

Maternal Obesity and Pregnancy Outcome

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Key words: obesity, pregnancy outcome, cesarean section, induction of labor

Abstract

Background: Obesity, a common condition in developed countries, is recognized as a threat to health.

Objectives: To describe the distribution of weight in pregnant women and evaluate the influence of obesity on pregnancy outcome in a high parity northern Israeli population.

Methods: The study included 887 women who gave birth in the Western Galilee Medical Center during the period August to November 1995. The patients were classified as underweight, normal weight, overweight, or obese according to body mass index. Maternal demographic, obstetric, and perinatal variables were compared. A control group of 167 normal weight women were matched with the obese group for maternal age, parity, and gestational age.

Results: Obese mothers had a higher incidence of gestational diabetes and pregnancy-induced hypertension compared to normal weight mothers (5.4% vs. 1.8%, and 7.2% vs. 0.6% respectively, $P < 0.01$), a higher rate of labor induction (20.4% vs. 10.2%, $P < 0.01$), and a higher cesarean section rate (19.6% vs. 10.8%, $P < 0.05$). There was also a significant difference in the prevalence of macrosomia in the offspring (16.8% vs. 8.4%, $P < 0.05$).

Conclusion: Obese pregnant women are at high risk for complications during delivery and therefore need careful pre-conception and prenatal counseling, as well as perinatal management.

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Obesity is a very common condition in developed countries [1] and frequently results in significant impairment of health. Associated with overeating, obesity is likely the product of the high fat diet prevalent in western countries [2]. Obesity is a well-known risk for cardiovascular disease; and the presence of hyperglycemia, hyperinsulinemia, and relative insulin resistance is a classic metabolic triad linked to the development of the overweight state [2]. In the non-pregnant state, well-established metabolic and hormonal changes are linked with the onset and maintenance of obesity.

Similar metabolic changes occur with advancing gestation and it is not surprising that they are more marked in obese gravidas, since age-adjusted rates of obesity for women of all races exceed those for men [2]. It is recognized that severe obesity is a hazard to the pregnant woman and her fetus, and the effects of obesity during pregnancy have been the subject of several investigations [3,4]. These health impacts of obesity in pregnancy beckon further research among obstetric gynecologists. The present study evaluates the effects of excessive maternal weight on pregnancy outcome in northern Israel, thus adding to our knowledge on the Israeli population.

Patients and Methods

The study population comprised 887 women who gave birth in the Western Galilee Hospital in Nahariya between August and November 1995. The patients were divided into four groups according to body mass index, as defined by the Food and Nutrition Board of the National Institutes of Medicine [5]. BMI is derived by dividing weight (kilograms) by height squared (meters). The definitions are as follows: underweight: BMI <19.8, normal weight: BMI 19.8–26, overweight: BMI 26.1–29, and obese: BMI >29.

Of the 887 women, 62 (7%) were classified as being underweight, 483 (54.5%) were of normal weight, 175 (19.7%) were overweight, and 167 (18.8%) were obese [Table 1]. The 167 obese pregnant women (BMI >29) constituted the study group. The 167 patients from the normal weight group (483 women) — matched for maternal age, parity and length of pregnancy — were selected as a control for this case-control study. The following data were reviewed:

- **Maternal variables:** maternal age, parity, weight, BMI, weight gain during pregnancy, and weight of fetus.
- **Obstetric complications:** twin pregnancy, gestational diabetes mellitus defined by at least two abnormal values using the 3 hours glucose tolerance test (fasting: 105 and 190–165–145 mg/dl after 1–2–3 hours (Williams Obstetrics, 20th edition), pregnancy-induced hypertension that develops as a consequence of pregnancy and regresses postpartum, blood pressure 140/90 mmHg or higher with

BMI = body mass index

Table 1. Maternal characteristics (mean \pm standard error)

