Atomic Spectroscopy
Article

Assessment of Serum Hyperglycosylated hCG Marker in a Pregnant Women with Pre-eclampsia

Abeer Saleh Hasan[1], Prof.Dr.Manal Kamal Rasheed[2], Ass.Prof.Dr.Najmah Mahmood Meran[3]
[1]Northern technical university- college of health and medical techniques- aldoor- department of optics– lecturer
[2]baghdad university – medicine college- professor
[3]assistance professors

ABSTRACT: The background: A possible biomarker for the early detection of pre-eclampsia is the level of hyperglycosylated human chorionic gonadotropin (hCG-h) during the first trimester. To confirm pre-eclampsia (PE), as in studies in the first trimester, and its subtypes early-onset, late-onset, severe, and mild PE in case-control research, this study sought to assess the performance of clinical risk variables, mean arterial pressure (MAP), and hCG-h in the third trimester.
Aim of study: Pre-eclampsia patients in the third trimester are being studied to see how well clinical risk indicators for mean arterial pressure (MAP) and hyperglycosylated hCG perform as pre-eclampsia predictors.
Patient and Method: This case control study was included 48 (16 patient excluded because chronic hypertension and GDM) pregnant women with preeclampsia and 44 (12 control was excluded because anemia and GDM) Normal pregnant women aged 20-40y. samples were collected from Baghdad teaching hospital period from June 2021 to September 2021.The blood sample directly transferred into disposable gel tube and left for 15-30 minutes at 37 ˚C for clot formation to evaluate H-HCG, method of measurement with ELISA Kit.
Result: The outcomes demonstrated that the level of serum Hyperglycosylated HCG in preeclampsia patient 238.53 ± 59.35 were significantly decreases (P value < 0.05) as comparison to healthy pregnant women.
Conclusion: Serum Hyperglycosylated HCG were significantly decreased among PE women than healthy women, so it can be used as a biomarker to predict preeclampsia.

Keywords: Pre-eclampsia in pregnant women,
Hyperglycosylated Human Chorionic Gonadotropin and preeclampsia,
High Albumin in Urine in association with preeclampsia,
Gestational Hypertension with exclusion of chronic hypertension and gestational diabetes melletus,
Urgent pregnant women comes to emergency and converted to cesarian section and Postpartum Complication.

INTRODUCTION

Pre-eclampsia is a complicated disorder that affects several systems during pregnancy, and it has unknown root causes. It affects 2 to 8% of pregnancies and contributes significantly to maternal and fetal morbidity and mortality globally [(10), (1)]. Etiology preeclampsia has a complex etiology that combines genetic, immunologic, and environmental components. Preeclampsia manifests as a wide variety of clinical signs, demonstrating the condition's heterogeneous nature [(23)].

Acute breathing difficulties (ARDS), severe preeclampsia with pulmonary edema, and eclampsia were the main reasons why the patients were transferred to the Critical Care Unit (CCU) [(31).]

Early detection of women who are at a higher risk of developing pre-eclampsia is crucial for planning their prenatal care and implementing preventive strategies.
clinical indicators of risk factors include, for example [(24)]:
syndrome caused by an antiphospholipid antibody.
a history of preeclampsia ,
persistent hypertension
Pregnancy-related diabetes

These risk variables have been used to identify pregnant women who are more likely to experience pre-eclampsia in the early stages of their pregnancies [(7)]. To effectively implement preventative strategies, a predictive test with high sensitivity and positive predictive value, incorporating maternal risk factors, biomarkers, and biophysical parameters, is needed. [(32)], [(20)]. Regardless of gestational age, the World Health Organization (WHO) defines intrauterine fetal death as the death of a fetus before it is entirely ejected or removed from the mother. This determination is made based on the absence of breathing or any signs of life following separation from the mother [(5)].

The placenta produces the hormone human chorionic gonadotropin (hCG), which has undergone intensive research for more than a century and is acknowledged as the first protein connected to pregnancy. Despite having a primary function in encouraging corpus luteal cells to produce more progesterone, hCG is not a single physiologically active molecule. Instead, it is made up of a collection of at least five varieties, each of which is produced by a separate cell and has a unique purpose. The hyperglycosylated version of hCG (H-hCG) is one of these variations and is essential for trophoblast invasion, placental development, and fetal growth. [21], [22].

