

Gestational Diabetes Mellitus: collaboration Between Nursing, Pharmacists, and Clinical Pathology in Diagnosis, Management, and Treatment.

Shaima Abdullah Aldossery¹
Sultsnanah Eid Matar Almulaire²
Ameerah Mahareb Salmanalhurayji³
Khaznah. Merweh. Alteel. Aldhafeeri⁴
Sultan Sayed Mogaees Almutairi⁵
Fouad Ibrahim Ali Shaiban⁶
Sultsnanah Eid Matar Almulaire⁷
Fatimah Hussain Majrashi⁸
Sultsnanah Eid Matar Almulaire⁹
Mohammed Safar Bin Hamdan Alotaibi¹⁰
Rami Mohammed Shoei Hamdi¹¹
Sami Hussain Tawhari¹²
Salihah Abdullah Saeed Alghamdi¹³
Khuluod Ali Mohammed Rezgallah¹⁴
Arif Thaar Alotaibi¹⁵

1. Ksa, Ministry Of Health, The First Health Cluster Public Health Administration Infection Control Department
2. Ksa, Ministry Of Health, JanoobAlkhalidyahHafarAlbatin
3. Ksa, Ministry Of Health, JanoobAlkhalidyahHafarAlbatin
4. Ksa, Ministry Of Health, Mental Health Hospital Hafr Al, Batin
5. Ksa, Ministry Of Health, Al-Busaira Health Center
6. Ksa, Ministry Of Health, South Abu Arish Primary Health Care Center
7. Ksa, Ministry Of Health, JanoobAlkhalidyahHafarAlbatin
8. Ksa, Ministry Of Health, AiradaHeaith Psychological Ln Jazan
9. Ksa, Ministry Of Health, JanoobAlkhalidyah. HafarAlbatin
10. Ksa, Ministry Of Health, East Dawadmi Health Center
11. Ksa, Ministry Of Health, Al Birk General Hospital
12. Ksa, Ministry Of Health, Al-Birk General Hospital
13. Ksa, Ministry Of Health, Imam Abdulrahman Al Faisal Hospital
14. Ksa, Ministry Of Health, Imam Abdulrahman Al Faisal Hospital
15. Ksa, Ministry Of Health, Primary Health Center Wedakh

Abstract:

Background: Gestational diabetes mellitus (GDM) is a metabolic disorder that typically arises in the second and third trimesters of pregnancy, characterized by glucose intolerance. It is increasingly prevalent due to factors such as obesity, sedentary lifestyles, and advanced maternal age. GDM can lead to severe complications such as preeclampsia, macrosomia, and neonatal hyperbilirubinemia. Furthermore, women with GDM are at higher risk of developing type 2 diabetes later in life. Early detection and proper management are critical in preventing both short-term and long-term maternal and fetal complications. However, existing diagnostic criteria and treatment options remain subjects of debate.

Aim: This article explores the collaboration between nursing, pharmacists, and clinical pathology in the diagnosis, management, and treatment of GDM. It aims to highlight how an interdisciplinary approach can improve patient outcomes and identify the need for optimized diagnostic and treatment protocols.

Methods: The article reviews the current literature on GDM, focusing on epidemiology, risk factors, pathogenesis, diagnosis, and treatment. The collaborative roles of nursing, pharmacy, and pathology professionals are discussed, emphasizing how each discipline contributes to GDM care.

Results: Key findings suggest that an integrated care approach improves early detection, better management of blood glucose levels, and a reduction in complications. The role of biomarkers in predicting and diagnosing GDM is highlighted, with specific markers such as leptin and adiponectin showing promise. Additionally, the article discusses various diagnostic methods and the importance of personalized care.

Conclusion: Collaboration among nursing, pharmacists, and clinical pathology is essential for the effective management of GDM. This teamwork ensures timely diagnosis, appropriate treatment, and continuous monitoring, improving both maternal and fetal health outcomes. Further research into biomarkers and new diagnostic criteria is needed to enhance early detection and treatment strategies.

Keywords: Gestational diabetes, nursing, pharmacology, clinical pathology, biomarkers, management, diagnosis, treatment.

Introduction:

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance that arises during the second and third trimesters of pregnancy, leading to hyperglycemia of varying severity [1]. The global prevalence of GDM is experiencing a significant rise, largely due to the increasing incidence of obesity, sedentary lifestyles, and advanced maternal age [2]. GDM affects between 2% and 38% of pregnancies [3], with rates being higher in racial and ethnic minorities compared to non-Hispanic white populations [4]. The variation in prevalence can also be attributed to differences in diagnostic criteria, with the International Association of Diabetes in Pregnancy Study Group (IADPSG) guidelines indicating a prevalence rate 2.4 times higher than the World Health Organization (WHO) guidelines from 1999 [5]. Moreover, studies have shown that the prevalence of GDM fluctuates with the seasons, with higher rates observed during the summer and lower rates in the winter months [6].

Untreated GDM can lead to both short-term and long-term complications for both the mother and the fetus. These include hypertension, preeclampsia, cesarean delivery, birth trauma, macrosomia, and neonatal hyperbilirubinemia [7, 8]. Children born to mothers with GDM are at a heightened risk for metabolic complications later in life [9]. Additionally, women with GDM are seven times more likely to develop type 2 diabetes mellitus (T2DM) than those with normal glucose tolerance [10], suggesting that the prevalence of GDM could serve as a predictor for the broader population's rate of T2DM [11]. The progression of T2DM is influenced by several factors, such as the severity of glucose abnormalities, the gestational age at GDM diagnosis, and whether insulin therapy was utilized during pregnancy [12]. However, a small proportion of women with GDM who have pancreatic beta-cell autoantibodies may develop type 1 diabetes mellitus postpartum [13].

Despite GDM being the most common metabolic disorder during pregnancy, there remains substantial debate regarding the diagnostic approaches, treatment options, and postpartum monitoring strategies [14, 15]. Early detection and treatment of GDM have been shown to improve pregnancy outcomes, emphasizing the need for optimized detection and treatment protocols [16, 17]. Unfortunately, current diagnostic criteria based on glucose homeostasis are not sufficient to predict or detect all cases of GDM [9]. Therefore, biomarkers for GDM must be specific, easily detectable in circulation, non-invasive, and unaffected by normal physiological and metabolic changes [9]. Consequently, the identification of novel biomarkers for early GDM detection could significantly enhance clinicians' ability to manage these patients and reduce the negative pregnancy outcomes associated with the condition.

Etiology and Pathogenesis of GDM

While the exact etiology of GDM remains unclear, several proposed mechanisms and risk factors help explain the progression of the disease.

Risk Factors for GDM

Epidemiological studies have identified several risk factors for GDM, although findings have been inconsistent [12, 18, 19, 20]. Obesity and a family history of diabetes are recognized as key risk factors. The risk of developing GDM is 2.14 times higher in obese women, 3.56 times higher in overweight women, and 8.56 times higher in women who are extremely obese compared to those with normal body weight [20]. Advanced maternal age is another well-established risk factor for GDM, although there is no consensus on a precise cut-off age [12]. The American Diabetes Association recommends considering age 25 as a risk factor for GDM [21]. GDM was found to be uncommon in pregnant women under the age of 20, but prevalent in 33.3% of women aged 20–29 and 58.3% of women aged 30–39 [19]. Interestingly, depression has recently been identified as a risk factor for GDM, with a 1.54-fold increased likelihood of developing the condition [22].

