Short-term obstetric outcomes in obese Maltese women

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Abstract

Introduction: Obesity is associated with specific increased perinatal risks to mother and child. The study sets out to identify and assess the risk in a high NIDDM prevalence population. *Methodology:* The obstetric outcomes of 1691 women with a body weight ≥ 85 kg and a pre-pregnancy BMI >30 and their 1721 infants were statistically compared to similar parameters in the background population of 18717 maternities and 18974 births. *Results:* Obese mothers were characteristically more likely to be older than 30 years of age [$p<0.0001$], multiparous [$p<0.0001$] with a history of previous miscarriages [$p=0.003$]. The antenatal period was more likely to be complicated by hypertensive disease [pre-existing and pregnancy-induced; $p<0.0001$], and gestational diabetes [$p<0.0001$]. They were less likely to suffer from accidental haemorrhage [$p=0.0074$]. These antenatal problems were not simply a determinant of maternal age. They did not appear to have a significantly higher risk of pre-existing diabetes [$p=0.3267$] and did not require an increased risk of assisted reproduction [$p=0.3953$]. The multiple pregnancy rates were also not statistically increased [$p=0.3121$]. The obese women were also more likely to require obstetric interventions with induction of labour [$p<0.0001$] and Caesarean section [$p<0.0001$]. There was a statistically lower rate of operative deliveries [$p=0.0007$]. The preterm delivery rates were not different [$p=0.947$] between the two groups of women. The infants born to obese women were at a statistically higher risk for macrosomia [more than 4.0 kg; $p<0.0001$] and lower risk for low birth weight [under 2.5 kg; $p=0.0248$]. They also had a statistically higher risk for congenital malformations. There was a slightly higher risk for respiratory distress although the differences did not reach statistical significance [$p=0.0596$]. There was not any significantly increased risk for perinatal loss [$p=0.8212$], shoulder dystocia [$p=0.5059$], and low APGAR scores at five minutes [$p=0.9989$]. There were no differences in the male: female ratios [$p=0.761$]. Infants born to obese women were less likely to be solely breastfeed, though the differences did not reach statistical significance [$p=0.0782$]. *Conclusion:* It appears that the obese pregnant woman and her infant are predisposed to adverse short-term obstetric outcomes similar to those found in gestational diabetics. This relationship may reflect determinants of the "Metabolic Syndrome". (Int J Diabetes Metab 14: 88-91, 2006)

Keywords: *complications, obesity, outcome, pregnancy*

Introduction

The Maltese population comprises a small island community in the Central Mediterranean with a high prevalence of obesity and maturity onset diabetes mellitus reflected in a high prevalence of gestational carbohydrate intolerance. A comprehensive epidemiological study conducted in 1981 has established that the mean Body Mass Index (BMI) was already in the overweight range (25-30) in females aged 25-44 years, while above the age of 45 years, females had a mean BMI in the obese range (>30). The same study showed that the overall prevalence of carbohydrate metabolism disorders in female individuals aged >15 years was 14.4% (DM 9.0%; IGT 5.4%). The rate was higher in the elderly. A similar high rate of gestational diabetes amounting to 11.5% using WHO diagnostic criteria has been identified in the pregnant Maltese population.[1,2] There is evidence to support the inclusion of obesity, even in the absence of gestational or pre-gestational carbohydrate intolerance, as a significant risk factor possibly related to the association with Syndrome X. A pilot study in the obese Maltese population has previously suggested an increased risk of hypertensive disease and carbohydrate intolerance, while their infants were more likely to be macrosomic.[3] The aim of the study was to identify the short-term outcome risk factors to the obese mothers and their infants in this high-risk population.

