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Intra uterine growth retardation (IUGR)

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ABSTRACT

Intrauterine growth restriction (IUGR) refers to a condition in which a fetus is unable to achieve its genetically determined potential size. This functional definition seeks to identify a population of fetuses at risk for modifiable but otherwise poor outcomes. This definition intentionally excludes of fetuses that are small for gestational age (SGA) but are not pathologically small. SGA is defined as growth at the 10th or less percentile for weight of all fetuses at that gestational age. Not all fetuses that are SGA are pathologically growth restricted and, in fact, may be constitutionally small. Similarly, not all fetuses that have not met their genetic growth potential are in less than the 10th percentile for estimated fetal weight (EFW). Of all fetuses at or below the 10th percentile for growth, only approximately 40% are at high risk of potentially preventable perinatal death. Another 40% of these fetuses are constitutionally small. Because this diagnosis may be made with certainty only in neonates, a significant number of fetuses that are healthy but SGA will be subjected to high-risk protocols and, potentially, iatrogenic prematurity. The remaining 20% of fetuses that are SGA are intrinsically small secondary to a chromosomal or environmental etiology.

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Examples include fetuses with trisomy 18, cytomegalovirus infection, or fetal alcohol syndrome. These fetuses are less likely to benefit from prenatal intervention, and their prognosis is most closely related to the underlying etiology.

KEY WORDS

Intrauterine growth restriction, Fetal growth restriction, Fetal programming, Management, Treatment, SGA.

INTRODUCTION

Intrauterine growth restriction (IUGR) is a common and complex obstetric problem. IUGR is noted to affect approximately 10-15 % of pregnant women IUGR is a diagnosis commonly made antenatally; however, some of these fetuses, especially if unscreened during pregnancy, may be detected only in the newborn period. It is very important for obstetricians and perinatologists to recognize growthrestricted fetuses, because this fetal condition is associated with significant perinatal morbidity and mortality. [(1)] IUGR results when foetus does not grow as per normal foetal growth. There are two types of IUGR babies i.e. symmetrical and asymmetrical. Symmetrical IUGR- when insult on foetal growth occur early. The size of head, body weight and length are equally reduced. Asymmetrical IUGR- the insult on the foetal growth occurs during late gestation producing a brain sparing effect, head circumference is relatively preserved compared to length and weight.

Small for gestational age- It is a statistical definition and denotes weight of infant being less than 2 standard deviation or less than the 10 percentile of the population. SGA and IUGR are considered synonymous. [(2)]

CAUSES OF INTRA UTERINE GROWTH RETARDATION-[(3)(4)]

Maternal causes of IUGR

- Chronic hypertension
- Pregnancy-associated hypertension
- Cyanotic heart disease
- Class F or higher diabetes
- Hemoglobinopathies
- Autoimmune disease
- Protein-calorie malnutrition
- Smoking
- Substance abuse
- Uterine malformations

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- Thrombophilias
- Prolonged high-altitude exposure

Placental or umbilical cord causes of IUGR

- Twin-to-twin transfusion syndrome
- Placental abnormalities
- Chronic abruption
- Placenta previa
- Abnormal cord insertion
- Cord anomalies
- Multiple gestations

IUGR occurs when gas exchange and nutrient delivery to the fetus are not sufficient to allow it to thrive in utero. This process can occur primarily because of maternal disease causing decreased oxygen-carrying capacity (eg, cyanotic heart disease, smoking, hemoglobinopathy), a dysfunctional oxygen delivery system secondary to maternal vascular disease (eg, diabetes with vascular disease, hypertension, autoimmune disease affecting the vessels leading to the placenta), or placental damage.

Perinatal Implications

IUGR causes a spectrum of perinatal complications, including fetal morbidity and mortality, iatrogenic prematurity, fetal compromise in labor, need for induction of labor, and cesarean delivery. In a cohort study in Sweden, a 10-fold increase in late fetal deaths was found among very small fetuses. Similarly, Gardosi et al noted in 1998 that nearly 40% of stillborn fetuses that were not malformed were SGA. Fetuses with IUGR who survive the compromised intrauterine environment are at increased risk for neonatal morbidity. Morbidity for neonates with IUGR includes increased rates of necrotizing enterocolitis, thrombocytopenia, temperature instability, and renal failure. These are thought to occur as a result of the alteration of normal fetal physiology in utero.

With limited nutritional reserve, the fetus redistributes blood flow to sustain function and to help in the development of vital organs. This is called the brain-sparing effect and results in increased relative blood flow to the brain, heart, adrenals, and placenta, with diminished relative flow to the bone marrow, muscles, lungs, GI tract, and kidneys. The brain-sparing effect

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may result in different fetal growth patterns.

In 1977, Campbell and Thoms introduced the idea of symmetric versus asymmetric growth. Symmetrically small fetuses were thought to have some sort of early global insult (eg, aneuploidy, viral infection, fetal alcohol syndrome). Asymmetrically small fetuses were thought to be more likely small secondary to an imposed restriction in nutrient and gas exchange. Investigators since then have disagreed on the importance of this differentiation. Dashe et al examined this issue among 1364 infants who were SGA (20% were asymmetrically grown, 80% symmetrically grown) and 3873 infants who were in the 25-75th percentile (ie, appropriate for gestational age). The symmetrically grown infants who were SGA had outcomes very similar to the infants who were appropriate for gestational age and very different prognoses than the asymmetrically SGA fetuses, thus reinforcing the concept of using growth parameters for diagnostic and outcome counseling.

