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Article

The Relationship Between Blood Glucose Level and other Biomarkers in Pregnant Women

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Abstract: This study aimed to determine the relationship between blood glucose levels and other biomarkers in pregnant women compared to non-pregnant women. This study was conducted in Kirkuk, northern Iraq, from January 2024 to April 2024. One hundred blood samples were collected and divided into two groups: 50 samples from non-pregnant women and 50 samples from pregnant women. The study revealed significant elevations in blood glucose, urea, and chloride levels in pregnant women, while serum potassium levels decreased significantly. The study did not reveal any statistically significant differences in serum potassium and creatinine levels between the two groups. These findings highlight changes in glucose metabolism, kidney function, and electrolyte balance during pregnancy, which are critical factors for fetal growth and development. Continuous monitoring of these parameters is crucial to maintaining maternal health and avoiding complications. Future studies require analyzing larger and more diverse populations, using advanced and cost-effective methods, to confirm and improve these findings.

Keywords: Kirkuk, Biomarkers, Biochemical Parameters, Pregnant Women

Introduction

Gestational diabetes is a public and cumulative impediment of pregnancy [1]. Furthermost women suffering from this complication develop everlasting metabolic disorders within a period after pregnancy [2]. Diabetes is the most public metabolic disorder through pregnancy. Gestational diabetes raises to changeable grades of glucose intolerance, which seems or is first identified through pregnancy. This type of diabetes is related with increased request for health facilities and a important economic load on society. It is also related with short- and long-term consequences through pregnancy and following years [3].

Pregnancy is a period of extraordinary alteration not physiologically but also neurologically. Though many maternal amendments emphasis on the cardiovascular, metabolic, and generative systems, the maternal brain undergoes similarly important renovation to funding the stresses of motherhood. These neurological variations are vital for the start of caregiving performances, attachment with the newborn, and adapting to the expressive and physical errands of parenthood [1].

Gestational diabetes(GD) usually appears during pregnancy, usually in the fifth or sixth month (i.e., between weeks 24 and 28) [2;3]. It then disappears after delivery, but it can sometimes persist throughout pregnancy. Gestational diabetes occurs as a result of elevated blood sugar levels due to changes in steroid hormones during pregnancy, which leads to a decreased response of the pancreas and its hormones due to hormones secreted by the placenta that counteract the action of insulin ([4;5]. Between 3% and 10% of pregnant women, or one in seven, develop it during pregnancy. Gestational diabetes usually disappears after delivery, but there is still concern that it could recur in the next pregnancy. A woman may develop type 2 diabetes due to repeated high blood sugar levels during each pregnancy[6;7].

Type 2: This type occurs when the mother has diabetes before pregnancy. It is a dangerous type for both the mother and the fetus [8;9]. The mother must be careful at this stage before considering pregnancy and during pregnancy until the baby is born [10]. During pregnancy, the body's need for insulin increases more than normal, and this need increases as the months of pregnancy progress until delivery [11]. The mother is treated with this condition by gradually increasing the amount of insulin, especially if she was taking insulin before pregnancy. This way, she can have a normal pregnancy and give birth to a healthy fetus [12;13].

Lifestyle variations are crucial in the supervision of gestational diabetes. First-line treatment in GD is medicinal nutrition treatment, together with weight management and physical commotion [14,15]. It has been advised that lifestyle alteration unaided is enough to control blood glucose in 70–85% of the women that were identified with GD [5]. How the diet should be collected for women with GD is a composite substance and still not totally established [16].

Lipid and triglyceride levels rise in pregnancy and do not pose difficulties for greatest women. Nevertheless, pregnancy-specific features and genetic aberrations, especially mutations, may affect in supraphysiological hypercholesterolaemia (HC) and simple hypertriglyceridaemia (sHTG). Cholesterol and vital fatty acids are important for usual fetal growth[17;18].The metabolic environment, ensuing from irregular glucose tolerance through pregnancy and its sequelae, exercises a mainly important effect on fetal development and, therefore, on the birth weight and fat physique of the newborn infants.[19,20;21].

