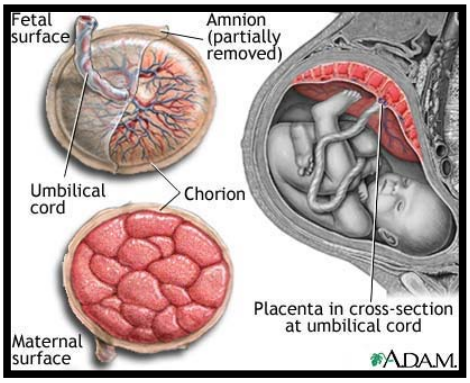
Gestational Diabetes Mellitus

(GDM)

Training Materials for Physicians

September, 2011 to February, 2012

(Universal blue circle symbol for diabetes)



HEALTH & DISEASE RESEARCH CENTER FOR RURAL PEOPLES, DHAKA, BANGLADESH

Supported by: World Diabetes Foundation, Denmark

ACKNOWLEDGEMENT

On behalf of the general people of Bangladesh, we are very grateful to World Diabetes Foundation for supporting our Project on Gestational Diabetes Mellitus. It is a first and only project (till now) in Bangladesh, which is addressing a critical health issue like Gestational Diabetes in Bangladesh.

In Bangladesh, most of the pregnant mothers are poor, non-educated and not aware about the Gestational Diabetes. We think this project will help to build up a strong relationship with those mothers to prevent the GDM. We also believe that, the activities of the project will draw the attention of national planner and think-tank to initiate their steps for Gestational Diabetes.

Actually, there is no specific data about the GDM in Bangladesh. We hope this project will discover a hidden painful data from the community of Bangladesh about GDM. That will help us to initiate a planning and develop a treatment strategy for GDM. We hope this kind of support from WDF will be continued in future for the wellbeing of Bangladeshi people.

PROGRAM SCHEDULE FOR PHYSICIAN ON GDM

Health and Disease Research Center for Rural Peoples (HDRCP) 14/15, 1st Floor, Probal Housing Ltd. Shekertak (Adjacent to Shekertak, Road-1), Mohammadpur Dhaka 1207, Bangladesh

Title: Training on Gestational Diabetes Mellitus for Physicians

Date	Time	Topics/subjects	Methods	Training materials	Facilitator	Remarks
First Day	9.00-9.40	**Induction and Eyes Breaking** - Participant's registration - Induction with participants - Welcome Speech.	- Lecture Method - Question and Answers - Eyes Breaking	- Used prescribed Format - Laptop Computer - Multi-media - Power point presentation - Banner	- HDRCRP's Training Unit - HDRCRP's Clinical Expert - Upazilla Parishad Chairman	
	9.40-10.45	**Brief induction on HDRCRP & WDF** - Activities - Aim and Objectives - How, why & where we work - About GDM project	- Lecture Method - PowerPoint presentation - Question and Answers - Brainstorming - Participatory facilitation	- Laptop Computer - Multi-media - Power point presentation - Poster	- Research Director of HDRCRP	
		Refreshment Break				
	11.00-12.30	**Basics of Gestational Diabetes** - Introduction - Definition - Epidemiology - The Endocrinology of pregnancy - Maternal pancreas - Maternal pancreas - Fetal endocrinology - Pathophysiology of GDM - Causes - Risk factors - Symptoms	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Participatory facilitation - Variety of games	- Laptop Computer - Multi-media - Flip chart - Posters - Leaflets - Banners	- Dr Md Abdullah Al Mamun MBBS, MD (Endocrinology)	
	12.30-2.00	**Management of GDM** - Target Group of GDM - Management during antenatal period - Management during labor and delivery - GDM Management team - Food habit - Diagnosis and screening - Treatment of GDM - Life style of GDM	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Participatory facilitation - Variety of games	- Laptop Computer - Multi-media - Flip chart - Poster paper - White Board - White board marker - Artliner - Leaflets - Banners	- Dr Samir Kumar Talukder Assistant Professor,Dept. of Endocrinology, Rangpur Medical College	
		Lunch and prayer Break				
	3.00-4.30	**Case Presentation (Practical Sessions)** - A pregnant lady with GDM - She shares her experience during pregnancy with GDM. - About her life style, Diet, treatment etc - About her insulin treatment	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Variety of games	- Laptop Computer - Multi-media - Flip chart - Poster paper - White Board - White board marker - Art liner	- DR Md Abdullah Al Mamun - Dr Samir Kumar Talukder	
	4.30-5.00	**Prescription writing for management of various part of GDM**	Participatory facilitation	- Oral discussion	- Dr Md Abdullah Al Mamun MBBS, MD (Endocrinology)	

Date	Time	Topics/subjects	Methods	Training materials	Facilitator	Remarks
Second Day	9.00-9.40	- Recapitulation of yesterday's sessions & discussions. - A brief description about today's presentations.	- Lecture Method - Question and Answers - Eyes Breaking	- Used prescribed Format - Laptop Computer - Multi-media - Power point presentation - Banner	- HDRCRP's Clinical Expert	
	9.40-10.45	- Management After delivery - GDM management team - Management of GDM - Follow up after pregnancy	- Lecture Method - PowerPoint presentation - Question and Answers - Brainstorming - Participatory facilitation	- Laptop Computer - Multi-media - Power point presentation - Poster	- Dr Md Abdullah Al Mamun MBBS, MD (Endocrinology)	
		Refreshment Break				
	11.00-12.30	- Glucose monitoring & insulin management - Complication in mother - Fetal development & growth - Obstetric management	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Participatory facilitation - Variety of games	- Laptop Computer - Multi-media - Flip chart - Posters - Leaflets - Banners	- Dr Samir Kumar Talukder Assistant Professor,Dept. of Endocrinology, Rangpur Medical College	
	12.30-2.00	- Insulin management during labor - Neonatal morbidity - Pregnancy with previously diagnosed diabetes - Prevention	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Participatory facilitation - Variety of games	- Laptop Computer - Multi-media - Flip chart - Poster paper - White Board - White board marker - Art liner - Leaflets - Banners	- Dr Md Abdullah Al Mamun MBBS, MD (Endocrinology) -	
		Lunch and prayer Break				
	3.00-4.30	**Case Presentation (Practical Sessions)** - A mother (who had GDM during her pregnancy) with her baby. - Management of baby after Delivery - Management of delivery - Follow up of mother after delivery.	- Lecture Method - PowerPoint presentation - Brainstorming - Group works - Question and Answers - Variety of games	- Laptop Computer - Multi-media - Flip chart - Poster paper - White Board - White board marker - Art liner - Leaflets	- DR Md Abdullah Al Mamun - Dr Samir Kumar Talukder	
	4.30-5.00	**Closing session**	Participatory facilitation	- Speech and feed back from the participants and facilitators	All participants	

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OBJECTIVE OF THE TRAINING

Gestational diabetes mellitus affects 2-8% of pregnancies, depending on the population studied. Recent data show that gestational diabetes mellitus (GDM) prevalence has increased by ~10-60% in several race/ethnicity groups during the part 20 years. A true increase in the prevalence of GDM , aside from its adverse consequences for infants in the newborn period, might also reflect or contribute to the current patterns of increasing diabetes and obesity, especially in the offspring. Therefore, the public health aspect of increasing GDM needs more attention.

Health and Disease Research Center for Rural Peoples (HDRCRP) in association with World Diabetes Foundation (WDF) plays an unique role in GDM advocacy and healthcare delivery in Bangladesh.

We hope, this training material will be very much helpful to the doctors in understanding GDM.

Gestational Diabetes

(Diabetes diagnosed during pregnancy)

INTRODUCTION:

Impaired glucose tolerance develops in 2–8% of pregnant women, usually during the second half of gestation. The frequency depends on ethnic group (highest in Asian-American, Latina, Native American, and Polynesian populations) and is increased in those with central obesity or a family history of diabetes. The mechanism of glucose intolerance in lean women results from sluggish first-phase insulin release coupled with excessive insulin resistance. In overweight women with gestational diabetes, insulin resistance increases more than in overweight controls, despite increased circulating insulin levels, so that insulin secretion is actually inadequate in relation to the hyperglycemia.

DEFINATION:

Gestational diabetes is a condition that occurs during pregnancy. Like other forms of diabetes, gestational diabetes involves a defect in the way the body processes and uses sugars (glucose) in the diet. Gestational diabetes, however, has a number of characteristics that are different from other forms of diabetes.

EPIDEMIOLOGY

GDM is the most common metabolic complication associated with pregnancy. GDM occurs in up to 14% of all pregnancies, resulting in approximately 200,000 cases annually in the United States. As the occurrence of T2DM has increased over the past few decades, an increase in the incidence of GDM has also been observed. Between 1994 and 2002, the incidence of GDM doubled. The rise in GDM can likely be attributed to improved screening and diagnostic tools, as well as to the climbing rate of obesity in the U.S. Excessive caloric intake and sedentary lifestyles are the major causative factors contributing to obesity.