During trophoblast invasion, H-hCG promotes the conversion of spiral artery walls into larger-diameter, low-resistance vessels, and the invasion of extravillous cells known as cytotrophoblast cells into the decidua. When trophoblast invasion and uterine artery remodeling are impaired, it leads to insufficient placental perfusion and restricted fetal growth, as the growth of the fetus heavily relies on nutrient availability. New methods for controlling and managing fetal growth limitation may be identified by comprehending the function of H-hCG in the development of the placenta. [8].

Human chorionic gonadotropin (hCG) that has been glycosylated more heavily, or has more sugar molecules linked to it, is known as hyperglycosylated hCG. Preeclampsia is a pregnancy condition that commonly develops after 20 weeks of gestation and is characterized by elevated blood pressure and organ damage [19].

Hyperglycosylated hCG is a term used to describe human chorionic gonadotropin (hCG) that has undergone increased glycosylation or has more sugar molecules attached to it. Preeclampsia is a pregnancy disorder marked by high blood pressure and organ damage that often appears after 20 weeks of gestation. [11].

The extracellular signal-regulated protein kinases 1 and 2 (Erk1/2) pathway is activated by the hCG signaling in endometrial stromal cells (ESCs) via the exchange protein Epac, which is activated by cyclic AMP (cAMP). This activation results in a temporary increase in the expression of progesterone receptor (PR) transcripts and proteins, as well as enhancing its function in transcription. [4]. The Ras-Raf-MEK-ERK signal transduction cascade depends on the protein serine/threonine kinase enzyme identified as Extracellular Signal-Regulated Kinases (ERK). A wide range of functions, including cell adhesion, cell cycle progression, cell migration, cell survival, differentiation, metabolism, proliferation, and transcription, are significantly regulated by this cascade. Additionally, ERK also plays a role in the process of mammalian embryo implantation. There is a suggestion that Osteopontin (OPN) regulates FoxM1, a protein involved in endometrial proliferation for the establishment of endometrial receptivity. Through the signaling pathway of the extracellular regulated protein kinases (ERK 1, and 2), the protein kinase B (PKB, AKT), and p 38 mitogen-activated protein kinase (p38MAPK, p38), FoxM1 is controlled by OPN. When the activities of ERK 1 and 2, AKT, and p38 are inhibited, the expression and localization of FoxM1 induced by OPN are suppressed. [13].

The emergence of hemochorial placentation in primates is intimately related to the development of both regular chorionic gonadotropin (hCG) and hyperglycosylated hCG. Previous studies on humans have revealed that regular HCG promotes the growth of spiral arteries, while hyperglycosylated HCG controls the invasion of trophoblast cells during implantation [22]. However, the advanced form of hemochorial placentation in humans is associated with a relatively high rate of pregnancy failures. This can be attributed to inadequate implantation, as the demanding nature of human embryo implantation relies on the proper production of hyperglycosylated HCG [21]. Failures in the invasion of hemochorial placentation result in oxygen deprivation, which can lead to preeclampsia and eclampsia, unique obstetric complications in humans. The lack of signaling from hyperglycosylated HCG is probably to blame for these problems. In conclusion, it is proposed that the evolutionary causes of these obstetric difficulties in humans are low quantities of regular HCG and hyperglycosylated HCG molecules. [18], [15].

A crucial diagnostic need for pre-eclampsia syndrome is hypertension. Preeclampsia patients have higher systemic vascular resistance and afterload than healthy pregnant women do, and their cardiac output and intravascular volumes are also lower. These factors all contribute to preeclampsia-related hypertension. This alteration is a result of numerous things. Conduit artery compliance declines, and the typical drop in nocturnal blood pressure is either reduced or eliminated [25].

