# Risk Factors of Obesity on Maternal and Perinatal Outcomes among Pregnant Women

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**Abstract:** Obesity constitutes a major risk factor for both maternal and fetal complications for mothers includes, hypertensive disorders, gestational diabetes, preeclampsia, eclampsia and higher frequency of cesarean section and fetal complications includes intrauterine death, low Apgar score and macrosomia. This study aimed to assess risk factors of obesity on maternal and perinatal outcomes among pregnant women. Prospective cohort study, the study subjects consisted of four groups; 27 women with normal BMI (group I: BMI 19.1–25 kg/m2), 25 overweight (group II: BMI 25.1–30 kg/m2), 73 obese (group III: BMI 30.1–40 kg/m2) and 25 morbidly obese women (group IV: BMI >40 kg/m2). The study was conducted in obstetric department, labor ward at zagazig university hospital. Women in group III and IV were characterized by higher systolic booking blood pressure and higher diastolic booking blood pressures, with mean differences being 27.9,14.2 respectively. Similar differences were found in booking diastolic blood pressures with mean difference being 12.13 and 1.83 respectively. Women in group III and IV are at increased risk at gestational diabetes with OR 2.72; and OR 10.1. Requirement of instrumental deliveries or lower segment caesarean section occurred more often in group II, III and IV with 2.07, 2.58 and 6.61 respectively. The birth weight in group III and IV are significantly higher with mean differences 1.54 and 1.72. Macrosomia occurred more often in all increased BMI groups, but significance was only found in women of group IV with OR 4.4. The present study had revealed that the obesity during pregnancy represents a major risk for adverse outcome for pregnant women. Maternal weight measurements need to be recorded during antenatal care and used consistently because BMI is obviously a risk factor for maternal complications during pregnancy, labor and postpartum period.
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**Key words:** Risk factors, Obesity, maternal, perinatal, outcome.

## 1. Introduction

Obesity is defined as a condition of excessive body fat, it is usual assessed clinically by body mass index (BMI), obtained by dividing weight by height squared. The World Health Organization (WHO) defines normal weight as a BMI of ‹24.9-18.5 overweight as a BMI of ‹29.9-25 and obesity as a BMI of 30 or greater. Obesity is further characterized by BMI into class I ‹(34.9 -30) class II ‹(39.9-35) and class III (greater than 40**) (World Health Organization, 2000)** the prevalence of obesity in pregnancy ranges from 1.8 to 1.%25.3 Obesity beforehand during pregnancy constitutes a major risk factor for both maternal and fetal complications **(Thadhani *et al.*, 1999**)

Maternal obesity, usually defined on the basis of pre-pregnancy BMI, is associated with a higher incidence of many pregnancy complications **(Birdsall *et al.*, 2009)**. As pregnancy progresses, this index are influenced by gestational weight gain in lean tissues, thus limiting its use in pregnancy. An alternative, the use of pre-pregnancy BMI as an indicator of obesity in pregnancy, may be complicated by the fact that the weight used for this calculation is frequently self-reported, producing inaccuracies **(Nucci *et al.*, 2001)**.

Maternal obesity carries significant risks for both mother and fetus, is considered an obstetrical risk factor leading to high frequency of complications during prenatal period, and increases the risk of several adverse outcomes of pregnancy. Therefore, there is a substantial need for the development of preventive actions **(Baeten *et al.*, 2001 & Kumari 2001)**. There are many criteria for definition of obesity in pregnancy and body mass index is one of mostly commonly used **(Baeten *et al*, 2001 & Guelinckx *et al.*, 2008)**. Many recent publications used pre pregnancy BMI as a risk factor for pregnancy and labor complications **(Bianco *et al.*, 1998 & Jensen *et al.*, 1999)**.

High pre-pregnancy BMI is associated with adverse obstetric outcome. Maternal complications include early miscarriage, pregnancy induced hypertension and preeclampsia, gestational diabetes, thrombo-embolic disease, infections, sleep apnea, prolonged labour, increased risk of interventions like induction of labour and operative delivery, shoulder dystocia and postpartum hemorrhage. Perinatal complications include birth defects (mainly neural tube defects), macrosomia, in-utero growth

restriction, still births, preterm birth and need for intensive care admission **(Bilal *et al.*, 2005 & Satpathy *et al.*, 2008 & Callaway *et al*, 2006)**.

The risk of developing hypertensive disorder and preeclampsia has been shown to increase according to levels of pre-pregnancy BMI **(Thadhani *et al.*, 1999)**. Obesity and diabetes play independent roles in determining fetal size. Women with gestational diabetes and normal body weight who control glycemia with diet, insulin or anti-diabetic drugs present an incidence of neonatal macrosomia comparable to that of women without diabetes. In addition, insulin treatment prevented macrosomia in overweight and obese women. On the other hand, in obese women on diet who managed to control glycemia, as well as in overweight and obese **(Leikin *et al*, 1987)**.