The report, Gestational diabetes mellitus in Australia, 2005-06, showed that in 2005-06, 4.6% of women aged 15-49 years who gave birth in hospital were diagnosed with GDM. 'This amounts to more than 12,400 women and their babies affected,' said Mardi Templeton of the Institute's Cardiovascular, Diabetes and Kidney Unit. The report found the incidence of gestational diabetes among all Australian women in the 15-49 year age bracket increased by over 20% between 2000-01 and 2005-06.

THE ENDOCRINOLOGY OF PREGNANCY

Introduction

Throughout pregnancy, the fetal-placental unit secretes protein and steroid hormones into the mother's bloodstream, and these alter the function of every endocrine gland in her body. Both clinically and in the laboratory, pregnancy can mimic hyperthyroidism, Cushing's disease, pituitary adenoma, diabetes mellitus, and polycystic ovary syndrome.

The endocrine changes associated with pregnancy are adaptive, allowing the mother to nurture the developing fetus. Although maternal reserves are usually adequate, occasionally, as in the case of gestational diabetes or hypertensive disease of pregnancy, a woman may develop overt signs of disease as a direct result of pregnancy.

Aside from creating a satisfactory maternal environment for fetal development, the placenta serves as a repository endocrine gland as well as a respiratory, alimentary, and excretory organ. Measurements of fetal-placental products in the maternal serum provide one means of assessing fetal well-being. This chapter will consider the changes in maternal endocrine function in pregnancy and during parturition as well as fetal endocrine development. The chapter concludes with a discussion of some endocrine disorders complicating pregnancy.

Fetal-Placental-Decidual Unit

The function of the placenta is to establish effective communication between the mother and the developing fetus while maintaining the immune and genetic integrity of both individuals. Initially, the placenta functions autonomously. By the end of the first trimester, however, the fetal endocrine system is sufficiently developed to replace placental function and to provide some hormone precursors to the placenta. From this time, it is useful to consider the conceptus as the fetal-placental unit.

The fetal-placental unit will be considered in three separate but related categories: (1) as a source of secretion of protein and steroid hormones into the maternal circulation; (2) as a participant in the control of fetal endocrine function, growth, and parturition; and (3) as a selective barrier governing the interaction between the fetal and maternal systems.

Ovarian Hormones of Pregnancy

The hormones produced by the corpus luteum include progesterone, 17-hydroxyprogesterone, and estradiol. The indispensability of the corpus luteum in early pregnancy has been demonstrated by ablation studies, in which luteectomy or oophorectomy before 42 days of gestation results in precipitous decreases in levels of serum progesterone and estradiol, followed by abortion. Exogenous progesterone will prevent abortion, proving that progesterone alone is required for maintenance of early pregnancy. After about the seventh gestational week, the corpus luteum can be removed without subsequent abortion owing to compensatory progesterone production by the placenta.

Because the placenta does not produce appreciable amounts of 17-hydroxyprogesterone, this steroid provides a marker of corpus luteum function.

Another marker of corpus luteum function is the polypeptide hormone relaxin, a protein with a molecular mass of about 6000. It is similar in its tertiary structure to insulin. Relaxin becomes detectable at about the same time as hCG begins to rise, and it maintains a maximum maternal serum concentration of about 1 ng/mL during the first trimester. The serum concentration then falls approximately 20% and is constant for the remainder of the pregnancy.

Pharmacologically, relaxin ripens the cervix, softens the pubic symphysis, promotes decidual angiogenesis, and acts synergistically with progesterone to inhibit uterine contractions. A major physiologic role for relaxin in human gestation has not been established. Luteectomy after 7 weeks of gestation does not interfere with gestation in spite of undetectable relaxin levels. Extraluteal production of relaxin by the decidua and placenta has been demonstrated, however, and local effects may be exerted without alteration of systemic hormone concentrations.

Human Chorionic Gonadotropin (HCG)

The first marker of trophoblast differentiation and the first measurable product of the placenta is hCG, a glycoprotein consisting of 237 amino acids. It is similar in structure to the pituitary glycoprotein hormones in that it consists of two chains: a common alpha chain, and a beta chain, The alpha chain is identical in sequence to the alpha chains of thyroid-stimulating hormone (TSH), follicle-stimulating hormone (FSH), and luteinizing hormone (LH).

In the early weeks of pregnancy (up to 6 weeks), the concentration of hCG doubles every 1.7–2 days, and serial measurements provide a sensitive index of early trophoblast function. Maternal plasma hCG peaks at about 100,000 mIU/mL during the tenth gestational week and then declines

Human Placental Lactogen (HPL)

A second placental polypeptide hormone, also with homology to a pituitary protein, is termed placental lactogen (hPL). hPL is a protein whose structures are similar to those of growth hormone (GH) and PRL. hPL is diabetogenic and lactogenic but it has minimal growth-promoting activity as measured by standard GH bioassays.

Other Chorionic Peptide Hormones & Growth Factors

Other chorionic peptides have been identified, but their functions remain poorly defined. Similarly, adrenocorticotropic hormone (ACTH)-like, lipotropin-like, and endorphin-like peptides have been isolated from placenta, but they have low biologic potency and undetermined physiologic roles. Activin, inhibin, corticotropin-releasing factor, and multiple peptide growth factors, including fibroblast growth factor (FGF), epidermal growth factor (EGF), platelet-derived growth factor (PDGF), and the IGFs—and many of their cognate receptors—have all been isolated from placental tissue. Placental growth factor (PlGF) and the related vascular endothelial growth factor (VEGF) have been suggested to play a role in placental angiogenesis, preeclampsia, and fetal growth.

Steroid Hormones:

In contrast to the impressive synthetic capability exhibited in the production of placental proteins, the placenta does not appear to have the capability to synthesize steroids de novo. All steroids produced by the placenta are derived from maternal or fetal precursor steroids.

Glucocorticoids from Maternal Adrenal Cortex

Total plasma cortisol concentrations increase to three times nonpregnant levels by the third trimester. Most of the change can be explained by increased corticosteroid-binding globulin (CBG). Which further causes anti-insulin effect in pregnant mother.

Androgens from Maternal Adrenal Cortex

In normal pregnancy, the maternal production of androgens is slightly increased. The most important determinant of plasma levels of specific androgens, however, appears to be whether or not the androgen binds to sex hormone-binding globulin (SHBG).

Progesterone:

The placenta relies on maternal cholesterol as its substrate for progesterone production. Fetal death has no immediate influence on progesterone production, suggesting that the fetus is a negligible source of substrate. Enzymes in the placenta cleave the cholesterol side chain, yielding pregnenolone, which in turn is isomerized to progesterone; 250–350 mg of progesterone is produced daily by the third trimester, and most enters the maternal circulation. The maternal plasma concentration of progesterone rises progressively throughout pregnancy and appears to be independent of factors that normally regulate steroid synthesis and secretion.

Estrogens

Estrogen production by the placenta also depends on circulating precursors, but in this case both fetal and maternal steroids are important sources. Most of the estrogens are derived from fetal androgens, primarily dehydroepiandrosterone (DHEA) sulfate. Fetal DHEA sulfate, produced mainly by the fetal adrenal, is converted by placental sulfatase to the free DHEA and then, through enzymatic pathways common to steroid-producing tissues, to androstenedione and testosterone. These androgens are finally aromatized by the placenta to estrone and estradiol, respectively.

MATERNAL ADAPTATION TO PREGNANCY:

As a successful "parasite," the fetal-placental unit manipulates the maternal "host" for its own gain but normally avoids imposing excessive stress that would jeopardize the pregnancy. The prodigious production of polypeptide and steroid hormones by the fetal-placental unit directly or indirectly results in physiologic adaptations of virtually every maternal organ system. Most of the commonly measured maternal endocrine function tests are radically changed. In some cases, true physiologic alteration has occurred; in others, the changes are due to increased production of specific serum binding proteins by the liver or to decreased serum levels of albumin. In addition, some hormonal changes are mediated by altered clearance rates due to increased glomerular filtration, decreased hepatic excretion of metabolites, or metabolic clearance of steroid and protein hormones by the placenta. The changes in endocrine function tests are summarized in Table1.

Table 1. Effect of Pregnancy on Endocrine Function Tests.		
	Test	**Result**
Pituitary		
FSH, LH	GnRH stimulation	Unresponsive from third gestational week until puerperium
GH	Insulin tolerance test	Response increases during the first half of pregnancy and then is blunted until the puerperium
	Arginine stimulation	Hyperstimulation during the first and second trimesters, then suppression
TSH	TRH stimulation	Response unchanged
Pancreas		
Insulin	Glucose tolerance	Peak glucose increases, and glucose concentration remains elevated longer
	Glucose challenge	Insulin concentration increases to higher peak levels
	Arginine infusion	Insulin response is blunted in mid to late pregnancy
Adrenal		
Cortisol	ACTH infusion	Exaggerated cortisol and 17-hydroxycorticosterone responses
	Metyrapone	Diminished response
Mineralocorticoids	ACTH infusion	No DOC response
	Dexamethasone suppression	No DOC response

MATERNAL PANCREAS

The nutritional demands of the fetus require alteration of maternal metabolic homeostatic control, which results in both structural and functional changes in the maternal pancreas. The size of pancreatic islets increases, and insulin-secreting cells undergo hyperplasia. Basal levels of insulin are lower or unchanged in early pregnancy but increase during the second trimester. Thereafter, pregnancy is a hyperinsulinemic state, with resistance to the peripheral metabolic effects of insulin. The increased concentration of insulin has been shown to be a result of increased secretion rather than decreased metabolic clearance. The measured half-life for insulin is unchanged in pregnant women. The effects of pregnancy on the pancreas can be mimicked by appropriate treatment with estrogen, progesterone, hPL, and corticosteroids.

