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Analysis of Adipokines and some Steroid Hormones in Myocardial Infarction

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Abstract:

The most common cause of death is cardiovascular disease (CVD), with ischemic heart disease being the most notable type. There is a propensity to raise the sensitivity of methods in contemporary laboratory for diagnosing of CVD, and assessing key as CVD bio-indicators. The urgent task is to seek for different indicators as a hopeful tool for early detection and monitoring of myocardial infarction in blood samples only. This study comprised 117 Volunteers, recorded with both genders in the age range of 32-64 years old. The volunteers were categorized into two groups: 67 patients of myocardial infarction, other group embraced 50 healthy individuals. The samples of blood were collected and directed for biochemical analysis to evaluate estradiol, testosterone, progesterone, adiponectin, leptin, and lipid profile [total cholesterol, triglycerides, high density lipoprotein (HDL), and low-density lipoprotein (LDL)] levels in each group. The following conclusion can be drawn from this study based on statistical assessment of bio-indicator parameters, significantly reduced of testosterone and HDL ($P < 0.001$) levels in myocardial infarction, within non-significantly elevated $P < 0.061$ of progesterone levels in myocardial infarction patients as compared with healthy individuals. The remaining biochemical tests indicated significantly elevated levels in patients with myocardial infarction such as estradiol ($P < 0.001$), adiponectin ($P < 0.001$), leptin ($P < 0.001$), total cholesterol ($P < 0.001$), triglycerides ($P < 0.001$) and LDL ($P < 0.001$) levels. Adipokines (adiponectin, leptin) and some steroid hormones (estradiol, testosterone) show crucial roles in the improvement of metabolic and cardiovascular diseases and may be utilized as bio-pointer for myocardial infarction exposure, medical conduct and severity. This acknowledgment offers early diagnosis of disease and progression.

Keywords: Adiponectin, Estradiol, Ipid profile, Leptin, Myocardial infarction, Progesterone, Testosterone.

Introduction:

Cardiovascular disease (CVD) is still the leading cause of death in both men and women.^{1,2} Myocardial infarction (MI) is delineated as a clinical incident in the scenery of myocardial ischemia, which showed signs of myocardial injury. In order to analyze the bio-indicators' implications with cardiac symptoms, electrocardiographic alterations were suggested, and/or imaging with echocardiography or nuclear revisions demonstrating harm of viable myocardium or fresh area wall sign anomaly³. One of the cardiovascular dangers is myocardial infarction obliges an emergency judgment and treatment manner⁴, with the prospective for substantial indisposition and death⁵. Jeopardy influence for metabolic deviations and predisposes to cardiovascular disease is

fatness⁶. Globally an uninterrupted rise in obesity occurrence⁷ with the cardiometabolic concerns, so they motivate attention to instigate of conceivable linked mechanisms⁸. Presently adipose tissue is measured as an endocrine organ, a complex arrangement tangled not just in fat storage but likewise in freeing numerous bio-active polypeptides termed adipokines. The adipokines task is to adjust the body weight, pressure of the blood, appetite monitoring, glucose homeostasis, and control inflammation⁹. Adipocytes formed from polypeptide hormone termed leptin, and it is measured as an adipokine pro-inflammatory tangled in the reducing of inflammatory state pronounced in heavy and fatness sickness. The most distinguishing secretory product is leptin, which is detected and

measured as an endocrine organ.^{10,11}. In addition, leptin adjusts the other cytokines pro-inflammatory such as interleukin-6 with tumor necrosis factor α ¹². Adiponectin, on the other hand, is an isoforms hormone made up of a 244-amino-acid protein excreted by diverse cell categories, for example adipocytes and endothelial cells. It is quite notorious the immersion of adiponectin in fatness allied dysthymia, linking diabetes the kind of type two and cardiovascular illness^{9,13}. The steroid is tangled in physiology and disease, interceding endocrine, reproductive tasks and acting part in sarcoma, neurological, pathways, and cardiovascular illnesses^{14,15}. A crucial gender hormone in males is a testosterone which is chiefly formed from the testes Leydig cells and enhanced by the hormone termed luteinizing¹⁶. Another gender hormone in charge termed estradiol which is the leading estrogen during propagative in female years together in the serum consists besides in the terms of active estrogen¹⁷. Progesterone is a gender steroid hormone tangled in the cycle of menstrual, pregnancy, etc.¹⁸. So the urgent task of this article is to seek for various bio-pointers (gender hormones and cytokines) as a hopeful tool for early detection and monitoring of myocardial infarction in blood samples only. The aim of the present study is to demonstrate the status of serum levels of Adipokines (adiponectin & leptin) and Steroid Hormones in patients with myocardial infarction and to compare it with those of healthy controls. This is the first study of its type conducted in Erbil/Iraq in MI patients.

