Int J Diabetes & Metabolism (2008) 16: 53-56

Maternal BMI and antenatal weight gain as determinants of obstetric outcome

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Abstract
Obesity during pregnancy has been shown to carry significant risk to both mother and child. The role of uncontrolled weight gain during pregnancy has not been fully elucidated. The study analysed the incidence of gestational hypertension and fetal macrosomia in various groups of mothers according to their BMI and antenatal weight gain. A definite statistically significant increased risk of both gestational hypertension and infant macrosomia was demonstrated with increasing BMI. This was compounded by increases in antenatal birth weight in all ranges of maternal BMI suggesting that adverse maternal metabolic parameters may contribute towards promoting the development of maternal and fetal complications. A restricted calorific intake in obese individuals during pregnancy may contribute towards decreasing the relative risks to both mother and child.

Key Words: *Anthropomorphy, fetal complications, macrosomia*

Introduction

The obese pregnant woman has been shown to have specific increased perinatal risks to mother and child. The fetal consequences of maternal obesity include excessive weight in the child.[1] The macrosomic infant has been shown to carry short-term consequences in the form of birth trauma to the infant.[2] Long-term consequences include the increased predisposition to develop Type 2 diabetes later on in life.[3] The pathophysiology behind the development of macrosomia may be attributable to a genetic predisposition towards obesity reflected by the maternal anthropometry and/or to intrauterine overfeeding especially in the presence of altered carbohydrate metabolism. This study sets out to investigate the role of antepartum weight gain reflecting nutritional intake on the development of macrosomia, irrespective of any possible genetic predisposition towards obesity.

Material and Methods

The study utilized the national database of all maternities delivering in the Maltese Islands during 1999-2006. Cases with absent data regarding height, pre-pregnancy weight, and weight at term were excluded from the study. The total number of patients analysed in the study thus amounted to 23424, or 72.4% of the total maternities. The population was subdivided into four basic groups on the basis of their BMI: BMI <20 kg/m^2 = 1484; BMI 20-24 kg/m^2 = 10070; BMI 25-29 kg/m^2 = 7300; and BMI >=30 kg/m^2 = 4567. Each group was further subdivided into different antenatal weight gain groups - <5 kg, 5-9 kg, 10-14 kg, and >=15 kg [Table 1]. The incidences of several maternal and infant outcome indicators were assessed by maternal BMI. The number of macrosomic infants defined as infants born with a weight of 4000 gm or more were identified in each of the sixteen groups. Furthermore the number of cases in each group of maternal gestational hypertension as a metabolic parameter of maternal adverse effect was identified. Statistical analysis within each group was carried out using the chi square and the student t tests as appropriate, with statistical significance being accepted at a probability <0.05.

Table 1: Study population subgroups

Study groups	Body Mass Index	Antepartum Weight gain	No.	%
A1		0-4 kg	31	2.1
A2	<20 kg/m^2	5-9 kg	310	20.9
A3		10-14 kg	583	39.3
A4		>=15 kg	560	37.7
A1-4		Total	1484	
B1		0-4 kg	412	4.1
B2	20-24	5-9 kg	2059	20.4
B3	kg/m^2	10-14 kg	3913	38.9
B4		>=15 kg	3686	36.6
B1-4		Total	10070	
C1		0-4 kg	503	6.9
C2	25-29	5-9 kg	1903	26.1
C3	kg/m^2	10-14 kg	2543	34.8
C4		>=15 kg	2354	32.2
C1-4		Total	7303	
D1		0-4 kg	727	15.9
D2	>30 kg/m^2	5-9 kg	1527	33.4
D3		10-14 kg	1335	29.2
D4		>=15 kg	978	21.4
D1-4		Total	4567	
A-D1		0-4 kg	1645	7.0
A-D2	Total	5-9 kg	5799	24.8
A-D3	population	10-14 kg	8374	35.8
A-D4		>=15 kg	7578	32.4
A-D1-4		Total	23424	

Received on: 7/11/2007
Accepted on: 9/06/2008

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Table 2: Outcomes by maternal BMI

