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Diagnosis of IUGR: Traditional Biometry

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An important advance in obstetric medicine will be the improved ability to identify pathologic states of fetal growth, determine their consequences, and implement appropriate interventions. In response to utero-placental insufficiency and under nutrition, the fetus makes physiologic, metabolic, and hormonal adaptations which influence growth, including reducing metabolic dependence on glucose and increasing oxygenation of other nutritional substrates including amino acids and lactate. These endocrine changes combined with reduced nutrient supply divert amino acids from protein synthesis and tissue growth, resulting in impaired somatic growth and diminished growth of kidneys, liver, and heart—the developing organs with the highest rates of cellular turnover. The obstetrician must be able to recognize and accurately diagnosis the fetus with intrauterine growth restriction (IUGR). Ultrasonography is the accepted standard for monitoring fetal growth. Serial ultrasound measurements can provide a reasonable estimate of fetal gestational age and weight based on individual and composite fetal biometric measurements. The purpose of this chapter is to discuss those traditional biometric measurements as they relate to the diagnosis of IUGR.

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Small for gestational age (SGA) is most commonly used to describe a newborn birth weight below the 10th percentile for gestational age and is a definition that can only be applied after birth. Low-birth-weight newborns (<2500 g) have been classified into three groups based on the work of Lubchenco and coworkers.[1,2] The three groups are: (1) preterm neonates: newborns delivered before 37 weeks who are appropriate size for gestational age (preterm AGA); (2) preterm and growth-restricted neonates: newborns delivered before 37 weeks who are small for gestational age (preterm SGA); and (3) term growth-restricted neonates: newborns delivered after 37 weeks who are small for gestational age (term SGA). Unfortunately, it is much less clear as to how to best define a growth-restricted fetus (IUGR) that is failing to reach its full growth potential. In the four decades since the introduction of obstetrical ultrasonography, there has been considerable debate as to what represents IUGR. Commonly used definitions include: estimated weight of fetus below the 10th percentile for gestational age, a fetus with an estimated weight (EFW) below the 3rd, 5th, or 15th percentile for gestational age, estimated weight 2 SD below the mean for gestational age, newborn ponderal index (weight divided by cube of length) below the 10th percentile for gestational age, or when the fetal abdominal circumference measurement is <2 SD below the mean for gestational age.[3]

IUGR infants have a significant risk of increased morbidity and mortality. Neonates born at term weighing between 1500 and 2500 g (<10th percentile) have a 5- to 30-fold increase in perinatal morbidity and mortality compared with infants with weights between the 10th and 90th percentile.[4] Those less than the 3rd percentile (<1500 g) at term have a 70- to 100-fold increase in poor perinatal outcomes. Manning and coworkers reported on the morbidity and mortality among 1560 SGA newborns and demonstrated marked increases as birth weight falls from the 10th to the 1st percentile.[5] Manning identified a cutoff of the 5th percentile as defining a high probability of compromise. Scott and Usher identified a similar relationship with a fetal death rate that rose 8-fold between the 10th and 3rd percentile and 20-fold below the 3rd percentile.[6] Of those newborns that survive, as many as 50% will have significant short- or long-term morbidity.[7,8] Survivors also experience an excess of neurodevelopmental problems during later childhood.

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(ACOG) defines IUGR as a fetus with an estimated weight below the 10th percentile for gestational age.[9] However, not all fetuses measuring less than the 10th percentile are at risk for adverse outcomes and may just be constitutionally small. Of those identified as <10th percentile in utero, as many as 70% will be normal or constitutionally SGA and not at risk of poor fetal or neonatal outcomes.[5] Usher and coworkers used a cutoff of <3rd percentile and missed a significant number of infants with clinically significant IUGR.[10] Seeds and Peng showed an increased odds ratio for perinatal morbidity with those fetuses <15th percentile; however, the false-positive rate was very high.[11] The fact that some fetuses with weights greater than the 10th to 15th percentile demonstrate both physical and biochemical characteristics of fetal growth restriction is another limitation of these definitions and is a reason some have proposed using the ponderal index as a better indicator of a newborn infant with IUGR.[12] It is also the reason for the interest in individualized fetal growth curves.