Pancreatic production of glucagon remains responsive to usual stimuli and is suppressed by glucose loading, although the degree of responsiveness has not been well evaluated.

The major role of insulin and glucagon is the intracellular transport of nutrients, specifically glucose, amino acids, and fatty acids. These concentrations are regulated during pregnancy for fetal as well as maternal needs, and the pre- and post feeding levels cause pancreatic responses that act to support the fetal economy. Insulin is not transported across the placenta but rather exerts its effects on transportable metabolites. During pregnancy, peak insulin secretion in response to meals is accelerated, and glucose tolerance curves are characteristically altered. Fasting glucose levels are maintained at low normal levels. Excess carbohydrate is converted to fat, and fat is readily mobilized during decreased caloric intake.

Amino acid metabolism also is altered during pregnancy at the expense of maternal needs. Because alanine, the key amino acid for gluconeogenesis, is preferentially transported to the fetus, maternal hypoglycemia leads to lipolysis.

The normal metabolic effects of pregnancy are to reduce glucose levels modestly but to reserve glucose for fetal needs while maternal energy requirements are met increasingly by the peripheral metabolism of fatty acids. These changes in energy metabolism are beneficial to the fetus and innocuous to the mother with an adequate diet. Even modest fasting, however, can cause ketosis, which is potentially injurious to the fetus.

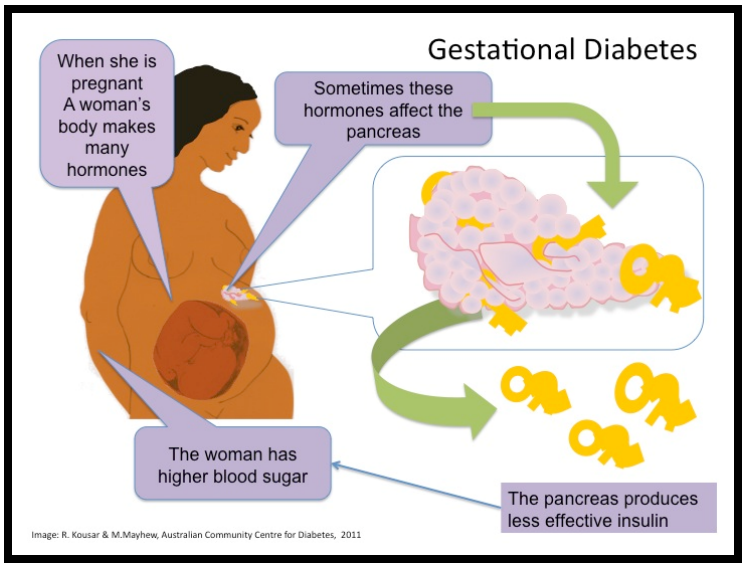

Figure 1: Pathogenesis of Gestational Diabetes.

FETAL ENDOCRINOLOGY

Because of the physical inaccessibility of the fetus, much of our information about fetal endocrinology is derived indirectly. Study of the fetal endocrine system is further complicated by the multiplicity of sources of the various hormones. Inferences from the behavior of adult endocrine systems are not transferable to the fetus, because target organs, receptors and other modulators develop at different times.

Dating of events in fetal development is usually given in "fetal weeks," which begin at the time of fertilization. Thus, fetal age is always 2 weeks less than gestational age.

Human fetal growth is influenced by endocrine and hemodynamic factors that dictate the distribution of nutrients between the mother and the conceptus. The main metabolic substrates for fetal and placental growth are glucose, lactate, amino acids, and lipids. A variety of placental transport proteins regulate the partitioning of these nutrients. In addition, placental hormones such as hPL, GH-variant, and IGF-I and IGF-II are secreted into the maternal and fetal circulations where they modulate energy metabolism and fetal growth.

The endocrine system is among the first to develop in fetal life.

So, all hormones are increased during pregnancy including insulin but we know except insulin all other hormones act in favor of increasing of glucose. As a result resistance against insulin become increased and all conditions in favor of making a mother diabetic.

PATHOPHYSIOLOGY OF GDM:

The main pathophysiologic defects that occur in GDM are the same as those observed with T2DM: marked insulin resistance and impairment of insulin secretion. The exact mechanisms responsible for these defects in GDM are not known.

All pregnancies are associated with an increase in insulin resistance and increased pancreatic insulin secretion as the pregnancy progresses. Skeletal muscle is the body's main site of glucose disposal and becomes insulin resistant during pregnancy. This insulin resistance begins in mid pregnancy and continues until the end of gestation. Pregnancies are also associated with a 200% to 250% increase in insulin secretion to maintain euglycemia in the mother. These metabolic changes are normal and provide adequate nourishment to the fetus. When maternal insulin secretion is unable to meet increased demand secondary to marked resistance, GDM results.

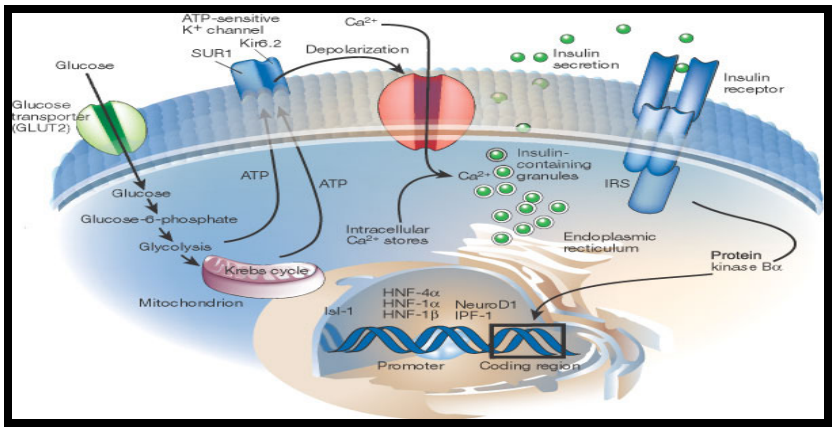

Figure2: Understanding the function of Beta cell in Pancreas

The cause for pancreatic beta-cell dysfunction and accompanying decrease in insulin secretion in GDM is categorized into three groups. The three groups are: 1) autoimmune, 2) monogenic, and 3) occurring on the background of insulin resistance. Additional maternal factors such as obesity also contribute to this insulin resistance. The exact maternal influences and the extent of their contribution are still poorly understood.

Placental hormones contribute to insulin resistance and secretion as well. The placenta is capable of producing a milieu of hormones and cytokines independently. Placental cytokines such as tumor necrosis factor alpha (TNFa), resistin, and leptin are known to contribute to the

insulin resistance of GDM. Important placental hormones include human chorionic somatomammotropin (HCS), cortisol, estrogen, progesterone, and human placental growth hormone (hPGH). HCS increases throughout pregnancy and stimulates maternal pancreatic insulin release. Placental overexpression of hPGH results in severe peripheral insulin resistance. It is thought that the cumulative effects of maternal and placental influences result in abnormalities in insulin signaling pathways, which lead to decreased glucose uptake and an increase in insulin resistance. The exact molecular processes of such remain unclear.

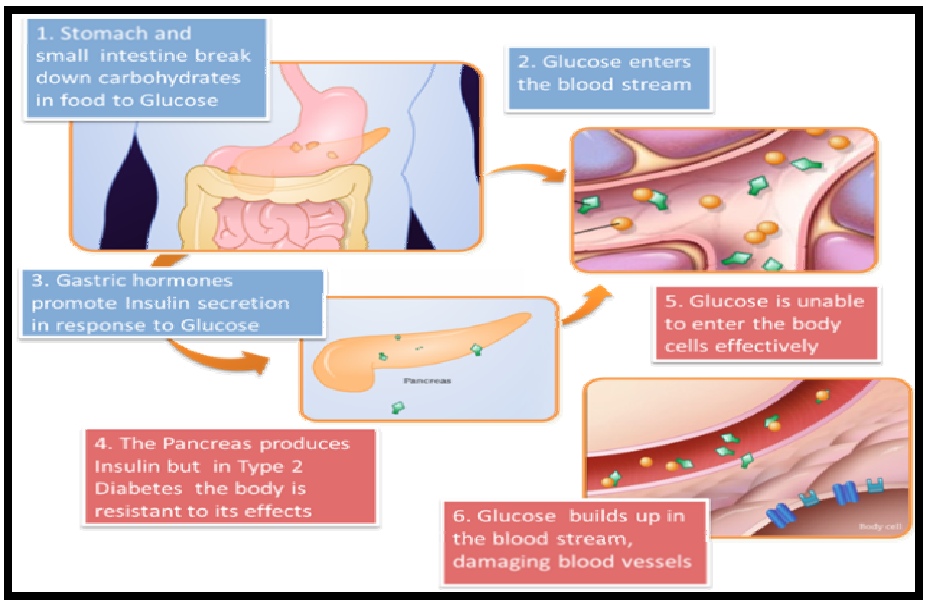

Figure 3: Mechanism of Type-2 Diabetes Mellitus (similar with GDM)

CAUSES:

Researchers don't yet know exactly why some women develop gestational diabetes. To understand how gestational diabetes occurs, it can help to understand how pregnancy affects body's normal processing of glucose.

