JBAAR

Journal of Bioscience and Applied Research

https://jbaar.journals.ekb.eg

SPBH

Association between Asprosin and Clusterin in Patients with Myocardial Infarction.

Ahmed Abdulkadhim Hamidy[1], Marwah A. K. Hameedi[2], Noor Ali Abdul Jabbar[3], Mohammed I Hamzah[4]

[1]Chemistry Department, College of Science, Mustansiriyah University, Baghdad – Iraq.

*E-mail: ahmed.abd.h@uomustansiriyah.edu.iq

[2]Missions and Cultural Relations Department, AI-Iraqia University, Baghdad, Iraq.

*E-mail: marwa.a.hamidi@aliraqia.edu.iq

[3]Biology Department, College of Science, Mustansiriyah University, Baghdad – Iraq.

*E-mail: nora.alkadi@uomustansiriyah.edu.iq

[4]Ph.D. Clinical chemistry, College of Medicine, Alnahrain University, Baghdad, Iraq.

*E-mail: Moh_alsafi75@nahrainuniv.edu.iq

DOI:10.21608/jbaar.2025.385738.1204

ABSTRACT

Background: The most common cause of acute myocardial infarction (AMI) is still plaque rupture. Elevation of ST segments. Myocardial infarction (STEMI) and elevation without ST-segment. There are two forms of AMI, Myocardial Infarction (NSTEMI) and AMI, each with unique clinical features. Asprosin is a newly discovered fasting-induced glucogenic adipokine that stimulates the liver to release glucose into the bloodstream. It has been linked to metabolic disorders like polycystic ovarian syndrome and type II diabetes mellitus. Most biological fluids contain the heterodimeric glycoprotein clusterin, which is produced by a variety of tissues. Based on clusterin's distribution and in vitro properties, several physiological roles have been suggested. A notable and distinctive characteristic of clusterin is its induction in various disease states, including but not limited to glomerulonephritis, polycystic kidney disease, renal tubular injury, and neurodegenerative conditions such as Alzheimer's disease, atherosclerosis, and myocardial infarction.

Keywords: Asprosin, clusterin, Lipid Profile, CK-MB, Myocardial infarction

---

INTRODUCTION

(1,655 per 100,000) People worldwide suffer from coronary artery disease (CAD), and by 2030, that number is predicted to rise to 1,845 per 100,000. With nine million deaths a year, it is the primary cause of death globally (1). Even with remarkable medical progress, CAD still has a negative economic impact on the world (2).

Myocardial necrosis brought on by a coronary artery blockage is known as an acute myocardial infarction (AMI). One typical symptom is chest pain. A diagnosis is typically made using the electrocardiogram (ECG) and the existence or lack of serological markers (3). If the coronary artery is still blocked, most of the region that is at risk turns necrotic. The cornerstone of current treatment to

Received: May 16, 2025. Accepted: July 30, 2025. Published: September 22, 2025

prevent myocardial tissue loss is coronary revascularization, which restores blood flow to the area at risk (4). It was recently revealed that the hormone Asprosin, a new hormone with a protein structure, controls the production of glucose from the liver. This hormone, which is released from white adipose tissue and stimulates glucose and insulin production during fasting, is the C-terminal cleavage product of profibrillin-1 protein. In addition, it increases hunger and fat storage by activating the hypothalamus nutrition center (5). According to certain earlier research, asprosin's protective properties on the myocardium aided in the healing of wounds. Additionally, it was discovered to have a considerable regulatory effect on left ventricular functioning (6). A novel measure for the severity of acute coronary syndrome and angina pectoris is asprosin (7). The body's white adipose tissue secretes asprosin, a novel hormone that was found in 2016 (8). The majority of body cells, including human cutaneous fibroblasts, pancreatic B cells, peripheral tissues, and organs, express the asprosin gene, Fibrillin1 (8-10). Most biological fluids contain the heterodimeric glycoprotein clusterin, which is produced by a variety of tissues. Considering clusterin's distribution and in vitro properties, several physiological roles have been postulated for it. Among these are membrane protection, lipid transport, sperm maturation, apoptosis initiation, complement modulation, hormone secretion, and promoting cell-cell interactions. One of the protein's most notable and unique properties is its commencement of illness states. such as polycystic kidney disease, renal tubular damage, glomerulonephritis, and neurodegenerative illnesses (11).

