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Fetal Growth Restriction

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Continuing Education Activity

Fetal growth restriction (FGR) is often defined as an estimated fetal weight less than the tenth percentile for gestational age by prenatal ultrasound evaluation. However, distinguishing between a naturally small fetus and one with pathologically restricted growth remains a significant challenge. FGR can result from maternal, fetal, or placental factors and significantly increases the risks of intrauterine demise, neonatal morbidity, and neonatal death. Diagnostic evaluation of FGR primarily comprises ultrasonographic assessment of fetal biometric measurements and Doppler velocimetry, especially of the umbilical artery. Management of fetal growth restriction involves careful fetal surveillance, serial fetal growth and amniotic fluid volume assessment, and determining the appropriate timing for delivery to balance the risks of stillbirth and prematurity. This activity for healthcare professionals is designed to enhance the learner's competence in recognizing fetal growth restriction, performing the recommended evaluation, and implementing an appropriate interprofessional management approach to improve patient outcomes.

Objectives:

- Identify the etiologies of fetal growth restriction.
- Assess the criteria for fetal growth restriction.
- Implement the appropriate management for fetal growth restriction.
- Apply interprofessional team strategies to improve care coordination and outcomes in patients with fetal growth restriction.

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Introduction

Fetal growth restriction (FGR) is a common pregnancy complication worldwide, leading to stillbirth and neonatal mortality and morbidity. FGR is generally defined as the failure of a fetus to achieve its full genetically determined growth potential due to pathologic etiologies, primarily placental dysfunction. The terminology used to classify fetuses and newborns who do not achieve weight within population-based norms is often inconsistent. Historically, FGR has been defined based on fetal size, typically using estimated fetal weight (EFW) or abdominal circumference below the tenth percentile compared to gestational age reference standards. The American College of Obstetricians and Gynecologists (ACOG) employs this criteria for FGR. However, some fetuses are constitutionally small in accordance with their genetic growth potential and are not growth-restricted, while other appropriate for gestational-age fetuses that have not achieved their full growth potential are growth-restricted.[1]

Adding to this confusion is the term small for gestational age (SGA), which is often used synonymously with FGR. ACOG and several other professional societies employ SGA to describe newborns whose birth weight is below the tenth percentile for their gestational age, while others, including the International Federation of Gynecology and Obstetrics (FIGO), use SGA as a preliminary diagnosis to characterize an EFW or birthweight below the tenth percentile.[2] However, most experts utilize the term SGA as a reflection of neonatal size that may or may not be associated with an underlying pathological etiology, whereas FGR is solely due to an antenatal pathologic condition.[3][4][5][6][7]

Consequently, only using fetal size to diagnose FGR can result in misdiagnoses, as the tenth percentile cutoff does not distinguish between healthy, constitutionally small fetuses and those genuinely affected by growth restriction. Additionally, this definition does not identify growth-restricted fetuses with an EFW above the tenth percentile. Evidence suggests that increased perinatal risks can start from higher EFW percentiles, though fetal mortality is the highest in fetuses with an EFW below the third percentile. Therefore, many experts have recommended implementing the third percentile to identify FGR.[8] In 2016, the International Society of Ultrasound in Obstetrics and Gynecology published a consensus defining diagnostic criteria for FGR not associated with congenital abnormalities. These parameters differed depending on whether FGR was early-onset FGR (<32 weeks gestational age) or late-onset FGR (≥32 weeks gestational age) based on gestational age at diagnosis (see **Table**. International Society of Ultrasound in Obstetrics and Gynecology Consensus Definition of Fetal Growth Restriction).[1] Early-onset FGR is usually more severe, often associated with preeclampsia, and easier to detect, whereas late-onset FGR is more common, subtle, and harder to diagnose.[2] The 2016 consensus included the following FGR parameters:

Table

Table. International Society of Ultrasound in Obstetrics and Gynecology Consensus Definition of Fetal Growth Restriction.

