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Gestational Diabetes and Insulin Use in Pregnancy

Sinisa Franjic*

Independent Researcher, Croatia

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***Corresponding author:** Siniša Franjić, Independent Researcher, Croatia

Abstract

Diabetes in pregnancy can significantly endanger the health of a pregnant woman, especially the vulnerable population and her child, and it is extremely important to talk about this disease. Diabetes mellitus or diabetes is a metabolic disease in which there is a chronic condition of elevated blood sugar levels (hyperglycemia) due to insufficient action of insulin, a hormone secreted by the pancreas. In some cases of the disease, insulin is not produced or is produced very little, and in other cases it is produced even in increased amounts, but the cells are resistant to its action, which is called insulin resistance. Sugar, or glucose, is the fuel that the human body needs to function properly. It is a source of energy for all the cells in our body and is created by the breakdown of carbohydrates in the digestive system. In order for our cells to be able to use the obtained glucose, they need insulin - a hormone that serves to transfer glucose to the cells. Precisely because of this, if the action of insulin is disturbed, the cells, but also the whole human body, do not function in a normal way. Because glucose cannot enter cells due to the ineffective action of insulin, it accumulates in the blood. High blood glucose levels damage small blood vessels in the kidneys, eyes, heart and nervous system. If diabetes is not treated, severe irreversible organ damage can occur. There are three main types of diabetes: type 1, type 2 and gestational diabetes. Pregnancy exacerbates existing type 1 and 2 diabetes, but also in some women, the disease may first appear in pregnancy, which is called gestational diabetes.

Keywords: Diabetes; Gestational diabetes; Insulin; Pregnancy; Health

Abbreviations: HPL: Human Placental Lactogen; DM: Diabetes Melitus; GDM: Gestational Diabetes Mellitus; ADA: American Diabetes Association; PCOS: Polycystic Ovary Syndrome; SHBG: Sex Hormone-Binding Globulin; WHO: World Health Organization

Introduction

Insulin is a pancreatic hormone that's essential for the regulation of glucose [1]. Lack of insulin and also the inability to adequately reply to insulin are primary causes of diabetes. Insulin contains a similar function in males and females. However, insulin's ability to control blood sugar is significantly more challenging in females than in males due to the influence of female hormones on blood sugar.

Insulin

a) Regulates the body's use of glucose [1]

b) Controls blood sugar levels

c) Regulates fat storage

d) Provides the signals required by the liver, muscles, and fat to take in glucose from the blood

e) Signals the liver to take in glucose and store it as glycogen

Maternal metabolism is directed toward supplying adequate nutrition for the fetus [2]. In pregnancy, placental hormones cause insulin resistance at tier that tends to parallel the expansion of the fetoplacental unit. because the placenta grows, more placental hormones are secreted. Human placental lactogen (HPL) and hormone (somatotropin) increase in direct correlation with the expansion of placental tissue, rising throughout the last 20 weeks of pregnancy and causing insulin resistance. Subsequently, insulin secretion increases to beat the resistance of those two hormones. within the nondiabetic pregnant woman, the pancreas can answer the demands for increased insulin production to keep up normal glucose levels throughout the pregnancy. However, the woman with glucose intolerance or diabetes during pregnancy cannot cope with changes in metabolism resulting from insufficient insulin to fulfill the needs during gestation.

Over the course of pregnancy, insulin resistance changes. It peaks within the last trimester to produce more nutrients to the fetus. The insulin resistance typically leads to postprandial

hyperglycemia, although some women even have an elevated fasting blood glucose level. With this increased demand on the pancreas in late pregnancy, women with diabetes or glucose intolerance cannot accommodate the increased insulin demand; glucose levels rise as a results of insulin deficiency, leading to hyperglycemia. Subsequently, the mother and her fetus can experience major problems.

In general, pregnancy increases medication requirements for adequate glucose control within the second and third trimesters [3]. Women with type 1 diabetes may become more brittle within the first trimester and thus prone to hypoglycemia. additionally, hyperemesis may complicate oral intake for diabetic pregnant women. Management of diabetic women having surgical abortions depends partly on plans for pain management. No changes in diet or medication are required for those having abortions under local anaesthesia. When deeper sedation requires preprocedure fasting, a common approach is to have the patient inject half her usual long-acting insulin dose the evening before and skip the morning dose. Ideally, a woman with diabetes is scheduled to have one in every of the first procedures of the day, in order that are going to be able to eat and take her usual dose of morning insulin afterwards; morning NPH will be active within the afternoon. Glucose is monitored frequently by finger stick before, during, and after the procedure, with insulin given as required until the patient resumes eating. Although sliding scales of insulin doses associated with blood glucose levels are used in hospitals for many years, little evidence supports this approach.

