

A Study of Apelin-36 and GST Levels with Their Relationship to Lipid and Other Biochemical Parameters in the Prediction of Heart Diseases in PCOS Women Patients

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Abstract:

This work studies the role of serum apelin-36 and Glutathione S-transferases (GST) activity in association with the hormonal, metabolic profiles and their link to the risk of cardiovascular disease (CVD) in healthy and patients' ladies with polycystic ovary syndrome (PCOS). A total of fifty-four (PCOS) patients and thirty-one healthy woman as a control have been studied. The PCOS patients were subdivided on the basis of body-mass-index (BMI), into 2-subgroups (the first group was obese-PCOS with BMI ≥ 30 and the second group was non-obese PCOS MBI <30). Fasting-insulin-levels and Lipid-profile, Homeostatic-model assessment-of-insulin-resistance (HOMA-IR), follicle-stimulating-hormone (FSH), luteinizing-hormone (LH), testosterone and serum Apelin-36 (AP-36) levels, GST-Activity were done for all groups. PCO patients showed higher concentricity of apelin-36 than healthy (160.43 ± 20.81 (pg/ml) versus 85.49 ± 17.85 (pg/ml), $P=0.008$), while GST-activity decreased in PCOS patients and was higher in the control (7.99 ± 1.19 (IU/L) versus 12.96 ± 1.90 (IU/L) respectively, with a P -value=0.022). Apelin-36 levels are directly interrelated with BMI and Very-low-density-lipoprotein (VLDL) in PCOS patients, but GST-activity levels correlated significantly negatively with (BMI) in PCOS patients. Moreover, obese-PCOS patients show increased AP-36 levels more than non-obese PCOS (185.76 ± 92.0 (pg/ml) versus 123.59 ± 27.65 (pg/ml), $P=0.127$) respectively, whilst the GST-activity was exhibited to be lower in obese-PCO patients more than in non-obese PCOS (6.99 ± 1.4 (IU/L) versus 9.44 ± 2.0 (IU/L), $P=0.102$). The data showed that AP-36 level is negatively associated with GST-activity in PCOS patients. AP-36 isn't legitimately ensured in the pathogenesis of PCO disorder, yet it may be included as an adipokine that is influenced by BMI. The oxidants increased because of the highly levels of VLDL and the lower in the activities of antioxidant, that may be a response to higher levels of oxidative stress. A decrease in the antioxidant capacity and an increase in AP-36 levels leads to an increased the risk of cardiovascular disease in PCOS patients.

Key words: BMI (Body mass index), Cardiovascular disease (CVD), Glutathione-S-Transferase (GST), resistance of insulin (IR) , Polycystic ovary syndrome (PCOS).

Introduction:

Polycystic ovary syndrome (PCOS), as a popular endocrine issue in procreative age, is a heterogeneous condition described by clinical symptoms, including reproductive, cardiac and psychological disorders (1, 2). Women with PCOS are at increased risk of developing chronic diseases such as uterine cancer, diabetes and cardiovascular disease. (3, 4). Different signs incorporate

hyperinsulinism, insulin resistance (IR), diabetes, hirsutism, weakness, and a condition of poor-quality inflammation (5-9). Obesity may impact the danger of ovarian cysts through insulin resistance (IR). Moreover, the compensatory hyperinsulinemia, causes expanded ovarian creation and stifles the sex hormone restricting globulin (SHBG), along these lines the androgen

bioavailability expands. Changed luteinizing hormone (LH) discharge assumes a significant job in the pathophysiology of PCOS and in spite of the fact that corpulence is commonly connected with relative decreases of (LH), higher (LH) has all the earmarks of being the best indicator of testosterone (T) among young women with heftiness. Other potential systems of weight related hyperandrogenemia incorporate upgraded androgen generation in extending the lipid lump and possible impact of unusual levels in each of cytokine and adipokine (10). Insulin resistance is the main cause of pathogenic factor in the background of increased metabolic disorders in women with PCOS, which is irregular menstruation and other metabolic manifestations associated with PCOS, but insulin resistance is not a standard diagnosis of the syndrome, yet one of its symptoms. (11). Greasy tissue works about as a member of the endocrine glands that produce an assortment of molecules signaling that direct conduct of nourishment, vitality use, digestion, generation, endocrine and immunological capacity (12). In this way, corpulence in ladies with PCOS is predominantly described by an expansion in the volume of fat cells (intemperate heftiness obesity) as opposed to expanding the quantity of fat cells (hyperplastic weight) (13). Apelin is an endogenous. Cytokine joined and transmitted through adipocytes (14), it is apparently an open controller in the lipid and glucose absorption and might be connected with the resistance of insulin. PCOS is also known to be connected in the company of extended IR (15). The Glutathione S-transferases (GST) is a gathering of multifunctionality proteins, that assume a focal job in the detoxification of electrophilic synthetic compounds and the hepatic evacuation of possibly destructive hydrophobic mixes from the blood. Oxidative stress (OS) happens because of an unevenness inters the creation of reactive oxygen species (ROS) and the framework antioxidant. In women, stress of oxidation may assume a job in infertility (16-17). Unevenness among oxidants and antioxidant causes OS coming about because of over generation of ROS or consumption of antioxidants. Operating system has been related with certain sicknesses, for example, cardiovascular malady, diabetes. A few examinations have additionally shown an expanded oxidative status and cardiovascular hazard in PCOS patient's (18).

