Ain Shams University
Institute of Postgraduate Childhood Studies
Medical Department

Lipid Abnormalities of Diabetic Children (Controlled and Uncontrolled)

A Thesis Submitted for Fullfilment of the Philosophy Degree in Childhood Studies (**Medical Department**)

By

Maher Abdel-Latife Rashed
M.B.B.Ch
Master Degree of Childhood Studies

Supervisors

Prof. Dr. / Ahmed Abdel-Moneem Khashaba
Prof. of Pediatrics
Faculty of Medicine - Banha University

Dr. / Omar El-Sayed El-Shorbagy
Ass. Pro. in Institute of Postgraduate Childhood Studies
Medical Department - Ain Shams University

Ain Shams University
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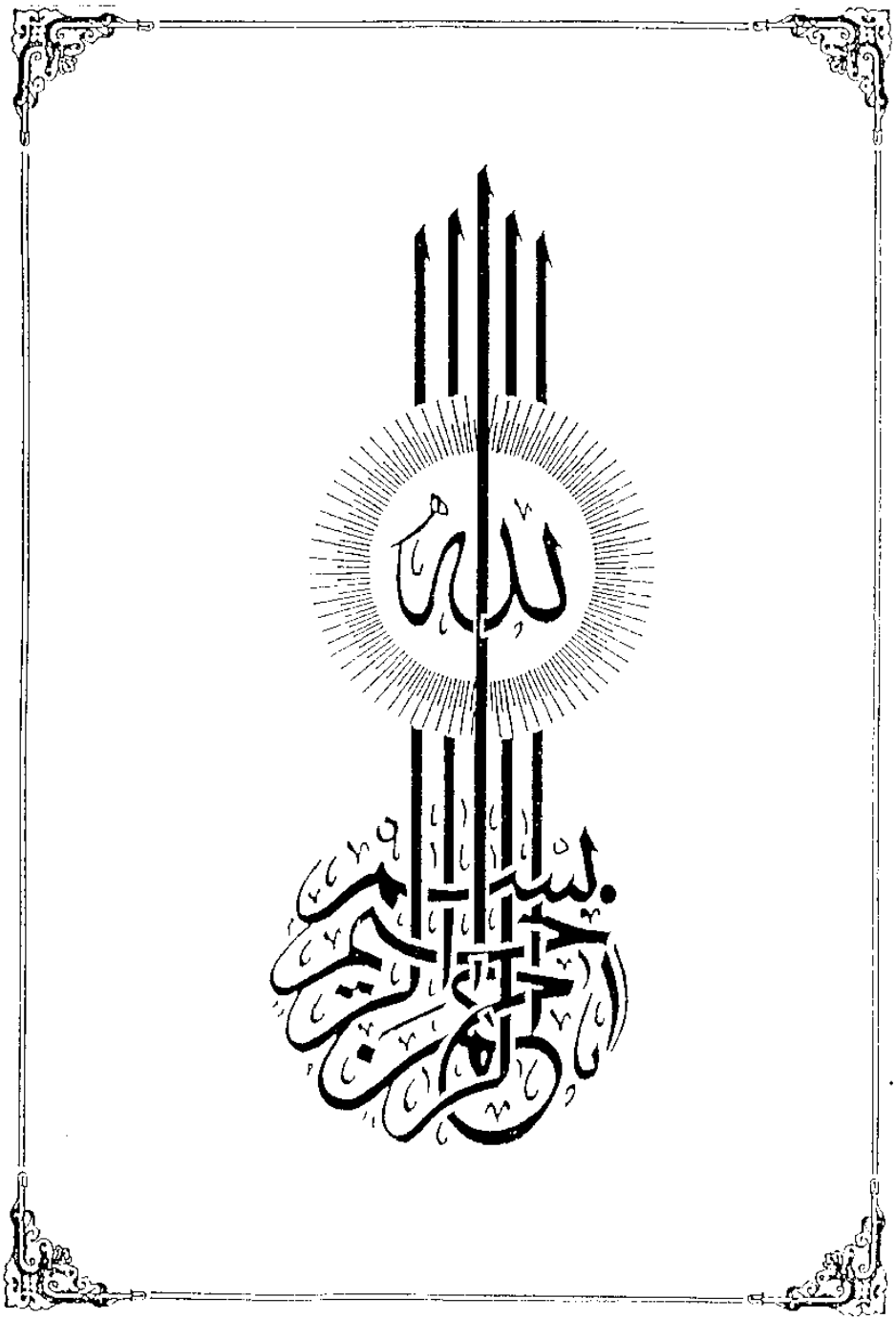

