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Review

Difficulties in the Diagnosis and Management of Fetal Growth Restriction

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Abstract

Fetal growth restriction (FGR) is an important topic in perinatal medicine that results from impaired placental function and has poor perinatal outcomes. However, there is a lack of consensus on diagnostic criteria and management strategies worldwide. While some clinicians consider only fetal size in the diagnosis, others disagree and recommend adding fetal growth velocity Doppler indices to the diagnostic criteria. Different strategies are followed for pregnancy follow-up and delivery decision-making. There are different opinions on which Doppler measurements of vessels such as the umbilical artery, middle cerebral artery, and ductus venosus should be performed during follow-up. To assess fetal well-being and to decide on delivery, methods such as cardiotocography, computed cardiotocography, biophysical profile scoring, and biophysical profile scoring are used or not used. The Society of Maternal-Fetal Medicine (SFMF) and the International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) published guidelines in the same year. However, their perspectives on fetal growth restriction were quite different. We basically analyzed the differences between these two guidelines and the reasons for the differences. As a result, we presented our own practice.

Keywords: Fetal growth restriction (FGR), Fetal size, Maternal-Fetal Medicine, ISUOG.

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Fetal growth restriction (FGR) is one of the leading causes of perinatal morbidity and mortality and is also associated with adverse neurodevelopmental outcomes.[1-3] Prematurity, which has the effect of increasing adverse obstetric outcomes, is common.[4] FGR is also associated with an increased risk of adult diseases such as hypertension, metabolic syndrome, insulin resistance, Type-2 diabetes, coronary heart disease, and stroke.[5] Because it is such an important issue, the Maternal-Fetal Medicine Society (SMFM) [6] and the International Society of Ultrasound in Obstetrics and Gynecology (ISUOG)[7] published clinical guidelines on the management of FGR in 2020. However, these two guidelines have deep disagreements regarding diagnosis, evaluation, monitoring, and timing of delivery.

FGR in antenatal care is a challenging issue for clinicians based on a multifactorial etiology. It is mainly due to maternal, fetal, or placental causes. The most common cause, and whatever the cause, the ultimate underlying mechanism is impaired placental development and function, resulting in reduced nutrient and oxygen supply.[8] Thus, fetal weight remains small for gestational age and fetal growth rate slows down. Estimated fetal weight (EFW) is defined by measurements of biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), and femur length (FL), most often with the help of the Hadlock formula.[9] EFW <10th percentile has most frequently been used to define a small for gestational age (SGA) fetus.[7] Perinatal morbidity and mortality rates increased in fetuses with EFW<10th percentile. AC<10. percentile is also used to estimate the

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fetal size and associated poor fetal outcomes.[6, 7] However, the vast majority of these infants are constitutionally small and do not have adverse perinatal outcomes.[6, 7] In addition, Doppler anomalies in Umbilical Artery (UA), Uterine Artery (UtA), Ductus Venosus (DV), and/or Middle Cerebral Artery (MCA) occur as a result of decreased uteroplacental perfusion and increased fetal systemic vascular resistance. In follow-up, tests such as cardiotocography (CTG), computerized cardiotocography (cCTG), or biophysical profile (BPP) that evaluate fetal well-being are also used. Utilizing these tools within a system in the management of FGR improves perinatal and neurodevelopmental outcomes.[10-13] Still, there is no worldwide consensus on which to use and how often.

FGR is divided into two main classes as early-onset (<32 weeks) and late-onset (≥32 weeks) according to the time of diagnosis. The reason for this classification is based on the differences in severity, natural history, Doppler findings, association with hypertensive complications, placental findings, and treatment between these two FGR phenotypes.[14, 15] Early FGR is often associated with preeclampsia, abnormal umbilical artery Doppler findings, and poor perinatal outcomes. In late FGR, however, there is milder placental dysfunction and consequently less likely to be associated with preeclampsia and changes in umbilical artery Doppler. A consensus statement based on the Delphi procedure[16] proposed separate definitions for early-onset and late-onset FGR. Both guidelines classify FGR as early and late FGR. However, in diagnostic criteria and follow-up tools, SFMF[6] takes a more traditional approach, while ISUOG[7] takes an approach that considers the Delphi procedure in diagnostic criteria and the time of occurrence of FGR in follow-up. In this review, we will evaluate the perspectives of the SFMF and ISUOG guidelines to FGR and describe our own practice.