Healthy Pregnancy vs. GDM Pregnancy

During a normal pregnancy, significant metabolic changes occur, including reduced insulin sensitivity and elevated fatty acids and glucose levels, which are necessary to meet the nutritional demands of the developing fetus [2]. As gestation progresses, there is a gradual increase in maternal and placental hormones such as progesterone, estrogen, prolactin, placental growth hormone, and human placental lactogen (hPL), leading to a state of insulin resistance [2]. Progesterone contributes to insulin resistance by inhibiting the PI3-kinase pathway and suppressing insulin receptor substrate 1 (IRS1) expression. Estradiol also induces insulin resistance through the membrane estrogen receptor (ER)-mediated activation of JNK and subsequent serine phosphorylation of IRS-1 [23]. Prolactin

mainly regulates β -cell function through the JAK-2/signal transducer and activator of transcription (STAT)-5 pathway [24]. Human placental growth hormone (hPGH) contributes to insulin resistance by enhancing the expression of the p85-regulatory unit of PI3K, thus reducing IRS-1-associated PI3K activity. Additionally, hPL induces insulin resistance by decreasing IRS-1 phosphorylation [25]. It has been reported that insulin responses to an intravenous glucose tolerance test are about threefold higher in late pregnancy compared to pre-pregnancy levels [26]. These changes in insulin sensitivity generally resolve within days of delivery, indicating that they are primarily mediated by placental hormones [27].

GDM typically develops between the 24th and 28th weeks of pregnancy and is associated with the failure of pancreatic β -cells to produce sufficient insulin, leading to varying degrees of hyperglycemia [12]. When maternal insulin production is inadequate to overcome insulin resistance, glucose intolerance arises, thereby increasing the likelihood of developing GDM [28]. Insulin resistance begins to develop as early as the second trimester and peaks by the third trimester, with an estimated 56% increase in insulin resistance in patients, primarily due to impaired insulin signaling in skeletal muscle and adipose tissue [29]. Additionally, glucose consumption has been shown to decrease by 40–60%, depending on body mass index (BMI) [30]. Along with β -cell dysfunction, alterations in the insulin pathway also occur. Under normal conditions, insulin binds to the insulin receptor on peripheral tissues, such as skeletal muscle, stimulating glucose uptake by cells. This interaction results in the autophosphorylation of the insulin receptor's tyrosine kinase domain, which facilitates the movement of glucose transporter type 4 (GLUT4) to the cell membrane, enabling glucose absorption. In pregnant women, the level of IRS1, a key molecule in insulin signaling, is reduced compared to non-pregnant women [31]. Furthermore, a significant reduction in glucose uptake is observed in the skeletal muscles of women with GDM compared to those with normal glucose tolerance, due to impaired autophosphorylation of the insulin receptor. In addition to environmental factors such as obesity, recent research suggests that genetic and epigenetic factors play an important role in the pathogenesis of GDM. Evidence indicates that genetics contribute to the recurrence of GDM in at least 30% of previously diagnosed women, genetic variations in insulin production and resistance, and ethnic heterogeneity, all of which are linked to an increased risk of GDM [32].

Diagnosis of GDM

The early identification and accurate prediction of gestational diabetes mellitus (GDM) are pivotal in mitigating pregnancy-related complications for both the mother and the fetus. However, there remains a lack of consensus regarding critical aspects such as the optimal timing for screening, the appropriate diagnostic tests, and the precise glycemic thresholds for diagnosing GDM [33]. Until 2010, the World Health Organization (WHO) and the American Diabetes Association (ADA) represented the primary authorities in the diagnosis of GDM. The ADA guidelines recommended a 100-gram oral glucose tolerance test (OGTT) between 14 and 18 weeks of gestation for women at high risk, and between 28 and 32 weeks for those at medium risk [1]. Presently, the diagnostic criteria proposed by the International Association of Diabetes and Pregnancy Study Groups (IADPSG) are widely adopted. These criteria were developed based on the outcomes of the Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) Study, an extensive international study involving 23,000 pregnant women [34]. The HAPO Study demonstrated that maternal glycemia, even below the threshold for GDM diagnosis, continued to pose risks for adverse maternal and fetal outcomes, necessitating a revision of the intervention criteria. Consequently, the IADPSG guidelines now recommend that all pregnant women undergo a fasting plasma glucose (FPG) test, glycosylated hemoglobin (HbA1c) measurement, or random plasma glucose evaluation at their first prenatal visit, with a threshold of ≥ 5.1 mmol/L indicating GDM [34]. These thresholds are lower than those proposed by previous guidelines. Today, the IADPSG criteria are the most frequently recommended, though some countries continue to utilize alternative standards. A detailed summary of the primary screening and diagnostic guidelines for GDM. In addition to the existing diagnostic protocols, the identification of specific biomarkers at various stages of pregnancy may enhance the screening process for GDM.

Prospective Diagnostic and Predictive Markers for GDM

During the course of GDM, adipose and placental tissues may release specific factors that contribute to inflammation and insulin resistance [9]. These biomarkers, which can be detected in maternal blood or urine, hold promise for predicting and diagnosing GDM. Furthermore, the combination of biochemical and molecular biomarkers in predictive models may facilitate the early detection of GDM and help mitigate the complications associated with the condition.

Biochemical Markers

Adipose-Related Markers

Adipose tissue produces hormone-like substances known as adipokines, many of which play a role in the development of diabetes [9]. Leptin, an adipocyte-derived hormone, is produced by adipocytes, the ovaries, and the

placenta, and influences glucose metabolism by inhibiting insulin action [35]. In both normal weight and obese women with GDM, leptin levels in the second half of pregnancy are markedly higher compared to those in non-GDM women [36]. In contrast, adiponectin, another protein produced by adipocytes, is known for its anti-inflammatory and insulin-sensitizing properties [37]. While adiponectin levels decrease during normal pregnancy, studies have shown that women with GDM exhibit significantly lower levels of adiponectin compared to women without GDM [37, 38]. Hypoadiponectinemia has been linked to a 4.6-fold increase in the risk of developing GDM and is inversely related to body mass index (BMI), insulin resistance, and leptin levels [39]. Consequently, the plasma adiponectin/leptin ratio (0.33) in the 6th to 14th week of pregnancy may serve as a predictive marker for GDM [40]. Additional adipokines that may prove useful for diagnosing GDM include visfatin, resistin, and omentin. Visfatin is believed to activate NF- κ B signaling, which may contribute to insulin resistance [9]. Elevated levels of visfatin are seen in the late first trimester [41], but its expression in the third trimester of GDM varies [42]. Resistin, associated with increased levels of pro-inflammatory molecules, has been found to be either decreased or unchanged in women with GDM [43]. Omentin-1, an adipokine produced by non-fat cells in adipose tissue, plays a role in relaxing vascular tone by enhancing endothelial nitric oxide (NO) production and reducing levels of high-sensitivity C-reactive protein (hs-CRP) and tumor necrosis factor (TNF) signaling [44]. Omentin-1 levels are significantly reduced in the second trimester of GDM, paralleling the pattern observed with adiponectin [45]. Furthermore, studies have indicated that women with GDM present with lower fasting ghrelin levels compared to non-pregnant women and pregnant women without GDM, especially in the first and third trimesters [46].

