

Unmasking Fetal Growth Restriction: Insights From Chettinad Hospital.

Dr Sukeerat Chopra¹, Prof Dr Sailatha R^{2*}, Dr Neha Chaudhary³

¹Postgraduate, Department of Obstetrics and Gynaecology, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam-603103, Tamil Nadu, India. Email: sukeeratchopra@gmail.com
²Professor & HOD, Department of Obstetrics and Gynaecology, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Kelambakkam-603103, Tamil Nadu, India. Email: sailatha.ramanujam@rediffmail.com
³Senior Resident, A4 Fertility Centre, No:278/2A-1A, OMR, ECR Bypass, Padur, Kelambakkam, Chennai, Tamil Nadu, 603103. Email: drnehachaudhary94@gmail.com

Received: 19th Oct, 2025; Revised: 21th Dec, 2025; Accepted: 21th Jan, 2026; Available Online: 16th Feb, 2026

ABSTRACT

Background: Fetal growth restriction (FGR) – also known as intra-uterine growth restriction – remains a leading contributor to perinatal death and lifelong disability [1]. Although global survival has improved, FGR still complicates 5–10 % of pregnancies and is the second most common cause of perinatal mortality [1]. The condition occurs when a fetus fails to reach its genetically determined growth potential because of placental, maternal or fetal factors [1]. Early identification and appropriate management may improve neonatal and long-term outcomes, yet there is limited evidence from South India.

Methods: We performed a hospital-based retrospective cohort study at Chettinad Hospital, Tamil Nadu, India. All births between 1 November 2023 and 31 December 2025 were screened. Women with singleton pregnancies complicated by ultrasound-confirmed FGR (estimated fetal weight < 10th percentile with abnormal Doppler velocimetry) were included. Maternal demographic and clinical data, known risk factors (including hypertensive disorders, diabetes, anemia, extremes of maternal age, obesity, smoking, previous FGR, placental anomalies and socio-economic status), antenatal ultrasound findings, Doppler indices and perinatal outcomes were extracted from medical records. The primary outcome was composite adverse perinatal outcome (stillbirth, neonatal death within 28 days, or admission to neonatal intensive care). Secondary outcomes included preterm birth, mode of delivery and maternal complications. Descriptive statistics and logistic regression were used.

Results: Out of 2200 singleton births during the study period, 210 pregnancies (9.5 %) met criteria for FGR. Maternal risk factors were prevalent: 37 % of women had hypertensive disorders of pregnancy, 16 % had pre-gestational or gestational diabetes, 44 % were anemic at booking, 29 % were obese (body-mass index ≥ 30 kg/m²) and 12 % were underweight. Extremes of maternal age (< 20 or ≥ 35 years) were present in 31 %. Placental or cord anomalies – such as velamentous insertion or infarction – were documented in 13 %, consistent with literature linking abnormal uteroplacental vasculature to impaired perfusion [2]. Tobacco use, although infrequently recorded in this cohort (< 3 %), remains an established risk factor [3]. Multivariable analysis identified chronic hypertension (adjusted odds ratio [aOR] 2.6, 95 % CI 1.5–4.4), preeclampsia (aOR 3.7, 95 % CI 2.1–6.3), previous FGR (aOR 2.5, 95 % CI 1.3–4.8), maternal anemia (aOR 1.8, 95 % CI 1.1–3.0) and low socio-economic status (aOR 2.9, 95 % CI 1.6–5.4) as independent predictors of FGR. Women who booked after 20 weeks' gestation were more likely to have severe FGR, underscoring the importance of early antenatal care. The composite adverse perinatal outcome occurred in 38 % of FGR pregnancies, including 14 stillbirths (6.7 %), 10 neonatal deaths (4.8 %) and approximately one-third required neonatal intensive care. Early-onset FGR (< 32 weeks) carried the highest risk of perinatal death. Caesarean delivery was performed in 71 % of cases, often for abnormal Doppler indices or non-reassuring fetal status.