	No.	BMI	Mean age (yr)	Gestational age at delivery (wk)	Mean weight gain (kg)	Parity
Underweight	62	18.56 \pm 0.11	25.06 \pm 0.60*	38.05 \pm 0.23	11.2 \pm 0.63	2/08 \pm 0.17
Normal weight	167	23.38 \pm 0.13	30.2 \pm 0.44	39.2 \pm 0.15	10.2 \pm 0.36*	25 \pm 0.12*
Overweight	175	27.4 \pm 0.06	29.02 \pm 0.39	39.32 \pm 0.11	8.48 \pm 0.33	3.29 \pm 0.15
Obese	167	32.42 \pm 0.24	30.5 \pm 0.45	39.06 \pm 0.15	7.79 \pm 0.36*	3.99 \pm 0.18*

* $P < 0.001$.

or without proteinuria or pathological edema, placenta previa, placental abruption, cephalopelvic disproportion, premature rupture of membranes, dysfunctional labor, cord around the neck, antepartum hemorrhage, postpartum hemorrhage, prematurity, and oligohydramnios.

- **Perinatal outcome:** intrauterine fetal death, pathological fetal heart rate, intrauterine growth restriction, preterm delivery, and macrosomia (birthweight 4,000 g or higher).
- **Therapeutic interventions:** induction of labor, episiotomy, amniotomy, manual removal of placenta, vacuum extraction, forceps, and cesarean section.

The statistical analysis included classical tests usually used in such studies. Differences of means were evaluated by the Student *t* test. Differences in rates were calculated using the chi-square test. Statistical significance was defined as $P < 0.05$.

Results

Table 2 shows the complications of pregnancy in the two groups. There was no statistically significant difference in the incidence of PROM between the obese and the normal weight women (19.2% vs. 12.5%), but PIH was significantly more common in the obese women, 7.2% vs. 0.6% ($P < 0.01$), as was GDM ($P < 0.08$, NS).

Data on labor are presented in Table 3. Labor was induced in 10.2% of the normal group and 20.4% of the obese group ($P < 0.05$). Among the reasons for induction, PIH showed the greatest difference (8 vs. 1), followed by post-term pregnancy (6 vs. 2), between obese and normal weight respectively. With regard to the techniques used for induction, prostaglandins or the Atad ripening catheter for unripe cervix were administered to 68% of the obese women versus 41% of normal weight women. Oxytocin was given to almost the same number from both groups.

The obese mothers had a significantly higher rate of cesarean section (19.6% vs. 10.8%, $P < 0.05$).

There were 56% elective cesarean sections in the normal weight group and 69% in the obese group. The main reasons were large babies and malpresentations in the obese group, and dysfunctional labor and placenta previa in the normal weight group. Postoperative morbidity (wound infection,

PROM = premature rupture of membranes

PIH = pregnancy-induced hypertension

GDM = gestational diabetes mellitus

Table 2. Maternal complications

	Normal weight (n=167)	Obese (n=167)	Significance
Placenta previa	4 (2.4%)	1 (0.6%)	NS
Placental abruption	3 (1.8%)	3 (1.8%)	NS
PROM	21 (12.5%)	32 (19.2%)	NS
Oligohydramnios	3 (1.8%)	5 (3%)	NS
Dysfunctional labor	11 (6.6%)	3 (1.8%)	$P < 0.03$
Cord around neck	10 (6%)	14 (8.4%)	NS
GDM	3 (1.8%)	9 (5.4%)	NS ($P < 0.08$)
PIH	1 (0.6%)	12 (7.2%)	$P < 0.01$
Chronic hypertension	—	4 (2.4%)	NS
CPD	—	3 (1.8%)	NS
APH	1 (0.6%)	1 (0.6%)	NS
PPH	—	1 (0.6%)	NS
Breech presentation	2 (1.19%)	9 (5.4%)	$P < 0.04$

NS = not significant, APH = antepartum hemorrhage, PPH = postpartum hemorrhage, CPD = cephalopelvic disproportion