Materials and Methods:

Blood samples were collected in Rzgary hospital/Erbil-Iraq between periods of Sep 2020 to Feb 2021. The totals of 117 volunteers were recruited to this scientific trial, registered of both genders in the age range of 32-64 years old. Volunteers were categorized into two groups: 67 patients of MI included 26 males (age range 32-64) with 41 females (age range 33-61), other groups embraced 50 healthy individuals (19 males and 31 females). The samples of blood were collected between 8.00-10.0 am. The serum was prepared by collecting 5 ml of the blood in a test tube without anticoagulant, the samples were separated by centrifugation, then directed for biochemical analysis to evaluate estradiol, testosterone, progesterone, adiponectin, leptin, and lipid profile (total cholesterol, triglycerides, HDL, and LDL) levels in each group. The ELISA (sandwich enzyme linked immunosorbent assay) was used to assess serum estradiol, testosterone, progesterone,

adiponectin, and leptin levels by Biotech company ELISA kit, while, Biolabo kit was utilized for lipid profile assess, which was determined by spectrophotometer instrument. The levels of the biochemical parameters in the samples were obtained by instructions supplied by the manufacturer. The program SPSS 25.0 was used for all statistical analyses.

Results:

The quantifiable data of 50 healthy subjects and 67 patients of myocardial infarction were enrolled for two different samples in this trial. The results of the blood parameters levels have been revealed in Figs. 1, 2. When compared to the control group, the testosterone and HDL ($P < 0.001$) levels in MI were considerably lower, within non-significantly elevated $P < 0.061$ of progesterone levels in MI patients. The remaining biochemical indicated significantly high levels in patients with MI as compared with control groups such as estradiol ($P < 0.001$), adiponectin ($P < 0.001$), leptin ($P < 0.001$), total cholesterol ($P < 0.001$), triglycerides ($P < 0.001$) and LDL ($P < 0.001$) levels.

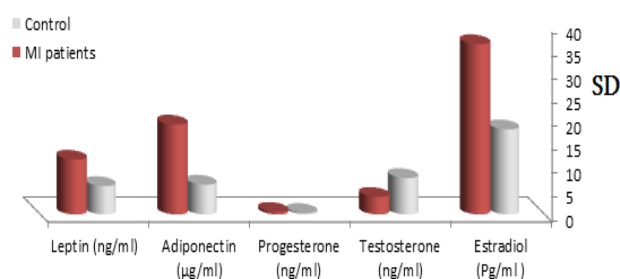


Figure 1. Adiponectin, leptin, estradiol, testosterone, and progesterone levels in control and patients groups with MI

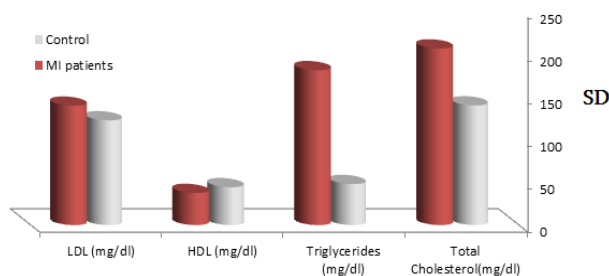


Figure 2. Total cholesterol, triglycerides, HDL, and LDL levels in control and patients groups with MI

Discussion:

In order understand the CVDs pathophysiology excessive progress has been prepared, and this has upgraded the CVDs prognosis and prevention¹. The