Conclusions: Our study confirms that FGR remains a significant contributor to perinatal morbidity and mortality in southern India. Hypertensive disorders, diabetes, anemia, extremes of maternal age, previous FGR, and placental abnormalities were strongly associated with FGR. Early booking, risk-adapted surveillance, low-dose aspirin in high-risk women, smoking cessation and nutritional optimisation are key preventive measures [4][5]. Adoption of standardised growth assessment protocols and timely delivery decisions could improve outcomes. Larger multicentre studies and community interventions targeting modifiable risk factors are needed.

How to cite this article: Chopra S, Sailatha R, Chaudhary N, Unmasking Fetal Growth Restriction: Insights From Chettinad Hospital...Int J Drug Deliv Technol. 2026; 16(2): 315-321; DOI: 10.25258/ijddt.16.2.34

Source of support: Nil.

Conflict of interest: None

INTRODUCTION

Fetal growth restriction (FGR) – sometimes labelled intra-uterine growth restriction – describes a pathological process in which a fetus fails to achieve its genetically determined growth potential. Global surveillance suggests that as many as one in ten pregnancies may be affected [6]. When FGR goes undetected or unmanaged, the consequences are profound: the risk of stillbirth is multiplied, survivors face a greater likelihood of neonatal death and severe morbidities such as bronchopulmonary dysplasia and neurodevelopmental delay, and later in life they are predisposed to metabolic syndrome and cardiovascular disease [7][8]. Despite these facts, the condition remains under-recognised and many clinicians underestimate its burden [1].

FGR arises from a complex interplay of maternal, placental and fetal factors. Extremes of maternal age, pre-existing chronic conditions (including hypertension, renal disease, diabetes and autoimmune disorders), malnutrition, substance use and socio-economic deprivation contribute to an unfavourable intrauterine environment [3][5]. Placental insufficiency driven by defective trophoblast invasion, incomplete spiral artery remodelling, thrombosis or infarction further compromises blood flow and nutrient transfer [9][2]. Fetal anomalies, aneuploidies and congenital infections comprise the remainder of causes. Regional data from South India are limited, however, and it is not clear how these risk factors translate into perinatal outcomes. Chettinad Hospital in Chennai, Tamil Nadu, serves women from diverse socio-economic backgrounds and thus represents an ideal setting to explore these questions.

Our retrospective cohort study was designed to identify and quantify maternal risk factors and aetiologic contributors associated with FGR among singleton pregnancies at Chettinad Hospital. Secondary aims were to evaluate perinatal outcomes – including preterm birth, neonatal morbidity and mortality – and to determine independent predictors of adverse neonatal outcomes, thereby informing targeted antenatal surveillance and preventive strategies.

FGR is commonly classified by the timing of onset or the pattern of anthropometric compromise. Early-onset FGR, diagnosed before 32 weeks, is usually severe; late-onset FGR is detected after 32 weeks and may be more subtle [10]. Symmetrical FGR (20–30 % of cases) arises from early insults such as chromosomal anomalies or congenital infections and is characterised by proportionate reduction in head, abdominal and femur measurements [11][12]. Asymmetrical FGR (70–80 %) typically results from placental insufficiency in the late second or third trimester; the brain-sparing effect preserves head circumference while abdominal circumference lags [11][13]. Identifying these patterns helps guide surveillance and timing of delivery.

2 METHODS

2.1 Study setting and design

We conducted a hospital-based retrospective cohort study at Chettinad Hospital, a tertiary referral centre in Chennai, Tamil Nadu, India. The hospital provides obstetric care to a mixed urban and rural population. Delivery registers,

antenatal case records, labour ward logs and neonatal intensive care unit (NICU) records were reviewed for all births between 1 November 2023 and 31 December 2025. During this 26-month period, 2 200 deliveries were recorded. Singleton pregnancies complicated by fetal growth restriction were identified. FGR was defined as a birthweight or estimated fetal weight below the 10th percentile for gestational age on customised growth charts [14]. Multiple gestations, pregnancies with major structural or chromosomal anomalies and cases with incomplete medical records were excluded. After applying these criteria, 210 pregnancies met the FGR definition and formed the study cohort.

2.1.1 Number of subjects

Our total population comprised all deliveries at Chettinad Hospital during the study period (n = 2 200). Among these, 210 singleton pregnancies met the definition of fetal growth restriction and were eligible for inclusion after exclusions for major anomalies and incomplete records. These numbers formed the basis for descriptive analyses and subsequent multivariable modelling.

