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New concepts in predicting, diagnosing and monitoring foetuses with intrauterine growth restriction

Nowe koncepcje w predykcji, diagnostyce oraz monitorowaniu płodów z wewnątrzmacicznym ograniczeniem wzrastania

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intrauterine growth restriction, constitutionally small infant, early-onset IUGR, late-onset IUGR

Słowa kluczowe

wewnątrzmaciczne zahamowanie wzrastania, konstytucjonalnie małe dziecko, wczesna hipotrofia, późna hipotrofia

S u m m a r y

A small-for-gestational-age (SGA) foetus is diagnosed when the estimated ultrasound weight is below a certain threshold; usually the 10th percentile. The first important step in the diagnostic process is to differentiate between intrauterine growth restriction due to dysfunction of the maternal-foetal unit and a constitutionally small, mostly healthy, foetus. Traditionally, the differentiation between these two clinical situations was based on the assessment of umbilical artery flow. These days we know that in order to distinguish between healthy, yet smaller foetuses, and small foetuses with an increased risk of intrauterine and perinatal complications, the ultrasound diagnostics should be extended by the cerebro-placental ratio and uterine artery flow evaluation. The next stage involves the differentiation between an early-onset IUGR occurring before 34 weeks gestation and a late-onset IUGR occurring after 34 weeks gestation. This gives rise to two phenotypically different clinical situations characterised by different course and prognostics. It also allows for the implementation of an appropriate management to minimise the risk of intrauterine foetal injury, death and iatrogenic prematurity. Based on contemporary literature, we present the most recent concept for the management in this type of pregnancy pathology.

S t r e s z c z e n i e

Mały płód rozpoznajemy wówczas, gdy szacowana ultrasonograficznie masa znajduje się poniżej pewnego progu, najczęściej jest to 10. percentyl. Pierwszym istotnym krokiem diagnostycznym jest zróżnicowanie między wewnątrzmacicznym ograniczeniem wzrastania wynikającym z nieprawidłowego funkcjonowania jednostki matczyno-płodowej a konstytucjonalnie mniejszym w większości przypadków zdrowym płodem. Tradycyjnie różnicowanie między tymi dwoma sytuacjami klinicznymi miało miejsce na podstawie oceny przepływu w tętnicy pępowinowej. Obecnie wiemy, że aby rozróżnić między płodami zdrowymi, ale mniejszymi, a tymi mniejszymi, które mają zwiększone ryzyko powikłań wewnątrzmacicznych i okołoporodowych, należy rozszerzyć spektrum diagnostyki dopplerowskiej o współczynnik mózgowo-łożyskowy oraz ocenę przepływów w tętnicach macicznych. Następnie rozróżniamy wczesną występującą przed 34. tygodniem postać IUGR oraz późną występującą po 34. tygodniu. Warunkuje to rozróżnienie między dwoma fenotypowo innymi postaciami, o odmiennym przebiegu i rokowaniu. Pozwala też na wdrożenie odpowiedniego postępowania, dzięki czemu minimalizuje się ryzyko uszkodzenia wewnątrzmacicznego płodu, zgonu oraz jatrogennego wcześniactwa. Na podstawie współczesnej literatury przedstawimy najbardziej współczesną koncepcję postępowania w tej patologii ciąży.

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INTRODUCTION

Intrauterine growth restriction is one of the main diagnostic and therapeutic challenges in perinatal medicine. One in five perinatal deaths, the same number of preterm labours before 34 weeks of gestation and 1/3 of morbidity cases among newborns are associated with the occurrence of this pathology (1, 2). SGA (Small for Gestational Age) and IUGR (Intrauterine Growth Restriction) are used interchangeably. However, it should be emphasised that their meaning is different. The term SGA was first used by neonatologists in 1967 to describe a newborn with a birth weight