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Material and Methods

The study reviewed the medical data records of all women delivering in the Maltese Islands during 1999-2003 [n = 20408 maternities resulting in 20695 births]. The medical data was made available by the National Obstetric Information System managed by the Department of Health Information [Malta]. Cases with a recorded pre-pregnancy body weight of 85 kg or more were identified. The Body Mass Indexes of these cases were then calculated to identify the study group with a BMI of 30 kg/m^2 or more. The medical records revealed a total of 1691 maternities in obese women resulting in 1721 births [26 twin and two triplet maternities]. The background population used for comparison [n = 18717 maternities and 18974 births] included all the women with a pre-pregnancy body weight of <85 kg, the women with a body weight of ≥85 kg but BMI <30 kg/m^2 and those whose pre-pregnancy body weight was not registered in the database. Screening for gestational diabetes is mainly dependant on historic and

Table 1: Maternal parameters and obstetric outcome

Maternal Parameters	Obese population BMI >=30 km/ m^2		Background population		*p value Obese vs background*
	No.	%	No.	%	
Maternal Age >=30 years	687	40.6	6735	36.0	*p<0.0001*
Previous Parity 1 or more	982	58.0	9042	48.3	*p<0.0001*
Previous miscarriages					
1 or more	322	19.0	3018	16.1	*p=0.0030*
2 or more	78	4.6	574	3.1	*p=0.0008*
Multiple Pregnancies	28	1.7	248	1.3	*p=0.3121*
Assisted Reproduction	17	1.0	146	0.8	*p=0.3953*
Accidental Haemorrhage	22	1.3	440	2.4	*p=0.0074*
Hypertensive disease:					
Pre-existing and gestational	347	20.5	1034	5.5	*p<0.0001*
Diabetes Mellitus					
pre-existing DM	9	0.5	79	0.4	*p=0.6452*
GIGT/GDM [*WHO criteria*]	188	11.1	202	1.1	*p<0.0001*
Induction of labour	778	46.0	6859	36.6	*p<0.0001*
Preterm delivery <37 wks	112	6.6	1253	6.7	*p=0.947*
Type of Delivery					
Elective Caesarean section	313	18.5	2354	12.6	*p<0.0001*
Emergency Caesarean section	293	17.3	2081	11.1	*p<0.0001*
Operative vaginal delivery	40	2.4	762	4.1	*p=0.0007*
Total Maternities	1692		18716		

Table 2: Maternal antenatal complications by age and weight

Maternal Parameters	Obese population <30 years age		Obese population =>30 years age		background population		*p value* <30 vs =>30yrs *Obese vs background*
	No.	%	No.	%	No.	%	
Hypertensive disease	178	17.7	174	25.3	1034	5.5	*p<0.0001*
Diabetes Mellitus							
pre-existing DM	3	0.3	6	0.9	79	0.4	*p=0.2113*
GIGT/GDM	90	9.0	101	14.7	202	1.1	*p<0.0001*
Total Maternities	1005		687		18716		

Table 3: Infant characteristics and outcome

Fetal Parameters	Obese population BMI >=30 km/ m^2		Background population		*p value Obese vs background*
	No.	%	No.	%	
Perinatal loss	17	1.0	171	0.9	*p=0.8212*
Shoulder Dystocia	10	0.6	83	0.4	*p=0.5059*
Low Birth Weight <2.5 kg	89	5.2	1252	6.6	*p=0.0248*
Macrosomia >4.0 kg	207	12.0	956	5.0	*p<0.0001*
RDS	53	3.1	440	2.3	*p=0.0596*
APGAR at 5 min <=6	29	1.7	314	1.7	*p=0.9989*
Infant Feeding - breast only	641	37.3	7495	39.5	*p=0.0782*
Sex					
Male	887	51.6	9705	51.1	
Female	832	48.4	9266	48.8	*p=0.7610*
Malformation	110	6.4	774	4.1	*p<0.0001*
Total births	1720		18975		

clinical risk criteria and is carried out in 7.2% of the whole pregnant population, identifying about 30.7% of expected cases.[4] The data analysis was carried out using SPSS package with statistical significance being tested using the Chi Square test. A probability value of <0.05 was taken to represent a significant correlation.

