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SEMINARS IN PERINATOLOGY

Timing Delivery of the Growth-Restricted Fetus

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Intrauterine growth restriction (IUGR) is commonly defined as an estimated fetal weight of less than the 10th percentile. While 70% of these are small for normal reasons and not at risk, 30% are pathologically small at risk for numerous complications including fetal death. In the late preterm IUGR fetus (>34 weeks), prematurity risks less and the risk of fetal demise becomes the primary concern. Pulsed-wave Doppler interrogation of the umbilical and middle cerebral artery is useful in reducing perinatal mortality, however, Doppler changes in these vessels of the IUGR fetus may not occur after 34 weeks gestation. There are no randomized trials addressing the timing of delivery of the IUGR fetus in the late preterm or early-term period. However, retrospective reports show an increase risk of fetal demise. While timing the delivery of the late preterm/early-term IUGR fetus requires consideration of multiple factors (e.g. degree of growth restriction, etiology, amniotic fluid volume, and biophysical and Doppler testing), available data suggests that delivery should occur by 37 to 38 weeks for singleton IUGR fetuses. In twin pregnancies with a co-twin IUGR fetus, chorionicity also impacts timing of delivery, but delivery should occur by 34-36 weeks.

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Small for gestational age (SGA) and intrauterine growth restriction (IUGR) are terms frequently used to describe the small fetus. SGA was defined by neonatologists in 1967 to categorize a newborn with a birth weight less than the 10th percentile.[1] Over time, SGA was adopted by obstetricians to broadly classify the under grown fetus regardless of etiology. Although the terms SGA, fetal growth restriction, and IUGR are often used interchangeably, the term IUGR will be used in this article because it reflects both the fetus and placenta and because it is the preferred term of both the American College of Obstetrics and Gynecology (ACOG).[2] An estimated fetal weight (EFW) less than the 10th percentile has been most widely applied as the threshold to define IUGR and has been used by ACOG.[2] Relative to the diagnosis, management and timing of delivery of the IUGR fetus, it is important to be mindful of 3 points regarding this definition. First, this definition, like many, is derived from population-based growth curves, and this does not take into account the individualized growth potential of individuals. As such, it will overdiagnose growth failure and will miss a small percentage of larger fetuses that have failed to achieve their growth potential and may be at risk.[3] Second, Approximately 70% of infants with a birth weight less than the 10th percentile are small but normally grown (constitutionally small), and are not at risk for adverse perinatal outcomes, leaving 30% that are truly IUGR and at risk.[4] Third, lower percentile cutoffs may be more highly associated with adverse perinatal outcomes.[5] Despite the limitations of the 10th percentile based on population growth curves, this cutoff is more sensitive in the identification of fetuses at increased perinatal risk.[6] Most evaluated neonatal complications are increased with decreasing birth weight percentile, even when addressed for pregnancies delivering at term. This is highlighted by a study of more than 137,000 deliveries at term from the North-West Thames regional database. The authors found increased rates of perinatal mortality, meconium staining, emergency cesarean delivery, transfer to the neonatal intensive care unit, as well as low Apgar scores and umbilical cord pH with reduced birth weight percentile (especially below 2 standard deviations).[7]

Although the focus of this article is on timing delivery of the late preterm and early-term IUGR fetus, most what we understand about the pathologic processes and timing of delivery for IUGR is based on studies that were performed on preterm (less than 34 weeks) fetuses. There are a limited

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Table 1 Determinants of delivery timing for the IUGR fetus

Classification and etiology
Behavioral responses
Nonstress testing
Biophysical profile
Fetal movement
Amniotic fluid volume
Doppler velocimetry
Interval growth
Gestational age
Maternal comorbidity(ies)

number of studies that address timing the delivery of the late preterm/early-term IUGR fetus, and it has historically been generally accepted that lower thresholds for delivery of the IUGR fetus should exist after 34 weeks of gestation.[8,9] Whether the pregnancy is preterm, near term, or at term, several factors are important to consider that influence the management of and delivery timing of the IUGR fetus.