Placenta-Related Markers

Placental factors are also implicated in the development of GDM. Sex hormone-binding globulin (SHBG), a glycoprotein expressed in the placenta that is involved in the transport of sex hormones, is suppressed by insulin [9]. SHBG levels have been shown to be negatively associated with both obesity and insulin resistance [47]. Notably, a decrease in plasma SHBG levels during the first trimester has been identified as an accurate biomarker for GDM [48]. A study by Nanda et al. observed a parallel decrease in SHBG levels and an increase in adiponectin levels during the 11th to 13th weeks of pregnancy, which was linked to a BMI greater than 30 kg/m² and a family history of diabetes mellitus (DM). Additionally, women who were administered insulin had even lower SHBG levels [49]. A reduction in plasma fetuin-A concentrations and an increase in hs-CRP levels have also been observed during the late first trimester [50]. Fetuin-A has been shown to interact with the insulin receptor tyrosine kinase, leading to the development of insulin resistance [51]. Moreover, afamin, a protein found in the liver and placenta and belonging to the albumin family, may act as a biomarker for abnormal lipid and glucose metabolism in the first trimester [52]. In this context, lower levels of ficolin-3, a lectin pathway activator found in both the liver and placenta, along with a higher ficolin-3/adiponectin ratio, have been associated with the prediction of GDM during the 16th to 18th week of pregnancy [37]. Additionally, follistatin, a regulator of follicle-stimulating hormone, is reduced in the third trimester of GDM pregnancies [53].

Urine Markers

Urine collected from pregnant women may contain potential prognostic and diagnostic biomarkers for GDM [9]. A study examining urine samples from women with GDM during the third trimester of pregnancy revealed a significant increase in 14 compounds related to tryptophan metabolism and steroid hormone production [54]. The upregulation of these pathways during GDM may exacerbate insulin resistance and could represent a response to oxidative stress and inflammation. In addition, elevated levels of 3-hydroxybutanoic acid (BHBA), valine, and alanine were detected in both the urine and plasma of women with GDM between the 12th and 26th weeks of pregnancy. Increased excretion of serotonin and its associated metabolites, such as 1-tryptophan, was also noted in these patients [55].

Molecular Markers

There is an increasing body of evidence suggesting that genetic markers, including microRNAs (miRNAs), single-nucleotide polymorphisms (SNPs), and DNA methylation, can serve as potential biomarkers for the early detection of gestational diabetes mellitus (GDM) [56]. Despite their promising applications, these molecular biomarkers face significant challenges that need to be addressed before their integration into clinical practice.

MicroRNA

MicroRNAs (miRNAs), approximately 22 nucleotides in length, are highly conserved non-coding RNA molecules that play a crucial role in the regulation of biological processes [57]. These molecules have been linked to genes involved in insulin signaling, as well as glucose and lipid metabolism [58]. miRNAs can be secreted from the placenta into the maternal bloodstream as early as the 6th week of pregnancy, potentially influencing placental growth and insulin signaling [58]. The secretion of miRNAs occurs either passively, associated with apoptotic bodies, or actively, through vesicles, exosomes, or lipoproteins [9]. Additionally, miRNAs are known to impact trophoblast proliferation, apoptosis, and angiogenesis [59], and their dysregulation in the placenta has been

associated with metabolic disorders, including GDM. A significant reduction in the serum levels of miR-29a, miR-132, and miR-222 has been observed in women with GDM (n = 28) during the 16th week of pregnancy compared to controls (n = 53) [60]. Specifically, miR-29a is involved in glucose and fatty acid metabolism, while miR-222 is linked to insulin resistance, and miR-132 is associated with insulin secretion and glucose homeostasis [61, 62]. However, other studies have reported elevated plasma levels of miR-222 in women with GDM (n = 13) compared to controls (n = 9) [63]. These discrepancies may arise from variations in biological specimens (serum/plasma), maternal age, or other unidentified factors. Furthermore, other miRNAs, such as miR-17-5p, miR-16-5p, and miR-20a-5p, have been found to increase during the first and second trimesters in women with GDM, and these miRNAs are implicated in the regulation of inflammation and insulin resistance [64, 65]. Similarly, unregulated plasma miR-21-3p levels have been associated with GDM between the 7th and 23rd weeks of pregnancy, and this miRNA has also been linked to insulin resistance and preeclampsia [66, 67]. Several miRNAs, along with their sensitivity and specificity, and these biomarkers have the potential to aid in the early prediction of GDM in maternal plasma during pregnancy.

Single-Nucleotide Polymorphisms

Single-nucleotide polymorphisms (SNPs) represent the most common form of genetic variation within DNA sequences and can have functional consequences, influencing protein function [68]. Various SNPs within genes involved in insulin production and resistance, as well as glucose and lipid metabolism, have been associated with an increased risk of GDM [1, 16]. Known mutations include those in genes such as adiponectin, glucokinase (GCK), glucokinase regulator (GCKR), insulin-like growth factor 2-binding protein (IGF2BP), insulin receptor substrate 1 (IRS-1), and peroxisome proliferator-activated receptor gamma (PPARG2) [16]. These studies have reported both positive and negative associations, as well as cases where no association was observed, suggesting that the results may be influenced by population characteristics and genotyping methodologies.

Epigenetic Modifications

Epigenetic modifications refer to alterations in the molecular structure of DNA that can influence gene expression without changing the underlying DNA sequence [71]. These modifications play a significant role in cellular processes associated with pathophysiological conditions [16]. Specifically, epigenetic changes can affect genes involved in beta-cell morphology, function, and proliferation, indicating that such modifications may contribute to impairments in insulin secretion and sensitivity, which are key features of GDM [56]. DNA methylation, the most studied form of epigenetic modification, occurs when a methyl group is added to a cytosine residue in a cytosine-phosphate-guanine (CpG) dinucleotide [71]. Several studies have indicated that DNA methylation patterns in the placenta and cord blood of women with GDM differ from those in women with normoglycemic pregnancies [72, 73]. For instance, Kang et al. demonstrated a decrease in IL-10 methylation in GDM, while serum IL-10 levels increased toward the end of pregnancy [74]. Additionally, DNA methylation may be involved in the pathogenesis of childhood cardiometabolic traits through the vascular adhesion molecule 1 (VCAM-1), which has been found to be elevated in offspring exposed to GDM in utero compared to those not exposed [75]. This elevation in VCAM-1 may be related to increased methylation of genes such as PYGO1 and CLN8 [75, 76]. Furthermore, DNA methylation near the leptin gene promoter has been shown to influence leptin levels in cord blood, with hypomethylation associated with elevated leptin levels in offspring [75]. Although DNA methylation holds promise as a diagnostic and prognostic marker, the majority of studies on this subject, particularly in the context of GDM, have involved small sample sizes and employed various methodologies to detect methylation, highlighting the need for further research to standardize these approaches.

Treatment of GDM

The management of gestational diabetes mellitus (GDM) plays a crucial role in improving glucose tolerance and mitigating the risks associated with GDM, including preeclampsia and macrosomia. The primary approach to managing GDM involves lifestyle modifications, such as dietary adjustments and increased physical activity. Women diagnosed with GDM are typically advised to undergo personalized dietary counseling, which commonly includes recommendations to limit carbohydrate intake to 33-40% of total caloric intake. For the majority of women, glycemic control can be achieved through diet alone; however, approximately 15% to 30% of patients may require insulin therapy [77]. The effects of physical exercise on glycemic control in GDM remain debated, with some studies indicating benefits, particularly from aerobic exercise, which can improve glycemic regulation in diabetic individuals. A general recommendation is for GDM patients to aim for at least 30 minutes of exercise most days of the week [78]. Additionally, careful monitoring of maternal weight gain is vital to reduce the risk of fetal macrosomia. Women who are obese, have GDM, and gain more than 40 lb (18.1 kg) during pregnancy face a 40% increased risk of delivering a macrosomic infant [79]. When lifestyle interventions fail to adequately control blood glucose levels, pharmacologic treatment is considered [80]. Insulin, as well as oral medications such as metformin

and glyburide, are commonly utilized in the management of GDM. Metformin dosage is based on glycemic control and generally begins at 500 mg taken once per day, either in the evening or twice daily. During pregnancy, the maximum daily dosage may reach 2500-3000 mg, which exceeds the dose typically used in non-pregnant individuals [2]. Recent meta-analyses have suggested that metformin may provide benefits for both pregnant women and their newborns, with no apparent adverse effects. Although metformin can cross the placenta, no conclusive evidence has been found to suggest teratogenic effects [81]. Nevertheless, further research is needed to strengthen the evidence base for the continued use of metformin in pregnancy.