Human chorionic gonadotropin (hCG) is a glycoprotein hormone produced by syncytiotrophoblast cells in humans or hominins during pregnancy. This hormone functions similarly to luteinizing hormone (LH), facilitating ovulation synchronization and assisting early pregnancy by promoting follicular maturation, corpus luteum function, and embryo implantation [22;23]. Cell signalling mechanisms play a crucial role in regulating glucose and electrolyte balance during pregnancy. Serum-glucocorticoid-induced kinase 1 (SGK1) is essential for cellular metabolism and ion transport, which may indirectly influence maternal adaptations throughout pregnancy [24]. This study aimed the estimation of several biomarkers (blood sugar, urea, creatinine, Serum Na+, Cl-, and K+) levels in pregnant women and non-pregnant women.

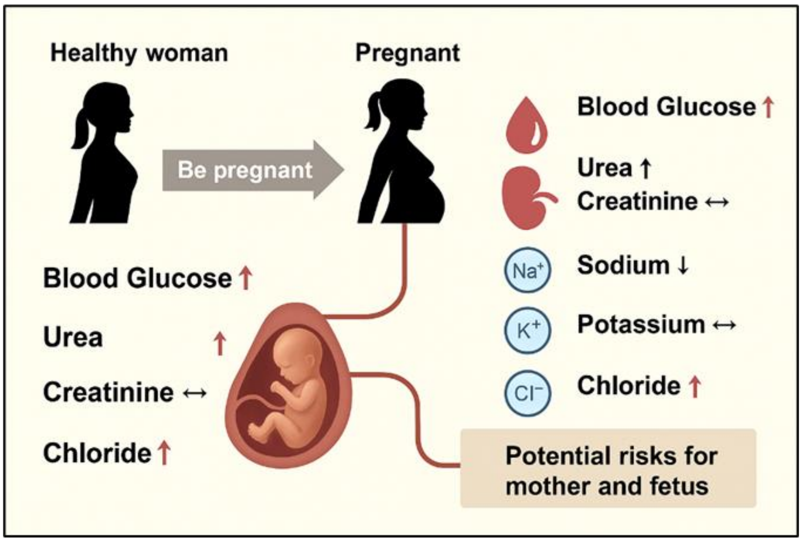

Figure 1. Physiological and biochemical changes during pregnancy in comparison to healthy women. Pregnancy is linked to elevated blood glucose, urea, and chloride levels, reduced sodium, and no notable alteration in creatinine and potassium. These alterations indicate maternal metabolic and renal adaptations that may provide potential dangers for both the mother and foetus.

Experimental Section

Collection of Blood Samples

Samples were collected from Kirkuk General Hospital's Laboratories. A number of 100 blood samples were collected from the vein, 50 samples were taken from pregnant women right before their labor, the other 50 samples were collected from mature women with no pregnancy as the control group. Blood was left to coagulate and then centrifuged to collect the serum for the next estimation steps. This research was conducted in the period between January 2024 and April 2024.

Methods

Blood glucose level was estimated following the enzymatic GOD-POD (glucose oxidase-peroxidase) method [25]. Urea was estimated using the enzymatic and colorimetric GOD-POD (Glucose Oxidase-Peroxidase) method [26]. The kinetic alkaline picrate (Jaffe) method has been utilized to estimate the creatinine level. Blood electrolytes were determined using the Jaffe method according to the literature [27].

Statistical Analysis

All data have been analyzed using the GraphPad Prism 8.0.2 program. The values of the studied parameters were expressed as (Mean ± SD).

Results and discussions

Table 1. Comparison of blood glucose, urea, creatinine, and electrolyte levels between pregnant and nonpregnant women.

Parameters	Pregnant Women (n=50)	Non-pregnant women (n=50)	P value
Blood Glucose (mg/dL)	164.86 ± 9.47	93.96 ± 7.23	P < 0.05
Blood Urea (mg/dL)	50.04 ± 10.89	34.79 ± 1.25	P < 0.05
Serum Creatinine (mg/dL)	0.98 ± 0.196	3.04 ± 1.64	0.278
Serum Na^+ (mEq/L)	139.50 ± 5.14	143.49 ± 18.67	P < 0.05
Serum K^+ (mmol/L)	3.68 ± 0.21	3.79 ± 0.41	0.239
Serum Cl^- (mmol/L)	109.05 ± 8.20	101.24 ± 7.35	P < 0.05