Body digests the food to produce sugar (glucose) that enters bloodstream. In response, pancreas — a large gland behind stomach — produces insulin. Insulin is a hormone that helps glucose move from bloodstream into body's cells, where it's used as energy.

During pregnancy, the placenta that connects growing baby to blood supply produces high levels of various other hormones. Almost all of them impair the action of insulin in cells, raising blood sugar. Modest elevation of blood sugar after meals is normal during pregnancy.

As baby grows, the placenta produces more and more insulin-blocking hormones. In gestational diabetes, the placental hormones provoke a rise in blood sugar to a level that can affect the growth and welfare of baby. Gestational diabetes usually develops during the last half of pregnancy — sometimes as early as the 20th week.

RISK FACTORS:

Any woman can develop gestational diabetes, but some women are at greater risk. Risk factors for gestational diabetes include:

Age greater than 25. Women older than age 25 are more likely to develop gestational diabetes.

Family or personal health history. Your risk of developing gestational diabetes increases if you have prediabetes — slightly elevated blood sugar that may be a precursor to type 2 diabetes — or if a close family member, such as a parent or sibling, has type 2 diabetes. You're also more likely to develop gestational diabetes if you had it during a previous pregnancy, if you delivered a baby who weighed more than 9 pounds (4.1 kilograms), or if you had an unexplained stillbirth.

E**xcess weight.** You're more likely to develop gestational diabetes if you're significantly overweight with a body mass index (BMI) of 30 or higher.

Non-white race. For reasons that aren't clear, women who are black, Hispanic, American Indian or Asian are more likely to develop gestational diabetes.

SYMPTOMS:

Gestational diabetes may not cause symptoms, one need to be tested for the condition. It may be surprised if one's test shows a high blood sugar. It is important for mother to be tested for gestational diabetes, because high blood sugar can cause problems for her and her baby.

Sometimes a pregnant women who has been living with another type of diabetes without knowing it. If she has symptoms from another type of diabetes that may include:

- Increased thirst
- Increased urination
- Increased hunger
- Blurred vision

Pregnancy causes most women to urinate more often and to feel more hungry. So having these symptoms dose not always mean that a women has diabetes. Talk with doctor if one has these symptoms, so that she can be tested for diabetes at any time during pregnancy.

SCREENING AND DIAGNOSIS:

Medical experts haven't established a single set of screening guidelines for gestational diabetes. Some question whether gestational diabetes screening is needed if you're younger than 25 and have no risk factors. Others say that screening all pregnant women — no matter their age — is the best way to catch all cases of gestational diabetes.

When to screen

Risk factors for gestational diabetes should be assessed early in your pregnancy.

High risk of gestational diabetes: if body mass index (BMI) before pregnancy was 30 or higher

or have a mother, father, sibling or child with diabetes —doctor may test for diabetes at first prenatal visit.

Average and low risk of gestational diabetes: a screening test for gestational diabetes during

second trimester — between 24 and 28 weeks of pregnancy

Strategies for diagnosis are outlined in following table:

Table 2: Screening and Diagnosis of Gestational Diabetes.
Risk for gestational diabetes mellitus should be ascertained at the first prenatal visit
Low risk:
Universal versus selective screening remains controversial; most diabetes organizations state that blood glucose testing is not normally required if all of the following characteristics are present:
Member of an ethnic group with a low prevalence of gestational diabetes mellitus
No known diabetes in first-degree relatives
Age < 25 years
Weight normal before pregnancy
No history of abnormal glucose metabolism or poor pregnancy outcome
Average risk:
Perform blood glucose testing at 24–28 weeks using one of the following
One-step protocol: 75 g, 2-hour oral glucose tolerance test on all women:
Fasting: < 95 mg/dL (5.3 mmol/L)
1 hour: < 180 mg/dL (< 10 mmol/L)
2 hours: < 155 mg/dL (< 8.6 mmol/L)
Two-step protocol: 50 g, 1-hour plasma glucose on all women: if test done in fasting state, threshold is > 130 mg/dL (> 7.2 mmol/L); if test done in fed state, threshold is 140 mg/dL (> 7.8 mmol/L). Then test with 100 g, 3 hours, in fasting state:
Fasting: < 95 mg/dL (< 5.3 mmol/L)
1 hour: < 180 mg/dL (< 10 mmol/L)
2 hours: < 155 mg/dL (< 8.6 mmol/L)
3 hours: < 140 mg/dL (< 7.8 mmol/L)
If one value is abnormal, repeat test in 4 weeks
High risk:
Perform testing as soon as feasible. If negative, repeat at 24–28 weeks

MANAGEMENT OF BABY AFTER DELIVERY

After delivery, mother and baby still need to be monitored closely. For the first few hours blood sugar level may be tested every hour. Usually blood sugar levels quickly return to normal. Baby's blood sugar level will also be monitored. If blood sugar levels were high during pregnancy , baby's body will makes extra insulin for several hours after birth. This extra insulin may cause baby's blood sugar to drop too low-hypoglycemia, if baby's blood sugar level drops too low, he or she may need extra sugar, such as oral feeding or glucose given intravenously. Blood glucose levels must be cheeked by heel prick within 30-60 minutes of birth and continued at regular intervals until one is sure that there is no risk for hypoglycemia.

Neonatal hypoglycemia is defined as blood glucose level less than 40 mg/dl in full term baby and less than 30 mg/dl in premature babies. If glucometer reading are between 25 and 45mg/dl give gastric or oral feedings of 10-15 ml glucometer reading are less than 25 mg/dl, i.v. 10% dextrose at the rate of 6 mg/kg/min id started. Bolus dose are to be avoided as this may stimulated the already over active pancreas to secrete more insulin and add to the problem. Baby's blood may also be cheeked for low calcium, high billirubin and extra red blood cells.

GDM MANAGEMENT TEAM:

The whole purpose of managing gestational diabetes is to maintain normal blood glucose levels. So that we have the best possible outcome for mother and baby. A healthy pregnancy with a healthy birth is the greatest of rewards. The disease it self is not addressed. Only the result of the condition can be controlled. In other words, there is no treatment that will reduce the resistance to insulin.

Therefore, we should concentrate all efforts in minimizing the effect of the disease. All that is needed is to keep blood glucose levels in the normal range to successfully managing gestational diabetes.

It is possible to have a healthy pregnancy with a healthy baby, even with gestational diabetes. Gestational diabetes management team consist of following personals.

° An obstetrician- a specialist in the care of pregnant women.

° A general practitioner who has an interest or training in gestational diabetes

°A family doctor who has training in gestational diabetes

°A medical nurse who is trained in the care of pregnant women.

°A certified professional midwife.

°A physicians assistant who has training in the care of pregnant women.

°A registered dietitian- every person has very different personal and nutritional needs, essential for on going assessment and counseling .

°A certified diabetes educator (CDE)- expand understanding of gestational diabetes and help to adjust to living with gestational diabetes.

°Other specialist-

- An endocrinologist

- A perinatologist.

Even though all these professionals are ready to assist, they cannot do it. If mother cannot make the decision to put their advice in to actions.

MANAGEMENT:

Diet plan: The patient should be placed on a diabetic meal plan modified for pregnancy: 25–35 kcal/kg ideal weight, 40–55% carbohydrate, 20% protein, and 25–40% fat. Calories are distributed over three meals and three snacks (Table 2). Most patients can be taught to count their carbohydrates and to read food labels.