Glyburide dosage is initiated at 2.5 mg every 12 hours and gradually increased to a maximum of 10 mg twice daily, depending on the patient's glycemic control [45]. If oral medications fail to achieve adequate blood glucose regulation, insulin therapy should be reconsidered. Insulin is a relatively large molecule that does not cross the placenta during pregnancy, making it a preferred treatment option. The type, dosage, and timing of insulin administration are tailored to the individual, considering factors such as the patient's weight, maternal age, and the time of day [80]. Despite its effectiveness, insulin is associated with higher costs and may pose challenges in terms of patient experience, limiting its use in some clinical settings. The management of GDM during pregnancy remains a subject of controversy due to the limited availability of data from large-scale randomized clinical trials. There is a pressing need for well-designed studies to provide evidence that can guide clinical decisions regarding the most effective approaches to GDM screening and treatment [82].

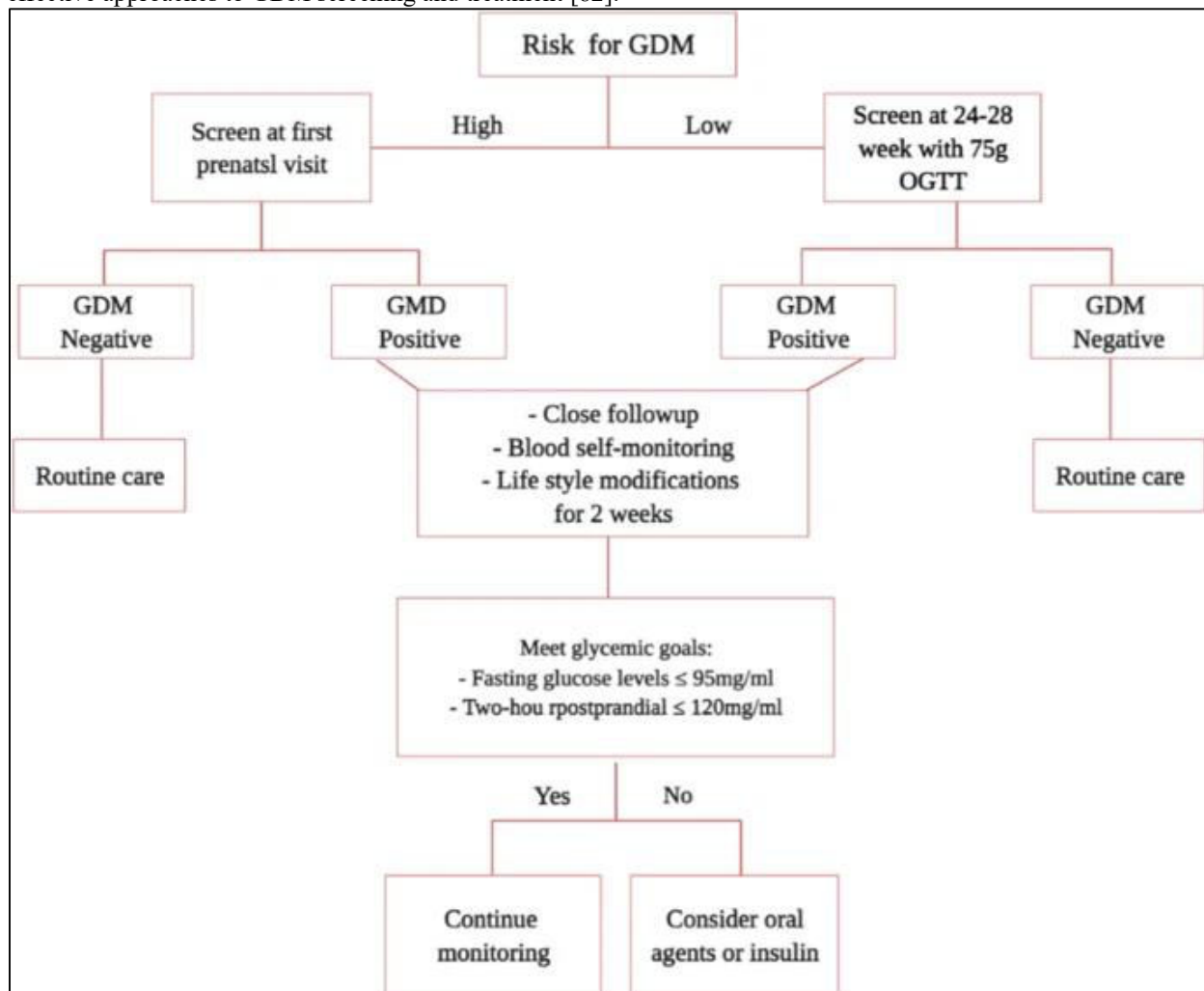


Figure 1: Management of GDM.

Nursing Interventions:

Gestational Diabetes Mellitus (GDM) is a condition characterized by elevated blood glucose levels that develop during pregnancy and usually resolve after delivery. However, it can pose significant risks to both the mother and the fetus, including the increased likelihood of macrosomia, preeclampsia, and the development of type

2 diabetes in later life. Nursing interventions for the GDM focus on managing blood glucose levels, educating patients, and promoting overall health for both mother and baby. The following nursing intervention plans outline the essential components for the care of women diagnosed with GDM.

1. Monitoring and Assessing Blood Glucose Levels

One of the primary nursing interventions for GDM is monitoring and maintaining blood glucose levels within the target range. Nurses play a crucial role in assessing the patient's blood glucose levels regularly, as this provides insight into the effectiveness of the current management plan. Nurses should educate patients on how to self-monitor blood glucose at home, teaching them the proper technique for using glucose meters and the significance of keeping track of readings. The nurse should stress the importance of adhering to the prescribed schedule for monitoring, especially after meals, when blood glucose levels are most likely to fluctuate. Regular glucose testing should be conducted as per the physician's recommendations, often at least four times per day: fasting, pre-meal, post-meal, and before bedtime. Blood glucose targets for GDM typically range between 60–95 mg/dL before meals and less than 120 mg/dL one to two hours after meals. Nurses must provide clear instructions regarding when to contact healthcare providers if blood glucose readings are outside the target range. Additionally, nurses should assist in interpreting these glucose readings, noting patterns, and collaborating with the healthcare team to adjust the treatment plan as necessary. This may involve insulin adjustments, changes in diet, or exercise modifications.