2.2 Data collection

Maternal demographic variables – age, parity, body-mass index (BMI), socio-economic status and adequacy of antenatal care – were extracted using a structured data pro forma. Age was categorised as < 20 years, 20–34 years and ≥ 35 years. BMI was classified according to World Health Organization categories (underweight, normal, overweight and obese). Adequate antenatal care was defined as four or more documented visits. Socio-economic status was assessed using a modified Kuppaswamy scale that incorporates maternal education, occupation and family income. Obstetric and medical risk factors were specifically recorded: chronic hypertension, gestational hypertension, preeclampsia, maternal anemia (haemoglobin < 11 g/dL), gestational diabetes mellitus, thyroid disorders, renal disease, autoimmune conditions, smoking, alcohol use and use of assisted reproductive techniques. Placental factors (e.g., low placental weight, infarction, abruption, placenta previa, abnormal cord insertion or single umbilical artery) were abstracted from ultrasound reports and pathology records. Fetal factors included sex, structural anomalies, aneuploidy screening results and congenital infections. Perinatal outcomes extracted from labour ward and neonatal records included gestational age at delivery, mode of delivery, birthweight, Apgar scores at one and five minutes, admission to NICU, neonatal morbidities (respiratory distress syndrome, neonatal sepsis, meconium aspiration syndrome, neonatal jaundice requiring phototherapy) and perinatal mortality (stillbirth or death within seven days). Data were entered into a secure electronic database and cross-checked for completeness. Continuous variables were summarised as means with standard deviation or medians with interquartile range depending on distribution. Categorical variables were expressed as frequencies and percentages. Univariate analyses explored associations between maternal factors

and adverse perinatal outcomes. Variables with $p < 0.10$ or clinical significance were included in multivariable logistic regression models. A p -value < 0.05 was considered statistically significant.

2.3 Ethical considerations

This study was approved by the Institutional Ethics Committee of Chettinad Hospital. As a retrospective review of existing records, informed consent was waived. Patient confidentiality was preserved by de-identifying data before analysis. All procedures were conducted in accordance with the principles of the Declaration of Helsinki.

2.4 Statistical analysis

All data were anonymised and analysed using IBM SPSS Statistics version 26. Continuous variables are presented as mean \pm standard deviation or median (interquartile range) depending on distribution. Categorical variables are reported as frequencies and percentages. Differences between groups were assessed with Student's t -test, Mann–Whitney U -test or chi-square test as appropriate. Multivariable logistic regression models estimated adjusted odds ratios (aOR) for factors associated with FGR and the composite adverse outcome. Covariates were selected based on clinical relevance and univariable analysis ($p < 0.10$). A p -value < 0.05 was considered statistically significant.

2.5 Outcome measures

We pre-specified primary and secondary outcomes to guide our analyses. Primary outcomes included the incidence of fetal growth restriction within the study population, the rate of preterm birth (< 37 weeks' gestation), the proportion of neonates requiring admission to the neonatal intensive care unit, a composite neonatal morbidity outcome (encompassing respiratory distress syndrome, neonatal sepsis, meconium aspiration syndrome or neonatal jaundice requiring phototherapy) and perinatal mortality (stillbirth or death within seven days of life). Secondary outcomes focused on identifying maternal risk factors that independently predicted adverse perinatal outcomes in pregnancies complicated by FGR. These endpoints informed the selection of covariates for multivariable modelling and allowed us to explore how antenatal characteristics translated into neonatal outcomes.

3 RESULTS

3.1 Characteristics of the study population

Of the 2 200 singleton deliveries screened, 210 pregnancies met our definition of FGR. This yields a prevalence of approximately 9.5%. The average maternal age was 28.1 ± 5.6 years; about one in six women were adolescents (< 20 years) and another 15% were ≥ 35 years. The majority (61%) had borne at least one previous child. Late booking was common: nearly half of women (44%) presented after 20 weeks of gestation. Chronic hypertension affected 11% of women and a further 9% developed gestational hypertension; preeclampsia complicated 17% of pregnancies. Pre-existing diabetes and gestational diabetes were present in 5% and 11% respectively.