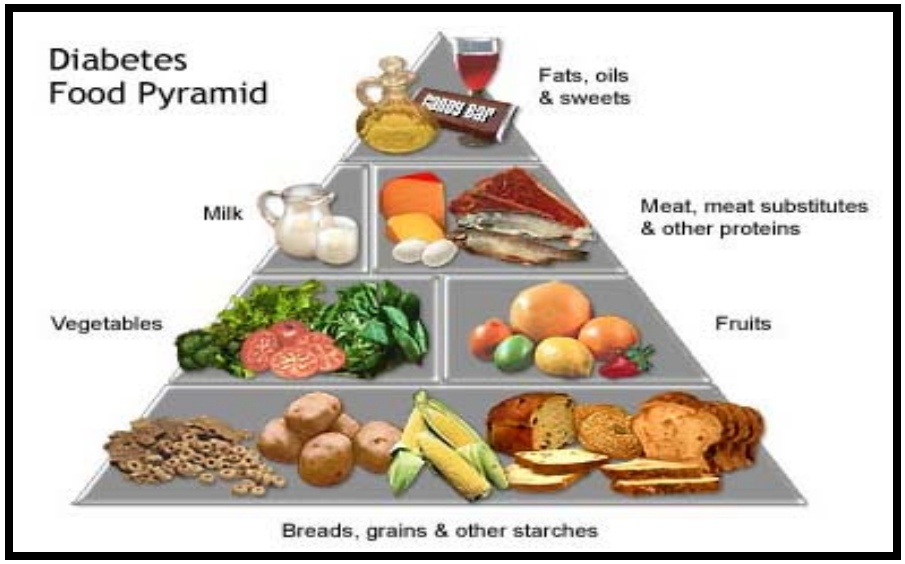

Figure 4: Food Pyramid for Diabetes Patient

The goal of therapy is not weight reduction but prevention of fasting and postprandial hyperglycemia. If fasting capillary blood glucose levels exceed 90–100 mg/dL (5–5.6 mmol/L) or if 1-hour or 2-hour postprandial glucose values are consistently greater (respectively) than 130 or 105 mg/dl (7.2 or 5.8 mmol/L), therapy is begun with human insulin.

As an alternative, consider the sulfonylurea glyburide, which crosses the human placenta poorly. Current research is evaluating its efficacy and safety when dietary therapy fails to produce normoglycemia.

Table 3. Management of Diet for Patients with Gestational Diabetes.
(1) Assess present pattern of food consumption.
(2) Balance calories with optimal weight gain.
(a) Caloric intake: 25–35 kcal/kg ideal weight.
(b) Weight gain: 0.45 kg (1 lb) per month during the first trimester; 0.2–0.35 kg (0.5–0.75 lb) per week during the second and third trimesters.
(3) Distribute calories and carbohydrates over 3 meals and 3 snacks; evening snack to include complex carbohydrate and at least one meat exchange.
(4) Use food exchanges to assess the amount of carbohydrate, protein, and fat:
(a) Carbohydrate: 40–55% of calories or 150 g/d.
(b) Protein: 20% of calories or 74 g/d.
(c) Fat: 25–40% of calories.
(5) Emphasize high-fiber, complex carbohydrate foods.
(6) Identify individual glycemic responses to certain foods.
(7) Tailor eating plans to personal needs.

Exercise :

Getting regular exercise, try to do at least 2.5 hours a week of moderate exercise. One way to do this is to be active 30 minutes a day, at least 5 days a weak. It's fine to be active in blocks of 10 minutes or more through out day and week. Regular moderate exercise during pregnancy helps body to use insulin better and helps to control blood sugar level. Low impact activates such as walking or swimming are especially good for pregnant women.

Figure5: Regular light exercise should be taken for Geststional Diabetes.

FOLLOW UP OF MOTHER AFTER PREGNANCY

Progression to type 2 diabetes later in life occurs in 5–50% of women with gestational diabetes. The wide range in incidence is influenced by body weight, family history, glucose levels, and the need for insulin treatment during pregnancy—and the choice of contraception and lifestyle after pregnancy. All patients with gestational diabetes should undergo a 75 g 2-hour glucose tolerance test at 6–10 weeks after delivery to guide future medical management. Follow-up protocols after pregnancy and criteria for the diagnosis of diabetes mellitus in the non-pregnant state are presented in Table 3.

Table 4. Follow-Up (after Pregnancy) of Patient with Gestational Diabetes Mellitus.

Encourage breast feeding.		
Monitor postprandial blood glucose occasionally to be sure it is < 180 mg/dL (< 10 mmol/L).		
Perform 75-g 2-hour oral glucose test at 6–12 weeks postpartum.		
Diagnosis	**Fasting Blood Glucose mg/dL (mmol/L)**	**Two-Hour Value mg/dL (mmol/L)**
Normal	< 100 (< 6.1)	< 140 (< 7.8)
Impaired glucose tolerance	100–125 (6.1–6.9)	140–199 (7.8–11.1)
Diabetes mellitus	> 125 (7.0)	> 199 (> 11.1)
Contraception: Barrier methods, Cu 7 IUD, low-dose birth control pills such as Ovcon 35, Triphasil (which do not affect glucose tolerance or lipid profiles).		
Use diet and exercise for women with impaired glucose tolerance and those with central body obesity. Impaired glucose tolerance implies a high risk of development of type 2 diabetes.		
Obtain annual blood glucose test of some kind and especially before the next pregnancy.		

Glucose Monitoring & Insulin Management

The goal of insulin therapy during pregnancy is to prevent both pre-prandial and postprandial hyperglycemia, but in patients with type 1 diabetes, caution must be used to avoid debilitating hypoglycemic reactions. Perinatal outcome is optimal if patients aim for fasting plasma glucose levels below 100 mg/dL (5.6 mmol/L) and postprandial levels below 130 mg/dL (7.2 mmol/L). In patients with type 1 diabetes and hypoglycemia unawareness, somewhat higher blood glucose targets should be selected.

Self-monitoring of capillary blood glucose should be done at home and in the workplace several times daily using glucose oxidase strips and portable reflectance colorimeters with memory capacity. Confirmation of long-term control is provided by sequential measurement of glycosylated hemoglobin and fructosamine.

Fifure 6: Regular Self-monitoring of capillary blood is a important part of GDM management.

Insulin: Most pregnant diabetic patients require at least two daily injections of a mixture of regular and intermediate insulin in order to prevent fasting and postprandial hyperglycemia.

Common insulin regimens are outlined in Table 4. The usual practice for initiation of insulin therapy in pregnant women with gestational diabetes mellitus or type 2 diabetes is to give two-thirds of the insulin before breakfast and one-third before supper.

More stringent regimens of administering short-acting subcutaneous insulin three times a day before meals and intermediate insulin at bedtime to control overnight and fasting glucose—or of continuous subcutaneous insulin infusion with a portable pump—is necessary to achieve normoglycemia in many women, especially those with type 1 diabetes.

Ultralente insulin has recently been discontinued by the manufacturer. Therefore, NPH is the only intermediate insulin available for basal coverage during pregnancy. In patients with type 1 diabetes requiring basal coverage during the day (especially those patients taking the short-acting analogs premeals), up to three small injections of NPH during the day may be necessary. Insulin glargine use during pregnancy has not been formally studied—anecdotal reports of its use during pregnancy have not reported any problems. These women also benefit from learning to self-adjust their doses of short-acting insulin based on planned carbohydrate load or premeal blood glucose levels.

Table 5. Illustration of Use of Home Blood Glucose Monitoring to Determine Insulin Dosage during Pregnancy.

Self-Monitored Capillary Blood Glucose		**Insulin Doses**
Fasting blood glucose	148 mg/dL (8.2 mmol/L)	14 units regular, 28 units intermediate
1 h after breakfast	206 mg/dL (11.4 mmol/L)	
1 h after lunch	152 mg/dL (8.4 mmol/L)	
1 h after supper	198 mg/dL (11.0 mmol/L)	9 units regular, 10 units intermediate
2–4 AM	142 mg/dL (7.9 mmol/L)	

Suggested changes based on pattern of blood glucose values over 2–3 days: slight increases in presupper intermediate insulin to control fasting blood glucose next day, in morning regular insulin to control postbreakfast glucose, and in presupper regular insulin to control postsupper hyperglycemia. Dose of morning intermediate insulin is adequate to control early afternoon blood glucose. When dose of presupper intermediate insulin is increased, patient should test to detect and prevent nocturnal hypoglycemia. One-hour postprandial testing is advised to detect the probable peaks of glycemic excursions. Patient should also test when symptoms of hypoglycemia appear.

Hypoglycemic reactions are more frequent and sometimes more severe in early gestation but are a risk at any time during pregnancy. Therefore, insulin-treated patients must use timely between-meal and bedtime snacks to prevent hypoglycemia—and patients with type 1 diabetes must keep glucagon on hand, and a member of the household must be instructed in the technique of injection. Hypoglycemic reactions have not been associated with fetal death or congenital anomalies, but they pose a risk to maternal health.

COMPLICATIONS IN MOTHER:

Vomiting of Pregnancy

In early gestation, diabetic gastroparesis or gastropathy can severely exacerbate the nausea and vomiting of pregnancy (hyperemesis gravidarum), which sometimes continues into the third trimester. Drugs stimulating gastric motility such as erythromycin may be useful, but many patients with this complication require hyperalimentation to achieve nutritional intake adequate for fetal development.

