is abnormal,but who has no symptoms or complications of diabetes.

3- Latent diabetic :

Is a person who has been diabetic in the past e.g. when pregnant, obese, or on steroid therapy, but whose carbohydrate tolerance is now normal.

4- Gestational diabetic :

Is a woman who becomes diabetic during pregnancy but ceases to be so afterwards.

5- Potential diabetic :

Is a person at special risk of becoming diabetic, e.g. because of strong family or obstetric history, in particular a history of having large babies or of unexplained fetal deaths. " Oakley; 1978 ".

Table (1): Show the main differences between " Insulin dependent and Non-insulin dependent" diabetes mellitus . (Foster, 1980) .

	Juvenile- onset (Insulin Dependent)	Maturity- onset "Non-Insulin Dependent "
Aged	Usually children and young adult	Middle- aged and elderly
Sex	Males- Females	Males - Females
Onset	Acute or subacute	Gradual
Symptoms	Present	May be absent
Nutrition	Normal	Often obese
Weight loss	Marked	Often absent
Ketosis and Coma	Common	Absent or slight
Response to Insulin	Sensitive	Relatively insensitive
Plasma Insulin	Absent or low	Normal or reduced
Response to oral agents	Absent	Present

* Non - insulin dependent diabetes mellitus is a mild from of diabetes that occur predominantly in adults but occasionally in juveniles and in which the 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. (Karam, 1981) .

* On the other hand, non- insulin dependent diabetes mellitus is subdivided into two subgroups which are distinguished by the absence or presence of obesity .

These subgroups are :

A- Non obese NIDDM :

These patients show generally an absent or blunted early phase of insulin release in response to glucose and may often be elicited in response to other insulinogenic stimuli such as acute intravenous administration of glucagon.

B- Obese NIDDM :

It is a secondary to extrapancreatic factors that produce insensitivity to endogenous insulin. Obesity is common in this type of diabetes as a result of excessive intake of calories, perhaps facilitated by hunger resulting from mild postprandial hypoglycaemia after excess insulin release.

On the other hand, Seth (1981), stated that the classification of diabetes mellitus and other categories of

glucose intolerance is as that :

- Diabetes mellitus (DM);
  - Insulin- dependent (type I DM).
  - Non- insulin- dependent (type II DM) .
- DM secondary to certain medical conditions or associated with genetic syndromes.
- Impaired glucose tolerance (IGT).
- Gestational diabetes mellitus.
- Previous abnormality of glucose tolerance.
- Potential abnormality of glucose tolerance.

Etiology of Type II Diabetes Mellitus :

Non -insulin dependent diabetes mellitus (NIDDM) is the commonest from of diabetes mellitus. About 80 % of the diabetic population belong to this category. Nevertheless our understanding of the pathogenesis is still poor. In most patients both abnormali insulin secretion and insulin resistance have been described. It is generally accepted that insulin deficiency results in hyperglycemia , whereas the importance of insulin resistance has been more controversal. However, recently it has been shown that patients with insulin receptor antibodies develop diabetes slowly because of insulin resistance (Flier, Kahn, and Roth, 1975), (Pedersen, Hjollund, and Beck- Nielsenel, Kromann, 1981). The relative significance of insulin deficiency and insulin resistance for development of NIDDM is still unclear.

Furthermore the question of which of the two abnormalities is the primary one has not yet been answered.

Insulin Secretion in NIDDM :

Concerning insulin secretion it is important to differentiate between lean and obese diabetics. In obese, the fasting plasma insulin and C- peptide concentrations very often are increased (Seltzer, et al., 1967) (Beck- Nielsenel, 1978) (Berson, Yallow, 1965). In non- obese subjects the fasting insulin values often are, normal, but they may be both decreased and increased (Seltzer, et al., 1967; Beck-Nielsenel et al., 1980).

The characteristic defect in insulin secretion in both lean and obese NIDDS in the reduced early (first 10 minutes) insulin release both after intravenous and per oral glucose stimulation (Beck-Nielsenel,1978; Berson, Yalow, 1965; Beck-Nielsenel, et al., 1980; Cerasi,et al., 1967).

In obese subjects the total amount of insulin secretion usually is normal or increased, whereas most lean diabetics are insulin deficient. The impairment often seem to be selective for glucose and,the beta cell are partially insensitive to glucose in both thin and obese diabetic subjects (Seltzer, et al., 1967, Vague et al., 1982).