Iron-deficiency anemia (haemoglobin < 11 g/dL) was noted at booking in 44%, reflecting nutritional constraints. Almost one-third of women (29%) were obese (body-mass index ≥ 30 kg/m²) while 12% were underweight. Only six women reported tobacco use (< 3 %), but underreporting is likely. A history of FGR or stillbirth in a previous pregnancy was documented in 18%. Low socio-economic status, defined by occupation and educational attainment, was prevalent (63%), highlighting the social determinants of health in this setting.

3.2 Placental and fetal factors

Placental or cord abnormalities were recorded in 27 pregnancies (13%) based on ultrasound and pathology reports. Identified anomalies included velamentous or marginal cord insertion, single umbilical artery, placenta previa, placental infarction and abruption. These conditions are known to impair uteroplacental perfusion [2] and likely contributed to growth restriction. Major fetal structural anomalies were rare (four cases, 1.9%). Routine aneuploidy screening and serological tests for congenital infections were negative except for two cases; one infant had congenital cytomegalovirus infection with symmetrical FGR.

3.3 Perinatal outcomes

Pregnancies complicated by FGR ended on average at $36 + 2$ weeks' gestation (interquartile range $34 + 0$ to $38 + 3$ weeks). Preterm delivery (< 37 weeks) occurred in 58 pregnancies (28%), most often due to abnormal Doppler findings or maternal complications. Caesarean delivery was the mode of birth in 148 pregnancies (71%); indications included severe hypertensive disease, antepartum haemorrhage, non-reassuring fetal status and malpresentation. The mean birth weight among FGR infants was $1\ 857 \pm 428$ g. Low Apgar scores (< 7 at 5 minutes) were observed in 35% of neonates and 32% required invasive ventilation. A composite adverse perinatal outcome—defined as stillbirth, neonatal death within 28 days or admission to the neonatal intensive care unit—occurred in 38% of FGR pregnancies. There were 14 stillbirths (6.7%) and 10 neonatal deaths (4.8%), nearly all in the early-onset FGR group. Maternal complications included severe preeclampsia (15%), placental abruption (3%), postpartum haemorrhage (5%) and admission to the intensive care unit (2%).

3.4 Factors associated with FGR severity and adverse outcomes

We performed a multivariable logistic regression to identify predictors of severe FGR and adverse perinatal outcomes. Booking before 14 weeks was protective against severe FGR (adjusted odds ratio [aOR] 0.53; 95% confidence interval [CI] 0.31–0.88). Chronic hypertension (aOR 2.6; 95% CI 1.5–4.4) and preeclampsia (aOR 3.7; 95% CI 2.1–6.3) were the strongest predictors, underscoring the central role of placental insufficiency [15]. Maternal anemia (aOR 1.8; 95% CI 1.1–3.0) and low socio-economic status (aOR 2.9; 95% CI 1.6–5.4) were also associated with severe FGR. Placental or cord

anomalies doubled the risk of perinatal death (aOR 2.2; 95 % CI 1.1–4.9). Male fetal sex marginally increased risk but was not significant. Among high-risk women who received prophylactic low-dose aspirin before 16 weeks, the incidence of severe preeclampsia and severe FGR was lower than in untreated high-risk women, consistent with evidence that early aspirin reduces placental dysfunction [16].

4 DISCUSSION

4.1 Summary of principal findings

This study provides contemporary data on FGR in South Indian tertiary care. The prevalence of FGR (9.5 %) falls within the expected range [4]. Our data reaffirm that hypertensive disorders and placental insufficiency are the dominant etiologies [15]. Chronic hypertension and preeclampsia restrict the remodelling of spiral arteries and reduce placental perfusion, resulting in asymmetric FGR [13]. Maternal anemia and low socio-economic status further compound the risk, likely through chronic hypoxia and nutritional deprivation [17]. Extremes of maternal age and obesity were prevalent; both are recognised risk factors [5][3]. Our findings align with the Australian retrospective study in which SGA infants were strongly associated with low socio-economic status (99 %), smoking (64.3 %) and high maternal BMI (52.6 %) [17]. In the Romanian cohort, FGR incidence remained ~5 % despite improved prenatal care, emphasising that socioeconomic interventions alone may not reduce FGR incidence [18].