Relative risks associated with IUGR using morbidity and mortality parameters, from the study by Bernstein et al, are as follows:

- Relative risk of death, 2.77; 95%
- Relative risk of respiratory distress syndrome, 1.19; 95%
- Relative risk of intraventricular hemorrhage, 1.13; 95%
- Relative risk of severe intravascular hemorrhage, 1.27; 95%
- Relative risk of necrotizing enterocolitis, 1.27; 95%

DIAGNOSIS

Criteria for diagnosis of IUGR

For most purposes, an EFW at or below the 10th percentile is used to identify fetuses at risk. Importantly, however, understand that this is not a definitive cutoff for uteroplacental insufficiency. A certain number of fetuses at or below the 10th percentile may be constitutionally small. In these cases, short maternal or paternal height, the neonate's ability to maintain growth along a standardized

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curve, and a lack of other signs of uteroplacental insufficiency (eg, oligohydramnios, abnormal Doppler findings) can be reassuring to the clinician and parents. Customized growth curves for ethnicity, parental size, and gender are in development so as to improve sensitivity and specificity of diagnosing IUGR.

Importantly, review the dating criteria before offering intervention to treat growth restriction in a fetus. If dates are uncertain or unknown, obtaining a second growth assessment over a 2- to 4-week interval may be of value unless strong supportive data or risk factors warrant an immediate change in management plans.

Screening the fetus for growth restriction

Although no single biometric or Doppler measurement is completely accurate for helping make or exclude the diagnosis of growth restriction, screening for IUGR is important to identify at-risk fetuses. Dependent upon the maternal condition associated with IUGR (see Maternal causes of IUGR) patients may undergo serial sonography during their pregnancies. An initial scan may be obtained in the middle of the second trimester (at 18-20 wk) to confirm dates, evaluate for anomalies, and identify multiple gestations. A repeat scan may be scheduled at 28-32 weeks' gestation to assess fetal growth, evidence of asymmetry, and stigmata of brain-sparing physiology (eg, oligohydramnios, abnormal Doppler findings).

Screening for IUGR in the general population relies on symphysis–fundal height measurements. This is a routine portion of prenatal care from 20 weeks' until term. Although recent studies have questioned the accuracy of fundal height measurements, particularly in obese patients, a discrepancy of greater than 3 cm between observed and expected measurements may prompt a growth evaluation using ultrasound. The clinician should be aware that the sensitivity of fundal height measurement is limited, and he or she should maintain a heightened awareness for potential growth-restricted fetuses. In an unselected hospital population, only 26% of fetuses that were SGA were suggested to be SGA based on clinical examination findings.

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Management and Delivery Planning

Once IUGR has been detected, the management of the pregnancy should depend on a surveillance plan that maximizes gestational age while minimizing the risks of neonatal morbidity and mortality. This should include steroid administration when at all feasible, based on the monitoring and delivery strategies. Fetal lung maturity studies by amniocentesis, in fetuses greater than 34 weeks', may additionally influence delivery timing.

The goal in the management of IUGR, because no effective treatments are known, is to deliver the most mature fetus in the best physiological condition possible while minimizing the risk to the mother. Such a goal requires the use of antenatal testing with the hope of identifying the fetus with IUGR before it becomes acidotic. Developing a testing scheme, following it, and having a high index of suspicion in this population when results of testing are abnormal is important. The positive predictive value of an abnormal antenatal test result in fetuses with IUGR is relatively high because the prevalence of acidemia and chronic hypoxemia is relatively high.

Although numerous protocols have been suggested for antenatal monitoring of IUGR fetuses, the mainstay includes a weekly non stress test (NST). Additional modalities may include amniotic fluid volume determination, biophysical profiles, and/or Doppler assessments. Other more complex protocols have been proposed. This protocol integrates multiple venous and arterial Doppler measurements and the biophysical profile score (BPS); this strategy may be used at institutions where these measurements are routinely obtained by qualified technicians. (5)

IUGR In *AYURVEDA*

In Ayurveda, certain disease entities related to fetus are mentioned in the form of garbhsosa, upvistaka, nagodara or upshuska and leenagarbha. By considering the sign symptomplogy of above fetal disorders, there is a lot of difference in opinions about its interpretation. Sarangdhara mentioned the upvistaka, nagodara and gudhagarbha under Astagarbhvyapata; so these are disorders directly related to fetus. Certain authors

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opine that upvistaka, nagodara and leenagarbha are the intrauterine fetal deaths. Then the question arises as why Acharyas mentioned mritgarabh separately. If we look into the treatment portion why garbhvridhikaraharavihar mentioned first then at last garbhapatan, there is a lot of controversy about the interpretation of these terminologies.

Conclusion

IUGR remains a challenging problem for clinicians. Most cases of IUGR occur in pregnancies in which no risk factors are present; therefore, the clinician must be alert to the possibility of a growth disturbance in all pregnancies. No single measurement helps secure the diagnosis; thus, a complex strategy for diagnosis and assessment is necessary. The ability to diagnose the disorder and understand its pathophysiology still outpaces the ability to prevent or treat its complications. The current therapeutic goals are to optimize the timing of delivery to minimize hypoxemia and maximize gestational age and maternal outcome. Further study may elucidate preventive or treatment strategies to assist the growth-restricted fetus.

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