Original Article

# Pregnancy Outcome among Obese Indians - A Prospective Cohort Study in a Tertiary Care Centre in South India

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## Abstract

**Background:** Obesity is a growing problem in the Asian subcontinent with Indians having increased propensity of developing obesity related complications like diabetes and hypertension later in life, notably at much lower levels of BMI than we have come to associate them with. The current study incorporates new Asian Indian guidelines for obesity in our pregnant women and assesses whether pregnancy complications are also similarly increased. There is a paucity of studies in this regard and hence the need for this study.

**Aims & Objectives:** To assess obesity related adverse outcome in pregnancy, in labour and immediate effects on the newborn using new Asian Indian guidelines for obesity.

**Materials & Methods:** 199 pregnant women attending JIPMER antenatal outpatient department at less than 16 wks of gestation were enrolled. They were subdivided into 3 groups: 99 in non-obese (BMI < 25 Kg/m$^2$), 81 as obese (BMI: 25-34.9 Kg/m$^2$), and 19 as morbidly obese (BMI ≥ 35 Kg/m$^2$). Hypertensive disorders in pregnancy and GDM were main outcomes while total LSCS, instrumental deliveries, induction rates, macrosomia, PPH, shoulder dystocia, birth asphyxia and wound sepsis were also studied.

**Results:** There was a significant increase in gestational diabetes among morbidly obese (26%) and obese (17%) compared to non-obese (4%)(p = 0.0023). Significant association of hypertensive disorders (p < 0.0001) was seen with obesity with significantly increased odds of among obese (OR: 3.6) and morbidly obese (OR: 13.9). There was a significant increase in LSCS as well as instrumental deliveries. Macrosomia, PPH, birth asphyxia and puerperal wound sepsis were also significantly higher among obese.

**Conclusion:** Obesity defined by Asian Indian guidelines (BMI ≥ 25 Kg/m$^2$) is associated with adverse outcomes in pregnancy, in labour and on the fetus at odds comparable to western studies with obesity taken as BMI ≥ 30 Kg/m$^2$.

**Keywords:** BMI, Diabetes, Hypertension, Obesity

## INTRODUCTION

Obesity, the silent epidemic worldwide has reached a stage where approximately 2.3 billion adults will be overweight and more than 700 million adults will be obese by 2015, as projected by WHO.[1] National Family Health Surveys in India indicated an increase in obesity from 10.6% in 1998-1999 to 14.8% in 2005-06.

Recent studies have shown that Asian Indians have more pre-disposition for truncal obesity as opposed to generalised obesity with increased amount of subcutaneous and intra-abdominal adipose tissue deposition as well as certain ectopic sites like in muscle and liver. Deurenberg-Yap et al.[2] determined absolute and relative risks for at least one cardiovascular risk factor (elevated triglycerides/hypertension/diabetes mellitus) for various categories of BMI and waist-hip ratio (WHR). Absolute risks for cardiovascular complications are high, ranging from 41 to 81% at low categories of BMI (22-24 kg/m$^2$) and WHR (0.80-0.85)(Odds ratio: 1.97-4.38). Interestingly these complications appeared in categories of BMI and

WHR well below the cut-off values of BMI and WHR recommended by WHO. South Asians settled overseas also were at increased risk of insulin resistance and cardiovascular complications than white Caucasians matched for BMI.[3] Hence experts met in New Delhi in 2008 to develop Asian Indian specific guidelines for defining and managing obesity.[4,5] In proceedings of 9th International conference on obesity in Sao Paolo as well, this need of developing ethnicity related cut-offs for obesity based on BMI was accepted.

