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Comparison Between Uterine and Umbilical Artery Doppler In Prediction Of Out-come Of Cases of Intra uterine Growth Restriction

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LIST OF ABBREVIATIONS

Abbrev.	Meaning
AC	Abdominal circumference
AEDV	Absent end-diastolic volume
AFV	Amniotic fluid volume
AGA	Appropriate for gestational age
BPD	Biparietal diameter
BPP	Biophysical profile
CPM	Confined placental mosaicism
CPR	Cerebroplacental
CW	Continuous wave Doppler
DV	Ductus venosus
EDD	Expected Date of Delivery
EFW	Estimated fetal weight
ELBW	Extremely Low birth weight
F/A ratio	Femur-to-abdomen ratio
FDA	Fetal Descending Aorta
FGR	Fetal growth restriction
FHR	Fetal heart rate
FVWs	Flow velocity waveforms
GA	Gestational age
Glu T gene	Glucose transport gene
HC	Head circumference
HS	Highly significant
IGF	Insulin like growth factor
IUGR	Intrauterine growth restriction
IVC	Inferior vena cava
IVH	Intraventricular hemorrhage
LBW	Low birth weight
LGA	Large for gestational age
MCA PI	Middle cerebral artery pulsatility index
NPV	Negative predictive value
Ob gene	Obesity gene

PI	The pulsatility index
PPV	Positive predictive value
PW	Pulsed wave Doppler
REDV	Reversed end-diastolic volume
RI	The resistance index
ROC	Receiver operator characteristic
S/D ratio	The Systolic / Diastolic ratio
SFH	Symphyseal fundal height
SGA	Small for gestational age
TCD	Transverse cerebellar diameter
U/S	Ultrasound
UA PI	Umbilical artery pulsatility index
UAV	Umbilical artery velocimetry
UV	Umbilical vein

INTRODUCTION

The estimation of pregnancy dates is important for the mother, who wants to know when to expect the birth of her baby, and for her health care providers, so they may choose the way in which to perform various screening tests and assessments. The three basic methods used to help estimate gestational age (GA) are menstrual history, clinical examination, and ultrasonography ***(Mongelli et al., ٢٠٠٥).***

Small for gestational age (SGA) babies are those whose birth weight lies below the ١٠th percentile for that gestational age. They have usually been the subject of intrauterine growth restriction (IUGR), formerly known as intrauterine growth retardation ***(Kurjak et al., ٢٠٠٦).***

Fetal growth is dependent on genetic, placental and maternal factors. The fetus is thought to have an inherent growth potential that, under normal circumstances, yields a healthy newborn of appropriate size. The maternal-placental-fetal units act in harmony to provide the needs of the fetus while supporting the physiologic changes of the mother. Limitation of growth potential in the fetus is analogous to failure to thrive in the infant ***(Bernstein and Gabbe, ١٩٩٦).*** Fetal growth restriction is the second leading cause of perinatal morbidity and mortality, preceded only by prematurity ***(Bernstein and Gabbe, ١٩٩٦).***

Small for gestational age fetuses can be categorized (according to etiology) into normal (SGA) where no structural anomalies, with normal liquor, normal umbilical artery waveforms (UADWs) and normal growth velocity. As well as abnormal (SGA) where those with structural or genetics abnormalities, in addition to the last category which is fetal growth restriction (FGR) those with impaired placental

function identified by abnormal (UADWs) and reduced growth velocity. ***(Bobrow, et al., ١٩٩٩).***

Doppler ultrasound imaging is presently used to follow-up and manages pregnancies complicated by intrauterine growth restriction, hypertension, diabetes mellitus, twin pregnancies, Rh immunization and fetal malformations. Umbilical, uterine and intracranial arteries are the most commonly studied fetal vessels in the case of intrauterine growth restriction ***(Makhseed et al., ٢٠٠٠).*** The vascular resistance in the maternal uterine, arcuate and umbilical arteries, and in the fetal descending aorta, is usually low, but is high in the fetal carotid and cerebral vessels ***(Makhseed et al., ٢٠٠٠).***

Doppler velocimetry of the uterine arteries reflects vascular impedance on the maternal side of the placental circulation. An increased pulsatility index (PI) and/or notch of the uterine arteries in the second trimester are correlated with FGR and pre-eclampsia later in pregnancy ***(papageorghiou et al ٢٠٠١).***

In growth-restricted pregnancies, Doppler examinations of the umbilical artery can identify pregnancies with increased vascular impedance on the fetal side of the placenta and thus select a group of women in need of increased surveillance ***(madazli and coworkers., ٢٠٠١)***. Increased vascular resistance in the umbilical artery will manifest as decreased diastolic flow, absent and/or reverse diastolic flow which are associated with high perinatal morbidity and mortality due to fetal hypoxia and acidosis ***(Makhseed et al., ٢٠٠٠).***

١-AIM OF THE WORK

The aim of this work is to compare the uterine and umbilical artery Doppler in prediction of the adverse perinatal outcome in pregnancies suspected of intra uterine growth restriction.

