

Factors Associated with Fetal Growth Restriction and Small-For-Gestational-Age Fetuses in a Private Tertiary Care Hospital

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ABSTRACT

Objective: To describe risk factors associated with fetal growth restriction (FGR) and small-for-gestational-age (SGA) fetuses and assess their presence in a private tertiary-care hospital population.

Methods: A retrospective observational cohort study included singleton pregnancies diagnosed with impaired fetal growth between April 2018 and September 2022 at a Maternal–Fetal Medicine unit. Cases were classified as early- or late-onset FGR or SGA according to standardized diagnostic criteria. Maternal characteristics, comorbidities, obstetric history, biochemical markers, and neonatal outcomes were analyzed using nonparametric statistics.

Results: Among 143 eligible patients, 15 (10.5%) had early-onset FGR, 47 (32.9%) late-onset FGR, and 81 (56.6%) SGA. Demographic characteristics were similar across groups. Most patients had normal pregestational BMI; however, inadequate gestational weight gain was frequent. Early-onset FGR was associated with significantly lower birthweight ($p = 0.000$). Preeclampsia correlated with lower neonatal weight, while assisted reproductive technology (ART) pregnancies showed higher mean birthweight.

Conclusions: In this cohort, inadequate gestational weight gain emerged as a frequent potentially modifiable factor. Strengthening nutritional counseling and multidisciplinary prenatal care may improve perinatal outcomes.

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INTRODUCTION

Fetal growth restriction (FGR) is defined as the failure of a fetus to reach its growth potential due to placental dysfunction (1). Standardized diagnostic criteria were established through an international Delphi consensus and later validated (1,2). International best-practice recommendations emphasize systematic screening, diagnosis, and surveillance to reduce perinatal morbidity and mortality (3).

FGR is categorized into early- and late-onset phenotypes according to gestational age at diagnosis (4,5). These phenotypes differ in severity, Doppler findings, placental pathology, and clinical progression (4,5).

FGR results from maternal, placental, or fetal conditions that impair nutrient and oxygen transfer (6). Maternal risk factors include advanced age, low BMI, chronic disease, substance use, and assisted reproductive technologies (3,6). Inadequate

gestational weight gain has been associated with increased risk of SGA and adverse neonatal outcomes (7).

Smoking increases the risk of SGA, while cessation reduces incidence and improves birthweight (8,9). Placental dysfunction accounts for a significant proportion of FGR cases (6,10,11). Chromosomal abnormalities and infections contribute to a smaller percentage (12,13).

Low-dose aspirin has been recommended in high-risk pregnancies to reduce preeclampsia and possibly FGR (14). First-trimester biomarkers such as pregnancy-associated plasma protein-A (PAPP-A) and β -hCG have been associated with placental-mediated complications (15,16). Combined screening models improve predictive performance (17).

Short-term consequences include stillbirth and prematurity (18). Increasing evidence supports fetal programming mechanisms linking adverse intrauterine environments to adult cardiovascular and metabolic disease (19–21).

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Advanced maternal age has been associated with increased SGA risk (22,23). Primigravidity may influence birthweight through immunologic mechanisms (24). Maternal nutrition has lifelong implications for offspring health (25). Recurrence risk of FGR ranges from 20–30% (26). Hypothyroidism has been linked to impaired placental vascularization (27). Smoking prevalence varies across populations (28). Aspirin may exert molecular effects beyond platelet inhibition (29). ART pregnancies have been associated with placental dysfunction and growth abnormalities (30).

Methods

Study Design and Population

A retrospective observational cohort study was conducted at the Maternal–Fetal Medicine Unit of Hospital Ángeles Lomas. Singleton pregnancies diagnosed with FGR or SGA between April 2018 and September 2022 were included. FGR and SGA were defined according to international consensus criteria (1,5). Patients with structural anomalies, chromosomal abnormalities, intra-amniotic infection, or incomplete records were excluded.

Data Collection

Maternal demographic variables included age, parity, pregestational weight, BMI, gestational weight gain, smoking, ART use, aspirin intake, hypothyroidism, thrombophilia, and preeclampsia.

Biochemical markers (MoM values of β -hCG and PAPP-A) were recorded.

Neonatal outcomes included gestational age at delivery, birthweight, length, sex, and Apgar scores.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics v25. Numerical variables were expressed as mean $\pm$ SD. Categorical variables were expressed as frequencies and percentages. Due to non-normal distribution, Mann–Whitney U and Kruskal–Wallis tests were applied. Spearman's rho was used for correlation analysis. Statistical significance was defined as $p < 0.05$.

RESULTS

A total of 196 patients were identified; 53 were excluded due to incomplete data, leaving 143 patients.