below the 10th percentile (3). Later, this term was adopted by obstetricians to describe foetuses with small birth weight regardless of aetiology. IUGR, in turn, refers to foetuses with growth rate reduction, and thereby the birth weight is causatively associated with the placental pathology. So far, there have been many definitions describing abnormal foetal growth. According to ASOG, these are foetuses with bodyweight below 10th percentile, whereas the WHO defines in the same way foetuses under 3rd percentile (4). The discrepancies in the definition make it difficult to determine the criteria for diagnosis and classification. The research on the natural course of intrauterine growth restriction poses a number of challenges. Firstly, the diagnosis often takes place after delivery. This concerns up to 3/4 of children born with intrauterine growth restriction (5). In the so-called low-risk populations, the diagnosis rate is only 15% (6). Such a low detection rate of foetuses with low birth weight is also observed in populations in which a routine reference test in the third trimester of pregnancy is performed (7, 8). It was shown that most intrauterine deaths that could have been avoided result from the failure to diagnose SGA prior to delivery (9). Secondly, IUGR diagnosis most often leads to earlier pregnancy termination, particularly in severe cases, and the greater the child's maturity, the better the chance of extrauterine development. Most qualitative and quantitative evidence for the significance of IUGR diagnosis comes from retrospective analyses of post-delivery foetuses born alive or dead (10). The need to improve IUGR definition by implementing the concept of foetal growth potential is postulated. It is also important to differentiate between IUGR and SGA foetuses, whose lower birth weight is primarily due to constitutional factors and does not increase the risk of pathology in pregnancy, labour or the neonatal period. Selection of foetal subpopulations with pathologically restricted growth constitutes a challenge for modern perinatology. In recent years, two types of intrauterine foetal growth restriction have been distinguished – early and late (10). The first one occurs before the 34 weeks of pregnancy and is associated with difficulties in pregnancy monitoring and termination. The second type primarily constitutes a significant diagnostic problem. Innovating ultrasound techniques, with a view to a very detailed description of foetal anatomy, placental function and ultrasound assessment of uterine, placental and foetal blood flow, allow for a very detailed differentiation between small but healthy foetuses and the foetuses with restricted growth due to placental dysfunction.

AETIOLOGY

An utmost care should be taken in the adequate diagnosis and managing a pregnancy characterised by disturbed foetal growth. Since most SGA cases are diagnosed in the third trimester of pregnancy and are associated with good prognostics, a precise analysis of risk factors should be performed (tab. 1) (11).

Determining the probable aetiology may reduce perinatal complications, in particular intrauterine death of anatomically normal foetuses. It should be noted that advanced age of the mother, diseases in the pregnancy and methods of assisted reproduction, which are well-known risk factors, have become more common. Intrauterine growth restriction is an important factor for intrauterine foetal death. The relative risk is 4.0 (95% CI = 2.8-5.7) when the pathology is diagnosed prenatally, and twice as high – 8.0 (95% CI = 6.5-9.9) if it is undiagnosed (2). In large population studies evaluating intrauterine deaths earlier growth retardation (weight < 10th percentile) accounted for over 50% of deaths after the exclusion of congenital defects. As a result of the analysis, the number of unexplained intrauterine deaths decreased from 65-70% (Wigglesworth classification) to about 15% (12, 13).

Diagnostic management in determining SGA aetiology involves a detailed medical history, an evaluation of foetal anatomy, an assessment of markers for chromosomal aberrations, serological status of the patient and a Doppler analysis of maternal-foetal flows. This allows for an identification of the cause underlying the disturbed growth, differentiation between healthy and affected SGA infants and determination of IUGR. The exact diagnosis will influence adequate management aimed at minimising potential complications.

The potential risk of morbidity in foetuses with hypotrophy can be classified into two groups: early and late (tab. 2) (11).