The purpose of this work: is to investigate the association between CVD risk factors with PCOS through measuring the apelin-36 concentration and the activity of the enzyme glutathione S transferase.

Subjects and Methods :

This study included fifty four patients with PCOS (their ages ranging from (18-38) years subdivided, on the basis of BMI level into 2 groups: first group was non-obese: BMI <30 (kg)/ (m²) (n= 22) and the second group was obese: BMI ≥ 30 (kg)/ (m²) (n= 32)). They were enlisted for this investigation after their endorsement in Kamal Al-Samaria Hospital, from January to December 2019. Furthermore, thirty-one healthy women (their ages range from 19 to 32 years) were considered as a control group. The patients with PCOS were identified depending on the 2003 criteria of Rotterdam (19) with at the least 2 of the symptoms biochemical hyperandrogenism clinical or amenorrhea, oligomenorrhea, and PCOS on ultrasound. Avoidance criteria included: - Patients taking pressure medication, diabetes medication or PCOS therapy, The exclusion criteria were: Non-exemplary intrinsic adrenal hyperplasia due to 21- α hydroxylase lack, metabolic, untimely ovarian disappointment, neoplasia of ovarian, acromegaly, or cardiac disease linked condition or other simultaneous medicinal ailments (e.g diabetes mellitus), meaning to go on a diet or a particular program of physical action. Weight Index (BMI) was determined utilizing the accompanying recipe: weight (kg) / height (m²). WHR demonstrated the distribution fat (20). Waist circumference (WC), which refers to central obesity, was estimated between the costal edge and iliac peak alignment of the umbilical pivot, while the Hip circuit estimated by the hip circumference was measured to the buttocks. This survey was approved through the scientific committee in College of Science for Women, and a verbal consent form was obtained from each participant enrolled in the study.

Laboratory methods: In this study (10) mL of the venous blood was taken from each woman (patient and healthy) through Vacutainer, then the blood was put in the gel tube during the early follicular stage (this includes the days 2-5 of the menstrual cycle), then, it was left to coagulate, then separated by centrifugation at three thousand (rpm) for 10 (mints) to get the serum. The obtained serum was used to check the centrifugation of fasting serum sugar (FSS) and also the lipid profile was measured manually using kit (human, Germany). The hormonal profile was done using VIDAS analyzer (Biomerieux, France), the remained serum was stored and preserved frosting at -40 °C for diagnosing insulin hormone at fasting using ELISA (Demeditec, Germany). Apelin-36 by ELISA (CUSABIO, China) and GST activity were measured manually using GSH (Sigma chemicals, USA).

Statistical analysis: A statistical software package was used to perform the analyses programming SPSS variant 23. The result was expressed as mean \pm SD. The significant difference between mean values was estimated by the Student t-test, Chi-square test, the correlation coefficient (r) between parameters was determined by analyzing linear regression. The point of statistical significance was noted when probability was $p < 0.05$, correlation analysis was used to test the linear relationship between parameters.

Results:

A sum of 54 women with PCOS and 31 healthy were studied. The statistics of hormonal and biochemical attributes of PCOS patients and the healthy are outlined in (Table 1). The proportion of LH/FSH, LH levels, fasting insulin, HOMA-IR, and T were essentially higher, though the FSH level was decreased in the PCOS groups compared with healthy, true to form ($p < 0.05$).

