Feto-Maternal Outcome in Supervised Pregnancies of the Overweight Parturient

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Abstract

Objective

To compare the feto-maternal outcome in obese and matched non-obese parturients

Materials and Methods

106 obese, pregnant women and an equal number of non–obese matched controls who booked and delivered in LASUTH were studied.

Biosocial data as well as feto-maternal parameters were recorded and analyzed.

Results

Increasing maternal age and parity were significantly associated with obesity in this study.

There was no significant association between maternal weight and either prematurity or postdatism.

PIH was the commonest antenatal complication in the obese group. None of the patients in the non-obese group had abnormal presentations.

Caesarean section was commoner and the incidence of spontaneous vertex delivery lower in the obese group.

The mean birth weight of babies in the obese group was significantly higher than that of the control group. 20.8% of the obese group had macrosomic babies compared with only 5.7% of the non obese group.

No statistically significant differences were demonstrated in the incidence of neonatal complications, estimated blood loss at delivery and duration of hospital stay.

Conclusions

Overweight parturients in this study tend to be older, of higher parity, prone to pregnancy induced hypertension, have a higher incidence of caesarean sections and are predisposed to having bigger babies.

Keywords: *Diabetes, obesity, fetus, pregnancy, Apgar, forceps*

Introduction

Obesity is a nutritional disorder that is characterized by excessive accumulation of fat in the subcutaneous tissues, omentum, viscera and muscles[1]. It is due to an imbalance between energy intake and expenditure such that excess energy is stored in the fat cells which enlarge or increase in number[2].

Obesity is defined as weight greater than the 95th percentile of the sex specific BMI for age. WHO defines obesity as a body mass index greater than 39kg/m^2. In obstetrics, the attainment of a critical weight of 90kg or more at some time during pregnancy is regarded as obesity while pregnant women weighing 114kg or more at anytime during pregnancy are regarded as massively obese.

Obesity has become a global epidemic with an estimated 1.3 billion people being overweight or obese[2]. The incidence of obesity in pregnancy is 7.4%[3]. Obesity until now has been a problem of developed countries but is now gradually spreading to developing countries.

There are a number of clinical problems that had been reportedly associated with obesity in pregnancy. They include higher incidences of spontaneous abortions, urinary tract infections, abnormal presentations, pregnancy induced hypertension, gestational diabetes mellitus and macrosomic babies. Others are increased risk of augmentation of labour, prolonged labour, obstructed labour, cephalopelvic disproportion, increased risk of delivery by caesarean section, wound dehiscence and sepsis, prolonged hospital stay, primary post-partum hemorrhage and deep vein thrombosis.

The effects of excess adipose tissue or the metabolic effects of increased adiposity possibly explain these occurrences. The metabolic effects of increased adipose tissue are exerted via increased release of free fatty acids and the production of adipocyte derived factors known as adipokines that have specific functions most of which are not clearly understood. These include the hormones leptin, and adinopectin, cytokines TGFx and IL-6, transcription factors and other adipokines angiotensinogen and resistin[2].

A lot of studies had reported adverse pregnancy outcomes in obese pregnant women but most of these findings were from studies carried out in the developed world countries. Few studies have been done on obese pregnant women in Nigeria. The question therefore is whether there is enough evidence to extrapolate the findings from the developed countries on the Nigerian situation. This prospective randomized case controlled study set out to determine the feto-maternal outcomes in obese pregnant Nigerian women using the matched non-obese patients as controls.

Materials and Methods

The study was conducted between 1st January 2006 and 31st March 2007. A total of 106 obese pregnant women were selected using a Body Mass Index (BMI) of 30kg/m2 as the cut off point for obesity. This was in conformity with the World Health Organization definition of obesity.[4] An equal number of non-obese controls (106) with BMI less than 25kg/m^2 were selected and matched as much as possible for age and parity. Both subjects and controls were followed up through the antenatal period to the post-partum period and discharge. Only patients who booked in LASUTH and delivered in the hospital were recruited for the study. Age, parity, antenatal complications, estimated gestational age at delivery, mode of delivery, birth weight, Apgar score at 1 and 5 minutes of the baby, neonatal outcome, post partum complications, estimated blood loss and duration of hospital stay were observed and documented.

The data was analyzed using SPSS version 11.0-Statistical Computer Software. Proportions and percentages were calculated for categorical variables. Pearson's Chi-square (a non-parametric) test was used to determine the significant differences. Level of statistical significance was set at $P < 0.05$. Some parameters were analyzed using the independent samples T-test and Mannwhitney U test.

