PERINATAL MORTALITY AND MORBIDITY IN DIABETES MELLITUS COMPLICATING PREGNANCY

Dissertation submitted for

M.D., Branch II

OBSTETRICS AND GYNAECOLOGY

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

FEBRUARY 2006

BONAFIDE CERTIFICATE

This is to certify that this dissertation titled '**Perinatal Mortality and Morbidity in Diabetes Mellitus Complicating Pregnancy'** has been done by **Dr. S. Sampathkumari** is her original work and this manuscript is an outcome based on the results of the study conducted by her at this institution. This dissertation is submitted in partial fulfillment of The Tamilnadu Dr.MGR Medical University, Chennai regulations for the award of the degree M.D., (Obstetrics & Gynaecology).

Dr. V. Madhini M.D., D.G.O., M.N.A.M.S.,
Director & Superintendent
Institute of Obstetrics & Gynaecology &
Govt. Hospital for Women and Children
Madras Medical College
Chennai.

ACKNOWLEDGEMENT

I sincerely wish to thank and record my deep sense of gratitude to the following persons for their valuable help and timely advices, without whose encouragement and guidance this study would not have progressed and reached the shape it is today.

Dr.Kalavathy Ponniraivan B.Sc.,M.D., Dean, Madras Medical College, Chennai
Dr. Shakundala Barathi M.D, D.G.O., (Retd) Director of IOG
Dr. V. Madhini M.D., D.G.O., M.N.A.M.S., Director & Supdt. of IOG, Chennai
Dr. R.S. Hariharan M.D., Professor of Diabetology, Govt. GH & Madras Medical College, Chennai
Dr.Arasan Seeralan M.D., Neonatalogist, I.O.G, Chennai and
Dr.Padmavathy Palanisamy M.D, D.G.O, Gynaecologist, I.O.G, Chennai

CONTENTS

INTRODUCTION

Introduction

" In every child who is born under no matter what circumstances and of no matter what parents, the potentiality of human race is born again, and in him too, once more and each of us our terrific responsibility towards human life" James Agee

Gestational Diabetes mellitus and other categories of glucose intolerance during pregnancy are one of the causes for increased perinatal mortality. But nowadays with pre conceptional counselling, early screening and advances in management of diabetic pregnancy have reflected in the continuous reduction mortality and morbidity in the infants of diabetic mothers**.**

Hypoglycemia, hyperbilirubinemia are managed with little difficulty. Respiratory Distress Syndrome and Macrosomia are now largely preventable with good glycemic control. The unresolved problem at present is the prevention of malformation in diabetic pregnancy.

With the above in mind the perinatal outcome at Institute of Obstetrics and Gynaecology, attached to Madras Medical College, Chennai was analysed for possible clues in effective management of diabetic mothers who turn up at the institute.

.

REVIEW OF LITERATURE

Review of Literature

History

The history of Diabetes Mellitus is as old as human kind. However, en Egyptian papyrus inscription of 1500 B.C has the first record of Polyuria related to diabetes. The disease was prevalent in the days of Hippocrates (466 – 372 B.C) and was considered rare and incurable. The symptom was noted as ‘ to make water much or often’ a condition known to be ‘wasting of the body’.

The naming of the disease as Diabetes is credited to Aretaeus of Cappadocia (AD 30 – 90). The term diabetes means to pass through or to siphon. The severity of the illness and the frustration and hopelessness it caused was not only to the patient but also to the physicians as well. Celsus (30 BC – AD50) described the individuals with diabetes as patients with discharge of urine greater than the amount of fluid taken in by mouth.

Avicenna (AD 980 – 1027) has observed in his writing about Arabian medicine that diabetic patients have irregular diet associated with thirst, mental exhaustion, inability to work and loss of sexual function. He was alarmed by these factors and believed that diabetes affected the liver and caused its enlargement. The Hindu writings of the sixth century refer to the ailment as honey urine.

Although observations on diabetes were seen in almost all parts of the world attempts on possible causes were witnessed only in the recent centuries. Sylvanus (1478 – 1555) opined that diabetes was a disease of blood and Willis Wyatt (1621 – 1675) explained that the sugar present in the urine of diabetics represented excretion of sugar that was initially in the blood. In 1682, Brunner created history bay making an animal model and his experiment was corroborated in Cawley’s (1688) diagnosis that the disease resulted from the injury to pancreas.

18^{th} century experiments paved the way for systematic treatment and therapeutic approach to deal with diabetes. Physicians by now were convinced that the disease was caused by the inability of the body to handle carbohydrates properly. It was Rolls in 1797, who had attempted to control the disease by dietary management. Bouchardat (1806 – 1866) advocated for dietary management and exercise by the use of green vegetables, fresh fats

substituting carbohydrates, avoidance of milk and alcohol. Naunyn too suggested dietary management, as he believed that when the patient is sugar free his tolerance level increases. Therefore the Alpha and Omega care of diabetes is dietary treatment and not drugs was his ultimate conclusion. Sheffer in 1895 suggested that the islets of Langerhans when diseased produced daibetes. Opie of U.S further amplified this in 1901. The degeneration of the islets of Langerhans in the pancreas altered the form and the function of the internal secretion that caused diabetes was his theory. Minkowski and von Mering experimented a dog by conducting pancreatectomy. Thus the work of Minkowski and the observation of Opie began to close the link between the islet cell disease and diabetes.

Discovery of Insulin

Paulesco, a Physiologist from Bucharest in1916 extracted pancreine from pancreas which when injected gave a temporary relief to the pancreatectomised dog. Zuelzer et al (1908) prepared a improvised version prepared an extract from the expressed juice of the pancreas. This was tested on pancreatectomized dogs with hyperglycemia and ketonuria, eight diabetic humans and four other humans with ketosis. This experiment came close to the discovery of insulin by Banting and Best in 1921.As we all know good science is created in the morning. Banting apparently awoke at 2.00 a.m. came up with the idea of preparing insulin.

Diabetes in Pregnancy

Until 1856 there was no report of DM complicating pregnancy. Blott had written that ‘true diabetes in inconsistent with conception’. In 1892, a report published by Duncan, described 22 pregnancies with diabetes. In 1909 Reel collected 66 cases, 27 % died at the time of labour or within 1 – 2 weeks afterwards, another 22 % died within 2 years, one-eighth of pregnancies ended in abortion and one-third that went to term the baby was born dead. Beunewitz quoted a series of 19 cases, 10 of which died during labour or shortly thereafter. This trend continued until the discovery of insulin.

Diabetes was considered as a disease with dismal prognosis and reproductive success was not common and pregnancy worsened the disease. In 1920 De Lee wrote that sterility was common among diabetes probably due to the atrophy of the uterus and ovaries, causing premature menopause, abortion and premature labour in 33 % of pregnancies. The foetus

died when pregnancies went to term with an overall perinatal mortality of 60 – 70 %. 30 % of the mothers died due to ketoacidosis.

The advent of Insulin

Insulin drastically reduced the maternal mortality from 45 % to just over 2 % shortly but the perinatal mortality did not come down rapidly. Very large babies and associated traumatic injury to the foetuses and the mothers during parturition was noy helped by insulin. The problem of neonatal hypoglycemia, congenital malformations, toxemia of pregnancy and the like continued to haunt affecting the outcome.

It was also observed that beyond 36 weeks of gestation the risk of still birth rose significantly – an alarming ten fold increase, and foetal mortality was about 25 %, if the birth weight was 3.2 kg or 70 % if the weight was 4.5 kg. Due to this diabetic mothers were delivered routinely on or before 36 weeks by section or by induction if foetal death had not already occurred or when maternal complications necessitated.

Following years saw the emergence of newer protocols aimed at improving the foetal outcome. White claimed in 1945 a 97 % foetal survival with normal hormonal balance and a 47 % survival in abnormal hormone balance. Pedersen of Copenhagen in 1954 showed that the foetal mortality rate was lower when being followed over a long period than in those who were seen first at or about the time of delivery. Advances like new types of insulin (long acting type and human insulin) and tools like estriol measurement, USG, antepartum foetal heart rate testing, foetal blood sampling techniques, glucose meters, insulin pumps, neonatal intensive care units are being used to improve the foetal outcome. Karlsson & Kjellmer's retrospective study established a relationship between glycemic control and perinatal mortality. This led to a new trend in diabetics care. Stringent maintenance of ambient glucose level resulted in average perinatal mortality rate less than 5 %. The cause of maternal deaths has shifted from diabetic ketoacidosis to cardiac renal complications. The impressive strides achieved in diabetic care these days have enabled expectant mothers to enjoy pregnancy performance and foetal outcome similar to that of non-diabetic patients. Yet problems like macrosomia and congenital malformations still remain unresolved. The incidence of birth defects is reported to be 6 – 12 %. Clinical and laboratory studies suggest that these malformations are caused by the derangement in metabolism during organogenesis.

Gestational Diabetes Mellitus

GDM defined as ‘ Carbohydrate intolerance of varied severity with the onset or first recognition during the present pregnancy’. It is estimated to occur in 2 – 5 % of pregnancies.

Classification

Classification of Diabetic mothers

Revised Whites’ Classification (1980)

I Gestational Diabetes
Abnormal GTT, but euglycemia maintained by diet alone
If diet alone is insufficient, insulin is required

II Pregestational Diabetes

NDDG Classification (1979)

Type I	IDDM	Insulin dependent DM
Type II	NIDM	Non Insulin dependent DM
Type III	GCI	Gestational Carbohydrate Intolerance
		Non Obese & Obese

Fuel Metabolism in normal pregnancy

Pregnancy is characterized by maternal adaptations such as facilitated insulin action during the first half of pregnancy and diabetogenic stress during the second half of the pregnancy. The metabolic changes of pregnancy in the fed state are dominated by a single characteristic; resistance to glucose lowering effects of insulin. These changes appear to be related to the effect of hormones including human placental lactogen, HPL, pregesterone, prolactin and cortisol. The progressive insulin resistance of pregnancy results in large part from metabolic actions of hormones. As a result, one of the main forces exerted by the conceptus on maternal metabolism causes the blunting of insulin’s action on glucose uptake in insulin target tissues (i.e., skeletal muscle and adipose tissue). The reduction in insulin-mediated glucose uptake that occurs in pregnancy results from inefficient coupling between activation of insulin cell surface receptors and translocation of GluT4 molecules to the cell surface.

The metabolic changes in fasting state in pregnancy are similar to that occurring during fasting in non-pregnancy but in a rapid and greater degree. Thus Freinkel has referred to the metabolic profile of fasted women as ‘accelerated starvation’. The two major components of accelerated starvation are (1) reduction in circulating glucose concentration and (2) accelerated lipolysis and ketogenesis.