2. Dietary Education and Counseling

Dietary management is a cornerstone of GDM treatment. Nurses should educate patients about the importance of a balanced diet in controlling blood glucose levels. A registered dietitian may work alongside the nursing team to create a personalized meal plan that includes appropriate caloric intake and macronutrient distribution, particularly focusing on carbohydrates. It is essential for women with GDM to understand the concept of carbohydrate counting and the role of fiber, protein, and fats in moderating blood sugar levels. Patients should be encouraged to eat small, frequent meals throughout the day, rather than large meals, to prevent blood sugar spikes. The nurse should provide clear guidance on foods to avoid, including high-glycemic index foods such as refined carbohydrates, sugary snacks, and processed foods. Additionally, nurses should monitor any signs of poor nutritional intake, such as weight loss or difficulty following the diet plan, and offer support or refer patients to nutrition counseling when necessary.

3. Physical Activity Guidance

Exercise is another important element in managing GDM. The nurse should educate patients about the benefits of regular physical activity in controlling blood glucose levels and improving insulin sensitivity. Patients should be advised to aim for at least 30 minutes of moderate-intensity aerobic exercise most days of the week, as this can significantly enhance glucose control. Activities such as walking, swimming, or stationary cycling are often recommended, as they are low-impact and accessible. However, it is essential to assess each patient's physical capability and any contraindications, such as pre-existing conditions like hypertension or musculoskeletal issues. Nurses should emphasize the importance of exercising safely, monitoring for symptoms such as dizziness, shortness of breath, or unusual fatigue, and stopping exercise if these occur. Furthermore, nurses should encourage patients to track their exercise habits, ensuring that they are consistent with their physical activity goals and adjusting intensity as needed.

4. Weight Management and Fetal Monitoring

Weight management is vital for GDM patients, especially those who are overweight or obese. Nurses should monitor maternal weight gain throughout pregnancy, helping to ensure that weight gain stays within recommended guidelines to prevent fetal macrosomia and other complications. Women with GDM should aim for a steady, controlled weight gain, avoiding excessive weight gain, which can exacerbate blood glucose control and increase the risk of complications. In addition to monitoring maternal weight, nurses should support fetal monitoring. Routine ultrasounds, non-stress tests, and other assessments should be carried out as recommended by the healthcare provider to track fetal growth and well-being. Nurses should assist in interpreting the results and communicate any concerns to the healthcare team. Special attention should be given to identifying signs of fetal distress or complications, such as excessive fetal growth (macrosomia) or low amniotic fluid levels.

5. Pharmacologic Therapy Management

When lifestyle interventions such as diet and exercise are insufficient to control blood glucose levels, pharmacologic therapy may be required. Nurses should assist in managing medication regimens, particularly insulin and oral hypoglycemic agents like metformin and glyburide. Nurses should educate patients on the proper use of insulin injections, including the correct technique for subcutaneous administration, rotation of injection sites, and storage of insulin. For patients using oral medications, nurses should ensure that the patient understands the dosage

schedule, potential side effects, and the importance of adhering to the prescribed regimen. Nurses should also monitor for adverse reactions, such as hypoglycemia or gastrointestinal issues, and report them promptly to the healthcare team. In addition, nurses should work with patients to adjust treatment plans when necessary, particularly if blood glucose levels remain uncontrolled or if side effects become problematic.

6. Psychosocial Support and Education

Nurses must recognize the emotional and psychological burden that GDM may place on patients. Anxiety, stress, and fear of complications can affect the mother's emotional well-being, and these concerns should be addressed throughout the care process. Nurses should provide supportive counseling and encouragement, addressing any fears or misconceptions about the condition and treatment. In addition to emotional support, nurses should ensure that patients have access to relevant educational resources about GDM, its potential risks, and the importance of managing the condition effectively for the health of both mother and baby. Education should be clear, tailored to the patient's level of understanding, and provided in a variety of formats (verbal, written, and digital) to reinforce learning.

7. Postpartum Follow-up Care

After delivery, GDM patients are at an increased risk of developing type 2 diabetes. Nurses should ensure that women with a history of GDM are screened for diabetes at 6–12 weeks postpartum, and that they continue to receive care and education on long-term lifestyle changes to prevent the progression to type 2 diabetes. Nurses should also emphasize the importance of breastfeeding, as it has been shown to improve maternal glucose metabolism and reduce the risk of developing type 2 diabetes. In conclusion, the nursing interventions for GDM are multifaceted and require an integrated approach to care. Nurses play a pivotal role in monitoring blood glucose levels, educating patients about diet and exercise, supporting pharmacological treatment, managing weight gain and fetal health, and providing emotional support. Through these interventions, nurses help to improve maternal and fetal outcomes and reduce the risks associated with GDM.

Conclusion:

Gestational diabetes mellitus (GDM) represents a growing concern for both maternal and fetal health, with increasing global prevalence linked to rising obesity rates, sedentary lifestyles, and advanced maternal age. The condition presents a significant risk for long-term complications, including hypertension, preeclampsia, macrosomia, and the increased likelihood of developing type 2 diabetes later in life. In light of these risks, early identification and effective management are critical to improving pregnancy outcomes and reducing complications for both the mother and the baby. Despite advancements in understanding the pathophysiology of GDM, there remains ongoing debate over optimal diagnostic strategies, treatment methods, and the best practices for postpartum monitoring. Current guidelines recommend routine screening for GDM, but variations in diagnostic criteria—such as those from the World Health Organization (WHO) and the International Association of Diabetes and Pregnancy Study Groups (IADPSG)—highlight the need for further standardization in global practices. Collaboration between different healthcare professionals—specifically nurses, pharmacists, and clinical pathologists—can significantly enhance the diagnosis, management, and treatment of GDM. Nurses play an essential role in monitoring blood glucose levels, educating patients on lifestyle modifications, and providing support throughout the pregnancy. Pharmacists are crucial in managing pharmacological interventions, including the prescription and monitoring of insulin or other medications, while clinical pathologists assist with accurate laboratory testing and the identification of biomarkers that could facilitate early detection. By working together, these healthcare professionals can ensure more comprehensive care and better outcomes for patients. Recent studies suggest that biomarkers, such as leptin, adiponectin, and sex hormone-binding globulin, hold potential as early diagnostic tools for GDM. These markers, which can be detected in maternal blood or urine, offer a promising avenue for improving the predictability and early detection of the condition, potentially allowing for earlier interventions. Furthermore, ongoing research into the genetic and epigenetic factors contributing to GDM may lead to more personalized care, tailoring management strategies based on individual risk profiles. In conclusion, the integrated approach involving nursing, pharmacology, and clinical pathology is essential in managing GDM effectively. This collaborative model not only ensures timely interventions and optimized treatment strategies but also fosters a holistic approach to patient care. Future research should focus on refining diagnostic criteria, exploring novel biomarkers, and enhancing the effectiveness of current management protocols to minimize the risks associated with GDM. As the prevalence of this condition continues to rise, improving interdisciplinary collaboration will be key in addressing the challenges it presents and ensuring better health outcomes for both mothers and their babies.

References:

1. Chiefari E, Arcidiacono B, Foti D, Brunetti A. Gestational diabetes mellitus: an updated overview. *J Endocrinol Investig.* 2017;40:899–909.

