Small for Gestational age

ESSAY

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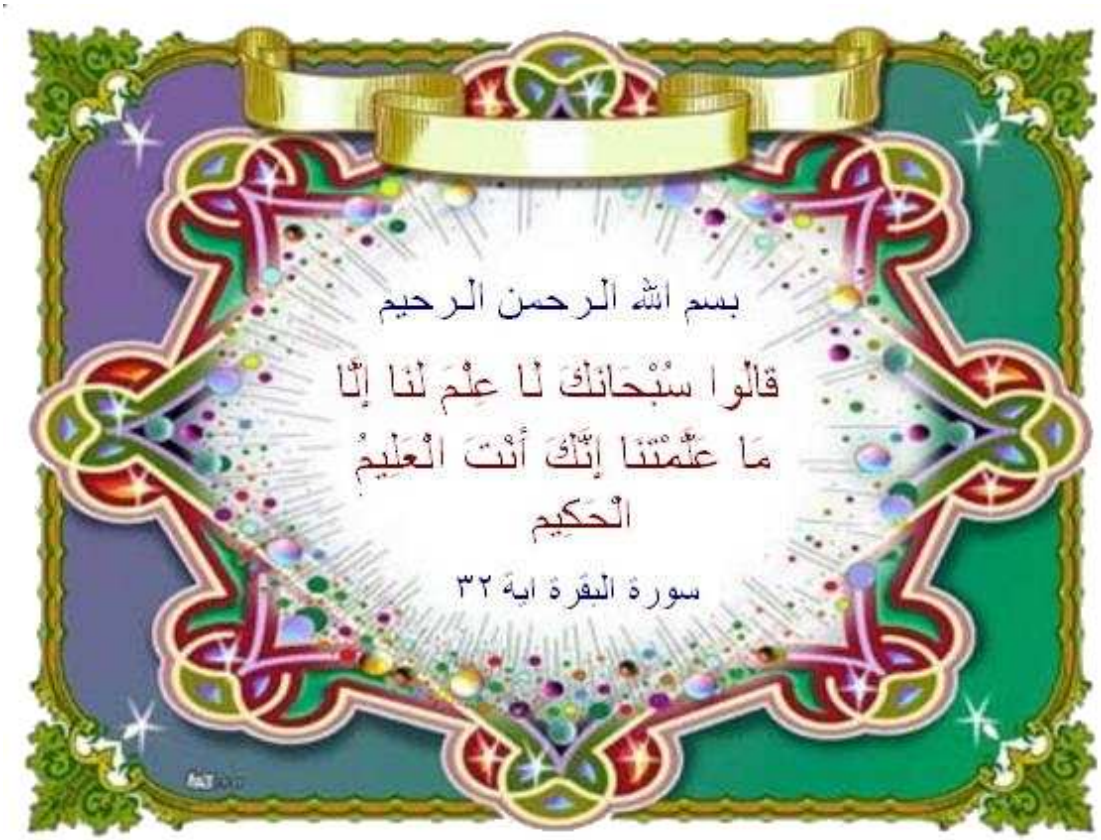
بسم الله الرحمن الرحيم
قالوا سُبْحانكَ لا عِلْمَ لنا إلّا
ما عَلَّمْتَنا إنَّكَ أنْتَ الْعَلِيمُ
الْحَكِيم
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Abstract

Normal fetal growth depends on maternal, fetal, placental, and external factors combined with genetically predetermined growth potential.
Categorization of decreased size by etiology is very important as not every small fetus has equal risk of adverse sequelae.
The early and accurate diagnosis of IUGR may reduce the incidence of morbidity and mortality of fetuses with this condition.
The question of when to deliver a growth restricted fetus has no straight forward answer.
The mode of delivery for fetuses with IUGR should be based entirely on standard obstetric practices. There is no evidence to support a policy of routine cesarean delivery for all fetuses with IUGR.

Key words

fetal growth – IUGR – Small for Gestational Age.

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BASMA AHMED

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List of abbreviations

11B-HSD2	11B-hydroxysteroid dehydrogenase 2
AC	Abdominal circumference
ASF	Abnormal small fetuses
AFP	Alpha-feto protein
AFI	Amniotic fluid index
AFV	Amniotic fluid volume
AGA	Appropriate for gestational age
BPP	Biophysical profile
BPD	Biparietal diameter
CPR	Cerebro placental ratio
EDF	End diastolic flow
EFW	Estimated fetal weight
FL	Femur length
FHR	Fetal heart rate
GTT	Glucose tolerance test
GRF	Growth restricted fetuses
HC	Head circumference
hCG	Human chorionic gonadotrophin
HPL	Human placental lactogen
IGF	Insulin growth factor
IGF-BP	Insulin growth factor binding proteins
IUGR	Intrauterine growth retardation
MCA	Middle cerebral artery
NSF	Normal small fetuses
E3	Oestriol
PI	Pulsatility index
RC/RP	Resistance brain/resistance placenta
RI	Resistance index
SGA	Small for gestational age
S/D	Systolic/diastolic ratio
UA	Umbilical artery

Introduction

Introduction

With respect to gestational age, a fetus may be preterm, term or post term with respect to size, the fetus or infant may be normally grown or appropriate for gestational age (**AGA),** overgrown and consequently large for gestational age (**LGA**) small for gestational age (**SGA) *(David et al, 1998)***.

Two terms requiring clarification are low birth weight (**LBW**) and small for gestational age **(SGA**).