Obese women even with normal glucose tolerance have a two-fold higher risk of giving birth to macrosomic babies since both condition are independently correlated to macrosomia. Given that the incidence of obesity is approximately ten-fold that of gestational diabetes, it is evident that maternal lifestyle exerts a great influence on the incidence of fetal macrosomia. Once again, there seems to be a quantitative relationship between maternal BMI and the risk of delivering a macrosomic/LGA neonate **(Ehrenberg *et al.*, 2004 & Beaten *et al.*, 2001)**.Women who failed to do so, irrespective of treatment, the incidence of macrosomia remained high. Macrosomia, as well as maternal heighten and weight, gestational age and number of prior deliveries, are considered reliable predictors of the risk of obstetrical events, such as shoulder dystocia and injury of the branchial plexus **(Yogev and Catalano, 2009)**.

Neonates born to obese mothers are at increased risk of admission to neonatal intensive care unit **(Heslehurst *et al.*, 2008)**. They are at increased risk of meconium aspiration and shoulder dystocia **(Cedergren, 2004)**. In addition, there is an association between maternal obesity and neonatal hypoglycaemia, jaundice and respiratory distress **(Callaway *et al.*, 2006)**. The relationship between maternal obesity and early neonatal death is unclear, with three studies confirming a relationship, and another showing a relationship between maternal obesity and early neonatal death only in primiparous mothers **(Kristensen *et al..*, 2005 & Galal 2002 & Weiss *et al.*, 2003)**.

**Aim of the study**

To assess risk factors of obesity on maternal and perinatal outcomes among pregnant women.

**Research question**

- What is the impact of obesity on maternal outcomes during pregnancy?
- What is the risk factors of obesity on fetal?

## 2. Subjects and methods

**Design and Setting** :

The study was a prospective cohort study; data were obtained by a detailed review of medical case notes of all women giving birth to singleton babies in the Augusts 2010 to the end of December 2010. This study was carried out at Department of Obstetric and Gynecology, labor ward at Zagazig University Hospital.

**Subjects:**

The study involved 150 Women attending for labor in Obstetric and Gynecologic Department during Augusts 2010 to the end of December 2010. The sample classified into four gropes (group I: 27 women with normal weight, group II: 25women overweight, group III: 73women obese, and group IV: 25women morbidly obese).

**Inclusion criteria**

Criteria for selection included:-

- Normal pregnancy with no pathological conditions
- Prime gravid and multi gravid.
- Any gestational age

**Excluded criteria**

Women excluded from the study who were complaining from:-

- Cardiac disease and respiratory diseases.
- Fetal with congenital malformation
- Multiple pregnancy
- Ante partum hemorrhage

**Methods of data collection:**

Patient assessment sheet was designed and used to collect the relevant data including:-

**A**-The socio demographic characteristic of the pregnant women were: age, parity, weight, height, body mass index (BMI) as Pregnant women were analyzed in the following four body mass index (BMI) categories (kg/m2) calculated at their first prenatal visit: group I: normal (19.1–25 kg/m2), group II: overweight (25.1–30 kg/m2), group III: obese (30.1–40 kg/m2) and group IV: morbidly obese (≥ 40 kg/m2).

**B-Medical and obstetric history:**

1-Complication in previous pregnancy as:- Diabetes mellitus, chronic hypertension.

2-Complication in the current pregnancy as: - Gestational diabetes (GD), hypertension, and mode of delivery.

-For the classification of hypertensive conditions

associated with pregnancy the following definitions were used: chronic hypertension, GH, pre-eclampsia both using ASSHP and ISSHP criteria, superimposed pre-eclampsia, severe pre-eclampsia, HELLP syndrome and eclampsia.

- In this current study we used the following definitions:

**Chronic hypertension:** Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on two consecutive measurements in the first half of the pregnancy.

**Gestational hypertension (GH):** Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on two consecutive measurements in the second half of pregnancy.

**Pre-eclampsia according to ASSHP:** Increase in blood pressure to at least 140/90 mmHg after the 20th week of gestation in previously normotensive women combined with proteinuria, and/or intrauterine growth restriction (IUGR) and/or elevated liver enzymes and/or thrombocytopenia.

**Pre-eclampsia according to ISSHP:** Increase in blood pressure to at least 140/90 mmHg after the 20th week of gestation in a previously normotensive woman, combined with proteinuria.

**Superimposed pre-eclampsia:** De novo proteinuria developing for the first time in the second half of pregnancy in women with chronic hypertension.

**Severe Pre-eclampsia:** Pre-clampsia according to ISSHP and MgSO4 given.

**HELLP syndrome:** Hemolysis, Elevated Liver Enzymes, Low Platelets.

**Eclampsia:** The occurrence of seizures and unconsciousness in a pregnant woman.

**Pregnancy-induced hypertensive disorder:** Gestational hypertension, pre-eclampsia, HELLP syndrome or eclampsia.

**2-Neonatal assessment sheet**

Variables of birth weight, apgar scores, and the neonatal complications were also determined.

**Main outcome measures**

Outcome variables included: booking demographics, booking blood pressures, glucose challenge test, hypertensive complications, pre-existing and gestational diabetes, instrumental deliveries, caesarean deliveries, birth weights, Apgar scores, and neonatal complications.