When advising women regarding medications and diet, providers should keep in mind that modest hyperglycemia poses no acute risk to women undergoing an abortion. "Loose" control of diabetes round the time of the procedure is preferred to "tight" control. as an example, a transient blood glucose concentration of 180 to 200mg/dL during an abortion isn't worrisome, whereas a blood glucose level of 30mg/dL is. Hence, providers should have food, intravenous glucose solutions, or glucagon available. After the procedure, the patient's medication requirements may decrease substantially. Coordination of care along with her medical provider is usually recommended, especially during this transitional time.

Effects

Any elevated blood glucose concentration within the maternal blood (hyperglycemia), as in diabetes (described within the discussion on the pancreas and diabetes mellitus), is harmful to the developing fetus [4]. In early pregnancy when the organ systems are developing, the hyperglycemia may cause congenital malformations or maybe result in fetal death. Later within the pregnancy, the additional glucose crossing the placenta into the fetal bloodstream causes the fetal pancreas to release more insulin, and also the extra glucose is metabolized to promote fetal growth. As a result, the fetus becomes larger than average size; the larger fetal kidneys secrete more urine, which contributes to the quantity of amnionic fluid and causes its volume to increase. Delivery of the oversized fetus is also tougher, possibly requiring a cesarean section. After delivery, the infant's blood glucose may fall precipitously (hypoglycemia) because the newborn infant's pancreas has been at home with handling a much higher intrauterine blood glucose concentration and has not had time to compensate for the lower postdelivery blood glucose. The infant also faces neonatal respiratory distress problems caused by inadequate surfactant, as described within the discussion on the respiratory system. For these reasons, recognition and treatment of hyperglycemia in pregnancy are within the best interest of both the mother and her baby.

Pregnant women should be evaluated on their initial visit for any risk factors— like obesity, gestational diabetes in a previous pregnancy, a family history of diabetes, and the other factors that may predispose her to gestational diabetes—and some form of screening test should be performed. the usual screening test consists of 50g of glucose solution given orally without relation to fasting status, followed by determining the concentration of glucose within the patient's blood one hour later. If the result exceeds a predetermined concentration, more comprehensive studies are performed to confirm the diagnosis of gestational diabetes, and a course of treatment is begun.

Pregnant women are routinely screened for gestational diabetes in many developed countries, during the second half of the second trimester, because it is related to adverse perinatal outcomes [5]. Treatment usually commences with dietary and exercise changes, with insulin common because the first-line medical treatment. However, only if critical illness is related to altered carbohydrate metabolism, screening for gestational diabetes while a pregnant woman is in ICU (intensive care unit) wouldn't be indicated. Rather, assessing blood glucose levels as one normally would during critical illness is required, keeping in mind that maternal hyperglycemia should be actively avoided.

GDM

According to the National Diabetes Data Group Classification, there are four sorts of diabetes [6]. they're type 1 Diabetes Melitus (DM), called insulin-dependent diabetes; type 2 DM, called insulin-resistant diabetes; diabetes dependent on other specific conditions like infection or drug induced; and gestational diabetes mellitus (GDM). GDM is defined as carbohydrate intolerance that's first recognized during pregnancy. There's a 50% risk of GDM turning to chronic DM within 5 yr after diagnosis if no lifestyle changes are made. Diabetes poses significant risks to maternal/fetal morbidity and mortality. Incidence of diabetes in pregnancy has increased because more women are delaying pregnancy until relatively late into their reproductive years. Currently the incidence is 4%-14% with GDM accounting for nearly 90%.

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At the first prenatal visit all women are screened for clinical risk factors when obtaining a history. If risk factors are identified like previous history of GDM, known impaired glucose metabolism, previous macrosomic baby (greater than 4000 g), and obesity (BMI greater than 30), early screening is usually recommended. An early screen that's negative is repeated for these high-risk women at 24-28 wk. Between 24 and 28 wk gestation, it's recommended that each one pregnant women be screened for GDM. A two-step screening process is currently supported by ACOG (American Congress of Obstetricians and Gynecologists). The approach begins with administration of 50 g of oral glucose solution with a 1-hr serum glucose measurement because the initial screening. Screening is suggested even for patients with low risk factors (age younger than 25 yr, not a member of an ethnic group at risk for developing type 2 DM, BMI but 25, no previous history of abnormal glucose tolerance, no previous history of adverse obstetric outcomes that are usually related to GDM, and no known diabetes during a first-degree relative [mother, father, siblings]).

For all women with type 1 DM, four to 5 daily insulin injections or an insulin pump are going to be required [7]. For women not on an insulin pump, insulin should lean as a part of a basal - bolus regimen with a long - acting basal insulin, usually given at night, and rapid or short - acting insulin boluses loving each meal. There's some evidence that the chance of hypoglycaemia for women with type 1 DM in early pregnancy is lessened with the use of an insulin pump and suitable patients should be considered for an insulin pump prior to pregnancy.

