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**GESTATIONAL DIABETES AND RISK OF CONGENITAL MALFORMATIONS:
EXPLORING THE ASSOCIATION AND CLINICAL IMPLICATIONS**

VASANTHA G*, PUSHPA A, ANJANA P, BHARGAVI R AND RATNASRI SAI P

Vignan Institute of Pharmaceutical Technology (Autonomous), Duvvada, Visakhapatnam
530049, Andhra Pradesh, India

***Corresponding Author: Dr. Galanki Vasantha: E Mail: drvasanthaniper@gmail.com**

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ABSTRACT

GDM is the most common pregnancy-related condition. Most of the time, pancreatic beta cell failure resulting from chronic insulin resistance impairs glucose tolerance, which leads to hyperglycemia. Obesity or being overweight, having an elderly mother, and having a family history of diabetes of any type are risk factors for GDM.

GDM dramatically raises the fetus's risk of congenital defects, including cardiac defects, renal abnormalities, and neurological issues. These anomalies can have long-lasting health consequences for the child. Commonly known as birth defects, or congenital abnormalities, congenital anomalies are conditions of prenatal origin that exist at birth and may affect the health, development, and/or life of a newborn. Before the seventh week of pregnancy, congenital abnormalities linked to diabetes develop.

A comprehensive strategy is needed to provide the best care possible for a person with GDM. This includes blood sugar control, dietary changes, nutritional monitoring, and weight gain management throughout pregnancy. Diet and more exercise are the main treatments for GDM. Treatment can be accelerated with the use of insulin, gliburide, and metformin. Standard diagnosis criteria, suitable education, and mother counseling are required to treat the problem. We intend to look at how GDM affects the fetus in light of the changing prognosis, prevalence, and prevention of the condition.

Keywords: Gestational diabetes mellitus, Foetal anomalies, Pre-conceptional blood sugar blood glucose monitoring, exercise, insulin therapy, pregnancy, obesity, macrosomia, hyperglycemia

INTRODUCTION

When glucose intolerance of varied severity begins or is initially identified during pregnancy, it is known as gestational diabetes mellitus (GDM). A higher risk of unfavourable obstetrical outcomes, such as macrosomia, shoulder dystocia, birth damage, preterm, perinatal mortality, and the necessity for a Caesarean section, is associated with this medical pregnancy problem [1]. Although Carrington originally used the phrase "gestational diabetes" in 1957, it gained more recognition in 1961 and 1964 as a result of John O'Sullivan's writings [2]. The earliest signs of GDM, a form of diabetes, appear in the second trimester [3] or the third trimester of pregnancy [4].

RISK FACTORS

Pregnancy-related hyperglycemia is more common as women age. GDM was found in 8.5% of women over 30 but 6.7% of pregnancies overall, according to Mosses *et al* [5]. According to Lao *et al.*, pregnant women between the ages of 35 and 39 had the highest risk of getting GDM when compared to those who were younger (OR 95% CI: 10.85 (7.72–15.25) vs. 2.59 (1.84–3.67) [6].

Pluriparity and the incidence of GDM continue to have a linear connection, even after accounting for the woman's age [7]. The chance of recurrence is more than six times

higher if GDM was present during a prior pregnancy [8]. When a woman's BMI is at least 30 kg/m², the prevalence of GDM is 12.3%. [9, 10]. The chance of GDM is approximately 3 times greater (95% CI 2.1-3.4) for class I obese women (BMI 30-34.99 kg/m²) and four times higher (95% CI 3.1-5.2) for class II obese women (BMI 35-39.99 kg/m²) than for women with a BMI < 30 kg/m² [21]. 11.6% of women with a history of GDM in their first-line ancestors. Up to 61% of instances of GDM are at increased risk when these two factors are combined [9, 10]. GDM pregnancies were more than twice as common in women who had previously had treatment for polycystic ovarian syndrome (PCOS) [12].

With recurrence rates as high as 84%, the largest risk factor for GDM is a history of the condition during a prior pregnancy [13]. Multiparity is one of the additional risk factors for GDM [14], twin pregnancy [15], previous macrosomia [16], Perinatal difficulties in the past [14], maternal small-for-gestational-age (SGA) or LGA [14], physical inactivity [17], low-fiber high-glycemic load diets [18], reduced intake of carbohydrates and increased dietary fat [19], and medications such as glucocorticoids and anti-psychotic agents [20]. GDM risk is also linked to maternal

hypertension in the prenatal and early stages of pregnancy [21].

DIAGNOSIS OF GDM

An OGTT is advised for expectant mothers between weeks 24 and 28 of pregnancy by the American Diabetes Association [22]. It is recommended by the International Association of Diabetes and Pregnancy Study Groups (IADPSG) that routine Diagnostic and screening for GDM be carried out between weeks 24 and 28 of pregnancy [23].