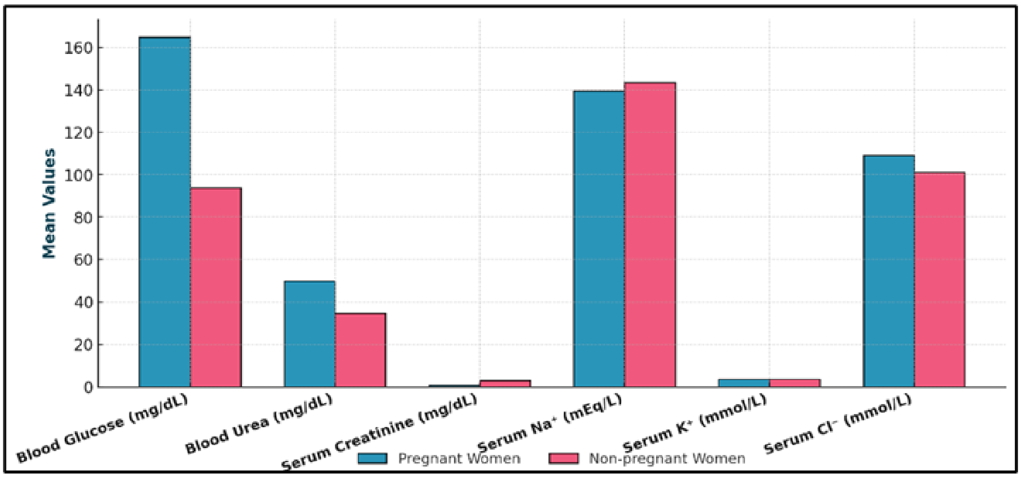

Figure 2. Comparison of biochemical parameters between pregnant and non-pregnant women.

Discussions

Table 1 compares the amounts of several blood components between pregnant and non-pregnant women. Pregnant individuals had elevated levels of blood glucose, blood urea, serum potassium, and serum chloride, alongside reduced levels of creatinine and serum sodium. Nonetheless, there was no notable disparity in blood creatinine and serum potassium levels between the two cohorts.

The metabolic alterations noted during pregnancy are, in part, analogous to other physiological stress states, including glutathione and malondialdehyde, which signify heightened free radical generation and modified antioxidant defenses [28]. Thus, pregnancy is associated with strenuous physical activity, which involves increased oxidative and inflammatory responses, potentially leading to marked metabolic changes. Furthermore, mitochondria are considered a key component of maternal-fetal energy metabolism. [29] This underscores the potential impact of mitochondrial bioenergetics on biochemical changes in glucose and electrolyte balance during pregnancy.

Blood glucose levels are markedly elevated in pregnant women relative to non-pregnant women (164.86 ± 9.47 vs. 93.96 ± 7.23 mg/dL, $P < 0.05$). The temporary rise in blood glucose levels during pregnancy is caused by steroid hormones, which are elevated during pregnancy and suppress the pancreas's sugar response and its associated hormones [30]. They also help secrete high levels of sugar from the liver into the blood, preventing it from entering tissue cells. Consequently, it rises as it remains in the blood [31]. Although gestational diabetes (GD) disappears after delivery as hormone levels return to normal, it is essential to monitor it, as it is a serious matter, as women with gestational diabetes are more susceptible to developing type 2 diabetes later on [32]. Factors like increased body fat percentage and other maternal characteristics can influence the development of hyperglycemia during pregnancy [32]. Factors like increased body fat percentage and other maternal characteristics can influence the development of hyperglycemia during pregnancy [33].

Blood urea levels are markedly elevated in pregnant women relative to non-pregnant women (50.04 ± 10.89 vs. 34.79 ± 1.25 mg/dL, $P < 0.05$). The lower blood urea levels in pregnant women are primarily due to increased renal blood flow and GFR, expanded plasma volume, increased protein synthesis, hormonal changes, and the fetus's demand for nitrogen. These physiological adaptations ensure that both the mother and the developing fetus maintain optimal metabolic balance during pregnancy [34]. A study comparing serum urea levels in pregnant and non-pregnant women revealed that pregnant women had considerably lower serum urea levels than their non-pregnant counterparts [35]. Another study conducted on pregnant women attending antenatal clinics reported a progressive decrease in urea levels as pregnancy advanced, with significantly lower values observed in the third trimester compared to non-pregnant women serving as the control group [36].