Tab. 1. Risk factors of foetal hypotrophy

Maternal	Foetal	Placental
Gestational, pregestational arterial hypertension Vascular diseases Autoimmune diseases Diabetes Viral, parasitic, less common bacterial infections Hypoxia (lung diseases, cyanotic heart diseases, anaemia, staying at heights, haemoglobinopathies) Toxins, medications (warfarin, anticonvulsants, anticancer agents) Congenital disorders or uterine myomas Throbophilias Underweight, starving, diet Obesity Socio-economic factors Family factors Tobacco smoking, alcohol, other drugs Assisted reproduction Medical history of hypotrophy, former pregnancies complicated with hypotrophy	Multiple pregnancy TTTS Chromosomal disorders Congenital disorders	Placental infarctions Deep vein thrombosis Placental abruption Placental villous oedema Placenta previa Abnormal umbilical cord insertion Two-vessel umbilical cord

In general, it can be stated that smaller foetuses run a higher risk of worse perinatal outcome, though this group should be divided into pathologically and physiologically (constitutionally) small foetuses. The

Tab. 2. Morbidity and mortality related to foetal hypotrophy

Foetal	Neonatal	Late
Risk connected with induced labour and preterm birth Threatening intrauterine foetal asphyxia Iatrogenic prematurity Intrauterine foetal death	Increased risk of: MAS syndrome Respiratory failure Hypoglycaemia NEC Thrombocytopaenia Thermoregulation disorders Kidney failure Risk associated with prematurity Risk connected with chromosomal disorders and congenital disorders Death	Probably increased risk of: Short stature Cerebral palsy Retarded development Behavioural and emotional disorders Lower intelligence quotient Ischemic heart disease Arterial hypertension Diabetes Hypercholesterol-aemia Stroke

latter group will be defined as SGA (small for gestational age). SGA are foetuses with estimated body weight (EFW) below the 10th percentile. This group was selected from the population due to a significantly higher morbidity risk in term infants with body weight under the 10th percentile compared to the average population risk (3). Foetuses with intrauterine growth restriction (IUGR) have a higher risk of intrauterine complications, such as hypoxia or death as well as low Apgar score at birth (7). They will comprise, to some extent, a subgroup of SGA in which the genetically programmed growth process was restricted by one or more risk factors connected with abnormal processes in the formation of maternal-foetal circulation. However, these are two separate groups, which is confirmed by a higher perinatal morbidity risk as compared to SGA. Therefore, IUGR should always be excluded in a group of foetuses with EFG < 10th percentile. What should differentiate foetuses with restricted growth from constitutionally small ones is abnormal flow indicating centralised circulation that reflects adaptive foetal changes due to chronic malnutrition of varying degree will differentiate foetuses with restricted growth from constitutionally small foetuses. Macroscopic, histological and biochemical indicators of placental disease as well as an increased co-existence of preeclampsia will be observed in these pregnancies. For over 20 years, abnormal, elevated umbilical artery resistance was the main factor differentiating between these two forms. In recent years, it has been shown that although this was true for foetuses with severe placental pathology, in cases of moderate insufficiency this method does not allow for a diagnosis of a substantial part of early and virtually all late cases of hypotrophy (14-16).

Therefore, the division into early and late-onset IUGR seems justified. The widespread use of this division would be of a special value in attempts to compare the results from different centres. Clinically, this classification is important due to different methods for the monitoring and management in such pregnancies.

Early-onset IUGR accounts for approximately 20-30% of all pregnancies complicated by intrauterine growth restriction, while in almost 50% of cases it is associated with the co-existence of preeclampsia. In this form, the degree of placental failure is high and exceeds 30%. Moreover, in most cases it is characterised by an abnormal spectrum of flows in the umbilical artery (17). In these foetuses, a typical sequence of changes in Doppler flow spectra are observed: increased resistance in the umbilical artery, lower resistance in the middle cerebral artery, circulatory centralisation, gradual lowering of A-wave in the ductus venosus, disappearance of the diastolic wave in the umbilical cord, etc. These changes indicate foetal adaptation to chronic hypoxia and, in effect, may precede intrauterine death by weeks. The aim of the medical management is to balance between the risk of hypoxia-related complications and intrauterine death and the risk of complications and neonatal death as a result of prematurity.