Early-onset FGR: 15 (10.5%)

Late-onset FGR: 47 (32.9%)

SGA: 81 (56.6%)

Mean maternal age was 30 ± 5 years. Mean pregestational BMI was within normal range in 79% of patients. However, 69% of women with normal BMI gained less weight than recommended.

Mean neonatal birthweight was $2,321 \pm 406$ g. Two fetal deaths occurred, both in the early-onset FGR group.

Birthweight differed significantly between groups ($p = 0.000$), with early-onset FGR showing the lowest values.

ART pregnancies showed higher mean birthweight ($p = 0.012$).

Preeclampsia during pregnancy was associated with a reduction of approximately 800 g in neonatal birthweight ($p = 0.000$).

Birthweight correlated positively with gestational age at delivery ($p = 0.688$, $p = 0.000$) and Apgar at 1 minute ($p = 0.181$, $p = 0.030$).

Variable	SGA (n = 81)	Early FGR (n = 15)	Late FGR (n = 47)	p-value
Maternal age (years)	30.1 ± 5	30.7 ± 4	30.3 ± 5	0.840
Age ≥ 35 years, n (%)	15	2	9	—
Primigravida, n	28	11	22	—
Prepregnancy weight (kg)	55.4 ± 8	46.9 ± 11	57.7 ± 8	0.881
Prepregnancy BMI (kg/m²)	21.6 ± 3	21.6 ± 4	22.4 ± 3	0.392
Gestational weight gain (kg)	10.1 ± 2	10.0 ± 3	9.6 ± 4	0.255
Inadequate GWG (normal BMI group), n (%)	78 (69%)*	—	—	—
Smoking during pregnancy, n	0	1	3	0.381
ART pregnancy, n	10	2	2	0.012†
Aspirin use, n	14	4	14	0.161
Preeclampsia during pregnancy, n	2	3	2	0.000†
Variable	SGA	Early FGR	Late FGR	p-value
Gestational age at delivery (weeks)	38.1 ± 2	35.2 ± 4	37.5 ± 2	—
Birthweight (g)	2480 ± 322	1851 ± 586	2198 ± 305	0.000†
Length (cm)	47.3 ± 2	42.9 ± 4	42.9 ± 4	—
Apgar 1 min	9	8	9	0.844

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Variable	SGA (n = 81)	Early FGR (n = 15)	Late FGR (n = 47)	p-value
Apgar 5 min	9	8	9	0.091
Fetal deaths, n	0	2	0	—

Variable	Mean Birthweight (g)	p-value
ART – No	2290	
ART – Yes	2604	0.012†
Preeclampsia – No	2359	

DISCUSSION

This study evaluated risk factors associated with impaired fetal growth in a private tertiary-care population.

Advanced maternal age was infrequent in this cohort compared to populations described in the literature (22,23). Although primigravidity has been linked to lower neonatal weight (24), differences were not statistically significant in this sample.

Most patients had normal pregestational BMI; however, inadequate gestational weight gain was common. Maternal nutrition has long-term metabolic implications for offspring (25), supporting the relevance of this finding.

Preeclampsia demonstrated a strong association with reduced birthweight, consistent with placental insufficiency mechanisms (6). Recurrence of FGR was low in this sample compared to reported recurrence risks (26).

Hypothyroidism was present in a notable proportion of patients, aligning with evidence linking thyroid dysfunction to impaired placental vascularization (27).

Smoking prevalence was lower than reported in other populations (28), limiting its detectable impact.

Although aspirin reduces preeclampsia risk (14,29), growth restriction occurred despite prophylaxis in some patients.

Contrary to some reports associating ART with placental dysfunction (30), ART pregnancies in this cohort showed higher birthweight, possibly reflecting closer surveillance.

Early-onset FGR was associated with fetal mortality, consistent with its known higher perinatal risk (3,18).

CONCLUSIONS

The studied population differed from classical high-risk profiles. Most women began pregnancy at favorable reproductive age, with normal BMI and low smoking prevalence. However, inadequate gestational weight gain was frequent and may represent a key modifiable factor.

Early detection and multidisciplinary prenatal care—including nutritional support—are essential to reduce adverse perinatal outcomes and potential long-term metabolic consequences.

Further studies with larger and more diverse populations are warranted to confirm these findings.