**Ethical consideration**

Data were collected from pregnant women without taking information such as names or civil registration numbers. As no intervention would occur, only oral patient's consent was taken.

**Pilot study:**

It was carried out on 10% of the sample (12 women) to clarify the validity and reliability of the questionnaire and then modification was done.

**Statistic analysis:**

The obtained data were coded, analyzed and tabulated, descriptive statistics as frequency and percentages were calculated using computer. Differences in characteristics between cases and controls were compared by either t-tests when variables are continuous and results are expressed as P- value and the mean difference with the corresponding 95% Confidence Interval, considering P50.05 statistically significant. Chi-squared tests were used for categorical variables, results were expressed as Chi-square value and Odds Ratio (OR) with the corresponding 95% Confidence Interval, considering P50.05 statistically significant. Statistics were performed by using SPSS version 15.0 and InStat version 3.

## 3. Results

The differences in booking characteristics between the four subgroups are presented in **Table 1.** Besides the 27 with normal BMI, 25 overweight, 73 obese and 25 morbidly obese women were identified. Booking details showed group IV to be significantly older with a mean difference of 7.3 (95% CI: 4.83–9.81) year. No significance was found in gravidity and parity. Booking systolic blood pressure was significantly higher in group III and IV with mean differences being 27.9 (95% CI: 19.3–36.5), 14.2 (95% CI: 7.24–21.19) respectively. Similar differences were found in booking diastolic blood pressures with mean difference being 12.13 (95% CI: 5.5–18.7), 1.83 (95% CI: 3.5–7.16) respectively. As BMI increases, pre-existing morbidity rate rises, reaching statistical significance in group IV (OR 4.22; 95% CI: 1.15–16.1). Looking at pre-existing morbidity in the combined group II– III shows an OR of 1.34 (95% CI: 0.36-5), 2.19(95% CI: 0.78-6.28) Not surprisingly, there was also a significant difference in the use of medication with OR 9.5 (95% CI: 2.26–43.1) for group IV and there was no significant difference in the use of medication with OR 1.86 (95% CI: 0.7–5.04) for the combined II–IV group

Are presented in **Table 2.** The hypertension (>140/90 mmHg) didn't occur more often in the overweight group, but was significantly more frequent in group III and IV with OR 3.5 (95% CI: 1.02-13.7) and 5.31 (95% CI: 1.22–24.8). With regard to hypertensive especially GH that occurred more often in group IV (OR 1.44; 95% CI: 1.28-7.6), whereas the frequency of pre-eclampsia (both using ASSHP and ISSHP definitions) is higher in group III

and IV, but these differences were not significant. In women without pre-existing hypertension, morbidly obese were also at a significantly higher risk of developing GH (OR 3.69; 95% CI: 1.56–8.76).The frequency of all diabetes was significantly higher in group III and IV with OR 6.57 (95% CI: 1.44–29.89) and 16.33 (95% CI: 3.58–74.49).The prevalence of gestational diabetes was significantly higher in group III and IV (OR 2.72;95% CI: 1.31–61.6 and OR 10.1; 95% CI: 1.1–238.6), primarily explained by a markedly higher prevalence of gestational diabetes requiring insulin treatment.

Data concerning the intrapartum period are presented in **Tables 3 and 4.** A spontaneous onset of labour occurred mostly in the control group, statistical significance was found in group III and IV, with ORs of respectively 2.58 (95% CI: 1.0-7.4) and 4.22 (95% CI: 1.15–16.16) for having spontaneous onset of labour. Induction of labour (IOL) was significantly more common in group IV (OR 2.75; 95% CI: 1.71-10.96). This difference appeared to be primarily explained by the more common use of oxytocin. Analyzing the mode of delivery [normal versus instrumental or lower segment caesarean section (LSCS)] showed a significantly higher risk of requiring an instrumental delivery or LSCS, respectively odds ratios of 2.07 (95% CI: 1.49-9.1), 2.58 (95% CI: 1.2–8.85) and 6.61 (95% CI: 1.62-28.7) for group II, III, IV. In multiparous women with previous vaginal birth, significance with regard to requiring instrumental delivery or LSCS was only found in groups III and IV group III OR 3.42 (95% CI: 1.71–18.3) IV OR 2.82 (95% CI: 1.88-9.31). Dystocia was the main reason to proceed to an elective LSCS in group IV. It occurred often in all BMI groups but significance was only in women of group IV with OR 40.0; 95% CI: 1.37-66.41). In comparison to the control group, III but not group IV had a significantly prolonged second stage; mean difference between group I and III being 15 min.

**Table (5)** descriptive the prenatal outcomes of pregnancy between studied groups. The Mean birth weights increased with rising BMIs with mean (SD) values of respectively 3.00(0.33), 3.46 (0.62), 3.53 (0.54) and 3.7 (0.78), being only statistically significant higher in the obese and morbidly obese groups (mean difference 1.54; 95% CI: 0.28-0.79 and 1.72; 95% CI: 1.4-0.99). Macrosomia occurred more often in all increased BMI groups, but significance was only found in women of group IV with OR 4.4 (95% CI: 1.10-16.5) Regarding the Apgar scores, a significant difference was found for 1 min Apgar score in group III; an Apgar score<5 after 1 min occurred more frequently in this group (OR 8.73; 95% CI: 1.71-66.62), whereas the other BMI groups did not have an additional risk.