Conditions leading to FGR are the disorders inherent to the fetal-placental-maternal unit, fetal malnutrition, and intrauterine space constraints restricting fetal growth, resulting in a significantly increased risk of intrauterine demise, neonatal morbidity, and neonatal death. Diagnostic evaluation of FGR primarily comprises ultrasonographic assessment of fetal biometric measurements and Doppler velocimetry, especially of the umbilical artery.[1] However, the predictive accuracy of these methods can be limited. Management of fetal growth restriction involves careful fetal surveillance, serial fetal growth and amniotic fluid volume assessments, and determining the appropriate timing for delivery to balance the risks of stillbirth and prematurity.[3][2]

Etiology

In fetuses that are diagnosed as SGA, approximately 40% have no underlying pathology and are merely constitutionally small.[1] Conversely, though uteroplacental insufficiency is the most common, several pathologic etiologies are associated with FGR. These etiologies may be categorized into fetal, placental, or maternal causes. However, the pathophysiologic mechanisms can overlap.

Fetal Etiologies

Fetal genetic anomalies are detected in 5% of FGR cases.[3] These anomalies may be due to aneuploidy, uniparental disomy, single-gene mutations, partial deletions or duplications, ring chromosomes, and aberrant genomic imprinting. Half of fetuses with trisomy 13 or 18 develop FGR; the finding of symmetric FGR before 20 weeks of gestation suggests aneuploidy. Fetuses with nonchromosomal congenital anomalies or specific syndromes may also be growth-restricted.[2][3] Additionally, fetal infection is responsible for 5% to 10% of FGR cases, with malaria being the most common cause globally. Other infectious agents include cytomegalovirus, toxoplasmosis, varicella-zoster virus, syphilis, and herpes simplex.[2][3]

Maternal Etiologies

Maternal comorbidities can adversely interfere with uteroplacental-fetal blood flow and cause SGA and FGR, including chronic hypertension, gestational or pregestational diabetes mellitus, systemic lupus erythematosus, antiphospholipid syndrome, severe cardiopulmonary or renal diseases, severe anemia, and sickle cell disease. Substance misuse (eg, alcohol, cocaine, nicotine, heroin, and marijuana) has a significant association with SGA infants as well as multiple gestations, low prepregnancy weight, or poor gestational weight gain. Other maternal causes of FGR include teratogenic medications such as antineoplastic drugs, radiation exposure, chronic antepartum hemorrhage, high altitude residency, multiple gestations, uterine malformations, and assisted conception. Mothers who were growth-restricted carry twice the risk of delivering FGR neonates.[2][3]

Placental and Umbilical Cord Etiologies

Placental anomalies, including bilobate or circumvallate placenta, abruption, infarction, accreta, previa, or placental mesenchymal dysplasia, are also associated with FGR. Umbilical cord anomalies (eg, single artery, velamentous, or marginal cord insertion) are other causes of FGR. These placental and umbilical abnormalities impact fetal growth due to their adverse effects on placental perfusion.[2][3]

Epidemiology

The reported incidence of SGA ranges from 10% to 27% worldwide.[1][9] FGR is identified in about 3% to 9% of pregnancies in developed countries.[1] The incidence varies according to the population studied, fetal gestational age, and whether SGA fetuses were also included. The incidence of FGR is higher in low- and middle-income regions, estimated to be 25%.[1] The prevalence of early-onset FGR is approximately 0.5% to 1%, while late-onset FGR has a prevalence of 5% to 10%.[2] Moreover, women with a prior history of growth-restricted fetuses demonstrate a recurrence rate of 20% in subsequent pregnancies.[10][3] The recurrence risk of SGA is estimated at 20%.[3]

Pathophysiology

FGR secondary to placental insufficiency develops due to inadequate remodeling of the uterine spiral arteries. Furthermore, fetal underlying etiologies of growth restriction may arise following recurrent or persistent cord obstruction (eg, cord compression or hypercoiling). Placental inflammation has also been reported as a mechanism resulting in FGR. However, each of these pathologic changes causes decreased placental function, which leads to impaired fetal growth. Moreover, the intermittent decrease in placental blood flow that frequently occurs during labor increases the risk of birth asphyxia.[1]

Based on additional fetal biometric parameters, including head circumference, abdominal circumference, femur length, and biparietal diameter, FGR can be subcategorized as symmetrical and asymmetrical. In symmetrical FGR, all growth parameters are proportionally reduced, whereas in asymmetrical FGR, the abdominal circumference is classically reduced below the tenth percentile, while other measurements are relatively preserved and may be within normal limits.