REFERENCES

- I. Gordijn SJ, Beune IM, Thilaganathan B, Papageorgiou A, Baschat AA, Baker PN, et al. Consensus definition of fetal growth restriction: a Delphi procedure. *Ultrasound Obstet Gynecol.* 2016;48(3):333–9.
- II. Molina LCG, Odibo L, Zientara S, Običan SG, Rodriguez A, Stout M, et al. Validation of Delphi procedure consensus criteria for defining fetal growth restriction. *Ultrasound Obstet Gynecol.* 2020;56(1):61–6.
- III. Melamed N, Baschat A, Yinon Y, Athanasiadis A, Mecacci F, Figueras F, et al. FIGO initiative on fetal growth: best practice advice. *Int J Gynecol Obstet.* 2021;152(S1):3–57.
- IV. Aviram A, Sherman C, Kingdom J, Zaltz A, Barrett J, Melamed N. Defining early vs late fetal growth restriction by placental pathology. *Acta Obstet Gynecol Scand.* 2019;98(3):365–73.
- V. Lees CC, Stampalija T, Baschat A, da Silva Costa F, Ferrazzi E, Figueras F, et al. ISUOG Practice Guidelines: diagnosis and management of SGA and FGR. *Ultrasound Obstet Gynecol.* 2020;56(2):298–312.
- VI. Nardoza LMM, et al. Fetal growth restriction: current knowledge. *Arch Gynecol Obstet.* 2017;295(5):1061–77.
- VII. Rasmussen KM, Yaktine AL. *Weight Gain During Pregnancy.* National Academies Press; 2009.
- VIII. Committee on Obstetric Practice ACOG. Tobacco and Nicotine Cessation During Pregnancy. *Obstet Gynecol.* 2020;135(5):221–9.
- IX. Committee on Practice Bulletins ACOG. Fetal Growth Restriction. *Obstet Gynecol.* 2019;133(76):168–86.
- X. Zur RL, Kingdom JC, Parks WT, Hobson SR. The Placental Basis of FGR. *Obstet Gynecol Clin North Am.* 2020;47(1):81–98.
- XI. Albu AR, et al. Predictive factors for intrauterine growth restriction. *J Med Life.* 2014;7(2):165–71.

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- XII. Longo S, et al. IUGR and infections. *Early Hum Dev.* 2014;90(S1):S42–4.
- XIII. Maulik D. Fetal growth restriction: The etiology. *Clin Obstet Gynecol.* 2006;49(2):228–35.
- XIV. Meher S, et al. Antiplatelet therapy before or after 16 weeks. *Am J Obstet Gynecol.* 2017;216(2):121–128.
- XV. Smith GCS. Universal screening for FGR. *Best Pract Res Clin Obstet Gynaecol.* 2018;49:16–28.
- XVI. Cignini P, et al. Predictive value of PAPP-A and free β -hCG on FGR. *Arch Gynecol Obstet.* 2016;293(6):1227–33.
- XVII. Chaemsathong P, Sahota DS, Poon LC. First trimester preeclampsia screening. *Am J Obstet Gynecol.* 2022;226(2):S1071–S1097.
- XVIII. Pallotto EK, Kilbride HW. Perinatal outcome and implications of IUGR. *Clin Obstet Gynecol.* 2006;49(2):257–69.
- XIX. Moreno-Fernandez J, et al. Early nutrition and fetal programming. *Nutrients.* 2020;12(12).
- XX. Wei Y, Schatten H, Sun QY. Environmental epigenetic inheritance. *Hum Reprod Update.* 2015;21(2):194–208.
- XXI. Lane RH. Fetal programming and adult disease. *Clin Perinatol.* 2014;41(4):815–31.
- XXII. Lean SC, et al. Placental dysfunction and advanced maternal age. *Sci Rep.* 2017;7(1).
- XXIII. Leader J, et al. Very advanced maternal age outcomes. *J Obstet Gynaecol Canada.* 2018;40(9):1208–18.
- XXIV. Robillard PY, et al. Primipaternities and birthweights. *J Reprod Immunol.* 2021;147.
- XXV. Marshall NE, et al. Nutrition in pregnancy and lifelong consequences. *Am J Obstet Gynecol.* 2022;226(5):607–32.
- XXVI. Blue NR, Page JM, Silver RM. Recurrence risk of FGR. *Obstet Gynecol Clin North Am.* 2021;48(2):419–36.
- XXVII. Serakides R, et al. FGR in hypothyroidism. *Reprod Fertil Dev.* 2012;24(7):923–31.
- XXVIII. Santiago M, et al. Smoking in pregnancy prevalence. *Rev Esp Salud Pública.* 2013;30(1):37–40.
- XXIX. Dutta S, et al. Molecular targets of aspirin in pregnancy. *Int J Mol Sci.* 2019;20(18).
- XXX. Manna C, et al. Placental dysfunction in ART pregnancies. *Int J Mol Sci.* 2022;23(2).