NORMAL FETAL GROWTH

Human fetal growth is characterized by sequential patterns of tissue and organ growth, differentiation, and maturation that are determined by maternal provision of substrate, placental transfer of these substrates, and fetal growth potential governed by the genome ***(Cunningham et al., ٢٠٠٥).***

The process of fetal growth comprises three consecutive and somewhat overlapping phases.

The first phase is the phase of cellular hyperplasia and encompasses the first ١٦ weeks of gestation.

The second phase, known as the phase of concomitant hyperplasia and hypertrophy, occurs between the ١٦th and ٣٢nd weeks and involves increases in cell size and number.

The third and final phase, called the phase of cellular hypertrophy, occurs between the ٣٢nd week and term and is characterized by a rapid increase in cell size. Quantitatively, normal singleton fetal growth increases from approximately ٥ g/day at ١٤ to ١٥ weeks of gestation to ١٠ g/day at ٢٠ weeks and ٣٠ to ٣٥ g/day at ٣٢ to ٣٤ weeks, after which the growth rate decreases ***(Resnik, ٢٠٠٢).***

In early fetal life the major determinant of growth is the fetal genome, but later in pregnancy environmental, nutritional, and hormonal influences become increasingly important ***(Holmes et al., ١٩٩٨).***

Fetal growth is the result of a complex interplay of various factors, which include genetic, environmental, maternal, nutritional placental & endocrine influence ***(Sacks, ٢٠٠٤).***

The importance of identifying the determinants of fetal growth is highlighted by the fact that fetal growth restriction remains the

second leading cause of perinatal mortality, and is further enhanced by the association between low birth weight (LBW) and adult-onset disease as cardiovascular diseases and diabetes ***(Barker, ١٩٩٨)***.

Factors that influences the fetal growth

١- *Maternal influences*

Various maternal factors affect fetal growth. These include maternal anthropometry, overall health, nutritional status, and genotype. Several studies have clearly demonstrated correlations between birth weight and maternal height, prepregnant weight, and weight gain during gestation ***(Thame, ٢٠٠٤).***

Good maternal health is essential for proper placental implantation and normal fetal growth and development, as it allows the woman to respond and adapt appropriately to changes related to the establishment and maintenance of pregnancy. Maternal health factors limiting oxygen and nutrient delivery to the fetus have a significant negative impact on fetal growth. For instance, women with cyanotic heart disease, preeclampsia, or significant pulmonary diseases tend to have smaller infants as well as an increased risk of LBW infants. One of the most common maternal medical conditions worldwide that alters fetal growth is anemia ***(Lone et al., ٢٠٠٤)***.

Maternal nutrition is responsible for the availability of nutrients for the fetoplacental unit. Its importance is highlighted by the fact that fetal growth restriction is seen as a result of severe maternal under nutrition in many developing countries and that the incidence of LBW is higher in women with eating disorder ***(Sacks, ٢٠٠٤).***

Because human maternal undernutrition has been associated with decreased placental volume, chorionic villous area, fetal capillary surface area, and volume density of trophoblasts. These all changes

correlated with LBW and thus represent a mechanism by which fetal growth is altered ***(Osgerby et al., ٢٠٠٤).***

At this time, it remains unclear whether nutritional deficiencies in specific dietary components have a greater impact on fetal growth compared with an overall deficiency ***(Ashworth and Antipatis, ٢٠٠١).***

What is becoming more evident, however, is the importance of micronutrient intake on fetal growth and the role that nutrient– gene interactions may have in this process. Some of the more important micronutrients studied to date include folate, Zinc, iron, copper, as well as vitamins E and A ***(Yue-Xin et al., ٢٠٠٠).***

٢- Genetic influences

Elements from both the maternal and the paternal genome are required for normal fetal growth and development. Recent data have demonstrated that, for certain genes, only one allele is functional. This is referred to as genetic imprinting, an epigenetic mechanism by which one of the two alleles of a gene is expressed according to its parental origin. The allele that is silenced is called imprinted that most maternally imprinted genes act as growth suppressors (e.g., H١٩, p٥٧), whereas paternal ones act as growth promoters e.g., insulin-like growth factor ٢, (IGF-٢) ***(Devriendt, ٢٠٠٤)***.

It has been postulated that imprinting occurs because of conflicts between the maternal and paternal genome and nutrient transfer to the fetus from the mother. Thus, paternally expressed genes result in fetal growth promotion at the expense of the mother, whereas genes that are maternally expressed would have the opposite effect ***(Devriendt, ٢٠٠٤)***. It has been shown that biallelic expression of IGF-٢ leads overgrowth of the fetus, which is recognized clinically as Beckwith–Wiedemann syndrome, characterized by large birth weight, organomegaly, macroglossia, and neonatal hypoglycemia. Deletion of