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REVIEW ARTICLE

UNDERSTANDING A THEORETICAL ANALYSIS OF THE PATHOPHYSIOLOGY AND EFFECTS OF GESTATIONAL DIABETES MELLITUS ON MATERNAL AND FETAL HEALTH: NARRATIVE REVIEW

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ABSTRACT

Typically occurring in the second or third trimester of pregnancy, gestational diabetes mellitus (GDM) is a common metabolic illness. It is brought on by a combination of placental hormone-induced insulin resistance and the absence of pancreatic 8-cell compensation. Due to factors including placental villous immaturity and hypertrophic decidual vasculopathy, which can hinder placental activity and fetal growth, the condition poses a risk to the health of both the mother and the fetus.

The complex connections between genetic susceptibility, environmental factors, and hormonal changes during pregnancy define the pathophysiology of GDM. It has also been discovered that genetic variations, such as polymorphisms in the VEGF gene, may contribute to the development of GDM. Additionally, insulin resistance and 8-cell dysfunction in GDM may be caused by epigenetic changes.

GDM has long-lasting impacts on both the mother and the unborn child that extend beyond the pregnancy period. Type 2 diabetes mellitus is more likely to strike women in their adult years if they have a history of GDM. GDM exposure during pregnancy increases a child's risk of having a metabolic syndrome as well as metabolic diseases like obesity and insulin resistance.

A key factor in reducing these hazards is early GDM screening and therapy. The identification of multiple potential biomarkers, such as SPINA-GBeta, that can be utilized to diagnose GDM early and create a customized treatment plan has been made possible by the understanding of the molecular processes involved in the disease's pathophysiology.

INTRODUCTION

Gestational diabetes After hypertension, diabetes is the second most common pregnancy-related illness [1]. Similar to type 1 diabetes, pre-gestational diabetes mellitus (PGDM) is a condition in which a person has diabetes before becoming pregnant. The condition known as gestational diabetes mellitus (GDM) is characterized by elevated blood sugar levels that initially appear during pregnancy [2].

Hyperglycemia in Pregnancy (HIP) was defined by the World Health Organization (WHO) in 2014 as having diabetes during or prior to pregnancy. Additionally, it is separated into two subtypes: gestational diabetes mellitus (GDM) and diabetes in pregnancy (DIP) [3]. Patients with gestational diabetes mellitus (GDM) are more likely to experience preterm delivery, hypertension, shoulder dystocia, large-for-gestational-age birth, infant hypoglycemia, and respiratory distress[4]. The incidence of gestational diabetes

mellitus in pregnant women who are obese or have a high body weight may be avoided by adopting weight loss techniques and changing one's lifestyle during pregnancy [5].

First-trimester screening can identify pre-existing diabetes and early-stage GDM, enabling prompt implementation of glucose management strategies [6]. Because it has been shown to reduce the risk of problems, changing one's lifestyle is the first step in treating GDM[7]. The first-line drug prescribed to those whose lifestyle modifications are insufficient to maintain their health is insulin normal levels of blood sugar[8].

Epidomeology

Iraq is a prominent nation in the prevalence of type 2 diabetes [9].

The Arab countries are similar in terms of language, culture and religion but they are also very fferent in terms of governance structures, economic potential and social conditions including the level of violence. The World Health Organization (WHO) and the International Diabetes Federation (IDF) have extensive information about the Arabian Gulf countries. The World Health Organization (WHO), which has six locations worldwide, has a regional station in Cairo. The IDF states that there are 21 countries and territories that make up the Middle East, North Africa, and Horn of Africa (MENA) Region. Among them, the IDF is affiliated with 29 regional diabetes organizations [10,11].

Analysis of several studies revealed that the Middle East and North Africa (MENA) region has the highest prevalence of gestational diabetes mellitus. The prevalence of GDM in this area ranged from 8.4% to 24.5%, with an average estimate of 12.9%. Europe has the lowest prevalence of the illness, followed by North America and the Caribbean, South and Central America, Southeast Asia, the Western Pacific, and Africa. These regions' respective median incidence rates were 11.7%, 11.7%, 11.2%, 8.9%, and 7.0% [12].

