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Short Review on Biomarkers for the Diagnosis of Gestational Diabetes

Ariel Pablo Lopez*

Department of Genetics, Molecular Biology Laboratory, Universidad de Buenos Aires, Argentina

Correspondence to: Ariel Pablo Lopez, Department of Genetics, Molecular Biology Laboratory, Universidad de Buenos Aires, Argentina; E-mail: aplopez@prensamedica.com.ar

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Introduction

About 14% of births, or 135,000 women annually, in the United States are affected by gestational diabetes mellitus (GDM), also known as "impaired glucose tolerance", which was initially identified during pregnancy [1]. GDM is a parental risk factor for developing type 2 diabetes [2]. GDM raises the likelihood of developing type 2 diabetes (T2DM) and a number of other disorders, as well as unfavorable maternal and fetal outcomes. The improvement of both maternal and fetal health will be made possible by early risk assessment to stop the progression of GDM and by advancements in biomarker testing for the detection of GDM. The value of spectroscopy is that it enables molecular information without the need for specific stains and dyes, which expedites and streamlines the required ex vivo and in vivo testing for medical treatments. Pregnancy's most frequent complication, GDM, has grown by even more than 30% in several developing nations during the past 20 years [3]. More than 10% of pregnancies are affected by GDM, which raises the possibility of pregnancy problems such as hypertension, fetal abnormalities, miscarriage, emergency caesarean sections, and the eventual onset of T2DM in children and mothers [4]. For low- and middle-income nations compared to high-income countries, the incidence of risk factors like overweight or obese people, sedentary behavior, poor diet, and diabetes grew more quickly [5]. The highest frequency of GDM (12.9%) is found in North Africa and the Middle East, according to estimations based on diagnostic criteria for each nation [6,7]. Throughout many nations and eras, diagnostic standards and routine screening techniques varied. The use of various methods in earlier attempts to discover biomarkers has proven restrictive. Spectroscopic methods have shown promise as instruments for biological research, and their use in clinical assessment has grown dramatically in recent years. These approaches' key benefits over traditional imaging methods include being less intrusive, reagent-free, and enabling in vivo evaluation and ex vivo research. Spectral data may be gathered in a matter of seconds, allowing for the quick identification and gathering of multidimensional data on important organs when surgery is not advised. It is characterized as the study of how electromagnetic radiation interacts with atoms and molecules to cause a change in their energy state, moving them from a stable to a more energetically excited state. Energy is emitted, absorbed, lost, or converted throughout this process. Moreover, spectroscopic techniques may be used to quantify component concentrations in samples as well as describe biological material in vitro and in vivo. Disease biomarkers can be found and created using spectroscopic techniques. Early biomarker identification improves therapies, which lower mortality and morbidity. Raman spectroscopy, Fourier transform infrared spectroscopy, elastic scattering spectroscopy, fluorescence spectroscopy, and nuclear magnetic resonance spectroscopy are only a few of the spectroscopic methods employed in the clinical sector [8,9].

Methods

To adjust to pregnancy, women have particular metabolic and cardiovascular changes. Even before the placenta develops into a functional organ, several changes happen very early on during pregnancy [10]. Maternal insulin sensitivity frequently rises, followed by lipid synthesis and fat accumulation in adipose tissue. In addition, variations in hormone and metabolic levels are linked to shifts in heart size, shape, and function. Furthermore, insulin receptors and signaling are enhanced. Cells react by creating more insulin to keep blood sugar levels normal. Progesterone and oestrogen are significant steroid hormones that affect insulin sensitivity, among other placental factors [11]. While progesterone decreases insulin-stimulated glucose absorption and oestrogen increases systemic insulin sensitivity, both hormones produce pancreatic hypertrophy. Moreover, they have opposing impacts on vascular and dietary physiology. The major energy source for the myocardium is lipids, which are converted into lipids by the hormone progesterone. Progesterone also inhibits the release of neuroactive hormones, including melatonin and serotonin, which improve insulin sensitivity and glucose tolerance [12]. Moreover, oxytocin decreases food intake, obesity, insulin and glucose intolerance, blood pressure, and heart oxygenation and inflammation [13]. Although GH curbs hunger by lowering hunger and peptide Y production during pregnancy, PRL increases appetite by blocking leptin. Placental factors control how the mother adjusts to the demands of her metabolism and cardiovascular system. T2DM is associated with several genetic variations of GDM. Mutants in insulin, the insulin signaling pathway, insulin-like economic expansion factor-2, glycogen synthase, the PRL-GH family, cell line nuclear factor-4A, plasminogen inhibitor

1 (PAI-1), and melatonin receptor 1B have historically been used to explain how the placenta plays a role in the development of GDM. It's important to note that obesity can cause GDM to develop. Despite the fact that body mass index (BMI) is unimportant for overweight women during infertility, this anthropomorphic factor has been linked to the development of GDM. Adipose tissue and the placenta can both produce a similar pattern of cytokines, which explains why obese women are more likely to be obese.