Late-onset IUGR accounts for 70-80% of all pregnancies complicated by intrauterine growth restriction and, in contrast to the early form, it coexists with preeclampsia in only 10%of cases. The severity of placental insufficiency is moderate, i.e. less than 30%. In most cases, the umbilical artery flow spectrum is normal, however, a relationship with abnormally lowered values of the cerebroplacental ratio (CPR) was observed. The lower value is due to reduced resistance in the middle cerebral artery and is expressed by MCA PI < 5 percentile (pulsatility index of middle cerebral arterial Doppler assessment). In fact, these foetuses will never exhibit an abnormal spectrum of flows in the ductus venosus. Although the course of this form is milder, it involves the risk of severe perinatal complications. This is reflected in the data suggesting a higher risk of intrauterine death compared to a healthy population (12), as well as the risk of foetal asphyxia and neonatal acidemia (18). This is likely to result from a reduced foetal tolerance to hypoxia due to the occurrence of systolic activity or a sudden growth in placental insufficiency in a full term pregnancy. From the point of view of medical management, late-onset IUGR is no longer problematic after it is diagnosed. An increase in late-onset IUGR diagnostic rate, which is 15-25% in low-risk populations and does not exceed 50% in most of analyses, remains a challenge (5, 19). Characteristic features of these two forms of hypotrophy are presented in table 3 (20).

Tab. 3. Two types of hypotrophy – characteristic features

Early hypotrophy	Late hypotrophy
Early onset severe hypotrophy	Moderate hypotrophy
Less common form	More common form
< 34 Hbd	> 34 Hbd
Main problem: management	Main problem: diagnosis
Severe placental insufficiency (> 30% of the placenta)	Moderate placental insufficiency (< 30% of the placenta)
Severe hypoxia: foetal adaptation	Moderate hypoxia: lack of foetal adaptation
High mortality and morbidity	Low mortality, moderate morbidity, unknown scale of the problem

DIAGNOSTICS

A proper medical history on early gestational stage is fundamental for prediction as well as early diagnosis of foetal growth disorders and it should include chronic and family diseases, such as: systemic lupus erythematosus, antiphospholipid syndrome, factors predisposing to hypercoagulability, chronic hypertension and chronic kidney diseases. Women with a pregnancy complicated by intrauterine growth restriction run a 50%-risk of recurrence, especially if they developed severe hypotrophy accompanied by gestational hypertension and preeclampsia. In such cases, it seems justified to conduct series of ultrasound tests in the third trimester of pregnancy. A similar refers to a history of intrauterine foetal death. The risk increases as over half of deaths are related to the co-existence of IUGR. It is particularly the case of intrauterine deaths before the 32 weeks of pregnancy (21). Diabetes, especially pregestational or developed in the first trimester, is also a risk factor for both macrosomia and restricted growth. It often co-exists with preeclampsia. The risk increases up to 50% in concomitant nephropathy. Such patients should also undergo serial ultrasound tests to evaluate foetal growth. The NHS additionally recommends prophylactic administration of acetylsalicistic acid in this group of patients. Obesity is also a risk factor, particularly if the assessment of foetal weight is based on centile charts evaluating the growth potential. If this is the case, the risk increases by about 50%. This group includes the so-called relatively smaller foetuses (relative to the mother). Although a thorough medical history is crucial for identifying a high-risk group, only 10% of children in this group will develop IUGR (23).

Gestational age should be verified in suspected growth disorders. About 20% of women have irregular menstrual cycles (24). An assessment of the crown-rump length (CRL) before 12 weeks of pregnancy, optimally between 8 and 12 weeks of pregnancy, when the foetal boundaries are best seen, is the most optimal test for verifying gestational age. If the difference between the delivery term based on the date of the last period and the ultrasound-based gestational age is more than 7 days, the term based on the ultrasound assessment is considered as the ultimate one.