GDM pregnant women were more likely to have caesarean sections, premature deliveries, and high blood pressure. Age progression, a family history of diabetes, the number of pregnancies, and an increasing body mass index (BMI) were risk variables associated with the development of gestational diabetes mellitus (GDM) [13].

According to one such study (2014), which involved 100 pregnant women, the prevalence of gestational diabetes mellitus (GDM) was 7%. Premature delivery, cesarean delivery, and hypertension throughout pregnancy were all more common in women with gestational diabetes mellitus (GDM). High body mass index (BMI), a family history of diabetes, advanced age, and an increased number of pregnancies were identified as risk factors for the development of gestational diabetes mellitus (GDM) (13). The prevalence of gestational diabetes mellitus (GDM) was recently investigated in Iraq [14].

among 120 women between the ages of 20 and 45. It was discovered that 13.3% of these women had GDM.None of the research have indicated the GDM crucial diagnosis criteria. Furthermore, hardly many pregnant women were enlisted. Furthermore, the political climate is making it more frequent for research to be undertaken insufficiently. The IDF's most recent version acknowledges the absence of HIP data in Iraq [15].

Etiology

It seems that a number of variables contribute to the etiology of gestational diabetes, including increased insulin resistance and malfunctioning or sluggish pancreatic beta cell response to glucose levels, which are caused by the placenta's hormone release. The main hormone associated with elevated insulin resistance in gestational diabetes mellitus (GDM) is human placental lactogen[16].

Growth hormone, prolactin, corticotropin-releasing hormone, and progesterone are other hormones linked to the development of this illness. Additionally, during pregnancy, the hormones lead to hyperglycemia and insulin resistance. While the precise cause of GDM is still unknown, a variety of potential contributing variables can be listed. The placenta secretes a variety of hormones to sustain the pregnancy in addition to providing the fetus with nutrition and water. Some hormones, including as estrogen, cortisol, and human placental lactogen, may inhibit the action of insulin. The contraintestinal effect is a well-known phenomenon that typically starts between weeks 20 and 24 of pregnancy [17]. The placenta's expansion causes a rise in the production of these hormones, raising the risk of insulin resistance. During pregnancy, the fetus need nourishment and placental hormones. However, some long-lasting inflammatory chemicals, such as tumor necrosis factor α, might inhibit insulin's action and result in the onset of insulin resistance (IR) [18].

Pathophysiology

The pathogenesis of gestational diabetes mellitus (GDM) includes β-cell malfunction and tissue insulin resistance.

GDM is typically the result of β-cell dysfunction in cases of prolonged insulin resistance during pregnancy. These deficiencies typically arise prior to conception and may get worse over time, increasing the risk of developing gestational diabetes. There are numerous more organs and systems that are both impacted by and involved in the development of GDM. The brain, skeletal muscle, liver, placenta, and adipose tissue are examples of the same [19].

Dysfunction of β-Cells

The primary function of β-cells is to generate the necessary amounts of insulin and store and release it in response to an increase in blood glucose levels. When β-cells are unable to produce enough insulin or react appropriately to an increase in glucose levels,

they malfunction. Chronic fuel excess is thought to lead to excessive and prolonged insulin production, which in turn induces β-cell malfunction [20]. However, the malfunction of β-cells may be caused by other intricate processes [21,22]. Problems can occur at any stage of the process, including during the pro-synthesis of insulin, post-translational changes, granule storage, blood glucose levels, or complex machinery that controls granule exocytosis [23] Insulin Resistance for a Prolonged Duration: When cells become less responsive to insulin, this is known as insulin resistance. This occurs as a result of a decrease in the migration of glucose transporter 4 (GLUT4) to the cell's plasma membrane, which transports glucose into the cell for energy production. Insulin-stimulated glucose absorption is 54% lower in gestational diabetes mellitus (GDM) than in a typical pregnancy [24.] Although the concentration of the insulin receptor itself is usually normal, GDM is characterized by impaired insulin signaling due to either an increase in serine/threonine phosphorylation or a decrease in tyrosine phosphorylation of the insulin receptor [25]. Furthermore, changes in the expression and phosphorylation of downstream regulators of insulin signaling, such as GLUT4, PI3K, and insulin receptor substrate (IRS)-1, are also associated with GDM [26].