When obesity is present, a number of factors may cause adipocytes to express more Clusterin. Following this, circulating clusterin affects the liver in a number of ways, including decreased ApoA1 expression, dyslipidemia, poor insulin signaling, and maybe increased steatosis and inflammation. It also affects macrophages, which may raise the risk of CVD and contribute to cardiometabolic syndrome (12).

Subjects, Supplies, and Procedures

Subjects

This investigation included fifty (50) patients of Acute Myocardial Infarction patients during the period August 2023 to November 2023from Ibn Al-Bitar hospital, Ibn Al-Nafes hospital, and Al- Al-Imamain AL-Kadhimain hospital. The Heart enzymes and an ECG were used to confirm the diagnosis of IHD, which was established according to the medical background of the patient and the presence or absence of ST elevation. contrasted with 25 volunteers who were healthy subjects (Control) and matched for age and sex.

Diagnostic criteria:

a. ST-elevation myocardial infarction patients
Twenty-five patients with chest pain and ECG changes, and high levels of cardiac enzymes diagnosed as STEMI.

b. Individuals with myocardial infarctions that are not ST-elevated
NSTEMI is the diagnosis made for 25 people who had chest discomfort and ECG abnormalities suggestive of ischemia.

c. Control group:
25 participants were chosen to serve as the control group. These participants will either appear to be in good health or have not recently complained of heart issues.

Criteria for inclusion:

According to the WHO (World Health Organization) definition of MI (myocardial infarction), any patient with a high troponin level

plus one of the following:

1- ECG (electrocardiogram) change of myocardial infarction (ST elevation myocardial infarction and non-ST elevation myocardial infarction).
Patients and controls were age and gender matched
2- chest pain or its equivalent ischemic.

Exclusion criteria:

Diabetes mellitus (DM), Liver disease, renal impairment, trauma, malignancies, any infectious disease, and rheumatoid arthritis.

Blood Sample Collection

Patients and a healthy control group provided five milliliters of blood in serum separator vacuum sealers. After being divided, sera were promptly kept at -20°C until examination.

Assessment of the blood lipid composition:

Total cholesterol (TC), total glycerides (TG), low-density lipoprotein (LDL-C), high-density lipoprotein (HDLC), and very low-density lipoprotein (vLDL-C) were among the lipid profile measurements. Using diagnostic kits from Randox in the United Kingdom, the lipid profile was measured, and the 500 nm absorbance was used to measure the color that formed.

Measurement of Clusterin, Asprosin in the serum, and creatine kinase (CK-MB):

The Human Asprosin ELISA kit and the Human Clustrin ELISA kit (Cusabio, China) were used as diagnostic kits to measure the asprosin and Clusterin levels. measure the CK-MB (Ichroma, China). **Analytical statistics:**

Under Windows XP, the statistical software for social sciences (SPSS) version 25 is used to code and enter all of the data. The mean and standard error were used to summarize descriptive data (SD). A p-value of less than 0.05 was regarded as statistically significant.

Results:

When compared to a group of 25 healthy control participants who were matched for age and sex, 50 subjects with acute myocardial infarction (25 patients with ST-MI and 25 patients with non-ST-MI) had their serum levels of Asprosin and Clusterin estimated.

The investigation's findings demonstrated a significant rise in the intensity of Asprosin and Clusterin in acute myocardial infarction (ST-MI & Non-ST-MI) compared with healthy control ($p<0.001$), and statistical analysis showed no significant difference within patients, ST-MI & Non-ST-MI $p>0.05$ as shown in Table 2 and Figures 2 and 3.

Accordingly to the levels of lipid profile shown in Table 1, Patients with acute myocardial infarction had triglyceride, LDL, VLDL, and total cholesterol levels that were noticeably higher. compared to healthy individuals ($p<0.05$). while HDL-C showed a significant decrease ($p < 0.05$). However, there was no discernible variation in the lipid profile levels between the ST-MI and non-ST-MI patients.

CK-BM and cardiac-Tnt levels were significantly higher in the myocardial infarction group of patients than in the control group, as indicated in Table 1. Additionally, these biomarkers are used to diagnose myocardial infarction.