A precise assessment of the foetal anatomy with the evaluation of chromosomal aberration markers, the assessment of anatomy and ultrasonography symptoms of TORCH infections should be performed in SGA foetuses. This allows for differentiating between healthy and affected SGAs. A total of 25% of foetuses with congenital disorders have low birth weight. The majority of foetuses with congenital cytomegalovirus infection have a reduced amount of amniotic fluid, which should be accounted for in differential diagnostics. Every SGA foetus should undergo tests to exclude infection from the TORCH complex. A total of 15% of SGA patients are associated with a genetic syndrome (25). The risk is higher if anatomical abnormalities are detected.

Anatomical defects, diagnosed growth restriction below 24 weeks pregnancy, severe growth restriction (< 3rd percentile) in the early third trimester of pregnancy (week 24-28), particularly when the risk of aneuploidy is > 1/1000 in the first trimester of pregnancy, are an indication for invasive diagnostics (26).

Pregnant patients with diagnosed foetal growth restriction should be closely monitored for preeclampsia as well as receive regular measurements of blood pressure and the assessment of proteinuria. In early-onset IUGR under 34 weeks of pregnancy, this relationship is very strong and concerns over 50% of cases (27).

MONITORING

Umbilical artery

The assessment of the umbilical artery flow spectrum is the most significant element in the monitoring of foetuses with disturbed growth. The progression of changes, from elevated resistance, through the absent-end diastolic velocity (AEDV) and reversed-end diastolic velocity (REDV), directly correlates with the risk of foetal damage and intrauterine death. There is much evidence in the literature to indicate that the assessment of umbilical flows improves the prognostics in high-risk pregnancies, by a reduction in intrauterine deaths by 29% (14). AEDV and REDV occur up to a week prior to a significant deterioration in the foetal condition. In this case, the risk of acidemia and poor perinatal outcomes can be predicted with a sensitivity and specificity of up to 60%. Therefore, pregnancy continuation over 30 weeks is not unjustified in REDV (28, 29).

Middle cerebral artery

The assessment of the middle cerebral artery flow provides information on the level of vasodilation and is an ultrasound marker for hypoxia. The reduced resistance in the middle cerebral artery is a relatively late symptom. Although the specificity of this symptom is acceptable, the sensitivity is quite low. The diagnostic value of this marker can be improved by calculating the cerebroplacental ratio. A relationship between an abnormal middle cerebral artery pulsatility index (MCA-PI) and poor perinatal and neurological outcomes was shown. Still, the opinions whether the prognostics for such foetuses truly improve as a result of an earlier delivery are contradictory (20). This marker has a special application in the assessment of late-onset IUGR, as these foetuses usually exhibit normal flow in the umbilical cord, and the reduced resistance in MCA is the only Doppler abnormality. A 6-fold increase in the risk of urgent Caesarean section was shown in these foetuses due to the risk of intrauterine foetal asphyxia. Such infants also have poorer neurobehavioral results in the second year of age (30, 31).

Cerebroplacental ratio

The CPR (cerebroplacental ratio) is reduced in an increased resistance in the umbilical cord or decreased

resistance in the middle cerebral artery. When its value falls below 5th percentile, this means that the foetus exhibits circulatory centralisation, which reflects the adaptation to hypoxia. It has been shown that foetuses with normal weight, but abnormal CPR values also show neurobehavioral problems at 18 months of age (32). One in four foetuses with the late-onset growth resistance has abnormal CPR values, which is associated with an increased risk of complications during labour induction (33).