## 4. Discussion

Obesity is a global epidemic now and that the prevalence of overweight and obesity is increasing worldwide at an alarming rate affecting both developed and developing countries **Tawfik *et al*. (2003)**. The prevalence of obesity in adults is very high in Egypt, particularly among women, and that the prevalence of diabetes and hypertension parallels that of obesity **Galal (2002)**. In the present study, the morbidity obese women have a significantly older than the other groups with a mean difference of 7.3 (95% CI: 4.83–9.81) year. No significance was found in gravidity and parity. This was supported by **Callaway *et al.* (2006)** who stated that the mean age of cases in our study was higher than controls, which suggests that women gain weight with age, and difference in parity was not significant, as we had excluded grand multigravidae.

The results of this study are broadly consistent with previous studies about maternal obesity and the occurrence of a big range of adverse outcomes, it has been reported that women categorized as obese and morbidity obese are at an increased risk of pre-eclampsia and gestational hypertension (GH) during their pregnancy compared with women who are of normal weight. this agreement with **Weiss *et al.* (2003) and Kumari (2001)** who found that a majority of studies have described an increased risk for gestational diabetes, gestational hypertension and preeclampsia associated with obesity. Moreover, **National High Blood Pressure Education Program (2004)** who stated that the hypertensive disorders of pregnancy, preeclampsia, and gestational hypertension, which complicate 6 to 8% of pregnancies, are leading causes of maternal and fetal morbidity and mortality. This finding in the same line with **OBrien *et al.* (2003)** who mentioned that during pregnancy, obese women face increased risk of developing hypertension, pre-eclampsia and gestational diabetes. Specifically, women with a BMI >30 kg/m2 have a two- to three-fold higher risk for developing pre-eclampsia, while this risk doubles for an increase in BMI prior to pregnancy by 5-7 kg/m2. On the other hand, **Sattar and Greer (2002)** who stressed that obesity and a previous pregnancy complicated by pre-eclampsia constitute the main risk factors for developing severe pre-eclampsia in the current pregnancy. Pre-eclampsia is also associated with an increased risk for coronary heart disease in later life.

**Table (1) Distributions of age, BMI, clinical data, pregnancy, preexisting morbidity and medication among pregnant women**

| Variables | BMI group I (N=27) | | BMI group II (N=25) | | BMI group III (N=73) | | BMI group IV (N=25) | |
|---|---|---|---|---|---|---|---|---|
| **Age (years)** | 21.52±1.99 | | 23.92±4.75 | | 25.1±5.17* | | 28.8±4.3* | |
| **Length (m)** | 1.61 ± .035 | | 1.62 ± .067 | | 1.60 ± .064 | | 1.54 ± .062 | |
| **Weight (kilo's)** | 61.35±1.93 | | 76.88± 8.27 | | 88.22 ± 9.36 | | 106.6 ± 9.13 | |
| **BMI** | 23.5±1.13 | | 28.59 ± 1.22 | | 34.30 ± 2.58 | | 44.1 ± 3.1 | |
| **Booking BP systolic (mmHg)** | 99 ± 11.8 | | **112.8 ±16.9*** | | **113.2 ±15.3*** | | **127.0 ± 18.6*** | |
| **Booking BP diastolic (mmHg)** | 76.6 ± 7.34 | | **76.4 ±13.8*** | | **78.4 ±12.1*** | | **88.8 ±13.6*** | |
| **Gravidity** | 1.67 ± 0.92 | | 1.34 ±1.3 | | 1.07 ± 1.1 | | 1.56 ±1.39 | |
| **Parity** | 1.33 ± 0.48 | | 1.56 ± 1.0 | | 1.64 ± 0.58 | | 1.96±1.1 | |
| **Pre-existing morbidity** | No | % | No | % | No | % | No | % |
| **None** | 19 | 70.4% | 16 | 64.0% | 38 | **52.1%** | 9 | **36.0%*** |
| **Depression** | 0 | 0% | 3 | 12.0% | 6 | 8.2% | 1 | 4.0% |
| **Cardiovascular** | 0 | 0% | 2 | 8.0% | 10 | 13.7% | 6 | 24.0% |
| **Diabetes** | 0 | 0% | 2 | 8.0% | 7 | 9.6% | 0 | 0% |
| **Epilepsy** | 0 | 0% | 0 | 0% | 0 | 0% | 1 | 4.0% |
| **Asthma** | 7 | 25.9% | 1 | 4.0% | 6 | 8.2% | 4 | 16.0% |
| **Other/combination** | 1 | 3.7% | 1 | 4.0% | 6 | 8.2% | 4 | 16.0% |
| **Medication** | | | | | | | | |
| • **None** | 19 | 70.4% | 17 | 68.0% | 47 | **64.4%** | 5 | **20.0%*** |
| • **Antidepressant** | 0 | 0% | 5 | 20.0% | 6 | 8.2% | 8 | 32.0% |
| • **Antihypertensive** | 0 | 0% | 2 | 8.0% | 7 | 9.5% | 6 | 24.0% |
| • **Antidiabetes** | 0 | 0% | 1 | 4.0% | 1 | 1.4% | 1 | 4.0% |
| • **Antiepileptics** | 0 | 0% | 0 | 0% | 0 | 0% | 1 | 4.0% |
| • **Antiasthma** | 7 | 25.9% | 0 | 0% | 4 | 5.5% | 0 | 0% |
| • **Other/combination** | 1 | 3.7% | 0 | 0% | 8 | 11.0% | 4 | 16.0% |