The terms IUGR, LBW and SGA are not synonymous but one of these definitions, where others may meet all three (**Snijders et al, 1993)**.

Fetal growth retardation defined as a birth weight less than the tenth percentile for the gestational age ***(David et al, 2002).***

Up to-70% of SGA infants are small simply due to constitutional factors determined by maternal ethnicity, parity, weight, or height.

The causes of SGA are multiple, involving many different factors. Studies in humans and animals have shown that the maternal environment is the most important determinant of newborn weight, accounting for some similarity in birth weights of siblings than does genetic affinity ***(David et al, 1998)***

The early and accurate diagnosis of IUGR may reduce the incidence of morbidity and mortality of fetuses with this condition **(*Rizzo Get al 2009)***

Clinical prenatal diagnosis remains difficult in spite of the fact that some clinical signs of IUGR have been recognized**(Lin 1998).**

Many clinical methods have been used in the detection of the small fetus. Abdominal palpation and symphyseal-fundal height are the most widely used methods; however they are poor indicators of fetal size. Twenty percent of assessments made immediately prior to delivery using the abdominal palpation method have an error of at least 450 gm (**Gardosi et al,1999**)

The advent of sonography has changed the practice of obstetrics by providing a window to the womb through which the anatomic structure of the fetus can be evaluated. The addition of Doppler flow studies of maternal and fetal vessels has provided a tool where the physiology of the maternal-fetal unit can be assessed. This information can provide the physician and the patient with vital information for a subsequent approach to the pregnancy. The use of fetal Doppler blood flow studies has become common in the evaluation and management of pregnancies complicated by conditions such as suspected fetal growth restriction (sciscione ***AC et al 2009)***

Chapter1

Normal fetal growth

Normal Fetal Growth

Size at birth is determined by two important factors: placental function and duration of pregnancy. Although low birth weight is associated with a variety of peri- and postnatal diseases, fetal overgrowth is also associated with significant neonatal and subsequent morbidity. Prevention of poor intrauterine growth and preterm labor relies upon problems being detected through routine antenatal assessments. Although ultrasonography is considered to be the most reliable method for assessing fetal growth and gestational age, it does suffer from some intrinsic limitations. For example, among the measurements routinely made at 18–20 wk gestation is femur length; however, ultrasonography detects only ossification and not cartilagenous mass at the end of the bone, which could introduce errors**(Paul Saenger et al 2007)**

Factors Influencing Intrauterine Growth

Normal fetal growth depends on maternal, fetal, placental, and external factors combined with genetically predetermined growth potential ***(Jena Miller et al 2008)***

A. Maternal nutrition

Several adverse factors are known to influence the growth of the developing fetus. For example, caloric restriction and insufficient maternal weight gain during gestation can result in the birth of infants of normal length but reduced weight (**Naeye RL et al 1973**). The influence of maternal nutrition in fetal development is complex, and the timing of

adverse nutritional status has an important impact on the shape or weight of the infant. In the Dutch Hunger Winter famine of 1944–45, for example, extreme malnutrition in mothers in their last trimesters of pregnancy gave rise to infants with a low ponderal index at birth (**Stein ZA, et al 1975**). There is limited evidence to suggest that nutritional supplementation during gestation can improve birth weight and reduce the fetal mortality rate (**Gluckman PD et al 2006**).

During the first two trimesters of pregnancy, maternal metabolism, mediated by placental and pituitary hormones, is directed toward energy storage and uteroplacental development. In addition to increased maternal food intake, first-stage insulin secretion typically increases by approximately 60%, whereas sensitivity to insulin and fasting glucose concentrations remains relatively normal (**Freemark M 2006**). The etiology of weight gain during early to midgestation is multifactorial but is likely to include a consequence of the ever-decreasing levels of pituitary GH, which normally inhibits adipogenesis, and the increasing levels of progesterone, prolactin, and placental lactogen, which stimulate food intake, fat storage, and insulin production (**Freemark M 2006**). The hyperinsulinemic state of early and midpregnancy promotes lipogenesis and the storage of fat and is associated with a rise in plasma leptin concentrations and a concomitant decrease in plasma lipid and IGF-I levels (**Freemark M 2006**).

In the later stages of pregnancy, although food intake and fat deposition continue to rise, changes in insulin production and action mean that maternal metabolism is redirected toward supporting fetal, placental, and

mammary growth. Maternal insulin resistance is typical of this stage of gestation. Insulin-mediated glucose utilization by skeletal muscle can drop by 40% or more in the third trimester of gestation, whereas a more modest reduction in insulin-stimulated glucose uptake by cardiac and adipose tissue is normal. Total body insulin sensitivity at this stage of pregnancy can be up to 70% lower than in nonpregnant women (**Freemark M 2006**). These changes in insulin activity during late gestation facilitate efficient storage of energy during times of nutritional abundance, while permitting rapid nutrient mobilization during periods of fasting.

B. Placental size and function

Throughout pregnancy, the size of the placenta changes and remains highly correlated with birth weight; small placentae generally give rise to small babies (**Teasdale F 1984)**. At approximately midgestation, the fetus and placenta are of similar weights, but from 32 wk, fetal growth exceeds that of the placenta and the fetal/placental weight ratio increases. It is unlikely that the size of the placenta causes fetal growth restriction (**Bryan SM 2006**), because the placenta is able to withstand functional inactivation of up to 40% of its villous population without affecting fetal growth (**Fox H 1997**) and is, in any case, capable of compensatory growth (**Hindmarsh PC et al 2000**).