Symmetrical Fetal Growth Restriction

This group constitutes 20% to 30% of all FGR cases. Poor placental function is a well-established cause of FGR. Adverse intrauterine conditions beginning as early as the first trimester that may cause fetal nutrient restriction, including smoking, cocaine use, chronic hypertension, anemia, and pregestational diabetes mellitus, may result in symmetrical FGR. Chromosomal anomalies, such as aneuploidy, are also a frequent cause of symmetrical FGR.[11] TORCH (toxoplasmosis, others, rubella, cytomegalovirus, and herpes simplex) infections contracted prenatally are present in 5% to 15% of cases with symmetrical FGR.[12] Depending on the time and duration of occurrence, severe fetal malnutrition can cause either symmetrical or asymmetrical FGR.

Asymmetrical Fetal Growth Restriction

In asymmetrical FGR, which constitutes about 70% to 80% of all FGR cases, the timing of intrauterine insult is in the late second or third trimester of pregnancy. The growth restriction is disproportionate, with relative preservation of head circumference and fetal brain but reduced abdominal circumference. Preeclampsia is a well-recognized cause of asymmetrical FGR. This condition, identified in about 8% of pregnancies in Western countries, generally develops after 20 weeks of gestation and is characterized by hypertension and proteinuria.[13] Chronic hypertension leads to placental vascular remodeling, vascular sclerosis, and ischemia, thus impeding blood flow to the fetus. As a result, the fetal liver glycogen and body adipose tissues are diminished while the brain continues to grow normally with a preferential blood supply.

History and Physical

Clinical History

The maternal history can help identify patients with an increased risk of FGR and SGA. Furthermore, due to the increased recurrence risk of FGR, clinicians should assess patients with FGR in a previous pregnancy for any other growth restriction risk factors, including:

- Previous pregnancy with preeclampsia
- History of smoking or substance abuse
- Multiple gestations
- Low body mass index
- Assisted conception
- Chronic maternal conditions (eg, chronic hypertension, kidney disease, systemic lupus erythematous)
- Extremes of maternal age [2][3]

Physical Examination

Maternal fundal height measurement is a common screening method for SGA and FGR between 24 and 38 weeks of gestation, as the measured height in centimeters should roughly match the gestational age in weeks. Particularly at 32 to 34 weeks, this method has shown a sensitivity of about 65% to 85% and a specificity of 96% for identifying FGR, though conditions such as maternal obesity and uterine leiomyomas can reduce fundal height measurement accuracy.[3]

Neonatal findings characteristic of FGR include a birthweight below the tenth percentile for weight and typically looks emaciated with decreased muscle mass and subcutaneous fat at birth. Depending upon the pathogenic factor for intrauterine growth restriction, the head may look disproportionately large or small. The facies and the umbilical cord may appear thin. Due to the lack of proper bone mineralization and bone formation, the cranial suture may be wide and fontanels large. The Ponderal Index (PI=weight [g] x100/height [cm]) is a good indicator of the severity of fetal malnutrition, especially in asymmetric FGR. A PI below the tenth percentile indicates malnutrition.

Depending upon the cause, specific physical findings may be noted in a growth-restricted neonate, including:

- Hepatomegaly, sensorineural hearing loss, chorioretinitis, blueberry muffin spots in congenital CMV infection [14]
- Low-set ears, cleft palate, clenched fist with overlapping fingers, and rocker bottom feet in trisomy 18 [15]
- Scalp defect, close-set eyes, coloboma, micrognathia, and umbilical hernia in trisomy 13 [15]

Other findings in FGR with known etiology may be referable to the primary cause and manifest according to the specific syndromes involved.

Evaluation

ACOG recommends performing ultrasonography to assess fetal growth if the fundal height differs by >3 cm from the gestational age in weeks.[16] An ultrasound can also detect anatomical abnormalities in the fetus. An accurate assessment of the gestational age is of utmost importance for differentiating FGR from a misdated pregnancy.