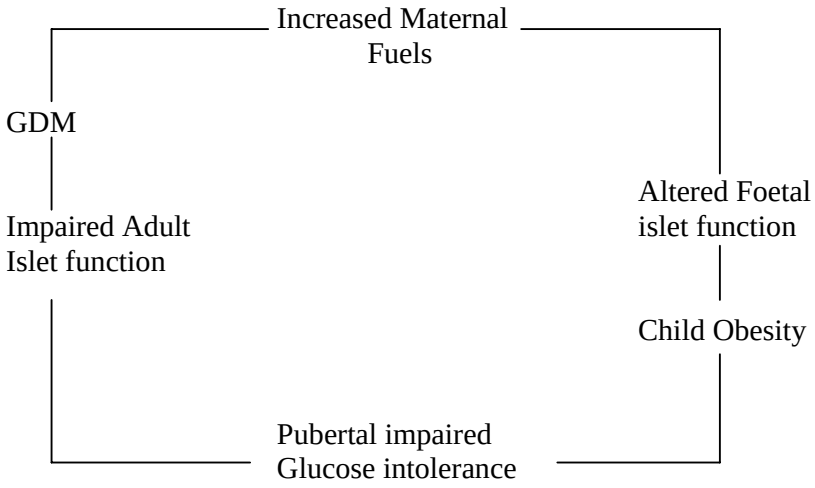
In short, facilitated anabolism in the fed state and accelerated starvation in the fasted state characterise the maternal fuel adaptations during pregnancy. These hormonal and metabolic changes are geared towards facilitated anabolism under the actions of insulin throughout pregnancy. However in the second half of the pregnancy, insulin resistance and diabetogenic stress necessitate compensatory increased insulin secretion. Hence the clinical expression of gestational diabetes, where this compensation is inadequate, usually occurs during the second half of pregnancy Fuel Metabolism in Diabetic Pregnancy

The effect of diabetic pregnancy on fuel metabolism is one of under utilisation of exogenous fuel in the fed state and over production from endogenous sources in the fasted state.

	Fasted State	Fed State
Normal pregnant state	Accelerated starvation	Facilitated anabolism
Diabetic Pregnant state	Hyper accelerated Starvation	Reduced facilitated anabolism

Diabetes begets diabetes

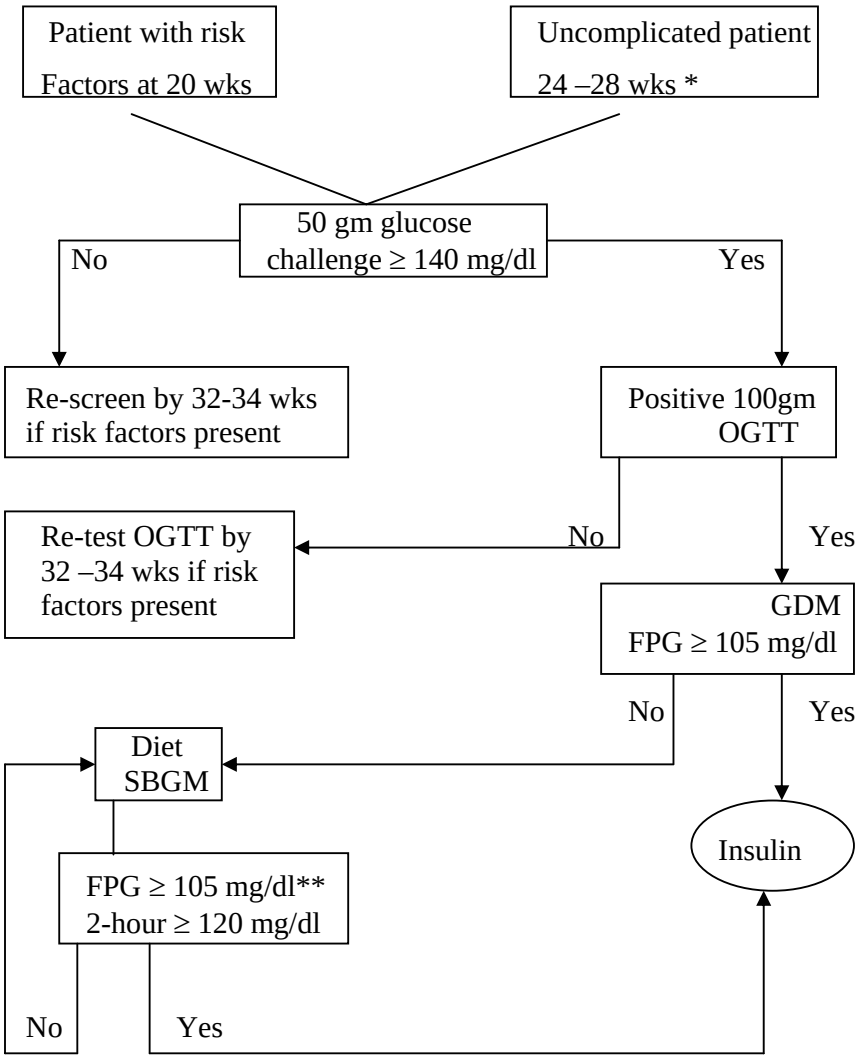
Reece suggests the chain of events in that many of the offsprings of diabetic mothers will be predisposed to GDM in second generation. This suggests that DM can predispose to more DM thus contributing to the overall increasing burden of DM in the population.



However this process is potentially preventable by research works more effectively done to find a solution for early diagnosis and prevention of GDM.

Screening

Screening for GDM is performed with 50 gm oral glucose load between 24 and 28 weeks followed by 1 hr. venous plasma glucose level. Screening test is performed when diabetologic effect of pregnancy is peaking[30]. If the test reveals a result ≥ 140 mg/d, patient is scheduled for 3 hr 100gm – OGTT.



SBGM = Self blood glucose monitoring
FPG = Fasting plasma glucose

* Screening is not required if the patient meets all of the following criteria:

1) Less than 25 years of age
2) Normal body weight
3) No first degree relatives with DM
4) Not of Hispanic, Native American, Asian, African – American ethnic groups

** Initiation of insulin therapy at FPG of 95 mg /dl has been advocated by some investigators

Diagnosis

Early detection and appropriate treatment of GDM provides the opportunity to prevent adverse outcomes for a mother and their children.

Diagnostic criteria for GDM using 100 gm OGTT

	O'Sullivan 21*	NDDG 48**	Carpenters & Coustan 49*
Fasting	90 mg/dl	105	95
1 hour	165 mg /dl	190	180
2 hour	145 mg /dl	165	155
3 hour	125 mg /dl	145	140

* Venous whole blood, Somogyi – Nelson analysis ** Plasma glucose oxidase

The diagnosis requires any two values to meet or exceed those listed above.

ACOG and the expert committee on the diagnosis classification of DM recommend NDDG criteria for the diagnosis of GDM. ACOG recommended GDM screening based on risk factors[32]. Others said that half of all patients GDM have no identifiable risk factors. Latest ACOG Technical Bulletin on Diabetes and Pregnancy suggest selective screening in low risk clinical setting such as teen clinics and universal screening in other settings[31].

Risk factors requiring Diabetic screening

1) Obesity (>15 % of non pregnant ideal body weight or BMI > 29 %)
2) Positive family h/o diabetics
3) History of still birth
4) History of delivery of large infant
5) Glycosuria
6) History of unexplained neonatal death
7) History of congenital anomaly
8) History of prematurity
9) H/o pre eclampsia as a multipara
10) Poly hydramnios
11) H/o traumatic delivery with associated neurogenic disorder of infant
12) Poor reproductive history
13) Chronic Hypertension
14) Recurrent UTI or severe moniliasis
15) Age > 30 years
16) H/o Diabetics in previous pregnancy

Maternal Morbidity

Many studies have documented an increase in pre eclampsia, Polyhydramnios and operative delivery in pregnancies complicated by GDM[33,34,35]. The Toronto Tri-Hospital Gestational project, a prospective cohort study evaluating maternal and foetal outcomes with increasing carbohydrate intolerance, observed significant association between glucose intolerance and increased incidence of caesarian delivery, pre eclampsia and length of maternal hospitalisation.

Women with GDM also have significant risk of developing diabetes later in life. Coustan and associates studied former GDM women and found diabetes or impaired glucose tolerance (GTT) in 6 % of the those tested 0 – 2 years, 13 % at 3 – 4 yrs, 15 % at 5 - 6 yrs and 30 % at 7 – 10 yrs post partum. Other studies have documented type 2 diabetes 3 – 5 years post partum in 30 – 50 % of women who had a pregnancy complicated by DM.[36, 37]

Peter and associates found that episodes of insulin resistance due to additional pregnancies increased the rate of developing type 2 diabetes independent of pregnancy – associated weight gain. They also found the relative risk for type 2 diabetes was 1.95 for every 10 pounds gained during follow-up adjusting for the number of pregnancies and other risk factors.[38]

Perinatal Mortality and Morbidity

Infants of mother with GDM (IGDM) are not at increased risk for congenital anomalies unless these women have pre-existing DM. However, IGDM do have an increased risk of perinatal mortality and morbidity including hyper bilirubinemia, macrosomia and birth trauma, hypocalcemia and hypoglycemia.

O'Sullivan observed a fourfold increase in perinatal mortality rates in pregnancies complicated by improperly managed GDM.[39] Several other studies have found an increased rate of still birth in untreated GDM.[40] Today in pregnancies identified and treated appropriately, intra uterine foetal demise is not increased in GDM.

Silverman studied the children of women with both pre GDM and GDM and found IGT in adolescent children were 13 times more frequency than control adolescents.[41] Silverman documented that by 8 years of age 50 % of children of diabetic mothers had weights above

the 90th percentile compared to children of women without diabetes[42]. Plagemann and associates confirmed that children of mothers with pre gestation and GDM have high incidence of obesity[43]. Pethitt and associates studied the children of Pima Indians form 5 – 19 yrs of age and found a significantly higher body weight as compared to control subjects.[44]

Diabetes and Foetal Growth

Maternal diabetes is characterised by increased plasma concentrations of glucose, free fatty acids, triglycerides and some amino acids. Pederson first proposed an explanation of the pathophysiology of the infant of diabetic mother, which has become known as the hyperglycemia hyperinsulinemia hypothesis.

Mother		Foetus	Neonate	Child
↓ Insulin Sensitivity	P L A C E N T A L	Macrosomia	Hypoglycemia	Obesity
↑ Plasma - Glucose -Aminoacid - Lipids		↑ insulin		IGT
		↑ Mixed Nutrients		Diabetes

Although embryonic cell proliferation is occurring at a very rapid rate and embryonic height is increasing rapidly during the period of embryogenesis, 95 % of eventual weight of human foetus is gained during the second half of the pregnancy. The disparity between the foetal and placental weight gain at 20 weeks of gestation can be viewed as suggestive evidence that factors other than the placental transport function are involved in controlling foetal growth.

Management

Dietary Therapy is the foundation for the treatment of GDM. ADA has recommended dietary therapy to start with 2000 – 2500 kcal /dl with 50 – 60 % carbohydrate (complex, high fibre) 10 – 20 % proteins and 25 – 30 % fats (< 10 % saturated). New ADA specify a protein level of 10 – 20 % calories but now allow greater flexibility in the levels of carbohydrate and fats.[45]

Jovanovic – Peterson associated found that former dietary recommendations lead to significant weight gain and postprandial hyperglycemia requiring insulin therapy in 50 % of patients.[46, 47] They suggested a diet with less calorie intake calculated by 30 kcal/kg for the normal sized individual's current pregnancy, 24 kcal/ kg for overweight patients and 12 kcal/ kg for morbidly obese women. Carbohydrate composition was < 45 % at breakfast, < 55 % at lunch and 50 % at dinner. Occasionally carbohydrate composition was decreased to 33, 45 and 40 % respectively for further improvement of glycemic control.