BMI group I: 19.1–25 kg/m2; BMI group II: 25.1–30 kg/m2; BMI group III: 30.1–40 kg/m2; BMI group IV > 40 kg/m2; Data are presented as percentages and mean (SD); BP, blood pressure. *Indicates a significant difference. Bold letter type indicates significant differences after combining subgroups versus reference group.

**Table (2) Distributions of hypertensive disorders and diabetes among pregnant women**

| Variables | BMI group I (N= 27 | | BMI group II (N=25) | | BMI group III (N=73 | | BMI group IV (N=25) | |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % |
| **Hypertension** | | | | | | | | |
| • **No** | 13 | 52.0% | 45 | 61.6% | 20 | 80.0% | 23 | 85.2% |
| • **Yes** | 12 | **48.0%*** | 28 | 38.4%* | 5 | 20.0% | 4 | 14.8% |
| Chronic Hypertension | 0 | 0% | 0 | 0% | 0 | 0% | 0 | 0% |
| Gestational Hypertension | 3 | **12.0%*** | 8 | 11% | 1 | 4.0% | 0 | 0% |
| Preeclampsia ASSHP | 4 | 16.0% | 8 | 11% | 2 | 8.0% | 2 | 7.4% |
| Preeclampsia ISSHP | 3 | **12.0%** | 10 | **13.7%** | 2 | 8.0% | 2 | 7.4% |
| Severe preeclampsia | 1 | 4.0% | 0 | 0% | 0 | 0% | 0 | 0% |
| HELLP | 0 | 0% | 2 | 2.7% | 0 | 0% | 0 | 0% |
| Eclampsia | 1 | 4.0% | 0 | 0% | 0 | 0% | 0 | 0% |
| Diabetes | | | | | | | | |
| No | **72.0%*** | 18 | 98.0%* | 65 | 92.0% | 23 | 92.6% | 25 |
| yes | 28.0% | 7 | 11% | 8 | 8.0% | 2 | 7.4% | 2 |
| Pre-existent diabetes | 4.0% | 1 | 1.4% | 1 | 4.0% | 1 | 3.7% | 1 |
| GD Diet | 8.0% | 2 | 2.7% | 2 | 4.0% | 1 | 0% | 0 |
| GD Insulin | 20.0% | 5 | 6.8% | 5 | 0% | 0 | 3.7% | 1 |
| TGDM | 28.0% | 7 | 9.6% | 7 | 4.0% | 1 | 3.7% | 1 |
| GCT | 16% | 4 | 16.4% | 12 | 12% | 3 | 11.1% | 3 |

BMI group I: 19.1–25 kg/m2; BMI group II: 25.1–30 kg/m2; BMI group III: 30.1–40 kg/m2; BMI group IV > 40 kg/m2; GH, gestational hypertension; BP, blood pressure; PIHD, pregnancy induced hypertensive disorder; GDM, gestational diabetes mellitus; *Indicates a significance difference. Bold letter type indicates significant differences after combining subgroups versus reference group.