Etiology and Risk Factors

FGR is caused by maternal, fetal, or placental causes. However, in at least 40% of FGR cases, the etiologic cause may not be identified. In infants with an underlying cause, FGR results from genetic disease in about one-third of infants and the remainder due to poor fetal environment, which is the result of placental and maternal causes.[17] Maternal causes are also considered risk factors and regardless of the cause, the underlying mechanism is placental insufficiency and dysfunction.[8]

Maternal Factors

The risk of FGR is increased in individuals with diseases such as cardiovascular diseases, preeclampsia, and gestational diabetes. The risk also increases in the presence of factors such as anemia, autoimmune diseases, drug use (antiepileptics, chemotherapy drugs, etc. ....), tobacco and alcohol use.[18, 19]

Fetal Factors

Fetal structural and chromosomal abnormalities are present in 20% of cases, especially in early-onset FGR (<32 GW). Chromosomal anomalies include trisomies (most commonly trisomy 13 and trisomy 18), triploidy (in the diandric type, the extra chromosome comes from the father, and partial mole and symmetric FGR are observed. In the digynic type, the extra chromosome comes from the mother and asymmetric FGR is observed). The presence of genetic syndromes and congenital infections can be considered among other causes.[20, 21]

Placental Factors

The most common placental cause that increases the risk of FGR is placental vascular insufficiency. Also, umbilical cord abnormalities and advanced placental maturation increase the risk.[21, 22]

Diagnosis and Management

FGR is defined as infants who fail to reach their predetermined genetic growth potential. The aim of prenatal care is to prevent FGR-related perinatal morbidity and mortality, poor neurodevelopmental outcomes, and long-term adult diseases.[3, 5, 6, 7] Ultrasonographic fetal weight measurement and Doppler examinations (UA, UtA, MCA, DV) are used to detect infants who do not reach their growth potential. In addition, tests including CTG, cCTG, amniotic fluid index (AFI), and biophysical scoring can be performed to determine fetal well-being and timing of delivery.

Ultrasonographic Fetal Weight Measurement

First, the gestational age should be confirmed. The gestational week is confirmed by comparing the gestational week according to the menstrual history, preferably with the gestational week obtained from the crown-rump length (CRL) measurement in the first-trimester ultrasonography. If there is a difference of more than 7 days between the two measurements, the week of gestation is based on the CRL measurement.[23]

On ultrasonography, estimated fetal weight (EFW) is calculated by measuring BPD, HC, AC, and FL, and then the measurement is standardized using different fetal growth nomograms.[24-26] The most commonly used method is based on the Hadlock formula, which includes standards that are not adjusted for race and gender.[9] We use the Hadlock formula in our clinic, too.

According to the SFMF guidelines, FGR is an estimated fetal weight (EFW) or abdominal circumference (AC) below the 10th percentile.[6] ACOG also accepts this definition and this

definition is widely used by clinicians in the USA.[19] Fetuses with a birth weight below the 10th percentile have an increased risk of stillbirth and perinatal mortality. Perinatal outcomes are poorer in fetuses with a birth weight below the 3rd percentile.[27, 28] Therefore, worldwide, fetuses with EFW<3rd percentile are considered FGR. It was also found that the diagnostic accuracy of fetuses with AC<10th percentile was similar to that of fetuses with EFW<10th percentile.[29] However, 18%-22% of fetuses considered FGR because EFW<10th percentile or AC<10th percentile are constitutionally small at birth with normal perinatal outcomes. Conversely, there are infants who are not considered FGR by this definition, but who do not reach their true growth potential and consequently have poor perinatal outcomes. In one study, the lowest perinatal mortality was found in infants with birth weight between the 70th and 90th percentile and there was an inverse relationship between birth weight below the 80th percentile and perinatal mortality.[27]