For women with type 2 DM, a basal - bolus regimen is additionally recommended because it offers the foremost flexibility around food timing and exercise and lessens the chance of hypoglycaemia in early pregnancy. For some women who have had type 2 DM for only a short time and are well controlled on diet and metformin alone, adequate glycaemic control could also be achievable prior to and early in pregnancy with either a twice - daily mixture (of a brief - and medium- acting insulin) or with just a rapid - acting insulin with meals. However, by the second trimester most women would require a basal - bolus regimen.

Continuing with the oral agent metformin for girls with type 2 DM before pregnancy is usually recommended by the United Kingdom clinical guidelines, although this is often not endorsed by other national guidelines. While there's no evidence that the sulphonylureas are teratogenic in early pregnancy, these agents are related to less good glycaemic control in pregnancy than insulin and less favourable pregnancy outcomes.

Gestational diabetes mellitus (GDM) is one in every of the foremost common obstetric complications, with incidence starting from 3 to 10% in developed countries [8]. There's currently an absence of consensus within the medical literature regarding screening for GDM because data fail to indicate that universal screening for GDM benefits the population.

Selective screening exempts women considered at low risk for GDM. ACOG states that although universal screening is that the most sensitive means of detection, certain low risk women may have the benefit of selective screening (SOR C). The American Diabetes Association (ADA) also endorses selective screening, generally performed at 24 to 28weeks' gestation with a 1-hour test (blood glucose measured 1 hour after oral ingestion of 50g glucose in 150mL fluid). The test is performed either fasting or non-fasting, although fasting may increase the probabilities of a false-positive screen. Women at higher risk for GDM may be screened at the initial prenatal visit, with follow-up testing done at 24 to 28weeks if the first test is normal.

If the 1-hour test is abnormal, a 3-hour glucose challenge test should be administered. The test is performed after an overnight fast. A 100-g glucose load is given orally, with blood drawn before ingestion and hourly for 3 consecutive samples. A diet containing a minimum of 150g of carbohydrates must be consumed for 3 days before testing. Carbohydrate depletion causes spuriously high glucose levels on the glucose challenge test. A diagnosis of GDM is created when elevation occurs with either the fasting glucose alone or with two or more of the 3- hour measurements.

Specific treatment including dietary advice and insulin for GDM reduces the danger of maternal preeclampsia and perinatal morbidity (composite outcome of death, shoulder dystocia, bone fracture, and nerve palsy). However, it's related to higher risk of labor induction. Intensive insulin therapy is related to lower rates of macrosomia and wish for cesarean, but increased risks for both maternal and neonatal hypoglycemia. Sulfonylureas (glipizide and glyburide) and metformin (Glucophage) are being used in women with preexisting type 2 diabetes, especially when polycystic ovarian disease is present.

PCOS

PCOS (polycystic ovary syndrome) could be a complex syndrome involving both a genetic and an environmental component with many possible presentations both physically and hormonally [9]. The core problem for all PCOS patients is increased intraovarian androgens. However, the pathway to the present increase is extremely complex and, in many cases, undefined. PCOS doesn't have one easily definable cause. People with PCOS have a genetic makeup that puts them at risk. The genetic background is complex, involving many areas of the genetic code. A number of the problem areas include heredity (so there is also a case history of PCOS, obesity, or adult-onset diabetes) but often the genetic abnormalities arise anew during the genetic formation of the person with PCOS. However, having the genetic background doesn't mean that an individual will automatically have PCOS. There has to make certain environmental factors that trigger the problem. There's no agreement on what environmental factors exist or how important are known factors. One environmental factor which may trigger PCOS is overweight/obesity and therefore

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the insulin resistance which frequently accompanies these conditions. PCOS is related to certain metabolic disorders which are a part of the metabolic syndrome. These include dyslipidemia and glucose intolerance. The prevalence of metabolic syndrome in adolescents has been estimated to be between 12% and 44%. amenorrhea as a presenting symptom in PCOS is unusual since the usual history is one among amenorrhea. However, both presentations have similarities like insulin resistance and increased intraovarian androgens. Obesity is present in 35–50% of adolescents with PCOS and in some patients seems to be a risk factor for PCOS with the link being insulin resistance. The increased androgen production increases the incidence of hirsutism and acne. Included within the diagnosis is hormonal testing to eliminate other causes of the clinical picture of PCOS like 17-0H progesterone for nonclassic adrenal hyperplasia and androgen profiles to eliminate the diagnosis of adrenal or ovarian tumors.