Serum creatinine was non-significantly decreased in pregnant women (0.98 ± 0.196 mg/dL) compared to non-pregnant women (3.04 ± 1.64 mg/dL) ($P=0.278$). The findings align with research by Kang et al. [37], indicating that serum creatinine levels often decline throughout pregnancy as a result of physiological glomerular hyperfiltration that occurs with advancing gestational age. Wiles et al. elucidated that in a typical pregnancy, augmented blood volume and renal function result in heightened filtration of creatinine from the bloodstream into the urine. In cases of renal impairment due to pre-eclampsia, the creatinine clearance value decreases [38].

The levels of serum sodium (Na^+) are significantly lower in pregnant women compared to non-pregnant women (139.50 ± 5.14 vs. 143.49 ± 18.67 mEq/L, $P < 0.05$). However, there is no significant difference in the levels of serum potassium (K^+) between pregnant and non-pregnant women (3.68 ± 0.21 vs. 3.79 ± 0.41 mmol/L, $P = 0.239$). The levels of serum chloride (Cl^-) are significantly higher in pregnant women compared to non-pregnant women (109.05 ± 8.20 vs. 101.24 ± 7.35 mEq/L, $P < 0.05$). Compared to other results by Otoikhila and Serik, the results of Na^+ have shown slightly elevated levels of Na^+ in the first, second and third trimester of the pregnancy in comparison to the non-pregnant women [39], which corresponds the findings in this article.

Literature have shown either a slight or a progressive increase in the concentration of serum K^+ in pregnant women compared to non-pregnant women. This increase could be attributed to some factors including the increased requirement for potassium during the fetal growth and the proper fetal development [40] as well as the protein synthesis, these factors can affect the level of serum K+ in women with pregnancy [41].

A non-significant decrease has been seen in Cl- concentration (109.05 ± 8.20, 101.24 ± 7.35 mmol/L, P = 0.239) for pregnant and non-pregnant women respectively. Lower serum Cl- have been reported in pregnant women compared to non-pregnant women according to the literature. Mohammed and Inuwa have shown that low serum Cl- (below 95 mmol/L) in younger pregnant women (aged 15 to 24 years) [42]. Another study illustrated that metabolic alkalosis, vomiting, diarrhea, congestive heart failure could significantly lower the serum Cl- in pregnant women [43].

Blood glucose levels are strongly correlated with serum urea in pregnant women, and poorly controlled blood sugar can elevate serum urea, increasing the risk of diabetic nephropathy. Although the correlation between blood glucose and serum creatinine is weaker, creatinine levels are generally higher in pregnant diabetic patients due to higher muscle mass and protein intake [44,45]. Multivariate analysis reveals that increased fasting blood glucose, glycated hemoglobin, urea, and creatinine significantly raise the odds of renal and cardiovascular complications in pregnant women with diabetes [46]. Normal pregnancy is associated with a progressive decrease in serum urea due to hemodilution and increased GFR, and serum creatinine also significantly decreases compared to non-pregnant women [47;48;49]. Mean levels of sodium, potassium, and chloride remain within normal ranges during pregnancy, though their relationships with other parameters were not specifically examined [50].