Uterine arteries

Abnormal trophoblast invasion is a typical phenomenon in severe early-onset IUGR and is connected with abnormally increased resistance in uterine arteries (34). It is postulated that the evaluation of uterine artery flows increases the value of the assessment of umbilical artery flows and middle cerebral artery flows in the risk assessment of negative perinatal outcomes. Although meta-analyses have shown that the assessment of uterine arteries has a limited value in IUGR and intrauterine death prediction, the measurement of resistance indexes in these vessels was included in some protocols for the management in growth disorders (20). The evaluation of uterine artery flows together with the assessment of biochemical markers is used to predict the risk of pre-eclampsia and IUGR in the first trimester of pregnancy. This method allows for implementing prophylaxis for such complications by administering 150 mg acetylsalicylic acid. This method is justified for the prediction of early, severe forms of IUGR (35).

Ductus venosus

The evaluation of the flow in the ductus venosus (DV) is the most powerful tool for predicting a short-term risk of intrauterine death in early-onset IUGR. This flow is abnormal only in advanced stages of foetal insufficiency (28, 29, 36). The lack of the A-wave or the increase in the resistance factor DV-PI over 95th percentile are associated with a high risk of intrauterine death of 40-100% for early-onset IUGR and are an indication for pregnancy termination due to its advancement after a completed steroid therapy (37). In 50% of cases, the abnormal spectrum in DV occurs before the loss of short-term variability in CTG computer records, and in 90% – before the changes in the biological profile occur, 48 to 72 hours earlier (38).

Isthmus aortae

Evaluation of the flow in the isthmus aortae and an identification of the reverse flow indicate an increased risk of intrauterine death and neurological complications. They occur about 7 days before the abnormalities in the ductus venosus (39).

CTG computer analysis

CTG computer analysis has a comparable sensitivity as the ductus venosus in predicting the deterioration in placental sufficiency, unlike conventional CTG, which generates 50% of false positive results.

Biophysical profile

Similar to conventional CTG assessment, the evaluation of biophysical profile also shows a high percentage of false positive results. A meta-analysis has shown the lack of benefits in the application of a biophysical profile in high-risk pregnancies (41).

Assessment of the volume of the amniotic fluid

The AFI assessment comprises an element of foetal biophysical profile evaluation. A meta-analysis of eighteen randomised studies showed that decreased AFI is associated with lower Apgar score in the 5th minute, however no relationship with acidosis or perinatal death in the case of SGA was observed (RR 1.6, 95% CI = 0.9-2.6) (42).

MANAGEMENT

The management will depend on the diagnosis. The below scheme of management summarises the scheme proposed by Eduardo Gratacos and Francesc Figueras research group, and comprises the most clear option available in the literature (20). The scheme will present the management in four clinical situations: healthy SGA infants, affected SGA infants, early-onset IUGR and late-onset IUGR.

Healthy SGA infants

Foetuses with excluded infectious and genetic causes exhibit a good prognosis. At a weight of 3rd-9th percentile and a normal spectrum of flows (UtA, UA, MCA, DV), the prognostics are favourable and do not necessitate the induction of delivery before 40 weeks of pregnancy. Such foetuses do not require flow or CTG monitoring more often than every 2 weeks. Induction by means of intracervical Folley catheter is recommended to prevent hyperstimulation.

Affected SGA infants

The management of foetuses with detected anatomical or genetic defect or TORCH infection will depend on the pathology underlying the cause of growth restriction. In the absence of other indications, a full term delivery is allowed.

Early-onset IUGR

STAGE I

In the first stage of early-onset IUGR, the foetus is small. At EFW < 3 percentile or EFW between 3-10 percentile, the foetus has an abnormal spectrum of flows in one of Doppler parameters (UtA, UA, MCA, CPR). In the absence of other abnormalities, the risk for the foetus is considered low prior to delivery date. Induced delivery is acceptable after 37 weeks of gestation, taking into account the risk of foetal asphyxia. Induction with the use of Folley catheter is recommended. Foetal monitoring should take place once a week.