Results

The obese mothers were statistically more likely to be elderly aged 30 years or more [*p<0.0001*], multiparous [*p<0.0001*] with a history of previous miscarriages [*p=0.003*]. The antenatal period was more likely to be complicated by hypertensive disease [pre-existing and pregnancy-induced; *p<0.0001*], and gestational diabetes defined by a 75g OGTT 2-hour glucose level greater than 8.6 mmol/l [*p<0.0001*]. Obesity plays an important role in risk identification resulting in a high screening rate in obese individuals when compared to the non-obese. This may in part account for the high statistical significance in gestational diabetes rates noted in the study. They did not appear to have a significantly higher risk of pre-existing diabetes [*p=0.3267*] and did not require an increased risk of assisted reproduction requiring ovulation stimulation agents or other specialised technology [*p=0.3953*]. The multiple pregnancy rates were also not statistically increased [*p=0.3121*]. The obese individual were statistically less likely to have an antenatal period complicated by accidental haemorrhage defined as antepartum third trimester haemorrhage other than that caused by placenta praevia [*p=0.0074*] [Table 1]. The obstetric intervention rates were altered in the obese population with a statistically higher rate of induction of labour [*p<0.0001*] and Caesarean section rates [*p<0.0001*]. There was a statistically lower rate of operative deliveries including forceps and ventouse deliveries [*p=0.0007*]. The preterm delivery rates were not different [*p=0.947*] between the two groups of women [Table 1].

The differences in maternal morbidity rates from diabetes mellitus and hypertension were only in part related to the relatively higher age noted in the obese women. Thus, women aged less than 30 years still had a statistically higher risk of suffering from both hypertensive disease [background population 5.5% vs obese <30yrs 17.7% vs obese =>30yrs 25.3%] and gestational diabetes [1.1% vs 9.0% vs 14.7%] when compared to the background population. The risk for the development of these disorders increased with age. Pre-existing diabetes was higher in the older obese women [0.9%] when compared to both the younger obese woman and the background population [0.3%]. However the difference did not reach statistical significance [Table 2].

The infants born to obese women were at a statistically higher risk for macrosomia [more than 4.0 kg; *p<0.0001*] and a statistically lower risk for low birth weight [under 2.5 kg; *p=0.0248*]. They also had a statistically higher risk for congenital malformations. There appeared to be a slightly higher risk for respiratory distress though the differences did not reach statistical significance [*p=0.0596*]. There was no significantly increased risk for perinatal loss [*p=0.8212*], shoulder dystocia [*p=0.5059*], and low APGAR scores at five minutes [*p=0.9989*]. There was also no differences in the male:female ratios [*p=0.761*]. Infants born to obese women were less likely to be solely breastfeed at discharge from the hospital [approximately 2nd day postpartum], although the differences did not reach statistical significance [*p=0.0782*] [Table 3].

Discussion

Obesity was originally identified to be a high risk situation in pregnancy by Matthews and Der Brucke in 1938.[5] Since then a vast number of reports relating to obesity in the pregnant patients have been published, but many of the studies had methodological problems that have resulted in conflicting results between the various studies. No less than 37 obstetric complications have been reported as being more prevalent in moderately to severely obese women when compared with women of normal weight.[6] The present study has confirmed that the obese pregnant woman is at an increased risk of significant complications arising from hypertensive disease, haemorrhage, and gestational diabetes. She is also more likely to require obstetric intervention in the form of induction of labour, Caesarean section, and operative vaginal delivery. Her infant is also at risk of being macrosomic and possibly of being at greater risk of having respiratory problems at birth. The study fails to control specifically for maternal age and parity, the obese individual apparently being more likely to be aged more than 30 years and be multiparous. While age did influence the incidence of maternal complication, the younger obese woman was also more likely to suffer from the antenatal complications of hypertensive disease and gestational diabetes. A previous pilot study, which controlled for both age and parity, carried out in a sample of Maltese pregnant women aged less than 30 years and having their first child also confirmed the higher risk for hypertension, recurrent glycosuria and increased infant weight.[3]