Diabetic Retinopathy

Pregnancy also affects diabetic retinopathy. Background diabetic retinopathy may develop or progress during pregnancy, but it usually regresses postpartum. If background retinopathy is already present in early pregnancy, the rate of progression to neovascularization over the course of the pregnancy (proliferative diabetic retinopathy) is 6% → 18% → 38%, depending on the extent of background retinopathy (mild → moderate→ severe preproliferative changes). The risk factors for progression to proliferative retinopathy include poor glycemic control before and during early pregnancy, rapid improvement in glycemic control during pregnancy, hypertension, and perhaps the many growth factors derived from placental tissue. These risks are an important reason to institute intensified preconception management of diabetes. During pregnancy, sequential ophthalmologic examinations are essential in women with type 1 or type 2 diabetes, and laser photocoagulation treatment of the retina may be necessary.

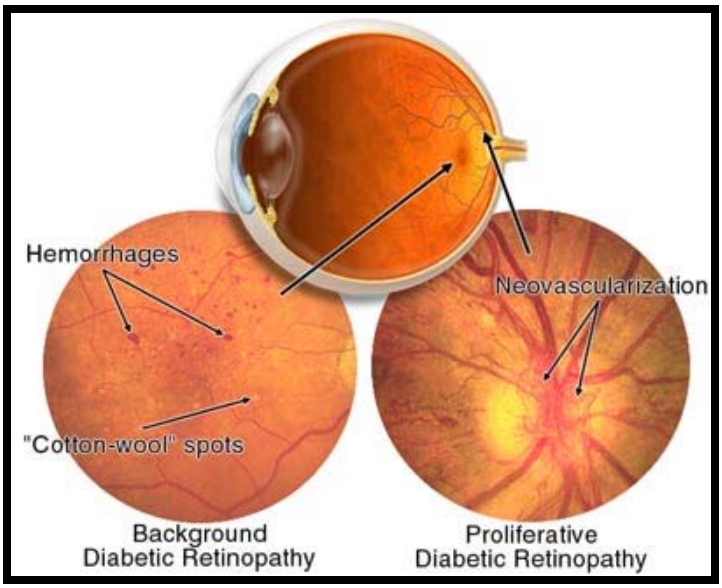

Figure 7 : Development of Diabetic Retinoplthy.

Diabetic Nephropathy

The risk of worsening of diabetic nephropathy during pregnancy depends on baseline renal function and the degree of hypertension. Total urinary albumin excretion does not increase substantially in normal pregnancy, but total urinary protein collections, which obstetricians have used to define preeclampsia, may show a twofold increase in uncomplicated gestation. Diabetic women with microalbuminuria (30–299 mg/24 h) may have worsening of the albuminuria during pregnancy with regression postpartum, and 15–45% develop the preeclamptic syndrome. Based on pooled data from several studies of pregnant diabetic women with a clinical level of proteinuria (24-hour urinary albumin > 300 mg) at the beginning of pregnancy, if initial renal function is preserved (serum creatinine < 1.2 mg/dL [< 106 μmol/L]; creatinine clearance > 80 mL/min with complete collection), then 15–20% are expected to show moderate decline during gestation, and 6% have renal failure at follow-up several years after pregnancy. The latter figure may not be different from the course of diabetic nephropathy in nonpregnant women with this level of initial renal function. If initial renal function in pregnancy is impaired (serum creatinine > 1.2 mg/dL [> 106 μmol/L]; creatinine clearance < 80 mL/min with complete collection), then 35–40% are expected to show further decline during pregnancy, and 45–50% have renal failure at follow-up several years later. Thus, careful preconception counseling is important for these patients and their family members.

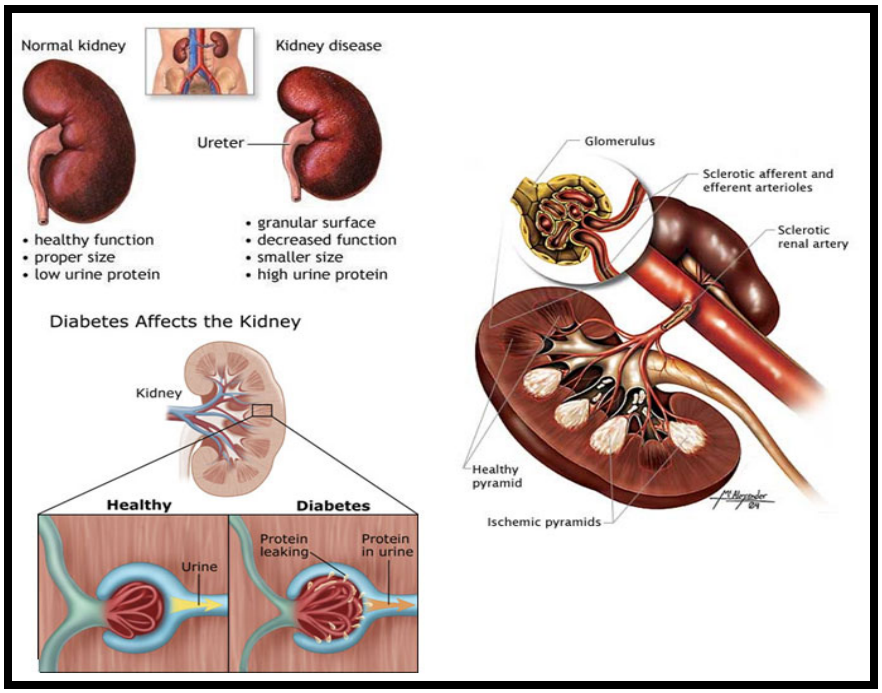

Figure 8: Diagram showing the development of Diabetic Nephropathy

Diabetic Neuropathy

The course of diabetic neuropathy is uncertain during pregnancy, and treatment may be relatively ineffective. The agents commonly used (amitriptyline, desipramine) may produce neonatal withdrawal symptoms.

Fetal Development & Growth

Congenital Anomalies

Major congenital anomalies are those that may affect the life of the individual or require major surgery for correction. The incidence in infants of poorly controlled diabetic mothers is 6–12%, compared with about 2% in infants born to diabetic women who begin pregnancy with normal glycohemoglobin or infants of a nondiabetic population. Because perinatal deaths due to stillbirth and respiratory distress syndrome have declined in pregnancies complicated by diabetes, the proportion of fetal and neonatal deaths ascribed to congenital anomalies has risen to over 50%. The types of anomalies most common in infants of diabetic mothers and their presumed time of occurrence during embryonic development are listed in Table 5. It is apparent that any intervention to reduce the incidence of major congenital anomalies must be applied very early in pregnancy. The finding that the excess risk of anomalies is associated with the group of diabetic women with elevated glycosylated hemoglobin early in pregnancy suggests that poor diabetic control is related to the risk of major congenital anomalies in their infants. Protocols of intensive diabetic management, instituted prior to conception and continued through early pregnancy, have resulted in significant reduction in the frequency of anomalies. Primary care physicians treating diabetic women of reproductive age should counsel them about the possibility and risks of pregnancy and help them achieve good glycemic control if pregnancy is desired.

Table 6. Congenital Malformations in Infants of Diabetic Mothers.[1]		
	Ratio of Incidences Diabetic vs Control Group	**Latest Gestational Age for Occurrence (Weeks after Menstruation)**
Caudal regression	252	5
Anencephaly	3	6
Spina bifida, hydrocephalus, or other central nervous system defects	2	6
Cardiac anomalies	4	
Transposition of great vessels		7
Ventricular septal defect		8
Atrial septal defect		8
Anal/rectal atresia	3	8
Renal anomalies	5	
Agenesis	6	7
Cystic kidney	4	7
Ureter duplex	23	7
Situs inversus	84	6

[1]Modified and reproduced, with permission, from Kucera J: Rate and type of congenital anomalies among offspring of diabetic women. J Reprod Med 1971;7:61; and Mills JL, Baker L, Goldman AS: Malformations in infants of diabetic mothers occur before the seventh gestational week: Implications for treatment. Diabetes 1979;28:292.

Neural Tube Defects

Ultrasonography in the first half of pregnancy confirms the dating of gestation and may detect neural tube defects (anencephaly, meningomyelocele) that occur with a higher incidence in infants of poorly controlled diabetic mothers. The physician should also screen all insulin-dependent pregnant women at 14–16 weeks of gestation for elevated serum alpha-fetoprotein levels that may suggest less severe cases of neural tube defects (eg, spina bifida). Later in pregnancy, at 18–22 weeks, sophisticated ultrasonographic examinations are used to detect congenital heart defects or other severe anomalies. Subsequent examinations at 26 and 36 weeks measure fetal growth and well-being.