Metabolic and Cardiovascular Conditions in Women with GDM

Specifically, in obese women, GDM is linked to the onset of postpartum metabolic syndrome. After delivery, cell dysfunction, insulin resistance, and fasting glucose all still exist. Adiponectin is absent, while E-selectin, ICAM-1, fibrinogen, interleukin-6, metalloproteinase-1 (TIMP-1), and PAI-1 levels are also elevated. There haven't yet been any epidemiological studies with a sizable, unselected sample of pregnant women whose blood glucose levels were checked before and during the pregnancy. Cardiac output also declines in the first hour following delivery before returning to normal after two weeks. A 66% increase in long-term cardiovascular damage was positively linked to GDM. Despite having a high BMI, women with GDM had a higher incidence of hospitalization for cardiovascular illness after giving birth. Compared to normal pregnancies, GDM is linked to significant prenatal morbidity and poor newborn outcomes. High plasma glucose and lipids in GDM women are linked to embryonic abnormalities and heart hypertrophy. Regardless of macrosomia, children and adolescents can have higher BMIs, glucose intolerances, and blood pressures, and they are more likely to experience GDM during their own pregnancies, which adds to the intergenerational loop of this illness [14].

GDM Diagnosis and Therapy

Women with GDM are encouraged to make lifestyle adjustments and are given the same care as women with GDM who are not obese. Before and throughout pregnancy, a diet comprising 30–35 kcal/kg, 33–40 L of carbohydrates, and exercise can help to maintain glucose homeostasis and ameliorate GDM pathophysiology. High age or BMI is already a symptom of medical need, but greater strenuous exercise (>60 min) might produce hypoglycemia and maternal hyperglycemia. Some people frequently experience hypoglycemia, which implies the necessity for different dosage strategies, such as large doses. Although metformin does not lower newborn hypoglycemia or macrosomia, it does reduce hyperglycemia and weight gain when combined with intermittent insulin injections [15]. Depending on the patient's age, history, stage of pregnancy, and existence of other conditions, pharmaceutical therapies may have a harmful impact on both the mother and the child. There is no standardized way to find GDM as of now. Therapeutically programmed advice identifying GDM by measuring glucose tolerance in the fasting state and after 1–2 hours of glucose overload is necessary because GDM may be anticipated when specific threshold values are achieved after glucose homeostasis has been quantified based on various factors. When hyperglycemia is identified by a hyperglycemia challenge test and verified by a further 1–3 hours of glucose excess, GDM is diagnosed using the two-step procedure.

Conclusion

The diagnosis and treatment of GDM, which impacts the cardiovascular and metabolic development of both the mother and the fetus, lack a consensus technique. GDM can be predicted by first trimester changes in plasma SHBG, adiponectin, RBP, afamin, ficolin-3, and specific miRNAs (miR-16-5p, miR-17-5p, and miR-20a-5p) [16,17]. Moreover, elevations in plasma FGF-21 and FABP in the third trimester can aid OGTT in the identification of GDM (Figure 1) [18]. Measurement of 5-anhydroglycitol can also forecast the onset of GDM. GDM-related cardiovascular impairment can be anticipated or identified. The panel of GDM biomarkers now includes vasatin, omentin-1, fetuin-A, IL-6, PAI-1, and FGF-21/23. With the ability to predict and categorize GDM with or without cardiovascular risk, personalized medicine would have the chance to address the main risk factors for disease recurrence, improving clinical outcomes and quality of life.

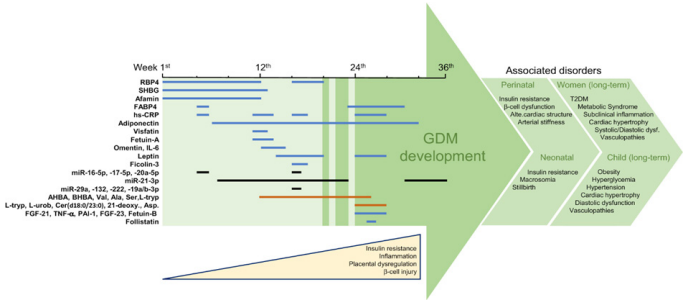

Figure 1: Prognostic and diagnostic biomarkers of GDM typically develop in the second trimester of pregnancy with increased inflammation, insulin resistance, placental dysregulation, and/or β-cell dysfunction and can be detected at weeks 24–28 by assessing glucose homeostasis. However, some specific proteins (blue lines), miRNAs (black lines), and metabolites (red lines) are released into the blood and/or urine during the (complicated) early stages of pregnancy and may act as GDM biomarkers. In particular, RBP, SHBG, afamin, FABP, hs-PCR, adiponectin, and several miRNAs (miR-16-5p, miR-17-5p, and miR-20a-5p) could be tested in early pregnancy, mainly in women with risk factors (obesity, advanced age, and previous GDM). In addition, visfatin, fetuin-A, omentin, leptin, ficolin-3, and certain metabolites (i.e., AHBA and L-Tryp) may be useful in mid-pregnancy, and FGF-21, PAI-1, fetuin-B, follistatin, and other metabolites [ceramide (d18:0/23:0), aspartame] may help in screening for GDM in the third trimester. Subsequently, early interventions for metabolic and cardiovascular diseases can mitigate postpartum (perinatal, neonatal, and chronic) diseases in women and their offspring [18].