significant increases in serum levels of estradiol, adiponectin, leptin, total cholesterol, triglyceride, and LDL were discovered in this study.. While testosterone and HDL levels were significantly diminished, within non-significantly elevated of progesterone levels in MI cases. Adiponectin diminished infection in many places such as cardiac myocytes, vascular endothelial cells, muscle cell (smooth), and macrophages^{19,20}. The anti-inflammatory action of adiponectin may perhaps be refereed by direct influences on inflammatory cells or from the conquest of the nuclear factor kappa B metabolism¹⁹. Due to the shielding activity of adiponectin in CVD and atherosclerosis, it is a noticeable pathophysiological shielding in various articles¹⁹⁻²¹. Some articles such as Ahmed et al.²², and Diah et al.²³ found that low concentrations of adiponectin in the proliferation of epicardial lipid bulk and coronary artery illness, these results are in line with the current study, that adiponectin concentrations reduced significantly ($p < 0.001$) as displayed in Fig. 1 in MI cases when compared with normal subjects. Although the researchers Achari and Jain exposed the contribution of adiponectin in fatness related with diabetes mellitus (type2) and CVD²⁴. Leptin is acknowledged to be principally tangled in reaction and resistance contrary to reduce the body lipid that could cause disability of survival and generative fitness. As a result, central nervous system signals to leptin are required for energy stores, along with homeostasis of normal energy, cardiovascular, etc.⁹. Some articles such as Martin et al.²⁵, Bickel et al.²⁶, Yang et al.²⁷ Katsiki et al.²⁸ and Weschenfelder et al.¹¹ observed enlarged levels of leptin levels in CVD patients, which is consistent with these existing study, that leptin amounts enhanced significantly ($p < 0.001$) in MI cases as demonstrated in Fig. 1. It is normally rumored that enriched estradiol concentrations in females are cardio-shielding. Peters and Woodward²⁹ explained that elevation amounts of estradiol were not linked with a lessened exposure of MI. This corresponded to our data showing significant ($P < 0.001$) increased levels of estradiol levels in sera samples in MI as compared with healthy subjects, which have been observed in Fig. 1. In the harmony with the current paper Lee et al.¹⁶ and Maggi, et al.³⁰ have been specified that cases with CVD have significantly elevated 17-beta estradiol levels and reduced testosterone concentrations. While Laughlin et al.³¹ and Tivesten et al.³² rate that elevated total death and CVD correlated with death were originated in reduced hormone testosterone concentrations. This was in agreement with this study, which demonstrated significantly ($P < 0.001$), low serum testosterone concentrations

level in cases with MI as observed in Fig. 1. Stagnant, the influences of progesterone on the CVD organism have been tiny revisions up to now. It has similarly been stated that reduced serum testosterone concentrations are linked with elevation of the occurrence of CVD in men³³. Progesterone acts as a chief part in CV and central nervous systems and influences mammary gland growth³⁴. Little linked and total jeopardies of evolving CVD is allied for most females who are using by combination estrogen-progestin oral contraceptives³⁵⁻³⁶. While Bernstein and Pohost established that taking the mixture of medroxyprogesterone and conjugated estrogen caused a more danger MI and stroke³⁷. The Chinese experimental exposed that, progesterone and C-reactive protein are connected to obesity³⁸. As similar to this existing trial that has been demonstrated the non-significant elevation $P < 0.061$ of serum progesterone concentrations in MI cases as seen in Fig. 1. In the Fig. 2 the results displayed that the significantly reduced of HDL levels ($P < 0.001$), within significantly raise levels of total cholesterol ($P < 0.001$), TG ($P < 0.001$) and LDL ($P < 0.001$) levels in MI cases as compared with control groups. This was agreeing with researches done by Ibrahim et al.³⁹, whom reported that the amounts of IL-1 α elevated in acute MI or with unstable angina. IL-1 α elevated positively with total cholesterol, triglycerides, LDL and LDL, whereas there was negatively reduced with HDL in acute MI or with unstable angina. Although Ahmed et al.⁴⁰ they indicated that serum triglycerides and cholesterol were positively increased and linked with cases have type two diabetes mellitus with CVD. Continually Kadhim and Jaddoue⁴¹ reported that the concentration of cholesterol elevated MI cases.

Conclusions:

The blood parameters levels of testosterone reduced significantly within non-significantly elevated progesterone levels, as reported in this paper, on what was the improvement concluded, and the study was not followed up on. So adipokines (adiponectin, leptin) and some steroid hormones (estradiol, testosterone) show crucial roles in the improvement of metabolic and cardiovascular diseases and may be utilized as bio-pointer for myocardial infarction exposure, medical conduct and severity. This acknowledgment offers an early analysis of illness and progression.

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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 republication attached with the manuscript.
- Authors sign on ethical consideration's approval
- Ethical Clearance: The project was approved by the local ethical committee in Dentistry College, Hawler Medical University.

Authors' contributions statement:

A. M. Y. (author) wrote and edited the manuscript with revisions idea, P. A. I. analysis the data with revisions idea. Ch. A. I. diagnosis the cases then collected the samples and doing the tests. Editing, investigation and proofreading were done by all investigators.