Results

The results are as shown in the tables and figure below.

69.8% of the obese patients were in the age group 37–40 years while 54.7% of the non-obese patients were in the age group 21–30 years. (Table I). Increasing age is directly and significantly associated with obesity ($p< 0.05$). 71.6% of the obese group were Para 2 and above compared with only 39.6% of the non obese group. (Table I). Increasing parity appears to be significantly associated with obesity ($p < 0.05$).

The mean estimated gestational age at delivery of the obese group was 39.44 + 2.31 weeks ranging from 30 to 42 weeks while the mean of the control group was 41.02 + 2.42 weeks ranging from 29 to 42 weeks (Table I). Neither prematurity nor postdatism was significantly associated with the subjects or the control groups.

Pregnancy Induced Hypertension (PIH) was the most common (24.5%) antenatal complication in the obese group (Table II). Compared to the incidence of 7.5% in the non-obese group, this was statistically significant. None of those in the non-obese group had

abnormal presentations compared to 5.7% of those in the obese group. The other antenatal complications appeared evenly distributed among the subjects and controls.

54.7% of the obese patients had caesarean delivery compared to only 39.6% in the non-obese group. The incidence of spontaneous vertex delivery was also higher in the non-obese group (58.5% vs 45.3%).

The mean birth weight of babies delivered by patients in the obese group was 3.47 + 0.79kg while that of the control group was 3.11 $^{+}$ 0.60kg. The mean birth weight of babies in the obese group was significantly higher than that of the control group by 0.36kg using the independent sample T-test. Also, using 4.0kg as the birth weight considered macrosomic, 20.8% of the obese group had macrosomic babies compared with only 5.7% of the non obese group (Figure 1). Even though the mean Apgar score of the non obese group was higher than that of the obese group, this was not statistically significant.

There were more neonatal complications in the obese group than in the non-obese group but this was not statistically significant ($p > 0.05$). Table III

The mean estimated blood loss of the obese group was higher than the control group though using the independent samples T-test the difference was not statistically significant.

The mean duration of hospital stay of the obese group was 6.17 + 2.81 days while that of the control group was 6.43 + 3.87 days. There was no statistical significance in the slight difference in mean duration of hospital stay of the two groups using the independence T-test.

Discussion

In a study involving obesity, the question will naturally be: "who is an obese pregnant woman?" A number of previous works[5] used the cutoff of 90kg body weight while others used the Body Mass Index (BMI) whereby obesity is defined as a BMI of greater than 27 kg/m2[6]. Sebire[1] et al defined normal weight as BMI 20-24.9kg/m^2, moderate obesity as BMI 25-29.9 kg/m^2 and very obese as BMI greater or equal to 30kg/m^2. Other workers would rather differentiate between being overweight (BMI 25-30kg/m2) and being obese (BMI > 30kg/m2)[7]. The World Health Organization and National Institute of Health in America recommend that obesity is measured by the body mass index where a BMI of 30-34.9 kg/m^2 is classified as Class 1 (mild obesity). 35-39.9kg/m^2 as Class II (moderate obesity) and greater than or equal to 40kg/m^2 as Class III (severe obesity)[8]. In this study we used a BMI of 30kg/m^2 as the cutoff for obesity. It is known that BMI is a better indicator of body composition than weight alone, being a more sensitive indicator of obesity in shorter women[9].

Increasing age and parity were found to be significantly associated with obesity in this study. The association with increasing age was earlier documented[3,10] while the association with increasing parity was also documented in a study from Nigeria[6]. Increasing age and multiparity are recognized as risk factors for obesity[9].

There is conflicting data in the literature regarding maternal obesity and preterm birth. Some studies showed increased risk[11,12]. Others showed a decreased risk or no risk at all[13,14]. In this study, neither prematurity nor postdatism was significantly associated with maternal obesity. In our opinion, the conflicting data pertaining to this parameter may be because of differences in the classification of obesity in the various studies.

The studies from Nigeria demonstrated significant relationships between maternal obesity and PIH[3,6]. Other much larger series from Nova Scotia, Canada and London confirmed the association[1,5]. In this study, an association between maternal obesity and PIH was demonstrated however, no other medical diseases were significantly commoner in the obese parturients. Maternal hemodynamic changes in obese mothers include higher arterial pressure, hemoconcentration and altered cardiac function[15]. In addition, the prevalence of hypertensive disorder and pre-eclampsia is higher in obese women.[16]

As in this study, a lot of other studies of the obese parturient had documented an increased caesarean section rate[1,3,5,6]. It is generally thought that the increase in the caesarean section rate may be related to an increased number of large for gestational age infants, suboptimal uterine contractions and increased fat deposition in the soft tissues of the pelvis leading to dystocia during labour[7]. However, some other workers had posited that the higher rate of caesarean section seems to be related to the higher rate of complications in the obese women rather than the obesity itself[17].

