International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Ravula PC et al. Int J Reprod Contracept Obstet Gynecol. 2026 Oct;15(10):4026-4033
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20263441

Original Research Article

Large-for-gestational-age births: maternal characteristics, risk factors and perinatal outcomes from a decade-long cohort

Pallavi Chandra Ravula[1]*, Vaishnavi Veloori[2]

[1]Department of Obstetrics, Fernandez Hospitals, Hyderabad, Telangana, India
[2]Fernandez Hospitals Education and Research Foundation, Hyderabad, Telangana, India

Received: 14 August 2026
Accepted: 11 September 2026

***Correspondence:**
Dr. Pallavi Chandra Ravula,
E-mail: drpallavi.r@fernandez.foundation

ABSTRACT

Background: Objectives were to describe the clinical profile and identify risk factors for large-for-gestational-age (LGA) births, defined by GROW customized birth-weight centile, comparing LGA with appropriately grown (AGA) births at a tertiary perinatal referral network in India.
Methods: This retrospective hospital-based study analysed 80,630 singleton births, live and stillborn, at 28 weeks gestation or more, to booked mothers between January 2013-December 2023, comprising 10,391 LGA and 70,239 AGA births. Multifetal pregnancies were excluded. Small-for-gestational-age births (<10th centile; n=9,842) were excluded and LGA were compared with AGA. Univariable and multivariable logistic regression identified independent risk factors.
Results: In this analysis 1 in 9 babies were LGA, and they were delivered by caesarean section far more often (62.7% vs 43.7%). LGA proportion was higher in diabetics than non-diabetic mothers (17.5% vs 11.2%). In the adjusted model (n=80,630; 10,391 events), independent risk factors for LGA were diabetes (adjusted odds ratio [aOR] 1.60), preterm birth (1.50), short stature (1.30), multiparity (1.16), assisted conception (1.12), obesity (1.09), advanced maternal age (1.08) and male baby (1.06). LGA birth was associated with higher odds of shoulder dystocia (aOR 4.54), birth injury (3.29), caesarean (2.07), neonatal hypoglycemia (1.46) and postpartum haemorrhage (1.29).
Conclusions: When contrasted with AGA, LGA was driven chiefly by maternal diabetes, with an association with preterm birth. The inverse association with hypertensive/placental disease persisted but was attenuated, indicating that part of the apparent "protective" effect seen against a non-LGA comparator is contributed by small-for-gestational-age births. LGA carries substantially higher caesarean rate and warrants anticipatory intrapartum planning.

Keywords: Large for gestational age, Fetal macrosomia, GROW customized centile, Gestational diabetes, India, Birth cohort

INTRODUCTION

Excessive fetal growth remains a common consequence of maternal obesity and diabetes, the prevalence of which continues to rise globally.[1,2] Two overlapping but distinct terms are used to describe it. Macrosomia is an absolute threshold, conventionally a birth weight of 4,000 g or, in some definitions, 4,500 or more applied without reference to gestation.[3,4] A baby is designated LGA when its birth weight lies above the 90th centile for its gestational age, so that a pre-term baby and a post-term baby are each judged against their own expected weight.[5]

The clinical case for identifying these babies is well established. Excessive fetal growth is associated with labour dystocia, shoulder dystocia and birth injury, higher rates of operative and caesarean delivery, postpartum haemorrhage and neonatal hypoglycaemia.[6,7] The consequences extend beyond the birth itself. Infants born large carry a predisposition to childhood obesity and

metabolic syndrome in later life, so that a single pregnancy exposure plausibly contributes to the intergenerational transmission of metabolic disease.[8] In India these considerations are not marginal. The pooled national prevalence of gestational diabetes mellitus is approximately 13% though estimates range from 4% to 19% depending on diagnostic criteria and whether sampling is hospital or community-based with wide regional variation, and diabetes in pregnancy is among the commonest comorbidities recorded in Indian tertiary obstetric practice.[9,10]

How largeness is defined therefore determines which babies are identified. As with LGA, population birth-weight standards misclassify constitutionally large but healthy babies while missing pathological overgrowth in babies of smaller mothers. A 3,600 g baby of a 150 cm mother and a 3,600 g baby of a 175 cm mother are not the same clinical event, yet a population centile treats them identically. This limitation is amplified in Asia, where maternal size and mean birth weight differ substantially from the populations in which conventional thresholds were derived, and where a single international standard performs poorly at the upper tail.[11] Customized centiles address this directly. The gestation-related optimal weight (GROW) model predicts an optimal weight for each pregnancy by adjusting for the physiological determinants of birth weight maternal height, booking weight, parity, ethnic origin and infant sex while excluding pathological variables such as diabetes, hypertension and smoking from the optimality calculation. Largeness is then judged against that individualized target rather than a population average, so that the LGA flag is enriched for metabolically driven rather than constitutional overgrowth.[12]

There is limited large-scale information on the profile and determinants of customized LGA from an Indian cohort of comparable size. We therefore analysed a large, decade-long, electronic-medical-record-based cohort from a tertiary perinatal network to describe the prevalence and clinical profile of customised-LGA births and to identify their independent risk factors.

METHODS

Study design, duration and approval

This retrospective observational hospital-based study analyzed births between January 2013 and December 2023 at a tertiary perinatal referral network in India. In accordance with standard hospital protocol, patients or their guardians completed a consent form for electronic data sharing at registration, and no identifiable patient parameters were used. The study adhered to the Declaration of Helsinki and was approved by the institutional ethics committee (EC Ref No 51-2026). Clinical information was recorded by trained obstetricians into a browser-based electronic medical records system using a standardized template.

Cases and comparison group

The records system was screened for all singleton births to booked mothers, both live and stillborn, with a gestational age at birth of 28 weeks or more and multifetal pregnancies were excluded. Within this base cohort, each birth was classified by its GROW customized birth-weight centile as LGA (≥90th), AGA (10th-<90th) or SGA (<10th). The present analysis compares the 10,391 LGA births with the 70,239 AGA births. The 9,842 SGA births were excluded so that LGA is contrasted with an AGA reference rather than with all non-LGA births.

Definition and data mining

LGA and AGA were defined from the customized-centile, which computes each baby's birth-weight centile after adjusting for maternal height, weight, parity and infant sex, so that the expected optimal weight is individualized to the mother. Maternal demographic details, systemic disorders and conception type, together with birth and neonatal outcomes, were extracted into a unified spreadsheet.

Statistical methods

Continuous variables were summarized as mean±SD and categorical variables as frequencies and percentages. Maternal and neonatal characteristics were compared between LGA and AGA births. The association of each candidate factor with LGA was first examined in univariable logistic regression (crude odds ratios), and all candidate factors were then entered simultaneously into a multivariable logistic regression model to estimate adjusted odds ratios (aORs) with 95% confidence intervals, fitted by maximum likelihood. An odds ratio above 1.0 indicates higher odds of LGA. Statistical significance was set at a two-sided $p<0.05$.

RESULTS

Prevalence and comparison groups

Within the cohort, 10,391 births were classified as LGA by the GROW customized centile (about one in nine of all classified births). These were compared with 70,239 AGA births, after excluding 9,842 SGA births. Because customization already accounts for maternal height, weight, parity and infant sex, this definition is enriched for metabolically driven rather than constitutional largeness.

Profile of LGA babies mothers

The 10,391 mothers of LGA babies were with a mean age of 28.7±4.1 years; 899 (8.7%) were aged 35 or more and 82 (0.8%) were teenaged. About 5,026 (48.4%), were multiparous. They had mean booking BMI of 27.0±5.1 kg/m²: 4,045 (38.9%) were overweight and 2,476 (23.8%) obese, while only 239 (2.3%) were underweight. The antenatal picture was dominated by diabetes in 3,829

(36.8%), 3,199 (30.8%) gestational and 630 (6.1%) pre-gestational. Hypertensive disorders were comparatively infrequent, affecting 1,147 (11.0%) overall, thyroid disorder (2,734 [26.3%]) and anemia (1,730 [16.6%]) were common, and 797 (7.7%) had conceived through assisted reproduction. Most pregnancies reached term: the mean gestation was 38.0±1.5 weeks, with 8,761 (84.3%) term, 1,226 (11.8%) preterm and 404 (3.9%) post-dated. Babies had a mean birth weight 3,536±394 g, with 984 (9.5%) macrosomic at 4,000 g or more and slightly more often male, 5,450 (52.4%). Delivery was frequently operative: 6,511 (62.7%) were by caesarean, 799 (7.7%) by assisted vaginal birth and 3,076 (29.6%) by spontaneous vaginal birth. The 1,275 (12.3%) babies were admitted to the neonatal unit, and stillbirth was at 23 (0.2%) (Table 1).

Comparison with AGA-maternal and neonatal profile

Relative to AGA, LGA babies were substantially heavier (mean birth weight 3,536 vs 2,965 g. They were delivered by caesarean considerably more often, 6,511 (62.7%) versus 30,711 (43.7%), and neonatal-unit admission was higher, 1,275 (12.3%) versus 6,902 (9.8%). Their mothers more often had diabetes (3,829 [36.8%] vs 18,014 [25.6%]), were multiparous (5,026 [48.4%] vs 30,830 [43.9%]), obese (2,476 [23.8%] vs 14,233 [20.3%]) or of advanced maternal age (899 [8.7%] vs 4,778 [6.8%]). Stillbirth was uncommon and similar in two groups, 23 (0.2%) versus 150 (0.2%). The comparison, with p values, (Table 2).

Univariable risk factors

In univariable analysis (Table 3), the strongest crude associations with LGA were diabetes (OR 1.69), preterm birth (OR 1.58), short stature (OR 1.37), advanced maternal age (OR 1.30), obesity (OR 1.23), multiparity (OR 1.20) and assisted conception (OR 1.18). Conversely, post-term birth (OR 0.53) and being underweight (OR 0.68) were associated with lower odds of LGA relative to AGA, and teenage pregnancy showed a similar inverse trend. Hypertension carried a modest crude association that did not persist on adjustment, while pre-eclampsia/eclampsia, anemia and connective-tissue disease were not significantly associated.

Independent risk factors

In the adjusted model (Table 4 and Figure 1), the strongest independent risk factor was diabetes (aOR 1.60 (1.53-1.67)), followed by preterm birth (1.50 (1.40-1.61)), short stature (1.30 (1.14-1.47)), multiparity (1.16 (1.11-1.21)), assisted conception (1.12 (1.04-1.22)), obesity (1.09 (1.04-1.15)), advanced maternal age (1.08 (1.00-1.17)) and male infant sex (1.06 (1.01-1.10)). Conversely, post-term birth (0.62 (0.55-0.68)), being underweight (0.77 (0.67-0.88)) and pre-eclampsia/eclampsia (0.72 (0.59-0.88)) were independently associated with lower odds of LGA. Among LGA and AGA births, the LGA proportion rose from 6,562/58,787 (11.2%) in non-diabetic to 3,829/21,843 (17.5%) in diabetic mothers (Figure 2), the diabetes-LGA relationship remaining the dominant factor after simultaneous adjustment for maternal build, parity, age and conception type.

Maternal and neonatal outcomes

LGA birth carried a distinctive pattern of mechanical and metabolic complications (Table 5). Caesarean section was far more common (6,511 [62.7%] vs 30,711 [43.7%]; aOR 2.07), and the size-related complications were markedly increased: shoulder dystocia (256 [2.5%] vs 383 [0.55%]; aOR 4.54), birth injury (36 [0.35%] vs 76 [0.11%]; aOR 3.29) and postpartum haemorrhage (900 [8.7%] vs 4,916 [7.0%]; aOR 1.29). Neonatal hypoglycaemia (201 [1.9%] vs 867 [1.2%]; aOR 1.46) and NICU admission (1,275 [12.3%] vs 6,902 [9.8%]; aOR 1.28) were also more frequent, while low Apgar scores among live births (51 [0.49%] vs 303 [0.43%]) and stillbirth (23 [0.22%] vs 150 [0.21%]; aOR 1.07) did not differ.

Table 1: Profile of LGA mothers and their babies.

Characteristics	LGA mothers, (n=10,391)
Maternal age, (in years), mean±SD	28.7±4.1
<20	82 (0.8%)
20-34	9,410 (90.6%)
≥35	899 (8.7%)
Primiparous	5,365 (51.6%)
Multiparous	5,026 (48.4%)
BMI kg/m²), mean±SD	27.0±5.1
Underweight (<18.5)	239 (2.3%)
Normal (18.5-24.9)	3,625 (34.9%)
Overweight (25-29.9)	4,045 (38.9%)
Obese (≥30)	2,476 (23.8%)
Short stature (<145 cm)	303 (2.9%)
Diabetes (any)	3,829 (36.8%)
Gestational (GDM)	3,199 (30.8%)
Pre-gestational (PGDM)	630 (6.1%)

Continued.

Characteristics	LGA mothers, (n=10,391)
Hypertensive disorder (any)	1,147 (11.0%)
Gestational hypertension	687 (6.6%)
Pre-eclampsia/eclampsia	130 (1.3%)
Thyroid disorder	2,734 (26.3%)
Anaemia	1,730 (16.6%)
Connective-tissue disease	57 (0.5%)
Assisted conception (ART)	797 (7.7%)
Gestation, weeks, mean±SD	38.0±1.5
Preterm (<37 week)	1,226 (11.8%)
Term (37-41 week)	8,761 (84.3%)
Post-dated (>41 week)	404 (3.9%)
Birth weight, g, mean±SD	3,536±394
Macrosomia (≥4000 g)	984 (9.5%)
Male infant	5,450 (52.4%)
Mode of delivery	
Spontaneous vaginal	3,076 (29.6%)
Assisted vaginal	799 (7.7%)
Caesarean	6,511 (62.7%)
NICU admission	1,275 (12.3%)
Stillbirth	23 (0.2%)

*Values are n (%) unless stated. Percentages are of the LGA group. LGA=Large for gestational age; GDM=Gestational diabetes mellitus; PGDM=Pre-gestational diabetes mellitus; NICU=Neonatal intensive care unit.

Table 2: Profile of LGA versus AGA births.

Characteristics	LGA	AGA	P value
Mean birth weight (g)	3,536	2,965	<0.001
Macrosomia ≥4000 g	9.5%	0.0%	<0.001
Stillbirth	0.22%	0.21%	0.873
NICU admission	12.3%	9.8%	<0.001
Caesarean delivery	62.7%	43.7%	<0.001
Multiparity	48.4%	43.9%	<0.001
Maternal age ≥35	8.7%	6.8%	<0.001
Teenage (<20)	0.8%	1.1%	0.009
Underweight (BMI<18.5)	2.3%	3.4%	<0.001
Obese (BMI≥30)	23.8%	20.3%	<0.001
Short stature (<145 cm)	2.9%	2.1%	<0.001
Hypertension	11.0%	10.0%	<0.001
Pre-eclampsia/eclampsia	1.3%	1.4%	0.401
Diabetes (any)	36.8%	25.6%	<0.001
Thyroid disorder	26.3%	25.4%	0.055
Anaemia	16.6%	16.8%	0.659
ART conception	7.7%	6.6%	<0.001
Connective-tissue disease	0.5%	0.6%	0.659
Male infant	52.4%	50.7%	0.001
Preterm birth (<37 week)	11.8%	7.8%	<0.001
Post-term birth	3.9%	7.1%	<0.001

*LGA 10,391; AGA 70,239; SGA (n=9,842) excluded. P values: Mann-Whitney U for birth weight; chi-square (or Fisher's exact where any expected cell <5, e.g. stillbirth) for categorical characteristics. Significant values (p<0.05) in bold. LGA= Large for gestational age; AGA=Average for gestational age; NICU=Neonatal intensive care unit; BMI=Body mass index; ART=Assisted reproductive techniques.

Table 3: Univariable associations with LGA (reference: AGA).

Factors	LGA/N	Rate	Crude OR (95% CI)
Multiparity	5,026/35,856	14.0%	1.20 (1.15-1.25)
Maternal age ≥35 years	899/5,677	15.8%	1.30 (1.20-1.40)
Teenage (<20)	82/832	9.9%	0.74 (0.59-0.93)
Underweight (BMI<18.5)	239/2,593	9.2%	0.68 (0.59-0.78)

Continued.

Factors	LGA/N	Rate	Crude OR (95% CI)
Obese (BMI≥30)	2,476/16,709	14.8%	1.23 (1.17-1.29)
Short stature (<145 cm)	303/1,806	16.8%	1.37 (1.21-1.56)
Hypertension	1,147/8,162	14.1%	1.12 (1.05-1.19)
Pre-eclampsia/eclampsia	130/1,080	12.0%	0.92 (0.77-1.11)
Diabetes (any)	3,829/21,843	17.5%	1.69 (1.62-1.77)
Thyroid disorder	2,734/20,598	13.3%	1.05 (1.00-1.10)
Anaemia	1,730/13,546	12.8%	0.99 (0.93-1.04)
ART conception	797/5,434	14.7%	1.18 (1.09-1.27)
Connective-tissue disease	57/467	12.2%	0.94 (0.71-1.24)
Male infant	5,450/41,086	13.3%	1.07 (1.03-1.12)
Preterm birth (<37 week)	1,226/6,706	18.3%	1.58 (1.48-1.69)
Post-term birth	404/5,409	7.5%	0.53 (0.48-0.58)

*LGA= Large for gestational age; AGA=Average for gestational age; ART=Assisted reproductive techniques

Table 4: Multivariable logistic regression-independent risk factors for LGA (reference: AGA).

Factors	aOR	95% CI	P value
Diabetes (any)	1.60	1.53-1.67	<0.001
Preterm birth (<37 week)	1.50	1.40-1.61	<0.001
Short stature (<145 cm)	1.30	1.14-1.47	<0.001
Multiparity	1.16	1.11-1.21	<0.001
ART conception	1.12	1.04-1.22	0.005
Obese (BMI≥30)	1.09	1.04-1.15	<0.001
Maternal age ≥35	1.08	1.00-1.17	0.047
Male infant	1.06	1.01-1.10	0.010
Hypertension	1.03	0.96-1.11	0.423
Anaemia	1.00	0.95-1.06	0.985
Thyroid disorder	0.98	0.94-1.03	0.434
Connective-tissue disease	0.86	0.65-1.14	0.310
Teenage (<20)	0.84	0.67-1.06	0.147
Underweight (BMI<18.5)	0.77	0.67-0.88	<0.001
Pre-eclampsia/eclampsia	0.72	0.59-0.88	0.001
Post-term birth	0.62	0.55-0.68	<0.001

*Adjusted n=80,630; 10,391 events; all factors entered simultaneously. Reference group: AGA. Connective-tissue disease defined as SLE, antiphospholipid syndrome or inflammatory arthritis; this grouping differs from the earlier all-non-LGA analysis and the estimate is imprecise. LGA=Large for gestational age; AGA=Average for gestational age; ART=Assisted reproductive techniques.

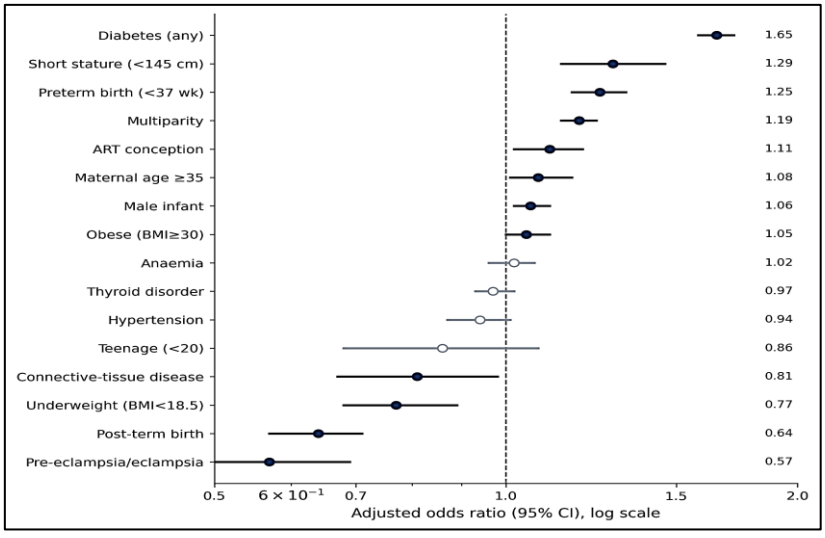

Figure 1: Adjusted odds ratios for LGA vs AGA (multivariable mode).

*Points=adjusted odds ratios; lines=95% CI; dashed line at OR=1. Filled markers p<0.05; hollow markers non-significant. Diabetes and preterm birth are the leading drivers; reference group is AGA births. LGA=Large for gestational age; AGA= Average for gestational age.

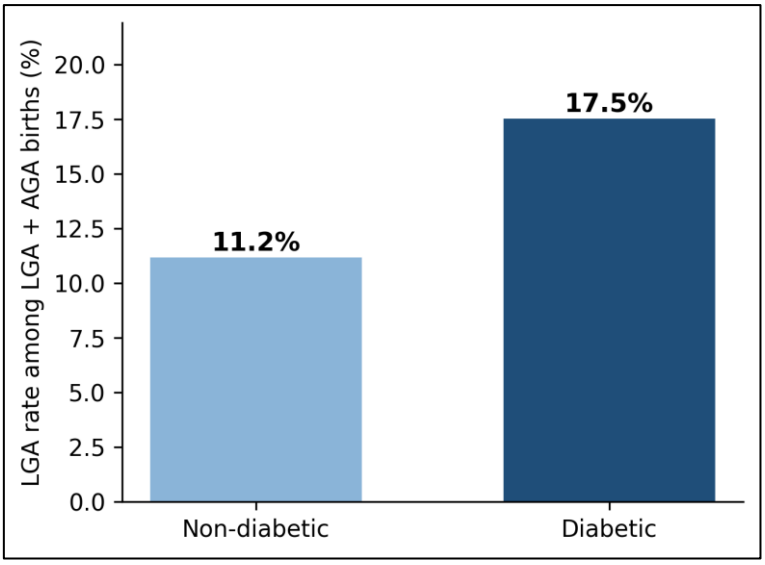

Figure 2: LGA proportion by maternal diabetes status (among LGA and AGA births).

*Among LGA and AGA births, the LGA proportion is higher in diabetic than non-diabetic mothers. LGA=Large for gestational age; AGA=Average for gestational age.

Table 5: Maternal and neonatal outcomes, LGA versus AGA.

Outcomes	LGA, N	AGA, N	aOR (95% CI)	P value
Caesarean section	6,511 (62.7%)	30,711 (43.7%)	2.07 (1.98-2.16)	<0.001
Postpartum haemorrhage	900 (8.7%)	4,916 (7.0%)	1.29 (1.20-1.39)	<0.001
Shoulder dystocia	256 (2.5%)	383 (0.55%)	4.54 (3.86-5.33)	<0.001
Birth injury	36 (0.35%)	76 (0.11%)	3.29 (2.20-4.91)	<0.001
Low Apgar score (live births)	51 (0.49%)	303 (0.43%)	1.21 (0.89-1.63)	0.220
Neonatal hypoglycaemia	201 (1.9%)	867 (1.2%)	1.46 (1.25-1.71)	<0.001
NICU admission	1,275 (12.3%)	6,902 (9.8%)	1.28 (1.20-1.36)	<0.001
Stillbirth	23 (0.22%)	150 (0.21%)	1.07 (0.69-1.66)	0.765

*Adjusted for maternal age, body mass index, parity and diabetes. Shoulder dystocia and birth injury are uncommon; p use Fisher's exact test where cells are sparse. Low Apgar is restricted to live births (10,368 LGA and 70,089 AGA), because every stillborn baby necessarily carries a low score and would otherwise duplicate stillbirth row. LGA=Large for gestational age; AGA=Average for gestational age.

DISCUSSION

In this decade-long cohort, 10,391 LGA births were compared with 70,239 AGA babies. In our cohort, customized LGA affected approximately one in nine singleton births (~11%), broadly comparable to the 9.4% reported in an earlier South Indian series and toward the upper end of the 4.3-22.1% range described for Asian populations, yet well above the 2.7% median for South Asia and below the 18.2% median of a recent 15-country analysis.[13-15] This spread reflects both genuine population differences and the growth standard applied, as our estimate derives from GROW centiles rather than the population-based references underlying most of these figures. Contrasting LGA with a cleanly normally grown reference sharpened two features that are blurred when all non-LGA births are used as the comparator: a materially stronger association with preterm birth, and an attenuated though still present, inverse association with hypertensive and placental disease.[16]

Diabetes was by far the dominant driver of customised LGA (aOR 1.60), consistent with the central role of maternal hyperglycaemia and fetal hyperinsulinaemia in accelerated fetal growth. A systematic review and meta-analysis of 156 studies including over 7.5 million pregnancies found that GDM was independently associated with an increased risk of LGA after adjustment for confounding factors. The adjusted odds of LGA were 1.57 among women managed without insulin and 1.61 in studies including insulin-treated women, confirming GDM as a significant independent predictor of excessive fetal growth.[16]

Obesity, short stature, multiparity, assisted conception, advanced maternal age and male sex were also independently associated, as expected for metabolically and constitutionally heavier babies. These metabolic associations were essentially unchanged from the all-non-LGA comparison, confirming their robustness to the choice of reference group.[12,13,16]

The association with preterm birth strengthened appreciably against an AGA reference (aOR 1.50, from 1.21 against all non-LGA). Van Zijl et al reported that LGA fetuses had a significantly higher risk of spontaneous preterm birth independent of maternal diabetes and other confounding factors. The proposed mechanism is uterine overdistension secondary to excessive fetal growth, which may trigger spontaneous labour or membrane rupture. This suggests that fetal overgrowth itself contributes to preterm birth risk and warrants closer surveillance.[18]

Because placental disease predisposes to growth restriction, a substantial part of the apparent "protective" effect of pre-eclampsia against LGA, when measured against all non-LGA births, is contributed by SGA babies in that comparator rather than by a direct relationship with normal growth.

LGA birth carried a distinctive pattern of mechanical and metabolic complications. Caesarean section was substantially more common (62.7% vs 43.7%; aOR 2.07), and the classic size-related complications were markedly increased shoulder dystocia more than four-fold (aOR 4.54) and birth injury more than three-fold (aOR 3.29) alongside a modest excess of postpartum haemorrhage (aOR 1.29). This pattern echoes systematic-review estimates of the maternal and neonatal complications of macrosomia, which pool increased risks of emergency caesarean, shoulder dystocia, obstetric brachial plexus injury and birth fracture. Although Beta et al demonstrated increased neonatal morbidity, including NICU admission, respiratory complications, birth trauma, and low Apgar scores in macrosomic infants, our study did not observe a significant increase in these outcomes among LGA neonates. This discrepancy may be explained by the use of a centile-based definition of LGA rather than absolute birth weight, early identification of fetal overgrowth, optimized obstetric management, and improved neonatal care in our tertiary referral network.[19]

Neonatal hypoglycemia (aOR 1.46), reflecting the fetal hyperinsulinism that accompanies maternal hyperglycemia, and neonatal-unit admission (aOR 1.28) were also increased, consistent with customized-centile analyses showing that large size for gestation identifies neonates at heightened risk of morbidity. Cartwright et al. reported that LGA infants had significantly higher neonatal morbidity, including increased rates of NICU admission, hypoglycemia, respiratory morbidity, and birth trauma, with risks increasing at higher birthweight centiles. These findings suggest that the degree of fetal overgrowth influences neonatal outcomes.[20]

Strengths and limitations

The strengths of this study include a very large cohort over a decade, structured electronic medical records, a customized growth standard that isolates metabolic from constitutional largeness, and a like-for-like AGA comparator. A like-for-like contrast against AGA confirms the metabolic drivers of overgrowth, reveals part of the hypertensive "protection" to be a comparator artefact, and unmasks a stronger link with preterm delivery a more valid basis for clinical inference. The data is single-network and retrospective and it may not represent the general population as the study site uses customized GROW charts for antenatal surveillance and birth centiles.

CONCLUSION

Compared with AGA, LGA in this large Indian cohort was driven chiefly by maternal diabetes and other metabolic factors and was more strongly associated with preterm birth than a non-LGA comparator. The inverse association with hypertensive/placental disease was attenuated once growth-restricted babies were excluded. Because LGA carries a substantially higher caesarean rate and a measurable excess of neonatal-unit admission, antenatal detection, optimization of glycaemic control and anticipatory intrapartum planning with attention to the timing of delivery, the most important routes to better outcomes.

ACKNOWLEDGEMENTS

Authors would like to thank to clinical team for their support and the Ms. Thankamani and Dr. Anthony Vipin Das for data collection and curation.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. McMurrugh K, Vieira MC, Sankaran S. Fetal macrosomia and large for gestational age. Obstet Gynaecol Reprod Med. 2024;34(3):66-72.
2. Kirakoya-Samadoulougou F, Ukwishaka J, Ngwasiri C, Subedi S, Hazel EA, Erchick DJ, et al. Prevalence of large-for-gestational age and macrosomia among livebirths in 23 low- and middle-income countries between 2000 and 2021: an individual participant data analysis. BJOG. 2025;132(8):S97-108.
3. American College of Obstetricians and Gynecologists. Macrosomia: ACOG Practice Bulletin No. 216. Obstet Gynecol. 2020;135(1):e18-35.
4. Damhuis SE, Ganzevoort W, Gordijn SJ. Abnormal fetal growth: small for gestational age, fetal growth restriction, large for gestational age: definitions and epidemiology. Obstet Gynecol Clin North Am. 2021;48(2):267-79.
5. Gardosi J, Francis A, Turner S, Williams M. Customized growth charts: rationale, validation and clinical benefits. Am J Obstet Gynecol. 2018;218(2):S609-18.
6. Beta J, Khan N, Khalil A, Fiolna M, Ramadan G, Akolekar R. Maternal and neonatal complications of fetal macrosomia: systematic review and meta-

analysis. Ultrasound Obstet Gynecol. 2019;54(3):308-18.
7. Robertson K, Vieira MC, Impey L. Perinatal outcome of fetuses predicted to be large-for-gestational age on universal third-trimester ultrasound in non-diabetic pregnancy. Ultrasound Obstet Gynecol. 2024;63(1):98-104.
8. Zhang Y, Liu P, Zhou W, Hu J, Cui L, Chen ZJ. Association of large for gestational age with cardiovascular metabolic risks: a systematic review and meta-analysis. Obesity (Silver Spring). 2023;31(5):1255-69.
9. Mantri N, Goel AD, Patel M, Baskaran P, Dutta G, Gupta MK, et al. National and regional prevalence of gestational diabetes mellitus in India: a systematic review and meta-analysis. BMC Public Health. 2024;24(1):527.
10. Ravula PC, Aziz N, Surapaneni T, Kolar G, Vavilala S, Fernandez E. Clinical profile, risk factor stratification and outcomes of the Fernandez birth cohort from India: a decade of experience. J Obstet Gynaecol India. 2025;75(5):422-9.
11. Pritchard N, Lindquist A, Hiscock R, Diksha P, Walker SP, Permezel M. Customised growth charts in large-for-gestational-age infants and the association with emergency caesarean section rate. Aust N Z J Obstet Gynaecol. 2019;59(3):380-6.
12. Gardosi J, Francis A, Turner S, Williams M. Customized growth charts: rationale, validation and clinical benefits. Am J Obstet Gynecol. 2018;218(2S):S609-18.
13. Jeyaseelan L, Yadav B, Silambarasan V, Vijayaselvi R, Jose R. Large for gestational age births among South Indian women: temporal trend and risk factors from 1996 to 2010. J Obstet Gynaecol India. 2016;66(1):42-50.
14. Harvey L, van Elburg R, van der Beek EM. Macrosomia and large for gestational age in Asia: one size does not fit all. J Obstet Gynaecol Res. 2021;47(6):1929-45.
15. Suárez-Idueta L, Ohuma EO, Chang CJ, Hazel EA, Yargawa J, Okwaraji YB, et al. Neonatal mortality risk of large-for-gestational-age and macrosomic live births in 15 countries, including 115.6 million nationwide linked records, 2000-2020. BJOG. 2025;132(8):S109-20.
16. Williams M, Turner S, Butler E, Gardosi J. Fetal growth surveillance-current guidelines, practices and challenges. Ultrasound. 2018;26(2):69-79.
17. Ye W, Luo C, Huang J, Li C, Liu Z, Liu F. Gestational diabetes mellitus and adverse pregnancy outcomes: systematic review and meta-analysis. BMJ. 2022;377:e067946.
18. van Zijl MD, Oudijk MA, Ravelli ACJ, Mol BWJ, Pajkrt E, Kazemier BM. Large-for-gestational-age fetuses have an increased risk for spontaneous preterm birth. J Perinatol. 2019;39(8):1050-6.
19. Beta J, Khan N, Khalil A, Fiolna M, Ramadan G, Akolekar R. Maternal and neonatal complications of fetal macrosomia: a systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2019;54(3):308-18.
20. Cartwright RD, Anderson NH, Sadler LC, Harding JE, McCowan LME, McKinlay CJD. Neonatal morbidity and small and large size for gestation: a comparison of birthweight centiles. J Perinatol. 2020;40(5):732-42.

Cite this article as: Ravula PC, Veloori V. Large-for-gestational-age births: maternal characteristics, risk factors and perinatal outcomes from a decade-long cohort. Int J Reprod Contracept Obstet Gynecol 2026;15:4026-33.