Our aim in the present study was to determine HCG-H in pregnant women with preeclampsia. We aimed to ensure HCG-H could predict pre-eclampsia in third trimester when combined with other markers depending on previous recommendation.

MATERIALS AND METHODS

Study Design case control of patients with preeclampsia: The patients with preeclampsia diagnosed by hypertension, albumin urine, edema and seizure in third trimester were divided into two groups Group A preeclampsia pregnant women (n=32) and Group B controlled pregnant women (n=32) Hyperglycosylated HCG (h-hCG) were evaluated after delivery with cesarean section. The study was done in Baghdad teaching hospital Baghdad /Iraq, period from June 2021 to September 2021 and sample was previously diagnosed with preeclampsia in emergency unite.

Collecting blood samples: The blood samples were collected each group (preeclampsia patient and controlled pregnant women). About (8-5) ml of blood in test tubes and serum was obtained by Centrifuge at 3000 rpm / minute and kept at -20°C to determine h-hCG concentration that is measured by ELISA method.

Ethical Issues

This study was done in College of Medicine Department of Biochemistry, University of Baghdad.

Data analysis: To do the statistical analysis, SPSS version 27 was used. Continuous variables are provided as mean and standard deviation, while categorical variables are shown as frequencies and percentages. The correlation between categorical variables was determined using the Fisher's exact test and Pearson chi-square tests. The means of the two groups were compared using an independent sample t-test. When the study's variables weren't normally distributed, the Mann-Whitney test was utilized to compare two groups. Significant ROC was defined as a p-value 0.05.

RESULT AND DISCUSSION

Table 1: The mean±SD differences of systolic and diastolic blood pressure (mmHg) according to studded group including (Preeclampsia patients and controlled pregnant women). There were significant differences between means of systolic and diastolic blood pressure (mmHg) according to the controlled pregnant women.

Table 1: The mean±SD differences of systolic and diastolic blood pressure (mmHg) according to study group

Study variables	Study group	N	Mean ± SD	t-test	P-value
Systolic blood pressure (mmHg)	Preeclampsia	32	165.47 ± 23.40	**12.191**	**<0.001***
	Control group	32	112.66 ± 7.29		
Diastolic blood pressure (mmHg)	Preeclampsia	32	100.47 ± 14.67	**10.07**	**<0.001***
	Control group	32	72.03 ± 6.33		

*P value ≤ 0.05 was significant.