Birth weights above the 90th percentile are commonly associated with babies of obese mothers[1,3,6,8,18,19]. In this study, using 4.0 kg as the birth weight considered macrosomic, 20.8% of babies in the obese mothers were macrosomic compared with only 5.7% in the non obese mothers. The original Pedersen hypothesis suggested that increased glucose concentrations in the mother with diabetes led to increased fetal growth[20]. Obesity is associated with increased maternal insulin resistance and fetal hyperinsulinaemia even in the absence of maternal diabetes[21]. Insulin resistant individuals have higher fasting plasma triglyceride levels and greater leucine turnover. Amino acids are insulin secretagogues and an increased flux of amino acids could stimulate foetal hyperinsulinaemia. Triglycerides are energy rich and placental lipases can cleave triglyceride and transfer free fatty acids to the

foetus[22,23,24]. The combination of an increased energy flux to the fetus and fetal hyperinsulinaemia may explain he increased frequency of large for gestational age infants seen in the obese non diabetic women studied.

Obi and Ebute documented significantly higher incidences of birth asphyxia, birth trauma, neonatal admission to intensive care unit and perinatal mortality in their series[6]. Another series from Nigeria however concluded that the risk of birth asphyxia and perinatal mortality were not increased by obesity[3]. In this study, even though there were more neonatal complications in the obese group, no statistical significance was demonstrated. The very large series in the United Kingdom could not determine whether there was an increase in neonatal morbidity[1]. This was attributed to the inconsistency in the recording of precise neonatal data. It has been suggested nevertheless that the relative risk of neonatal death is greater in preterm infants born to obese mothers than to the non-obese.[25] This may be secondary to the altered metabolic milieu in obesity reducing the infant's ability to adapt to postnatal life.

The issue of blood loss at either normal delivery or caesarean section has been researched in the obese and non obese parturients. Most studies found a higher incidence of postpartum hemorrhage in the obese patients[1]. It is suggested that the increased risk of postpartum hemorrhage in obese women, even after accounting for such predisposing factors as caesarean section may be explained by more bleeding from the relatively larger area of implantation of the placenta usually associated with a large for gestational age foetus[1]. In this study though there was no statistically significant difference between the average blood losses in both subjects and controls. The generally accepted difficulty in the accurate assessment of blood loss at delivery may explain this observation.

Conclusions

This study shows that in many ways the obese parturient in our setting tends to have a more adverse pregnancy outcome than the non obese. This makes a clear case for paying attention to pre-conception weight control as well as reasonable eating and moderate exercise in pregnancy. Maternal obesity should necessarily be an indication for referral to a specialist facility. In addition, recognition of this difference in outcome between the obese and the non obese should call for a closer monitoring of the former in pregnancy and labour.

References

1. **Sebire NJ, Jolly M, Hamis JP et al**. Maternal obesity and pregnancy outcome. A study of 287213 pregnancies in London; *Int J Obes*. August 2001; 25(8):1175-82

2. **Goedecke JH, Jennings CL, Lambent EV**. Obesity in South Africa; 1995-2005: 65-78

3. **Olayemi OO, Umuem CO and Aimakhu CO**. Obstetric performance of Nigerian obese parturients. *Trop J Obstet Gynaecol*. April 2002; 19(1): 17-20.

4. **World Health Organization**. Obesity: Preventing and Managing the Global Epidemic-Report of a WHO Consultation on Obesity, Geneva, June 3-5, 1997. Geneva: World Health Organization; 1998.

5. **Robinson HE, O'Connell CM, Joseph KS, McLeod NL**. Maternal Outcomes in Pregnancies Complicated by Obesity. *Obstet Gynaecol* 2005; 106 (6): 1357-1364.

6. **Obi SN and Obute EA.** Pregnancy outcome in the obese Nigerian. *Trop J Obstet Gynaecol.* April 2004: 32-5.

7. **Yu CKH, Teoh TG, Robinson S.** Obesity in pregnancy. *Br J Obstet Gynaecol* 2006. 113(10): 1117-1125.