Many of these molecular changes persist after conception[27].
Placental Transport: Insulin resistance is caused by hormones and cytokines released by the placenta during pregnancy. Additionally, the placenta, which separates the mother and the child, is vulnerable to high blood sugar levels and the consequences that follow in cases of gestational diabetes mellitus (GDM).
The placenta's absorption of glucose, amino acids, and lipids may likely be impacted by this. Since glucose is the primary energy source, the fetus and placenta both need it. Therefore, glucose should always be readily available. Insulin is not necessary for the passage of glucose across the placenta. GLUT1 uses carriers to promote glucose transport that is sodium-independent [28].

The placenta continues to express the insulin receptor, and insulin signaling can affect the metabolism of glucose in the placenta. The placenta is readily exposed to glucose and is extremely vulnerable to maternal hyperglycemia. This establishes a clear link between macrosomia and better fetal development [29].

There is no text provided. The movement of proteins and amino acids across the placenta is another important factor that affects the fetus's growth. High levels of systems A and L are associated with gestational diabetes mellitus (GDM). Pro-inflammatory cytokines such as TNF-α and IL-6 may also have an impact on these variables. GDM may result from changes in the transport of amino acids brought on by an excess of proteins [30]. There is no text provided.Lastly, lipids: Although hyperglycemia is usually associated with diabetes mellitus (DM), the topic of hyperlipidemia in relation to this disease has been brought up by the rising incidence of gestational diabetes mellitus (GDM) in relation to obesity. On the other hand, glucose pathways only account for 9% of the alterations in placenta gene expression during gestational diabetes mellitus (GDM), while lipid pathways account for 67%. Unlike T1DM, placental lipid gene activation is only associated with GDM [31].

Gestational Diabetes Screening

There is no text provided.Lastly, lipids: Although hyperglycemia is usually associated with diabetes mellitus (DM), the topic of hyperlipidemia in relation to this disease has been brought up by the rising incidence of gestational diabetes mellitus (GDM) in relation to obesity. On the other hand, glucose pathways only account for 9% of the alterations in placenta gene expression during gestational diabetes mellitus (GDM), while lipid pathways account for 67%. Unlike T1DM, placental lipid gene activation is only associated with GDM [32].

Furthermore, women with a history of GDM are recommended to have a prediabetes or diabetes test performed at least every three years with this group. using an oral glucose challenge test between weeks 24 and 28 of pregnancy at a weight of 50 grams in one hour. If the findings are abnormal, more than or equivalent to 130 mg/dL (7.22 mmol/L) or 140 mg/dL (7.77 mmol/L), a follow-up test utilizing a 100-g, 3-hour oral glucose tolerance test is necessary. The values for the test should be: values of 180 mg/dL in the first hour. In the second hour, more than 155 mg/dL. more than 140 mg/dL during the third hour. Gestational diabetes is diagnosed when there are two or more abnormal outcomes [33].

In order to identify pregestational diabetes or early gestational diabetes mellitus, the American Diabetes Association suggests that a screening can be established by all overweight or obese women with one or more of the risk factors listed below: Insufficient physical activity, a first-degree family

afflicted with diabetes, and a high-risk racial or ethnic group.

Previously given birth to a child weighing at least 4,000 grams.

Previously, gestational diabetes and hypertension. < HDL levels < 35 mg/dL. Triglyceride level greater than 250 mg/dL. Women afflicted with polycystic ovarian syndrome. An elevated A1c hemoglobin level exceeding 5.7%. Impaired glucose tolerance test. Reduced blood sugar levels when fasting. Cardiovascular illness history in the past Other diseases that relate to insulin resistance The American Diabetes Association claims that the hemoglobin A1C test is useful, yet due to its reduced sensitivity, it may not be appropriate to use it alone [34].