**Currently recommended cut-offs of BMI by WHO[1]**

Normal : 18.5-24.9 kg/m$^2$
Overweight : 25.0-29.9 kg/m$^2$
Obesity : ≥30 kg/m$^2$

**Consensus meeting statement based on various studies all over India[6]**

Normal BMI : 18.5-22.9 kg/m$^2$
Overweight : 23.0-24.9 kg/m$^2$
Obesity in Indians : ≥25 kg/m$^2$

Gestational diabetes mellitus is a major complication in pregnancy associated with obesity. About 17% of obese women show GDM in pregnancy compared to 1-3% women in normal BMI.[7] Hypertensive disorders of pregnancy are significantly higher among obese women. O'Brien et al.[8] demonstrated that the risk of pre-eclampsia is typically doubled with every 5-7 kg/m$^2$ increase in prepregnancy BMI. According to RCOG obesity is the most important risk factor for thrombo-embolism in pregnancy. Risks of APH chiefly placental abruption are also increased with obesity. Obesity also increases chances of multifetal gestation and preterm labour.[9]

Sebire et al.[9] showed that the caesarean section rate for obese women was over 20% compared to 10% for normal weight women. Shoulder dystocia and maternal injuries are increased with obesity.[10] Usha Kiran[11] found that women with BMI ≥ 30 kg/m$^2$ were at increased risk of post dated pregnancy and induction of labour. Postpartum complications including PPH and lactational dysfunction are also increased.

Vricella et al[12] reported significant risks of anaesthesia complications in obesity (insufficient duration of regional anaesthesia, increased conversions to general anaesthesia, need of additional I.V analgesia, increased rates of post-dural puncture headache, high spinal and profound intra-op hypotension.

Congenital anomalies mainly neural tube defects[13] and congenital heart diseases[14] are increased with obesity in pregnancy even after excluding chances of anomalies with simultaneous diabetes. Hyperinsulinemia is the main risk factor along with elevated uric acid, endogenous estrogen and triglycerides.[15]

## MATERIALS AND METHODS

This was a prospective cohort study undertaken in the department of Obstetrics & Gynaecology in JIPMER, Pondicherry - a tertiary care hospital in southern India for a period of two years. There were two exposure groups of pregnant women: one with BMI < 25 kg/m$^2$ and second with BMI > 25 kg/m$^2$ matched for gestational age and parity. It was approved by institutional ethics committee. Informed consent was obtained from all the participants of the study.

### Sample size

The sample size was estimated using the standard formula by Limeshow et al (1990). To calculate the sample size, hypertensive disorders in pregnancy was considered as the main variable and the relative risk was taken as 1.92 in BMI > 25 kg/m$^2$ group as compared to < 25 kg/m$^2$.[16] The sample size was estimated with 80% power at 5% level of significance. The minimum sample size required for this study was estimated as 80 in each group.

Pregnant women up to 16 wks of gestation on first visit to JIPMER antenatal outpatient were enrolled over a period of 1 year from October 2010 to September 2011.Women with pre-existing hypertension, diabetes and thrombophilias were excluded. Multiparous obese women undergoing caesarean section in present pregnancy due to doubtful previous LSCS scar integrity were also not included under total caesarean section parameter of pregnancy outcome.

Hypertensive disorders in pregnancy and GDM were primary outcomes studied while instrumental deliveries, total caesarean sections, multiple gestation, preterm labour, APH, abortions and stillbirths, maternal injuries and shoulder dystocia during labour were secondary outcomes. Congenital anomalies, macrosomia in newborn was also assessed. Puerperal sepsis and venous thrombo-embolism if any was assessed.

### Brief procedure

BMI was calculated in their first visit (wt in kg/htin m$^2$) along with their blood pressure. The women attended prenatal care every month until 28 weeks of pregnancy, fortnightly henceforth till 36 weeks and weekly thereafter till delivery. Gestational diabetes was estimated by 50 gm GCT at 24-28 weeks followed by GTT as per NDDG guidelines if 1 hr GCT value exceeded 140 mg/dl. Antepartum haemorrhage was defined as bleeding from or

into the genital tract after 28 weeks of gestation. Preterm delivery was defined as delivery prior to 37 completed weeks. Macrosomia was defined by birth weight 4000 gms or greater. Low APGAR score was considered by a score less than 5 at 1 minute. Follow-up was done till delivery & labour and perinatal outcomes assessed till postnatal day 7.

Chi-square test was used to compare the categorical data between the groups. The Kolmogorov-Smirnov Test was used to test the distribution of continuous variables and accordingly appropriate parametric (Independent Students t test) or non parametric (Mann Whitney U test) test was used for comparing the continuous variables. Odds ratios were calculated to approximate relative risks of BMI on adverse outcomes. All statistical analyses were done at 95% confidence interval and the P value of <0.05 was considered as statistically significant.