Table 1. The biochemical and morphological factors for the three groups under investigation

X=VS, and the values are Mean ± SD. n: number of cases; A mean and standard deviation were used to display the data; One-way ANOVA; P < 0.001 indicates high significance; P >0.05 indicates not significant. ; S: significant.

Parameters	Control	ST-MI	Non-ST-MI	P(ANOVA)-(T-Test)
NO.	25	25	25	……
Age (yrs)	55.35±9.18	51.81±7.33	53.77±8.61	NS
BMI (Kg/m²)	25.61±3.51	26.17±2.87	26.88±4.37	ST-MI x Non ST-MI : p =0.139 patients x C: P = 0.256
SBP (mmHg	125.53±4.33	128.83±8.87	131.26±13.64	ST-MI x Non ST-MI: p > 0.05 patients x C: P < 0.05
DBP (mmHg)	82.18±3.09	85.66±6.31	81.58±8.52	ST-MI x Non ST-MI: p =0.112 patients x C: P = 0.423
TC (mg/dl)	170.66 ±25.37	230.17 ±55.18	210.19 ±33.57	ST-MI x Non ST-MI: p =0.09 patients x C: P < 0.002
TG (mg/dl)	138.16±10.87	171.92±32.35	159.24±39.86	ST-MI x Non ST-MI: p =0.109 patients x C: P = 0.001
LDL -C (mg /dl)	78.05±15.44	146.11±54.76	121.16±26.09	ST-MI x Non ST-MI: p =0.07 patients x C: P = 0.001
VLDL -C (mg /dl)	24.42±2.78	35.12±8.39	30.67±9.09	ST-MI x Non ST-MI: p =0.288 patients x C: P = 0.0017
HDL -C (mg /dl)	53.44±11.64	31.86±3.21	38.86±6.16	ST-MI x Non ST-MI: p =0.416 patients x C: P = 0.001
CK – MB (ng /ml)	5.88±2.45	46.18±11.26	37.74±8.66	ST-MI x Non ST-MI: p =0.137 patients x C: P = 0.001
Tnt (ng/ml)	0.21± 0.77	0.54±0.64	0.36±0.96	ST-MI x Non ST-MI: p =0.04 patients x C: P = 0.001

Table 2: The Biochemical Parameters Asprosin and Clusterin in individuals with acute myocardial

Biochemical Parameters	Control	ST-MI	Non-ST-MI	P(ANOVA)-(T-Test)
NO.	25	25	25	……
Asprosin (ng/ml)	6.35 ± 2.77	21.78±7.09	16.53±9.66	ST-MI x Non st-MI: p =0.228 patients x C: P < 0.001
Clusterin (ng/ml)	18.11±4.99	66.55±7.34	33.41±9.47	ST-MI x Non st-MI: p =0.089 patients x C: P < 0.001

Sig. P < 0.05; elevated. Sig. P < 0.01; P> without an asterisk ** Less than 0.001 high sig., 0.05 * less than 0.01 sig.

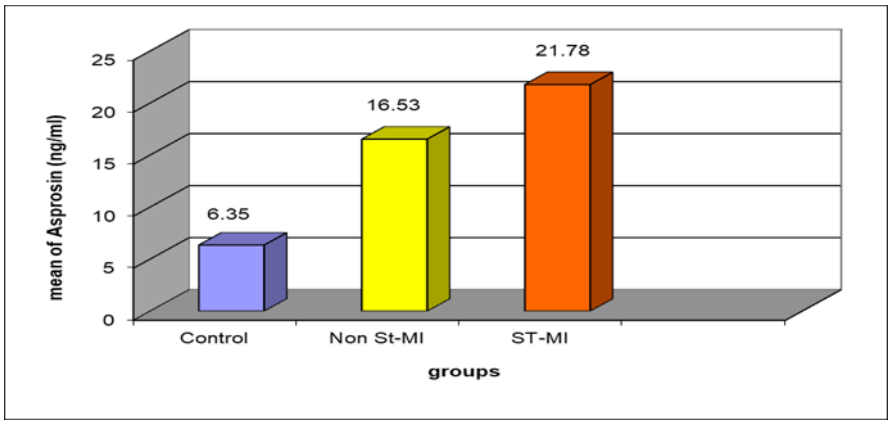

Figure 1. Asprosin mean values in patients with acute myocardial infarction compared to healthy individuals