2. Johns EC, Denison FC, Norman JE, Reynolds RM. Gestational diabetes mellitus: mechanisms, treatment, and complications. *Trends Endocrinol Metab.* 2018;29:743–54.
3. Alesi S, Ghelani D, Rassie K, Mousa A. Metabolomic biomarkers in gestational diabetes mellitus: a review of the evidence. *Int J Mol Sci.* 2021;22:5512.
4. Lawrence JM, Contreras R, Chen W, Sacks DA. Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999–2005. *Diabetes Care.* 2008;31:899–904.
5. Jenum AK, Mrøkrød K, Sletner L, Vange S, Torper JL, Nakstad B, et al. Impact of ethnicity on gestational diabetes identified with the WHO and the modified International Association of Diabetes and Pregnancy Study Groups criteria: a population-based cohort study. *Eur J Endocrinol.* 2012;166:317–24.
6. Moses RG, Wong VCK, Lambert K, Morris GJ, Gil FS. Seasonal changes in the prevalence of gestational diabetes mellitus. *Diabetes Care.* 2016;39:1218–21.
7. Burlina S, Dalfrà MG, Lapolla A. Short- and long-term consequences for offspring exposed to maternal diabetes: a review. *J Matern Neonatal Med.* 2019;32:687–94.
8. Lowe WL, Scholtens DM, Lowe LP, Kuang A, Nodzenski M, Talbot O, et al. Association of gestational diabetes with maternal disorders of glucose metabolism and childhood adiposity. *JAMA J Am Med Assoc.* 2018;320:1005–16.
9. Lorenzo-Almorós A, Hang T, Peiró C, Soriano-Guillén L, Egido J, Tuñón J, et al. Predictive and diagnostic biomarkers for gestational diabetes and its associated metabolic and cardiovascular diseases. *Cardiovasc Diabetol.* 2019;18:140.
10. Damm P, Houshmand-Oeregaard A, Kelstrup L, Lauenborg J, Mathiesen ER, Clausen TD. Gestational diabetes mellitus and long-term consequences for mother and offspring: a view from Denmark. *Diabetologia.* 2016;59:1396–9.
11. Zhu Y, Zhang C. Prevalence of gestational diabetes and risk of progression to type 2 diabetes: a global perspective. *Curr Diab Rep.* 2016;16:1–11.
12. Choudhury AA, Devi Rajeswari V. Gestational diabetes mellitus—a metabolic and reproductive disorder. *Biomed Pharmacother.* 2021;143:112183.
13. Ikeoka T, Sako A, Kuriya G, Yamashita H, Yasui I, Horie I, et al. Type 1 diabetes mellitus diagnosed during follow-up of gestational diabetes mellitus in the early postpartum period. *Intern Med.* 2018;57:3413–8.
14. Dalfrà MG, Burlina S, Del Vescovo GG, Lapolla A. Genetics and epigenetics: new insight on gestational diabetes mellitus. *Front Endocrinol.* 2020;11:602477.
15. Liu X, Wu N, Al-Mureish A. A review on research progress in the application of glycosylated hemoglobin and glycated albumin in the screening and monitoring of gestational diabetes. *Int J Gen Med.* 2021;14:1155–65.
16. Dias S, Pfeiffer C, Abrahams Y, Rheeder P, Adam S. Molecular biomarkers for gestational diabetes mellitus. *Int J Mol Sci.* 2018;19:2926.
17. Addison B, Belalcazar LM. Introduction of IADPSG criteria for the screening and diagnosis of gestational diabetes mellitus results in improved pregnancy outcomes at a lower cost in a large cohort of pregnant women: the St. Carlos gestational diabetes study. *Diabetes Care.* 2014;37(9):2442–50.
18. Li G, Wei T, Ni W, Zhang A, Zhang J, Xing Y, et al. Incidence and risk factors of gestational diabetes mellitus: a prospective cohort study in Qingdao, China. *Front Endocrinol.* 2020;11:636.
19. Ngala RA, Fondjo LA, Gmagna P, Gharthey FN, Awe MA. Placental peptides metabolism and maternal factors as predictors of risk of gestational diabetes in pregnant women. A case-control study. *PLoS ONE.* 2017;12:e0181613.
20. Chu SY, Callaghan WM, Kim SY, Schmid CH, Lau J, England LJ, et al. Maternal obesity and risk of gestational diabetes mellitus. *Diabetes Care.* 2007;30:2070–6.
21. Care ADA-D. Classification and diagnosis of diabetes. *Diabetes Care.* 2017;40:S11–24.
22. Larrabure-Torrealva GT, Martinez S, Luque-Fernandez MA, Sanchez SE, Mascaro PA, Ingar H, et al. Prevalence and risk factors of gestational diabetes mellitus: findings from a universal screening feasibility program in Lima, Peru. *BMC Pregnancy Childbirth.* 2018;18:1–9.
23. Wada T, Hori S, Sugiyama M, Fujisawa E, Nakano T, Tsuneki H, et al. Progesterone inhibits glucose uptake by affecting diverse steps of insulin signaling in 3T3-L1 adipocytes. *Am J Physiol Endocrinol Metab.* 2010;298:E881–8.
24. Gorvin CM. The prolactin receptor: diverse and emerging roles in pathophysiology. *J Clin Transl Endocrinol.* 2015;2:85–91.