## RESULTS

A total of 199 women were included with 99 fulfilling the inclusion criteria in BMI <25 kg/m$^2$ non-obese group. One hundred women were enrolled in ≥25 kg/m$^2$ group as obese. Thirty six women were lost to follow up and were excluded. Obese women were further subdivided as follows (Figure 1):

- 81 patients with BMI between 25-34.9 Kg/m$^2$ (obese)
- 19 patients with BMI ≥ 35 Kg/m$^2$ (morbidly obese)

The mean age (23.49 ± 3.9 in non-obese and 24.86 ± 3.9 in obese) and parity in both the groups was similar.

The overall incidence of gestational diabetes (GDM) among the study subjects was 11.5% and GDM increased with increase in BMI (P < 0.05). The incidence of GDM among morbidly obese (26%) and obese group (17%) was significantly higher with an OR of 8.5 and 5 respectively when compared with normal group (Table 1). The overall incidence of hypertensive disorders was 38% and it was significantly associated with increasing BMI (Figure 2). The Odds Ratio (OR) for the hypertensive disorders among the study subjects in morbidly obese and obese in comparison with normal BMI subjects was 13.9 and 3.6 respectively.

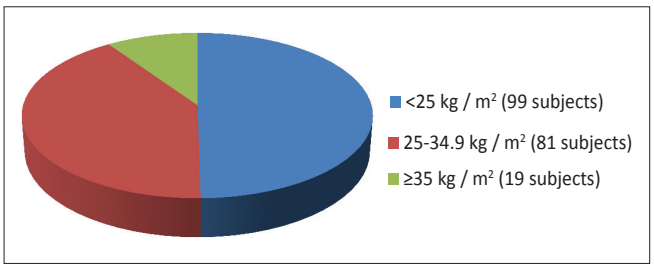


**Figure 1: Distribution of study subjects**

The incidence of gestational hypertension (GHT) and pre-eclampsia/eclampsia among study subjects is 28.4% and 18% and it is significantly higher in obese compared to normal subjects (Table 2). The rate of IUGR (Table 3) was higher in morbid obese (21%) when compared with obese (8.65%) and normal group (8.1%). The OR of the IUGR in morbid obese was 3.03 when compared to the normal and the OR in obese group was 1.08. The abortion rate among the study subjects was 2% and the abortion rates were comparable between the BMI groups (Table 3) as were multiple gestation, APH and preterm deliveries (Table 4). 37% were induced and it increased with increase in BMI (OR: 1.3 and 3.9) with statistical significance seen with morbid obesity.

27% of all underwent LSCS and the proportion of LSCS significantly increased with increase in BMI (Table 4). Of all the women undergoing LSCS, 78% underwent emergency LSCS (Table 5). Emergency LSCS was particularly increased with increasing BMI (OR: 2.6 and 7.6). Cephalopelvic disproportion was the predominant cause of elective LSCS among obese women while fetal distress (72%) and failed induction of labour (22%) main causes for emergency LSCS. The overall instrumental delivery rate was 18.9%. The rates in morbid obese (37.5%) and obese (27.1%) was significantly higher than in normal group (11%)(P < 0.05)(Table 4).

The incidence of maternal injuries was 4%. The incidence of macrosomia in morbidly obese group was 10.53% which was significantly higher when compared to normal group (1.02%) but it was not significantly different in comparison with obese group (2.47%). The mean birth weight among obese women was 2840 grams compared to 2630 grams among non-obese. There were no cases of shoulder dystocia in morbidly obese group with caesarean section being done for the macrosomic fetuses in that group which accounts for the non-significant association between obesity and shoulder dystocia (Table 3). Overall incidence of PPH was 6.5% and it significantly increased with increasing BMI (OR: 3.2 and 22.4 with obesity and morbid obesity)