Restricting caloric intake by 50 % (1200 kcal/ dl) improves glycemic control but produces ketonemia and ketonuria.[48] Moderate (33 %) caloric restriction reduces macrosomia rates without adversely affecting neonatal outcomes or causing ketonemia.

Gunderson reviewed intensive nutritional therapy, emphasising a limit on total carbohydrate and distribution of carbohydrate throughout the day at several meals and snacks in order to achieve normal blood glucose levels and prevent starvation ketosis.

Insulin

ACOG criteria for initiating insulin therapy included testing plasma glucose level ≥ 105 mg/ dl and 2 hour plasma postprandial levels ≥ 120 mg/ dl. Lower rate of macrosomia when fasting is > 95 mg.[49] However, prophylactic insulin treatment is not advised in-patients where fasting and postprandial values remain within the recommended range.

Insulin Dosing

Type	A.M	P.M
NPH	4/9 of total insulin	1/6 of total insulin at bedtime
Regular	2/9 of total insulin	1/6 of total insulin at dinner
Total	2/3	1/3
Total daily insulin is calculated by		0.7 U/ kg – 6 – 8 weeks
		0.8 U/ kg – 18 – 26 weeks
		0.9 U/ kg – 26 – 36 weeks
		1.0 U/ kg – 36 – 40 weeks

Adopted from Jovanovic – Peterson
Review of GDM and low calorie diet, Diabetes Mel. Rev., 12: 287 – 308, 1996.

There are no specific studies declaring one type of insulin or certain regimen as superior affecting any perinatal outcome. Physician should expect to increase the insulin dosage as pregnancy program and insulin resistance increases. No published guidelines are available to help family physicians treat patients with GDM who require insulin. When necessary, collaborative care by obstetricians or perinatologists is advisable.

Lispro insulin is rapid onset, earlier peak and shorter duration of effect than regular insulin. This pattern of action is similar to healthy pancreatic insulin release in response to a meal.[50] Lispro is considered a pregnancy category B drug.[51] Recently a letter in New England Journal of Medicine described two cases of long anomalies in patients taking Lispro.[52]

Exercise has been suggested as an adjuvant therapy in GDM since glycemic control has been shown to improve with exercise regimens.

Oral Hypoglycemic Medications

Oral drug has not been recommended because of potential teratogenicity and transport of glucose across the placenta (causing prolonged neonatal hypoglycemia).[53] Although first generation hypoglycemic agents (chlorpropamide [Diatend] tolbutamide) shown to cross placenta, recent in vitro and vivo existence has determined the glyburide (micronase) does not enter foetal circulation.[54,55]

RCT $\rightarrow$ Glyburide therapy resulted in comparable maternal outcome (glycemic control, caesarian delivery) and neonatal outcome (macrosomia, hyperglycemia, ICU admission). ACOG[56] and the ADA[57] agree that glyburide should not be practiced for the treatment of GDM until additional RCTS support its safety and effectiveness.

Delivery Timing

Patients with uncomplicated, diet controlled GDM are allowed to progress to term unless other complications arise. Amniocentesis to document foetal pulmonary maturity if delivery before 39 weeks indicated.

Foetal surveillance should begin at 40 weeks gestation with twice weekly NST in pregnancy complicated by diet controlled GDM. Antenatal testing initiated earlier at 32

weeks gestation in GDM requiring insulin or pregnancy or in pregnancy complicated by hypertension or prior stillbirth. Foetal movement counting should be performed by all patients after 28 weeks gestation.

During labour and delivery, maternal plasma glucose levels are monitored at the bedside every 1 – 2 hours. Plasma glucose levels should be kept < 90 mg/ dl in order to decrease the risk or severity of neonatal hypoglycemia.[28]

Postpartum

GDM are at high risk for developing type 2 diabetes in future, they should be tested for diabetes six weeks after delivery via testing blood glucose measurement on two occasions or two hour oral 75 g glucose tolerance test.

Normal	< 140 mg/ dl
140 – 200 mg	IGT
> 200 mg	Diagnostic of Diabetes.

Screening for diabetes repeated annually especially for those who had elevated fasting blood sugar during pregnancy.[29]

Breast-feeding improves glycemic control and should be encouraged in women who had GDM.[58]

Contraception should be discussed, because women who have diabetes during one pregnancy are likely to have the same condition in a subsequent pregnancy. There are no limits on the use of hormonal contraception in patients with a history of GDM.

AIMS AND OBJECTIVES

Aims and Objectives

1. To know the incidence of DM complicating pregnancy in Women & Children Hospital, Institute of Obstetrics and Gynaecology, attached to Madras Medical College**,** Chennai.

2. To know the Perinatal Mortality and Morbidity in DM complicating pregnancy.

3. To know the maternal outcome in DM complicating pregnancy.

4. To compare the mortality with GDM and known Diabetes preceding pregnancy

MATERIALS & METHODS

Materials and Methods

Materials and Methods

This present prospective study has been conducted at the Institute of Obstetrics and Gynaecology, Women and Children Hospital, Egmore, attached to Madras Medical College, Chennai during August 2004 to July 2005.

All cases delivered in labour ward were taken in to consideration. Both Gestational Diabetes and Diabetes preceding pregnancy were included. Patients in all age groups, parity and presentation with confirmed increased sugar value were included along with associated maternal complication cases.

Methods:

All patients with h/o Diabetes complicating pregnancy were assessed with:-

I. Thorough history regarding
   1. Routine – for age and socio economic status
   2. Obstetric history – parity, previous Obstetric history
   3. Positive family h/o Diabetes
   4. H/o still birth, weight of previous babies
   5. H/o any congenital anomaly or unexplained death
   6. When was the disease detected – period of detection
   7. H/o diabetes in previous pregnancy and
   8. Type of treatment taken/ given

II. Clinical Examination regarding
   1. Nutritional status / Anemia/ Obesity
   2. Vital signs
   3. Abdominal Examination regarding GA, Hydramnios, Contraction, weight of babies, foetal presentation and foetal well being
   4. Vaginal examination wherever needed to assess the mode of delivery.

III. Laboratory Investigation
   Ultra Sonogram
   Hb, PCV
   Fasting blood sugar on the day of delivery
   Monitoring fasting and post prandial sugar level
   Glycemic profile

Post op/ Postnatal blood sugar

Routine blood sugar, bilirubin, calcium, electrolytes for all babies

X-ray chest, ECG & Echo for all babies

All patients were admitted for observation and delivery – term and nearing term. Mode of delivery either by vaginal or abdominal route was decided upon information such as history, size of baby, associated factors, glycemic control, foetal condition in uterus and maternal complication apart from the age and weight of the babies.

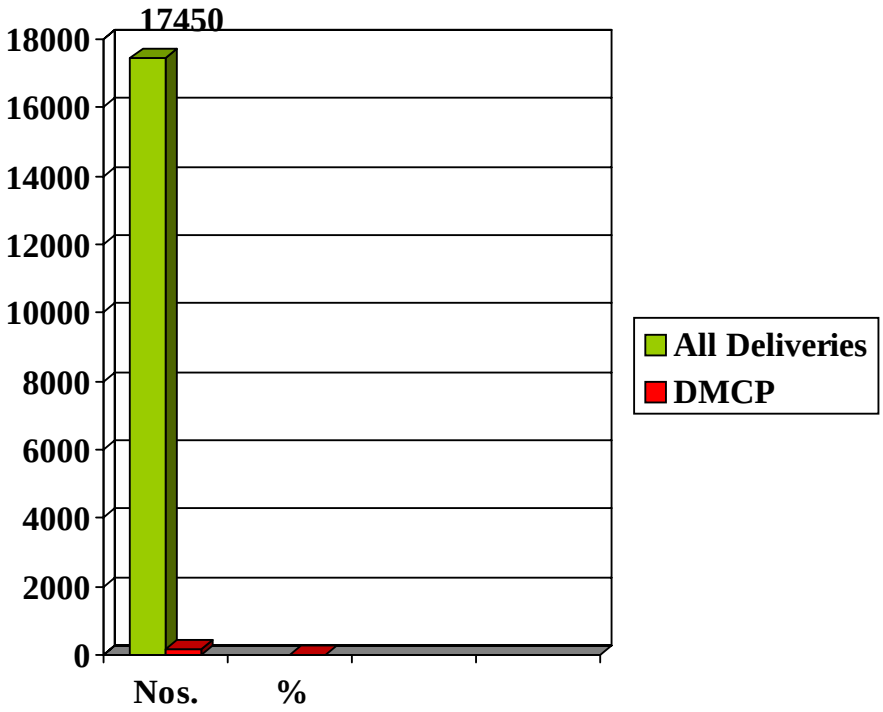
Patients selected for vaginal delivery were watched vigilantly in all the 3 stages of labour, carefully monitored to detect the foetal distress at the earliest. In cases where the abdominal route was decided some were elective / emergency depending on previous history, associated factors, infatibility status, bad obstetric history with weight of baby, glycemic control, fasting blood sugar were analysed.

Corticosteroids were administered for pre term and Diabetologists' opinions were sought and obtained regularly. The relationship between perinatal outcome and complication in labour was documented and babies were admitted in neonatal units for care and further investigation.

Foetal variables that influence the morbidity and mortality were asphyxia, malformations, RDS, Sepsis, Hypoglycemia, Hyperbilirubinaemia, Hypercalcimia, Macrosomia, Polycythemia and Cardiac Evaluation.

Postnatal blood sugar for mother was ascertained and insulin was stopped almost in 90% of cases and the cases were followed up in Diabetogy OP and Postnatal OP after 6 weeks.

Incidence


17450
18000
16000
14000
12000
10000
8000
6000
4000
2000
0
Nos.
%
All Deliveries
DMCP

Results and Observation

Incidence

Total no. of Deliveries:	17,450
No. of deliveries complicated by Diabetes:	153
Incidence in IOG**: 0.87 %**	

Deliveries complicated by Diabetes:

Gestational Diabetes:	120	78.4%
Diabetes Mellitus complicating	33	21.56%

Age & DM

	< 30 years Nos.	%	> 30 years Nos.	%	Statistical Significance
DM n = 33	14	44.6	19	55.4	**Chi Square Value** 5.89
GDM n = 120	81	67.5	39	32.5	**'P' Value** 0.0152278

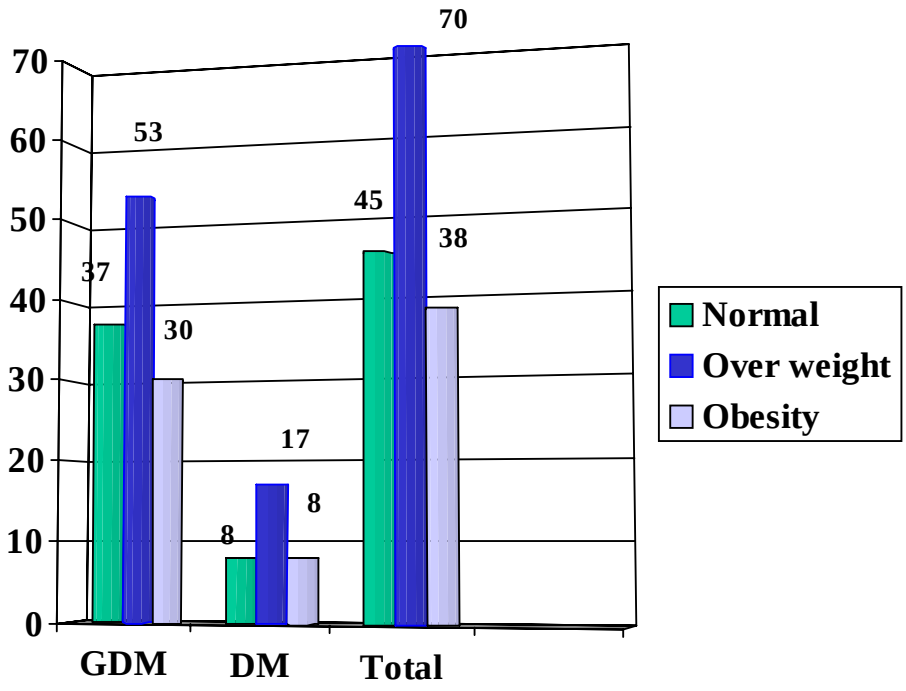
Incidence of known diabetics is higher in the higher age group of more than 30 yrs. i.e., 19 out of 33. In the case of GDMs, patients less than 30 years are more than double of patients who are more than 30 years as evidenced in the 'P' and Chi square values.

Parity in Diabetes

No. of deliveries	153 (DMCP)	17450 (All)	%
Primi	41	9630	0.43
G_2 & G_3	95	7743	1.23
> G_4	17	77	22.08

When this incidence of parity in DM complicating pregnancies is compared with the total deliveries, gravida 2 & 3 account for about 62 % in DMCP and is about 18 % more than the percentage in all deliveries. Again G4 and more in DMCP is 7 % more than that of all

Incidence with BMI



deliveries. In so far as the primis are concerned the DMCP incidence is less than half of all deliveries.

	Primi Nos.	%	> 1 para Nos.	%	Statistical Significance
No. of deliveries n = 17450	9630	55.18	7820	44.72	**Chi Square Value** 48.24
Diabetics n = 153	41	26.79	112	73.21	**'P' Value** 0.0000…

The chi square value and the corresponding P value validate the statistical significance.

Weight of the patient

n = 153

Weight	No.	%	GDM	DM
Less than 50 kg	15	9.8	10	5
51 – 69 kg	87	56.87	72	15
More than 70 kg	51	33.35	38	13

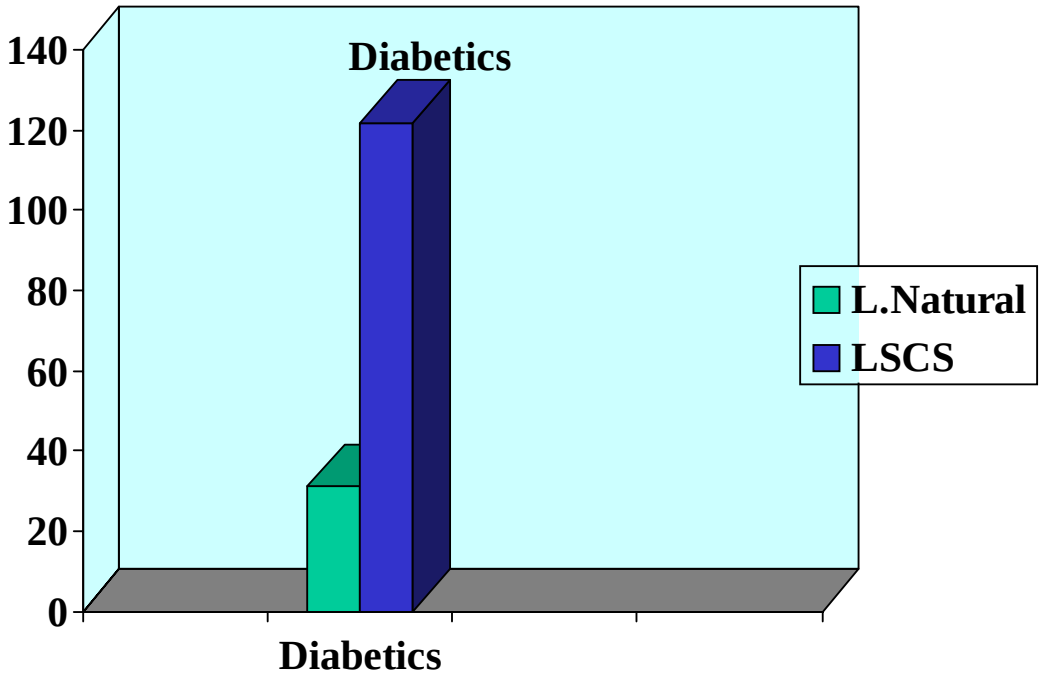
One third of the patients weighed more than 70 kg in weight and their babies weighed about 3.5 to 4 kg. Maximum weight of the mother was 121 kg.

Incidence with BMI of the patient

B M I		GDM Nos.	%	DM Nos.	%	Total Nos.	%
Normal	18.5 – 24.9	37	30.83	8	24.24	45	29.42
Over Weight	25.0 – 29.9	53	44.17	17	51.52	70	45.72
Obesity	> 30	30	25	8	24.24	38	24.86

Insulin resistance is the probable cause for over weight and obesity in DM. Body Mass Index based on the National Heart, Lung and Blood Institute in 1998 had indicated the values for normal and obese patients. The table above shows that nearly 70 % in GDM and 75 % in DM are over weight.

Mode of Delivery



Family History

Among 153 patients, 68 patients gave history of Diabetes in the family - either the mother or father or both. The incidence of diabetes in the family is : 44.4 %.

Total DMCP 153	GDM 120	Known DM 33
Family h/o +ve 68	44	24
Percentage 44.4	36.6	72.7

It is imperative to note that 72.7 % of patients with known GDM have a family history of DM.

Booked Diabetics

Total no. of deliveries	153
Booked at I O G	13
Booked at Private Hospitals	140

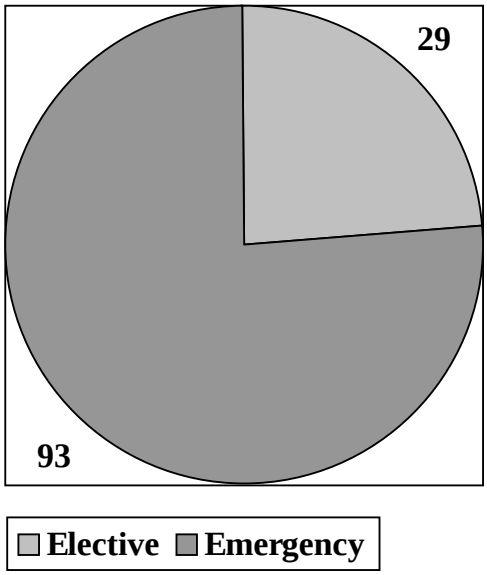
All cases that reported at I O G are booked at private hospitals and report here only when nearing term and the chance of having safe deliveries could not be ensured as we do not know how well they had followed the advises regarding Diabetes complicating pregnancy. Rise in blood sugar level in the III trimester often leads to perinatal mortality and morbidity.

Mode of Delivery

	No. of deliveries n = 17450		Diabetics n = 153		Statistical Significance
	Nos.	%	Nos.	%	**Chi Square Value**
L. Natural	9661	55.3	31	20.3	74.12
LSCS	7789	44.6	122	79.7	**'P' Value** 0.0000…

The incidence of LSCS in DM complicated pregnancies is drastically higher (35% more) when compared with routine cases. And, the 'P' value is zero making the study and the contents statistically significant.

Emergency vs Elective



Emergency Vs Elective Caesarian

	All deliveries N = 7789		DMCP N = 122		Statistical Significance
	Nos.	%	Nos.	%	
Elective	773	9.9	29	23.7	**Chi Square Value** 23.78
Emergency	7016	90.1	93	76.2	**'P' Value** 0.0000…

Incidence of elective LSCS in DM complicating pregnancy is more than double as in the normal cases. The 'P' Value confirms the statistical significance.

Gestational age - Term

Total no. of deliveries			153	
Patients delivered beyond 38 weeks			129	84.32 %
	GDM	Known DM	Total	%
Vaginal	13	6	19	14.72 %
LSCS	90	20	110	85.27 %

Whether the patient is GDM or Known DM, the incidence of LSCS is certainly high at 85 %. Among GDM patients it is seven times higher to vaginal and in DM patients it is more than three times that go for LSCS instead of vaginal births.

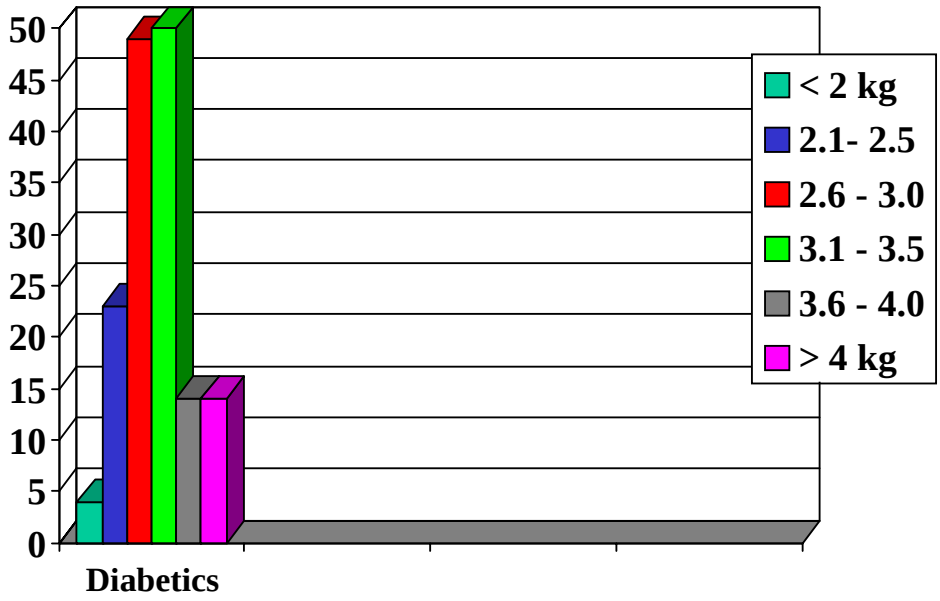
Gestational age - Pre term

Incidence of pre term deliveries was 15.68 % i.e., 24 out of 153 patients were delivered in less than 37 weeks and among them…

Vaginal		LSCS	
12	50 %	12	50 %
GDM	Pre GDM	GDM	Pre GDM
9	3	8	4

In pre term deliveries the mode of delivery was of equal distribution, be it vaginal or LSCS but in the known DM group the LSCS rate was marginally high – 4 out of 12 compared to 3 out of 12.

Baby Weight


50
45
40
35
30
25
20
15
10
5
0
Diabetics
< 2 kg
2.1- 2.5
2.6 - 3.0
3.1 - 3.5
3.6 - 4.0
> 4 kg

Incidence of operative delivery depending upon the type of AGT during pregnancy

	N = 122 Nos.	%	Statistical Significance
GDM N = 120	98	81.6	**Chi Square Value** 0.79
Known DM N = 33	24	72.7	**'P' Value** 0.3751045

LSCS rate in both GDM and known DM is equal and the 'P' value denotes the same.

Weight of Baby

Weight	Diabetics No.	%	Total Deliveries Nos.	%
< 2 kg	4	2.59	2012	11.53
2.1 – 2.5 kg	23	14.94	4547	26.05
2.6 – 3.0 kg	49	31.82	6144	35.20
3.1 – 3.5 kg	50	32.47	3923	22.48
3.6 – 4.0 kg	14	9.09	722	4.13
> 4.0 kg	14	9.09	102	0.58
Total	154		17450	

In our study, more than 18 % of delivered babies weighed more than 3.5 kgs. The maximum baby weight was 4.8 kg. In a random sampling of 200 babies the mean weight of the babies was 2.5 kg with a standard deviation of 0.5. 2 SD = 2.5 ± 1.0. The average weight fell between 1.5 kg $\rightarrow$ 3.5 kg. Babies weighing > 3.5 kg were considered as Macrosomia.

Among 4 babies of less than 2 kgs, 2 were pre term and 2 were IUGR with severe PIH.

Babies Presentation

Among 153 deliveries, Cephalic presentation were 143 and breech were 10. Only one twin was reported.

Type of treatment

Insulin Treatment		129	84.31 %		
Meal Plan		21	13.7 %		
Oral Drugs		1	0.6 %		
Insulin (129)		Meal Plan (21)		Oral (1)	
GDM	DM	GDM	DM	GDM	DM
99	32	21	-	-	1

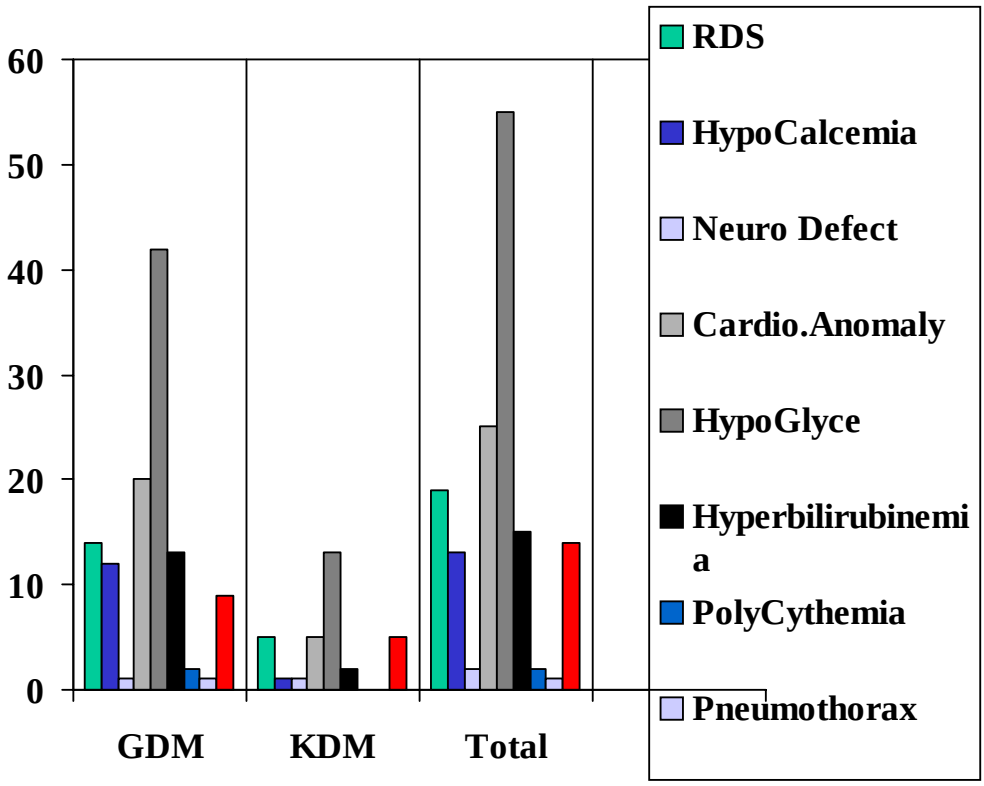
Though two GDM patients were detected they delivered before treatment could be started. One was still born and the other was normal. Insulin doses were adjusted each month depending on blood sugar value. No pre GDM patient was on meal plan and the lone oral drug patient was a known DM. Of all GDM patients 99 (83 %) were on insulin and another 21 (17 %) were on meal plan.

Dose of insulin

	< 20 U	21 – 50 U	>50 U
Known DM	14	9	7
GDM	70	24	5

The above table shows the dosage given to the patient in the last few days of delivery. Insulin doses were calculated based on blood sugar levels. Fasting >95 and >140 in glycemic profile were taken for adjustment. Minimum to start with is 4 U. If fasting level is high long acting insulin level was increased, if before dinner, and bedtime glucose level increased intermediate acting insulin level is adjusted The total dose given is 2/3 in morning and 1/3 in the evening.

Perinatal Morbidity



Perinatal Morbidity

Common perinatal morbidity seen in DM complicating pregnancies were as follows:

Morbidity	GDM	Known GDM	Total	%
Respiratory Distress	14	5	19	12.3
Hypo Calcemia	12	1	13	8.5
Congenital Malformation				
Neurology Defect	1	1	2	1.2
Cardiac Evaluation	20	5	25	16.27
Hypoglycemia	42	13	55	35.71
Hyperbilirubinimia	13	2	15	9.7
Polycythemia	2	-	2	1.2
Pneumothorax	1	-	1	0.6
Macrosomia	9	5	14	9.1

Hypoglycemia, Respiratory Distress and Hypocalcemia are the common earlier complications encountered in infants born to Diabetes Mellitus mothers.

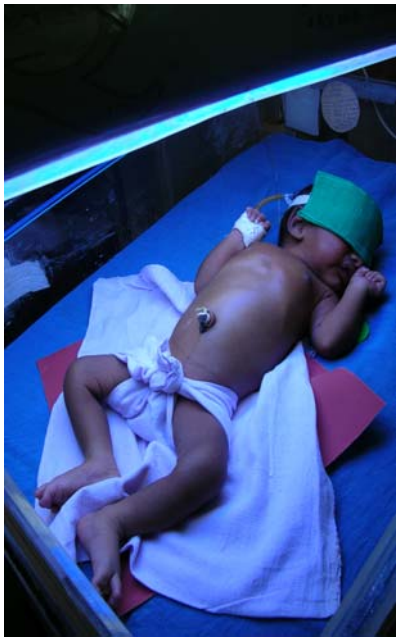
Among cardiac evaluation, x-ray findings revealed Cardiomegaly in 25 cases. Echo reported 21 small PDAs, L $\rightarrow$ R shunt with IAS, IVS intact / small ASD L $\rightarrow$ R shunt. 2 cases were reported with small closing muscular VSD without PHT and 1 PFO L $\rightarrow$ R shunt. All these cardiac cases need to be evaluated again after six months for anomalies, as by that time most of the cardiac problems would have settled.

Hyperbilirubinemia is encountered mostly in day 2. Among 15 cases 2 had severe icterus and had exchange transfusion.

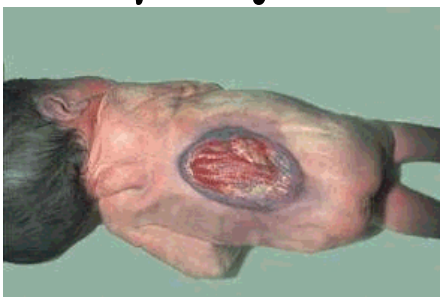
Congenital Malformation – Neurological defect was noticed in 2 cases of known DM, one with Spinabifida with Lumbosacral region small Meningomyelocele. There was also one case in the GDM group, which had Sacral Agenesis with Anorectal anomaly.

Not withstanding these, Macrosomia is yet another complication that Obstetricians have to contend with. 14 cases of macrosomia were reported. > 3.5 kg (Standard Mean Value) weighing babies account for 28 cases, which is above normal. In our study some babies had more than one complication.

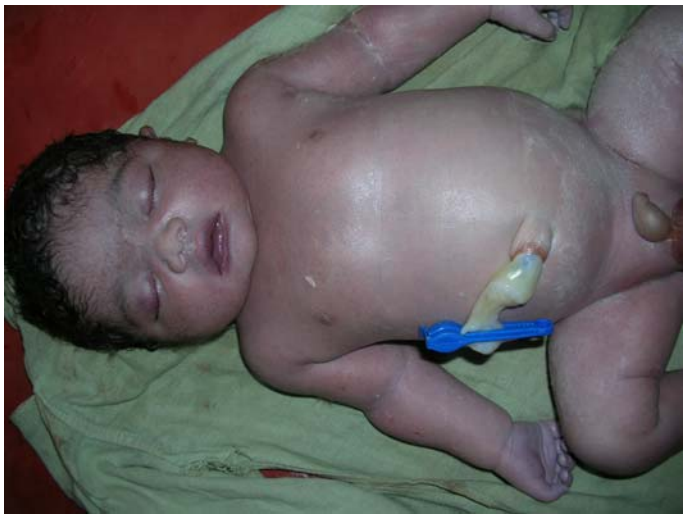
Phototherapy



Myelomeningocele



Stillbirth-Macrosomia



Perinatal Mortality

	DMCP	ALL Deliveries
Total no of babies:	154	17450
Mortality:	10	1184
Mortality Rate:	64.94 / 1000	67.95 / 1000

Mortality rate was more or less equal among DM complicating pregnancy and normal deliveries.

GDM / Pre GDM – Perinatal Mortality

Mortality	10	
Mortality among GDM babies	7 / 121	57.85 / 1000
Mortality among known DM babies	3 / 33	90.9 / 1000

Perinatal mortality is 91 % in known GDM cases when compared with GDM mothers (57.85 %).

Perinatal Mortality based on treatment

Of the 10 mortality babies		
8 were on insulin	1 was on meal plan and	1 was on oral drug.

Perinatal Mortality GA and mode of delivery

50 %	- 5 / 10 babies - pre term	
50 %	- 5 / 10 babies - term.	
Vaginal	2 / 4 Pre term	1 / 5 Term
LSCS	3 / 4 pre term	4 / 5 Term

Among pre term mortality LSCS mode of delivery was slightly more than vaginal, whereas in the mortality rate among term babies, LSCS rate was found to be very high.

Term IUD	2	(One macerated baby)
Pre term IUD	2	(One fresh death)
Term still birth	2	
Perinatal death term	1	
Perinatal death	1	
Congenital Anomaly	2 – One GDM & One Known DM	

Totally there were 4 IUDs, 2 stillbirths, 2 perinatal deaths and 2 of congenital anomaly.

Antenatal Complication

66 patients among 153 had antenatal complications. No morbidity was reported.

Total deliveries 153

Complications that are Pregnancy induced:

Hyper tension	27	17.6 %
Hypothyroid	8	5.2 %
Asthmatic	3	1.96 %
Epilepsy	1	0.65 %
Breech	10	6.5 %
Twins	1	0.65 %
Hydramnios	16	10.4 %

PIH is one of the common complications associated with GDM. PIH was so severe it resulted in IUGR in 2 cases. The PIH incidence is 17.6 %.

Postnatal Complication

Among 153 deliveries only 6 patients had postnatal complications.

Atonic PPH – Sub total Hysterectomy	1
Hyperglycemia – Ketoacidosis	4
Radiculopathy – Hyperglycemia	1

Ketoacidosis with postoperative blood sugar of > 240 mg with acetone ++. But patient subsided within 2 days on insulin therapy. Radiculopathy case could not be controlled and insulin was continued even after discharge (10 days). PPH is not associated with DM.

DISCUSSION

Discussion

The study done at Institute of Obstetrics and Gynaecology attached to MMC, Chennai from August 2004 to July 2005 showed the incidence of Diabetes Mellitus complicating Pregnancy as 0.87 %, which corresponds to normal study.

Study Incidence	Mangala et al[1], St.John's Bangalore	Hode et al
0.87%	0.7 %	0.15 – 12.3%

Prevalence of GDM

Author	Location	Prevalence
Abell, Beishar[2]	Australia	0.7
O. Sullivan[3]	Boston	2.5
Mage[4]	Seattle	3.2 – 5.0
Dooley[5]	Chicago	3.5 – 5.5
Sacks[6]	Los Angeles	3.4
Berkowitz[7]	Manhattan	4.6
Murphy[8]	Alaska	5.8
Natum[9]	Los Angeles	7.1
Benjamin[10]	Zuni, New Mexico	14.3
Sessiah V[11]	Trivandrum	15
,,	Erode	18.8
,,	Chennai	15

Type of Diabetes

Distribution is compared with other studies as given below.

Our study		Mangala et al		Sobhande et al[12]		Dume et al[13]	
Total 153		38		185		Caucas	Indonesia
GDM 120	7 8 %	26	68 %	139	75.4 %	40%	91(70%)
DM 33	22 %	12	32 %	66	24.8 %	60%	37(30%)

Age & DM

Maternal age elicited among 153, less than 20 years were 8 cases, 87 (56.8 %) in 20 –29 years and another 58 (37.9 %) belong to 30 – 40 years.

< 20 yrs	0.7 %	0.7 % *
(21 – 29 yrs	56.86 %	
30 – 40 yrs	37 %	3.8

In our study P value of >30 yrs old patients among the total 153 was significant for both GDM and Known DM – 39 / 81 and 19 / 14. (* Largest study based on screening - Coustan et al[14)]

Parity In Diabetes

Primi para's among 153 patients were only 26.7 %. Common parity was G2 – G3. More than G4 with one live/ no baby was about 11.11 % due to the disease complication. The general incidence of G4 is only 4.41 %.

Maternal Weight

Maternal obesity is an independent and more important risk factor for large infants with GDM and glucose intolerance (Leronadu et al, 1996) [19]. As per BMI except for 30.8 % rest were 69 % overweight among GDM and 76 % in Known DM.

BMI	Our study	Sunebaeg et al[15]
> 23.9	69 %	54.9 %
Normal	30.8 %	45.1 %

Family history of DM and Booked cases

68 out of 153 patients gave history of mother or father or both with DM and were on either oral or insulin. Among this group 73 % came under pre GDM, proof that pre GDM has a higher incidence for h/o of DM in the family. No single case was left unbooked and most of them were booked elsewhere, referred to IOG during III trimester / near term. Only 13 were booked at IOG. Increase in fasting blood sugar in III trimester leads to increased perinatal mortality and morbidity. 35.2 % (55) cases had increased fasting blood sugar nearing term, which resulted in perinatal mortality and mortality. Stillbirth could be due to increase in fasting blood sugar (both in GDM as well as pre GDM) – *Jobstone & Colleagues 1990* – and 2 stillbirths were noticed in this study.

Mode of Delivery

Among 153 patients labour natural was 31 (20.3 %) and LSCS was 122 (79.7 %). LSCS rate among foetal population was about 44.6 %.

Indication for greater incidence of LSCS were increased primary section, CPD, Macrosomia, elderly primi, BOH, Breech, GDM with PIH, Foetal distress and Cord presentation.

	Our Study	Mangala et al	Krishies et al[16]	Sobhande et al[12]	Hansen et al1[17]
LSCS	79.7 %	74 %	51 %	48 %	45.2 %
L.Natural	20.3 %	26 %	31 %	52 %	54.8 %

Among LSCS, incidence of emergency LSCS and elective were 76.21 % and 28.7%, which compared favourably with the total emergency LSCS and elective LSCS rate of 90 % and 90.9 %.

Gestational Age and Delivery

There were 24 pre term deliveries and about 129 (84 %) term deliveries. The incidence of LSCS is high both in pre term and term deliveries.

	Our Study	Mangala et al	Kristiane et al[16]	Hansen et al[17]
Pre term	15.68 %	11 %	46 %	20.6 %
Incidence	24/ 153	4/ 38	78/ 166	

Weight of baby

Babies of > 4kg were 14 and those more than 3.5 were 14, while it was 18 % in diabetes it was only 4.7 % of all total deliveries.

Macrosomic infants of diabetic mothers are described anthropometrically different from other large gestational age infants (McFarland & associates 2000, Modanlou[20] & colleagues 1982).

	Our study	Mangala et al	Tea cs et al[18]	Kitzmiller et al
Weight > 4 kg	9.09 %	36.8 % (no – 38)	3.8 % (no – 216)	11 %

Presentation

Cephalic presentation was noticed in 143 patients and 10 with breech. There was also a twin, with both babies hypoglycemic, treated with 10 % dextrose.

56 (36.36 %) normal babies were without any abnormalities as they were under good glycemic control. These babies were also kept in pre term for 48 hours, advised for warmth, breast feed, inj. Vit.K 1 amp IM and glucose level monitored at 4, 6, 24 and 48 hours.

Management

More than 85 % were on insulin and 21 were on meal plan and one continued oral drug. All known DM, except one was on insulin. Among GDM, 99 were on insulin and 21 on meal plan. Perinatal mortality and morbidity increased among patients on meal plan and patients who had insulin only for 4 days prior to delivery. The lone patient on oral drug also resulted in perinatal mortality. Fasting glucose > 110 mg will not respond to Glyburide therapy (Conway et al[21], 2004)

	Our study		Teacs et al[18]	
Insulin	131/ 153	85 %	210	40 %
Meal Plan	21/ 153			80 %

Perinatal Morbidity

Respiratory Distress Syndrome (RDS), Hypoglycemia, Hyperbilirubinemia, Hypocalcemia were the common problems faced in this study.

Insulin is generally administered in two to three times depending on blood sugar value. Combination of intermediate acting and regular insulin is given before breakfast and dinner (2:1 ratio). Patient was given 2/3 of their total dose with breakfast and remaining 1/3 in the evening as a combined dose with dinner or split in to components with regular insulin at dinnertime and with intermediate acting insulin at bedtime. (0.7 U/kg I trimester, 0.8 U/kg at II trimester and 1 U/kg at III trimester).

Hypoglycemia was reported in 55 cases and they all had increased blood sugar in the III trimester. Patients with glucose level of < 35 mg, within 1 hour, were given 10 % Dextrose 6 cc, iv bolous followed by 10 % Dextrose 180 ml, 200 ml for 0 – 24 hours, depend on

blood sugar level continued even for 3 days. Rapid decrease in plasma glucose level after delivery is characteristic of infants of diabetic mothers. It is attributed to hyperplasia of the foetal β islet cells induced by chronic maternal hyperglycemia. Taylor (2002) found neonatal hypoglycemia < 45 mg before second feeding was related to maternal blood sugar exceeding 145 mg / day during labour. Prompt recognition and treatment of hypoglycemic infant helped in minimising sequlae.

The incidence of Hyperbilirubinemia was 9 % compared to Mangala et al study that reported 19 %. One patient had exchange transfusion due to increased bilirubin level within 24 hrs. Phototherapy was given for 4 days. Other cases of hyperbilirubin were also treated with phototherapy.

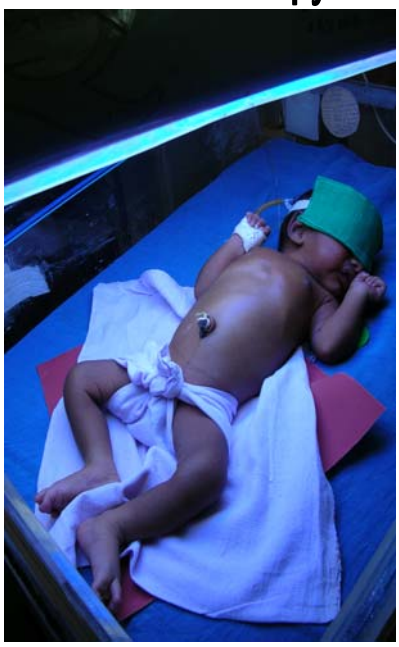
Patients' outcome associated with good diabetic control

Study	Our Values	Game et al	Kitzmiller et al	Levenot et al
Patients	153	260	134	120
Fasting B.S	95 – 120	109 – 140	105 – 121	145 – 153
PNM / 1000	64.94 %	46 %	37 %	42 %
Macrosomia	9 %	22 %	11 %	12 %
Hypoglycemia	35 %	39 %	49 %	28 %

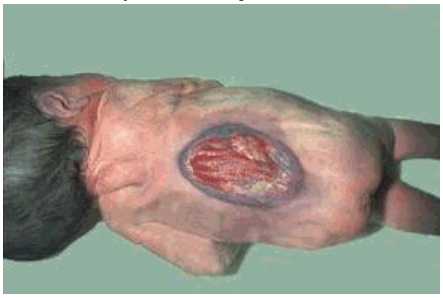
Patients who had jitteriness were examined carefully and their calcium levels were found to be < 7 mg and the treatment was 5 to 7.5 ml /kg, IV infusion of calcium gluconate for 24 hours and 2 ml / kg x 3 days. Oral calcium 60 ml / kg was administered till the calcium level rose to normal. Each 5ml contains 125 elemental calcium. Morbidity complications are of increased distribution among the known diabetic compared to GDM patients.

All babies of DMCP were admitted in pre term ward and evaluated for cardiology problem using x-ray chest, ECG and Echo. Cardiomegalies, PDA, ASD with L $\rightarrow$ R shunt were reported in 25 cases. But most of cardiology problems will settle down by six months (Way, 1979). As per echo, Cardiomegaly is reported in 25. Small PDA with L $\rightarrow$ R shunt and small ASD with L $\rightarrow$ R shunt in 21 cases. These problems may get resolved in six months, if persisting they may surface after this period as malformations (Way et al, J. Paed. 95: 1020, 1979). Pederson et al 853 IDM babies found 1.7 % cardiac malformations. Roward et al 470 DM 4 % cardiac malformation (50 % mainly VSD) Driscol & Pederson proved VSD as the single most anomaly.

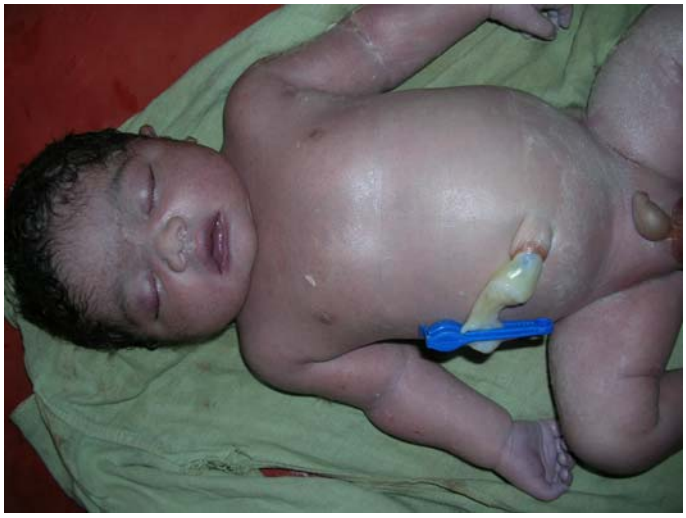
Phototherapy



Myelomeningocele



Stillbirth-Macrosomia



Perinatal Mortality

The perinatal mortality was 10 among 154 babies and the **PNM Rate** of DMCP was **64.94** /1000. The **Normal PNMR** was 1184 / 17450 – **67.85** /1000. Due to pre conception counselling and good glycemic control the PNM Rate is lower in DMCP.Mortality has been 90.9 % in Known DM and 57.8 % in GDM. This shows that glycemic control is well maintained in GDM compared to known DM.

Our Study	Sobhande et al [12]	Mizal et al
10/ 153	10/ 185	
6.4 %	5.7 %	10.5 %

Congenital Malformations among 154 babies were 2 with confirmed malformations (1.2 %) – one with Spinabifida, Myelomeningocele feature suggestive of Arnold Chiari syndrome and the other with Sacral agenesis and anorectal anomaly.

Adashi and Co-worker study 18 month reported 113 Diabetic pregnancy - 81 GDM (diet controlled), 6 (insulin) and 26 pre gestational insulin dependant diabetic insulin. Rate of congenital malformation: 5.3 % diet controlled GDM. NO malformation in offsprings of women with either GDM / known DM receiving insulin. According to White DM related malformation was 3 – 4 %. Caudal regression syndrome was pathognomonic of diabetic induced malformation.

One on oral drugs and one on meal plan resulted in mortality. Five were pre term and five were term. Incidence of mortality in pre term is comparatively higher than that of term babies.

Among two term IUDs, one was macerated whose mother was on meal plan with irregular check up. Patient reported at IOG with loss of foetal movement delivered labour naturally. The other term IUD was on human insulin only for the last 10 days. LSCS was resorted to as the fasting blood sugar was higher.

Among two pre term IUDs one was on oral drug, pre GDM and on insulin. Previous baby for this patient had myelomeningocele and died. Other pre term IUD was with previous LSCS glycemic control was not good.

Of the two stillbirths, one was GDM and other Pre GDM. Both babies were about 4 kg. Pregnancies in women with overt diabetes are at greater risk for foetal death. The danger is not apparent for those who have only postprandial hyperglycemia (Lucas & Co worker, 1993, Sheffield, 2002).

One GDM with congenital anomaly spina bifida, myelomeningocele at lumbar region was on insulin since fifth month. She was terminated with bougie application at seventh month, delivered normally and other with sacral agenesis with anorectal anomaly was on insulin before conception, delivered by LSCS.

O'Sullivan observed fourfold increase in PNMR in prognosing improperly managed GDM. Several other studies have found an increased rate of still births in untreated GDM. Today in pregnancies identified and treated appropriately, intra uterine foetal is not increased.

Antenatal

66 among 153 patients had associated complications – 43.1 %. Common complications were breech, Hydramnios, Hypertension, Hypo thyroid. Rare were asthmatic, epilepsy and twins.

	Our study	Mangala et al	Hansen et al[17]
PIH	17.6 %	31.5 %	20.6 %
	N = 153	38	491

Hypertension (17.6 %) induced or exacerbated by pregnancy is the major complication that most often forces delivery in diabetic women. Special risk factors for Pre eclampsia include any vascular complication and pre existing proteineuria with or without chronic hypertension. Sibbai & colleagues, 2000 reported 11 % class B, 22 % Class C, 21 in class D and 36 in class FR. Interestingly the incidence of Pre eclampsia does not seem to be related to glucose control (Garner & Co authors, 1990).

Ketoacidosis reported only in four cases (1.3 %). Common incidence of ketoacidosis is about 1 % of Diabetic pregnancies. It remains one of most serious complications (Garner, 1995). One patient went in atonic PPH and ended in sub total hysterectomy. This patient was on meal plan. Fasting blood sugar on the day of surgery was 110 mg.

More recent reports indicated that improved glycemic control is associated with significant reduction in various obstetric complications. DKA 0.7 %(Kilvert et a)l, Hydramnios 2 % and pre term labour 8.1 %.

One patient of known DM had weakness of lower limb (Radiculopathy). Postoperative blood sugar was increased and it was controlled with insulin drip was maintained for 10 days according to blood sugar level and advised to continue treatment consulting diabetologist.

SUMMARY

Summary

Incidence of diabetes:	0.87 %
Percentage of GDM: 78.4 %. Known DM:	21.56 %
Distribution among G2 & G3:	62.09 %
≥ G4:	11.11 %
Over weight and obesity distribution is: GDM:	69.1 %
Known DM:	75.7 %
Term delivery:	84.3 %
Pre term delivery:	15.68 %
Family History +ve GDM:	36.6 %
Known DM:	72.7 %
Mode of Delivery L.Natural:	20.3 %
LSCS:	79.7%
Among LSCS emergency LSCS:	76.2 %
Macrosomia :	9.09 %
Mode of Treatment- Insulin:	84.3 %
Meal Plan:	13.7 %
Oral drug:	0.6 %
Morbidities in DM babies were	
Respiratory Distress Syndrome:	12 %,
Hypoglycemia	35.9 %
Hypocalcemia	18 %
Hyperbilirubinemia	9.7 %
Congenital Malformation – Neurological Defect:	1.2 %
Cardiac Evaluation:	16.27 %
Perinatal Mortality:	10 / 154
GDM :	57.85/ 1000,
KDM :	90.9/ 1000
PNMR – Diabetes Mellitus complicating Pregnancy:	64.94 / 1000
Normal:	67.95 / 1000

Among 10 babies, 2 were term IUD, 2 pre term IUD, 2 still births,

2 Perinatal deaths (term & pre term) and 2 congenital malformation deaths.

Maternal associated complications were: PIH 17.6 %

Hypothyroid: 5.2 %

Hydramnios: 10.4 %)

Associated postnatal complication was only in 6 cases.

4 with Ketoacidosis corrected,

1 with Radiculopathy - continued treatment,

1 ended in PPH $\rightarrow$Subtotal.

CONCLUSION

Conclusion

Despite the changing trends in obstetrics, it is mandatory to have an uptodate knowledge, diagnosis, appropriate decision and management in labour. A careful intervention will avoid long-term complications; help reduce perinatal mortality and late sequalae.

The Perinatal Mortality Rate of Diabetes Mellitus Complicating Pregnancy is more or less equal to normal babies. Perinatal outcome, which is little higher in DM preceding pregnancy, can be overcome by pre conceptional counselling and good glycemic control.

Pre conceptional glycemic control and control around the critical period of organogenesis (7^{th} – 8^{th} week) though done at present needs to be carried out vigorously and effectively so as to help in lowering the incidence of congenital malformation.

The incidence of caesarian section can be reduced in Diabetes Mellitus complicating pregnancy to an extent by carefully monitoring the glycemic control, which in turn will lower the macrosomia rate, and by minimizing primary section.

Thus with the use of frequent home glucose monitoring, diet, insulin and team approach it is possible to achieve euglycemia in most diabetic pregnancies and prevent much of the perinatal mortality and morbidity.

APPENDIX

Proforma

Name: Age: Ht: Wt:

Socio Economic Status: IP No.:

Address

Rural / Urban Income Level:

Reference: Booked: Yes / No

Gravida: Para: Abortions:

Antenatal Check up: Regular / Irregular

Malpresentation:

I. Obstetrical History

Previous GDM: Yes / No When Diagnosed: I / II/ III Trimester

Mode of Treatment:

History of other complication:

Mode of delivery: Vaginal / LSCS Indication:

Baby: Term/ Pre term/ IUGR / Big Baby Weight of baby:

Complication of baby: When stopped treatment:

II. Known DM

Age at diagnosis of Diabetes Mellitus

Type of DM: Type I / II/ Other Mode of Treatment:

Complication of DM before conception:

Complication of DM after conception:

Associated Complication:

III. GDM Time of diagnosis: I Trimester / II / III

Complication associated: PIH / Hypothyroid/ Asthma/ Hydramnios

Mode of Treatment: Insulin/ Meal Plan/ Oral

Type & dose of insulin:

Mother: Duration of stay in hospital

Mode of delivery: Indication of induction

Baby: Weight: Length: APGAR HC:

Anomalies: AC:

Morbidities: Hypocalcemia/ Hyperbilirubinemia/ Hypoglycemia

Respiratory Disorder/ Cardiac Evaluation

Placenta:

Bibliography

1. Mangala R, Mhaskar A, Mhaskar R, Lillian T, Narayanan R, Perinatal outcome of Pregnancy complicated by Biabetes Mellitus, Int. Journal Diab, in Developing Countries 1991 (Vol II) page 22-27
2. Abell DA, Beischer NA, Evaluation of three-hour oral glucose tolerance test in detection of sinificant hyperglycemia & hypoglycemia in pregnancy, Diabetes 24; 874-80, 1973
3. O'Sullivan JB, Maban CM, Charles D, Dardrow R; Screening criteria for high risk gestation Diabetic patients, Am.J Obstt.Gynecol 116; 895-900,1973
4. Magee MS, Walden CE, Benedeth JJ, Knopp RH, Influence of diagnostic criteria on the incidence of gestational diabetes and perinatal morbidity, JAMA 269; 609-15,1993
5. Dooley SL,Metzer BE, Cho NH; Gestational Diabetes Mellitus: influence of race on disease prevalence and perinatal outcomes in US population. Diabetes care 40 (Sept 2) 25-29, 1991
6. Sacks DA, Abu-Fadil S, Karten GJ, Forsythe AB,Hacket JR, Screening for Diabetes, Obstt.Gynecol 70; 89-93
7. Bercowitz G, Roman SH, Lapinski RH, Alvarezm; Maternal characteristics on neonatal outcome and diagnosis of gestational diabetes Am.J Obstt & Gynecol 167; 976-82, 1992
8. Murphy NJ, Balkow LR, Scharer CD, Lanier AP,: Prevalence of DM in pregnancy among yup'ik Eskimos 1987-88, Diabetic care 16 (Suppl 1) 315-17, 1993
9. Natum GG, Huttaker BJ; Correlation between first and early thired trimester glucose screening test results, Obstt. Gynecol 76; 709-13,1990
10. Benjamin E, Winters D, Mayfield J, Goldes D,: Diabetes in pregnancy in Zuni Indian women, Diabetic care 16; 1231 –35, 1993
11. Sessaih V, Balaji V, Madhuri Balaji, Sanjeevi CB: Gestational Diabetes Mellitus in India, JAPI Vol 52; Sept 2004
12. Sobhande AA, KSKardu M, Archibong El; Complication of pregnancy & foetal outcome in diabetic patients managed in tertiary hospital, Saudi Arabia; West African J of Med, 2005 Jan-March (II) (13 T related article)
13. Dume et al: Foetal & maternal outcome in Indonesian compared to Caucasian women, Dept of Diabetic Medicine University of of hospital trust, UK
14. Coustal DR, Nelson C, Carpenter: maternal age and screening for GDM – A population based study; Obstt . Gynecol 73: 557, 1989
15. Sunebag A, Berne C, Lindmark G, Ewald U; Upsala University children hospital, Sweden: GDM – Perinatal outcome with policy of internal and intensive insulin therapy; Ups J. Medi.Science, 1991: 96 (3) 185-98 –related article
16. Kristensen J, Vesigard M, Wisborg K, Kesmodil U, Seeher NJ, Pregnancy weight & the risk of still birth & neonatal death; Perinatal epidemiology Research Unit, Denmark, BJOG 2005 April 112 (4) 403-8 Related article
17. Hansen, Person, Neonatal Morbidity in GDM, Dept of Women and Child, St.Goran's Hospital, Sweden, 24. Diabetic Care 1998, Aug 21 Suupl:2; 879-84
18. Tae CS, Wang HW, Thock, Heng TL, Rajasoorya C, Dept. Maternal Foetal Medicine, Kandang Kerbaum Hospital, Singapore; Ann Acad Med- Singapore 1990 July 19 (4) 459-62
19. Leonardi MR Botton SP, Increased incidence of large for gestational age infants and attributable to Gestational Diabetes; Am.J. Obstt . Gynecol 174; 393, 1996

20. Ms Farland MB, Langer O, Fazione et al, Anthropometric and body comparison difference in large for gestational age but not appropriate gest. age with without diabetes ; J.Obstt& Gyne Vol 7; 231, 2000
21. Conway DL, Gonzals O, Skiver D, Use of glyburide for the treatment of GDM. The San Antonio experience, J. Maternal Foetal Neonatal Medicine 15; 51 2004,\
22. Way GL, natural History of Hypertrophic cardiomyopathy in infants of DM; J Paediactric, 95: 1020, 1979
23. Taylor R, Kee C, Kyne Gizebalski D, et al Clinical outcome of pregnancy in women wih type I diabetes; Obstt. Gynecology 99; 537, 2002
24. Lucas MJ, Lowe TN, Bore L, et al, Class A Gestational Diabetes-Meaningful Diagnosis ? Obstt > Gyn 82: 260, 1993
25. Sibbai BM, Carithi S, Harth, Foetal Risk of pre eclampsia & adverse neonatal outcome among women with Pre gestational diabetes mellitus, Am.J. Obstt . Gynecol 189: 864, 2000
26. Garner RR, D Alton Dudly DK et al, Pre eclampsia in diabetic pregnancy, Am.J.Obstt. Gynecol 163: 505,1990
27. Garner P, Type II DM & Pregnancy, Lancet 346: 157,1995
28. Kuhl, Anderson GE, Hertel J, Molsted-Pederson L, Metabolic events in infants and diabetic mother during first 24 hours after birth, Acta J. Paediactrics of Scandinavia, 71: 19-25,1982
29. Kim C, Newton KM, Knopp RH,, GDM & incidence of type 2 diabeties systemic review, Diabetics Care 2002; 25, 1862-8
30. Jovanovic –Peterson L, Peterson CM, Review of GDM & low calorie diet & physical exercise as therapy. Diabetes Metab. Review 12; 287-308, 1996.
31. American College of Obstetricians & Gynecologist, Diabetes & Pregnancy, ACOG Technical Bulletin # 200. Washington DC., ACOG, 1994.
32. Coustan DR: GDM in diabetes in America, 2nd Edition, Harris ML,Cowie CC, Stern MP. National institute of health, National Institute of Diabetes, Digestive & Kidney Disease 1995, P 703 –17
33. Magee MS, Walden CE, Benedeth TJ, Knopp RK; Influence of diagnostic criteria on the incidence of gestational diabetes and perinatal morbidity, JAMA 269; 609 – 15, 1993
34. Sermer M, Naylor D, Farne D,Bringer A, Chen E, for Toronto TriHospital Gestational Diabetes investigator; impact of increasing carbohydrate intolerance on maternal –foetal outcomes in 3637 women without gestational diabetes. Am J Obstet Gynecol 173: 146 – 50, 1995
35. Deveciana M, Major CA, Morgan MA, Evan AT, Postprandial versus preprandial blood glucose monitoring in women with gestational diabetes mellitus requiring insulin therapy, N.England J. Med 333: 1237 –41,1995
36. O'Sullivan JB, Mahan CM; Criteria for the oral glucose tolerance test in pregnancy diabetes 13; 278 –85, 1964
37. Mestman JH, Anderson GV, Guadalupe V, Follow up study of 369 subjects with abnormal carbohydrate metabolism during pregnancy; Obstt Gynecol 39; 421-25,1972
38. Peters RK, Kjol SL, Xiang A, Long term diabetogenic effect of single pregnancy in women with previous GDM, Lancet 347; 227-30,1996
39. O'Sullivan JB, Charles D, Mahan CM, Dandrow RV, Gestational Diabetes and perinatal mortality rate, Am.J.Obstt. Gynecol 166; 901-904, 1973

40. Pettitt DJ, Knowler WC, Baird HR, Bennett PH, Gestational diabetes infant and maternal complications of pregnancy in relation to third trimester glucose tolerance in Pima indians, Diabetic care 3; 458-64,1980
41. Silverman BL, Metzger BE, Cho NH, Loeb CA, Impaired glucose tolerance in adolescent offspring of diabetic mothers, Diabetic care 18: 611-17,1995
42. Silverman BL, Rizzo T, Green OC, Cho NH, Ogata ES, Long term prospective evaluation of offspring of diabetic mothers, Diabetic Care 40: 121-25, 1991
43. Plagemann A, Harder T, Kohlhoff R, Rohde W, Dorner G; Overweight and obesity in infants of mothers with long term insulin dependent diabetics or gestational diabetes Int.J.Obesity 21: 451-56,1997
44. Pettitt DJ, Knowler WC, Bemet PH, Aleck KA, Baird HR; Obesity in offspring of diabetic Pima Indian women despite normal birth weight, Diabetic care 10: 76-80, 1987
45. Coustan DR, Carpenter MW, O'Sullivan PS, Carr SR, GDM predictors of subsequent disordered glucose metabolism Am.J.Obstt. Gynecol 168; 1139-45, 1993
46. Mulford MI, Jovanovic – Peterson CM, Alternative therapies for the management of GDM, Clinical Perinatology 20: 619-34,1993
47. Jovanovic-Peterson L, Peterson CM, Dietary manipulation as a prinmary treatment strategy of pregnancies complicated by Diabetes, J.Am. Coll of Nutrition 9; 320-25,1990
48. Magee MS, Knopp RH, Bebedett TJ, Metabolic effect of 1200 Kcal diet in obese pregnant women with GDM, Diabetes 39: 234-40, 1990
49. Langer O, Management of GDM,Clin Perinatol, 200, 1-15, 1993
50. Holleman F, Hoekstra J, Insulin Lispro N.Eng J.Med, 337; 176-83, 1997
51. Humalog Package insert, Indianapolis, Indian Eli-Lilly and Co
52. Diamond T, Kormas N;Possible adverse foetal effect on insulin Lispro, N.Eng J.Mrd 337; 1009,1997
53. Zielger, Graffon TF, Hansen DK; The effect of Tolbutamide on rat embryonic development in vitro, Tetrology 1993; 48, 45-51
54. Elliot BP. Schenker S, Langer O, Johnson R, Comparitive placental transport of oral hypoglycemic agents in humans- a model of human placental drug transfer; Am.J.Obstt.Gynecol 1994; 171, 653-60
55. Langer O, Conway DL, Berkes MD, Comparison of glyburide and insulin in women with GDM, N.Eng.J.Med 2000, 343: 1134-38
56. ACOG Practice Bulletin, GDM, Num 30, Dec-1994, Obstt & Gynecol 2001, 98: 523-38
57. Walkinshaw SA, Dietary regulations for Gestational diabetes, Cochrane Database Syst Rev 2003 (2) CD 000070
58. Kjol SL, Henry O, Lee RM, Buchanan TA, Effect of lactation on glucose and liquid metabolism in women with recent GDM; Obstt & Gynecol 1993; 82, 451-5

Abbreviations used

RDS	Respiratory Distress Syndrome
GDM	Gestational Diabetes Mellitus
IDDM	Insulin Dependent Diabetes Mellitus
NDDM	Non insulin Dependent Diabetes Mellitus
GCI	Glucose Corbohydrate Intolerance
DM	Daibetes Mellitus
KDM	Known Diabetes Mellitus
GTT	Glucose Tolerance Test
IGT	Impaired Glucose Tolerance Test
ADA	American Diabetics Association
ACOG	American College of Gynecologists and Obstetricians
ICU	Intensive Care Unit
NST	Non Stress Test
PPH	Post Partum Haemorrhage
ECG	Electro Cardio Graph
PDA	Patent Ductus Arteriosis
ASD	Atrial Septal Defect
VSD	Ventricular Septal Defect
HC	Head Circumferance
AC	Abdominal Circumference
IUD	Intra Uterine Death
DKA	Diabetic Keto Acidosis
USG	Ultra Sono Gram
PIH	Pregnancy Induced Hypertension
T	Term
PT	Pre Term
Hyd	Hydramnios
Hth	Hypo Thyroid
HT	Hyper Tension
HyB	Hyper Bilirubinemia
Br	Breech
C	Hyper Calcemia
G	Hyper Glycemia