

FETAL MACROSOMIA:RISK FACTORS,DIAGNOSIS AND IMPACT ON LABOUR

THESIS

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ABSTRACT

Macrosomia can be defined as birth weight equal to or exceeding 4000gm. The etiology of fetal macrosomia is believed to be multifactorial. Fetal macrosomia has an important impact on both fetus and mother. Maternal complications include arrest disorders, protraction disorders, and instrumental delivery with more obstetric lacerations, post partum hemorrhage, puerperal infections, cesarean delivery and shoulder dystocia. Fetal complications include birth injuries, asphyxial injuries, neonatal hypoglycemia as well as childhood and adolescent obesity. Accurate prenatal diagnosis of macrosomia is important for planning and timing of the method of delivery. Diagnosis can be done through risk assessment, clinical estimation of fetal weight and Ultrasonography. Preventive factors of fetal macrosomia include reduction of pre-pregnancy weight and weight gain during pregnancy, limitation of post term pregnancy and control of diabetes. The management of patients with suspected fetal macrosomia is controversial. Elective cesarean delivery and labor induction have been proposed as interventions to prevent maternal and perinatal complications.

Key words: fetus, macrosomia, diabetes, shoulder dystocia.

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LIST OF ABBREVIATIONS

AC: Abdominal Circumference

ACOG: American College of Obstetrics and Gynecology

AFI: Amniotic Fluid Index

BMI:Body Mass Index

BPD: Biparietal Diameter

BWT: Birth Weight

CRL: Crown Rump Length

CNS:Central Nervous System

CS: Cesarean Section

CT: Computerized Tomography

DFE: Distal Femoral Epiphysis

EFW: Estimated Fetal Weight

FDL: Femur Diaphysis Length

FL: Femur Length

GA:Gestational Age

GDM: Gestational Diabetes Mellitus

GTT: Glucose Tolerance Test

HA: Head profile Area

HC: Head Circumference

IDDM: Insulin Dependent Diabetes Mellitus

IGF-I: Insulin like Growth Factor I

IGF-II: Insulin like Growth Factor II

LGA: Large for Gestational Age

MRI: Magnetic Resonance Imaging

NICU: Neonatal Intensive Care Unit

NIDDM: Non Insulin Dependent Diabetes Mellitus

NS: Not Significant

OFD: Occipito Frontal Diameter

PGA: Poly Glycolic Acid

PHE: Proximal Humeral Epiphysis

Pro-CS: Prophylactic Cesarean Section

SD: Shoulder Dystocia

STM: Soft Tissue Mass

TC: Thoracic Circumference

TCD: Trans Cerebellar Diameter

ThC: Thigh Circumference

TrC: Trunk Circumference

UC: Umbilical Cord

UK: United Kingdom

VD: Vaginal Delivery

WE: Weight Estimation

AIM OF WORK

In the present study our aim of work is to study the problem of fetal macrosomia with its risk factors,maternal and fetal complications and its impact on labour.

INTRODUCTION

Fetal macrosomia is defined as fetus that is of large size for gestational age equal to or greater than the 90th percentile.

Large for gestational age fetuses can be a result of genetic variance, excessive growth secondary to overweight mothers, excessive maternal weight gain during pregnancy and gestational diabetes or insulin dependent diabetes mellitus(**Haram et al ,2002**).

There is some evidence of increased perinatal mortality and morbidity rates in cases of macrosomia. A number of problems during delivery ,such as prolonged duration of delivery ,an increased risk of cesarean section and postpartum hemorrhage have been widely reported(**Boulet et al, 2006**).

Unless there are additional indications ,there is currently no solid evidence to support induced labour or elective cesarean section in non diabetic patient with suspected fetal macrosomia. Instead, it is recommended that spontaneous delivery should be awaited ,or the labour should be induced only when indicated (**Impey et al ,2000**).