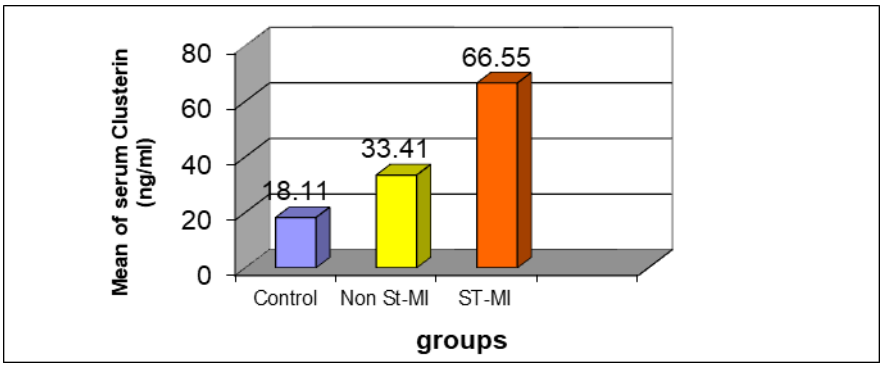

Figure 2: Mean of serum levels of Clusterin in all study groups.

Discussion

When the myocardium does not receive the oxygen it needs from the coronary artery, it can lead to acute myocardial infarction (AMI), which is defined as the necrosis of the heart muscle (13,14). ACS encompasses UA, STEMI, and NSTEMI, and includes acute myocardial infarction. A thrombus blocking the coronary artery is the cause of about 90% of MI cases (15,16). The assessment of cardiac biomarkers is the first step toward diagnosing AMI. It has helped physicians diagnose patients and plan treatments, which has decreased the fatality rate (16). Because cardiac troponins have a high specificity, they have replaced cytosolic enzymes in the diagnosis of myocardial infarction (MI). Additionally, biochemical biomarkers, such as CK-MB or troponins, have been introduced to estimate the likelihood of future MI episodes. These biomarkers are known as cardiac risk indicators (17,18). The current investigation sought to determine the relationship between myocardial infarction severity and blood levels of clusterin and asprosin. All study participants provided serum samples, from which Asprosin and Clusterin levels were determined. White adipocytes are the primary source of asprosin, a hormone that was only recently identified (19). White adipose tissue secretes asprosin, a recently identified orexigenic adipokine that acts centrally and controls glucose metabolism (20). The C-terminal cleavage product of profibrillin, mainly produced by white adipocytes

but potentially removed by β-pancreatic cells, was recently discovered as asprosin. The human placenta (21), the stomach fundus's surface epithelial cells, the kidneys' cortical distal tubule, and the heart's cardiomyocytes in rats (22) are among the tissues that express it. Asprosin stimulates appetite and affects adiposity by traversing the hepatic glucose release pathway of G-protein cyclic adenosine monophosphate (cAMP)/protein kinase A (PKA) and crossing the blood-brain barrier to activate the hypothalamic feeding circuits. Additionally, research has linked circulating asprosin to many metabolic diseases, such as obesity (29), diabetes mellitus (24-28), and polycystic ovarian syndrome (PCOS) (26,30). It has been suggested that asprosin is (ST-MI & Non ST-MI). The mean value of Asprosin in the serum of the healthy control group was (6.35 ± 2.77). While the mean value for serum Asprosin within the ST-MI patients group was (21.78±7.09), the mean value for serum Asprosin within the NST-MI patients group was(16.53±9.66). Table 2 displays a statistically significant difference (P value<0.001) between the sick groups and the healthy control group. This work agrees with earlier research by Yuan M and Six AJ, which hypothesizes that asprosin may be a predictor of cardiovascular illnesses and may indicate the severity of acute coronary syndrome in patients with unstable angina pectoris (32,33). Additionally, the current findings are consistent with a prior investigation conducted by Moradi. (34), which revealed that patients with atherosclerosis had a markedly elevated asprosin concentration. Asprosin's precise function is unknown, but it has been suggested that high levels of this hormone could improve heart muscle cells' mitochondrial respiration, shielding the heart from hypoxia (35,36). The current study's findings demonstrated that, in comparison to healthy controls, acute myocardial infarction patients had considerably higher levels of the lipid profile. The findings of this investigation are consistent with a previous study that demonstrated an independent relationship between triglycerides and lipid levels (37,38). Moreover, asprosin production by pancreatic cells has been linked to hyperlipidemic conditions; this initiates pathways that

