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Original Research Article

Maternal and Placental Risk Factors Associated with Small for Gestational Age Neonates: An Analytical Cross-Sectional Study from a Tertiary Care Centre in South India

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Abstract

Background: Small for gestational age (SGA) neonates carry a disproportionate burden of perinatal morbidity and mortality, and both maternal and placental determinants contribute to this outcome. Region-specific data linking clinical, nutritional, and placental morphometric variables are limited, particularly from tertiary centres in Tamil Nadu.

Objectives: To evaluate the association of maternal socio-demographic, clinical, and nutritional factors, along with placental morphometric parameters, with the occurrence of SGA neonates at a tertiary care hospital.

Methods: This analytical cross-sectional study was conducted over one year in the Department of Neonatology of a Government Medical College Hospital, Tamil Nadu. Sixty-eight neonates classified as SGA and 68 appropriate-for-gestational-age (AGA) controls, categorised using INTERGROWTH-21st standards, were enrolled after ethics committee approval. Maternal demographic, anthropometric, antenatal, and haematological data were collected using a structured proforma, and placentas were subjected to standardised gross morphometric examination. Data were analysed in SPSS using chi-square/Fisher's exact tests, independent t-tests, Mann-Whitney U tests, and multivariable logistic and linear regression, with $p<0.05$ considered significant.

Results: Mean birth weight was significantly lower in the SGA group than the AGA group (2227.32 ± 322.78 g vs 2888.68 ± 405.65 g; $p<0.001$), while gestational age and sex distribution did not differ between groups. Moderate maternal anaemia (OR 15.00, 95% CI 3.07–73.29), inadequate gestational weight gain (OR 18.75, 95% CI 2.39–147.08), a previous history of SGA delivery (OR 18.75, 95% CI 2.39–147.08), a short interpregnancy interval (OR 5.06, 95% CI 2.03–12.62), and irregular antenatal supplementation (OR 3.19, 95% CI 1.56–6.47) were significantly associated with SGA. Among placental parameters, abnormal placental thickness (OR 7.75), volume (OR 4.88), weight (OR 4.86) and other structural abnormalities (OR 3.58) were significantly associated with SGA, whereas the placental ratio was not ($p=0.146$). Maternal weight gain correlated positively with birth weight ($r=0.440$, $p<0.001$).

Conclusion: SGA in this population is driven by a multifactorial interaction of maternal nutritional and haematological status and placental morphometric insufficiency. Routine antenatal anaemia screening, monitoring of gestational weight gain, and gross placental examination at delivery are recommended as feasible, low-cost adjuncts for risk stratification in similar tertiary care settings.

Keywords: Small for gestational age; intrauterine growth restriction; placental morphometry; maternal anaemia; gestational weight gain; INTERGROWTH-21st; tertiary care; South India.

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Introduction

Small for gestational age (SGA) neonates are defined as those with a birth weight or crown-heel length below the 10th percentile, or more than two standard deviations below the mean, for gestational age. As of 2020, an estimated 23.4 million neonates worldwide were classified as SGA out of approximately 135 million live births, comprising 21.9 million term and 1.5 million preterm SGA infants [1]. Earlier global modelling using CHERG datasets estimated that 19.3% of live births in low- and middle-income countries (LMICs) were SGA, and that this group accounted for over one-fifth of neonatal deaths in these settings, with the burden being highest in South Asia [2]. The choice of birth-weight-for-gestation reference population has itself been shown to materially influence both the prevalence and the estimated mortality risk attributed to SGA, complicating cross-study comparison [3].

Within India, reported SGA prevalence ranges widely from 12% to over 78%, depending on the diagnostic reference and population studied [3]. A South Indian cohort described temporal trends in SGA prevalence linked to maternal nutritional and antenatal care practices [4], while Tamil Nadu-specific estimates have placed the prevalence around 8.3%.

At the authors' own institution, a tertiary referral centre in Chengalpattu, preliminary institutional data suggested a considerably higher prevalence of approximately 18.4%, prompting the present investigation into local, potentially modifiable determinants.