Table 1: Levels (Mean \pm SD) of Apelin-36 and GST activity, lipid profile, atherogenic index and hormones (FSH, LH, Testosterone, insulin, HOMA-IR) of ladies with PCOS and controls

Parameters	Polycystic ovary syndrome (PCOS) No. (54)	Healthy control No. (31)	P value
FSH (mIU/ml)	4.49 \pm 1.73 (1.4-8.8)	7.06 \pm 1.85 (4.3-9.6)	*0.0001
LH (mIU/ml)	7.91 \pm 2.63 (2.7-16.2)	5.74 \pm 1.64 (3.1-8.7)	*0.0001
LH/FSH ratio	2.14 \pm 1.30 (0.4-7.7)	0.90 \pm 0.43 (0.3-1.7)	*0.0001
Testosterone (Ng/ml)	0.61 \pm 0.24 (0.2-1.3)	0.44 \pm 0.19 (0.1-0.9)	*0.001
Insulin (μ IU/mL)	30.16 \pm 4.18 (2.2-99.1)	22.81 \pm 3.47 (0.4-67.0)	0.180
HOMA-IR	6.56 \pm 0.91 (0.4-24.5)	4.66 \pm 0.78 (0.09-16.0)	0.119
HDL (mmol/L)	1.12 \pm 0.46 (0.4-3.1)	2.28 \pm 0.33 (1.7-2.8)	*0.0001
atherogenic index (mmol/L)	3.41 \pm 0.88 (1.9- 6.7)	3.08 \pm 0.44 (2.2- 3.8)	*0.0001
VLDL (mmol/L)	0.77 \pm 0.41 (0.1-1.8)	0.39 \pm 0.13 (0.1-0.6)	*0.0001
Apelin -36 (pg/ml)	160.43 \pm 20.81 (1.5-505.2)	85.49 \pm 17.85 (0.9-309.6)	*0.008
GST Activity (IU/L)	7.99 \pm 1.19 (0.5-33.3)	12.96 \pm 1.90 (2.0-48.9)	*0.022

*high statistically significant at ($p < 0.05$).

The level of apelin-36 and enzyme activity and another parameter are shown in Table 2.

Table 2: Levels (Mean \pm SD) of Apelin-36 and GST activity in Obese Polycystic ovary syndrome and Non-Obese polycystic ovary syndrome groups

Parameters	Obese polycystic ovary syndrome (PCO) group (1)	Non-Obese polycystic ovary syndrome (PCO) group (2)	P value
Apelin -36 (pg/ml)	185.76 \pm 92.0 (4.6-505.2)	123.59 \pm 27.65 (1.5-405.7)	0.127
GST Activity (IU/L)	6.99 \pm 1.4 (0.5-33.3)	9.44 \pm 2.0 (1.04-31.2)	0.102
Insulin (μ IU/mL)	35.13 \pm 33.19 (2.2-99.1)	22.94 \pm 25.78 (2.4-96.6)	0.106
HOMA-IR	7.52 \pm 7.15 (0.4-24.5)	5.17 \pm 6.0 (0.4-22.3)	0.198

* no statistically significant difference in serum Apelin -36 level, GST Activity, Insulin and, HOMA-IR of ($p > 0.05$) in Obese PCOS group compared to non- Obese PCOS group.

The relationship between apelin-36 and all parameters is shown in Table 3.

Table 3: correlation between apelin-36 and some studied variables included the one which is sig (P) & correlated (R)

		Apelin -36
		Polycystic ovary syndrome (PCOS)
		No. (54)
Age (years)	R	0.010
	P	0.943
BMI (Kg/m2)	R	0.426**
	P	0.001
WHR	R	0.092
	P	0.509
FBS	R	-0.078
	P	0.574
Cholesterol	R	0.160
	P	0.248
Triglycerides	R	0.161
	P	0.246
HDL	R	-0.223
	P	0.105
LDL	R	-0.132
	P	0.340
VLDL	R	0.161
	P	0.246
FSH	R	-0.226
	P	0.100
LH	R	0.220
	P	0.109
LH/FSH ratio	R	0.246
	P	0.073
Testosterone	R	0.640**
	P	0.0001
Insulin	R	0.287*
	P	0.035
HOMA IR	R	0.268
	P	0.050
GST activity	R	-0.231
	P	0.093

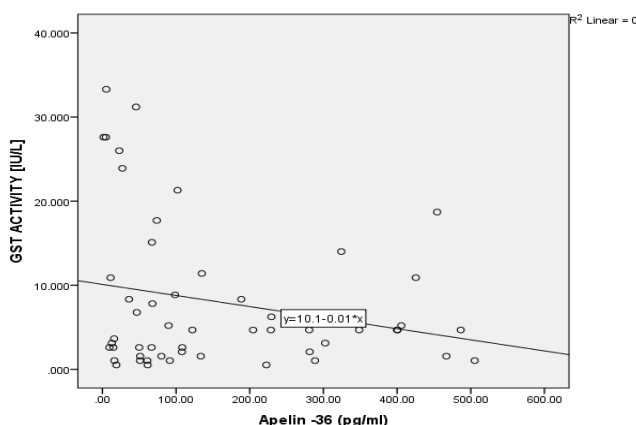


Figure (1): Not strong negative correlation Significant Significant between apelin-36 and GST Activity in PCO