Although 50–70% of PCOS patients have insulin resistance [10], it's not one in all the diagnostic criteria of PCOS. the subject deservedly receives much attention, as many of the clinical signs and symptoms of PCOS is also attributed to excess insulin exposure. The precise molecular basis for insulin resistance is unknown, but it appears to be a postreceptor defect. There's tissue specificity of insulin resistance in PCOS: muscle and adipose tissue are resistant, while the ovaries, adrenals, liver, skin, and hair remain sensitive. The resistance to insulin in skeletal muscle and adipose tissue leads to a metabolic compromise of insulin function and glucose homeostasis, but there's preservation of the mitogenic and steroidogenic function in other tissues. The effect of hyperinsulinemia on the sensitive organs leads to downstream effects seen in PCOS, like hirsutism, acanthosis nigricans, obesity, stimulation of androgen synthesis, increase in bioavailable androgens via decreased sex hormone-binding globulin (SHBG), and, potentially, modulation of LH secretion.

Insulin resistance may be a component of the World Health Organization (WHO) definition of the metabolic syndrome, which may be a cluster of risk factors for cardiovascular disease. The WHO defines the metabolic syndrome because the presence of glucose intolerance or insulin resistance, with a minimum of two of the following: hypertension, dyslipidemia, obesity, and microalbuminuria. Women with PCOS are 4.4 times more likely to have the metabolic syndrome, so it becomes prudent to screen these patients, especially in those with insulin resistance.

Lipid abnormalities are more prevalent in PCOS patients. There may be a big increase in total cholesterol, ldl cholesterol, and triglycerides, and a decrease in hdl cholesterol compared to weight-matched controls. The dyslipidemia, impaired glucose intolerance, central obesity, hyperandrogenism, and hypertension seen in PCOS patients greatly increase the chance for cardiovascular disease. based on this risk profile, women with PCOS have a sevenfold increased risk of myocardial infarction.

Management

Prevention of hyperglycemia through rigorous control of blood sugar level is that the mainstay of treatment within the pregnant woman with pregestational diabetes [11]. This is often best accomplished by careful preconceptional counseling and achievement of normal HbA1c levels before pregnancy in pregestational diabetics, frequent (usually 4–5 times per day) home glucose level monitoring, adjustment of diet, and regular exercise.

Non–weight-bearing or low-impact exercise may be initiated or continued. Even short episodes of exercise will sensitize the patient's response to insulin for approximately 24 hours. All care providers should stress the importance of diet. Soluble fiber provides satiety and improves both the quantity of insulin receptors and their sensitivity. Carbohydrate restriction improves glycemic control and will enable a patient to realize her glycemic goals using diet and activity. Calories are prescribed at 25–35kcal/kg of actual weight, generally 1800–2400kcal/d. Diet should be approximately 40% carbohydrate, 40% fat, and a pair of 0% protein usually divided into 3 meals and 2 or 3 snacks per day. A bedtime snack is especially important to prevent nocturnal hypoglycemia. When postprandial values exceed the targets, it's important to review all recent food intake and to adjust food choice, preparation, and portion size.

Self-monitoring of fasting, 1- or 2-hour postprandial, and nighttime blood glucose levels using a glucose meter provides instant feedback to assess the patient's diet and behavior. When the glycemic goals are met, the feedback may be a powerful motivator. Diet and/or activity errors are identified and corrected as needed. Optimal glucose levels during pregnancy are fasting levels of 70–95mg/dL and 1-hour postprandial values <130–140mg/dL or 2-hour postprandial values <120mg/dL.

A minimum of two visits to a dietitian improves education and active participation regarding diet. Food records are useful. The dietitian reviews content and calories and suggests the way to include favorite ethnic foods to improve compliance. Other family members should be encouraged to participate within the dietary education because their understanding and support increase the prospect for a successful diet. Often, the other family members will benefit from the healthful diet changes. Additional follow-up visits between patient and dietitian are important when glycemic goals don't seem to be reached, weight change is simply too great or too small, or the patient has difficulty maintaining the diet.

Conclusion

Gestational diabetes is diabetes that is first diagnosed in pregnancy. It most often occurs in the second trimester of pregnancy, primarily due to insulin resistance, which is potentiated by hormones produced by the placenta. Insulin resistance is a disorder of glucose metabolism when there is a weakening of the peripheral effect of insulin whose main task is

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to facilitate the transfer of glucose from the blood to target tissues (liver, muscle, fat tissue). The consequence of the insufficient peripheral effect of insulin is manifested in enhanced pancreatic function and increased secretion of insulin from the beta cell with the aim of maintaining the balance of blood glucose levels. Gestational diabetes occurs during pregnancy in women whose pancreatic function is insufficient to overcome pregnancy-related insulin resistance. Among the main consequences are increased risks of preeclampsia, macrosomia and cesarean delivery and their associated morbidities.

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