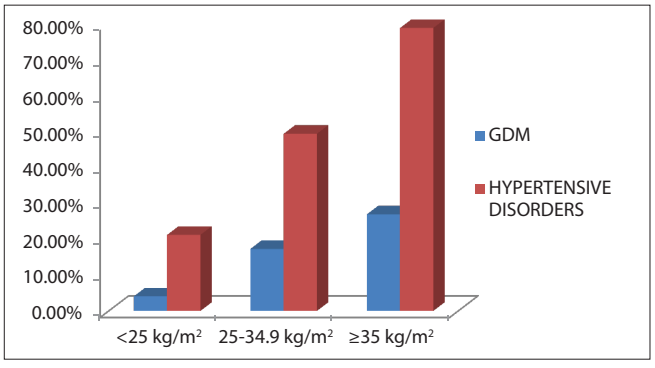


**Figure 2: Distribution of GDM and hypertensive disorders among study groups**

**Table 1: Obesity, GDM and hypertensive disorders**

| Groups | GDM present | OR (95% CI) | Hypertensive disorders | OR (95% CI) |
|---|---|---|---|---|
| <25 kg/m² | 4 (4.05%) | Reference | 21 (21.2%) | Reference |
| 25-34.9 kg/m² | 14 (17.29%) | 5 (1.564-15.75) | 40 (49.4%) | 3.6 (1.89-6.94) |
| ≥35 kg/m² | 5 (26.92%) | 8.5 (2.63-35.44) | 15 (78.9%) | 13.9 (4.12-46.43) |

**Table 2: Gestational hypertension and pre-eclampsia in different groups**

| BMI category | GHT (%) [a] | OR (95% CI) | PE/Eclampsia(%)[b] | OR (95% CI) | Total |
|---|---|---|---|---|---|
| <25 kg/m² | 15 (16.1%) | | 6 (7.14%) | | 99 |
| 25-34.9 kg/m² | 24 (36.9%) | 2.36 (1.14-4.88) | 16 (28.1%) | 5.07 (1.84-13.95) | 81 |
| ≥35 kg/m² | 10 (71.4%) | 6.22 (2.12-17.87) | 5 (55.55%) | 16.25 (3.43-76.07) | 19 |
| Total | 49 (28.4%) | | 27 (18%) | | 199 |

a-excluding pre-eclampsia/eclampsia, b-excluding gestational hypertension

**Table 3: Fetal complications and obesity**

| Fetal complications | BMI categories | Total (%) | OR (95%C I) |
|---|---|---|---|
| IUGR | <25 Kg/m² | 8 (8.1%) | |
| | 25-34.9 kg/m² | 7 (8.65%) | 1.08 (0.37-3.11) |
| | ≥35 kg/m² | 4 (21.1%) | 3.03 (0.81-11.34) |
| Abortions | <25 Kg/m² | 3 (3%) | |
| | 25-34.9 kg/m² | 1 (1%) | |
| | ≥35 kg/m² | 0 | |
| Perinatal mortality | <25 Kg/m² | 7 (7.1%) | |
| | 25-34.9 kg/m² | 7 (8.65%) | |
| | ≥35 kg/m² | 2 (10.53%) | |
| Congenital malformations | <25 Kg/m² | 1 | |
| | 25-34.9 kg/m² | 3 | |
| | ≥35 kg/m² | 1 | |
| Macrosomia | <25 Kg/m² | 1 (1.02%) | |
| | 25-34.9 kg/m² | 2 (2.47%) | 2.48 (0.22-27.89) |
| | ≥35 kg/m² | 2 (10.53%) | 11.5 (0.99-134.4) |
| Shoulder dystocia[c] | <25 Kg/m² | 1 (1.2%) | |
| | 25-34.9 kg/m² | 4 (6.78%) | |
| | ≥35 kg/m² | 0 | |
| Birth asphyxia | <25 Kg/m² | 15 (15.1%) | |
| | 25-34.9 kg/m² | 19 (23.4%) | 1.74 (0.81-3.64) |
| | ≥35 kg/m² | 8 (42.1%) | 4.07 (1.41-11.8) |