Unfortunately, despite advances in ultrasound technology , our longstanding experience in obtaining fetal biometric measurement and research efforts to date ,the diagnosis of macrosomia still remains problematic(**Chauhan et al, 2005**).

DEFINITION

Several definitions of fetal macrosomia exist, the most common: an absolute birth weight greater than 4500gm, an absolute birth weight greater than 4000gm, a birth weight greater than 90th centile for gestational age. These definition based on the measurement of infants after delivery (retrospective diagnosis).

The American College of Obstetric and Gynecology defines macrosomia as infant with absolute BW > 4500gram irrespective of gestational age or other demographic variable.

A grading system of macrosomia has been described. This grading system has been used in the research setting to determine maternal and neonatal outcome associated with fatal macrosmia.

Macrosmic infant classified in to 3 grades:

- Grade 1: 4000-4499gm
- Grade 2: 4500-4999gm
- Grade 3: > 5000gm.

(Boule et al, 2003)

INCIDENCE:

Globally: the incidence of macrosomic newborn is increasing.

Fetal macrosomia occurs with an incidence of approximately (8-10%) in developed countries. **(Wollschlaeger et al, 1999)**

PATHOGENESIS OF FETAL

MACROSOMIA

Determination of the mechanism that leads to inutero overgrowth has proved elusive because our knowledge has significantly expanded as a result of the generation of experimental mouse models engineered to disrupt the expression of one or more genes, and by detailed molecular and genetic analysis of infants and children with overgrowth syndromes. Studies, which were done on mice, have largely defined the essential role of insulin like growth factors (IGF 1 and IGF II), insulin and their receptors in embryonic and fetal growth
(Sotos,1997)

Hyperinsulinism:

Hyperinsulinism has long been associated with fetal overgrowth. The precise mechanism by which excessive insulin stimulates overgrowth however remains unclear **(Sacks,2007)**.

Although maternal hyperglycaemia appears to be the most common cause of fetal hyperinsulinism, substrates other than glucose such as branched-chain amino acids may stimulate insulin production. Also a stimulatory effect of insulin binding antibodies of maternal origin can not be excluded **(Persson et al, 1986)**.

Fetal hyperinsulinemia occurs in uncontrolled diabetic mothers, and in infants with primary pancreatic pathology, such as islet cell hyperplasia and islet adenomatosis **(Sotos,1997)**.

Regardless of the aetiology, infants with hyperinsulinism often exhibit an increased birth weight. However increased linear growth in- utero, while clearly occurring in some of these infants, is less well documented. It is, therefore, not certain whether all of these

infants are truly overgrown, that is, have somatic cellular hyperplasia. In many of these infants, the increased weight may be due to increased adipose tissue and glycogen stores, which can readily be attributed to the action of insulin **(Verma et al,1997)**.

Macrosomia or fetal obesity is a frequent complication of pregnancy in diabetes mellitus. Several alterations observed in carbohydrate and lipid metabolism in macrosomic infants of diabetic mothers are thought to be a consequence of maternal hyperglycemia leading to fetal hyperinsulinaemia. Macrosomic infant of diabetic mother are prone to the development of glucose intolerance, obesity and diabetes during childhood and adulthood **(Merzouk and Khan,2003)**.

Both clinical and experimental evidences support the central role of hyperinsulinism in the development of fetal macrosomia **(Schwartz et al,1994)**.

Insulin(C-peptide) should be determined in cord blood to define the degree of hyperinsulinemia. Glucose should be determined in plasma at birth and at regular intervals thereafter for detection of neonatal hypoglycaemia. It should be noted that diagnosis of hypoglycaemia cannot be based on glucose measurements with reagent strips **(Schwartz,1999)**.

Nonetheless, when the glucose intolerance of diabetic pregnant women is well controlled, fetal hyperinsulinism does not occur or is minimal and these infants exhibit neither increased birth size nor an increased incidence of other consequences of the disorder **(Barbour, 2003)**.

Relation between insulin-like growth factors and fetal macrosomia: