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Original Research Article

Maternal and perinatal outcomes in obese versus non-obese pregnant women at a tertiary care hospital in Western India: a prospective comparative study

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ABSTRACT

Background: Maternal obesity is increasing worldwide and in India and is associated with adverse pregnancy outcomes. This study compared maternal and perinatal outcomes in obese (body mass index ≥25 kg/m²) and non-obese (body mass index 18.5–24.9 kg/m²) pregnant women in a tertiary care hospital using Indian BMI cut-offs.

Methods: A prospective comparative analytical study was conducted in the Department of Obstetrics and Gynaecology, Krishna Institute of Medical Sciences, Karad, Maharashtra. A total of 204 women were enrolled and equally allocated to an obese group (n=102) and a non-obese group (n=102). Maternal outcomes included hypertensive disorders of pregnancy (HDP), gestational diabetes mellitus (GDM), mode of delivery, postpartum haemorrhage (PPH) and wound infection. Perinatal outcomes included preterm birth, birth weight, Apgar scores and neonatal intensive care unit (NICU) admission. Data were analysed using Chi-square test, Fisher's exact test and Student's t-test, with p<0.05 considered statistically significant.

Results: Obese women had higher rates of pregnancy-induced hypertension (25.5% vs 7.8%), eclampsia (3.9% vs 0%), GDM (29.4% vs 0%), lower-segment caesarean section (51.0% vs 37.3%), PPH (15.7% vs 3.9%) and preterm delivery (23.5% vs 11.8%). Neonates of obese mothers more often had macrosomia (17.6% vs 0%), low Apgar score at 1 minute (25.5% vs 13.7%) and 5 minutes (11.8% vs 3.9%), and NICU admission (41.2% vs 13.7%).

Conclusions: Maternal obesity defined by Indian BMI cut-offs is associated with markedly increased risks of GDM, HDP, operative delivery, PPH, macrosomia and NICU admission, supporting targeted preconception counselling and intensified antenatal surveillance in obese women.

Keywords: Body mass index, Caesarean section, Gestational diabetes mellitus, Hypertensive disorders of pregnancy, Maternal obesity, Perinatal outcomes

INTRODUCTION

Obesity has evolved from a local public health problem in affluent nations into a global pandemic affecting almost all countries, including those with a background of undernutrition such as India.[1] The prevalence of obesity among women of reproductive age is rising rapidly, and about one in six pregnant women worldwide is now classified as obese.[2] In South Asia this transition is particularly concerning: National Family Health Survey (NFHS-5, 2019–2021) data show that the combined prevalence of overweight and obesity (body mass index [BMI] ≥25 kg/m²) among Indian women aged 15-49 years increased from about 12.6% in 2005-06 to 24% in 2019-21.[3] Among urban Indian women of reproductive age, this burden exceeds 33%, with marked variation across states and socioeconomic groups.[4]

The adverse obstetric impact of maternal obesity is well documented. Obese pregnant women have higher risks of gestational diabetes mellitus (GDM), hypertensive disorders of pregnancy (HDP), dysfunctional labour, operative delivery, postpartum haemorrhage (PPH) and wound infection, and these risks are accentuated in low- and middle-income settings with constrained access to specialist care.[5-7] Infants of obese mothers are more likely to have macrosomia, preterm birth, low Apgar scores and a need for neonatal intensive care unit (NICU) admission.[8,9]

Multiple mechanisms link maternal obesity to these outcomes. Excess adipose tissue creates a chronic pro-inflammatory and insulin-resistant state, with altered adipokines and endothelial dysfunction, which amplifies the physiological insulin resistance of pregnancy and predisposes to GDM and hypertensive disorders.[10-13] Obesity-related myometrial dysfunction and altered prostaglandin signalling further increase the risk of labour dystocia, induction failure and caesarean section.[14]

An important consideration in India is the choice of BMI cut-offs. Asian populations, including Indians, develop metabolic complications at lower BMI values than Western populations.[15] National and regional guidelines therefore define overweight as BMI 23-24.9 kg/m^2 and obesity as BMI ≥25 kg/m^2.[16] However, many Indian obstetric studies still apply the World Health Organization general criterion of BMI ≥30 kg/m^2 for obesity, which underestimates both the true prevalence and the clinical burden of obesity in pregnancy.

Despite the scale of the problem, there is limited prospective, comparative evidence from Indian tertiary care hospitals particularly from semi-urban western India using Indian BMI thresholds and concurrently evaluating a broad range of maternal and perinatal outcomes.[17] This evidence is needed to inform local risk stratification, counselling and referral pathways. The present study was designed to prospectively compare maternal and perinatal outcomes in obese (BMI ≥25 kg/m^2) and non-obese (BMI 18.5-24.9 kg/m^2) pregnant women admitted for delivery at a tertiary care teaching hospital in western India, using Indian (Asian) BMI criteria to generate clinically applicable evidence for similar South Asian obstetric settings.

METHODS

Study design and setting

This prospective, comparative analytical study was conducted in the Department of Obstetrics and Gynaecology, Krishna Institute of Medical Sciences (KIMS), Karad, Maharashtra, India, a tertiary care teaching hospital affiliated to Krishna Vishwa Vidyapeeth (Deemed to be University), Karad. KIMS serves a large and diverse obstetric population from the Satara and Sangli districts of western Maharashtra. The study period was from 1st July 2024 to 30th June 2025. Ethical clearance was obtained from the Institutional Ethics Committee before initiation of the study, and written informed consent was taken from all participants.

Study population

Pregnant women admitted to the Department of Obstetrics and Gynaecology for delivery or attending antenatal care at the same institution during the study period were screened for eligibility. BMI was calculated as weight (kg) divided by height squared (m^2). Maternal weight was recorded at the first antenatal visit or from pre-pregnancy records when available, and height was measured using a calibrated stadiometer. Based on BMI, women were stratified into two groups: obese (BMI ≥25 kg/m^2; study group) and non-obese (BMI 18.5-24.9 kg/m^2; control group).

Inclusion criteria

Women were eligible if they were aged 18-40 years, had a singleton pregnancy, and belonged to either the obese group (BMI ≥25 kg/m^2) or the non-obese group (BMI 18.5–24.9 kg/m^2), and if they provided written informed consent and were available for follow-up until delivery.

Exclusion criteria

Women were excluded if they were aged <18 or >40 years, had BMI <18.5 kg/m^2, carried multiple gestations, were unwilling to participate, or were lost to follow-up before delivery.

Sample size

Sample size was calculated using data from Kutchi et al.[18] comparing perinatal outcomes in obese and non-obese pregnant women, applying the formula for comparison of two means: $n = (SD_o + SD_{no})^2 \times (Z_{1-\alpha/2} + Z_{1-\beta})^2/D^2$, where SD_o = 582.62 (standard deviation in the obese group) and SD_{no} = 425.04 (standard deviation in the non-obese group). With a 95% confidence level ($Z_{1-\alpha/2}$ = 1.96) and 80% power ($Z_{1-\beta}$ = 0.84), a minimum of 102 participants per group was required. Accordingly, 204 women were enrolled, with 102 in each group.

Study procedure and data collection

After enrolment and written informed consent, participants underwent structured assessment including sociodemographic details, obstetric history and anthropometric measurements. Maternal weight recorded at the first antenatal visit or from available pre-pregnancy records and measured height were used to calculate body mass index (BMI) as weight (kg)/height (m)^2. Based on Asian (including Indian) BMI criteria, women were classified into obese (BMI ≥25 kg/m^2) and non-obese (BMI 18.5-24.9 kg/m^2) groups.[1,16,18]

All participants were followed prospectively until delivery. Maternal outcomes assessed included hypertensive disorders of pregnancy, pre-eclampsia, eclampsia, gestational diabetes mellitus, postpartum haemorrhage, wound infection and mode of delivery.

Neonatal outcomes evaluated included gestational age at delivery (preterm: <37 completed weeks), birth weight category (low birth weight: <2500 g; normal birth weight: 2500–3999 g; macrosomia: ≥4000 g), Apgar scores at 1 and 5 minutes (low: <7; satisfactory: ≥7), congenital anomalies, intrauterine death and neonatal intensive care unit (NICU) admission.

Outcome measures

Primary outcome measures included: (1) obstetric complications (gestational diabetes mellitus, hypertensive disorders of pregnancy, postpartum haemorrhage and wound infection); (2) mode of delivery; (3) duration of hospital stay; and (4) immediate perinatal outcomes (intrauterine death, congenital anomalies, birth weight, gestational age, Apgar scores and NICU admissions). The secondary outcome was to generate region-specific clinical evidence regarding obesity-related obstetric risks using Indian BMI cut-offs to support antenatal risk stratification and counselling practices.

Statistical analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarised as mean ± standard deviation and compared between groups using Student's independent-samples t-test. Categorical variables were expressed as frequencies and percentages and compared using the Chi-square test; Fisher's exact test was used when expected cell counts were <5. A two-tailed p value <0.05 was considered statistically significant. Institutional Ethics Committee, Krishna Vishwa Vidyapeeth (Deemed to be University), Karad, Maharashtra, India. All procedures complied with institutional ethical standards and the Declaration of Helsinki.

RESULTS

Baseline characteristics and sociodemographic profile

The study enrolled 204 pregnant women; 102 (50.0%) were obese (BMI ≥25 kg/m^2) and 102 (50.0%) were non-obese (BMI 18.5-24.9 kg/m^2). Equal group allocation improved comparability and reduced group-size bias. Obese women were significantly older than non-obese women (mean age 29.92±6.58 vs 26.72±6.25 years; p<0.0001). Age distribution also differed, with a higher proportion of obese women in the 38-42-year group (21.57% vs. 5.88%) and more non-obese women in the 18-22-year group (32.35% vs. 16.67%). Sedentary occupation was more frequent among obese women (51.0% vs 15.7%), whereas manual labour was more common in non-obese women (51.0% vs 5.9%) (all p<0.0001). These data are summarised in Table 1.

Table 1: Baseline demographic and occupational characteristics of obese and non-obese pregnant women.

Variable	Non-obese (n=102)	Obese (n=102)	Test statistic	P value
Mean age (years±SD)	26.72±6.25	29.92±6.58	t = 3.58	<0.0001*
Age 18-22, N (%)	33 (32.35)	17 (16.67)	χ^2 = 15.84	0.003*
Age 23-27, N (%)	18 (17.65)	20 (19.61)		
Age 28-32, N (%)	33 (32.35)	33 (32.35)		
Age 33-37, N (%)	12 (11.76)	10 (9.80)		
Age 38-42, N (%)	6 (5.88)	22 (21.57)		
Sedentary work, N (%)	16 (15.7)	52 (51.0)	χ^2 = 59.47	<0.0001*
Manual labour, N (%)	52 (51.0)	6 (5.9)		
Homemaker/unemployed, N (%)	25 (24.5)	34 (33.3)		

n: number of participants; SD: Standard deviation; t: Student's t-test statistic; χ^2: Chi-square test statistic; *Statistically significant (p <0.05). Test statistics and p values for age and occupational distribution refer to overall group comparison

Table 2: Hypertensive disorders of pregnancy and gestational diabetes mellitus in obese and non-obese women.

Complication	Non-obese (n=102)	Obese (n=102)	Test statistic	P value
Pregnancy-induced hypertension, N (%)	8 (7.8)	26 (25.5)	χ^2 = 10.52	0.001*
Pre-eclampsia, N (%)	8 (7.8)	16 (15.7)	χ^2 = 3.12	0.077
Eclampsia, N (%)	0 (0)	4 (3.9)	Fisher's	0.043*
Gestational diabetes mellitus, N (%)	0 (0)	30 (29.4)	Fisher's	<0.0001*

n: number of participants; χ2: Chi-square test statistic; Fisher's: Fisher's exact test; *Statistically significant (p<0.05)

Hypertensive disorders of pregnancy and gestational diabetes mellitus

Hypertensive disorders of pregnancy were more common in the obese group. PIH occurred in 25.5% of obese women compared with 7.8% of non-obese women (p=0.001). Eclampsia was seen only in obese women (3.9% vs 0%; p=0.043). Pre-eclampsia was more frequent in obese women (15.7% vs 7.8%), although this difference was not statistically significant (p=0.077). GDM occurred exclusively in the obese group (29.4% vs 0%; p<0.0001). These findings are detailed in Table 2.