In present study shows that serum Clusterin level increases with myocardial comparing individuals with infarction, compared to a healthy group of controls. These results agree with other results done by (39). Who first study claimed that patients with premature coronary artery disease (PCAD) had higher serum clusterin levels. Clusterin is crucial for the cardiovascular system; in this study, MI patients had higher serum levels of clusterin. The mean value of Clusterin in the serum of the healthy control group was (18.11±4.99). while the mean value for serum Clusterin within the STEMI patients group was (66.55±7.34), and the mean value for serum Clusterin within the NSTEMI patients group was(33.41±9.47). There was a significant difference between patient groups with health control (P value<0.001), as shown in Table 2. These results agree with another previous study that found increased Clusterin levels in the heart and serum at the early stage after myocardial infarction (MI). (40,41). The result of the current investigation agrees with earlier research performed by Min-Jung Kang (42). Serum clusterin was significantly increased in AMI patient samples compared to levels in healthy controls.

Conclusion:

As recommended, serum concentrations of Asprosin and Clusterin could be assessed as diagnostic biomarkers in acute myocardial infarction patients and be beneficial in the diagnosis of early-stage myocardial infarction patients. Additionally, measurements of asprosin and clusterin are highly helpful in predicting and preventing complications for patients with angina. These parameters have been implicated in the pathophysiology of myocardial infarction.

Conflict of interest: NIL

Funding: NIL

REFERENCES

1. Khan MA, Hashim MJ, Mustafa H, Baniyas MY, Al Suwaidi S, AlKatheeri R, et al. Global epidemiology of ischemic heart disease: results from the global burden of disease study. Cureus. 2020;12:e9349.

2. Gheorghe A, Griffiths U, Murphy A, Legido-Quigley H, Lamptey P, Perel P. The economic burden of cardiovascular disease and hypertension in lowand middle-income countries: a systematic review. BMC Public Health. 2018; 18:1.

3. A. Cranz, "Broken Heart Strings - Psychological Stress in Cardiac Patients after Chordae Tendineae Rupture," thesis, p. 24,2019.

4. M. J. M. Silvis et al., "Immunomodulation of the NLRP3 Inflammasome in Atherosclerosis, Coronary Artery Disease, and Acute Myocardial Infarction," J. Cardiovasc. Transl. Res., vol. 14, no. 1, pp. 23–34, 2021.

5. C. Duerrschmid, et al., Asprosin is a centrally acting orexigenic hormone, Nat. Med. 23 (2017) 1444–1453.

6. L. Zhu, et al., Expression and purification of Asprosin and its effects on cardiac function in mice, Acta Lab. Anim. Sci. Sin. 25 (2017) 368–372.

7. A.C. Acara, et al., A novel biochemical marker for predicting the severity of ACS with unstable angina pectoris: asprosin, Am. J. Emerg. Med. 36 (2018) 1504–1505.

8. Ugur, K., Aydin, S. Saliva and blood asprosin hormone concentration associated with obesity. Int J Endocrinol 2019. Article ID 2521096.

9. You, M., Liu, Y., Wang, B., Li, L., Zhang, H., He, H., et al., Asprosin induces vascular endothelial-to-mesenchymal transition in diabetic lower extremity peripheral artery disease. Cardiovasc Diabetol 2022; 21(1): 1-15.

10. Shabir, K., Brown, J. E., Afzal, I., Gharanei, S., Weickert, M. O., Barber, T. M., et al., Asprosin, a novel pleiotropic adipokine implicated in fasting and obesity-related cardiometabolic disease: Comprehensive review of preclinical and clinical evidence. Cytokine Growth Factor Rev. 2021; 60: 120-132.

11. Rosenberg, M. E., & Silkensen, J. Clusterin: physiologic and pathophysiologic considerations. The international journal of biochemistry & cell biology,1995; 27(7), 633-645.