References:

1. Ueda K, Fukuma N, Adachi Y, Numata G, Tokiwa H, Toyoda M, et al. Sex differences and regulatory actions of estrogen in cardiovascular system. *Front Physiol.* 2021; 2: 1-9. <https://doi.org/10.3389/fphys.2021.738218>
2. Mair J, Lindahl B, Hammarsten O, Müller C, Giannitsis E, Huber K, et al. How is cardiac troponin released from injured myocardium *Eur Heart. J Acute Cardiovasc.* 2018; 7(6): 553-560, <https://doi.org/10.1177/2048872617748553>
3. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA et al. Fourth Universal Definition of Myocardial Infarction. *J Am Coll Cardiol.* 2018; 72(18): 2231-64.
4. Zeng J, Huang J, Pan L. How to balance acute myocardial infarction and COVID-19: the protocols from Sichuan Provincial People's Hospital. *Intensive Care Med.* 2020; 46: 1111-13.
5. Anderson JL, Morrow DA. Acute myocardial infarction. *New Eng J Med.* 2017; 376(21): 2053-64. <https://doi.org/10.1056/NEJMra1606915>
6. Kachur S, Lavie CJ, de Schutter A, Milani RV, Ventura HO. Obesity and cardiovascular diseases. *Minerva Med.* 2017; 108: 212-228.
7. Zhu J, Su X, Li G, Chen J, Tang B, Yang Y. The incidence of acute myocardial infarction in relation to overweight and obesity: a meta-analysis. *Arch Med Sci.* 2014;10(5):855-862. doi:10.5114/aoms.2014.46206
8. Fuster JJ, Ouchi N, Gokce N, Walsh K. Obesity-induced changes in adipose tissue microenvironment and their impact on cardiovascular disease. *Circ Res.* 2016; 118: 1786-1807.
9. Recinella L, Orlando G, Ferrante C, Chiavaroli A, Brunetti L, Leone S. Adipokines: new potential therapeutic target for obesity and metabolic, rheumatic, and cardiovascular diseases. *Front Physiol.* 2020; 11: 578966. doi: 10.3389/fphys.2020.578966.
10. Funcke JB, Scherer PE. Beyond adiponectin and leptin: Adipose tissue-derived mediators of inter-organ communication. *J Lipid Res.* 2019; 60: 1648-1684.
11. Weschenfelder C, Schaan de Quadros A, Lorenzon dos Santos J, Garofallo SB, Marcadenti A. Adipokines and adipose tissue-related metabolites, nuts and cardiovascular disease. *Metabolites.* 2020; 10(1): 32. doi: 10.3390/metabo10010032.
12. Pandit R, Beerens S, Adan RAH. Role of leptin in energy expenditure: the hypothalamic perspective. *Am J Physiol Regul Integr Comp Physiol.* 2017; 312: R938-R947.
13. Achari AE, Jain SK. Adiponectin, a therapeutic target for obesity, diabetes, and endothelial dysfunction. *Int J Mol Sci.* 2017; 18:1321. doi: 10.3390/ijms18061321.
14. Funder JW. Primary aldosteronism. *Hypertension.* 2019; 74: 458-466.
15. Dwyer AR, Truong TH, Ostrander JH, Lange CA. 90 years of progesterone: steroid receptors as MAPK signaling sensors in breast cancer: let the fates decide. *J Mol Endocrinol.* 2020; 65: T35-T48.
16. Lee JH, Shah PH, Uma D, Salvi DJ, Rabbani R, Hamid P. Testosterone replacement therapy in hypogonadal men and myocardial infarction risk: systematic review & meta-analysis. *Cureus.* 2021; 13(8): e17475. doi: 10.7759/cureus.17475
17. Iwakura A, Shastry S, Luedemann C, Hamada H, Kawamoto A, Kishore R, et al. Estradiol enhances recovery after myocardial infarction by augmenting incorporation of bone marrow-derived endothelial progenitor cells into sites of ischemia-induced neovascularization via endothelial nitric oxide synthase-mediated activation of matrix metalloproteinase-9. *Circulation.* 2006; 113: 1605-1614. <https://doi.org/10.1161/CIRCULATIONAHA.105.553925>.
18. Zhang J, Abou-Fadel JS. Calm the raging hormone - a new therapeutic strategy involving progesterone-signaling for hemorrhagic CCMs. *Vessel Plus.* 2021; 5: 48.
19. Nigro E, Scudiero O, Sarnataro D, Mazzarella G, Sofia M, Bianco A, et al. Adiponectin affects lung epithelial A549 cell viability counteracting TNF α and IL-1 β toxicity through AdipoR1. *Int J Biochem Cell Biol.* 2013; 45: 1145-53. doi: 10.1016/j.biocel.2013.03.003
20. Subedi A, Park PH. Autocrine and paracrine modulation of microRNA-155 expression by globular adiponectin in RAW 264.7 macrophages: involvement of MAPK/NF- κ B pathway. *Cytokine.* 2013; 64: 638-41. doi: 10.1016/j.cyto.2013.09.011
21. Villarreal-Molina MT, Antuna-Puente B. Adiponectin: antiinflammatory and cardioprotective effects. *Biochimie.* 2012; 94: 2143-49. doi: 10.1016/j.biochi.2012.06.030
22. Ahmed HH, Shousha WG, El-Mezayen HA, Emara IA, Hassan ME. New biomarkers as prognostic