Mode of delivery and postpartum complications

Mode of delivery differed significantly between the groups (p=0.033). LSCS was more frequent in obese women (51.0% vs 37.3%), while normal vaginal delivery was less common (37.3% vs 53.9%). Rates of assisted delivery were similar (11.8% vs 8.8%). PPH occurred more often in obese women than in non-obese women (15.7% vs 3.9%; p=0.008). Wound infection was more common in the obese group (13.7% vs 5.9%), but this difference did not reach statistical significance (p=0.057). Mean duration of hospital stay did not differ significantly between groups. Table 3 presents these data.

Table 3: Mode of delivery and postpartum complications in obese and non-obese women.

Variable	Non-obese (n=102)	Obese (n=102)	Test statistic	P value
Normal vaginal delivery, N (%)	55 (53.9)	38 (37.3)	$\chi^2 = 6.82$	0.033*
Assisted/instrumental delivery, N (%)	9 (8.8)	12 (11.8)		
LSCS, N (%)	38 (37.3)	52 (51.0)		
Postpartum hemorrhage, N (%)	4 (3.9)	16 (15.7)	$\chi^2 = 7.14$	0.008*
Wound infection, N (%)	6 (5.9)	14 (13.7)	$\chi^2 = 3.62$	0.057
Hospital stay, days (Mean±SD)	5.44±1.87	5.93±1.97	t = 1.28	0.203

n: number of participants; LSCS: Lower Segment Caesarean Section; SD: Standard deviation; χ2: Chi-square test statistic; t: Student's t-test statistic; * Statistically significant (p <0.05). Mode of delivery test statistics and p-values refer to overall group comparison

Preterm delivery and birth weight

Preterm delivery was significantly more frequent among obese women (23.5% vs 11.8%; p=0.029). Birth weight distribution showed a significant association with maternal obesity (p<0.0001). Macrosomia (≥4000 g) was observed only among neonates of obese mothers (17.6% vs 0%), whereas the prevalence of low birth weight was similar in both groups (9.8% vs 11.8%). Intrauterine death and congenital anomalies occurred only in the obese group but were rare and not statistically significant. Table 4 summarises neonatal birth outcomes.

Table 4: Preterm delivery and birth weight distribution by maternal BMI category.

Neonatal outcome	Non-obese (n=102)	Obese (n=102)	Test statistic	P value
Preterm delivery (<37 weeks), N (%)	12 (11.8)	24 (23.5)	$\chi^2 = 4.76$	0.029*
Low birth weight (<2500 g), N (%)	12 (11.8)	10 (9.8)	$\chi^2 = 21.38$	<0.0001*
Normal birth weight (2500-3999 g), N (%)	90 (88.2)	74 (72.5)		
Macrosomia (≥4000 g), N (%)	0 (0)	18 (17.6)		
Intrauterine death, N (%)	0 (0)	2 (2.0)	Fisher's	0.497
Congenital anomaly, N (%)	0 (0)	4 (3.9)	Fisher's	0.495

n: number of participants; χ^2: Chi-square test statistic; Fisher's: Fisher's exact test; Low birth weight: birth weight <2500 g; Macrosomia: birth weight ≥4000 g; *Statistically significant (p<0.05). Birth weight distribution test statistics and p values refer to overall group comparison

Table 5: Neonatal condition at birth: Apgar scores and NICU admission by maternal BMI category.

