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Review Article

Fetal growth restriction: aetiology, screening, diagnosis and management

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ABSTRACT

Foetal growth restriction (FGR) is a pathological condition that refers to a foetus that fails to reach his/her genetically predetermined growth potential. FGR remains a leading contributor to perinatal mortality and morbidity and metabolic syndrome in later life. The purpose of this review is to provide the summary statements of the aetiology of FGR and to establish a framework for screening, diagnosis, and management of pregnancies affected with foetal growth restriction. For this study, published literature was retrieved through searches of PubMed (Medline), Highwire, Google Scholar, Scopus, Cochrane Database of Systematic Reviews. Conference/Seminar proceedings, textbooks, previously published systematic reviews, controlled clinical trials, high quality prospective and retrospective observational studies, research articles and unpublished studies were also used for this study. The identification of FGR begins with assessment of maternal, foetal, placental and environmental risk factors and the diagnosis is made by Ultrasound biometry examination. Functional assessment of placental and foetal circulation by Doppler velocimetry and blood flow volume, together with computerized assessment of foetal heart rate variability, are key examinations in early and late FGR to assess severity of the disease and monitor foetal wellbeing. Appropriate timing of delivery in early FGR might change the outcome, and appropriate monitoring in late and term FGR might avoid unnecessary interventions.

Keywords: Foetal growth restriction, Foetal abdominal circumference, Doppler velocimetry, Screening, Diagnosis, Management

INTRODUCTION

Fetal growth restriction (FGR) is a pathological condition that refers to a fetus that fails to reach his/her genetically predetermined optimal growth potential and constitutes a major clinical and public health problem, mainly in the developing world. Traditionally, an estimated foetal weight (EFW) or abdominal circumference (AC) below the 10th percentile raises concerns over suboptimal intrauterine growth, however the distinction between normal and pathologic growth often cannot reliably be made at this arbitrary cut-off. In addition, approximately 70% of foetuses below the 10th percentile will have a normal perinatal outcome.[1] The risk of adverse outcome is proportional to the degree of growth restriction with those below the 3rd percentile and/ or abnormal umbilical artery Doppler measurements at greatest risk of morbidity or mortality.[2] In addition, analysis of foetal growth trajectories has been identified as an important factor in the differentiation between physiological small-for-gestational age (SGA) and pathological intrauterine growth restriction.[3]

Fetal growth restriction is associated with increased perinatal mortality and morbidity due to higher risk of intrauterine fetal demise (IUFD), intrapartum morbidity, and increased rate of iatrogenic prematurity (before 34 weeks of gestation).[4,5] Besides the short-term

complications such as polycythemia, hypoglycemia, hypothermia, and respiratory difficulties due to induced prematurity, FGR newborns have a higher risk of long-term complications such as developmental delay, and behavioral dysfunctions.[6,7] Moreover, an increasing number of reports suggests the causative link between FGR and metabolic syndrome in adulthood.[8]

Historically, foetal growth restriction has been categorized as symmetric or asymmetric. Symmetric foetal growth restriction refers to foetuses with equally poor growth velocity of the head, the abdomen and the long bones. Asymmetric foetal growth restriction refers to infants whose head and long bones are spared compared with their abdomen and viscera.[9] The clinical observations made by Dashe et al found that among small-for-gestational age foetuses with no anatomic abnormalities, only those that were asymmetrically small were associated with increased pregnancy-induced maternal hypertension before 32 weeks and caesarean delivery for abnormal heart rate patterns compared with those of foetuses average for gestational age.[10] Additionally, asymmetric small-for-gestational age foetuses sustained more adverse neonatal composite outcomes compared to symmetric small-for-gestational age or average for gestational age foetuses.

AETIOLOGY

It is important to identify etiological factors in FGR because this directs the managing physician to an early diagnosis. Conditions that result in foetal growth restriction broadly consist of maternal, foetal, placental, and environmental disorders**.**

Maternal factors

Women at extremes of reproductive age (<16 years, >40 years), are at increased risk for FGR.[11] Social factors, such as ethnicity, poverty status, age at menarche, maternal height, and net maternal weight gain have an independent effect on birth weight in adolescent mothers.[12] Maternal weight at birth, low pre-pregnancy weight, and poor weight gain during pregnancy are positively associated with increase in FGR.[13,14] Women with lower socioeconomic status commonly have poor nutritional status, maternal anaemia, and poor prenatal care and substance abuse problem, which affect foetal growth. Maternal heavy cigarette smoking and excess amount of alcohol consumption throughout the pregnancy, especially in third trimester, is associated with FGR.[15-18] The use of illicit drugs like heroin and cocaine and exposure to various medications, such as warfarin, anticonvulsants, antineoplastic agents can result in FGR.[19-21]