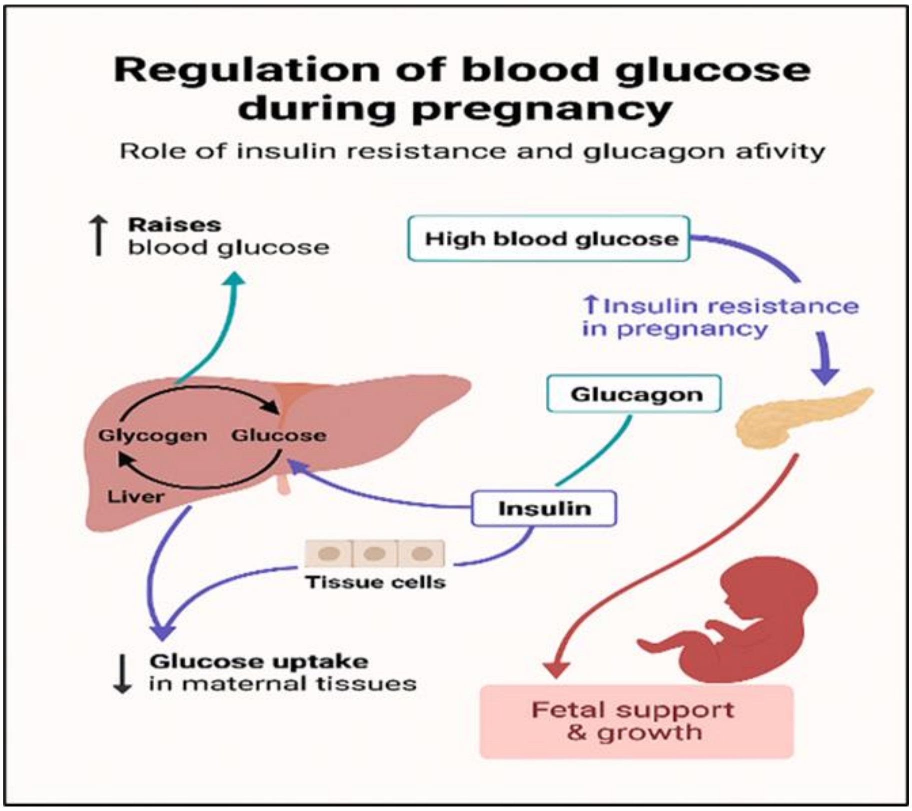

Figure 3. Regulation of blood glucose during pregnancy. Pregnancy is linked to heightened insulin resistance, resulting in higher maternal blood glucose levels. Glucagon facilitates glycogenolysis in the liver, whereas insulin enhances glucose absorption in maternal tissues. Decreased maternal glucose absorption guarantees enhanced glucose availability for the foetus, therefore facilitating foetal growth and development.

It is noteworthy to mention that this study has been conducted only on a specific group of pregnant women who have shown significant increased blood glucose, all other parameters were tested for the targeted group to compare the results to non-pregnant women.

Conclusion

This study compared blood components between pregnant and nonpregnant women, finding higher levels of blood glucose, urea, and serum chloride, but lower serum sodium levels in pregnant women. Serum creatinine and potassium levels showed no significant differences. Elevated glucose and greater glycemic variability are due to physiological changes and higher hyperglycemia risk. Increased blood urea aligns with enhanced renal function and protein synthesis. Lower sodium is likely from hemodilution and hormonal changes, while higher chloride suggests altered electrolyte balance. Potassium homeostasis is maintained. These results highlight the need for monitoring during pregnancy and further research to confirm findings and explore mechanisms.

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REFERENCES

[1] Y. T. Chiang, J. H. Chen, and K. H. Chen, "The molecular and cellular basis of physiological changes in pregnancy and its implications in neurologic and ophthalmic pathologies," *Int. J. Mol. Sci.*, vol. 26, no. 11, p. 5220, 2025, doi: 10.3390/ijms26115220.

[2] A. H. Rad, A. F. Yaseri, and G. Chatrnour, "The influence of maternal physical activity on gestational diabetes mellitus," *J. Renal Endocrinol.*, vol. 10, no. 1, p. e25141, 2024, doi: 10.34172/jre.2024.25141.

[3] H. Rashidi, K. Kalantari, H. Shahbazian, and S. Nouhjah, "The relationship between fasting plasma glucose in the first trimester of pregnancy and the incidence of gestational diabetes in Iran," *Diabetes Metab. Syndr. Clin. Res. Rev.*, vol. 15, no. 4, p. 102193, 2021, doi: 10.1016/j.dsx.2021.102193.

[4] R. Deori, C. Deori, J. Das, and T. Kutum, "Metabolic changes and biochemical monitoring during pregnancy," in *Current Trends in the Diagnosis and Management of Metabolic Disorders*, CRC Press, 2023, pp. 29–47.

[5] M. K. Bohn and K. Adeli, "Physiological and metabolic adaptations in pregnancy: importance of trimester-specific reference intervals to investigate maternal health and complications," *Crit. Rev. Clin. Lab. Sci.*, vol. 59, no. 2, pp. 76–92, 2022, doi: 10.1080/10408363.2021.1978923.

[6] E. C. Francis, D. Dabelea, B. M. Ringham, K. A. Sauder, and W. Perng, "Maternal blood glucose level and offspring glucose–insulin homeostasis: what is the role of offspring adiposity?," *Diabetologia*, vol. 64, pp. 83–94, 2021, doi: 10.1007/s00125-020-05294-2.