STAGE II

At this stage, AECD or an early wave in the isthmus aortae occurs. The delivery should take place after

Tab. 4. Stage based system of monitoring

Stage	I	II	III	IV
Criteria	EFW < p3 or UtA PI > p95 or UtA PI > p95 or CPR < p5	UA-AEDV or reverse AoI	UA-REDV or DV PI > p95	DV (a rev) Declarations in CTG cCTG STV < 3 ms
Monitoring	1/week UA + MCA	2/week UA + MCA + DV	24-48 hrs UA + MCA + DV/cCTG	Every 12-24 hrs UA + MCA + DV/UV + cCTG
Pregnancy termination	Induced delivery	Caesarean section	Caesarean section	Caesarean section
Time of pregnancy termination	> 37 Hbd	34-37 Hbd	> 30 weeks	26-28 weeks

Tab. 5. Late hypotrophy

	Constitutional SGA	Late-onset hypotrophy	
Criteria	EFW > p3	1 abnormal parameter out of: EFW, UA, UV, UtA, BPP	MCA < p5
Monitoring	1/2 weeks (UtA) + UA + MCA	1/week (UtA) + UA + MCA	2/week (UtA) + UA + MCA + DV
Pregnancy termination	Spontaneous delivery or inducED delivery	InducED delivery	
Time of pregnancy termination	40-41	37-38	34-37

34 weeks of gestation. As the risk of a Caesarean section in the case of induced delivery exceeds 50%, it is recommended to perform an elective Caesarean section after once steroid therapy is completed. Monitoring should take place twice a week.

Stage III

At this stage, REDV occurs or DV-PI exceeds 95th percentile. Despite the risk of intrauterine death and neurological complications in the foetus, the risk is believed to be not as high to justify pregnancy termination before 30 weeks of gestation. The delivery should be performed by means of Caesarean section after completed steroid therapy. Monitoring should be performed by every 24-48 hours.

Stage IV

CTG readings show spontaneous decelerations, short-term variability drops below 3 ms or a reverse wave in the ductus venosus. While spontaneous decelerations may occur sporadically, their repetitiveness in subsequent readings may indicate the necessity to perform an urgent Caesarean section. The remaining two parameters indicate a significant risk of intrauterine death within 3-7 days and severe foetal neurological complications. Delivery by Caesarean section is preferred after 26 weeks of gestation. The complication-free survival rates exceed 50% after 26-28 weeks of pregnancy. If this gestational age cannot be reached, interdisciplinary parental consultation is recommended.

Late-onset IUGR

The management in late-onset IUGR is analogical to the first stage of early-onset IUGR. Foetuses diagnosed with growth restriction later than 34 weeks of gestation, with body weight < 3rd percentile and normal Doppler parameters, or between 3rd and 9th percentile and at least one abnormal parameter (UtA, UA, MCA, CPR) are qualified in this group. The foetuses are monitored once a week and born after 37 weeks of gestation, and in the case of MCA < 5th percentile – twice a week, accounting for the flow in DV. Induced delivery should take place between 34 and 37 weeks of gestation.

If preeclampsia symptoms occur at any IUGR stage, the pregnant patient should be qualified one stage higher and an adequate management in line with the guidelines should be implemented.

The management in pregnancies complicated by foetal hypotrophy is summarised in tables 4 and 5.

CONCLUSIONS

The lack of unified terminology, guidelines and recommendations is the main challenge in the prediction, diagnostics and management in foetuses with growth restriction. Therefore, study meta-analyses are very difficult. The implementation of local recommendations for the diagnosis and management in this type of pathologic pregnancy significantly improves perinatal outcomes, particularly in relation to the prevention of intrauterine deaths. In the case of early-onset IUGR, foetuses should remain under the care of a centre experienced in Doppler assessment and with a neonatology department able to meet the needs of premature infants. Here, the benefits and the risk arising from chronic hypoxia and prematurity are balanced. Late-onset IUGR primarily causes problems with diagnosis. Most of these pregnancies will be around the date of delivery at the time of diagnosis, therefore the management no longer poses a significant clinic problem. It is still unknown what the long-term implications for such infants are. This definitely requires further research.

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