**Table (3) Distribution of mode of delivery among pregnant women**

| Variables | BMI group I (N= 27) | | BMI group II (N=25) | | BMI group III (N=73) | | BMI group IV (N=25) | |
|---|---|---|---|---|---|---|---|---|
| Gestational Age | 37.71 ± 3.37 | | 37.88 ± 2.85 | | 38.34 ± 2.95 | | 37.47 ± 2.59 | |
| Gestational <37 | 0 | 0% | 7 | 28% | 13 | 17.8% | 6 | 24% |
| Gestational >41 | 1 | 3.7% | 3 | 12% | 8 | 11% | 1 | 4% |
| First stage(min) | 980.29 ±594.65 | | 902.40 ± 835.23 | | 721.99 ±518.51 | | 629.33 ±465.72 | |
| Second stage(min) | 19.8 ±7.07 | | 19.40 ±15.29 | | 34.38* ±23.61 | | 22.2 ±20.0 | |
| First + second stage nulliparous | 1368.64±128.66 | | 1114.72±900.84 | | 851.43±629.30 | | 465.00±701.48 | |
| First + second stage multiparous | 197.50 ±112.41 | | 432.85 ±270.59 | | 580.32 ±311.25 | | 694.58 ±444.33 | |
| Second>1hrs | 0 | 0% | 1 | 4% | 7 | 9.5% | 2 | 8% |
| Second>2hrs | 0 | 0% | 1 | 4% | 1 | 1.4% | 1 | 4% |
| **Onset of labor** | | | | | | | | |
| Spontaneous | 19 | 70.4% | 13 | 52% | 35 | 48%* | 9 | **36%*** |
| IOL | 6 | 22.2% | 7 | 28% | 23 | 31.5% | 11 | **44%*** |
| Elective LSCS | 2 | 7.4% | 5 | 20% | 15 | 20.5% | 5 | 20% |
| **IOL** | | | | | | | | |
| Prostaglandin | 3 | 11.1% | 2 | 8% | 11 | 15.0% | 4 | 16% |
| Oxytocin | 1 | 3.7% | 1 | 4% | 2 | 2.6% | 2 | 8% |
| ARM | 2 | 7.4% | 3 | 12% | 4 | 5.4% | 2 | 8% |
| Combination | 0 | 0% | 3 | 12% | 6 | 8.2% | 3 | 12% |
| Other | 0 | 0% | 1 | 4% | 1 | 1.3% | 0 | 0% |
| **Augmentation** | | | | | | | | |
| Prostaglandin | 0 | 0% | 0 | 0% | 1 | 1.4% | 7 | 28% |
| Oxytocin | 2 | 7.4% | 2 | 8% | 8 | 11% | 3 | 12% |
| ARM | 4 | 14.8% | 3 | 12% | 10 | 13.7% | 4 | 16% |
| Combination | 0 | 0% | 2 | 8% | 4 | 5.5% | 3 | 12% |
| Other | 0 | 0% | 1 | 4% | 0 | 0% | 0 | 0% |
| **Mode of delivery** | | | | | | | | |
| Normal | 22 | 81.5% | 17 | 68%* | 46 | 63%* | 10 | **40%*** |
| Ventouse | 1 | 3.7% | 1 | 4% | 1 | 1.4% | 0 | 0% |
| Forceps | 1 | 3.7% | 1 | 4% | 2 | 2.6 | 1 | 4% |
| Elective LSCS | 1 | 3.7% | 5 | 20% | 16 | 22% | 9 | 36% |
| Emergency LSCS | 2 | 7.4% | 1 | 4% | 8 | 11% | 5 | 20% |
| **Mode of delivery Nuillparous** | 10 | 37% | 13 | 52% | 26 | 35.6% | 9 | 36% |
| Normal | 0 | 0% | 1 | 4% | 1 | 1.4% | 0 | 0% |
| Ventouse | 0 | 0% | 0 | 0% | 1 | 1.4% | 0 | 0% |
| Forceps | 0 | 0% | 0 | 0% | 4 | 5.5% | 8 | 11% |
| Elective LSCS | 2 | 7.4% | 0 | 0% | 4 | 5.5% | 0 | 0% |
| Emergency LSCS | | | | | | | | |
| **Mode of delivery multiparous** | 12 | 44.4% | 4 | 16% | 20 | 27.4% | 1 | 4% |
| Normal | 1 | 3.7% | 0 | 0% | 0 | 0% | 0 | 0% |
| Ventouse | 1 | 3.7% | 1 | 4% | 1 | 1.4% | 1 | 4% |
| Forceps | 1 | 3.7% | 5 | 20% | 12 | 16.4% | 1 | 4% |
| Elective LSCS | 0 | 0% | 1 | 4% | 4 | 5.5% | 5 | 20% |
| Emergency LSCS | | | | | | | | |

BMI group I: 19.1–25 kg/m2; BMI group II: 25.1–30 kg/m2; BMI group III: 30.1–40 kg/m2; BMI group IV > 40 kg/m2; GH, gestational hypertension; BP, blood pressure; PIHD, pregnancy induced hypertensive disorder; GDM, gestational diabetes mellitus; *Indicates a significance difference. Bold letter type indicates significant differences after combining subgroups versus reference group.

**Table (4) indications of CS among pregnant women**

| Variables | BMI group I (N= 27) | | BMI group II (N=25) | | BMI group III (N=73) | | BMI group IV (N=25) | |
|---|---|---|---|---|---|---|---|---|
| **Indication of CS** | | | | | | | | |
| Post date | 0 | 0% | 0 | 0% | 9 | **12.3%** | 0 | 0% |
| Fetal distress | 1 | 3.7% | 0 | 0% | 4 | **5.5%** | 0 | 0% |
| Severe preeclampsia | 1 | 3.7% | 0 | 0% | 3 | 4.1% | 0 | 0% |
| Breech | 0 | 0% | 0 | 0% | 2 | 2.7% | 0 | 0% |
| Dystocia | 1 | 3.7% | 2 | 8.0% | 1 | 1.4% | 8 | 32.0%* |
| Previous CS | 2 | 7.4% | 1 | 4.0% | 4 | 5.5% | 1 | 4.0% |
| combination | 1 | 3.7% | 3 | 12.0% | 1 | 1.4% | 0 | 0% |

BMI group I: 19.1–25 kg/m2; BMI group II: 25.1–30 kg/m2; BMI group III: 30.1–40 kg/m2; BMI group IV > 40 kg/m2. *Indicates a significant difference.