Table 3. Obstetric interventions

	Normal weight	Obese	Significance
Induction of labor	17(10.2%)*	34(20.4%)*	< 0.01
Manual removal of placenta	1(0.6%)	—	NS
Manual exploration of uterus	2(1.2%)	3(1.8%)	NS
Cesarean section	18 (10.8%)**	32 (19.6%)**	0.04
Episiotomy	27 (16.2%)	26 (15.6%)	NS
Amniotomy	5 (3%)	2 (1.2%)	NS
Vacuum extraction	2 (1.2%)	1 (0.6%)	NS

* $P < 0.01$ ** $P < 0.05$

blood loss, urinary tract infection, aspiration) was present in 2% in the normal weight group and 15% in the obese group ($P < 0.01$). Maternal complications after vaginal delivery (vaginal laceration, postpartum hemorrhage) were present in 1.2% of normal weight women and 2.3% of obese women. Table 4 presents the neonatal outcome, showing that obese mothers delivered twice as many macrosomic infants (birthweight $\geq 4,000$ g) than the normal weight mothers (16.8% vs. 8.4%, $P < 0.02$). Indications for admission to the neonatal intensive care unit showed more prematurity and hyperbilirubinemia and less respiratory distress syndrome in normal weight compared to obese mothers.

Our study showed that obese mothers were more likely than their non-obese counterparts to have medical induction of labor (20.4% and 10.2% respectively, $P < 0.02$). There

Table 4. Neonatal characteristics

	Underweight mothers	Normal weight mothers	Overweight mothers	Obese mothers (normal weight vs. obese)	Significance
Weight (g)	3048±65.5	3270±52	3427±39.7	3400±48	NS
Twins	1 (1.6%)	4 (2.4%)	3 (1.7%)	7 (4.2%)	NS
IUFD	—	1 (0.6%)	2 (1.1%)	1 (0.6%)	NS
Pathol FHR	5 (8%)	9 (5.4%)	10 (5.7%)	15 (8%)	NS
IUGR	4 (6.5%)	5 (3%)	4 (2.3%)	5 (3%)	NS
Prematurity	2 (3.2%)	8 (4.8%)	3 (1.7%)	13 (7.8%)	NS
Macrosomia	3 (4.8%)	14 (8.4%)	15 (8.6%)	28 (16.8%)	<i>P</i> <0.03
NICU	8 (12.9%)	31 (18.6%)	12 (6.9%)	15 (8.9%)	<i>P</i> <0.02

IUFD = intrauterine fetal death, Pathol FHR = pathological fetal heart rate, NICU = admitted to neonatal intensive care unit

were no significant differences between the groups in the prevalence of pathological fetal heart rate, prematurity (almost significant, *P*<0.06), fetal death *in utero*, or IUGR. Significantly more infants born to normal weight mothers were admitted to the neonatal intensive care unit than infants born to obese mothers, 18.6% vs. 8.9% (*P*<0.05).

Discussion

The influence of obesity in pregnancy is well described in the medical literature [6–8]. In this study, the first on this entity in a northern Israeli population, we used the BMI criterion for evaluating body weight; Perlow and others [7] defined massive obesity as a pregnancy weight over 300 lbs, while Ekblad and Grenman [6] defined abnormal maternal pregnancy weight as being 20% over ideal body weight for height. The prevalence of obesity in our study population was 18.9%. In general, obese women gained less weight during pregnancy than mothers with normal body build. Obese women gained a mean of 7.79±0.36 kg, while those underweight gained 11.2±0.63 kg, figures similar to those reported by Ekblad and Grenman [6]. The optimal weight gain during pregnancy in obese mothers, as recommended in the American College of Obstetricians and Gynecologists' technical bulletin [8–10], is no more than 15 lbs (6.79 kg), while in underweight women the recommended optimal weight gain is 12.48–18.12 kg.

Another interesting finding was the higher parity of obese women in our population than observed by Ekblad and Grenman [6] and Perlow et al. [7].