[7] J. Lin, H. Jin, and L. Chen, "Associations between insulin resistance and adverse pregnancy outcomes in women with gestational diabetes mellitus: a retrospective study," *BMC Pregnancy Childbirth*, vol. 21, pp. 1–10, 2021, doi: 10.1186/s12884-021-04006-x.

[8] T. L. Hernandez, J. E. Friedman, and L. A. Barbour, "Insulin resistance in pregnancy: implications for mother and offspring," in *Insulin Resistance: Childhood Precursors of Adult Disease*, 2020, pp. 67–94, doi: 10.1007/978-3-030-25057-7_5.

[9] K. Beardsall and A. L. Ogilvy-Stuart, "Developmental physiology of carbohydrate metabolism and the pancreas," in *Maternal-Fetal and Neonatal Endocrinology*, Academic Press, 2020, pp. 587–597, doi: 10.1016/B978-0-12-814823-5.00034-9.

[10] Y. Y. Sun, J. Juan, Q. Q. Xu, R. N. Su, J. E. Hirst, and H. X. Yang, "Increasing insulin resistance predicts adverse pregnancy outcomes in women with gestational diabetes mellitus," *J. Diabetes*, vol. 12, no. 6, pp. 438–446, 2020, doi: 10.1111/1753-0407.13013.

[11] R. A. Karasneh, F. H. Migdady, K. H. Alzoubi, S. I. Al-Azzam, Y. S. Khader, and M. B. Nusair, "Trends in maternal characteristics, and maternal and neonatal outcomes of women with gestational diabetes: A study from Jordan," *Ann. Med. Surg.*, vol. 67, p. 102469, 2021, doi: 10.1016/j.amsu.2021.102469.

[12] W. Bao et al., "Long-term risk of type 2 diabetes mellitus in relation to BMI and weight change among women with a history of gestational diabetes mellitus: a prospective cohort study," *Diabetologia*, vol. 58, pp. 1212–1219, 2015, doi: 10.1007/s00125-015-3537-4.

[13] M. V. Diaz-Santana, K. M. O'Brien, Y. M. M. Park, D. P. Sandler, and C. R. Weinberg, "Persistence of risk for type 2 diabetes after gestational diabetes mellitus," *Diabetes Care*, vol. 45, no. 4, pp. 864–870, 2022, doi: 10.2337/dc21-1430.

[14] J. M. Yamamoto et al., "Gestational diabetes mellitus and diet: a systematic review and meta-analysis of randomized controlled trials examining the impact of modified dietary interventions on maternal glucose control and neonatal birth weight," *Diabetes Care*, vol. 41, no. 7, pp. 1346–1361, 2018, doi: 10.2337/dc18-0102.

[15] L. Rasmussen et al., "Diet and healthy lifestyle in the management of gestational diabetes mellitus," *Nutrients*, vol. 12, no. 10, p. 3050, 2020, doi: 10.3390/nu12103050.

[16] H. D. McIntyre, P. Catalano, C. Zhang, G. Desoye, E. R. Mathiesen, and P. Damm, "Gestational diabetes mellitus," *Nat. Rev. Dis. Primers*, vol. 5, no. 1, p. 47, 2019, doi: 10.1038/s41572-019-0098-8.

[17] E. P. Davis and A. J. Narayan, "Pregnancy as a period of risk, adaptation, and resilience for mothers and infants," *Dev. Psychopathol.*, vol. 32, no. 5, pp. 1625–1639, 2020, doi: 10.1017/S0954579420001121.

[18] C. Li et al., "Comparison of the effectiveness and safety of insulin and oral hypoglycemic drugs in the treatment of gestational diabetes mellitus: a meta-analysis of 26 randomized controlled trials," *Gynecol. Endocrinol.*, vol. 38, no. 4, pp. 303–309, 2022, doi: 10.1080/09513590.2021.2015761.

[19] M. Lende and A. Rijhsinghani, "Gestational diabetes: overview with emphasis on medical management," *Int. J. Environ. Res. Public Health*, vol. 17, no. 24, p. 9573, 2020, doi: 10.3390/ijerph17249573.