**Table (5) Distribution of prenatal outcomes of pregnancy among pregnant women**

| Variables | BMI group I (N= 27) | | BMI group II (N=25) | | BMI group III (N=73) | | BMI group IV (N=25) | |
|---|---|---|---|---|---|---|---|---|
| Apgar score 1 min | 7.06 ± 0.82 | | 6.72 ±1.28 | | 6.70 ±1.34* | | 6.93 ±1.16 | |
| Apgar score 5 min | 8.76 ± 0.97 | | 8.04 ±1.65 | | 8.00 ±1.44* | | 8.33 ± 1.11 | |
| Baby weight | 3.00 ± 0.33 | | 3.46± 0.62 | | 3.53 ± 0.54* | | 3.7 ±0.78* | |
| Complication | No | % | No | % | No | % | No | % |
| No | 21 | 77.7% | 19 | 76% | 31 | 42.5% | 8 | 32% |
| Fetal distress | 1 | 3.7% | 1 | 4% | 8 | 11% | 0 | 0% |
| Low apgar score | 1 | 3.7% | 0 | 0% | 24 | 32.8%* | 3 | 12% |
| Meconium stained | 1 | 3.7% | 1 | 4% | 4 | 5.5% | 0 | 0% |
| Neonatal death | 0 | 0% | 0 | 0% | 0 | 0% | 1 | 4% |
| Low birth weight | 1 | 3.7% | 1 | 4% | 2 | 2.7% | 0 | 0% |
| Macrosomia | 1 | 3.7% | 1 | 4% | 4 | 5.5% | 12 | 48%* |
| combination | 1 | 0% | 2 | 8% | 0 | 0% | 1 | 4% |

BMI group I: 19.1–25 kg/m2; BMI group II: 25.1–30 kg/m2; BMI group III: 30.1–40 kg/m2; BMI group IV > 40 kg/m2; Data are presented as percentages and mean (SD); Apgar score, baby weight. *Indicates a significant difference. Bold letter type indicates significant differences after combining subgroups versus reference group.

Our study found that obese women were at increased risk of developing gestational diabetes compared with women with a normal weight. The frequency of all diabetes was significantly higher in obese and morbidity obese explained by a markedly higher prevalence of gestational diabetes. These study findings are matching with **Yogev and Catalano (2009)** who reported that approximately 3-10% of obese women will be affected by gestational diabetes. Although many factors contribute to this, such as ethnic origin, age and family history, obesity constitutes an independent risk factor as the incidence of gestational diabetes is two- to three-fold higher in obese and overweight as compared to normal weight women. Moreover, obesity and diabetes play independent roles in determining fetal size.

It was observed that there was no significance difference detected among the four groups of our study, related glucose challenge (GCT) test, who control glycemia with diet, insulin, anti-diabetic drugs. On the same line **Berger *et al.* (2002)** stated that, obese women on diet who managed to control glycemia, as well as in overweight and obese women who failed to do so, irrespective of treatment. Therefore, testing in women with risk factors early in pregnancy is recommended.

The present study found that spontaneous onset of labour occurred mostly in the control group, statistical significance was found in obese and morbidly obese women and induction of labour (IOL) was significantly more common in morbidly obese women. This difference appeared to be primarily explained by the more common use of oxytocin. These finding were incongruent with **Schrauwers & Dekker (2009)** who stated that Spontaneous onset of labour occurred only in a minority of obese and morbidly obese women. These findings probably reflect an underlying 'angst' or pressure to act for the maternity care provider when confronted with a term pregnant patient of impressive dimensions. In particular the use of prostaglandins is very common in these women. Also **Bhattacharya *et al* (2007)** found an increased risk of induced labour in morbidly obese patients. The frequent use of prostaglandins is concerning since there is no Cochrane evidence suggesting that this is an effective and safe strategy to reduce intrapartum complications, quite the contrary data by for instance **Usha *et al.* (2005)** demonstrated that IOL is often the starting

point in the cascade of events leading to intrapartum complications. These findings were in contrast with **Chereshneva *et al* (2008)** found that there was no difference in the rates of spontaneous onset of labour in the three groups. This suggests that overweight and obese women were just as likely to start labouring, but were less likely to deliver vaginally compared with women with a normal BMI.

As a result, analyzing the mode of delivery [normal versus instrumental or lower segment caesarean section (LSCS)] showed a significantly higher risk of requiring an instrumental delivery or LSCS in obese and morbidity obese with OR exceeding 6 for morbidly obese patients. These findings were supported by **Joshua *et al.* (2004)** reported that the morbidly obese patients were more likely to have an operative vaginal delivery and LSCS than patients with a BMI less than 30. However, once again the OR was less than 2.0. On the same line **Aelvoet *et al.* (2008)** stated that the global epidemic of obesity in developed countries has been recognized as one of the key drivers in the global increase in LSCS. In this respect **Heslehurst *et al.* (2008)** pointed to that as maternal BMI increases, both the caesarean section rate and the rates of operative and complicated vaginal delivery increase. Also **Schrauwers & Dekker (2009)** who found that the obese is a major risk factor for operative vaginal delivery and LSCS, with OR exceeding 5 for morbidly obese patients.