Neonatal parameter	Non-obese (n=102)	Obese (n=102)	Test statistic	P value
Apgar <7 at 1 minute, N (%)	14 (13.7)	26 (25.5)	$\chi^2 = 4.51$	0.034*
Apgar <7 at 5 minutes, N (%)	4 (3.9)	12 (11.8)	$\chi^2 = 4.06$	0.044*
NICU admission, N (%)	14 (13.7)	42 (41.2)	$\chi^2 = 18.64$	<0.0001*

n: number of participants; χ^2: Chi-square test; NICU: Neonatal Intensive Care Unit; Apgar scores assessed at 1 and 5 minutes post-delivery; Low Apgar defined as <7 at 1 and 5 minutes; *Statistically significant (p <0.05)

Neonatal condition at birth and NICU admission

Neonates of obese mothers had poorer early adaptation at birth. Low Apgar score (<7) at 1 minute occurred in 25.5% of neonates in the obese group versus 13.7% in the non-obese group (p=0.034), and at 5 minutes in 11.8% versus 3.9% respectively (p=0.044). NICU admission was required in 41.2% of neonates born to obese mothers, compared with 13.7% in the non-obese group (p<0.0001). These outcomes are presented in Table 5.

DISCUSSION

This prospective comparative study using Indian (Asian) BMI cut-offs (BMI ≥25 kg/m^2) demonstrates that maternal obesity is associated with a broad spectrum of adverse outcomes, including significantly higher rates of gestational diabetes mellitus, hypertensive disorders of pregnancy, caesarean section, postpartum haemorrhage, preterm birth, macrosomia, low Apgar scores and NICU admission. These findings confirm that, even at BMI thresholds lower than the conventional WHO definition of obesity, obese pregnant women in an Indian tertiary care setting experience a substantial excess burden of obstetric and perinatal morbidity.

The higher mean age (29.92±6.58 vs. 26.72±6.25 years, $p<0.0001$) and predominance of sedentary occupations (51.0% vs. 15.7%, $p<0.0001$) in the obese cohort in this study mirror NFHS-based and other national data, which link older age, urban residence and higher socioeconomic status with obesity among Indian women.[3,4,19] This reinforces the importance of targeted screening and counselling for high-risk subgroups in antenatal clinics.

GDM occurred exclusively in obese women (29.4% vs. 0%, $p<0.0001$). This corroborates extensive literature demonstrating that obesity is among the most potent risk factors for GDM, mediated through adipokine-driven insulin resistance.[10-12] The pro-inflammatory state of obese adipose tissue, characterised by elevated Tumor Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6), and leptin, with reciprocally diminished adiponectin, amplifies the physiological insulin resistance of pregnancy beyond the adaptive threshold, resulting in overt hyperglycaemia.[11,20] A meta-analysis by Zhang et al in 2024 demonstrated that obese women face an approximately 3.5-fold elevated odds of GDM (OR 3.46; 95% CI 3.05-3.94) compared to normal-weight counterparts, directly mirroring the absolute risk difference of 29.4% observed in the present study.[14] These data support universal early GDM screening for women with BMI ≥25 kg/m^2 in Indian practice.

Similarly, the significantly higher rates of PIH (25.5% vs. 7.8%, $p=0.001$) and eclampsia (3.9% vs. 0%, $p=0.043$) among obese women reflect the established link between obesity, endothelial dysfunction and abnormal placentation, and are comparable to rates reported in the large retrospective cohort study from a Plovdiv tertiary centre, which reported the incidence of hypertensive disorders to be 26.64% in women with BMI ≥30 compared to 6.79% in those with BMI <30.[21,22] Crucially, our findings demonstrate that these risks are present even at the lower obese threshold (BMI ≥25 kg/m^2) employed in the current study, reaffirming the clinical utility of Indian BMI criteria for risk stratification. The exclusive occurrence of eclampsia in the obese group in this cohort, while based on small absolute numbers (n=4), underscores the potential severity of hypertensive sequelae in this population.

The elevated caesarean section rate in the obese group aligns with international and Indian studies that attribute this trend to combined effects of macrosomia, failed induction and anaesthetic complexity.[14,23] Gudipally et al (2022), using the LIFE cohort from India, demonstrated a three-fold increased risk of CS in obese women (OR 3.13; 95% CI 1.56-6.29).[24] The high operative delivery rate in the obese group has downstream implications for resource allocation, including blood bank availability, anaesthetic services, and skilled surgical teams, particularly in resource-constrained tertiary centres.