Fetal Growth Rate

There are several methods for monitoring fetal growth rate: longitudinal growth charts[29] and customized growth charts,[26] which assess deviation from growth rate charts. There are studies suggesting that slowing of fetal growth rate in the third trimester is associated with poor perinatal outcomes. Decreased growth velocity is normally taken as a drop of more than 50 percent for AC or, more commonly, EFW between consecutive ultrasound scans.[30]

Doppler Velocity Measurements

The rationale for Doppler velocity measurements in FGR: It shows uteroplacental function on the maternal side. In uteroplacental insufficiency, there is maladaptation of the spiral arteries and abnormality of the villous vascular tree. The uterine artery (UTA) and umbilical artery (UA) are evaluated by Doppler velocity measurements.[7] On the fetal side, the progression of cardiovascular adaptation from hypoxia to acidemia is demonstrated. middle cerebral artery (MCA) and ductus venosus (DV) are evaluated with Doppler.

Uterine artery pulsatility index (PI) >95th percentile is associated with placental insufficiency and impaired maternal vascular perfusion.[31]

A progressive increase in UA PI indicates impaired gas and nutrient exchange at the placental surface and increased fetal afterload resistance. Loss of UA end-diastolic flow (UA-EDF) and, most recently, UA reverse end-diastolic flow (UA-REDF) are signs of increasing severity of residual placental insufficiency. UA Doppler has a key role in the monitoring of FGR and the timing of delivery.[6, 7]

Absent or inverted a-wave in the ductus venosus (DV) indicates increased intraatrial pressure and myocardial ischemia due to increased placental vascular resistance. It is an important tool in terms of timing of delivery, especially in the management of early-onset FGR.[10, 11] Because in early FGR, the clinician is in a decision process between the risk of stillbirth and premature FGR delivery.

ISUOG[7] recommends the international Delphi consensus decisions[16] for the diagnostic criteria of FGR. Accordingly, the diagnostic criteria for FGR are different in early-onset (before 32 weeks of gestation) and late-onset (after 32 weeks of gestation) FGR, provided that congenital anomalies are absent:

Delphi diagnostic criteria in early-onset FGR:

- AC/EFW <3rd percentile or UA-AEDF alone is a diagnostic criterion alone or
- AC/EFW between the 3rd and 10th percentile: accompanied by UtA-PI>95th percentile and/or UA-PI>95th percentile.
- Delphi diagnostic criteria in late-onset FGR:
- AC/EFW<3rd percentile is the diagnostic criterion alone or
- Two of the following 3 findings: AC/EFW between the 3rd and 10th percentile, slowing of the AC/EFW follow-up growth rate by more than 2 quartiles (50th percentile), CPR<5th percentile or UA PI>95th percentile.

FGR is a challenging subject of obstetrics that should be managed by maternal-fetal medicine specialists in adequately equipped centers.[6, 7] Detailed fetal ultrasonography should be performed at the time of diagnosis and amniocentesis and microarray examination should be recommended to the family, especially in early-onset FGR, in the presence of polyhydramnios or fetal structural anomalies.[7, 20, 22] Doppler velocity measurements and fetal well-being Should Be Evaluated And The Resulting Data Should Be Carefully Analyzed.

Management Strategies in Early-Onset FGR

It is less frequent (30%) than late-onset FGR. However, it represents more severe placental insufficiency. Therefore, the fetus may be very small, Doppler abnormalities are prominent, the perinatal mortality rate is high and it is associated with maternal comorbidities including hypertension and preeclampsia.[14-16] In early-onset FGR, increased resistance in UAA, UA, and MCA Doppler are early changes and occur weeks before severe cardiovascular and metabolic deterioration. With increasing severity, UA-AEDF and UA-REDF occur. The rate of transition from UA-AEDF to UA-REDF indicates the severity of placental insufficiency.[32] In early-onset FGR, late deterioration is the absence or inversion of the a-wave in DV. This cardiovascular and metabolic deterioration occurs before or simultaneously with STV abnormalities. This is followed by abnormal BPP

scoring, spontaneous recurrent decelerations on CTG, and finally stillbirth.[33] Especially in early-onset FGR, maternal factors such as exacerbation of maternal hypertension also influence the decision to deliver.[6, 7] The Trial of Randomized Umbilical and Fetal Flow in Europe (TRUFFLE) study[11] showed that multidisciplinary counseling (by neonatology and maternal-fetal medicine specialists), follow-up in a tertiary center under a specific protocol, and timing of delivery reduce poor perinatal outcomes.[10]