- factors for cardiovascular complications in type 2 diabetic patients. *Indian J Clin Biochem.* 2020; 35: 54-62. doi: 10.1007/s12291-018-0784-4
23. Diah M, Lelo A, Lindarto D, Mukhtar Z. Plasma concentrations of adiponectin in patients with coronary artery disease and coronary slow flow. *Acta Med Indones.* 2019; 51: 290-95.
24. Achari AE, K Jain S. Adiponectin, a therapeutic target for obesity, diabetes, and endothelial dysfunction. *Int J Mol Sci.* 2017; 18(6): 1321. doi: 10.3390/ijms18061321.
25. Martin SS, Blaha MJ, Muse ED, Qasim AN, Reilly MP, Blumenthal RS, et al. Leptin and incident cardiovascular disease: The Multi-ethnic Study of Atherosclerosis (MESA). *Atherosclerosis.* 2015; 239: 67-72.
26. Bickel C, Schnabel RB, Zeller T, Lackner KJ, Rupprecht HJ, Blankenberg S, et al. Predictors of leptin concentration and association with cardiovascular risk in patients with coronary artery disease: Results from the AtheroGene study. *Biomarkers.* 2017; 22: 210-218.
27. Yang H, Guo W, Li J, Cao S, Zhang J, Pan J, et al. Leptin concentration and risk of coronary heart disease and stroke: A systematic review and meta-analysis. *PLoS One* 2017; 12: e0166360.
28. Katsiki N, Mikhailidis DP, Banach M. Leptin, cardiovascular diseases and type 2 diabetes mellitus. *Acta Pharmacol Sin.* 2018; 39(7): 1176-88. doi: 10.1038/aps.2018.40
29. Peters SAE, Woodward M. Oestradiol and the risk of myocardial infarction in women: a cohort study of UK Biobank participants. *Int J Epidemiol.* 2021; 50(4): 1241-49. doi: 10.1093/ije/dyaa284.
30. Maggi M, Wu FC, Jones TH, Jackson G, Behre HM, Hackett G et al. Testosterone treatment is not associated with increased risk of adverse cardiovascular events: results from the Registry of Hypogonadism in Men (RHYME). *Int J Clin Pract.* 2016; 70:843-52. doi:10.1111/ijcp.12876
31. Laughlin GA, Barrett-Connor E, Bergstrom J. Low serum testosterone and mortality in older men. *J Clin Endocrinol Metab.* 2008; 93: 68-75. doi: 10.1210/jc.2007-1792
32. Tivesten A, Vandenput L, Labrie F, Karlsson MK, Ljunggren O, Mellström D, et al. Low serum testosterone and estradiol predict mortality in elderly men. *J Clin Endocrinol Metab.* 2009; 94: 2482-8. doi:10.1210/jc.2008-2650
33. Khera M, Miner M, Jaffe J, Pastuszak AW. Testosterone therapy and cardiovascular risk: a critical analysis of studies reporting increased risk. *J Sex Med.* 2021; 18: 83-98. doi: 10.1016/j.jsxm.2020.10.019
34. Nagy B, Szekeres-Barthó J, Kovács GL, Sulyok E, Farkas B, Várnagy Á, et al. Key to life: Physiological role and clinical implications of progesterone. *Int J Mol Sci.* 2021; 22: 11039. <https://doi.org/10.3390/ijms222011039>.
35. Okoth K, Singh J, Thomas G, Nirantharakumar K. Association between the reproductive health of young women and cardiovascular disease in later life: *BMJ* 2020; 371. *MJ* 2020; 371 doi: <https://doi.org/10.1136/bmj.m3502>
36. Shufelt CL, Bairey Merz CN. Contraceptive hormone use and cardiovascular disease. *J Am Coll Cardiol.* 2009; 53: 221–31.
37. Bernstein P, Pohost G. Progesterone, progestins, and the heart. *Rev Cardiovasc Med.* 2010; 11(4): 228-36.
38. Nie L, Wei D, Liu P, Zhang L, Fan K, Song Y, et al. C-Reactive protein mediates the effect of serum progesterone on obesity for men and postmenopausal women in henan rural cohort study. *J Inflamm Res.* 2021; 14: 633–644. doi: 10.2147/JIR.S293882
39. Ibrahim AE, Ibrahim ZI, Thamer H. Comparison study of Interleukin-1 alpha between unstable angina and acute myocardial infarction patients. *Baghdad Sci J.* 2013; 10(3): 915-20.
40. Ahmed HH, Shousha WG, El-Mezayen HA, Emara IA, Hassan ME. New biomarkers as prognostic factors for cardiovascular complications in type 2 diabetic patients. *Indian J Clin Biochem.* 2020; 35: 54-62. doi: 10.1007/s12291-018-0784-4.
41. Kadhim HY, Jaddoue BA. Assessment of risk factors for myocardial infarction and its relationship with some variables. *Baghdad Sci J.* 2010; 7(1): 784-87.

