Ain Shams University
Institute of Post Graduate Childhood Studies

ABNORMAL IRON DISTRIBUTION IN INFANTS OF DIABETIC MOTHERS

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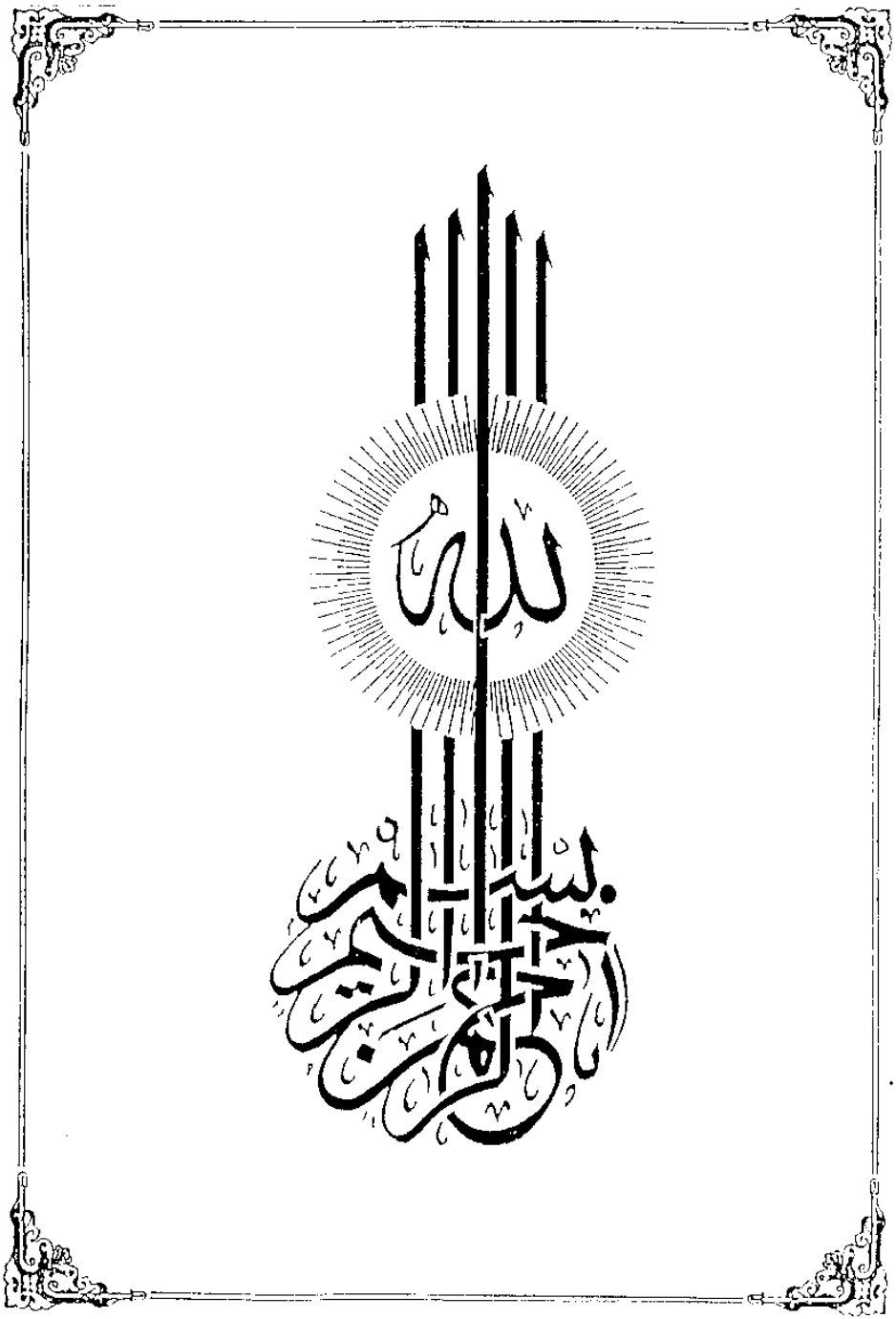

DISCUSSION AND JUDJMENT COMMITTEE

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LIST OF ABBREVIATIONS

AGA	= Appropriate for gestational age
BFU-E	= Burst Forming-Unit-Erythroid
BW	= Birth Weight
CFU-E	= Colony Forming Unit-Erythroid
CNS	= Central nervous system
CS	= Cesarean section
DNA	= Desoxy nucleic acid
EPO	= Erythropoietin
G	= Gram
GA	= Gestational age
GDM	= Gestational diabetes mellitus
Hb	= Hemoglobin
HbA_1C	= Glycosylated hemoglobin
H,C	= Head circumference.
IDDM	= Insulin dependent diabetes mellitus
IDMs	= Infant of diabetic mothers
IQ	= Intelligence quotient
Kg	= Kilogram
LGA	= Large for gestational age
L/S ratio	= Lecithin/sphengomylin
MAC	= Midarm circumference
mg	= Milligram
mRNA	= Massenger ribonucleic acid
Mu	= Micro unit
ng	= nanogram

NICU	= Neonatal intensive care unit.
NVD	= Normal Vaginal Delivery
RDS	= Respiratory distress syndrome
rHUEP	= Recombinant human EPO
RIAs	= Radioimmunoassays
S.C.	= Subcutaneous
SD	= Standard deviation
TIBC	= Total iron binding capacity
Ug	= microgram
WKs	= Weeks
WHO	= World health organization

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INTRODUCTION

Pregnancy in a mother with diabetes constitutes a high-risk pregnancy and the infant of the diabetic mother (IDM) is an infant at high risk requiring special observation in the immediate neonatal period **(Black, 1991)**.

In spite of being macrosomic, infant of diabetic mother is considered fragile angle. He is liable to many complications, such as hypoglycemia, hypocalcemia, respiratory distress; Jaundice, polycythemia, renal vein thrombosis and others **(Lemons and Vargas, 1981)**.

Newborn infants at risk for intrauterine hypoxemia or placental vascular insufficiency appear to have reduced iron stores at birth. These groups include infants of diabetic mothers **(Chockalingam, et al., 1987)**.

Amarnath et al.,(1989) observed that 90% of infants who are large for gestational age and have hypoglycemia at birth, have reduced serum ferritin and iron concentrations, and 67% have elevated total iron binding capacity.

The degree of serum iron abnormalities in hypoglycemic large for gestational age infants, whether born to diabetic mother or not, has been directly correlated with the degree of fetal macrosomia and neonatal hypoglycemia **(Amarnath et al., 1989)**. This raises the possibility that fetal hyperinsulinemia or hyperglycemia wich are directly related to macrosomia and neonatal hypoglycemia, may also involved in the genesis of the iron abnormalities **(Georgieff et al., 1990)**.

It is possible that placental vascular insufficiency could contribute to abnormal iron distribution in IDMs by reducing maternal fetal iron transfer or by causing fetal hypoxemia. Chronic hypoxemia causes a redistribution of iron from serum and storage pools into an expanding erythrocyte mass **(Chockalingam et al., 1987)**.

Infants of diabetic mothers are often hypoxemic in utero and have a high prevalence of polycythemia at birth. Progessively abnormal iron profiles were associated with higher glycosylated fetal Hb values, greater degree of macrosomia, increased hemoglobin and erythropoietin concentrations **(Ruth et al., 1990)**.

Also, the severity of these iron abnormalities is inversely related to the mother glycemic control during pregnancy **(Georgieff et al., 1990)**.