The obese Maltese woman and her infant have a similar outcome indicator profile to that of the woman suffering from gestational diabetes even when an oral glucose tolerance test carried out in the third trimester proves normal glucose metabolism as defined by WHO criteria.[7] The relationship suggests that the underlying pathophysiology for the development of complications is insulin-resistance, these women being candidates for Metabolic Syndrome. Insulin-resistant individuals may initially compensate by producing increased insulin levels and thus control their blood glucose levels during their tolerance test. Metabolic Syndrome or Syndrome X in the young female has a multifaceted presentation. It can present as polycystic ovarian disease with menstrual disturbances that give rise to subfertility and an increased risk of recurrent miscarriage. While the present study failed to show a relationship between obesity and the use of assisted reproductive methods, the obese individual was shown to be statistically more likely to have a history of a previous miscarriage. The increased risk of hypertension during pregnancy in the obese individuals may also be related to the cardiovascular complications known to be associated with the Metabolic Syndrome in later life.

The effects of maternal obesity on the infant appear to be mainly that of a significant tendency towards macrosomia. The classic relationship between the maternal hyperglycaemia causing fetal hyperinsulinism causing macrosomia has long been accepted,[8] although this has been tempered by important contributions from other maternal fuels. Women with a high BMI and the associated insulin resistance are more likely to experience an increase in maternal glucose which could result in higher secretion of insulin in the fetus, eventually producing a bigger infant.[9] It is more difficult to explain the development of macrosomia in the presence of a normal glucose tolerance test and euglycaemia since the fetus of the euglycaemic obese woman can be presumed to have been exposed to normal mean glucose values and thus produced normal levels of fetal insulin and insulin-like growth factors. However, the development of macrosomia has been observed in fetuses born to obese women with normal glucose tolerance.[10,11] This suggests that the mechanisms promoting excessive fetal growth are more complex than the classic fetal hyperinsulinaemia causation, and the relationship of obesity with macrosomia may reflect an inherent insulin-resistance syndrome that, at the time of glucose tolerance testing, shows metabolic compensation. The relationship between placental receptors to maternal insulin and growth of the infant has still to be fully elucidated. The role of maternal lipids must also be elucidated. Studies have shown that maternal metabolic parameters generally associated with maternal overweight were independent determinants of macrosomia. Gestational diabetes was not independently associated with fetal macrosomia. Blood parameters known to be associated with the metabolic syndrome, such as high serum insulin and non-high density lipoprotein (HDL)-cholesterol and low serum HDL-cholesterol, were shown to be associated with an increased risk of macrosomia independent of BMI, weight gain, placental weight and gestational diabetes.[12] The birth of a macrosomic infant has been shown to be a predictor for the mother developing the metabolic syndrome later on in life.[13]

The macrosomic infant has been shown to have short-term adverse outcomes arising from problems during delivery.[14] Attitudes towards intrapartum management with higher induction rates to prevent postdates and higher Caesarean section delivery rates contribute towards decreasing previously described associated complications such as shoulder dystocia. However, even more important than the short-term complications are the long-term consequences of macrosomia. The macrosomic infant has been shown to be at a higher risk of developing the metabolic syndrome in adult life as a result of the altered intrauterine milieu interieur.[15] It thus appears that obesity remains a high risk situation in pregnancy resulting not only from the increased association of pregnancy medical complications of hypertension and diabetes, but also from the metabolic milieu brought on by the underlying Syndrome X. These as yet unexplained alterations, possibly related to increased fuel supply and hyperinsulinaemia, may also be the cause for the observed raised risk of congenital anomalies in infants of obese mothers, even in the absence of pre-existing diabetes.[16]

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