References

1. Kim C, Newton KM, Knopp RH (2002) Gestational diabetes and the incidence of type 2 diabetes: a systematic review. Diabetes Care 25: 1862-1868. https://doi.org/10.2337/diacare.25.10.1862

2. Ferrara A (2007) Increasing prevalence of gestational diabetes mellitus: a public health perspective. Diabetes Care 30: S141-S146. https://doi.org/10.2337/dc07-s206

3. Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications: Report of a WHO Consultation. Part 1, Diagnosis and Classification of Diabetes Mellitus. [https://apps.who.int/iris/handle/10665/66040] [Accessed March 20, 2023]

4. Zhu Y, Zhang C (2016) Prevalence of gestational diabetes and risk of progression to type 2 diabetes: a global perspective. Curr Diab Rep 16: 7. https://doi.org/10.1007/s11892-015-0699-x

5. Coustan DR (1995) Gestational diabetes. In Harris MI, Cowie CC, Stern MP, Boyko EJ, Reiber GE, et al (eds) Diabetes in America. U.S. Govt. Printing Office, Washington, DC.

6. Hagbard L, Svanborg A (1960) Prognosis of diabetes mellitus with onset during pregnancy: a clinical study of seventy-one cases. Diabetes 9: 296-302. https://doi.org/10.2337/diab.9.4.296

7. Stowers JM, Sutherland HW, Kerridge DF (1985) Long-range implications for the mother: the Aberdeen experience. Diabetes 34: 106-110. https://doi.org/10.2337/diab.34.2.s106

8. Ward WK, Johnston CL, Beard JC, Benedetti TJ, Halter JB, et al. (1985) Insulin resistance and impaired insulin secretion in subjects with histories of gestational diabetes mellitus. Diabetes 34: 861-869. https://doi.org/10.2337/diab.34.9.861

9. Thorpe LE, Berger D, Ellis JA, Bettegowda VR, Brown G, et al. (2005) Trends and racial/ethnic disparities in gestational diabetes among pregnant women in New York City, 1990–2001. Am J Public Health 95: 1536-1539. https://doi.org/10.2105/AJPH.2005.066100

10. Dornhorst A, Paterson CM, Nicholls JSD, Wadsworth J, Chiu DC, et al. (1992) High prevalence of gestational diabetes in women from ethnic minority groups. Diabet Med 9: 820-825. https://doi.org/10.1111/j.1464-5491.1992.tb01900.x

11. Yang X, Hsu-Hage B, Zhang H, Yu L, Dong L, et al. (2002) Gestational diabetes mellitus in women of single gravidity in Tianjin City, China. Diabetes Care 25: 847-851. https://doi.org/10.2337/diacare.25.5.847

12. Zargar AH, Sheikh MI, Bashir MI, Masoodi SR, Laway BA, et al. (2004) Prevalence of gestational diabetes mellitus in Kashmiri women from the Indian subcontinent. Diabetes Res Clin Pract 66: 139-145. https://doi.org/10.1016/j.diabres.2004.02.023

13. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, Mebazaa A (2016) Physiological changes in pregnancy: review articles. Cardiovasc J Afr 27: 89-94. https://doi.org/10.5830/CVJA-2016-021

14. Musial B, Fernandez-Twinn DS, Vaughan OR, Ozanne SE, Voshol P, et al. (2016) Proximity to delivery alters insulin sensitivity and glucose metabolism in pregnant mice. Diabetes 65: 851-860. https://doi.org/10.2337/db15-1531

15. Barbour LA, McCurdy CE, Hernandez TL, Kirwan JP, Catalano PM, et al. (2007) Cellular mechanisms for insulin resistance in normal pregnancy and gestational diabetes. Diabetes Care 30: S112-S119. https://doi.org/10.2337/dc07-s202

16. Richardson AC, Carpenter MW (2007) Inflammatory mediators in gestational diabetes mellitus. Obstet Gynecol Clin North Am 34: 213-224. https://doi.org/10.1016/j.ogc.2007.04.001

17. Cong J, Fan T, Yang X, Squires JW, Cheng G, et al. (2015) Structural and functional changes in maternal left ventricle during pregnancy: a three-dimensional speckle-tracking echocardiography study. Cardiovasc Ultrasound 13: 6. https://doi.org/10.1186/1476-7120-13-6

18. Lorenzo-Almorós A, Hang T, Peiró C, Soriano-Guillén L, Egido J, et al. (2019) Predictive and diagnostic biomarkers for gestational diabetes and its associated metabolic and cardiovascular diseases. Cardiovasc Diabetol 18: 140. https://doi.org/10.1186/s12933-019-0935-9