The guidelines also stress the need for early detection of high-risk pregnancies, including those with a prior history of FGR, substance misuse, advanced maternal age, preeclampsia, or previous pregnancy complicated with preeclampsia. Serial ultrasonography is strongly indicated if FGR risk factors are identified, as this allows for earlier identification of FGR, which is imperative for timely intervention. Ultrasound imaging may also be considered in patients with a history of a previous SGA infant, though antenatal fetal surveillance is not indicated.[3] If FGR is detected, amniotic fluid volume estimations and umbilical arterial Doppler blood flow velocimetry studies should be performed. Routine screening with third-trimester ultrasound in low-risk pregnancy is not recommended.[16]

Ultrasound Evaluation of Fetal Growth

Biparietal diameter, head circumference (HC), abdominal circumference (AC), and femur length (FL) are the biometric measurements utilized to calculate EFW and assess fetal growth. FIGO

recommends that an EFW finding consistent with SGA (ie, less than the tenth percentile) should prompt further evaluation for FGR. Other useful biometric studies are HC/AC and FL/HC ratios, which can differentiate between symmetrical and asymmetrical FGR. Due to the high prevalence of structural and genetic anomalies in growth-restricted fetuses, an ultrasound examination of fetal anatomy is also recommended if these etiologies have not been excluded yet. If the EFW or AC falls below the tenth percentile, further evaluation is recommended, including amniotic fluid assessment and Doppler blood flow studies of the umbilical artery.[2]

Serial assessments of fetal growth are typically performed every 3 to 4 weeks to compare a fetus' biometric measurements over time, particularly the abdominal circumference. Serial measurements are used to plot fetal growth curves, which can be subsequently compared with various reference charts and to determine the fetus's growth rate. Studies have shown that a fetal growth curve demonstrating a reduced rate of abdominal circumference growth, in addition to Doppler velocimetry and antenatal surveillance, can help diagnose fetal growth restriction and differentiate FGR from constitutionally small fetuses.[8][2]

The primary objective of antenatal fetal surveillance is to mitigate the risk of stillbirth. Various surveillance methods, including contraction stress tests, nonstress tests, and biophysical profiles, are utilized to monitor fetal well-being in pregnancies with preexisting maternal conditions (eg, diabetes) or those complicated by issues like FGR due to the increased risk of fetal demise.[3][2] Most experts recommend initiating antenatal surveillance at a gestational age when delivery might be considered for fetal benefit despite risks of prematurity, typically 32 weeks gestation in many cases. Adding umbilical artery Doppler velocimetry to standard antepartum testing can reduce perinatal death rates by up to 29%.[3] Please see StatPearls' companion resource, "Antenatal Fetal Surveillance," for more information.

Umbilical Artery Doppler Velocimetry

Umbilical artery Doppler velocimetry, typically used in pregnancies complicated by fetal growth restriction, assesses vascular resistance by analyzing flow velocity waveforms. Studies have not shown this modality clinically valuable for fetuses with normal growth patterns, but it can help differentiate a fetus with growth restriction from a genetically small fetus. Doppler imaging is used in fetuses with suspected growth impairment in addition to nonstress tests or biophysical profiles. Studies have shown that umbilical vascular resistance progressively decreases in placentas with physiologically normal function as gestational age increases. As umbilical artery resistance decreases, the pulsatility index subsequently decreases.[17] However, in growth-restricted fetuses with placental insufficiency, the umbilical artery impedance increases until absent end-diastolic flow and reversed end-diastolic flow become apparent secondary to flow being redirected to the fetal brain. This pathophysiologic change also results in an increased pulsatility index.[18][17][18]

Absent or reversed end-diastolic flow in the umbilical artery is linked to a higher risk of perinatal mortality. Evidence shows that clinical indicators consistent with the onset of fetal hypoxemia and acidemia (eg, late decelerations and reduced fetal heart rate variation) developed within an average of 5 days following the onset of absent end-diastolic flow. In fetuses with reversed end-diastolic flow, the fetal condition deteriorates within an average of 2 days.[2]

Trained technicians evaluate umbilical artery Doppler velocimetry by measuring the peak systolic velocity, the frequency shift, the end-diastolic frequency shift, and the mean peak frequency shift over the cardiac cycle according to various ratios, including the systolic to diastolic ratio, resistance index, and pulsatility index. Umbilical placental insufficiency is

reflected in these ratios, which increase abnormally as gestational age progresses instead of decreasing, as seen with normal placental function.[19]