The rate of cesarean section is high in most developed countries, and obesity is one of several important determinants for this operation. Our study concurred with other reports [6,7] of significantly higher rates of cesarean section in obese women. We found a strikingly higher frequency of postoperative complications in women of the obese group. Obese women are at higher risk for both chronic hypertension and preeclampsia during pregnancy. In our study 7.2% of obese women and 0.6% of non-obese women had

PIH, and 2.4% of obese women suffered from chronic hypertension. The obese mothers were more likely than non-obese mothers to have medically induced labor (20.4% and 10.2% respectively), suggesting that obesity is a factor in medical induction.

With regard to the offspring, Naeye [4] reported increased perinatal mortality among infants of obese mothers. Although in other studies the incidence of intrauterine fetal death was lower in obese women [11], we found no such difference between obese and control mothers. Macrosomia is more common among infants of obese women, which is possibly related to obesity and gestational diabetes [12]. The incidence of macrosomia in obese women in this investigation was indeed higher than in the normal weight group. It is difficult to explain why more infants born to normal weight mothers were admitted to the neonatal intensive care unit since most indications for admission were similar in both groups.

In conclusion, the results of our study indicate that obesity carries a significantly increased risk for complications during delivery. Obesity also presents a health hazard to mothers for the rest of their lives. Even if no other pregnancies are planned, such patients should be advised that postpartum nutritional counseling to encourage weight loss may be the best course for long-term health.

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IUGR = intrauterine growth restriction

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Experience is a hard teacher because she gives the test first, the lesson after.

Vernon Sanders-Law

Capsule



Are somatic symptoms really due to depression?

A team of researchers explored the relationship between depression and somatic symptoms. They based their experiment on the hypothesis that a patient is more likely to present with somatic symptoms (headache, constipation, weakness, and back pain) than depressive symptoms (loss of appetite, feelings of worthlessness, and guilt). Another point examined was whether non-western cultures and developing countries are more likely to deny psychological symptoms and to seek help for somatic complaints. This behavior may be found more frequently in a culture where a negative stigma is attached to psychological distress.

Defining somatization in three different ways, the team screened patients with depression based on these definitions in several primary care centers in 14 countries on 5 continents. Their goal was to examine the relationship between different ways of presenting somatic symptoms and depression. They also attempted to correlate behavioral differences between cultures in the way depression is manifested. The first definition, "somatic presentation" of depression, refers to the parameter that many patients with psychiatric disorders seek care for somatic symptoms. They found that although there were certain centers in non-western countries with higher rates of somatized depression, statistically the form of somatization did not vary significantly according to geographic or economic classification of the centers. The second definition, "somatosensory amplification," accounts for the influence psychological stress has on one's perception of somatic symptoms. This definition applies to patients with psychological disorders who report multiple (at least three) unexplained somatic symptoms. This view holds that somatization is a psychological defense mechanism against the expression of depressive

symptoms, and would be most prevalent in cultures where psychiatric disorders carry a negative connotation. It was reported that depressed individuals were more likely to report unexplained symptoms than those without psychological problems. However, it is also possible that these results are due to differences in reporting rather than correlation between depression and somatic symptoms. The relationship between depression and unexplained somatic symptoms were again consistent between centers. The third definition was denial of psychological symptoms when directly questioned. On examining the patient's tendency to deny psychological symptoms, no differences in patient admission or denial were found between centers. Sixty percent of patients initially presented with somatic symptoms, but acknowledged psychological disturbances when asked.

Results showed that 85% of patients with major depression met at least one of the definitions of somatization, but only 4% met all three. There were no cultural differences in the types of symptoms reported and no differences in rates of depression between non-western and western countries. However, the agreement among centers was statistically no greater than could be expected by chance. It was found that somatic symptoms are universally central to depressive episodes, and somatization of depression was greater in walk-in centers than in patients having a prior relationship with their physician. The authors conclude that an individual's initial report of somatic symptoms may be due not to an unwillingness to acknowledge psychological disturbances, but as a route to initially seek help.

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