Determinants of Delivery of the IUGR Fetus

Factors that influence whether the IUGR fetus should be delivered are listed in Table 1. Understanding the value that each of these determinants brings to the management and delivery of the IUGR fetus helps one understand the current health and developmental status of the fetus. Furthermore, these factors can assist the practitioner in determining whether continued intrauterine life or removal from a hostile intrauterine environment is more appropriate. These factors are reviewed as they pertain to the delivery indication, and in many circumstances are applicable to the preterm, near term and term IUGR fetus.

Classification and Etiology

IUGR can be classified as symmetric or asymmetric, with the latter characterized by a head-to-abdominal circumference ratio of greater than the 95th percentile. This categorization may be helpful in understanding the etiology and to stratify risk, but its clinical utility has not been clearly demonstrated. For example, IUGR because of a placental cause is typically asymmetric but may be symmetric if the insult occurs early in gestation. The underlying etiology is more important and may assist with determination of timing of delivery. For instance, fetal aneuploidy or congenital viral infection may not have outcomes altered by delaying delivery until term. More specifically, the presence of a lethal condition would result in maternal preference with safety taking precedence in timing the delivery. The fetus with IUGR because of uteroplacental disease is most amenable to management with biophysical testing and Doppler velocimetry studies. This diagnosis is usually one of exclusion. However, various phenotypic signs, especially in combination, are suggestive of a uteroplacental cause. These are delineated in Table 2.

Behavioral Responses

A number of methods for assessing fetal well-being, including the nonstress test (NST), biophysical profile (BPP), and maternal perception of fetal movement (FM), can be grouped under behavioral responses. The IUGR condition impacts the fetal response in these tests compared with controls. A decrease in maternal perception of FM has been reported to be a concerning sign for fetal health and helps physicians identify fetuses at increased risk for fetal distress in labor.[10] However, FM assessment is more objectively performed by ultrasound and biophysical profile testing. Approximately 80% of normally developed fetuses at 32 weeks will demonstrate fetal heart rate reactivity and biophysical profile test scores of 8/8 or 8/10. However, IUGR fetuses resulting from uteroplacental dysfunction with secondary chronic hypoxia demonstrate slow maturation of the central nervous system that leads to a delay in all the behavioral responses of the biophysical profile and NST, most noticeably between 28 and 32 weeks gestation. Other findings seen with IUGR compared with normally developed fetuses include an elevated fetal heart rate (FHR) and lower short/long term variability.[11-18]

Although central nervous system maturation is delayed, centrally regulated responses to hypoxia remain preserved.[17] With fetal hypoxemia, there will be a decrease in overall fetal activity, a progressive loss of individual biophysical profile components, and often a gradual decline in amniotic fluid volume.[19,20] With persistent hypoxemia and developing acidemia, fetal breathing motions, body movements, and tone decrease and finally cease.[21] IUGR fetuses delivered before 34 weeks generally receive betamethasone for fetal benefit if delivery is not emergent. Importantly, antenatal corticosteroids influence behavioral responses, including temporary reductions in FHR variability, fetal movements, and fetal breathing motions on days 2 and 3 after administration.[21,22] A reactive NST reflects an absence of fetal acidemia and correlates well with a low risk of fetal demise.[23,24] In contrast, a biophysical profile score of 4 or less, repetitive decelerations, or computerized FHR monitor showing a nonreactive tracing are asso-

Table 2 Phenotype of the IUGR Fetus with Uteroplacental Disease

Asymmetric biometric growth
Exclusion of structural abnormalities
Evidence of brain-sparing effect
Head size maintained
Reduced MCA Doppler index or CPR
Oligohydramnios
Abnormal umbilical artery Doppler velocimetry
Elevated Doppler index
Absent (AEDF) or reversed (REDF) end-diastolic flow
Abnormal venous Doppler velocimetry
Increased venous Doppler indexes
Umbilical venous pulsation
Abnormal biophysical testing
Spontaneous recurrent late decelerations
Abnormal biophysical profile score (≤4)

CPR, cerebral-placental ratio; MCA, middle cerebral artery.