The American Diabetes Association (ADA), the American College of Obstetricians and Gynaecologists (ACOG), the Diabetes Canada Clinical Practice Guidelines (DCCPG), India's Diabetes in Pregnancy Society Group (DIPSI), The European Association of the Study of Diabetes (EASD), The Australian Diabetes in Pregnancy Society (ADIPS), the International Federation of

Gynaecology and Obstetrics, World Health Organisation (WHO), the National Institute for Health and Care Excellence (NICE) and the International Association of the Diabetes and Pregnancy Study Groups (IADPSG).¹

Using a 1-hour post-load measurement, there are no set standards for diagnosing diabetes mellitus in pregnancy. The level of total blood glucose is denoted by^{2, 3} Suggests the IADPSG one-step or two-step method; serum or plasma glucose concentration is measured one hour following an oral glucose load of 50 g (GCT) for first screening. Those who exceed the threshold perform either a 100 g OGTT.⁴ The alternative strategy, the IADPSG which uses an OGTT of 75 g without fasting is listed in the preferred technique.⁵ Makes use of a 75 g non-fasting OGTT

The criteria for diagnosing GDM according to several scientific bodies are shown in **Table 1**.

Table 1: The criteria for diagnosing GDM according to several scientific bodies

Criteria	Fasting mg/dL (mmol/L)	1h mg/dL (mmol/L)	2h mg/dL (mmol/L)	3h mg/dL (mmol/L)	Number of Values for Diagnosis
ADA/ACOG ³ 2003,2018	95(5.3)	180 ¹ (10.0 ¹)	155(8.6)	140(7.8)	2
ADIPS 2014	92(5.1)	180(10.0)	153(8.5)	-(-)	1
DCCPG 2018 ⁴	95(5.3)	-(10.6)	-(9.0)	-(-)	1
DIPSI 2014 ⁵	-(-)	-(-)	140(7.8)	-(-)	1
EASD 1991	110 ¹ /126 (6.1 ¹ /7.0)	-(-)	162 ¹ /180 (9.0 ¹ /10.0)	-(-)	1
FIGO 2015	92(5.1)	180(10.0)	153(8.5)	-(-)	1
WHO 1999	110 ² /126 (6.1 ² /7.0)	-(-)	120 ² /140 (6.7 ² /7.8)	-(-)	1
WHO 2013	92(5.1)	180(10.0)	153(8.5)	-(-)	1
IADPSG/WHO	92(5.1)	180(10.0)	153(8.5)	-(-)	1
NICE	-(5.6)	-(-)	-(7.8)	-(-)	

PATHOGENESIS OF GDM

In addition to genetic, environmental, and dietary factors, the two most significant factors that may contribute to the development of GDM are insulin resistance and beta cell dysfunction.

INSULIN RESISTANCE:

As in type 2 diabetes, insulin resistance and decreased insulin production relative to patient needs play a significant role in the pathogenesis of GDM. We see GDM in women who are lean and fat [24]. Obese women already have pre-pregnancy insulin resistance, which overlaps with pregnancy-induced insulin resistance. The initial stage of insulin synthesis is also impaired in women who are slim [25]. Insulin resistance is a result of pregnancy-related overproduction of insulin-antagonistic hormones (cortisol, placental hormones), which by the end of the third trimester is close to type 2 diabetes [26]. Moreover, the onset of gestational diabetes is predicted by hyperleptinemia during the first few weeks of pregnancy [27]. Meanwhile, hyperleptinemia is caused by increased leptin levels, which are a hallmark of GDM [28]. Therefore, pre-pregnancy BMI is a better indication of leptinemia than GDM itself [29]. Compared to pregnant women without problems in their glucose metabolism, pregnant women with gestational diabetes had

lower levels of adiponectin, regardless of their pre-pregnancy BMI. [30] It has been shown that low adiponectin levels in the first and second trimesters of pregnancy are indicative of the onset of diabetes during pregnancy [31].

B-CELL DYSFUNCTION

There are conflicting findings when analyzing insulin secretion abnormalities in GDM. The mechanism of beta-cell hypertrophy and proliferation, which causes a 300% increase in insulin release during the first two trimesters of physiological pregnancy, is insufficient to explain GDM [32]. The etiology of GDM has been shown to involve autoimmune and genetic factors, such as anti-insulin and/or anti-insulin antibodies, which raise the risk of developing DM1 and later autoimmune diabetes in adults (LADA) [33]. In both DM2 and GDM, they are associated with a decreased initial phase of insulin production, which leads to a decrease in beta cell mass and dysfunction and, eventually, GDM [34].

TREATMENT

For the optimum care of a person with GDM, a comprehensive approach is required. This includes blood sugar control, nutritional monitoring, dietary changes, and weight gain management throughout pregnancy. Adequate exercise, dietary changes, and lifestyle enhancements can cure up to 70% to 85% of individuals with gestational diabetes.[35]. In

15% to 30% of cases, taking medication is required. Among these are oral hypoglycemics and insulin.