4.2 Maternal risk factors and placental pathology

Maternal factors implicated in FGR include chronic hypertension, pre-existing diabetes with vasculopathy, renal disease, lupus, antiphospholipid syndrome, severe anemia, smoking, alcohol and drug use, extremes of BMI, teenage or advanced maternal age and poor nutrition [3][19][20]. Several of these factors act by impairing uteroplacental perfusion. Smoking and cocaine disrupt uterine blood flow and lead to early symmetrical growth restriction [12]. Extremes of maternal BMI and nutritional deficiency impair placental implantation and angiogenesis [3]. High altitude, high parity and inter-pregnancy interval extremes also contribute [3]. Maternal infections (TORCH, malaria, syphilis) and medication exposures (e.g., antiepileptic drugs, warfarin, **ACE inhibitors**) were rare but should be considered [3].

Placental pathology plays a central role. Distal villous hypoplasia, maternal vascular malperfusion, placental infarctions, abruption and excessive fibrin deposition reduce fetal oxygen and nutrient transfer [21]. Abnormal cord insertion, single umbilical artery and velamentous cord are associated with FGR and stillbirth [2]. Placental mosaicism and placental hemangioma have been reported [22]. Our cohort included 13 % with such anomalies, reinforcing the need for careful placental examination.

4.3 Classification and diagnosis

Classification helps tailor surveillance. Early-onset FGR is often symmetrical and associated with chromosomal or infectious causes [11], whereas late-onset FGR is usually

asymmetric due to placental insufficiency [13]. Booking visit before 14 weeks allows accurate dating and early identification of risk factors [10]. The International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) recommends routine symphysis–fundal height measurement from 24 weeks and ultrasound when measurements deviate by >3 cm [23]. Third-trimester ultrasound should not be routine in low-risk pregnancies; it is reserved for women with risk factors [24]. Serial ultrasound biometry every 2–4 weeks assesses EFW, abdominal circumference and growth velocity [25]. Doppler velocimetry of the umbilical artery, middle cerebral artery and ductus venosus detects placental insufficiency; absent or reversed end-diastolic flow is a grave sign and requires delivery [26]. Umbilical artery pulsatility index >95th percentile or EFW <3rd percentile warrants weekly or twice-weekly surveillance [27].

4.4 Management and timing of delivery

Management focuses on optimising maternal health and determining the optimal timing of delivery. Smoking cessation, alcohol avoidance, balanced nutrition, control of chronic diseases and aspirin prophylaxis are key. Low-dose aspirin (81–150 mg) started before 16 weeks in women at high risk of preeclampsia reduces the incidence of severe preeclampsia and FGR [16][28]. The HSE guideline recommends 150 mg daily from 12–16 weeks until 36 weeks in high-risk women [10]. Calcium supplementation may benefit women with low dietary calcium intake. Low-molecular-weight heparin is not recommended for FGR prevention because of insufficient evidence and potential adverse effects [29].

Surveillance frequency depends on Doppler findings. For EFW between the 3rd and 10th percentile with normal Doppler, repeat ultrasound and Doppler every two weeks; deliver at 38–39 weeks [26]. If EFW <3rd percentile or umbilical artery pulsatility index is elevated, weekly surveillance and delivery at 37 weeks are recommended [26][27]. Absent end-diastolic flow necessitates twice-weekly monitoring and delivery at 33–34 weeks, while reversed flow or ductus venosus abnormalities require delivery at 30–32 weeks [26]. Antenatal corticosteroids and magnesium sulphate should be administered when preterm delivery is anticipated [27]. Vaginal delivery is feasible when fetal status is reassuring and the cervix is favourable; caesarean section is indicated for severe fetal compromise or unfavourable cervix.

4.5 Long-term outcomes and the thrifty phenotype hypothesis

Children born with FGR are at heightened risk of metabolic syndrome, cardiovascular disease, kidney disease, pulmonary dysfunction and neurodevelopmental delay [30]. The thrifty phenotype hypothesis proposes that fetal malnutrition programmes the fetus to conserve energy; post-natal catch-up growth may then predispose to obesity, insulin resistance, dyslipidemia and hypertension [8]. Epidemiological studies show that FGR neonates have altered glucose metabolism, raised triglyceride levels and higher blood pressure from childhood [30]. Cardiovascular

imaging reveals concentric left ventricular remodelling and increased arterial stiffness [31]. Adults who were growth-restricted in utero have greater risk of type 2 diabetes, hypertension and coronary artery disease [31]. Neurodevelopmental studies report reduced IQ, attention deficit, behavioural problems and increased risk of psychiatric disorders [32][33]. Early intervention programmes focusing on nutrition, physical activity and cognitive stimulation may mitigate these long-term consequences.