Maternal disease and systemic conditions such as reduced uteroplacental blood flow, reduced oxygen-carrying capacity, or decreased nutrition to the fetus, chronic hypertension, preeclampsia, pregestational diabetes, chronic renal insufficiency, systemic lupus erythematous (SLE), antiphospholipid syndrome, renal diseases, autoimmune diseases and acquired thrombophilia, chronic maternal hypoxemia due to pulmonary disease, cardiac disease have been associated with FGR.[22-25] maternal malnutrition, protein deficiency and gastrointestinal conditions can cause lower birth weight because of decreased nutrition to the foetus. Other maternal causes include uterine factor, maternal periodontal disease, and genetic conditions, such as angiotensinogen gene mutations.[26-28]

Foetal factors

Fetal causes are less common in FGR and include genetic causes, congenital malformations, foetal infection, or other causes, including multiple pregnancies. Genetic causes include various abnormalities such as chromosomal abnormalities, autosomal abnormalities, ring chromosome structural alterations and single gene disorders. Congenital malformations, including congenital heart disease, diaphragmatic hernia, abdominal wall defects, renal agenesis or dysplasia, anencephaly, and single umbilical artery, are associated with FGR.[29-32] Common infections accounts for FGR foetuses are viral (rubella, cmv, herpes, varicella, herpes zoster, HIV) parasitic infections (toxoplasmosis, syphilis, malaria) and bacterial infections (chlamydia, mycoplasma, listeria, and tuberculosis).[33,34] Other viral, parasitic, and bacterial infections are associated with direct cell damage, or transplacental passage causing foetal infection, or placental vascular insufficiency. Multiple pregnancies and the risk of fetal growth restriction depend on a variety of factors such as chorionicity, number of fetuses, presence of congenital anomaly, umbilical cord abnormalities, unequal placenta, presence of twin transfusion syndrome, conjoint twin, and acardia.[35-37]

Placental factors

Normal placental development and functional integrity are essential for normal fetal growth. There is extensive evidence demonstrating that placental insufficiency accounts for FGR.[38,39] The relative decrease in placental mass and function and immunological disturbances at the maternal-fetal interface can result in the development of FGR.[40] placental causes of FGR include placental abruption, placenta accreta, placental infarction, placental villous obliteration, circumvallate placenta, placental hemangioma, placenta previa, placental mosaicism, single umbilical artery, and velamentous cord insertion.[41,42]

Environmental factors

Environmental risk factors for FGR may include exposure to radiation, chemicals and use of drugs, such as the anticonvulsants (phenytoin and trimethadione), warfarin, and heroin etc. Environmental risk factors such

as high altitude areas which are exposed to chronic hypoxia cause FGR.[43,44]

Screening and diagnosis

Effective screening for intrauterine growth restriction requires patient's history, physical examination, and general laboratory tests during routine antenatal care. Accurate dating is a prerequisite for pregnancy care and the tracking of foetal growth. This should be established from a careful history and correlated with the results of early ultrasound examinations in the first or second trimester. Foetal growth is estimated during routine antenatal care using symphysis–fundal height (SFH) measurement followed by the foetal ultrasound examination (foetal biometry).[45-47] Measurements of foetal head circumference (HC), abdominal circumference (AC) and femur length (FL) allow for the comparison of individual growth and local reference values or customized growth charts.[48] Beside the comparison with standard growth curves and an auxologic assessment of head to abdomen proportion, these measurements allow the calculation of estimated foetal weight (EFW).[49,50] The validity of both symphysis-fundal height (SFH) determined by clinical examination and estimated foetal weight (EFW) determined by ultrasound to identify FGR foetuses may be improved though customized standards that are designed to identify foetuses that measure < 10th percentile of their expected genetic growth potential.[51,52] This approach adjusts the growth curve percentiles for anthropomorphic variables such as maternal height, weight, parity, and foetal sex.[53] evidence from controlled, non-randomized trials supports this approach.[54,55] in addition, customized growth percentiles are better correlated with adverse pregnancy events and reduce the frequency of additional testing because of false-positive screening information.[56-58] ultrasound assessment, using uterine artery doppler and placental morphology, is of value to distinguish a subset of pregnancies at high risk of early severe FGR.[59-61] In women with risk factors for intrauterine growth restriction, uterine artery doppler screening at 19 to 23 weeks may identify pregnancies at risk of antepartum stillbirth and preterm delivery due to intrauterine growth restriction and placental disease.[62] In pregnancies in which intrauterine growth restriction due to uteroplacental vascular insufficiency is diagnosed, maternal surveillance for the development of severe preeclampsia with adverse features is warranted.[63,64] Evaluation of placental function by umbilical artery doppler is a clinical standard to distinguish between SGA and FGR.[65,66]