Outcomes	A BMI <20	B BMI 20-24	C BMI 25-29	D BMI >30	A-D Total
BMI [kg/m^2] Mean ± SD	18.41 ± 0.92	22.24 ± 1.36	26.63 ± 1.40	33.9 ± 3.99	25.64 ± 5.14
Maternal Age [years] Mean ± SD	26.17 ± 5.90 p<0.0001	27.65 ± 5.94	28.47 ± 6.02 p<0.0001	29.02 ± 6.16 p<0.0001	28.08 ± 6.05
Antenatal weight gain [kg] Mean ± SD	13.39 ± 5.33 p=0.19	13.19 ± 5.47	12.32 ± 5.74 kg p<0.0001	10.22 ± 5.97 kg p<0.0001	12.36 ± 5.75
Artificial Reproduction p=0.82	13 0.9%	107 1.1%	72 1.0%	51 1.1%	243 1.0%
Pre-existing DM p=0.04	19 1.3%	13 0.1%	32 0.4%	10 0.2%	74 0.3%
Gestational DM p<0.0001	15 1.0%	66 0.7%	102 1.4%	204 4.5%	387 1.7%
Gestational hypertension p<0.0001	22 1.5%	331 3.3%	425 5.8%	689 15.1%	1467 6.3%
Infant birth weight [gm] Mean ± SD	3066 ± 496 p<0.0001	3200 ± 486	3293 ± 510 p<0.0001	3340 ± 552 p<0.0001	3248 ± 513
Macrosomia p<0.0001	28 1.9%	390 3.9%	493 6.8%	432 9.5%	1343 5.7%
Caesarean delivery p<0.0001	294 19.8%	2300 22.8%	2001 27.4%	1674 36.7%	6269 26.8%
Dystocia p<0.05	3 0.2%	35 0.3%	34 0.5%	29 0.6%	101 0.4%
Respiratory Distress p=0.02	31 2.1%	211 2.1%	190 2.6%	130 2.9%	562 2.4%
Stillbirth & Neonatal death p=0.10	10 0.7%	55 0.6%	59 0.8%	39 0.9%	163 0.7%

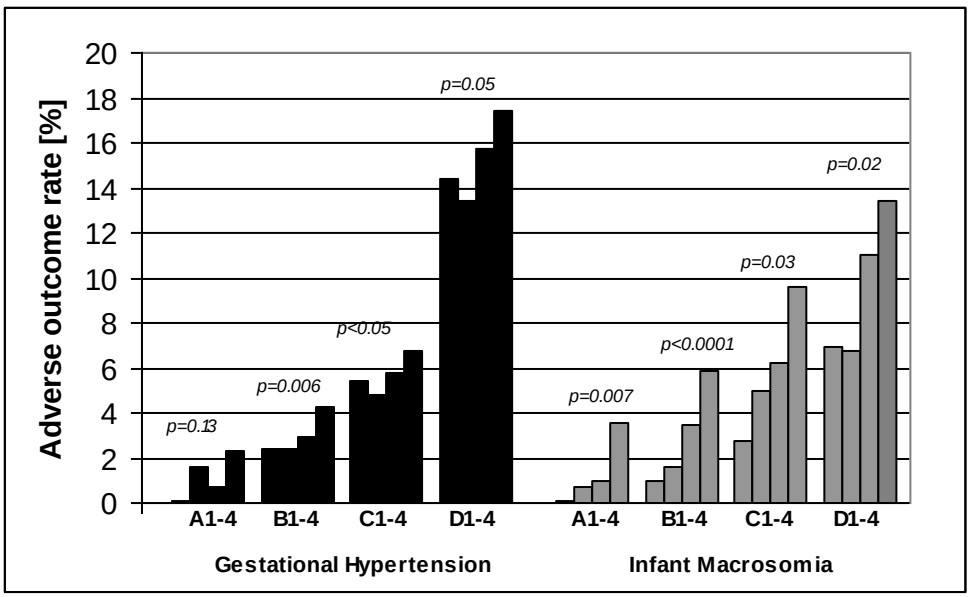

Figure 1: Adverse outcomes by antenatal weight gain and BMI

Results

Body Mass Index alone is a definite determinant adverse maternal condition reflected by the increasing development of gestational hypertension and fetal condition as determined by increasing rates of macrosomia [Table 2]. The mean antenatal weight gain appeared to be statistically increased with increasing BMI values when compared to the weight gain in the lean BMI group. The incidence of gestational hypertension increased significantly with increasing BMI from a rate of 1.5% in those women with a BMI <20 kg/m^2 to 15.1% in those women with a BMI >30 kg/m^2 [p<0.0001]. The mean maternal age seemingly increased statistically with increasing maternal BMI. Pre-existing diabetes, mainly IDDM, was statistically more common in the lean group of mothers [p=0.04]. Conversely diagnosed gestational diabetes was statistically more common in the severely obese group [p<0.0001]. The latter observation, however, may reflect a greater predisposition for the obese individuals to be screened by an OGTT when compared to the lean group. There was no statistically significant difference in the need for artificial reproductive techniques to be used to achieve a pregnancy. The mean infant birth weight similarly increased progressively with increasing BMI, all the differences being statistically significant when compared to the lean group. The incidence of macrosomia was also shown to increase progressively

with increasing BMI from 1.9% in those women with a BMI of <20 kg/m^2 to 9.5% in those women with a BMI >30 kg/m^2 [p<0.0001]. The incidence of caesarean delivery was significantly increased in the obese individual [p<0.0001]. In spite of the increase in caesarean births, dystocia was still higher in the obese individuals [p<0.05]. The incidence of respiratory distress was also significantly increased [p=0.02]. There was no statistically significant increase in the stillbirth and neonatal death rates.