c-percentage calculated after excluding LSCS

**Table 4: Other maternal complications and obesity**

| Maternal complications | BMI categories | Total (%) | OR (95% CI) |
|---|---|---|---|
| LSCS[x] | <25 Kg/m² | 13 (14.6%) | Reference |
| | 25-34.9 kg/m² | 22 (33.8%) | 2.99 (1.37-6.5) |
| | ≥35 kg/m² | 11 (68.7%) | 12.86 (3.84-43.13) |
| APH | <25 Kg/m² | 2 (2%) | |
| | 25-34.9 kg/m² | 2 (2.47%) | |
| | ≥35 kg/m² | 2 (10.53%) | |
| Multiple gestation | <25 Kg/m² | 2 (2.03%) | |
| | 25-34.9 kg/m² | 7 (8.65%) | |
| | ≥35 kg/m² | 1 (5.27%) | |
| Preterm delivery | <25 Kg/m² | 12 (12.13%) | |
| | 25-34.9 kg/m² | 14 (17.29%) | 1.5 (0.66-3.49) |
| | ≥35 kg/m² | 3 (15.79%) | 1.4 (0.34-5.37) |
| Induction of labour[a] | <25 Kg/m² | 31 (31.6%) | |
| | 25-34.9 kg/m² | 30 (38.9%) | 1.3 (0.69-2.4) |
| | ≥35 kg/m² | 9 (64.2%) | 3.94 (0.93-18.6) |
| Instrumental deliveries[b] | <25 Kg/m² | 10 (11.6%) | |
| | 25-34.9 kg/m² | 16 (27.11%) | 2.83 (1.98-6.78) |
| | ≥35 kg/m² | 3 (37.5%) | 4.56 (0.94-22.06) |
| Maternal injuries | <25 Kg/m² | 3 (3%) | |
| | 25-34.9 kg/m² | 2 (2.5%) | |
| | ≥35 kg/m² | 3 (15.3%) | |
| PPH | <25 Kg/m² | 2 (2.1%) | |
| | 25-34.9 kg/m² | 5 (6.2%) | 3.2 (0.6-16.9) |
| | ≥35 kg/m² | 6 (31.6%) | 22.4 (4.08-122.8) |
| Wound sepsis | <25 Kg/m² | 2 (2%) | |
| | 25-34.9 kg/m² | 5 (6.2%) | 3.2 (0.60-16.9) |
| | ≥35 kg/m² | 3 (15.8%) | 9.1 (1.41-58.78) |

x-excluding instrumental deliveries, a-percentages and odds calculated after excluding elective LSCS in each BMI groups, b-percentages and odds calculated after excluding LSCS from BMI groups

(Table 4). Birth asphyxia was also significantly increased with morbid obesity (OR: 4.7). Perinatal mortality rates were comparable in the three groups. While no significant association was found between the congenital anomalies and obesity, the sample size was also small for the purpose. Likewise there was not a single case with venous thrombo-embolism among the study subjects.

The overall prevalence of wound sepsis (including post LSCS wound discharge and episiotomy wound gape) was 5%. It increased with obesity (OR: 3.2) significantly among the morbidly obese (OR: 9.1).

## DISCUSSION

Risk of GDM was observed with obesity (OR: 5) with odds rising to eight fold with morbid obesity similar to other studies.[17] Along with hyperinsulinemia, maternal obesity is associated with hyperlipidemia, which enhances oxidative stress with decreased prostacyclin and more peroxide production, resulting in vasoconstriction and platelet aggregation, which increases the risk of hypertensive disorders of pregnancy. With a fourfold increase in hypertensive disorders, this study estimated a significant increase in gestational hypertension and pre-eclampsia similar to other studies.[16,18] Increase in risk of antepartum haemorrhage was noticed in morbidly obese women, chiefly due to placental abruption. Cedergren[18] however

**Table 5: LSCS in BMI groups**

| Groups | Elective LSCS(%) x | OR (95% C I) | Emergency LSCS(%) y | OR (95% C I) | Total |
|---|---|---|---|---|---|
| <25 kg/m² | 1 (1.3%) | | 12 (13.6%) | | 13 |
| 25-34.9 kg/m² | 4 (7%) | 5.74 (0.62-52.8) | 18 (29.5%) | 2.65 (1.16-6.02) | 22 |
| ≥35 kg/m² | 5 (50%) | 76 (7.4-781.5) | 6 (54.5%) | 7.6 (2.02-28.86) | 11 |
| Total | 10 | | 36 | | 46 |

x-excluding instrumental deliveries and emergency LSCS, y-excluding instrumental deliveries and elective LSCS

could not find any increased risk for placental abruption in his study with much larger numbers of morbidly obese women. Placenta previa too was less frequent in other studies.[19,20]