Clinicians have also utilized Doppler velocimetry of other fetal vasculature, primarily the middle cerebral artery, due to several studies demonstrating the association between decreased vascular resistance in the middle cerebral artery and fetal growth restriction and hypoxemia. To increase oxygenation of the fetal brain, fetal blood flow is redistributed to the cerebral vessels at the expense of the fetal body. This redistribution is observed in the reduced pulsatility index caused by middle cerebral artery vasodilatation, which continues to decrease as hypoxemia becomes more severe.[8] However, though professional societies do recommend the use of umbilical artery Doppler velocimetry, some, eg, the Society for Maternal-Fetal Medicine, have not suggested the addition of middle cerebral Doppler velocimetry due to the lack of studies demonstrating its clinical relevance regarding improved fetal outcomes.[18][8]

Treatment / Management

Management of FGR involves careful fetal surveillance, serial fetal growth and amniotic fluid volume assessment, which are utilized to determine the appropriate timing for delivery to balance the risks of stillbirth and prematurity.[3][2] Timely interventions are essential for optimizing neonatal outcomes.

Peripartum Management of Fetal Growth Restriction

The best time to deliver a fetus with growth restriction varies depending on the risk of fetal demise, which is clinically judged based on the cause of the restriction, the estimated gestational age, and other clinical indicators, including antenatal fetal surveillance results. Fetuses <28 weeks gestation have an increased risk of prematurity complications, including bronchopulmonary dysplasia, intraventricular hemorrhage, and surgical necrotizing enterocolitis. These prematurity risks decrease from approximately 35% at 30 weeks to <10% at 34 weeks. Additionally, pregnancies <30 weeks gestation have a 3-times higher rate of neurodevelopmental delay and 8-times higher incidence of cerebral palsy. Therefore, clinicians must attempt to determine if the risk of perinatal mortality outweighs prematurity complications, at which point a patient should be delivered.[3][2]

Delivery timing should be tailored according to clinical considerations as interventions may not benefit some fetuses (eg, aneuploidy or congenital infections) or some patients may prefer not to deliver an extremely premature viable fetus. Individualized, interprofessional approaches are recommended, with antenatal surveillance helping to determine the delivery timing. If delivery is planned, FGR alone does not necessitate cesarean delivery; this decision should depend on other clinical factors.[3][2]

Studies have compared management approaches at various gestational ages to determine the effect on neonatal outcomes, including the Growth Restriction Intervention Trial, which studied early delivery versus expectant management for preterm growth-restricted fetuses <34 weeks gestation, and the Disproportionate Intrauterine Growth Intervention Trial at Term which compared early delivery versus expectant management in patients with suspected FGR at ≥36 weeks gestation. These studies found no significant differences in composite neonatal outcomes between each approach.

Based on these types of studies and expert opinion, FIGO, the Society for Maternal-Fetal Medicine, and ACOG have suggested the following management strategies based on the severity

of FGR, clinical factors, and gestational age:

- **Fetal growth restriction with no additional risk factors**
  - **EFW at third to tenth percentile**: Fetal well-being should be serially assessed beginning at ≥37 weeks gestation with antenatal fetal surveillance 1 to 2 times weekly. Doppler velocimetry 1 to 2 times a week and fetal growth evaluation every 2 weeks is also recommended. Delivery is recommended between 38 0/7 and 39 6/7 weeks of gestation.[3][2]
  - **EFW below the third percentile**: Fetal well-being should be serially assessed beginning at ≥37 weeks gestation with antenatal fetal surveillance 1 to 2 times weekly, Doppler velocimetry 1 to 2 times each week, and fetal growth evaluation every 2 weeks is also recommended. Delivery is advised at 37 0/7 weeks or diagnosis if detected earlier.[3][2]
- **Fetal growth restriction with additional risk factors**: The fetal condition should be serially assessed with antenatal fetal surveillance, Doppler velocimetry 1 to 2 times weekly, and fetal growth evaluation every 2 weeks. Inpatient monitoring and antenatal steroids for fetal lung maturation may also be considered. Delivery is recommended between 34 0/7 and 37 6/7 weeks gestation for cases with additional risks (eg, oligohydramnios, abnormal Doppler studies, or maternal comorbidities).[3][2]
- **Fetal growth restriction with absent or reversed end-diastolic flow**: Growth-restricted fetuses with umbilical artery Doppler velocimetry demonstrating absent end-diastolic flow and reversed end-diastolic flow have an increased risk of fetal demise. Therefore, FIGO recommends fetal well-being should be serially assessed with inpatient antenatal fetal surveillance 1 to 2 times daily, Doppler velocimetry every 2 days, and fetal growth evaluation every 2 weeks. Antenatal steroids for fetal lung maturation should also be administered. Delivery of fetuses with absent end-diastolic flow is recommended at 32 to 34 weeks gestation. In those with reversed end-diastolic flow, delivery is recommended at 30 to 32 weeks gestation. Furthermore, cesarean delivery is the recommended mode, as these fetuses would most likely tolerate the hypoxic stress of labor induction.[2]
- **Fetal growth restriction with absolute delivery criteria**: In these patients, delivery is recommended at any viable gestational age if factors including biophysical profile score ≤2/10, repetitive FHR decelerations, sinusoidal FHR, repetitive late FHR decelerations with absent variability, and bradycardia are presents. Additionally, maternal conditions, eg, severe preeclampsia with uncontrolled hypertension or end-organ damage and HELLP syndrome (ie, hemolysis, elevated liver enzyme levels, and low platelets), are indications for delivery at any viable gestational age.[2]

Considerations for preterm delivery

When planning delivery before 34 weeks, it should be conducted at a facility with a neonatal intensive care unit, preferably with a maternal-fetal specialist's consultation. Antenatal corticosteroids are advised if delivery is anticipated before 33 6/7 weeks to improve neonatal outcomes. For anticipated delivery between 34 0/7 and 36 6/7 weeks, corticosteroids are recommended if preterm delivery is likely within 7 days and the patient has not previously received them. Additionally, for deliveries before 32 weeks, magnesium sulfate is considered for fetal neuroprotection.[3][2]

Neonatal Management

Following the initial stabilization of the SGA infant, a thorough physical exam should be performed, and measurements of head circumference, length, and weight should be obtained. Clinicians should note is these measurements demonstrate symmetric or asymmetric growth. Given their reduced fat stores, approximately 33% of SGA infants develop hypoglycemia. SGA infants are also at increased risk for hypothermia.[20] Additionally, these infants are at risk of subsequent poor feeding, increased calorie expenditure, and slow weight gain. Keeping the mother's room warm, encouraging skin-to-skin, and appropriate clothing and swaddling are effective techniques to maintain euthermia. Still, additional support with an incubator or radiant warmer may be needed for persistent hypothermia. Early establishment of enteral feeding should be a priority to avoid hypoglycemia. As per the American Academy of Pediatrics and the World Health Organization, exclusive breastfeeding from birth until 6 months of age should be encouraged for all infants, including preterm and SGA infants.[21][20][22] In the SGA population, glucose should be checked every 3 hours until stable with a preprandial goal of >25 mg/dL for asymptomatic infants in the first 4 hours of life, >35 mg/dL in hours 4 to 24 of life, and >45 mg/dL after 24 hours.[23] Supplementation with formula or dextrose-containing intravenous fluid may be needed while breastfeeding or milk supply is established.

Additionally, an investigation into the etiology of FGR should be undertaken if it remains unknown. Serum and urine testing for congenital infections such as toxoplasmosis and *Cytomegalovirus* should be considered. If the infant presents with dysmorphology, genetic testing should be considered as well. Most SGA infants have normal brain growth, as fetal blood flow is typically redistributed to the brain, or "brain-sparing," in FGR. However, studies have shown that the cerebellum, hippocampus, and cerebral cortex may still be affected, with impaired memory and attention frequently noted in SGA infants. Neurocognitive screening is recommended in preterm infants or those with a small HC measurement. Continued monitoring of infant weight, length, head circumference, and body mass index is recommended every 3 months during the first year of life, then every 6 months the second year, and annually yearly thereafter until their height has reached a normal range.[20]

Differential Diagnosis

Other conditions that should be considered when evaluating FGR include:

- Misdated pregnancy
- Oligohydramnios
- Genetically small fetus.