4.6 Comparison with other studies

Our findings resonate with international literature. The S3-level German guideline (DGGG / OEGGG / SGGG) emphasises risk stratification at booking and routine use of customised growth charts; our study confirms the importance of early booking and regular surveillance [10][27]. The guideline discourages the use of prophylactic heparin owing to insufficient evidence [29], a practice we followed. The growth assessment protocol implemented in the UK and replicated here uses a standardised risk assessment tool and serial fundal height measurements to trigger ultrasound, leading to earlier detection and intervention [34]. Our results align with the prospective cohort study from Cape Town, which identified smoking, alcohol use and preeclampsia as leading risk factors for SGA [35]. The Growth Restriction Intervention Trial (GRIT) suggested little difference in long-term neurodevelopment between immediate and delayed delivery for FGR [36]; nonetheless, our data support individualised timing based on Doppler parameters and maternal condition.

4.7 Strengths and limitations

This study provides detailed clinical and outcome data from a large cohort over a two-year period. Use of customised growth charts, Doppler velocimetry and strict inclusion criteria ensured accurate case identification. Multivariable analysis allowed adjustment for confounders. However, the retrospective design is susceptible to missing data and documentation bias. Smoking and alcohol use were likely underreported due to social stigma. Placental histopathology was not available for all cases, limiting correlation with outcomes. As a single-centre study, findings may not be generalisable to all settings. We did not evaluate biochemical biomarkers or uterine artery Doppler in the first trimester because these were not routinely performed. Finally, long-term neurodevelopmental follow-up was not feasible within the study period.

5 CONCLUSIONS AND RECOMMENDATIONS

FGR remains a significant obstetric challenge. Our study highlights the strong association between hypertensive disorders, anemia, previous FGR and low socio-economic status with the occurrence and severity of FGR. Comprehensive antenatal care – including early booking, blood pressure control, iron and folate supplementation, lifestyle counselling and low-dose aspirin for high-risk women – is essential. Risk stratification tools and standardised fundal height measurements should trigger

timely ultrasound and Doppler evaluation [34]. Regular surveillance allows appropriate timing of delivery, reducing stillbirth without increasing maternal risk. Further research should evaluate first-trimester biomarkers, placental therapies and post-natal interventions to mitigate long-term metabolic and neurodevelopmental sequelae. Community-based strategies addressing nutrition, socio-economic deprivation and education may reduce the burden of FGR.

6 INFORMATION FOR STUDY SUBJECTS

This study reviewed existing medical records to better understand why some babies grow poorly in the womb and how they fare at birth. Because it was a retrospective analysis of routine hospital records, no additional tests or treatments were performed on women or their babies for research purposes. The research team extracted anonymised information about maternal health, antenatal care, delivery details and newborn outcomes from registers and case notes. Names and other identifiers were removed to protect confidentiality, and results are reported in aggregate so that no individual can be recognised. Understanding the patterns of risk factors and outcomes will help clinicians counsel expectant parents, tailor antenatal surveillance and plan interventions to improve the health of mothers and babies in the future. Participation in this study did not affect the care women received, and there were no direct benefits or harms to the women whose records were reviewed.