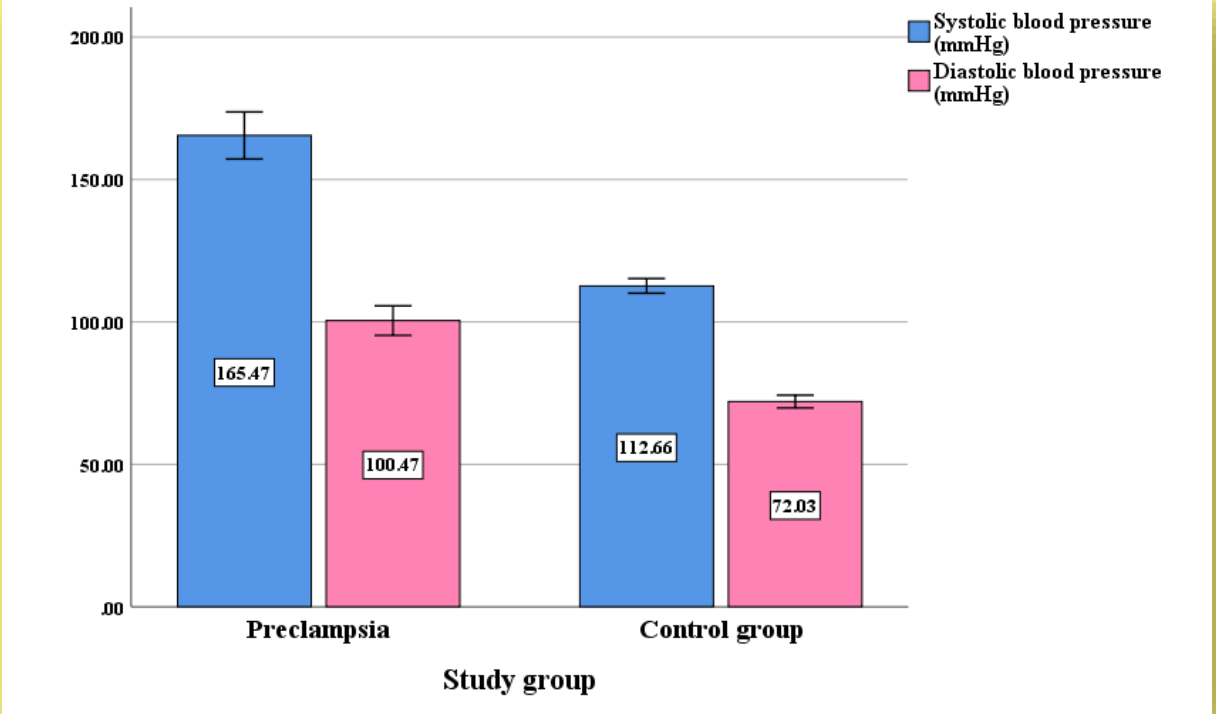

Figure 1: The mean differences of systolic and diastolic blood pressure (mmHg) according to study group (NO.=64, P<0.001*)

Hypertension serves as a critical diagnostic criterion for pre-eclampsia syndrome. In comparison to healthy pregnant women, increased systemic vascular resistance and afterload, combined with decreased cardiac output and intravascular volumes, are the main causes of hypertension in preeclampsia. This change is the result of numerous reasons. Conduit artery compliance decreases, and the regular drop in blood pressure during the night is reduced or eliminated. [9] [25]. Although the renin-angiotensin-aldosterone system (RAAS) is activated, preeclampsia results in decreased levels of renin, angiotensin II (Ang II), and aldosterone compared to a normal pregnancy, though they are still greater than nonpregnant people. Additionally, norepinephrine and Ang II sensitivity are increased [30]. reasons why preeclampsia can develop hypertensive problems despite having a reduced amount of RAAS components, are twofold. Firstly, in a normal pregnancy, reactive oxygen species downregulate AT1R. However, in preeclampsia, AT1R forms a heterodimer with bradykinin receptor B2, resulting in increased pressor effects of Ang II [29]. Second, placental hypoxia aids in the creation of circulating AT1R antibodies, which in turn improves vasoconstriction by activating endothelin-1, increasing sensitivity to circulating Ang II, and increasing placental production of sFlt-1 and sEng. [12].

A promising predictor of pre-eclampsia with early start is serum hyperglycosylated human chorionic gonadotropin (hCG-h), according to studies [17].

The downregulation of heme oxygenase-1 leads to a decrease in carbon monoxide generation, resulting in further increases in the release of sFlt-1 and sEng [6]. Blood pressure rises because of elevated peripheral vascular resistance, which is a result of soluble fms-like tyrosine kinase-1 (sFlt-1). To address the relative hemoconcentration in preeclampsia, the sympathetic nervous system, RAAS, and endothelin-1 are activated, which in turn intensifies vasoconstriction by increasing vasoconstrictors like thromboxane A1 and endothelins while decreasing vasodilators such as prostacyclin and nitric oxide [2], [6], [26]. Due to oxidative stress, these abnormalities cause endothelial dysfunction and prevent endothelium-dependent vasodilation. [28], [6]. An observational study conducted in the Netherlands, involving 205 women with preeclampsia, discovered that hypertension can persist for up to 2 years after delivery. The time needed for hypertension to subside was strongly connected with the severity of preeclampsia & the interval until delivery. Target organ damage from pre-eclampsia, such as heart failure, pulmonary edema, impaired renal function, and acute injury to the kidneys, as well as brain damage and stroke, is exacerbated by hypertension. [2], [27], [3]. Study findings reveal a significant association between intrauterine fetal death and preeclampsia, suggesting a heightened susceptibility to adverse pregnancy outcomes [5].

