The relationship of weight gain and foetal macrosomy in gestational diabetics

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ABSTRACT

A retrospective analysis of the causal relationship between weight gain in pregnancy and foetal macrosomy was conducted on obese and non obese gestational diabetics who completed term under our care. One hundred and twenty five gestational diabetics were studied in retrospect with regard to their caloric intake, insulin requirements, glycemic control, net weight gain, timing and mode of delivery and the outcome of pregnancy, the birth weight and neonatal complications.

The gestational diabetics were categorised into those with large for gestational age infants (LGA) and those with appropriate for gestational age infants (AGA). The groups were further subdivided as Obese, (O) and Non obese (NO) on the basis of BMI and per cent of ideal body weight.

The data obtained revealed that foetal macrosomy has a direct causal relationship with weight gain in pregnancy; restriction of weight gain in pregnancy is associated with birth of AGA infants.

INTRODUCTION

The current tenets of nutritional therapy in gestational diabetics include the advocation of a balanced meal plan in order to attain the desired weight gain in pregnancy and achieve and maintain euglycemia with a view to obviate both maternal as well as foetal morbidity (1). In keeping with these conventional tenets we could achieve most goals of therapy but could not obviate the morbidity peculiar to the infant of the diabetic mother (IDM) in obese pregnant diabetics (Figure 1). However, on restricting the net weight gain in pregnancy by a controlled calorie meal plan (CCMP) we could achieve all goals, including the obviation of neonatal complications. These gestational diabetics had a bad obstetric history and had previously delivered large for gestational age (L.G.A.) infant (2). Consequent to our experiences with the CCMP, we have routinely employed the same in all gestational diabetics entrusted to our care. It was in this context, that we decided to conduct a retrospective analysis of our experience

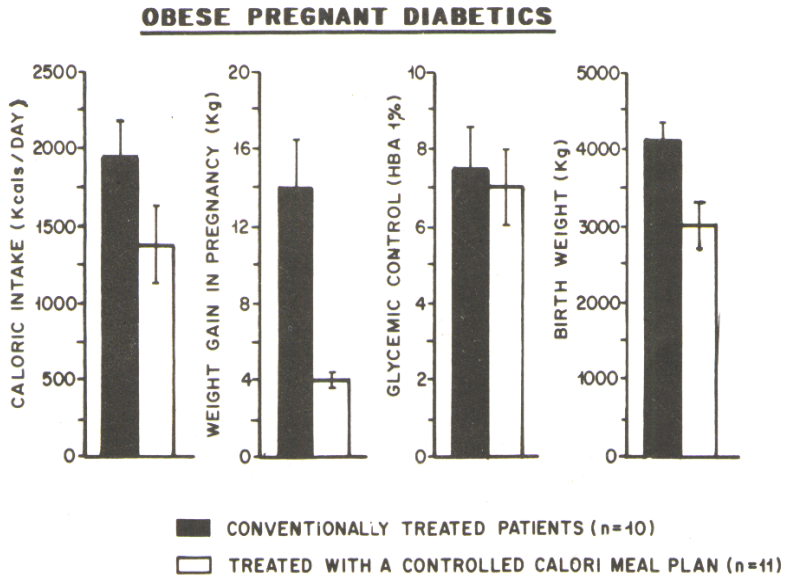

Fig. 1. The effect of Net Weight Gain in Pregnancy on the Glycemic Control of the mother and the Birth Weight of the Infant of the Obese Diabetic Mothers

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Table 1
Gestational Diabetics with large for gestational age infants

A: Previous Pregnancies

Parameter	Obese GDs with LGA infants (n=14)	Non-obese GDs with LGA infants (n=16)
Successful outcome	13 out of 31	6 out of 21

B: Index Pregnancy

Parameters	Obese GDs with LGA infants (n=14)	Non-obese GDs with LGA infants (n=16)	p value
BODY MASS INDEX (kg/M^2)	29.5 + 9.8	22.5 + 2.8	0.05
CALORIC INTAKE (kcals/day) INITIAL AT TERM	 1860 ± 408* 1530 ± 252*	 2080 ± 321* 1500 ± 100*	 NS NS
WEIGHT GAIN (kg)	10.5 ± 3.3	12.5 ± 4.4	NS
GLYCEMIC CONTROL Mean Post Lunch Blood glucose (mg/dl) INITIAL AT TERM	 166 ± 43@ 123 ± 36#	 166 ± 38@ 102 ± 27#	 NS NS
INSULIN REQUIREMENTS Units/day	62 (n=6)	44 (n=4)	0.05
TIMING OF DELIVERY (weeks)	35.3	35.3	
MODE OF DELIVERY L.S.C.S. Normal/Assisted	 14 0	 14 2	
BIRTH WEIGHT (kg)	3.8 ± 0.6 90^{th} centile	3.5 ± 1.0 90^{th} centile	NS
NEONATAL COMPLICATIONS Hypoglycemia Respiratory Distress Hyperbilirubinemia Hypothermia	 3 2 3 1	 2 1 1 0	

*versus **and @versus # are statistically significant ($p < 0.05$)

Table 2
Gestational Diabetics with appropriate for gestational age infants

A: Previous Pregnancies

Parameter	Obese GDs with LGA infants (n=47)	Non-obese GDs with LGA infants (n=48)
Successful outcome	6 out of 22	16 out of 67

B: Index Pregnancy

Parameters	Obese GDs with LGA infants (n=47)	Non-obese GDs with LGA infants (n=48)	p value
BODY MASS INDEX (kg/M^2)	30.5 + 3.9	29.0 + 6.0	0.05
CALORIC INTAKE (kcals/day)			
INITIAL	1860 + 400*	1980 + 300*	NS
AT TERM	1575 + 165*	1575 ± 165*	NS
WEIGHT GAIN (kg)	6.4 + 4.1	9.0 + 3.5	NS
GLYCEMIC CONTROL Mean Post Lunch Blood glucose (mg/dl)			
INITIAL	174 + 68@	159 + 57@	NS
AT TERM	123 + 7.0#	102 + 32#	NS
INSULIN REQUIREMENTS Units/day	33 (n=8)	24 (n=3)	
TIMING OF DELIVERY (weeks)	37.4	38	
MODE OF DELIVERY			
L.S.C.S.	30	24	
Normal/Assisted	17	24	
BIRTH WEIGHT (kg)	2.8 + 0.4 $10\text{-}90^{th}$ centile	2.8 + 0.4 $10\text{-}90^{th}$ centile	
NEONATAL COMPLICATIONS			
Hypoglycemia	0	0	
Respiratory Distress	0	0	
Hyperbilirubinemia	0	0	

*versus **and @versus # are statistically significant ($p < 0.05$)

regarding the role of maternal weight gain in pregnancy in the genesis and obviation of foetal macrosomy. The objective of the study was to evaluate the effect of net weight gain on the glycemic control, insulin requirements, timing and mode of delivery in the mother and birth weight of the infant and neonatal complications peculiar to IDMs, while on a controlled calorie meal plan.

MATERIAL AND METHODS

One hundred and twenty-five pre-gestational and gestational diabetics who had successfully completed term under our care between January, 1985 and November, 1990 were analysed in retrospect with regard to their caloric intake, net weight gain in pregnancy, insulin requirements, glycemic control, timing and mode of delivery, the birth weight and neonatal complications peculiar to the IDM.

The patients were detailed regarding their medical illness and food intake, followed by a thorough clinical examination. A record of their height, pre-gestational and existing weight and vital functions were noted.

The patients were diagnosed as obese (Body Mass Index > 25 kg/m^2 or percentage body weight > 120%) and non-obese (Body Mass Index 18-25 kg/m^2 and percentage body weight < 110%).

The laboratory investigations included a haemogram, assessment of glycemic status (OGTT, fasting and post cibal blood glucoses, glycated hemoglobin) and renal function (BUN, creatinine and urinalysis). The patients were taught home blood glucose monitoring, examination of urine for glucose and ketones by ketodiastix. They were explained the basic principles of nutrition in diabetes and pregnancy and the use of an individualised dietary exchange system. Those requiring insulin were familiarised with its usage. The patients were advised a controlled clorie meal plan (CCMP). These are low calorie diets designed to restrict weight gain in pregnancy and obviate the morbidity peculiar to IDMs (2). The diets are designed to prevent ketosis and intrauterine growth retardation, which may occur with caloric restriction in pregnancy (3,4). Ketosis is prevented by ensuring an adequate intake of carbohydrate (a minimum of 180 g/d of carbohydrates incorporated in CCMP). Urine was checked daily for ketones, which if positive in the absence of glycosuria, an increase in the carbohydrate intake was made. Intrauterine growth retardation is prevented by providing an optimal amount of carbohydrate and protein intake and growth of the foetus monitored by serial ultra sonographic examination. Any evidence of intrauterine growth retardation was appropriately handled by dietary manipulation which was found to be effective even in the last month of pregnancy (5).

Descriptive statistics were employed for calculating the mean and standard deviation. The two independent sample 't' test was employed for testing the mean. A value of less than 0.05 was considered to be statistically significant.

RESULTS

Results are presented in Table 1 and 2. As seen from these tables, obese and non obese diabetics who had he usual desired weight gain in pregnancy, delivered large for gestational age infants with neonatal complications peculiar to IDMs in spite of the fact that euglycemia was attained and maintained in this group. The obese subjects required significantly larger doses of insulin when compared to non-obese subjects for achieving a similar degree of glycemic control.

Obese and non-obese gestational diabetics in whom weight gain was restricted during pregnancy, delivered appropriate for gestational age infants without the neonatal complications peculiar to IDMs. They also attained and maintained euglycemia during pregnancy. The obese diabetics required significantly larger doses of insulin when compared to non-obese diabetics.

DISCUSSION

Foetal macrosomy and weight gain in pregnancy amongst gestational diabetics appear to have a positive correlationship, for it was observed that weight gain beyond the 50^{th} percentile was associated with birth of a large for gestational age infant and neonatal complications in the IDM, whereas restricting weight gain in pregnancy to less than the 25th percentile was associated with the obviation of foetal macrosomy and complications thereof. The recommendations for weight gain in pregnancy are based on the Harvard standards (6); the present study demonstrates the inefficacy of applying these standards in our country and highlights the need for the development of norms for weight gain in our country.

Restricting weight gain in pregnancy appears to be an age old practice employed by the South African pedis (7) in women who had borne large babies

earlier or had suffered from cephalo-pelvic disproportion. Caloric restriction led to birth of smaller babies and thus obviated intra-uterine foetal deaths and facilitated normal deliveries in these women.

The problems associated with caloric restriction in pregnancy (3,4,5) can easily be circumvented by employing the controlled calorie meal plan, which is a safe and effective mode of therapy in gestational diabetics. Several authorities have expressed their views on the need to curtail the caloric intake in gestational diabetics (8,9,10), thus obviating foetal macrosomy.

In spite of our efforts to minimise weight gain in all our patients by using hypocaloric diets, our patients can be stratified into those whose weight gain was usual (Table 1) and those in whom the weight gain could be restricted (Table 2). The patients in the former group demonstrated foetal macrosomy. They were usually late referrals and demonstrated accelerated foetal growth. We also found that it was difficult to retard foetal growth in these late-referral patients. The other reasons for difference in the weight gain in the two groups is attributed to poor compliance, incorrect dietary recalls, excessive salt and water intake, edema of pregnancy and possibly the hormonal changes that take place in pregnancy (12).

We conclude by suggesting that excessive weight gain in pregnancy may be one of the several determinants of foetal macrosomy in pregnancy (13,14,15). The restriction of weight gain in obese as well as non obese gestational diabetics is associated with obviation of both foetal macrosomy and the neonatal complications peculiar to the infant of the diabetic mother.

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