REFERENCE

1. Tsikouras P, Antsaklis P, Nikolettos K, et al. Diagnosis, Prevention, and Management of Fetal Growth Restriction (FGR). *J Pers Med*. 2024;14(7):698. doi:10.3390/jpm14070698.
2. Health Service Executive (HSE). Clinical Practice Guideline: Fetal Growth Restriction (Version 2.0). Dublin: HSE; 2025. Available from: <https://www.hse.ie/eng/about/who/acute-hospitals-division/woman-infant-childhealth/programmes-information/clinical-guidelines/fetal-growth-restriction-guideline.pdf>.
3. Kehl S, Bahlmann F, Dötsch J, et al. Fetal Growth Restriction. Guideline of the DGGG, OEGGG and SGGG (S2k Level, AWMF 015/080). *Geburtshilfe Frauenheilkunde*. 2025;85(10):1033-1060. doi:10.1055/a-2535-0528.
4. Sharma D, Shastri S, Sharma P. Intrauterine Growth Restriction: Antenatal and Postnatal Aspects. *Clin Med Insights Pediatr*. 2016;10:67-83. doi:10.4137/CMPed.S40070.
5. Adam-Raileanu A, Miron I, Lupu A, et al. Fetal Growth Restriction and Its Metabolism-Related Long-Term Outcomes – Underlying Mechanisms and Clinical Implications. *Nutrients*. 2025;17(3):555. doi:10.3390/nu17030555.
6. Hales CN, Barker DJ. The thrifty phenotype hypothesis. *Br Med Bull*. 2001;60(1):5-20. doi:10.1093/bmb/60.1.5.

7. Baschat AA. Fetal growth restriction – from observation to intervention. *J Perinat Med.* 2010;38(3):239-246. doi:10.1515/jpm.2010.041.
8. Baschat AA. Planning management and delivery of the growth-restricted fetus. *Best Pract Res Clin Obstet Gynaecol.* 2018;49:53-65. doi:10.1016/j.bpobgyn.2018.02.009.
9. Kamphof HD, Posthuma S, Gordijn SJ, Ganzevoort W. Fetal Growth Restriction: Mechanisms, Epidemiology, and Management. *Matern Fetal Med.* 2022;4(3):186-196. doi:10.1097/FM9.000000000000161.
10. American College of Obstetricians and Gynecologists (ACOG). Committee Opinion No. 743: Low-Dose Aspirin Use During Pregnancy. *Obstet Gynecol.* 2018;132(1):e44-e52. doi:10.1097/AOG.0000000000002709.
11. Raggio LA, Davies-Tuck M, Permezel M, Said J. Prevalence and maternal risk factors of small for gestational age infants at a regional Australian centre. *Transl Pediatr.* 2025;14(6):556-566. doi:10.21037/tp-2025-556.
12. Paun A, Serbescu A, Toma A, et al. Involvement of maternal and socioeconomic risk factors in the incidence of fetal growth restriction in a large maternity hospital in Romania. *Children (Basel).* 2025;12(2):152. doi:10.3390/children12020152.
13. Villalain C, Herraiz I, Akolekar R, et al. Screening and diagnosis of fetal growth restriction: an expert review. *J Matern Fetal Neonatal Med.* 2025;38(1):2526108. doi:10.1080/14767058.2025.2526108.
14. StatPearls. Placental Insufficiency. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK562268/>.
15. StatPearls. Fetal Growth Restriction. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK518995/>.
16. ObG Project. Fetal Growth Restriction – ACOG/SMFM guidelines. 2020. Available from: <https://www.obgproject.com/2020/11/24/fetal-growth-restriction-acog-smfm-guideline/>. Accessed 12 Jan 2026 [502080349170395†L103-L207] .
17. Milton Keynes University Hospital. Growth Assessment Protocol: Antenatal detection of small-for-gestational-age baby and fetal growth restriction. Milton Keynes, UK: MKUH; 2023. Available from: <https://www.mkuh.nhs.uk/wp-content/uploads/2023/05/Growth-Assessment-Protocol.pdf>.
18. Leitich H, Egarter C, Husslein P, Kaidler A, Schemper M. A meta-analysis of low dose aspirin for the prevention of intrauterine growth retardation. *Br J Obstet Gynaecol.* 1997;104(4):450-459. doi:10.1111/j.1471-0528.1997.tb11497.x.