12. Yu B, Yang Y, Liu H, Gong M, Millard RW, Wang YG, . Clusterin/Akt upregulation is critical for GATA-4 mediated cytoprotection of mesenchymal stem cells against ischemia injury. PLoS ONE.2016; 11:e0151542.

13. Bęćkowski, M. Acute coronary syndromes in young women–the scale of the problem and the associated risks. Kardiochirurgia i torakochirurgia polska= Polish journal of cardio-thoracic surgery,2015; 12(2), PP.134.

14. Ugwu, C., Nwankwo, S., Meludu, S., & Nnodim, J. Assessment of the risk of myocardial infarction among undergraduate students in a Nigerian tertiary institution. International Journal of Healthcare and Medical Sciences,2016; 2(11), PP. 60-65.

15. Azab, A., & Elsayed, A. Acute Myocardial Infarction Risk Factors and Correlation of its Markers with Serum Lipids. J Appl Biotechnol Bioeng,2017; 3(4),PP. 00075.

16. Alqahtan, A., Alshareef, S., Al-Hadlaq, Z., Almalki, A., ALHAYYAN, K., ALOUFI, S., Bin Barghash, A., ALMUDHISH, F., ALARAG, M., ALAMRI, A., ALANAZI, S., Alshahrani, S. A Non–ST-Segment Elevation Myocardial Infarction: An Emergent Condition- Main Role of Emergency Providers and Clinical Pathologists: A Review. *Journal of Medical and Life Science*, 2024; 6(4): 713-724. doi: 10.21608/jmals.2024.425349

17. Mythili, S., & Malathi, N. Diagnostic markers of acute myocardial infarction. Biomedical Reports,2015; 3(6), PP.743-748.

18. Faraj, H. Serum NT-pro BNP and Other Biochemical Markers in Patients with Cardiorenal Syndrome (CRS) in Thi-Qar province- Iraq. *Journal of Bioscience and Applied Research*, 2024; 10(4): 686-694. doi: 10.21608/jbaar.2024.311161.1064

19. Romere C, Duerrschmid C, Bournat J, Constable P, Jain M, Xia F, et al., Asprosin, a fasting-induced glucogenic protein hormone. Cell. 2016;165:566–579.

20. Mazur-Bialy AI. Asprosin-A Fasting-Induced, Glucogenic, and Orexigenic Adipokine as a New Promising Player. Will It Be a New Factor in the Treatment of Obesity, Diabetes, or Infertility? A Review of the Literature. Nutrients. 2021;13(2):620. https://doi. org/10.3390/nu13020620

21. Yu Y, He JH, Hu LL, Jiang LL, Fang L, Yao GD, et al. Placensin is a glucogenic hormone secreted by human placenta. EMBO Rep. 2020;21:e49530.

22. Kocaman N, Kuloğlu T. Expression of asprosin in rat hepatic, renal, heart, gastric, testicular, and brain tissues and its changes in a streptozotocin-induced diabetes mellitus model. Tissue Cell. 2020;66:101397.

23. Duerrschmid C, He Y, Wang C, Li C, Bournat JC, Romere C, et al. Asprosin is a centrally acting orexigenic hormone. Nat Med. 2017;23:1444–53.

24. Zhang L, Chen C, Zhou N, Fu Y, Cheng X. Circulating asprosin concentrations are increased in type 2 diabetes mellitus and independently associated with fasting glucose and triglyceride. Clin Chim Acta. 2017;489: 183–8.

25. Wang Y, Qu H, Xiong X, Qiu Y, Liao Y, Chen Y, et al. Plasma Asprosin concentrations are increased in individuals with glucose Dysregulation and correlated with insulin resistance and first-phase insulin secretion. Mediat Inflamm. 2018;2018:9471583.

26. Li X, Liao M, Shen R, Zhang L, Hu H, Wu J, et al. Plasma Asprosin levels are associated with glucose metabolism, lipid, and sex hormone profiles in females with metabolic-related diseases. Mediat Inflamm. 2018;2018: 7375294.

27. Groener JB, Valkanou A, Kender Z, Pfeiffenberger J, Kihm L, Fleming T, et al. Asprosin response in hypoglycemia is not related to hypoglycemia unawareness but rather to insulin resistance in type 1 diabetes. PLoS One. 2019;14:e0222771.