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Abbreviations

Apo	apoproteins (Apolipoproteins)
ATP	Adenosine triphosphate
C	Cholesterol
CAD	Coronary artery disease
CE	Cholesterol ester
CDM	Controlled diabetes mellitus
FCH	Familial combined hyperlipidemia
FFA	Free fatty acid
PL	Phospholipids
HDL	High density lipoproteins
HTL	Hepatic triglyceride lipase
IDDM	Insulin dependent diabetes mellitus
LCAT	Lecithin cholesterol acyl transferase enzyme
LDL	Low density lipoprotein
LPL	Lipoprotein lipase
NC	Normal control subjects
NIDDM	Non insulin dependent diabetes mellitus
NIH	National Institute of Health
TC	Total cholesterol
TG	Triglycerides
VLDL	Very low density lipoproteins
UDM	Uncontrolled diabetes

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Introduction

Atherosclerosis is the most important cause of morbidity and mortality in long standing diabetes mellitus (West,1978). There is a considerable evidence from intervention trials in non-diabetic populations that lowering serum cholesterol reduces the risk of coronary heart disease. Although no such trials have been performed in the diabetic population, said Betteridge (1989), the evidences from non-diabetic populations show such consistency that it is likely to be applicable in the diabetic population; which was also reported by Dunn (1982). He has also pointed out that changes in lipoprotein levels are major factor in the accelerated atherosclerosis which is so prevalent in patients with diabetes in developed world .

Patients with insulin dependent diabetes mellitus are at increased risk for coronary heart disease. Factors that may enhance the risk include dyslipidemia, hypertension and hyperglycemia. Unique abnormalities in the composition and metabolism of lipoproteins may occur in IDDM patients (Garg, 1994) .

Hyperlipidemia plays a crucial role in the etiology of atherosclerosis and coronary heart disease and its prognostic sequelae. It was noticed that the process of atherosclerosis begins as early as the first decade of life (Kwiterovich, 1990); as lipids play an essential physiological role in the cardiovascular system. Cholesterol and phospholipids are integral components of cell membranes; triglycerides are the main vehicles for the transport of fatty acids from the liver and intestine to, for example, the myocardium to provide energy or the endothelium to act as a substrate for prostaglandin synthesis (Thompson,1993).

Mann (1993), has reported that data are accumulating suggested that atherosclerosis begins in early life with formation of ' fatty streaks ' which can be seen in the aorta of children and teenagers; mainly at the

bifurcation points. These fatty streaks were also described by McGill (1968). He has also reported that nearly all children by 3 years of age have some degree of aortic fatty streaks. These begin to increase in size rapidly after eight years of age and involve approximately about fifteen percent of the aortic intimal surface by the fifteen years of age.

Dyslipidemia has been clarified by Thompson, (1993) as the pathogenic potential of excess or deficient numbers of lipoprotein particles in plasma. He has classified dyslipidemia into primary dyslipidemia and secondary dyslipidemia. Primary dyslipidemia has been further classified into familial hypercholesterolemia (FH), type III hyperlipoproteinemia (remnant lipoproteinemia), familial defective apolipoprotien B (FDB) and familial combined hyperlipidemia (FCH). Secondary dyslipidemia can reflect either increased production of lipoproteins (e.g. VLDL in alcohol-induced hypertriglyceridemia and LDL in nephrotic syndrome) or decreased clearance (e.g. the acquired defect of receptor-mediated LDL uptake seen in hypothyroidism). Other causes of secondary dyslipidemia include diabetes mellitus, chronic renal failure, usage of thiazide diuretics and beta adrenergic blocking drugs.

Aim of the study

The aim of the work is to study diabetic children in an attempt to reveal the relationship between plasma lipids and diabetes mellitus (controlled and uncontrolled).

Hypothesis:

* Does diabetes mellitus predisposes to dyslipoproteinemia?
* Is dyslipoproteinemia of diabetes mellitus prediapose to atherosclerosis?
* Does control of diabetes mellitus affect plasma lipid profile?
* Can we consider the control of plasma lipids as a major line of management of diabetic patients?