8. **Nadine M, Geeta K.** Obesity and pregnancy: A care plan for management. The Internet Journal of Gynaecology and Obstetrics. 2008. 9(2).

9. **Wolfe HM, Zador IE, Gross TL, Martier SS, Sokol RJ.** The clinical utility of maternal body mass index in pregnancy. *Am J Obstet Gynecol*. 1991. 164: 1306-1310.

10. **Lissner L, Johansson SE, Qvist J, Rossner S, Wolk A.** Social mapping of the obesity epidemic in Sweden. *Int J Obes Relat Metab Disord* 2000. 24: 801-805.

11. **Baeten JM, Bukusi EA, Lambe M**. Pregnancy complications and outcomes among overweight and obese nulliparous women. *Am J Public Health*. 2001. 91: 436-440.

12. **Jensen DM, Damm P, Sorensen B et al**. Pregnancy outcome and prepregnancy body mass index in 2459 glucose-tolerant Danish women. *Am J Obstet Gynecol*. 2003. 189: 239-244.

13. **Cnattingius S, Bergstrom R, Lipworth L, Kramer M**. Pre-pregnancy weight and the risk of adverse pregnancy outcomes. *N Engl J Med*. 1998. 338: 147-152.

14. **Sheiner E, Levy A, Menes TS, Silverberg D, Katz M, Mazor M**. Maternal obesity as an independent risk factor for caesarean delivery. Paediatr Perinat Epidemiol 2004. 18: 196-201.

15. **Tomoda S, Tamura T, Sudo Y, Ogita S**. Effects of obesity on pregnant wome: maternal hemodynamic change. *Am J Perinatol*. 1996. 13: 73-78.

16. **Sibai BM, Ramada MK, Chari RS, Friedman SA**. Pregnancies complicated by HELLP syndrome (Hemolysis, Elevated Liver enzymes and Low Platelets): subsequent pregnancy outcome and long-term prognosis. *Am J Obstet Gynecol* 1995. 172: 125-129.

17. **Perlow JH, Morgan MA, Montgomery D, Towers CV, Porto M**. Perinatal outcome in pregnancy complicated by massive obesity. *Am J Obstet Gynecol*. 1992. 167: 958-962.

18. **Weiss JL, Malone FD, Emig D. et al**. Obesity, obstetric complications and caesarean delivery rate- a population-based screening study. *Am J Obstet Gynecol*. 2004. 190: 1091-1097.

19. **Cedergreen MI**. Maternal morbid obesity and the risk of adverse pregnancy outcome. *Obstet Gynecol*. 2004. 103: 219-224.

20. **Pedersen J, Bojsen-Moller B, Poulsen.** Blood sugar in newborn infants of diabetic mothers. *Acta Endocrinol* 1954: 15: 33-52.

21. **Hoegsberg B, Gruppuso PA, Coustan DR**. Hyperinsulinaemia in macrosomic infants of nondiabetic mothers. Diabetes Care. 1993: 16; 32-36.

22. **Portman OW, Behrman RE, Soltys P.** Transfer of free fatty acids across the primate placenta. *Am J Physiol* 1969: 216; 143-147.

23. **Robinson S, Coldham N, Gelding SV. et al.** Leucine flux is increased whilst glucose turnover is normal,in pregnancy complicated by gestational diabetes mellitus. Diabetologia. 1992: (Suppl 1) A 683.

24. **Thomas CR.** Placental transfer of non-esterified fatty acids in normal and diabetic pregnancy. *Biol Neonate*. 1987. 51: 91-101.

25. **Lucas A, Morley R, Cole TJ. et al**. Maternal fatness and viability of preterm infants. *Br Med J*. 1998. 296: 1495-1497.

TABLE 1: AGE, PARITY AND GESTATIONAL AGE DISTRIBUTION IN SUBJECTS AND CONTROLS

	Obese No (%)	**Non-Obese No (%)**
AGE (Years)		
21 – 30	28 (26.4%)	58 (54.7%)
31 – 40	74 (69.8%)	46 (43.4%)
41 – 50	4 (3.8%)	2 (1.7%)
PARITY		
0	2 (1.9%)	0
1	28 (26.4%)	64 (60.4%)
2	40 (37.7%)	26 (24.5%)
3	16(15.1%)	8 (7.5%)
4	10 (9.4%)	6 (5.7%)
5	10(9.4%)	2 (1.9%)
ESTIMATED GESTATIONAL AGE (weeks)		
28 – 30	2 (1.9)	4 (3.8%)
31 – 33	6 (5.6%)	4 (3.8%)
34 – 36	12(11.4%)	10 (9.4%)
37 – 39	56 (52.8%)	48(45.3%)
40 – 42	30 (28.3%)	40 (37.7%)
TOTAL	**106 (100%)**	**106 (100%)**