Table 2: The mean±SD differences of H-HCG (ng/ml) according to study group including (Preeclampsia patients and controlled pregnant women). There were significant differences between means of H-HCG (ng/ml) according to the study group.

Table 2: The mean±SD differences of H-HCG (ng/ml) according to study group

Study marker	Study group	N	Mean ± SD	t-test	P-value
H-HCG (ng/ml)	Preeclampsia	32	238.53 ± 59.35	**-2.113**	**0.039***
	Control group	32	274.14 ± 74.63		

*P value ≤ 0.05 was significant.

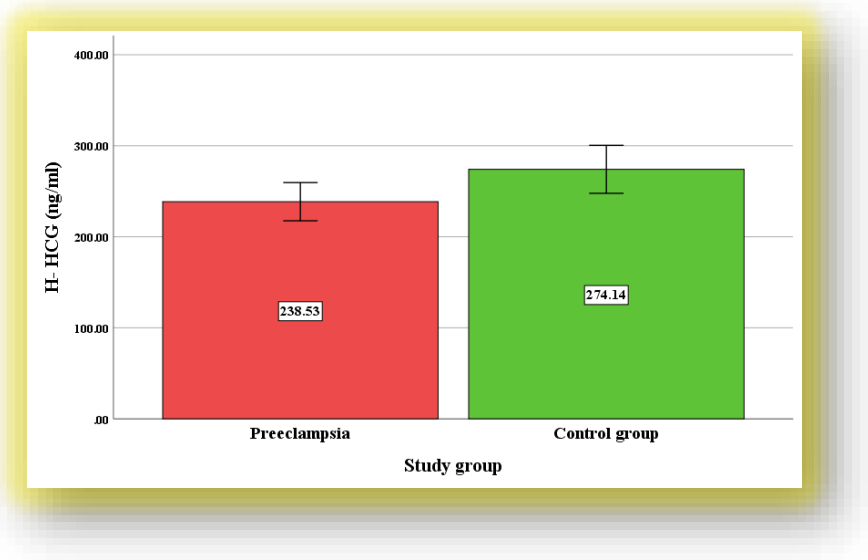

Figure 2: The mean differences of H-HCG (ng/ml) according to study group

(NO.=64, 0.039*)

Research suggests that hyperglycosylated hCG may play a role in the development and progression of preeclampsia. It is believed to have angiogenic properties, meaning it can affect the growth of blood vessels. In normal pregnancies, the balance between pro-angiogenic factors (promoting blood vessel growth) and anti-angiogenic factors (inhibiting blood vessel growth) is maintained. However, in preeclampsia, this balance is disrupted, leading to abnormal placental development and impaired blood vessel formation [(9)]. Hyperglycosylated hCG has been found to have increased expression in the placenta and maternal serum of women with preeclampsia. It is thought to contribute to the dysregulation of angiogenesis by influencing the production of pro-angiogenic and anti-angiogenic factors [(21)].

Another research is still needed to fully understand the exact mechanisms and significance of hyperglycosylated hCG in preeclampsia. However, it is a promising area of study that may provide insights into the pathophysiology and potential diagnostic or therapeutic approaches for preeclampsia in the future [(19)].

Table 3: The mean ± SD differences of study markers including (H- HCG (ng/ml) according to severity of Preeclampsia including (moderate and severe). There were no significant differences between means of study markers according to severity of Preeclampsia.

Table 3: The mean ± SD differences of study markers according to severity of Preeclampsia (NO.=32)

Study marker	Severity of Preeclampsia	N	Mean ± SD	t-test	P-value
H-HCG (mIU/ml)	Moderate	16	224.41 ± 69.17	-1.364	0.183
	Severe	16	252.65 ± 45.48		

The serum level of maternal β-hCG was non-significant raised in pre-eclampsia in comparison to controlled and parallel with the severity of pre-eclampsia, that's mean the maternal serum level of β-hCG plays one of the important roles in pathogenesis of pre-eclampsia and its severity [(14)].

Table 4: The mean±SD differences of study markers including (H- HCG (ng/ml),) according to albuminuria including (positive and negative). There were no significant differences between means of study markers according to albuminuria.