The increased risk of PPH observed here is consistent with previous reports of higher haemorrhage risk among obese women, particularly following caesarean delivery, likely mediated by macrosomia-related uterine overdistension impairing myometrial contractility, a higher burden of operative deliveries requiring larger uterine incisions, GDM-related placental abnormalities, and a generalised prothrombotic and pro-inflammatory state.[25-27]

From a perinatal perspective, the finding that macrosomia occurred exclusively in obese women (17.6% vs. 0%, $p<0.0001$) is clinically and metabolically significant. The hyperinsulinaemic foetal milieu created by maternal GDM, in which excess maternal glucose crosses the placenta and stimulates foetal insulin secretion, drives excessive fat deposition and organomegaly, producing macrosomia.[20,28] Macrosomia, in turn, amplifies the risk of birth trauma, shoulder dystocia, emergency CS, and PPH, thereby propagating a vicious cycle of mother-infant morbidity. The nearly doubled preterm delivery rate in obese women (23.5% vs. 11.8%, $p=0.029$) likely reflects medically indicated preterm deliveries due to GDM-related foetal compromise, worsening hypertensive disease, or foetal macrosomia.[29]

The highly significant elevation in NICU admission rates (41.2% vs. 13.7%, $p<0.0001$) represents one of the most consequential findings of this study, with direct implications for neonatal care resource planning. The mechanisms underpinning increased NICU admission in this cohort likely include neonatal metabolic derangements secondary to GDM (hypoglycaemia, polycythaemia), respiratory distress associated with preterm birth and macrosomia, and the sequelae of birth asphyxia reflected in the significantly lower Apgar scores at both 1 minute ($p=0.034$) and 5 minutes ($p=0.044$). These observations are consistent with data from Almutairi et al (2024), who found in a systematic review that neonates of obese mothers faced substantially elevated risks of NICU admission, respiratory distress syndrome, and neonatal mortality.[9]

The present study carries several implications for clinical practice. The application of Indian BMI thresholds (obesity: BMI ≥25 kg/m^2) captures a substantially larger and clinically high-risk obstetric sub-population than WHO global criteria (obesity: BMI ≥30 kg/m^2). It provides region-specific data from a semi-urban tertiary care hospital in western India, helping to fill a documented gap in the literature. The co-occurrence of multiple adverse outcomes, GDM, HDP, LSCS, PPH, macrosomia, and NICU admission, within the same obese cohort suggests that obesity functions as an upstream, multiplicative risk amplifier rather than a singular risk factor, necessitating intensive multidisciplinary antenatal surveillance.

At the same time, limitations must be acknowledged: the single-centre design may limit generalisability; the sample size, though adequate for major outcomes, may be underpowered for rare events such as eclampsia and intrauterine death; pre-pregnancy BMI was used for classification in cases where this data was available, but some participants' BMI was based on early antenatal weight, potentially introducing slight misclassification; and potential confounders such as parity, gestational weight gain, diet and pre-existing comorbidities were not fully adjusted for. Future multi-centre studies incorporating detailed metabolic profiling and longer-term maternal and child follow-up would strengthen the evidence base for policy and guideline development in India.

This prospective comparative study confirms that maternal obesity, defined using Indian (Asian) BMI thresholds (BMI ≥25 kg/m^2), is independently associated with a wide range of adverse maternal and perinatal outcomes, including GDM, PIH, eclampsia, increased LSCS rates, PPH, preterm birth, fetal macrosomia, low Apgar scores and substantially higher NICU admissions. Maternal BMI based on Indian cut-offs should therefore be incorporated into routine risk stratification and used to trigger intensified antenatal surveillance, early screening for GDM and HDP, and heightened intrapartum and neonatal vigilance. Pre-conception weight optimisation, lifestyle counselling and careful monitoring of gestational weight gain are essential components of strategies to reduce the growing obstetric and neonatal burden of maternal obesity in South Asian settings.

CONCLUSION

Maternal obesity defined by Indian BMI cut-offs is associated with markedly increased risks of GDM, HDP, operative delivery, PPH, macrosomia and NICU admission, supporting targeted preconception counselling and intensified antenatal surveillance in obese women.

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