[20] S. Hao et al., "Changes in pregnancy-related serum biomarkers early in gestation are associated with later development of preeclampsia," *PLoS One*, vol. 15, no. 3, p. e0230000, 2020, doi: 10.1371/journal.pone.0230000.

[21] D. Mavreli, M. Theodora, and A. Kolialexi, "Known biomarkers for monitoring pregnancy complications," *Expert Rev. Mol. Diagn.*, vol. 21, no. 11, pp. 1115–1117, 2021, doi: 10.1080/14737159.2021.1971078.

[22] M. Chandra and A. A. Paray, "Focus: Pregnancy and child development: Natural physiological changes during pregnancy," *Yale J. Biol. Med.*, vol. 97, no. 1, p. 85, 2024, doi: 10.59249/JTIV4138.

[23] J. Li, X. Zhu, W. Zhu, L. Li, H. Wei, and S. Zhang, "Research progress on the impact of human chorionic gonadotropin on reproductive performance in sows," *Animals*, vol. 14, no. 22, p. 3266, 2024, doi: 10.3390/ani14223266.

[24] P. H. Høj, J. Møller-Sørensen, A. L. Wissing, and F. A. A. Alatraktchi, "Electrochemical biosensors for monitoring of selected pregnancy hormones during the first trimester: A systematic review," *Talanta*, vol. 258, p. 124396, 2023, doi: 10.1016/j.talanta.2023.124396.

[25] W. I. Abo-Elenien, S. G. Badawy, O. Abouelenin, F. K. Hussein, and S. Kumari, "Role of sgk1 in cancer: a bibliometric analysis from 2013 to 2023," *Egypt. J. Med. Hum. Genet.*, vol. 25, p. 142, 2024, doi: 10.1186/s43042-024-00611-5.

[26] S. P. Sharma, A. P. Anjankar, and A. Kale, "Comparison of glucose levels using glucometer and GOD-POD method in diabetic patients," *Int. J. Clin. Biochem. Res.*, vol. 4, no. 1, pp. 6–10, 2017, doi: 10.18231/2394-6377.2017.0002.

[27] R. S. T. Bowden and M. O. J. McCarthy, "A comparison of some clinical laboratory methods of estimating the urea level in the blood of the dog," *J. Small Anim. Pract.*, vol. 6, no. 4, pp. 257–260, 1965, doi: 10.1111/j.1748-5827.1965.tb04337.x.

[28] V. Albert, A. Subramanian, K. Rangarajan, and R. M. Pandey, "Agreement of two different laboratory methods used to measure electrolytes," *J. Lab. Physicians*, vol. 3, no. 2, pp. 104–109, 2011, doi: 10.4103/0974-2727.86843.

[29] H. A. Salman, F. K. Hussein, and S. J. Abdulrahman, "A comparison of glutathione and malondialdehyde concentrations in athletes engaged in certain sports," *Thamar Univ. J. Nat. Appl. Sci.*, vol. 9, no. 1, pp. 39–42, 2024, doi: 10.59167/tujnas.v9i1.2053.

[30] M. C. Al-Noaemi and M. H. F. Shalayel, "Pathophysiology of gestational diabetes mellitus: the past, the present and the future," *Gestational Diabetes*, vol. 6, pp. 91–114, 2011.

[31] R. H. Ali, F. K. Hussein, D. M. Nasar, and A. A. S. Salem, "The relevance of mitochondrial DNA mutation in human diseases and forensic sciences," *Al-Nahrain J. Sci.*, vol. 28, no. 1, pp. 96–106, 2025, doi: 10.22401/ANJS.28.1.11.

[32] A. Nigam, N. Varun, S. Sharma, Y. P. Munjal, and A. Prakash, "Glycaemic profile in the second and third trimesters of normal pregnancy compared to non-pregnant adult females," *Obstet. Med.*, vol. 13, no. 1, pp. 30–36, 2020, doi: 10.1177/1753495X18807549.

[33] H. D. McIntyre, J. Fuglsang, U. Kampmann, S. Knorr, and P. Ovesen, "Hyperglycemia inpregnancy and women's health in the 21st century," *Int. J. Environ. Res. Public Health*, vol. 19, no. 24, p. 16827, 2022, doi: 10.3390/ijerph192416827.