MANAGEMENT

When intrauterine growth restriction is diagnosed, surveillance should be initiated. Serial ultrasound estimation of foetal weight (every 2 weeks), along with umbilical artery doppler studies should be initiated. If available, a placental assessment and other doppler studies such as middle cerebral artery, umbilical vein, and ductus venosus can be performed. Increased frequency of surveillance may be required.[67] if foetal growth starts to plateau, amniotic fluid index starts to decline, or foetal tone or gross movements are diminished or absent, then more intensive surveillance (e.g., 2 to 3 times per week) or admission to hospital and delivery planning is required. Abnormal umbilical cord doppler (e.g., absent or reversed end-diastolic flow) in the presence of intrauterine growth restriction is an ominous finding that requires intervention and possible delivery.

If delivery was not indicated prior to 37 weeks in a patient diagnosed with intrauterine growth restriction, expectant management with close foetal and maternal surveillance versus delivery should be discussed after 37 weeks.[68] If growth continues along growth curve: conduct weekly biophysical profile and umbilical artery doppler; add growth every 2 weeks; consider delivery near term (38 to 40 weeks) if no other issues.

If growth plateaus or stops <34 weeks: administer corticosteroids; increase surveillance to 2 to 3 times per week; consider hospitalization; consider maternal-foetal medicine consultation; consider neonatal consultation. FGR foetus < 34weeks with abnormal umbilical artery doppler studies, consider MCA and dv studies. Delivery is recommended when umbilical artery, MCA, and dv doppler studies becomes abnormal and NST is abnormal. NST can be used selectively if the BPP is abnormal. If abnormal doppler studies (e.g., absent or reversed end-diastolic flow) and normal BPP and NST: continue intensive monitoring with BPP and umbilical dopplers 2 to 3 times per week; deliver if BPP or umbilical dopplers worsen or if MCA/dv are abnormal. If the period of gestation is >34 weeks and the AFV and DVP, BPP, and doppler studies are normal: conduct weekly surveillance and discuss delivery or on-going monitoring after 37 weeks. If abnormal fluid (AFV <5 cm or DVP <2 cm), BPP, and/or doppler studies: consider delivery.[69]

CONCLUSIONS

Management of FGR remains one of the main challenges for the obstetricians, both for the complexities of management of severe early FGR and for the diagnostic difficulties in late and term FGR. Ultrasound biometry examination is crucial for an accurate diagnosis of FGR. Pregnancies at risk of FGR should be considered for longitudinal ultrasound monitoring beyond the routine ultrasound screening at 20 weeks of gestation. Functional assessment of placental and foetal circulation by Doppler velocimetry and blood flow volume, together with computerized assessment of foetal heart rate variability, are key examinations in early and late FGR to assess severity of the disease and monitor foetal wellbeing. Appropriate timing of delivery in early FGR might change the outcome, and appropriate monitoring in late and term FGR might avoid unnecessary interventions. Following delivery, pathological

examination of the placenta may provide key insights into the underlying cause. Maternal thrombophilia testing and a review of the results of both the placental pathology and pertinent neonatal investigations may refine the presumptive cause of FGR that could alter the management plan in a subsequent pregnancy. Further research is needed to illuminate the preventive strategies and treatment strategies to assist the growth-restricted foetus.

Abbreviations

AC: Abdominal circumference
EFW: Estimated foetal weight
FGR: Foetal growth restriction
FL: Femur length
HC: Head circumference
IUFD: Intrauterine foetal demise
SFH: Symphysis fundal height
SGA: Small-for-gestational age
SLE: Systemic lupus erythematous

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