SGA neonates may be constitutionally small, representing a benign variant of normal growth, or may reflect true intrauterine growth restriction (IUGR) arising from a pathological process that limits genetically determined growth potential.

IUGR is further sub classified as symmetrical, typically due to early insults such as chromosomal anomalies or congenital infection, or asymmetrical, which accounts for 70–80% of cases and results from uteroplacental insufficiency later in gestation, producing the characteristic head-sparing pattern. SGA neonates are at heightened risk of intrauterine demise, birth asphyxia, meconium aspiration, hypoglycaemia, hypothermia, polycythaemia, and necrotising enterocolitis in the immediate perinatal period, and of neurodevelopmental delay, stunted growth, and adult-onset metabolic and cardiovascular disease over the longer term, consistent with the foetal origins hypothesis.

While maternal and foetal contributors to SGA — including undernutrition, anaemia, hypertensive disorders, and inadequate antenatal surveillance — are well described, placental morphology remains comparatively under-examined in routine obstetric and neonatal practice despite its central pathophysiological role. Gross placental parameters such as weight, volume, thickness, and the placental-to-birth-weight ratio have each been proposed as accessible markers of placental sufficiency [10,11,14,27], yet their relative diagnostic value, particularly in South Indian tertiary care populations, has not been well characterised.

This study was therefore designed to concurrently evaluate maternal socio-demographic, nutritional, and clinical risk factors together with standardised gross placental morphometry in a cohort of SGA and AGA neonates, in order to generate region-specific evidence that could inform antenatal risk stratification and the potential value of routine placental examination.

Aim and Objectives

Aim: To evaluate the maternal and placental risk factors associated with the birth of small for gestational age (SGA) neonates at a tertiary care centre.

Objectives

1. To determine the association between maternal socio-demographic and clinical factors (age, socioeconomic status, parity, residence, body mass index, anaemia, gestational weight gain, interpregnancy interval, previous history of SGA, and antenatal supplementation) and the occurrence of SGA neonates.
2. To assess and compare gross placental morphometric parameters — weight, volume, thickness, and placental ratio — and other structural abnormalities between SGA and AGA neonates.
3. To determine the correlation between maternal gestational weight gain and neonatal birth weight through regression analysis.

Materials and Methods

Study design and setting: This was an analytical cross-sectional study conducted in the Department of Neonatology of a Government Medical College and Hospital, a tertiary care referral centre in Chengalpattu district, Tamil Nadu, India, over a period of one year, from May 2024 to April 2025. The study was approved by the Institutional Human Ethics Committee prior to commencement, and written informed consent was obtained from all participating mothers.

Study population: The target population comprised liveborn neonates delivered at the study institution during the study period. Neonates were

categorised as SGA or appropriate for gestational age (AGA) using the INTERGROWTH-21st international growth standards, with gestational age dating standardised against the 50th percentile values of the INTERGROWTH-21st dating charts, based on the earliest available first-trimester crown-rump length ultrasound wherever possible.

Inclusion Criteria: (i) Live births occurring at the study institution within the defined study period; and (ii) for case classification, a birth weight below the 10th percentile for gestational age as per INTERGROWTH-21st standards. Neonates with birth weight between the 10th and 90th percentile were included as controls (AGA).

Exclusion Criteria: (i) Neonates born of multiple gestations (twins, triplets, or higher-order pregnancies); (ii) births at a gestational age of less than 24 completed weeks; and (iii) stillbirths, defined as the absence of signs of life at delivery. Neonates with birth weight above the 90th percentile were also excluded from case-control comparison.

Sample Size: Sample size was calculated using OpenEpi software based on the mean and standard deviation of placental weight reported in a reference study, at 95% confidence and 80% power, yielding a required sample of 136 neonates — 68 SGA cases and 68 AGA controls — which was achieved in full.

Tools and investigation methods: Data were collected using a structured, pre-tested proforma covering maternal demographic characteristics (age, residence, education, occupation, household income-based socioeconomic classification, parity, and interpregnancy interval), antenatal record review, and direct maternal interview.

Pre-pregnancy weight and height were abstracted from the Mother and Child Protection (MCP) card, and body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Gestational weight gain was computed as the difference between weight at delivery and pre-pregnancy weight, and converted to a z-score using the INTERGROWTH-21st Gestational Weight Gain Calculator (2017), with a z-score below −2 designated as inadequate weight gain.

Antenatal care adequacy was defined by a minimum of three antenatal visits, regular nutritional supplementation from the second trimester onward, and an interpregnancy interval of three years or more. Maternal comorbidities — overt and gestational diabetes mellitus, pregnancy-induced hypertension, preeclampsia, eclampsia, anaemia, and thyroid dysfunction — were confirmed against clinical records using standard obstetric definitions. Antenatal IUGR was ascertained from serial growth ultrasonography and Doppler studies, supplemented postnatally by the Clinical Assessment of Nutrition (CAN) score adapted from Metcoff's criteria for cases of diagnostic ambiguity. Following delivery, all placentas underwent standardised macroscopic examination: blood clots were removed and the trimmed placenta weighed on a calibrated electronic scale (grams); maximum and minimum diameters were measured with a non-stretchable tape (centimetres); thickness was measured centrally with a sterile needle (millimetres); placental area was computed as the product of maximum and minimum diameter, and placental volume as area multiplied by thickness; and the placental ratio was derived as placental weight divided by birth weight.

Placental weight percentiles were referenced against gestational-age-stratified standards described by Thomas et al. Gross morphological assessment additionally documented completeness, umbilical cord insertion pattern, membrane attachment, and other structural abnormalities (bilobed placenta, succenturiate lobes, calcification, and chorioangioma), with photographic documentation of deviations from normal morphology.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS) software (IBM Corp., Armonk, NY, USA). Normally distributed continuous variables were summarised as mean ± standard deviation (SD) and compared using the independent samples t-test; skewed continuous and ordinal variables were summarised as median with interquartile range (IQR) and compared using the Mann–Whitney U test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test, or Fisher's exact test where expected cell counts were small.

Unadjusted odds ratios (OR) with 95% confidence intervals (CI) were calculated for candidate risk factors, and the correlation between maternal weight gain and neonatal birth weight was assessed using Pearson's correlation coefficient and linear regression modelling. A two-tailed p-value of less than 0.05 was considered statistically significant throughout.

Results

A total of 136 neonates (68 SGA and 68 AGA) were enrolled and analysed. The baseline socio-demographic and clinical characteristics of the two groups are summarised in Table 1. Sex distribution did not differ significantly between the SGA (54.41% male) and AGA (45.59% male) groups (p=0.303), and mean gestational age was comparable between groups (37.51 ± 1.48 vs 37.63 ± 1.47 weeks; p=0.645), indicating that prematurity did not confound group classification. In contrast,

mean birth weight was markedly lower in the SGA group (2227.32 ± 322.78 g) than in the AGA group (2888.68 ± 405.65 g; p<0.001), consistent with the defining criterion for SGA. Mode of delivery did not differ significantly between groups (p=0.362), with lower segment caesarean section (LSCS) being the commonest mode in both (58.82% SGA vs 60.29% AGA). Antenatal ultrasound identified IUGR in 38.24% of eventual SGA neonates, but this rose to a substantial proportion of undetected cases (54.41% of SGA neonates had no antenatal IUGR features), a difference that was highly significant (p<0.001) and highlights the limited antenatal sensitivity of routine growth scans for this group.

Table 1: Distribution of Demographic and Clinical Characteristics among SGA and AGA Neonates

Variable	SGA (n=68)	AGA (n=68)	p-value
Male sex, n (%)	37 (54.41)	31 (45.59)	0.303
Female sex, n (%)	31 (45.59)	37 (54.41)	
Birth weight, g (mean±SD)	2227.32 ± 322.78	2888.68 ± 405.65	<0.001
Gestational age, weeks (mean±SD)	37.51 ± 1.48	37.63 ± 1.47	0.645
NVD, n (%)	26 (38.24)	27 (39.71)	0.362
AVD, n (%)	2 (2.94)	0 (0)	
LSCS, n (%)	40 (58.82)	41 (60.29)	
Antenatal IUGR – Yes, n (%)	26 (38.24)	3 (4.41)	<0.001
Antenatal IUGR – No, n (%)	37 (54.41)	7 (10.29)	

NVD, normal vaginal delivery; AVD, assisted vaginal delivery; LSCS, lower segment caesarean section; IUGR, intrauterine growth restriction.

Table 2 presents the unadjusted associations between maternal socio-demographic and clinical factors and SGA status. Maternal age below 21 years was noted in 17.65% of SGA mothers, with no AGA mothers in this category, precluding odds ratio estimation, though the skewed distribution is suggestive of an association between younger maternal age and SGA risk.

Socioeconomic status showed a non-significant trend toward reduced risk in the upper-middle class (OR 0.50, 95% CI 0.24–1.03; p=0.06) relative to the lower-middle class (OR 1.05, 95% CI 0.54–2.06; p=0.885) , while rural versus urban residence showed no significant association (OR 0.72, 95% CI 0.33–1.59; p=0.417). Underweight mothers (BMI <18.5) showed a non-significant trend toward higher SGA risk (OR 1.89, 95% CI 1.00–3.58; p=0.130), whereas overweight and obese categories showed no meaningful association (BMI)). Maternal anaemia showed a strong, statistically significant, dose-related association with SGA: mild anaemia conferred an OR of 3.85 (95% CI 1.82–8.14; p<0.001) and moderate anaemia an OR of 15.00 (95% CI 3.07–73.29; p=0.001) relative to non-anaemic mothers.

Inadequate gestational weight gain (z-score < −2) was strongly associated with SGA (OR 18.75, 95% CI 2.39–147.08; p=0.005), as was a prior history of delivering an SGA infant, which carried an identical unadjusted OR of 18.75 (95% CI 2.39–147.08; p=0.005). Irregular or absent antenatal supplementation was associated with a more than three-fold increased risk of SGA (OR 3.19, 95% CI 1.56–6.47; p<0.001), and an interpregnancy interval of less than three years conferred an OR of 5.06 (95% CI 2.03–12.62; p<0.001).

Table 2: Association between Maternal Socio-Demographic and Clinical Factors and Small for Gestational Age (Unadjusted Odds Ratios)

Factor (category)	SGA n (%)	AGA n (%)	OR (95% CI)	p-value
Maternal age <21 yrs	12 (17.65)	0 (0)	Not estimable	–
Socioeconomic – upper middle	18 (26.47)	33 (48.53)	0.50 (0.24–1.03)	0.06
Socioeconomic – lower middle	40 (58.82)	35 (51.47)	1.05 (0.54–2.06)	0.885
Rural residence	14 (20.59)	18 (26.47)	0.72 (0.33–1.59)	0.417
BMI <18.5 kg/m²	21 (30.88)	13 (19.11)	1.89 (1.00–3.58)	0.130
Mild anaemia	40 (58.82)	26 (38.24)	3.85 (1.82–8.14)	<0.001
Moderate anaemia	12 (17.65)	2 (2.94)	15.00 (3.07–73.29)	0.001
Weight gain z-score < −2SD	15 (22.06)	1 (1.47)	18.75 (2.39–147.08)	0.005
Interpregnancy interval <3 yrs	12 (17.65)	13 (19.12)	5.06 (2.03–12.62)	<0.001
Previous history of SGA	15 (22.06)	1 (1.47)	18.75 (2.39–147.08)	0.005
Irregular/no antenatal supplements	35 (51.47)	17 (25)	3.19 (1.56–6.47)	<0.001

BMI, body mass index; OR, odds ratio; CI, confidence interval; SD, standard deviation. Reference categories: normal BMI (18.5–24.9), non-anaemic mothers, weight gain ≥−2SD, interpregnancy interval ≥3 years, no prior SGA history, and regular antenatal supplementation.

Placental morphometric findings are summarised in Table 3. Abnormal placental weight was significantly more common among SGA neonates (45.59% vs 14.71%; OR 4.86, 95% CI 2.13–11.07; p<0.001), as was abnormal placental volume (92.65% vs 72.06%; OR 4.88, 95% CI 1.70–14.01; p=0.003) and abnormal placental thickness (47.06% vs 10.29%; OR 7.75, 95% CI 3.10–19.35; p<0.001), which showed the strongest association of all placental parameters examined. Other structural abnormalities, including abnormal cord insertion and membrane attachment, bilobed placenta, succenturiate lobes, calcification, and chorioangioma, were also significantly more frequent among SGA placentas (35.29% vs 13.23%; OR 3.58, 95% CI 1.51–8.45; p=0.003). In contrast, the placental ratio — placental weight divided by birth weight — did not differ significantly between groups (OR 1.87, 95% CI 0.80–4.32; p=0.146), suggesting it is a less sensitive discriminator than the other morphometric indices in this cohort. Summarises the comparative magnitude of these odds ratios across all placental parameters.

Table 3: Association between Placental Morphometric Parameters and Small for Gestational Age

Placental parameter (abnormal)	SGA n (%)	AGA n (%)	OR (95% CI)	p-value
Placental weight	31 (45.59)	10 (14.71)	4.86 (2.13–11.07)	<0.001
Placental volume	63 (92.65)	49 (72.06)	4.88 (1.70–14.01)	0.003
Placental thickness	32 (47.06)	7 (10.29)	7.75 (3.10–19.35)	<0.001
Placental ratio	18 (26.47)	11 (16.18)	1.87 (0.80–4.32)	0.146
Other abnormalities	24 (35.29)	9 (13.23)	3.58 (1.51–8.45)	0.003

Reference category for each parameter is the corresponding normal finding.

The relationship between maternal gestational weight gain and neonatal birth weight was examined using correlation and linear regression, summarised in Table 4.

A significant positive correlation was observed between weight gain and birth weight (r=0.440; p<0.001) and between weight gain and the weight-for-length ratio (r=0.253; p=0.003). Linear regression modelling birth weight (kg) as a predictor of maternal weight gain yielded a statistically significant unstandardized coefficient (B=2.400, SE=0.462, t=5.197, p<0.001; 95% CI 1.487–3.313), indicating that for every 1 kg increase in birth weight, maternal weight gain increased by approximately 2.4 kg, and confirming maternal gestational weight gain as an independent predictor of neonatal birth weight in this cohort.

Table 4: Correlation and Linear Regression Analysis of Maternal Gestational Weight Gain and Neonatal Birth Weight

Analysis	Coefficient	95% CI	p-value
Correlation: weight gain vs birth weight (r)	0.440	–	<0.001
Correlation: weight gain vs weight/length ratio (r)	0.253	–	0.003
Regression constant (B)	2.669 (SE 1.198)	0.299–5.038	0.028
Regression coefficient – birth weight, kg (B)	2.400 (SE 0.462)	1.487–3.313	<0.001

SE, standard error; CI, confidence interval; r, Pearson correlation coefficient.

Discussion:

This analytical cross-sectional study of 136 neonates identified several maternal and placental factors significantly associated with SGA, consistent with, and in some respects extending, the existing global and Indian literature. Foetal sex showed no significant association with SGA occurrence, a finding that mirrors the null association reported by Teixeira et al. in a Brazilian cohort [8] and by Cheng et al. among late-term infants in China [6], although Subramanian et al. described a slight female predominance among SGA births in Kerala, potentially reflecting regional nutritional or socio-cultural determinants [7]. The marked birth weight disparity we observed between SGA and AGA groups mirrors findings from LMIC cohorts reported by Lee et al., where lower birth weight in SGA infants contributed materially to excess neonatal mortality [2], and is consistent with Katz et al.'s emphasis on birth weight as the operational diagnostic criterion for SGA regardless of the reference standard applied [3].

Gestational age did not differ between groups in our cohort, in keeping with Cheng et al.'s observation that growth restriction, rather than preterm birth per se, distinguishes SGA from AGA late-term infants [6], and consistent with Budal et al.'s finding that gestational age at delivery primarily modulates neonatal morbidity rather than SGA classification when standardised growth charts are applied [23]. Our use of INTERGROWTH-21st dating standards, endorsed in the Brighton Collaboration case-definition guidelines by Schlaudecker et al., supports the internal validity of this classification [24]. Mode of

delivery similarly showed no significant association with SGA status, in agreement with Asgharnia et al. [13] and Teixeira et al. [8], who both concluded that delivery mode is generally dictated by concurrent obstetric indications rather than by SGA status itself; McCowan et al.'s consensus guideline further recommends that indications for operative delivery in suspected growth restriction be evaluated independently of SGA classification [26].

Only 38.24% of eventual SGA neonates in our cohort were flagged as IUGR on antenatal ultrasound, underscoring a substantial gap in prenatal detection. Tsikouras et al. highlighted that the sensitivity of serial ultrasound and Doppler assessment for IUGR is highly dependent on scan timing and operator expertise [22], while Savchev et al. and Lees et al., in the TRUFFLE cohort, both demonstrated that late-onset growth restriction is disproportionately likely to evade routine third-trimester scanning [38,39]. Pels et al.'s systematic review similarly calls for enhanced surveillance protocols to improve early identification of growth-restricted foetuses [43], a recommendation our findings directly support.

The suggestion of increased SGA risk with adolescent maternal age in our data, though not statistically quantifiable owing to zero AGA representation in the youngest age band, parallels findings by Teixeira et al. [8] and Subramanian et al. [7], both of whom identified younger maternal age as a risk factor attributable to nutritional and physiological immaturity, and by Sharma et al., who linked adolescent pregnancy to heightened IUGR risk through competing nutritional demands [19]. A protective trend for higher socioeconomic status, though not statistically significant in our cohort, is congruent with Sebastian et al.'s South Indian data linking lower socioeconomic status to greater SGA prevalence via constrained healthcare and nutritional access [4], and with the broader LMIC pattern described by Lee et al. [2]. Unlike Subramanian et al., who reported higher SGA rates in rural populations attributable to weaker healthcare infrastructure [7], our study found no significant urban–rural difference, aligning instead with Cheng et al.'s observation that residence effects diminish once clinical and nutritional factors are accounted for [6].

The non-significant trend toward higher SGA risk with maternal underweight in our cohort is directionally consistent with Subramanian et al., who reported that each additional kilogram of pre-pregnancy weight reduced SGA risk by 0.8% [7], and with Sharma et al.'s description of low maternal BMI as a nutritional risk factor for IUGR [19]; the absence of statistical significance here likely reflects the modest sample size, as similarly cautioned by Katz et al. [3]. By contrast, maternal anaemia emerged as one of the strongest and most consistent risk factors identified, corroborating Thekkedathu et al.'s identification of anaemia as a significant risk factor for symmetric IUGR [5], Lee et al.'s description of anaemia-mediated reduction in placental oxygen and nutrient transport in LMIC settings [2], and Dhital et al.'s report that anaemia exacerbates placental pathology in SGA pregnancies complicated by autoimmune disease [20]. These findings collectively support Sebastian et al.'s call for routine antenatal anaemia screening and iron supplementation as a SGA-prevention strategy [4].