BLOOD GLUCOSE MONITORING

Daily self-glucose monitoring at home is recommended by most organisations. Postprandial and fasting blood glucose levels should currently be self-monitored every day. The American Diabetes Association (ADA) states that the ideal blood sugar levels are 140 mg/dL or 120 mg/dL one to two hours after a meal, and 95 mg/dL during a fast. Pre-prandial glucose monitoring is most beneficial for people who already have diabetes [36].

DIETARY MODIFICATIONS

Low-glycemic index diets, low-carbohydrate diets, low-unsaturated fat diets, high-fiber diets, soy-based diets, calorie-restricted diets, and the DASH diet (dietary methods to treat hypertension) are among the dietary solutions that are covered in the literature. Nutritional recommendations should focus on a balanced diet that contains 20% protein, complex carbohydrates, healthy fats, and appropriate portion sizes [37].

PHYSICAL EXERCISE

Regular exercise and physical activity have been advised and promoted even for pregnant women with GDM. Reduced risk of gestational diabetes, larger-than-normal babies, high blood pressure issues, preterm

birth, and foetal growth limitation are all advantages of moderate activity during pregnancy [38]. Additionally, pregnancy-related lifestyle modifications affect the postpartum period and reduce the incidence of postpartum depression [39].

PHARMACOTHERAPY FOR GDM MANAGEMENT:

15% to 30% of GDM patients have insufficient blood glucose control, necessitating the use of medication, despite the recommendations for dietary and lifestyle changes [36]. Usually, if hyperglycemia continues throughout the day after 10–14 days of dietary and lifestyle changes, pharmaceutical courses should be taken into consideration. To manage hyperglycemia, patients with gestational diabetes mellitus are given insulin and oral medicines [40]. During pregnancy, insulin offers the most stable outline. Sulfonylureas like glyburide and metformin are among the oral drugs that have been studied. Insulin and other large molecules cannot cross the placenta. It has been shown that glyburide and metformin can cross the placental barrier and get to the foetus [41].

PHARMACOLOGICAL TREATMENT:

Patients should receive pharmacological treatment if they are unable to meet glycaemic objectives with a well-balanced diet and the

correction of dietary mistakes [42]. Insulin therapy is the safest treatment option, according to the majority of research, and OAD (orally administered medications) should only be used when the patient refuses insulin therapy or it is not available [43]. Subcutaneous injections are used to administer insulin therapy in the functional intensive insulin therapy (FIIT) model [44]. It has been established that using human insulin during pregnancy is safe [45].

Glibenclamide and metformin are now used orally. Despite crossing the placenta, metformin and glibenclamide (glyburide) are not thought to be teratogenic [46]. Metformin by itself or in combination with extra insulin was found to be unrelated to an increase in perinatal problems [47]. Several follow-up studies based on this research assessed the safety and efficacy of metformin use in GDM [48].

CONGENITAL ANOMALIES:

Table 2: Congenital Anomalies

CATEGORY	ANOMALIES
CARDIAC ANOMALIES	Infants of women with pregestational diabetes mellitus are two to five times more likely to develop congenital heart disease, with atrioventricular septal anomalies being the most common type, according to a thorough assessment by Simeone <i>et al.</i> [49]
RENAL ANOMALIES	Congenital kidney and urinary tract abnormalities (CAKUT) are responsible for 50% of juvenile instances of chronic kidney disease and up to 20% of all birth defects identified during pregnancy. CAKUT are frequently separated into three groups based on their embryologic origin: abnormalities of outflow, anomalies of renal embryonic migration, and renal parenchymal malformations [50]
GROWTH DISORDER	The growth condition known as macrosomia is brought on by excessive intrauterine growth. There is evidence that neonates born large for gestational age (LGA) are more likely than other neonates to develop obesity in their early adult years, puberty, and childhood [51]
LUNG ANOMALIES	Babies delivered to women with gestational diabetes have been repeatedly shown to have delayed lung maturity and an elevated risk of respiratory distress syndrome [52]
NEUROLOGICAL ANOMALIES	Congenital abnormalities: Spina bifida, encephalocele, and anencephaly are neural tube malformations [53] Cognitive impairment: Shortfalls in linguistic, memory, and intellectual skills [54]. Motor disorders: Inadequate fine and gross motor control and delayed motor development [55] Attention-deficit hyperactivity disorder, autism spectrum disorder, and schizophrenia are examples of psychosocial disorders. [56]

CONCLUSION

The most prevalent metabolic disorder during pregnancy, gestational diabetes mellitus, continues to have a major influence on global health. Because of its substantial influence on the health of expectant mothers and their unborn children, this condition, which is characterized by high blood sugar levels

during pregnancy, requires our attention and a deeper knowledge. Additionally, the impact of GDM on children is a serious worry. This analysis highlights the increased risk of obesity, hypertension, and insulin resistance in children of women with GDM, diseases that can last into adulthood. For proactive prevention and control, it is essential to

comprehend these intergenerational health implications.

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