In the TRUFFLE study, the decision to deliver in early-onset FGR was based on late ductus venosus abnormalities and/or decreased short-time variability (STV) on cCTG or repetitive decelerations in fetal heart rate. ISUOG[7] adopts the recommendations of this study for the decision to deliver. However, cCTG is not a tool used worldwide. According to ISUOG, when cCTG is not available, if the biophysical profile score is 4 or less, delivery should be decided in echoing FGR. We also use biophysical profile scoring in our center. The SFMF,[6] on the other hand, does not recommend any of the tools of ductus venosus, cCTG, or biophysical profile scoring in early-onset FGR. In both early and late-onset FGR, CTG is used in addition to UA Doppler, which is used worldwide (also recommended by ISUOG). False positivity of CTG alone for fetal distress is common. This necessitates the need for additional methods in the decision-making process, especially when prematurity is taken into account. For these reasons, we use UA, DV Doppler, and biophysical profile scoring in early-onset FGR. Different intervals such as 1-2 weeks have been proposed as follow-up intervals.[6, 7] In the absence of Doppler abnormalities, we apply Doppler and biophysical profile scoring to FGR at 1-week intervals. As recommended by other guidelines,[6, 7] we measure fetal weight at 2–3-week intervals. In early-onset FGR, we hospitalize the patient in case of UA-AEDF or UA-REDF. Both ISUOG and SFMF[6, 7] recommend UA Doppler at 2-3 days intervals in this situation. Similarly, we hospitalize the patient and perform Doppler examination 2 days apart and CTG 3 times a day. If the CTG is nonreactive, we do biophysical profile scoring. We also administer betamethasone to the mother for lung maturation, similar to the recommendations of the other two guidelines,[6, 7] if preterm delivery is highly probable and has not been used previously. Apart from fetal indications, maternal obstetric emergencies such as placental abruption, severe pre-eclampsia, eclampsia, and HELLP syndrome are indications for emergency delivery even at the limit of viability (24-26 weeks of gestation).[6, 7] In addition, recurrent decelerations on CTG are indications of labor after 26 weeks of gestation.[6, 7] As recommended by ISUOG,[7] we recommend delivery when the biophysical score is 4 or less and this finding is recurrent. In early-onset FGR, our labor indication protocol according to gestational week is as follows:

- 26-30 weeks of gestation: Absence of a wave or inverted a-wave on DV,[7]
- 30-32 weeks of gestation: UA-REDF,[6, 7]
- 32-34 weeks of gestation: UA-AEDF. SFMF[6] and ISUOG[7] recommend delivery between
- 33-34 weeks of gestation in case of UA-AEDF.

In these Doppler anomalies, we recommend cesarean delivery as recommended by both guidelines.