25. Barbour LA, Shao J, Qiao L, Leitner W, Anderson M, Friedman JE, et al. Human placental growth hormone increases expression of the P85 regulatory unit of phosphatidylinositol 3-kinase and triggers severe insulin resistance in skeletal muscle. *Endocrinology*. 2004;145:1144–50.
26. Catalano PM, Tyzbir ED, Wolfe RR, Calles J, Roman NM, Amini SB, et al. Carbohydrate metabolism during pregnancy in control subjects and women with gestational diabetes. *Am J Physiol Endocrinol Metab*. 1993;264:E60–7.
27. Plows JF, Stanley JL, Baker PN, Reynolds CM, Vickers MH. The pathophysiology of gestational diabetes mellitus. *Int J Mol Sci*. 2018;19:3342.
28. Kramer CK, Swaminathan B, Hanley AJ, Connelly PW, Sermer M, Zinman B, et al. Each degree of glucose intolerance in pregnancy predicts distinct trajectories of β -cell function, insulin sensitivity, and glycemia in the first 3 years postpartum. *Diabetes Care*. 2014;37:3262–9.
29. Colomiere M, Permezel M, Riley C, Desoye G, Lappas M. Defective insulin signaling in placenta from pregnancies complicated by gestational diabetes mellitus. *Eur J Endocrinol*. 2009;160:567–78.
30. Sivan E, Chen X, Homko CJ, Reece EA, Boden G. Longitudinal study of carbohydrate metabolism in healthy obese pregnant women. *Diabetes Care*. 1997;20:1470–5.
31. Nguyen-Ngo C, Jayabalan N, Salomon C, Lappas M. Molecular pathways disrupted by gestational diabetes mellitus. *J Mol Endocrinol*. 2019;63:R51–72.
32. Yuen L. Gestational diabetes mellitus: challenges for different ethnic groups. *World J Diabetes*. 2015;6:1024.
33. Agarwal MM. Gestational diabetes mellitus: an update on the current international diagnostic criteria. *World J Diabetes*. 2015;6:782.
34. Basri NI, Mahdy ZA, Ahmad S, Abdul Karim AK, Shan LP, Abdul Manaf MR, et al. The World Health Organization (WHO) versus the International Association of Diabetes and Pregnancy Study Group (IADPSG) diagnostic criteria of gestational diabetes mellitus (GDM) and their associated maternal and neonatal outcomes. *Horm Mol Biol Clin Investig*. 2018;34:1–8.
35. Katsiki N, Mikhailidis DP, Banach M. Leptin, cardiovascular diseases and type 2 diabetes mellitus review. *Acta Pharmacol Sin*. 2018;39:1176–88.
36. Boyadzhieva M, Atanasova I, Zacharieva S, Kedikova S. Adipocytokines during pregnancy and postpartum in women with gestational diabetes and healthy controls. *J Endocrinol Investig*. 2013;36:944–9.
37. Yuan XS, Shi H, Wang HY, Yu B, Jiang J. Ficolin-3/adiponectin ratio for the prediction of gestational diabetes mellitus in pregnant women. *J Diabetes Investig*. 2018;9:403–10.
38. Bozkurt L, Göbl CS, Baumgartner-Parzer S, Luger A, Pacini G, Kautzky-Willer A. Adiponectin and leptin at early pregnancy: association to actual glucose disposal and risk for GDM—a prospective cohort study. *Int J Endocrinol*. 2018;2018:62–70.
39. Cseh K, Baranyi É, Melczer Z, Kaszás E, Palik É, Winkler G. Plasma adiponectin and pregnancy-induced insulin resistance. *Diabetes Care*. 2004;27:274–5.
40. Thagaard IN, Krebs L, Holm JC, Lange T, Larsen T, Christiansen M. Adiponectin and leptin as first trimester markers for gestational diabetes mellitus: a cohort study. *Clin Chem Lab Med*. 2017;55:1805–12.
41. Ferreira AFA, Rezende JC, Vaikousi E, Akolekar R, Nicolaides KH. Maternal serum visfatin at 11–13 weeks of gestation in gestational diabetes mellitus. *Clin Chem*. 2011;57:609–13.
42. Rezvan N, Hosseinzadeh-Attar MJ, Masoudkabir F, Moini A, Janani L, Mazaherioun M. Serum visfatin concentrations in gestational diabetes mellitus and normal pregnancy. *Arch Gynecol Obstet*. 2012;285:1257–62.
43. Megia A, Vendrell J, Gutierrez C, Sabaté M, Broch M, Fernández-Real JM, et al. Insulin sensitivity and resistin levels in gestational diabetes mellitus and after parturition. *Eur J Endocrinol*. 2008;158:173–8.
44. Yang RZ, Lee MJ, Hu H, Pray J, Wu HB, Hansen BC, et al. Identification of omentin as a novel depot-specific adipokine in human adipose tissue: possible role in modulating insulin action. *Am J Physiol Endocrinol Metab*. 2006;290:E1253–61.
45. Abell SK, Shorakae S, Harrison CL, Hiam D, Moreno-Asso A, Stepto NK, et al. The association between dysregulated adipocytokines in early pregnancy and development of gestational diabetes. *Diabetes Metab Res Rev*. 2017;33:e2926.
46. Stranak Z, Krofta L, Haak LA, Vojtěch J, Hašlík L, Feyereisl J. Prenatal parameters to estimate outcome and respiratory morbidity in fetuses with isolated left-sided congenital diaphragmatic hernia. *Eur J Obstet Gynecol Reprod Biol*. 2016;206:e110–1.
47. Ding EL, Song Y, Malik VS, Liu S. Sex differences of endogenous sex hormones and risk of type 2 diabetes: a systematic review and meta-analysis. *J Am Med Assoc*. 2006;295:1288–99.
48. Zhang T, Du T, Li W, Yang S, Liang W. Sex hormone-binding globulin levels during the first trimester may predict gestational diabetes mellitus development. *Biomark Med*. 2018;12:239–44.

49. Nanda S, Savvidou M, Syngelaki A, Akolekar R, Nicolaides KH. Prediction of gestational diabetes mellitus by maternal factors and biomarkers at 11 to 13 weeks. *Prenat Diagn.* 2011;31:135–41.
50. Kansu-Celik H, Ozgu-Erdinc AS, Kisa B, Findik RB, Yilmaz C, Tasci Y. Prediction of gestational diabetes mellitus in the first trimester: Comparison of maternal fetuin-A, N-terminal proatrial natriuretic peptide, high-sensitivity C-reactive protein, and fasting glucose levels. *Arch Endocrinol Metab.* 2019;63:121–7.
51. Farhan S, Handisurya A, Todoric J, Tura A, Pacini G, Wagner O, et al. Fetuin-A characteristics during and after pregnancy: Result from a case control pilot study. *Int J Endocrinol.* 2012;2012:36–41.
52. Köninger A, Mathan A, Mach P, Frank M, Schmidt B, Schleussner E, et al. Is Afamin a novel biomarker for gestational diabetes mellitus? A pilot study. *Reprod Biol Endocrinol.* 2018;16:1–11.
53. Náf S, Escote X, Ballesteros M, Yañez RE, Simón-Muela I, Gil P, et al. Serum activin A and follistatin levels in gestational diabetes and the association of the activin A-follistatin system with anthropometric parameters in offspring. *PLoS ONE.* 2014;9:e92175.
54. López-Hernández Y, Van Oostdam ASH, Toro-Ortiz JC, López JA, Salgado-Bustamante M, Murgu M, et al. Urinary metabolites altered during the third trimester in pregnancies complicated by gestational diabetes mellitus: relationship with potential upcoming metabolic disorders. *Int J Mol Sci.* 2019;20:1186.
55. Leitner M, Fragner L, Danner S, Holeschovsky N, Leitner K, Tischler S, et al. Combined metabolomic analysis of plasma and urine reveals AHBA, tryptophan and serotonin metabolism as potential risk factors in Gestational Diabetes Mellitus (GDM). *Front Mol Biosci.* 2017;4:84.
56. Yahaya TO, Salisu T, Abdulrahman YB, Umar AK. Update on the genetic and epigenetic etiology of gestational diabetes mellitus: a review. *Egypt J Med Hum Genet.* 2020;21:1–13.
57. O'Brien J, Hayder H, Zayed Y, Peng C. Overview of microRNA biogenesis, mechanisms of actions, and circulation. *Front Endocrinol.* 2018;9:402.
58. Poirier C, Desgagné V, Guérin R, Bouchard L. MicroRNAs in pregnancy and gestational diabetes mellitus: emerging role in maternal metabolic regulation. *Curr Diab Rep.* 2017;17:1–10.
59. Morales-Prieto DM, Ospina-Prieto S, Schmidt A, Chaiwangyen W, Markert UR. Elsevier trophoblast research award lecture: Origin, evolution and future of placenta miRNAs. *Placenta.* 2014;35:S39–45.
60. Zhao C, Dong J, Jiang T, Shi Z, Yu B, Zhu Y, et al. Early second-trimester serum miRNA profiling predicts gestational diabetes mellitus. *PLoS ONE.* 2011;6:e23925.
61. He A, Zhu L, Gupta N, Chang Y, Fang F. Overexpression of micro ribonucleic acid 29, highly up-regulated in diabetic rats, leads to insulin resistance in 3T3-L1 adipocytes. *Mol Endocrinol.* 2007;21:2785–94.
62. Shi Z, Zhao C, Guo X, Ding H, Cui Y, Shen R, et al. Differential expression of micrnas in omental adipose tissue from gestational diabetes mellitus subjects reveals mir-222 as a regulator of *era* expression in estrogen-induced insulin resistance. *Endocrinology.* 2014;155:1982–90.
63. Tagoma A, Alnek K, Kirss A, Uibo R, Haller-Kikkatalo K. MicroRNA profiling of second trimester maternal plasma shows upregulation of miR-195-5p in patients with gestational diabetes. *Gene.* 2018;672:137–42.
64. Zhu Y, Tian F, Li H, Zhou Y, Lu J, Ge Q. Profiling maternal plasma microRNA expression in early pregnancy to predict gestational diabetes mellitus. *Int J Gynecol Obstet.* 2015;130:49–53.
65. Fernandes T, Magalhães FC, Roque FR, Phillips MI, Oliveira EM. Exercise training prevents the microvascular rarefaction in hypertension balancing angiogenic and apoptotic factors: role of microRNAs-16, -21, and -126. *Hypertension.* 2012;59:513–20.
66. Wander PL, Boyko EJ, Hevner K, Parikh VJ, Tadesse MG, Sorensen TK, et al. Circulating early- and mid-pregnancy microRNAs and risk of gestational diabetes. *Diabetes Res Clin Pract.* 2017;132:1–9.
67. Hocaoglu M, Demirer S, Senturk H, Turgut A, Komurcu-Bayrak E. Differential expression of candidate circulating microRNAs in maternal blood leukocytes of the patients with preeclampsia and gestational diabetes mellitus. *Pregnancy Hypertens.* 2019;17:5–11.
68. Consortium T international H. A second generation human haplotype map of over 3.1 million SNPs. *Nature.* 2005;449:851–61.
69. Tarnowski M, Malinowski D, Pawlak K, Dziedziczko V, Safranow K, Pawlik A. GCK, GCKR, FADS1, DGKB/TMEM195 and CDKAL1 gene polymorphisms in women with gestational diabetes. *Can J Diabetes.* 2017;41:372–9.
70. Jamalpour S, Zain SM, Mosavat M, Mohamed Z, Omar SZ. A case-control study and meta-analysis confirm glucokinase regulatory gene rs780094 is a risk factor for gestational diabetes mellitus. *Gene.* 2018;650:34–40.
71. Lim DHK, Maher ER. DNA methylation: a form of epigenetic control of gene expression. *Obstet Gynaecol.* 2010;12:37–42.