Table 4: The mean±SD differences of H- HCG (mIU/ml) according to albuminuria (N=32)

Study marker	albuminuria	N	Mean ± SD	t-test	P-value
H- HCG (mIU/ml)	Positive	27	240.02 ± 51.98	0.212	0.841
	Negative	5	230.46 ± 98.16		

An imbalance in angiogenic factors, which causes systemic endothelial dysfunction, is a hallmark of preeclampsia. Due to related disorders including albuminuria and hypertension, chronic renal disease may accelerate the development of preeclampsia. Additionally, renin-angiotensin-aldosterone and complement systems, as well as glycocalyx integrity, might be affected by chronic kidney disease [(31)]. By generating acute kidney injury, endothelial cell damage, and podocyte loss, preeclampsia can further develop into kidney disease. Preeclampsia may represent a sizable sex-specific risk factor for chronic renal disease, it is important to note. Future diagnostics and therapies that are advantageous for all women may be found by developing a mechanistic understanding of how chronic kidney disease raises the risk of preeclampsia. [(16)].

Table 1: The sensitivity and specificity of study markers to predict Preeclampsia.

Study markers	Cutoff value	AUC	Sensitivity	Specificity	P value

H-HCG (ng/ml)	**≤ 266.99**	**0.66**	**71.9%**	**59.4%**	**0.028***

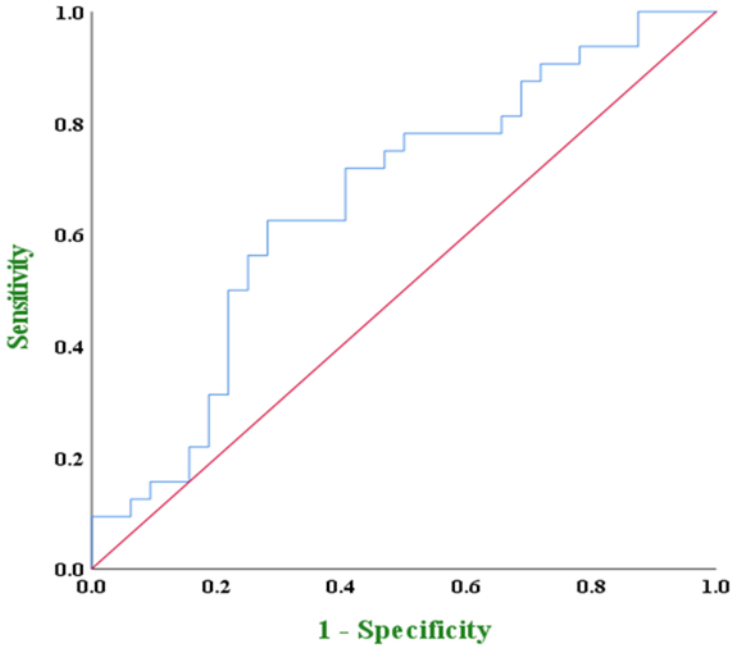

Figure 1: ROC curve for the sensitivity and specificity of H-HCG (ng/ml) to predict Preeclampsia.

CONCLUSION:

Our study found that lower level of hCG-h was associated with the late-onset PE and non-severe PE. Our study by using multivariate regression analyses for h-hCG the: cutoff was ≤ **266.99** AUC was 0.66, and sensitivity 71.9% at 59.4% specificity with p value of 0. 028.So it can be used as A biomarker to predict pre-eclampsia in assessment of other biomarker.

AUTHOR CONTRIBUTION

Study conception: Abeer Saleh Hasan, Dr. Manal Kamal Rasheed & Dr. Najmah M. Miran
Study design: Dr. Manal Kamal Rasheed & Dr. Najmah M. Miran
Acquisition of data analysis: Abeer Saleh Hasan,
Interpretation of data: Dr. Manal Kamal Rasheed, Dr. Najmah M. Miran & lecturer Abeer Saleh Hasan
Drafting of manuscript: Dr. Manal Kamal Rasheed & Lecturer Abeer Saleh Hasan

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