Macrosomia

Many fetuses of poorly controlled diabetic mothers are macrosomic (large for dates), with increased fat stores, increased length, and increased abdomen-to-head or thorax-to-head ratios. The hypothesis that fetal macrosomia results from the causal chain of maternal hyperglycemia →fetal hyperglycemia →fetal hyperinsulinemia →fetal macrosomia has been confirmed by clinical and experimental studies. Macrosomic infants of diabetic mothers have significantly higher concentrations of C peptide in their cord sera or amniotic fluid

(representing endogenous insulin secretion) than do those with birth weights appropriate for gestational age. Monkey fetuses with insulin-releasing pellets implanted in utero become macrosomic. However, in human pregnancies, the determinants of fetal hyperinsulinemia may be more than maternal hyperglycemia. Other metabolic substrates that cross the placenta, such as branched-chain amino acids, are insulinogenic and may play a role in fetal macrosomia, and transplacental lipids could contribute to fat deposition.

Figure 9: Left is normal healthy baby and right side is macrosomic large baby.

The level of maternal glycemia is related to birth weight adjusted for gestational age, and prevention of maternal hyperglycemia throughout pregnancy can reduce the incidence of macrosomia and birth trauma. The glycemic threshold for fetal macrosomia seems to be *postprandial* peak values above 130 mg/dL (7.2 mmol/L). On the other hand, excessively tight glycemic control (average peak postprandial blood glucose levels < 110 mg/dL [6.1 mmol/L]) can be associated with insufficient fetal growth and small-for-date infants, which may also induce complications in the neonatal period.

Polyhydramnios

Polyhydramnios is an excess volume of amniotic fluid (> 1000 mL, often > 3000 mL). It may cause severe discomfort or premature labor and is most often associated with fetal macrosomia. The excess volume of amniotic fluid is not related simply to the concentration of glucose or other solutes in amniotic fluid or to excess fetal urine output as measured by change in bladder size on ultrasonography. Other possible factors include decreased fetal swallowing, decidual and amniotic fluid prolactin, and as yet unknown determinants of the complicated multicompartmental intrauterine transfer of water. Polyhydramnios is rare in women with well-controlled diabetes.

Growth Retardation

In contrast to fetal macrosomia, the fetus of a woman with diabetes of long duration and vascular disease may suffer intrauterine fetal growth restriction related to inadequate uteroplacental perfusion. All body diameters may be below normal on ultrasonographic measurements, but the abdominal circumference is especially affected, and oligohydramnios and abnormal Doppler flow measurements of the umbilical cord are common. In these patients provision of adequate rest, meticulous control of hypertension (target < 135/85 mm Hg), maintenance of normal blood glucose levels, and intensive fetal surveillance are all essential for success.

Figure 10: Some still birth babies from Diabetic mother.

Intrauterine Death

Prior to the 1970s, the incidence of apparently sudden intrauterine fetal demise in the third trimester of diabetic pregnancies was at least 5%. Because the risk increased as pregnancies approached term, iatrogenic preterm delivery was instituted, but the incidence of neonatal deaths from respiratory distress syndrome increased. Except for congenital malformations, the cause of stillbirth is often not obvious. The risk is greater with poor diabetic control, and the incidence of fetal death exceeds 50% if ketoacidosis develops in the mother. Some instances of fetal demise are associated with preeclampsia-eclampsia, which is a common complication in diabetic pregnant women. Fetal death has also been associated with pyelonephritis, which is now largely prevented by screening for and treating asymptomatic bacteriuria. Other than these known risk factors, one can presume—based on experimental studies—that the combination of fetal hyperglycemia and hypoxia leads to acidosis and myocardial dysfunction. Good glycemic control in diabetic women greatly reduces the risk of stillbirth.

Obstetric Management of GDM mother

Monitoring

Technologic advances have led to techniques for detecting fetal hypoxia and preventing stillbirth (see Table 6). Most simply, the infrequency of fetal movement as noted in regular fetal kick counts (few than four per hour) may indicate fetal jeopardy. More rigorous analysis of fetal activity patterns using ultrasonography is known as the "fetal biophysical profile," which assesses gross body movements, the tone of the limbs, and chest wall motions as well as reactivity of the fetal heart rate (FHR) and the volume of amniotic fluid. The measurement of maternal estriol levels for fetal evaluation is now of only historical interest. The presence of FHR accelerations and long-range variability on the nonstress test (NST) and the absence of late decelerations (lower FHR persists after the contraction subsides) on the contraction stress test (CST) indicate that the fetus is well oxygenated. However, the predictive value of a normal result is valid only for a short duration in diabetic women with unstable metabolic control or hypertension. These patients may have to be hospitalized for daily fetal testing. Generally, the NST and CST are sensitive screening tests, and abnormal results of FHR monitoring in these tests overestimate the diagnosis of fetal distress. Therefore, it is wise to obtain additional evidence of fetal jeopardy (by biophysical ultrasonographic assessment) before cesarean delivery is recommended in preterm pregnancies. In term gestation with abnormal fetal testing, there is little to be gained by continuing the pregnancy.

Table 7. Schedule of Obstetric Tests and Procedures.		
	Risk Based on Glycemic Control, Presence of Vascular Disease	
Procedure	**Low Risk**	**High Risk**
Ultrasound to date gestation	8–12 weeks	8–12 weeks
Prenatal genetic diagnosis	As needed	As needed
Targeted perinatal ultrasound; fetal echocardiography	18–22 weeks	18–22 weeks
Fetal kick counts	28 weeks	28 weeks
Ultrasound for fetal growth	28 and 37 weeks[1]	Every 3–8 weeks
Antepartum FHR monitoring, backup with biophysical profile	36 weeks, weekly	27 weeks, 1–3 per week
Amniocentesis for lung	. . .	35–38 weeks
Induction of labor	41 weeks[2]	35–38 weeks

[1]Not needed in normoglycemic, diet-treated women with gestational diabetes mellitus.

[2]Earlier for obstetric reasons or for impending fetal macrosomia.

Timing of Delivery

Unless maternal or fetal complications arise, the goal for delivery in diabetic women should be 38–41 weeks in order to reduce neonatal morbidity from preterm deliveries. On the other hand, the obstetrician may wish to induce labor before 39 weeks if there is concern about increasing fetal weight. Before a preterm delivery decision (< 37 weeks) is made—or at 37–38 weeks in women with poor glycemic control—fetal pulmonary maturity should be determined. Tests for maturity using amniocentesis predict a low risk of neonatal respiratory distress syndrome and include the lecithin/sphingomyelin (L/S) ratio, phosphatidylglycerol, and other biochemical or physical assays of surfactant activity. In pregnancies complicated by hyperglycemia, fetal hyperinsulinemia can lead to low pulmonary surfactant apoprotein production. The lowest risk of respiratory distress syndrome is attained by delaying delivery (if possible) until 38–41 weeks and minimizing the need for cesarean sections.

Route of Delivery

Once fetal lung maturity is likely, the route of delivery must be selected based on the usual obstetric indications. If the fetus seems large (> 4200 g) on clinical and ultrasonographic examination of diabetic women, cesarean section probably should be performed because of the possibility of shoulder dystocia and birth trauma. Otherwise, induction of labor is reasonable, because maternal and peripartum risks are fewer following vaginal delivery. Once labor is under way, continuous FHR monitoring is essential. Maternal blood glucose levels greater than 150 mg/dL (8.3 mmol/L) can be associated with intrapartum fetal hypoxia.

INSULIN MANAGEMENT DURING LABOUR:

The diabetic parturient may be unusually sensitive to insulin during active labor and delivery, and severe maternal hypoglycemia is possible if delivery occurs sooner than anticipated and a high dose of subcutaneous intermediate-acting insulin was previously administered. Protocols for continuous low-dose intravenous insulin administration during labor or prior to cesarean delivery are used to achieve stringent control of blood glucose in order to reduce the incidence of intrapartum fetal distress and neonatal metabolic problems (Table 7). A cord blood glucose level at delivery correlates positively with the higher maternal levels, and there is no upper limit on placental transfer of glucose. During labor, maternal plasma glucose can usually be kept below 110 mg/dL (6.1 mmol/L) with 1–2 units of regular insulin and 7.5 g of dextrose given intravenously every hour. If cesarean section is necessary, insulin management is similar, and infants do equally well with general, spinal, or epidural anesthesia as long as the diabetic parturient does not receive rapid high-volume loads of glucose-containing intravenous solutions.

Table 8. Protocol for Intrapartum Insulin Infusion.[1]

Intravenous fluids		
If blood glucose is > 130 mg/dL (> 7.2 mmol/L), infuse mainline Ringer's lactate at a rate of 125 mL/h.		
If blood glucose is < 130 mg/dL (< 7.2 mmol/L), infuse mainline Ringer's lactate to keep vein open and begin Ringer's lactate and 5% dextrose at a rate of 125 mL/h controlled by infusion pump.		
Insulin infusion		
Mix 25 units of regular human insulin (U100) in 250 mL NaCl 0.9% and piggyback to mainline. The concentration is 1 unit/10 mL. Adjust intravenous insulin hourly according to the following table when the blood glucose is > 70 mg/dL (> 3.9 mmol/L).		
Blood Glucose mg/dL (mmol/L)	**Insulin (units/h)**	**Infusion (mL/h)**
< 70 (< 3.9)	None	None
71–90 (3.9–5)	0.5	5
91–110 (5.1–6.1)	1	10
111–130 (6.2–7.2)	2	20
131–150 (7.3–8.3)	3	30
151–170 (8.4–9.4)	4	40
171–190 (9.5–10.6)	5	50
> 190 (> 10.6)	Call MD and check urine ketones	

[1]Protocol useful also for diabetic pregnant women who are "NPO" or being treated with beta-adrenergic tocolysis or corticosteroids. The scale dosages may need to be doubled for the latter. Boluses of short-acting insulin must be used to cover meals.