28. Baykus Y, Yavuzkir S, Ustebay S, Ugur K, Deniz R, Aydin S. Asprosin in umbilical cord of newborns and maternal blood of gestational diabetes, preeclampsia, severe preeclampsia, intrauterine growth retardation, and macrosemic fetus. Peptides. 2019;120:170132.

29. Wang M, Yin C, Wang L, Liu Y, Li H, Li M, et al. Serum Asprosin concentrations are increased and associated with insulin

resistance in children with obesity. Ann Nutr Metab. 2019;75:205–12.

30. Alan M, Gurlek B, Yilmaz A, Aksit M, Aslanipour B, Gulhan I, et al. Asprosin: a novel peptide hormone related to insulin resistance in women with polycystic ovary syndrome. Gynecol Endocrinol. 2019;35:220–3.

31. Wen, M. S., Wang, C. Y., Yeh, J. K., Chen, C. C., Tsai, M. L., Ho, M. Y., et al., The role of asprosin in patients with dilated cardiomyopathy. BMC Cardiovasc Disord. 2020; 20(1): 1-8.

32. Yuan M, Li W, Zhu Y, Yu B, Wu J. Asprosin: A Novel Player in Metabolic Diseases. Front Endocrinol (Lausanne). 2020;11:64. https:// doi.org/10.3389/fendo.2020.00064

33. Six AJ, Backus BE, Kelder JC. Chest pain in the emergency room: value of the HEART score. Neth Heart J. 2008;16(6):191- 6. https:// doi.org/10.1007/BF03086144

34. Moradi, N., Fouani, F. Z., Vatannejad, A., Bakhti Arani, A., Shahrzad, S., Fadaei, R. Serum levels of aprosin in patients diagnosed with coronary artery disease (CAD): a case-control study. Lipids Health Dis. 2021; 20(1):1-8.

35. Zhang L, Chen C, Zhou N, Fu Y, Cheng X. Circulating asprosin concentrations are increased in type 2 diabetes mellitus and independently associated with fasting glucose and triglyceride. Clin Chim Acta. 2017;489:183–188.

36. Lee T, Yun S, Jeong JH, Jung TW. Asprosin impairs insulin secretion in response to glucose and viability through TLR4/JNK-mediated inflammation. Mol Cell Endocrinol. 2019; 486:96–104.

37. Ben-Yehuda, O., Chen, S., Redfors, B., McAndrew, T., Crowley, A., Kosmidou, I., et al., Impact of large periprocedural myocardial infarction on mortality after percutaneous coronary intervention and coronary artery bypass grafting for left main disease: an analysis from the EXCEL trial. Euro Heart J. 2019; 40(24): 1930-1941.

38. Yameny, A. Lipid profile metabolism, pathophysiology, clinical correlations, and therapeutic strategies in cardiovascular and metabolic diseases. *Pharaonic Journal of Science*, 2025; *1*(1): 80-91. https://doi.org/10.71428/PJS.2025.0107

39. Zhu, H., Liu, M., Zhai, T., Pan, H., Wang, L., Yang, H., ... & Zeng, Y. High serum clusterin levels are associated with premature coronary artery disease in a Chinese population. Diabetes/metabolism research and reviews,2019; 35(4), e3128.)

40. Sansanwal P, Li L, Sarwal MM. "Inhibition of intracellular clusterin attenuates cell death in nephropathic cystinosis". Journal of the American Society of Nephrology. 2015; 26 (3): 612–25.

41. Lin CC, Tsai P, Sun HY, Hsu MC, Lee JC, Wu IC, Tsao CW, Chang TT, Young KC. "Apolipoprotein J, a glucose-upregulated molecular chaperone, stabilizes core and NS5A to promote infectious hepatitis C virus virion production". Journal of Hepatology. 2014; 61 (5): 984–93.

42. Min-Jung Kang, Yunseo Seong, Joyeta Mahmud, Binh Thanh Nguyen. Obscurin and Clusterin Elevation in Serum of Acute Myocardial Infarction Patients. Bulletin of the Korean Chemical Society,2020; 41 (3), 266-273.