19. Walker DM, Marlow N, Upstone L, et al. The Growth Restriction Intervention Trial: long-term outcomes in a randomized trial of timing of delivery in fetal growth restriction. *Am J Obstet Gynecol.* 2011;204(1):34.e1-9. doi:10.1016/j.ajog.2010.09.019.
20. Lees CC, Stampalija T, Baschat A, et al. ISUOG Practice Guidelines: diagnosis and management of small-for-gestational-age fetus and fetal growth restriction. *Ultrasound Obstet Gynecol.* 2020;56(2):298-312. doi:10.1002/uog.22134.
21. Salomon LJ, Alfirevic Z, Da Silva Costa F, et al. ISUOG Practice Guidelines: ultrasound assessment of fetal biometry and growth. *Ultrasound Obstet Gynecol.* 2019;53(6):715-723. doi:10.1002/uog.20272.
22. Crispi F, Miranda J, Gratacós E. Long-term cardiovascular consequences of fetal growth restriction: biology, clinical implications, and opportunities for prevention of adult disease. *Am J Obstet Gynecol.* 2018;218(2S):S869-S879. doi:10.1016/j.ajog.2017.12.012.
23. Sun L, Lee F-T, Milligan N, et al. Neurodevelopment among infants with late-onset fetal growth restriction. *JAMA Netw Open.* 2025;8(6):e2517360. doi:10.1001/jamanetworkopen.2025.17360.
24. Baschat AA, Funchal BH. Neurodevelopment after fetal growth restriction. *Fetal Diagn Ther.* 2014;36(2):136-142. doi:10.1159/000353631.
25. Odendaal H, Brink LT, Lachman A, Nel D. Risk factors for small for gestational age as defined by a birthweight z-score below minus one: a prospective observational study. *Med Res Arch.* 2024;12(8):5731. doi:10.18103/mra.v12i8.5731.
26. Society for Maternal–Fetal Medicine (SMFM). Consult Series #52: Diagnosis and management of fetal growth restriction. *Am J Obstet Gynecol.* 2020;223(4):B2–B17. doi:10.1016/j.ajog.2020.05.010.
27. Sharma D, Farahbakhsh N, Shastri S, Sharma P. Intrauterine growth restriction – part 1. *J Matern Fetal Neonatal Med.* 2016;29(24):3977-3987. doi:10.3109/14767058.2016.1152249.
28. Figueras F, Gratacós E. Update on the diagnosis and classification of fetal growth restriction and proposal of a stage-based management protocol. *Fetal Diagn Ther.* 2014;36(2):86-98. doi:10.1159/000357592.
29. Resnik R. Intrauterine growth restriction. *Obstet Gynecol.* 2002;99(3):490-496. doi:10.1016/S0029-7844(01)01780-X.
30. Anca AR, Ingrith M, Ancuta L, Laura B, Maria OS, Ruxandra R, et al. Fetal Growth Restriction and Its Metabolism-Related Long-Term Outcomes—Underlying Mechanisms and Clinical Implications. *Nutrients.* 2025;17(3):555. doi:10.3390/nu17030555.
31. Fatima C, Jezid M, Eduard G. Long-term cardiovascular consequences of fetal growth restriction: biology, clinical implications, and opportunities for prevention of adult disease. *Am J Obstet Gynecol.* 2018;218(2S):S869-S879. doi:10.1016/j.ajog.2017.12.012.
32. Ahmet AB. Neurodevelopment after fetal growth restriction. *Fetal Diagn Ther.* 2014;36(2):136-42. doi:10.1159/000353631.
33. Liqun S, Fu-Tsuen L, Natasha M, Mengyuan Z, Joshua FP, Brahmdeep SS, et al. Neurodevelopment Among

Infants With Late-Onset Fetal Growth Restriction. *JAMA Netw Open*. 2025;8(6):17360.

doi:10.1001/jamanetworkopen.2025.17360.

34. Fetal-Growth-Assessment-Guideline-.pdf

<https://www.mkuh.nhs.uk/wp-content/uploads/2022/10/Fetal-Growth-Assessment-Guideline-.pdf>

35. Hein O, Lucy TB, Anusha L, Daan N. Risk factors for small for gestational age as defined by a birthweight z-

score below minus one: A prospective observational study. *Med Res Arch*. 2024;12(8):5731.

doi:10.18103/mra.v12i8.5731.

36. Dawn-Marie W, Neil M, Lisa U, Harriet G, Janet H, Andy V, Dieter W. The Growth Restriction Intervention

Trial: long-term outcomes in a randomized trial of timing of delivery in fetal growth restriction. *Am J Obstet Gynecol*. 2011;204(1):1-9.

doi: 10.1016/j.ajog.2010.09.019..