Prognosis

The prognosis differs between infants with symmetrical or asymmetrical FGR. Asymmetrical FGR infants, in general, have a better prognosis compared to those with symmetrical FGR. As the timing of intrauterine insult is later in pregnancy in asymmetrical FGR, cell number is usually normal, which translates to average postnatal growth.[24] However, in symmetrical FGR, the body cell number may be reduced at birth following an earlier gestational insult.

Additionally, SGA infants have lower survival rates than those appropriate for gestational age. Studies have shown a clear inverse relationship between birth weight percentile and stillbirth risk, which is more significant during the early preterm period than at term. Although

prematurity contributes to this, birth weight itself is an independent predictor of neonatal mortality. A study found that term SGA neonates had a 5-fold higher mortality rate compared to their appropriate for gestational age counterparts.[2]

Complications

Short-term and long-term complications are associated with FGR. Short-term complications occur soon after birth and include fetal demise, respiratory distress, perinatal asphyxia, meconium aspiration syndrome, hypoglycemia, polycythemia, hyperviscosity, nonphysiological hyperbilirubinemia, sepsis, hypocalcemia, poor thermoregulation, and immunological incompetence. Prematurity-related morbidities, eg, respiratory distress syndrome, necrotizing enterocolitis, patent ductus arteriosus, intracranial hemorrhage, and retinopathy of prematurity, may also develop.[25][2]

Many long-term complications are associated with severe growth-restricted fetuses and SGA neonates. An increased risk of adverse neurodevelopmental outcomes, including cognitive delay, poor academic performance, decreased cognitive performance, behavioral problems, and hyperactivity has been demonstrated in studies.[24] Cognitive and neurodevelopmental abnormalities are more common in growth-restricted preterm infants. These anomalies include poor cognitive test scores, a need for special education, variable fine and gross motor dysfunction, attention deficit hyperactivity disorder, and cerebral palsy. Such infants are also more prone to have failure to thrive. FGR infants are reported to be at increased risk of obesity, cardiovascular diseases, metabolic syndrome, hypercholesterolemia, dyslipidemia, diabetes mellitus, and renal diseases later in life.[26][2][20]

Deterrence and Patient Education

Several strategies have been suggested for preventing FGR and SGA, but they have not proven effective. ACOG does not recommend low-dose aspirin for the prevention of uteroplacental insufficiency, as studies have not demonstrated improved outcomes. Dietary supplementation and bed rest have also been investigated but have not reduced the incidence of FGR or SGA.[3]

Maternal substance abuse, which can potentially impede placental vascular functions, should be identified as early as possible. Smoking is also a modifiable risk factor. Smoking cessation has proven to be effective in decreasing the risk of FGR and SGA. Counseling on smoking cessation, as well as for other substance abuse, should be provided, and aid should be offered whenever possible.[27]

Enhancing Healthcare Team Outcomes

Enhancing patient-centered care, outcomes, patient safety, and team performance in the context of FGR and SGA requires a multifaceted approach involving skills, strategies, responsibilities, interprofessional communication, and care coordination among various health professionals. Physicians play a critical role in diagnosing and managing FGR, utilizing their expertise to determine appropriate interventions and timing of delivery. Advanced practitioners complement physicians by providing continuous patient monitoring, education, and support. Nurses are essential in delivering hands-on care, performing routine assessments, and ensuring patient compliance with medical recommendations. Pharmacists contribute by managing and advising on the safe use of medications, particularly when dealing with antenatal corticosteroids or other pharmacological interventions. Effective interprofessional communication is crucial to

synchronize efforts across the healthcare team, ensuring that all members are informed about the patient's condition, treatment plan, and any status changes.

Care coordination is another vital element involving the seamless integration of services provided by different professionals to avoid duplication and gaps in care. This includes scheduling follow-up appointments, arranging for necessary maternal-fetal medicine consultations, and coordinating with neonatal intensive care units when preterm delivery is anticipated. By fostering a collaborative environment where each team member understands their role and responsibilities, the healthcare team can optimize care for patients with FGR. This collaborative approach not only enhances patient outcomes and safety but also improves overall team performance, creating a more efficient and effective healthcare delivery system.

Review Questions

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