Odds for IUGR was increased but no significant association of multiple gestation with obesity (p: 0.13); a trend also noticed in other studies.[21] There was no increased risk of preterm labour which was an outcome similar to other studies with a small sample size. Bodnar had demonstrated increased risk of spontaneous and idiopathic preterm births with weight loss in class 1 and 2 obese women.[22] Hendler[23] had predicted fewer total and spontaneous preterm births with maternal prepregnancy BMI ≥ 30 Kg/m².

Morbidly obese women were more likely to undergo induction of labour (OR: 3.9). High rates of induction for postdatism and oligohydramnios even in non-obese women might account for the non-significant association with obesity (OR: 1.3 in obese compared to non-obese). Usha Kiran[11] however had found that women with BMI ≥ 30kg/m² were at increased risk for postdated pregnancy (OR: 1.4) and hence induction of labour (OR: 1.6).

Obesity was a significant risk factor for both elective and emergency c-sections with odds increased manifold with morbid obesity. Fyfe et al.[24] reported significant risk of prelabour c-sections (p = 0.02) as well as increased rates of caesarean delivery in first stage (OR: 2.89) among obese. They reported similar rates of second stage c-sections among both obese and non-obese.

Rode et al.[25] had demonstrated a fivefold increase in odds of instrumental deliveries among obese similar to this study. Sahu et al.[20] had reported significantly higher rates of macrosomia among morbidly obese women (p = 0.02). Sheiner et al.[26] felt that after having adjusted for diabetes mellitus, no significant association was found between macrosomia and obesity alone. Besides, Catalano[27] had already demonstrated that GDM can be a confounding factor in a study between macrosomia and obesity. In our study there was a significant association between macrosomia and morbid obesity (OR: 2.48 and 11.5 in obese and morbidly obese respectively). Inspite of the higher rates of macrosomia, there was no significant increase in shoulder dystocia as most of the obese mothers with macrosomia underwent LSCS. There was also no significant increase noted with perineal tears, c-section angle extensions and other maternal injuries among obese in our study (p > 0.05).

Postpartum haemorrhage (atonic) rates were significantly raised with obesity in our study. Prolonged duration of labour, increased instrumental deliveries and c-sections were directly related to atonicity. Sebire et al[9] reported a 44% increased risk of PPH with BMI > 30 Kg/m². Cedergren felt this increase in PPH may be due to increased surface area of placental implantation associated with LGA babies while Nuthalapathy[21] felt it could be due to large volume of distribution and decreased bio-availability of uterotonic agents in obese women. Birth asphyxia with or without convulsions was significantly seen among newborns of obese women in this study (p = 0.02). No significant association with perinatal mortality was however noted. Cedergren[18] demonstrated an increased risk for meconium aspiration and birth asphyxia among newborns of obese (OR: 2.85 and 2.52 respectively).

Wound sepsis rates were significantly raised (p = 0.03) in morbidly obese women. Alanis et al.[28] had demonstrated higher risks of post caesarean wound gape, discharge and seroma formation among the morbidly obese. There was no maternal mortality in the present study. With limited puerperal follow-up, no cases of venous thromboses were seen in any of the groups.

## CONCLUSION

Pregnancy complications related to obesity is a growing problem with complications arising at a BMI ≥ 25 Kg/m² at rates comparable to western definition of obesity (BMI ≥ 30 Kg/m²). This study reflects the need of new Indian guidelines of weight restriction to be taken more seriously and a larger, prospective trial taking ethnic differences into consideration, is the need of the hour. The small sample size and short span of this study as well as the selection bias associated with a tertiary care institute are limitations. Division of obese women into obese (BMI 25-34.9 Kg/m²) and morbidly obese (≥35 kg/m²) was a bit arbitrary considering lack of categorical definition in new Asian Indian guidelines, however was necessary for

better interpretation of results and was similar to other studies.[16,29]

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