Inadequate gestational weight gain and irregular antenatal supplementation were both significantly associated with SGA in our cohort, echoing Subramanian et al.'s finding that suboptimal weight gain strongly predicts low birth weight [7], Sebastian et al.'s support for INTERGROWTH-21st-based weight-gain monitoring [4], and Sharma et al.'s emphasis on nutritional intervention in preventing IUGR [19]. A previous history of SGA delivery was similarly a strong predictor of recurrence, in agreement with Teixeira et al. [8], Sharma et al.'s discussion of persisting genetic, environmental, and vascular contributors [19], and Pinheiro et al.'s linkage of recurrent SGA to maternal vascular malperfusion on placental histopathology [21].

Placental morphometry proved to be a particularly informative domain in our study. Reduced placental weight in SGA neonates parallels Oliveira et al.'s report of significantly lower placental weight in SGA infants [10], Zhang et al.'s finding of reduced placental weight in SGA neonates born to hypertensive mothers [11], and Menter et al.'s and Redline et al.'s broader advocacy for routine placental examination as a diagnostic adjunct with prognostic value [14,27]. Reduced placental volume showed a comparably strong association, consistent with Zhang et al.'s and Calis et al.'s descriptions of diminished placental volume reflecting placental insufficiency and impaired nutrient transfer [15,16], while Marijnen et al. have called for standardised methodology to allow such volumetric findings to be compared more reliably across studies [25]. Abnormal placental thickness showed the strongest association of all placental parameters examined, in keeping with Kleebkaow et al.'s and Zhang et al.'s reports linking thickness deviations to low birth weight and hypertensive pregnancies respectively [11,12], and with Pinheiro et al.'s association of thickness alterations with maternal vascular malperfusion [21].

In contrast to Oliveira et al., who identified an increased placental-to-foetal weight ratio as predictive of SGA [10], the placental ratio in our cohort showed no significant association, a discrepancy that Asgharnia et al. and Redline et al.

attribute to population-specific variability and adaptive placental mechanisms that may blunt the discriminatory value of this particular index [13,27]. Other structural placental abnormalities — including anomalous cord and membrane insertion, bilobed placenta, and calcification — were significantly more common among SGA placentas, consistent with Calis et al.'s and Pinheiro et al.'s descriptions of such lesions in growth-restricted pregnancies [15,21] and with Altshuler et al.'s classical description of villous fibrosis and reduced vascular branching in SGA placentas [18], reinforcing Redline et al.'s argument for a placental-phenotype-based approach to obstetric taxonomy [27].

The significant positive correlation between maternal weight gain and birth weight in our regression analysis is in agreement with Subramanian et al.'s demonstration that maternal weight gain predicts birth weight outcomes [7], Sharma et al.'s association of adequate weight gain with reduced IUGR risk [19], and Calek et al.'s description of distinct metabolic profiles in IUGR infants linked to maternal nutritional status [28].

Beyond the immediate perinatal period, the long-term consequences of SGA documented in the wider literature — developmental impairment [32], elevated neurologic risk among very preterm SGA infants at school age [33], persistent somatic and cognitive deficits into adulthood [34], cardiovascular programming [36], and a spectrum of metabolic and respiratory neonatal morbidities [37] — underscore the importance of the modifiable, region-specific risk factors identified in the present study for downstream prevention efforts.

Conclusion:

This study demonstrates that SGA at this tertiary care centre arises from a multifactorial interplay of maternal nutritional, haematological, and obstetric determinants together with placental morphometric insufficiency. Moderate maternal anaemia, inadequate gestational weight gain, a previous history of SGA delivery, a short interpregnancy interval, and irregular antenatal supplementation were the most robust maternal predictors, while reduced placental weight, volume, and thickness, along with other structural placental abnormalities, were the most informative placental correlates; the placental ratio, by contrast, was not a discriminating index in this cohort. The finding that over half of SGA neonates lacked antenatal ultrasonographic evidence of IUGR highlights an important gap in current screening protocols. Taken together, these results support the incorporation of routine antenatal anaemia screening, structured monitoring of gestational weight gain, and standardised gross placental examination at delivery into obstetric and neonatal care pathways in similar tertiary care settings, as low-cost, feasible measures to improve the identification and management of growth-restricted neonates.

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