IUGR has been further divided by some into symmetrical and asymmetrical, depending on the particulars of the individual fetal biometry. Approximately 20% to 30% of IUGR fetuses are symmetrically small (Type I), whereas 70% to 80% exhibit clinical characteristics of asymmetric growth restriction (Type II).[13,14] Symmetrical IUGR is believed to be an early onset condition associated with abnormal growth involving all biometric measures: skeletal, head, and abdomen. Asymmetrical IUGR is generally thought to have a later onset with relative sparing of the skeletal and head measurements but a decreased abdominal circumference: "head sparing IUGR." This is primarily a reflection of less glycogen storage, diminished liver size, and reduced subcutaneous fat. Symmetric IUGR has been described as being associated with conditions that reduce the absolute number of fetal cells, such as chromosomal aneuploidy or early onset congenital infections. Asymmetric IUGR is more often associated with utero-placental insufficiency, a redistribution of fetal blood to vital organs and a reduction in fetal cell growth rather than number. Unfortunately, there is a great deal of overlap between these two entities. Many fetuses with Down syndrome will have fairly normal fetal biometry, whereas more extreme forms of utero-placental insufficiency with early onset, such as antiphospholipid antibody syndrome, severe uncontrolled hypertension, or sickle cell anemia, can be associated with symmetrical reductions in fetal biometry. The gestational age at the onset of interaction of the pregnancy with the high risk factor is more important than the nature of the specific risk factor itself.

Diagnosis of IUGR

Patient History

Accurate diagnosis offers the best opportunity to reduce complications associated with IUGR. A thorough history is necessary to identify potential causes. IUGR may result from a variety of fetal, maternal, or placental origins. Fetal causes include chromosomal disorders, genetic syndromes, and confined placenta mosaicism, which account for approximately 5% to 10% of cases. Polygenic or multifactorial congenital malformations are found in another 10% to 20% of IUGR fetuses, and congenital viral infection (CMV or Toxoplasmosis) is responsible for another 10%. Maternal causes may include chronic or pregnancy-induced hypertension, diabetes with microvascular disease, sickle cell disease, renovascular disease, collagen vascular diseases, congenital or acquired thrombophilias, poor nutrition, smoking, and drug or alcohol abuse, which collectively account for about 25% to 35% of all IUGR fetuses. Early-onset severe preeclampsia and chronic hypertension with superimposed preeclampsia are two of the most common and profound causes of IUGR in nonanomalous fetuses. Finally, uteroplacental insufficiency is often a diagnosis of exclusion but a relatively common cause of IUGR. The placentae of IUGR fetuses will frequently be smaller size for gestational age, demonstrate abnormal function, or both. Electron microscopy of the placentae of IUGR fetuses reveals abnormalities of the terminal villous compartment, which explains the increased vascular impedance seen clinically as abnormal umbilical artery Doppler velocimetry.[15] Structural malformations of the placenta and cord such as placenta previa, accrete, abruption, infarction, circumvillate placenta, chorioangioma, velamentous cord insertion, a single umbilical artery, and monochorionicity are all associated with IUGR.

Whichever test is chosen to diagnose IUGR, it will have a higher positive predictive value if the patient's history includes one or more risk factors for IUGR. Understanding the relationship between disease prevalence in a given population and an increasing positive predictive value of any diagnostic test will help the practitioner interpret findings and choose the appropriate intervention.

Physical Examination

Diagnosis by physical examination alone is limited, and IUGR is frequently missed or incorrectly diagnosed. Studies have shown significant variation in detection rates using fundal height measurements. It is estimated that between 41% and 86% of SGA babies are detected by using serial measurements of fundal height.[16] Fundal height measurements are taken by using a tape measurement in centimeters from the upper edge of the symphysis pubis to the top of the uterine fundus. The uterine height in centimeters generally equals the gestational age in weeks and a measurement of ≥4 centimeters below the expected gestational age suggests growth restriction.[16] External palpation to estimate fetal weight tends to fall within 500 g of the actual birth weight in 80% to 85% of pregnancies, and 69% fall within 10% of the actual birth weight.[17] This error increases when attempting to estimate birth weight in the lower ranges (<2500 g) and in preterm gestations.[18,19]

Ultrasound Criteria

The ultrasound estimated fetal weight using multiple fetal biometric measurements is the most common method for establishing the diagnosis of IUGR. Again, the most commonly used definition of IUGR is a sonographically EFW that