The American College of Obstetricians and Gynecologists (ACOG) advises against blood glucose levels exceeding 95 mg/dL during fasting, 130–140 mg/dL within an hour of meals, and 120 mg/dL within two hours after meals during pregnancy. Monitoring of blood sugar levels during the postpartum period is suggested and this is 24 to 72 hours after birth. Removal of placenta typically reduces insulin resistance, and this can be used to reduce the insulin or hypoglycemic drug requirements. The goal of glucose treatment is to produce a high blood glucose level to a euglycemic range. In order to exclude the onset of type 2 diabetes, an oral glucose tolerance test, 75g, should be undertaken 412 weeks after child birth [35].

Treatment Management

Medical nutrition therapy or MNT is a new emerging field that is used to cure and prevent GDM. No predetermined rules can exist since dietary recommendations should be customized. The most suitable individual to initiate and manage MNT is a seasoned GDM manager or any other qualified individual. The other nonpharmacologic intervention is to use a daily 30-minute physical activity to women who either have no medical or obstetric contraindications associated with it [36].

The American Dietetic Association holds that a licensed dietitian is expected to provide nutritional counseling and come up with a tailored plan based on the BMI of the patient. Doctors can employ the following three fundamental nutritional notions to offer guidance in situations in which dietitians have failed:

The amount of exercise advised in the case of people with GDM is caloric allocation or the distribution of calories and intake of carbs. 30 minutes of moderate-intensity aerobic exercise a minimum of five days per week, or at least 150 minutes per week [37]. Insulin is the primary intervention in the ADA guidelines on the prevention of GDM. The insulin therapy has been seen as the usual therapy in gestational diabetes when diet and exercise fail to attain normal levels of glucose [38]. During fasting, insulin is added to the management if blood glucose levels are higher than or equal to 95 mg/dL, 140 mg/dL during one hour, or 120 mg/dL during two hours. This is useful in establishing an optimal metabolic control. Two oral hypoglycemic drugs include metformin and gliburide which are increasingly being used by women with gestational diabetes although they have not been approved by the FDA. Glibride is available at 2.5 mg starting dose and 20m at maximum. In the metformin therapy, the starting dose of 500mg and maximum dose 2500mg are recommended. Basal insulin dose may be calculated on the basis of weight of patient and adding 0.2 units per kilogram per day. When blood glucose level increases following a meal, one can take regular insulin or rapid acting insulin earlier with the starting amount of two to four units [39]. In the first trimester, the total daily insulin requirement is 0.7 units/kg, in the second, 0.8 units/kg, and in the third, 0.9 to 1.0 units/kg. The patient should divide the daily insulin dosage by half and take half of it as regular insulin or rapid-acting insulin before all the three meals and the remaining half as fundamental insulin before going to bed. Aspart and Lispro are acceptable to use in pregnancy,Short-acting insulin is associated with reduction in hypoglycemia and With long-lasting insulin, nighttime hypoglycemia is reduced [40].

Complications

There are two types of difficulties associated with developing gestational diabetes: Among the fetal problems are: Macrosomia and hypoglycemia in neonates polycythemia dystocia of the shoulders elevated bilirubinemia Increased prenatal mortality was linked to neonatal respiratory distress syndrome. low calcium levels[41].

Among the maternal problems are: High blood pressure, preeclampsia, A higher chance of getting diabetes mellitus and giving birth by cesarean section[42].

CONCLUSION

Gestational Diabetes Mellitus (GDM) is a serious health issue that has complicated pathophysiological

processes that affect the health of both the mother and the unborn child. In this review of the literature, we have been able to examine the underlying pathogenesis of insulin resistance during pregnancy, which includes changes in hormones and genetic predispositions. The consequences of GDM are far-reaching and translate into the emergence of the high risks of maternal complications including preeclampsia, section and the emergence of type 2 diabetes after childbirth. Moreover, fetal health is put under threat as well, possible outcomes include macrosomia, neonatal hypoglycemia and long-term metabolic issues. The risks can be reduced by early diagnosis, good management, and thorough knowledge of GDM. Lifestyle modification, timely monitoring and suitable medical management interventions should be of great help to the mother and child. The future studies must focus on better explanation of the long-term outcomes of GDM and development of new measures related to prevention and treatment of GDM, which will guarantee the improved health results to both mothers and fetuses.

DECLARATION

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Conflict of Interest

None to declare.

Patients consent

All the patients in this study have given their informed consent for publication.

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