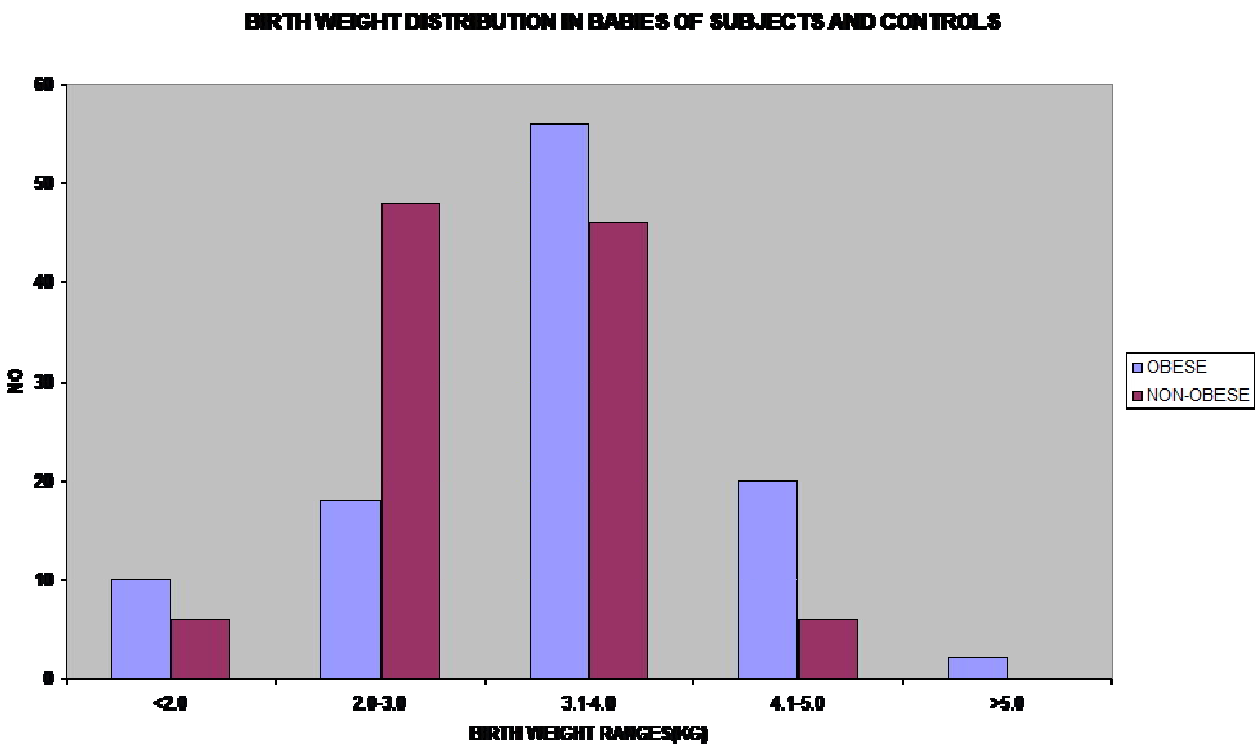

FIGURE 1: RANGES OF BIRTH WEIGHTS IN BABIES OF SUBJECTS AND CONTROLS

Table II: Antenatal Complications

Complications	**Obese** **No (%)**	**Non-Obese** **No (%)**
PIH	26 (24.5%)	8 (7.5%)
GDM	8 (7.5%)	2 (1.9%)
Febrile illness	6 (5.7%)	10 (9.4%)
Unstable lie	4 (3.8%)	-
Cough	2 (1.9%)	4 (3.8%)
IUGR	-	4 (3.8%)
Abnormal presentation	6 (5.7%)	-
UTI	-	6 (5.7%)
Vaginitis	-	6 (5.7%)
Eclampsia	-	2 (1.9%)
None	54 (50.9%)	64 (60.3%)
TOTAL	**106 (100%)**	**106 (100%)**

Table III: Neonatal Outcome

	Obese **No (%)**	**Non-Obese** **No (%)**
Neonatal sepsis	4	2
Neonatal jaundice	6	2
Perinatal Asphyxia	4	-
Macrosomia	2	-
Diarrhea	-	2
Purulent conjunctivitis	-	2