Thus, increasing BMI alone increases the relative risks of the mother developing gestational hypertension and for the infant to be macrosomic. These risks appear to be further statistically increased with increasing antenatal weight gain in all subgroups [Figure 1] confirming that the woman most at risk of developing gestational hypertension and having a macrosomic infant is the grossly obese woman whose antenatal weight gain is excessively disproportionate.

Discussion

The Maltese population comprises a small island community in the Central Mediterranean with a high prevalence of obesity and Type 2 diabetes mellitus reflected in a high prevalence of gestational carbohydrate intolerance.[4,5] The aetiology for the high prevalence of obesity and type 2 diabetes in this population has not been completely elucidated, though several epidemiological studies have suggested an adverse intra-uterine nutritional environment as a contributor.[6] A genetic element may also be functional, though this has yet to be identified in this high risk population.[7] The genetics of NIDDM are still not well understood though evidence for a genetic susceptibility to developing NIDDM has been presented, and defects in both insulin production and action are suspected.[8] Other receptor genes such as the B3-adrenergic receptor gene have been suggested as factors towards the development of weight gain, insulin resistance and the earlier onset of NIDDM. This gene polymorphism is predominantly expressed in adipose tissue and regulates lipid metabolism and thermogenesis, so its impairment may lead to obesity through its effect on the energy expenditure of fat tissue. The reported data so far remains conflicting.[9]

The obese pregnant Maltese woman has been shown to have specific increased perinatal risks to mother and child. Obese mothers were characteristically more likely to be elderly, multiparous with a history of previous miscarriages. Their antenatal period was more likely to be complicated by hypertensive disease and gestational diabetes. The obese women were also more likely to require obstetric interventions. Their infants were more likely to be macrosomic.[1] The increased predisposition towards gestational hypertension probably reflects an inherent tendency towards developing essential hypertension as a feature of the Metabolic Syndrome later on in life in a process similar to the relationship between gestational diabetes and the eventual onset of Type 2 DM. The inter-relationship between maternal obesity and carbohydrate intolerance to fetal macrosomia has been repeatedly demonstrated in several populations, including the Maltese. The incidence of fetal macrosomia, defined as a birth weight greater than 4000 gm, has been reported in the Maltese population to have decreased from 11.5% in 1983-86 to 5.9% in 1999-2002. While this fall may be attributed in part to an increase in the obstetric intervention rate with earlier timing of delivery, changes in the socio-biological characteristics of the mothers such as age at delivery patterns may also have played a determinant role.[10] Macrosomic babies have been shown to be more common in the older obese previously diabetic woman aged more than 30 years who had at least one previous pregnancy or miscarriage. The larger size of the infant imposes greater risks of birth trauma to the infant and the necessity of operative intervention to achieve a safe delivery.[2] This has again been demonstrated in this study.

The predisposition towards macrosomia in the Maltese population may thus be attributed to adverse intrauterine nutritional environment possibly superimposed on a genetic predisposition. Women with an elevated BMI, and a predisposition towards developing the Metabolic Syndrome later on in life, may have an inherent genetic predisposition that is made worse with increasing body weight brought on by an excessive unhealthy diet. The interrelationships between pre-pregnancy BMI and weight gain during pregnancy on the developing fetus have not been adequately investigated. The present study has confirmed that the increasing BMI predisposes to both the development of gestational hypertension and infant macrosomia. The tendency for an obese individual to give birth to a macrosomic infant may suggest a genetic element. However, the relationship between the various maternal metabolic parameters generally associated with maternal overweight and the development of macrosomia still needs to be elucidated fully. Studies have shown that maternal metabolic parameters generally associated with an elevated maternal BMI were independent determinants of 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.[11] The present study has further shown that irrespective of the pre-pregnancy BMI, excessive weight gain during pregnancy, presumably brought on by an uncontrolled unhealthy diet, also predisposed towards the development of gestational hypertension in the mother and macrosomia in the infant. This observation suggests that adverse maternal metabolic parameters contribute towards promoting the development of maternal and fetal features associated with an underlying Metabolic Syndrome.

The obese individual with an elevated BMI has definite risks to herself and her infant. These risks are augmented by excessive weight gain during pregnancy. Prenatal counselling of these women should aim at body weight reduction prior to becoming pregnant. Careful dietary advice during pregnancy aimed at reducing the excessive calorific intake in obese individuals may contribute towards decreasing the relative risks observed in both mother and child.

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