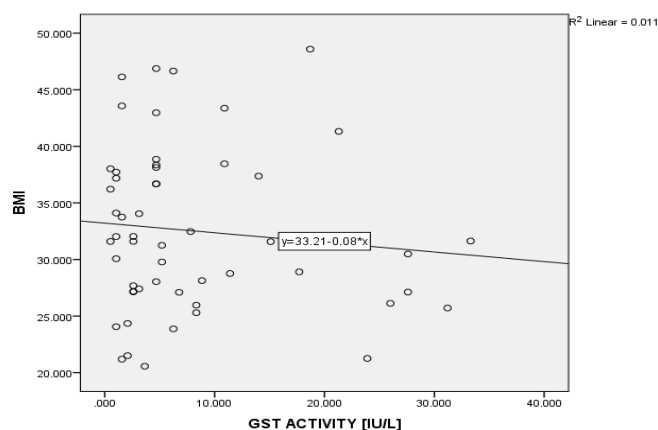


Figure (2): negative r & p value correlation between BMI and GST

Discussion:

PCO disorder is an endocrine and metabolic agitating impact which has an effect on 6% to 20% of women in the productive age (21), it is one of the novel delineated adipokines emitted through development in the human's adipocytes. Apelin plays an important role in increasing myocardial contractility and works to control blood flow, and cardiac tissue remodeling (22). This assumes an opener employment in the guideline of typical sugar and fat digestion and related with IR (23). Glutathione-S-transferase (GST) is a detoxification enzyme essential for a cellular protection against oxidative damage (24). In the present study, AP-36 levels were found to rise in patients with PCOS, when compared with the healthy group. It has also been found that levels of apelin-36 were higher in obese patients more than non- obese PCOS as shown in the Fig. 1. This result may be due to the inflammation of lipid cells in PCOS owing to differences ability in production an equal amount between HDL cholesterol and LDL cholesterol, this leads to increasing the levels of LDL and VLDL, especially in heart arteries and in the area of the waist and hip. It has been noted that the higher the ratio of waist circumference to the hip circumference, the greater the concentration of Ap-36 in sera of patients. These outcomes were in concurrence with Sun *et al.*, (25) who found increase in the AP-36 level in obese PCOS patients when compared with non- obese PCOS patients. In the present study, it has also been found that GST activity was lower in PCOS patients when compared with the healthy group. It has been found that the activity of enzyme is lower in obese PCOS patients more than non- obese PCOS patients. The decreased activity of this antioxidant enzyme in patients may be due to the increased production of oxidants in the body as a result of increased LDL fat in the body leading to a risk of atherosclerosis and heart attack. These outcomes are in agreement with Moti, Mahtab *et al.* (24, 26), who found increased oxidative stress and decrease in the level of antioxidants in PCOS patients when compared with healthy group. In the present study, it has been observed that Apelin -36 levels were emphatically associated with BMI as shown in Fig. 2, testosterone and insulin in PCOS groups. There was non-noteworthy connection between AP-36 and HOMA-IR, TG, VLDL, LH, LH/FSH proportion in PCOS subgroups as shown in (Table-3). Depending on our results, AP-36 isn't legitimately ensured in the PCOS pathogenesis, yet it might be included as (an adipokines) influenced through body mass index. Our outcomes accord with Chang *et al.*, (23) and Choi *et al.*, (27) who proclaimed no relationship

between AP-36 levels and HOMA-IR. This might be expected to the Hemostatic model assessment of insulin resistance dependent on FBS and fasting insulin levels. In which apelin improve the glucose digestion in the human body by expanding glucose usage in the insulin by increase the tissues sensitivity, doubtlessly in an insulin-free way as opposed to through hindrance of hepatic glucose yield (27). These actualities may be behind the absence or weakness of the relationship between apelin-36 levels and HOMA-IR. The difference between published studies might be credited to the distinctions in age, ponder structure, hereditary attributes. In this manner, further investigations are required for further proofs with various hereditary basis.