NEONATAL MORBIDITY:

Planning for the care of the infant should be started prior to delivery, with participation by the pediatrician or neonatologist in decisions about timing and management of delivery. In complicated cases, the pediatrician must be in attendance to learn about antenatal problems, to assess the need for resuscitation, to identify major congenital anomalies, and to plan initial therapy for the sick infant if required.

Respiratory Distress Syndrome

Infants of mothers with poorly controlled diabetes have an increased risk of respiratory distress syndrome. Possible reasons include abnormal production of pulmonary surfactant or connective tissue changes leading to decreased pulmonary compliance. However, in recent years, the incidence of respiratory distress syndrome has declined from 24% to 5%, probably related to better maternal glycemic control, selected use of amniotic fluid tests, and delivery of most infants at term (see above). The diagnosis of respiratory distress syndrome is based on clinical signs (grunting, retraction, respiratory rate > 60/min), typical findings on chest x-ray (diffuse reticulogranular pattern and air bronchograms), and an increased oxygen requirement (to maintain the PaO_2 at 50–70 mm Hg) for more than 48 hours with no other identified cause of respiratory difficulty (heart disease, infection). Survival of infants with respiratory distress syndrome has dramatically improved as a result of advances in ventilation therapy and intrapulmonary administration of surfactant.

Hypoglycemia

Hypoglycemia is common in the first 48 hours after delivery of previously hyperglycemic mothers and is defined as blood glucose below 30 mg/dL (1.7 mmol/L) regardless of gestational age. The symptomatic infant may be lethargic rather than jittery, and hypoglycemia may be associated with apnea, tachypnea, cyanosis, or seizures. Hypoglycemia has been related to elevated fetal insulin levels during and after delivery. Infants of diabetic mothers may also have deficient catecholamine and glucagon secretion, and the hypoglycemia may be related to diminished hepatic glucose production and oxidation of free fatty acids. The pediatrician attempts to prevent hypoglycemia in "well" infants with early feedings of 10% dextrose in water by bottle or gavage by 1 hour of age. If this is not successful, treatment with intravenous dextrose solutions is indicated. There are usually no long-term sequelae of episodes of neonatal hypoglycemia.

Other possible problems in infants of diabetic mothers include hypocalcemia less than 7 mg/dL (1.75 mmol/L), hyperbilirubinemia greater than 15 mg/dL (256 μmol/L), polycythemia (central hematocrit > 70%), and poor feeding. These complications are also somehow related to fetal hyperglycemia and hyperinsulinemia and probably to intermittent low-level fetal hypoxia. Improved control of the maternal diabetic state has reduced their incidence.

Pregnancy with previously diagnosed Diabetes

The hormonal and metabolic effects of pregnancy are associated with increased risks of both hypoglycemic insulin reactions and ketoacidosis. Increasing amounts of insulin are usually required to control hyperglycemia throughout gestation.

If diabetes is poorly controlled in the first weeks of pregnancy, the risks of spontaneous abortion and congenital malformation of the infant are increased. Later in pregnancy, polyhydramnios is also common in women with poorly controlled diabetes and may lead to preterm delivery. Fetal hypoxia may develop in the third trimester if blood glucose levels frequently exceed 180 mg/dL (10 mmol/L). In poorly controlled patients, careful fetal monitoring must be used to prevent stillbirth. The high incidence of fetal macrosomia (birth weight > 90th percentile for gestational age) associated with maternal hyperglycemia and fetal hyperinsulinemia increases the potential for traumatic vaginal delivery; cesarean deliveries are more common in these cases. Fetal intrauterine growth restriction may occur in diabetic women with vascular disease or those with relative hypoglycemia induced by overzealous treatment.

Neonatal risks linked to maternal glycemic control include respiratory distress syndrome, hypoglycemia, hyperbilirubinemia, hypocalcemia, and poor feeding. Although these problems are usually limited to the first days of life, excess maternal glucose and β-hydroxybutyrate levels with the fetus in utero have been related to diminished performance on intelligence and psychomotor testing during subsequent childhood development. However, if women with diabetes adhere to a program of careful management and surveillance, they have greater than 95% chance of delivering a healthy child.

PREVENTION

There are no guarantees when it comes to preventing gestational diabetes — but the more healthy habits one can adopt before pregnancy, the better. If one have had gestational diabetes, these healthy choices may also reduce your risk of having it again in future pregnancies or developing type 2 diabetes down the road.

Eat healthy foods: Foods should be high in fiber and low in fat and calories. Focus on fruits, vegetables and whole grains. Strive for variety to help the achievement of the goals without compromising taste or nutrition.

Keep active. Exercising before and during pregnancy can help protect against developing gestational diabetes. Aim for 30 minutes of moderate activity on most days of your week. Take a brisk daily walk. If anybody can't fit a single 30-minute workout into busy day, several shorter sessions can do just as much good. Park in the distant lot when run errands. Get off the bus one stop before reach the destination. Taking of every step increases the chances of staying healthy.

Lose excess pounds before pregnancy. Doctors don't recommend weight loss during pregnancy because body is already working overtime to support baby's development. But if anyone is planning to get pregnant, losing extra weight beforehand may help you have a healthier pregnancy. Focus on permanent changes to eating habits. Motivation by remembering the long-term benefits of losing weight, such as a healthier heart, more energy and improved self-esteem.

RESPONSIBILITY OF HEALTH PROFESSIONALS (PHYSICIANS, NURSES, PARAMEDICS AND HEALTH WORKERS) TO PREVENT THE GESTATIONAL DIABETES MELLITUS:

1) Aware all female patients (15 to 45 years) about GDM during your daily practice.
2) Advice FBS and GTT to all pregnant mother during 18^{th} and 28^{th} week of pregnancy.
3) After diagnosis of GDM, treat the patient according to proper guide line.
4) Advice all GDM affected patients for USG of fetus to detect any anomalies of the fetus and weight.
5) After delivery follow up the mother for evaluating her Diabetic status.
6) Advice all GDM affected mother to do FBS and PPBS for diagnosis of type -2 Diabetes Meellitus.
7) Give special attention about blood pressure and self monitoring of blood glucose.
8) Measure urinary albumin routinely.
9) Empower your patient to titrate their insulin dose according to Home monitoring of blood glucose by proper counseling.
10) Educate your patients about the route of delivery, harmful effects of GDM on maternal and fetal health.

RESPONSIBILITY OF SOCIAL LEADERS (POLITICAL LEADERS, RELIGIOUS LEADERS AND VOLUNTEERS) TO PREVENT THE GESTATIONAL DIABETES MELLITUS:

1) Discuss about the harmful effect of the GDM among the general population in various public meeting and religious meeting.
2) Discuss about the prevention of GDM among the various class of general population.
3) Take initiation to raise the awareness about GDM in local school and colleges.
4) If needed, take step to make small committee to make awareness about GDM among the community people.
5) Give input to help for making national planning preventing GDM.

ACTIVITIES OF HDRCRP IN PICTURE ABOUT GESTATIONAL DIABETES MELLITUS

Picture-1: Awareness Training Program among College Students about GDM

Picture-2: One of the Awareness Training Program among College Students about GDM

Picture-3: National Advocacy Meeting with Physicians, Nurses, NGO Officials, Journalists & others professionals about GDM

Picture-4: A Part of National Advocacy Meeting with Physicians, Nurses, NGO Officials, Journalists & others professionals about GDM

Picture 5: A part of Participants of National Advocacy Meetings

Picture-6: One of the Advocacies Meeting with Physicians about GDM

Picture-7: Awareness Training Program among School Students about GDM

Picture-8: One of the Community Level Awareness Rising Training Program about GDM

Picture-9: A part of the Community Level Awareness Rising Training Program about GDM

Picture-10: One of the Advocacy Awareness Training Program about GDM

Picture-11: One of the Advocacy Awareness Training Program about GDM

Picture-12: one of the Awareness Training Program among College Students About GDM

Picture-13: One of the Community Level Awareness Rising Training Program about GDM

Picture-14: Community Level Baseline Survey Program for search GDM

Picture-15: One of the Community Level Baseline Survey Program for search GDM

Picture-16: One of the Community Level Baseline Survey Program for search GDM

Picture-17: One of the Community Level Baseline Survey Program for search GDM

Picture-18: One of the Community Level Baseline Survey Program for search GDM

Picture-19: One of the Health Professional Awareness Training Program about GDM

Picture-20: A part of the Health Professional Awareness Training Program about GDM