Late-Onset FGR

It develops after 32 weeks of gestation. It is responsible for 70% of FGRs. The fetus may not be very small as placental insufficiency is milder. Maternal comorbidities such as hypertension and preeclampsia are less frequent. Doppler anomalies are less prominent. Increased resistance is usually not encountered in UA. However, the fetus is less resistant to hypoxia in the later weeks. Fetal pregnancy loss may occur in the UA without increased PI resistance. The decrease in CPR, which is the MCA PI/UA PI ratio, is due to the diversion of blood flow to the brain and is called the brain-protective effect. It is the main characteristic of late-onset FGR. The PORTO study[3] was a large prospective study evaluating the optimal management of FGR infants between 24 GW and 37 GW. They found that CPR and multivessel Doppler improved perinatal outcomes. According to their study, CPR had 66% sensitivity and 85% specificity in predicting poor perinatal outcomes and was not always in the same commonly accepted order of Doppler abnormalities. The ISUOG guideline[6] uses CPR in late-onset FGR and accepts the threshold value indicating abnormality as the 5th percentile. However, there is no adequate standardization in this regard, and in a large systematic review, the sensitivity and specificity of CPR in predicting poor perinatal outcomes were found to be lower compared to the PORTO study.[3, 34] Furthermore, it is not used as a criterion for the timing of delivery. For these reasons, the SFMF[6] does not use CPR. Randomized prospective studies are needed to ensure adequate standardization. However, in late-onset FGR, increased UA resistance is usually not observed and fetal death can occur within a few days even with a CTG reactive and/or biophysical profile score of 10/10. CTG and biophysical profile scoring do not inform the clinician about the frequency of follow-up. CPR should be used to get an idea of when the fetal condition may deteriorate. For these reasons, we use CPR in late-onset FGR. However, we use the CPR threshold value simply as CPR<1 instead of the 5th percentile. Although it is not an ideal threshold, we think it is useful in managing the disease. In late-onset FGR, if CPR<1, poor perinatal outcomes are imminent. Doppler and biophysical profile scoring at 2-3 day intervals should be considered. Hospitalization should be considered if the

patient cannot be adequately cooperated with or comes from a remote location. Between 34 and 36 weeks, if there is a high probability of late preterm labor, betamethasone administration for fetal lung maturity is controversial and recommended by SFMF[6] but not by ISUOG.[7] We administer betamethasone as a single dose in patients with a high probability of delivery within 1 week (maternal comorbidities such as pre-eclampsia, EFW <3 percentile, UA PI >95 percentile, CPR<1, biophysical profile score=6), unless it has been administered previously.

The stillbirth rate is less in late-onset FGR. However, stillbirth can occur without obvious signs and is observed more frequently than early-onset FGR (70%). Moreover, according to the TRUFFLE study, the frequency of neurodevelopmental disorders observed in early-onset FGR does not decrease in late-onset FGR.[35] For these reasons, we recommend follow-up at 1-week intervals as in FGR. At each follow-up, we check UA PI, MCA PI, and CPR, and also perform biophysical profile scoring. Our follow-up protocol is similar to the ISUOG guidelines.[7] However, cCTG is not available in our center. SFMF, on the other hand, recommends follow-up with UA Doppler and NST similar to early FGR and fetal weight measurement at 3-4 week intervals.[6]

The Intrauterine Growth Intervention Trial at Term (DIGITAT) study on the timing of delivery in near-term late-onset preterm FGR compared labor induction and expectant management in pregnancies with suspected FGR beyond 36 weeks of gestation. Here, pregnancies with UA-AEDF were excluded and perinatal outcomes were similar between the two groups. Only neonates in the induction group received more intermediate care. In those over 38 weeks of gestation, the results were similar. Therefore, as recommended by both SFMF[6] and ISUOG:[7]

- We recommend induction of labor for 38-39 gestational weeks pregnant women with EFW between 3-10 percentile and without significant Doppler abnormalities.

SFMF[6] recommends delivery at 37 weeks of gestation and ISUOG[7] recommends delivery between 36 and 37w6d for pregnant women with EFW<3 persantiles or UA PI>95 persantiles. Like SFMF, we also recommend induction of labor at 37 weeks gestation.

Conclusion

In conclusion, there is deep disagreement in the management of FGR. The main reason for this is that Doppler indices are not yet standardized. Prospective randomized studies on Doppler indices are needed to reduce stillbirths, poor perinatal outcomes, and poor neurodevelopmental outcomes. Furthermore, FGR should be followed up in tertiary centers with a multidisciplinary approach.