According to mode of delivery, the present study noted that a normal vaginal delivery occurred in only 40% of morbidly obese patients with 56% delivering by elective or emergency LSCS.in the same line with the foregoing **Hibbard *et al* (2006) and Chu *et al.* (2007)** pointed to that a normal vaginal delivery occurred in only 36.7% of morbidly obese patients with 56.6% delivering by elective or emergency LSCS. These published data do suggest that it might be time to start large randomized controlled trials on the optimum mode of delivery in morbidly obese women not in spontaneous labour at 40week's gestation.

As the length of labour (combined first and second stage) increases with BMI (significant for obese women), but morbidly obese not have a significantly prolonged second stage. This result coincided with **Schrauwers & Dekker (2009)** who stressed that the length of labour (combined first and second stage) increases with BMI (significant for obese women), except for the morbidly obese. It might be that this unexpected finding in morbidly obese women reflects doctors 'stress' in the management of these very high risk patients, i.e. one has a lower threshold to resort to an emergency LSCS (38.3% in this cohort).

According to findings of the current study, Dystocia was the main reason to proceed to an elective LSCS in morbidity obese women. It occurred often in all BMI groups but significance was only in women of morbidity obese. This is agreement with **Joshua *et al.* (2004)** who found that obese patients may have difficulty completing the second stage of labor secondary to soft tissue dystocia, and operative vaginal delivery may be used to expedite delivery in such a situation. Moreover, **Mazouni *et al.* (2006)** stated that obesity is a significant maternal risk factor for shoulder dystocia, but also specified that the risk is caused by discrepancy between maternal height or weight and infant weight. In contrast, **Robinson *et al.* (2003)** found that obesity cannot be seen as an independent risk factor and they stated that fetal macrosomia is the single most powerful predictor in their study. In contrast with **Usha *et al.* (2005) and Abenhaim *et al.* (2007)** who showed that the strongest predictors of shoulder dystocia are related to fetal macrosomia rather than obesity.According to the current study results, the Mean birth weights increased with rising BMIs, being only statistically significant higher in the obese and morbidly obese groups. Macrosomia occurred more often in all increased BMI groups, but significance was only found in women of morbidly obese women. In the same line with **Sebire *et al.* (2001)** who found that both obese and morbidly obese patients have a significantly increased risk for birth weight greater than 4500g compared with controls. These findings are important to remember when clinically estimating fetal weight in the labor room. The clinician should know that a large fetus is not necessarily more common in the obese and morbidly obese population, but a macrosomic fetus is. Moreover **Doherty *et al.* (2006) and Callaway *et al.* (2006)** who established association between increasing maternal BMI and increasing birth weight. Also **Catalano & Ehrenberg, (2006)** mentioned that maternal obesity is well known to be associated with macrosomia. This relationship exists regardless of the definition used for macrosomia, including >90th centile, >4.5 kg. in another study conducted by **Abenhaim *et al.* (2007) and Arendas *et al.* (2008) and Gartier *et al.* (2008)** emphasized that higher mean birth weight and macrosomia was found in our overweight patients, and the obesity was a risk factor for macrosomia (OR 2.1).

Regarding the Apgar scores, a significant difference was found for one minute and after five minute Apgar score in the obese women, with the mean apgar after one and five minute was (6.70±1.34 and 8.00±1.44) for the same group. In this regard **Dereure *et al.* (2000)** highlighted that Apgar scores are slightly more in infants of obese mothers than in

infants of normal mothers. Moreover **Tosson and AL-Hussaini, (2005)** revealed that the mean Apgar scores after one minute and after five minute (8.80±1.26 vs. 9.23±1.33 and 9.69±0.53 vs. 9.42±1.95) for obese and non- obese pregnant women.

**Conclusion**

The burden of overweight and obese pregnant women in our population is high. There is a strong association of high pre-pregnancy BMI with diabetes and hypertension. It should be regarded as a high risk state because of its association with adverse obstetric outcome

**Recommendation**

Before pregnancy**:**

Body Mass Index should become a recorded measure for all patients, just as other vital signs are documented. It is important for women to be counseled about being at a healthy weight before becoming pregnant. This counseling could be accomplished at routine gynecological, family planning, or family practice visits. Women should be counseled on their weight, if out of the normal range, and should be informed of their increased risks once becoming pregnant if they are at an unhealthy weight.

During pregnancy:

Inform and counsel women about the health risks associated with overweight and obesity. Screen for hypertension and diabetes mellitus in women who are at risk. Maternal weight measurements need to be recorded during antenatal care and used consistently because pregnancy-increased BMI is obviously a risk factor for maternal complications during pregnancy, labor and postpartum. We can conclude that prepregnancy normal weight women with increased BMI during pregnancy need special follow-up and counseling in pregnancy and delivery. The counseling of overweight and obese pregnant women and specific guidelines are of particular interest for the obstetrician.

**After Pregnancy (postpartum):-**

**Recommendations for** "for ALL women":

• Encourage breastfeeding

• Counsel women to return to a healthy weight

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