72. Finer S, Mathews C, Lowe R, Smart M, Hillman S, Foo L, et al. Maternal gestational diabetes is associated with genome-wide DNA methylation variation in placenta and cord blood of exposed offspring. *Hum Mol Genet.* 2014;24:3021–9.
73. Reichetzeder C, Dwi Putra SE, Pfab T, Slowinski T, Neuber C, Kleuser B, et al. Increased global placental DNA methylation levels are associated with gestational diabetes. *Clin Epigenet.* 2016;8:1–10.
74. Kang J, Lee CN, Li HY, Hsu KH, Wang SH, Lin SY. Association of interleukin-10 methylation levels with gestational diabetes in a Taiwanese population. *Front Genet.* 2018;9:222.
75. Elliott HR, Sharp GC, Relton CL, Lawlor DA. Epigenetics and gestational diabetes: a review of epigenetic epidemiology studies and their use to explore epigenetic mediation and improve prediction. *Diabetologia.* 2019;62:2171–8.
76. West NA, Kechris K, Dabelea D. Exposure to maternal diabetes in utero and DNA methylation patterns in the offspring. *Immunometabolism.* 2013;1:1–9.
77. Dolatkah N, Hajifaraji M, Shakouri SK. Nutrition therapy in managing pregnant women with gestational diabetes mellitus: a literature review. *J Fam Reprod Heal.* 2018;12:57–72.
78. Padayachee C. Exercise guidelines for gestational diabetes mellitus. *World J Diabetes.* 2015;6:1033.
79. Black MH, Sacks DA, Xiang AH, Lawrence JM. The relative contribution of prepregnancy overweight and obesity, gestational weight gain, and IADPSG-defined gestational diabetes mellitus to fetal overgrowth. *Diabetes Care.* 2013;36:56–62.
80. Lende M, Rijhsinghani A. Gestational diabetes: overview with emphasis on medical management. *Int J Environ Res Public Health.* 2020;17:1–12.
81. Bao LX, Shi WT, Han YX. Metformin versus insulin for gestational diabetes: a systematic review and meta-analysis. *J Matern Neonatal Med.* 2021;34:2741–53.
82. Karami, M., Mousavi, S. H., Rafiee, M., Heidari, R., & Shahrokhi, S. Z. (2023). Biochemical and molecular biomarkers: Unraveling their role in gestational diabetes mellitus. *Diabetology & Metabolic Syndrome*, 15(1), 5.

سكري الحوامل: التعاون بين التمريض، الصيدلة، وعلم الأمراض السريري في التشخيص، الإدارة، والعلاج

الملخص:

الخلفية: يُعد سكري الحوامل (GDM) اضطرابًا استقلابيًا يظهر عادةً في الثلث الثاني والثالث من الحمل، ويتميز بعدم تحمل الجلوكوز. ينتشر هذا المرض بشكل متزايد بسبب عوامل مثل السمنة، أنماط الحياة الخاملة، وتقدم عمر الأم. قد يؤدي سكري الحوامل إلى مضاعفات خطيرة مثل تسمم الحمل، ضخامة حجم الجنين، وفرط بيليروبين الدم لدى حديثي الولادة. بالإضافة إلى ذلك، فإن النساء المصابات بسكري الحوامل أكثر عرضة للإصابة بداء السكري من النوع الثاني لاحقًا. يعد الكشف المبكر والإدارة السليمة أمرين حاسمين للوقاية من المضاعفات قصيرة وطويلة المدى لكل من الأم والجنين. ومع ذلك، لا تزال معايير التشخيص وخيارات العلاج المتاحة موضع جدل.

الهدف: تستكشف هذه المقالة التعاون بين التمريض، الصيدلة، وعلم الأمراض السريري في تشخيص، إدارة، وعلاج سكري الحوامل. وتهدف إلى تسليط الضوء على كيفية تحسين النتائج الصحية من خلال نهج متعدد التخصصات، وتحديد الحاجة إلى تحسين بروتوكولات التشخيص والعلاج.

المنهجية: تستعرض المقالة الأدبيات الحالية حول سكري الحوامل، مع التركيز على علم الوبائيات، عوامل الخطر، الآليات المرضية، التشخيص، والعلاج. وتناقش الأدوار التعاونية لمهنيي التمريض، الصيدلة، وعلم الأمراض، مع التأكيد على كيفية مساهمة كل تخصص في رعاية سكري الحوامل.

النتائج: تشير النتائج الرئيسية إلى أن نهج الرعاية المتكاملة يحسن الكشف المبكر، وإدارة أفضل لمستويات الجلوكوز في الدم، وتقليل المضاعفات. تم تسليط الضوء على دور المؤشرات الحيوية في التنبؤ والتشخيص، مع التركيز على مؤشرات معينة مثل اللبتين والأديبونكتين التي تُظهر إمكانات واعدة. بالإضافة إلى ذلك، تناقش المقالة طرق التشخيص المختلفة وأهمية الرعاية المخصصة.

الخاتمة: يُعد التعاون بين التمريض، الصيدلة، وعلم الأمراض السريري ضروريًا للإدارة الفعالة لسكري الحوامل. يضمن هذا العمل الجماعي التشخيص في الوقت المناسب، العلاج المناسب، والمراقبة المستمرة، مما يُحسن النتائج الصحية لكل من الأم والجنين. هناك حاجة إلى مزيد من البحث حول المؤشرات الحيوية ومعايير التشخيص الجديدة لتعزيز استراتيجيات الكشف المبكر والعلاج.

الكلمات المفتاحية: سكري الحوامل، التمريض، علم الأدوية، علم الأمراض السريري، المؤشرات الحيوية، الإدارة، التشخيص، العلاج.