Disclosures

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References

1. Dudink I, Hüppi PS, Sizonenko SV, Castillo-Melendez M, Sutherland AE, Allison BJ, et al. Altered trajectory of neurodevelopment associated with fetal growth restriction. Experimental neurology 2022;347:113885.
2. Miller SL, Huppi PS, Mallard C. The consequences of fetal growth restriction on brain structure and neurodevelopmental outcome. The Journal of physiology 2016;594(4):807-23.
3. Unterscheider J, Daly S, Geary MP, Kennelly MM, McAuliffe FM, O'Donoghue K, et al. Optimizing the definition of intrauterine growth restriction: the multicenter prospective PORTO Study. American journal of obstetrics and gynecology 2013;208(4):290.e1-.e6.
4. Pallotto EK, Kilbride HW. Perinatal outcome and later implications of intrauterine growth restriction. Clinical obstetrics and gynecology 2006;49(2):257-69.
5. Crispi F, Miranda J, Gratacós E. Long-term cardiovascular consequences of fetal growth restriction: biology, clinical implications, and opportunities for prevention of adult disease. American journal of obstetrics and gynecology 2018;218(2):S869-S79.
6. Martins JG, Biggio JR, Abuhamad A, Medicine SfM-F. Society for Maternal-Fetal Medicine Consult Series# 52: diagnosis and management of fetal growth restriction:(replaces clinical guideline number 3, April 2012). American journal of obstetrics and gynecology 2020;223(4):B2-B17.
7. Lees C, Stampalija T, Baschat A, da Silva Costa F, Ferrazzi E, Figueras F, et al. ISUOG Practice Guidelines: diagnosis and management of small-for-gestational-age fetus and fetal growth restriction. Ultrasound in Obstetrics & Gynecology 2020;56(2):298-312.
8. Burton GJ, Jauniaux E. Pathophysiology of placental-derived fetal growth restriction. American journal of obstetrics and gynecology 2018;218(2):S745-S61.
9. Hadlock FP, Harrist RB, Martinez-Poyer J. In utero analysis of fetal growth: a sonographic weight standard. Radiology 1991;181(1):129-33.
10. Bilardo CM, Hecher K, Visser GH, Papageorghiou A, Marlow N, Thilaganathan B, et al. Severe fetal growth restriction at 26–32 weeks: key messages from the TRUFFLE study. Ultrasound in Obstetrics & Gynecology 2017;50(3):285-90.
11. Lees C, Marlow N, Arabin B, Bilardo CM, Brezinka C, Derks J, et al. Perinatal morbidity and mortality in early-onset fetal growth restriction: cohort outcomes of the trial of randomized umbilical and fetal flow in Europe (TRUFFLE). Ultrasound in obstetrics & gynecology 2013;42(4):400-8.
12. Stampalija T, Arabin B, Wolf H, Bilardo CM, Lees C, Brezinka C,