[34] S. S. Msollo, H. D. Martin, A. W. Mwanri, and P. Petrucka, "Prevalence of hyperglycemia in pregnancy and influence of body fat on development of hyperglycemia in pregnancy among pregnant women in urban areas of Arusha region, Tanzania," *BMC Pregnancy Childbirth*, vol. 19, pp. 1–9, 2019, doi: 10.1186/s12884-019-2463-8.

[35] C. Higgins, "Urea and the clinical value of measuring blood urea concentration," *Acutecaretesting.org*, vol. 22, pp. 1–6, 2016.

[36] T. Forrester, A. V. Badaloo, C. Persaud, and A. A. Jackson, "Urea production and salvage during pregnancy in normal Jamaican women," *Am. J. Clin. Nutr.*, vol. 60, no. 3, pp. 341–346, 1994, doi: 10.1093/ajcn/60.3.341.

[37] E. Tesfa et al., "Maternal serum uric acid, creatinine and blood urea levels in the prediction of pre-eclampsia among pregnant women attending ANC and delivery services at Bahir Dar city public hospitals, northwest Ethiopia: A case-control study," *Heliyon*, vol. 8, no. 10, 2022.

[38] J. Kang et al., "Gestational age-specific serum creatinine can predict adverse pregnancy outcomes," *Sci. Rep.*, vol. 12, no. 1, p. 11224, 2022, doi: 10.1038/s41598-022-15450-w.

[39] K. Wiles, K. Bramham, P. T. Seed, C. Nelson-Piercy, L. Lightstone, and L. C. Chappell, "Serum creatinine in pregnancy: a systematic review," *Kidney Int. Rep.*, vol. 4, no. 3, pp. 408–419, 2019, doi: 10.1016/j.ekir.2018.10.015.

[40] O. C. Otoikhila and S. A. Seriki, "A comparative study of serum sodium and potassium levels across the three trimesters of pregnancy," 2023.

[41] J. Tomala, A. Jendryczko, and P. Kossowski, "Serum potassium levels during pregnancy," *Zentralbl. Gynakol.*, vol. 116, no. 5, pp. 285–288, 1994.

[42] M. I. Mohammed and Y. Inuwa, "Determination of serum chloride ion concentration in pregnant women from Minjibir local government area, Kano State Nigeria," *Chemsearch J.*, vol. 3, no. 1, pp. 19–23, 2012.

[43] J. B. Wallach, *Interpretation of Diagnostic Tests*. Philadelphia, PA, USA: Lippincott Williams & Wilkins, 2007.

[44] S. A. Bamanikar, A. A. Bamanikar, and A. Arora, "Study of serum urea and creatinine in diabetic and nondiabetic patients in a tertiary teaching hospital," *J. Med. Res.*, vol. 2, no. 1, pp. 12–15, 2016.

[45] D. A. Mahmoud and S. S. Saleh, "Determination of the level of homocysteine and antioxidants in the blood serum of women with gestational diabetes," *Solid State Technol.*, vol. 64, no. 1, pp. 1129–1135, 2021.

[46] A. A. Choudhry, S. G. Rajeshwari, A. Gururaja, K. Prabhu, and N. Ravishankar, "Retrospective correlation of fasting glucose and glycated hemoglobin with serum electrolytes, urea and creatinine in diabetic patients," *Asian Pac. J. Trop. Dis.*, vol. 4, pp. S385–S388, 2014, doi: 10.1016/S2222-1808(14)60475-8.

[47] D. M. Kava and H. D. Lad, "Comparative study of assessment of renal function in pregnant women with non-pregnant women," *MedPulse Int. J. Biochem.*, vol. 11, no. 3, pp. 90–95, 2019.

[48] A. T. Bawah et al., "Effect of pregnancy on the metabolism of creatinine, urea and uric acid among pregnant women at the Volta Regional Hospital," 2018.

[49] D. S. Saleh, I. H. Saleh, and D. A. Mahmood, "Evaluation of some hormones and trace elements in renal failure patients," *Open Access Res. J. Chem. Pharm.*, vol. 6, no. 2, pp. 001–010, 2025, doi: 10.53022/oarjcp.2025.

[50] O. Akinloye, O. M. Obikoya, A. I. Jegede, D. P. Oparinde, and A. O. Arowojolu, "Cortisol plays central role in biochemical changes during pregnancy," *Int. J. Med. Biomed. Res.*, vol. 2, no. 1, pp. 3–12, 2013.