تحليل الأديبوكينات وبعض هرمونات الستيرويد في احتشاء عضلة القلب

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الخلاصة:

الموت الشامل بسبب أمراض القلب والأوعية الدموية هو السبب السائد للوفاة، ومرض القلب الإقفاري هو السبب البارز. هناك ميل لزيادة حساسية الأساليب في المختبر المعاصر لتشخيص الأمراض القلبية الوعائية، وتقييم المفتاح كمؤشرات حيوية للأمراض القلبية الوعائية. المهمة العاجلة هي البحث عن مؤشرات مختلفة كأداة للكشف المبكر عن احتشاء عضلة القلب ومراقبته. ضمت هذه المحاولة 117 متطوعاً، تم تسجيلهم من كلا الجنسين في الفئة العمرية من 32 إلى 64 عاماً. تم تصنيف المتطوعين إلى مجموعتين: 67 مريضاً من احتشاء عضلة القلب، واحتضنت المجموعات الأخرى 50 فرداً سليماً. تم جمع عينات الدم وتوجيهها للتحليل الكيميائي الحيوي لتقييم مستويات هرمون الاستراديول والتستوستيرون والبروجسترون والأديبونكتين واللبتين والدهون في كل مجموعة. بناءً على التقييم الإحصائي لمعاملات المؤشر الحيوي، يمكن الإعلان عن الاستنتاج التالي من هذه الدراسة، وهو انخفاض كبير في مستويات هرمون التستوستيرون والبروتين الدهني عالي الكثافة (P < 0.001) في احتشاء عضلة القلب، ضمن $P > 0.061$ المرتفعة بشكل ملحوظ من مستويات البروجسترون في عضلة القلب مرضى الاحتشاء بالمقارنة مع الأفراد الأصحاء. أشارت الاختبارات الكيميائية الحيوية المتبقية إلى مستويات مرتفعة بشكل ملحوظ في المرضى الذين يعانون من احتشاء عضلة القلب مثل استراديول (P < 0.001)، أديبونكتين (P < 0.001)، ليبتين (P < 0.001)، الكلي الكوليسترول والكوليسترول (P < 0.001)، الدهون الثلاثية (P < 0.001) ومستويات البروتين الدهني منخفض الكثافة (P < 0.001). تُظهر الأديبوكينات (أديبونكتين، ليبتين) وبعض هرمونات الستيرويد (استراديول، هرمون التستوستيرون) أدواراً حاسمة في تحسين أمراض التمثيل الغذائي والقلب والأوعية الدموية ويمكن استخدامها كمؤشر حيوي للتعرض لاحتشاء عضلة القلب، والسلوك الطبي والشدة. يقدم هذا الإقرار التشخيص المبكر للمرض وتطوره.

الكلمات المفتاحية: أديبونكتين، استراديول، نسب الدهون، ليبتين، احتشاء عضلة القلب، بروجسترون، هرمون تستوستيرون.