Conclusion:

The study concludes the following: -

1. Insulin and HOMA-IR, AP-36 are high levels in obesity PCOS patients than non- obesity.
2. PCOS issue is related with metabolic disorder and dyslipidemia, cardiovascular hazard worker particularly raised TG which serve to build the substrate with the expectation of complimentary radicals which are not killed by the blemished cancer prevention agent framework.
3. Low capacity of antioxidant may share an increased the risk of illnesses of cardiovascular in young women with PCOS. An effective decrease at antioxidants may be due to high body bad fat (VLDL) and low good fat intake. as Additionally, it may be due to atherosclerotic factors in addition to famous peril worker like resistance of insulin, high blood pressure, increased body mass index (obesity), and fat in the blood. In this way, treatment with cellular enhancements in the basic stages of infection may be useful as an optional treatment to avoid oxidative damage.

Authors' declaration:

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine ours. Besides, the Figures and images, which are not mine ours, have been given the permission for re-publication attached with the manuscript.
- The author has signed an animal welfare statement.
- Ethical Clearance: The project was approved by the local ethical committee in University of Baghdad.

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مستويات Apelin-36 وأنزيم GST وعلاقتها بالدهون وغيرها من المعايير الكيموحيوية في التنبؤ بأمراض القلب لدى النساء المصابات بمتلازمة تكيس المبايض

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3 مستشفى كمال السامرائي ، وزارة الصحة ، بغداد ، العراق

الملخص:

هذا العمل يتضمن دراسة دور الألبين-36 وفعالية انزيم الكلوتاثيون وعلاقتها بالهرمونات والعوامل ذات الصلة بالتمثيل الغذائي، وارتباطها بخطر الإصابة بأمراض القلب الوعائية في النساء الأصحاء والنساء المصابات بمتلازمة تكيس المبايض. اشتملت الدراسة على 54 مصابة بمتلازمة تكيس المبايض و31 امرأة بصحة جيدة (المجموعة الضابطة). مرضى PCOS تم تقسيمهم حسب مؤشر كتلة الجسم BMI الى مجموعتين (المجموعة الأولى يعانون من السمنة المفرطة $BMI \geq 30$ والمجموعة الثانية لا يعانون من السمنة $BMI \leq 30$). AP-36, GST-activity, Fasting-insulin, HOMA-IR, Testosterone, LH, FSH, Lipid-profile تم قياس المتغيرات التالية لجميع المجاميع. أظهرت السيدات المصابات بمرض تكيس المبايض ارتفاع في تركيز مصل Apelin-36 أعلى من السيدات الأصحاء (20.81 ± 160.43 مقابل 17.85 ± 85.49 pg/ml، $P=0.008$)، بينما كان فعالية انزيم GST منخفضة في المرضى ومرتفعاً في الأصحاء (7.99 ± 1.19 IU/L مقابل 1.90 ± 12.96 IU/L، $P=0.022$). ترتبط مستويات APLEN-36 ارتباطاً مباشراً بمؤشر كتلة الجسم و VLDL في المرضى، لكن مستويات فعالية انزيم GST مرتبطة عكسياً مع (BMI) في المرضى. علاوة على ذلك، وجد ان هنالك زيادة في مستويات AP-36 في مرضى PCOS البدناء أكثر من مرضى PCOS غير البدناء (92.0 ± 185.76 مقابل 27.65 ± 123.59 pg/ml، $P=0.127$)، في حين وجد أن نشاط أنزيم GST كان منخفضاً في مرضى PCOS البدناء أكثر من مرضى PCO غير البدناء (6.99 ± 1.4 IU/L مقابل 2.0 ± 9.44 IU/L، $P=0.102$). يتضح من المعلومات التالية بأن مستوى AP-36 يرتبط عكسياً بفعالية أنزيم GST في مرضى تكيس المبايض. AP-36 لا يرتبط مباشرة في التسبب في اضطراب PCO، ومع ذلك فقد يتم إدراجه باعتباره مادة دهنية تتأثر بمؤشر كتلة الجسم. أن زيادة المواد المؤكسدة بسبب زيادة مستوى VLDL وانخفاض فعالية إنزيمات مضادات الأكسدة قد تكون استجابة للمستويات العالية للأجهد التأكسدي. ان انخفاض قدرة مضادة الأكسدة وزيادة تركيز AP-36 قد يؤدي الى توسع خطر الإصابة بأمراض القلب الوعائية لدى السيدات المصابات بمتلازمة تكيس المبايض، بالإضافة إلى عوامل الخطر، مثلاً:- مقاومة الأنسولين، ارتفاع ضغط الدم، السمنة المفرطة، ارتفاع مستوى الدهون.

الكلمات المفتاحية: مؤشر كتلة الجسم (BMI)، أمراض القلب والأوعية الدموية (CVD)، الجلوتاثيون-اس-ترانسفيراز (GST)، مقاومة الأنسولين (IR)، متلازمة تكيس المبايض (PCOS).