et al. Is middle cerebral artery Doppler related to neonatal and 2-year infant outcome in early fetal growth restriction? American journal of obstetrics and gynecology 2017;216(5):521. e1-. e13.
13. Stampalija T, Thornton J, Marlow N, Napolitano R, Bhide A, Pickles T, et al. Fetal cerebral Doppler changes and outcome in late preterm fetal growth restriction: prospective cohort study. Ultrasound in obstetrics & gynecology 2020;56(2):173-81.
14. Aviram A, Sherman C, Kingdom J, Zaltz A, Barrett J, Melamed N. Defining early vs late fetal growth restriction by placental pathology. Acta obstetricia et gynecologica Scandinavica 2019;98(3):365-73.
15. Figueras F, Gratacós E. Update on the diagnosis and classification of fetal growth restriction and proposal of a stage-based management protocol. Fetal diagnosis and therapy 2014;36(2):86-98.
16. Gordijn S, Beune I, Thilaganathan B, Papageorghiou A, Baschat A, Baker P, et al. Consensus definition of fetal growth restriction: a Delphi procedure. Ultrasound in Obstetrics & Gynecology 2016;48(3):333-9.
17. Wollmann HA. Intrauterine growth restriction: definition and etiology. Hormone Research in Paediatrics 1998;49(Suppl. 2):1-6.
18. Maulik D. Fetal growth restriction: the etiology. Clinical obstetrics and gynecology 2006;49(2):228-35.
19. Restriction FG. ACOG practice bulletin, number 227. Obstet Gynecol 2021;137(2):e16-e28.
20. Bamfo JE, Odibo AO. Diagnosis and management of fetal growth restriction. Journal of pregnancy 2011;2011.
21. Suhag A, Berghella V. Intrauterine growth restriction (IUGR): etiology and diagnosis. Current Obstetrics and Gynecology Reports 2013;2(2):102-11.
22. Hendrix N, Berghella V, editors. Non-placental causes of intrauterine growth restriction. Seminars in perinatology; 2008: Elsevier.
23. Gabbe S. Intrauterine growth restriction. Obstetrics: Normal and Abnormal Pregnancies 1996:923-44.
24. Committee Opinion No 700: Methods for Estimating the Due Date. Obstet Gynecol 2017;129(5):e150-e4.
25. Grantz KL, Hediger ML, Liu D, Louis GMB. Fetal growth standards: the NICHD fetal growth study approach in context with INTERGROWTH-21st and the World Health Organization Multicentre Growth Reference Study. American journal of obstetrics and gynecology 2018;218(2):S641-S55. e28.
26. Salomon L, Alfirevic Z, Da Silva Costa F, Deter R, Figueras F, Ghi Ta, et al. ISUOG Practice Guidelines: ultrasound assessment of fetal biometry and growth. Ultrasound in obstetrics & gynecology 2019;53(6):715-23.
27. Blue NR, Beddow ME, Savabi M, Katukuri VR, Chao CR. Comparing the Hadlock fetal growth standard to the Eunice Kennedy Shriver National Institute of Child Health and Human Development racial/ethnic standard for the prediction of neonatal morbidity and small for gestational age. American Journal of Obstetrics and Gynecology 2018;219(5):474. e1-. e12.
28. Monier I, Ego A, Benachi A, Ancel P-Y, Goffinet F, Zeitlin J. Comparison of the Hadlock and INTERGROWTH formulas for calculating estimated fetal weight in a preterm population in France. American journal of obstetrics and gynecology 2018;219(5):476. e1-. e12.
29. Moraitis AA, Wood AM, Fleming M, Smith GC. Birth weight percentile and the risk of term perinatal death. Obstetrics & Gynecology 2014;124(2 PART 1):274-83.
30. Levytska K, Higgins M, Keating S, Melamed N, Walker M, Sebire NJ. Placental pathology in relation to uterine artery Doppler findings in pregnancies with severe intrauterine growth restriction and abnormal umbilical artery Doppler changes. American journal of perinatology 2017;34(05):451-7.
31. Crimmins S, Desai A, Block-Abraham D, Berg C, Gembruch U, Baschat AA. A comparison of Doppler and biophysical findings between liveborn and stillborn growth-restricted fetuses. American journal of obstetrics and gynecology 2014;211(6):669. e1-. e10.
32. Raju TN, Mercer BM, Burchfield DJ, Joseph Jr GF. Periviable birth: executive summary of a joint workshop by the Eunice Kennedy Shriver National Institute of child health and human development, Society for Maternal-Fetal medicine, American Academy of pediatrics, and American College of obstetricians and Gynecologists. American journal of obstetrics and gynecology 2014;210(5):406-17.
33. Group E. Incidence of and risk factors for neonatal morbidity after active perinatal care: extremely preterm infants study in Sweden (EXPRESS). Acta paediatrica 2010;99(7):978-92.
34. Vollgraff Heidweiller-Schreurs C, De Boer MA, Heymans M, Schoonmade L, Bossuyt P, Mol B, et al. Prognostic accuracy of cerebroplacental ratio and middle cerebral artery Doppler for adverse perinatal outcome: systematic review and meta-analysis. Ultrasound in Obstetrics & Gynecology 2018;51(3):313-22.
35. Van Wassenaer-Leemhuis A, Marlow N, Lees C, Wolf H, investigators T. The association of neonatal morbidity with long-term neurological outcome in infants who were growth restricted and preterm at birth: secondary analyses from TRUFFLE (Trial of Randomized Umbilical and Fetal Flow in Europe). BJOG: An International Journal of Obstetrics & Gynaecology 2017;124(7):1072-8.