Original Article

Correlation of Apelin and Leptin with Troponin I Titer among Patients with Acute Myocardial Infarction in Babylon Province

Ahmed Fessl Falih Al-Baaj[1], Mufeed Jalil Ewadh[1,2], Shokry Faaz Alsaad[3]

[1]Department of Biochemistry, University of Babylon, College of Medicine, Hilla, Iraq, [2]Department of Medical Laboratories Techniques, Al-Amal College for Specialized Medical Sciences, Karbala, Iraq, [3]Department of Internal Medicine, University of Babylon, College of Medicine, Hilla, Iraq

Abstract

Background: Acute myocardial infarction (AMI) is a clinical condition characterized by the necrosis of heart muscle tissue resulting from an imbalance between myocardial demand and coronary blood flow. **Objectives:** The aim of the present study was to determine the levels of serum APLN and leptin in AMI patients and their correlation with TnI titer among different types of AMI (STEMI and NSTEMI). **Materials and Methods:** In total, 60 patients with AMI were enrolled in this study and divided into two groups according to the type of AMI. Group 1 (G1) ST-elevation myocardial infarction (STEMI) and group 2 (G2) non-ST-elevation myocardial infarction (NSTEMI). In addition to 60 participants as a control group. The period the study extended from March 2022 to February 2023. This work was done at the Department of Biochemistry, College of Medicine University of Babylon and Marjan Medical City in Hilla City, Iraq. Determination of serum apelin (APLN) and leptin was done by Human ELISA kits. Determination of troponin I (TnI) titer was done by cobas e411 from Roche diagnostic. **Results:** The results of the current study show positive correlations in STEMI and NSTEMI groups between TnI with both APLN and leptin among AMI patients ($r = 0.910$, $r = 0.902$, $r = 0.476$, $r = 0.389$). **Conclusion:** patients with AMI in Babylon province have high levels of APLN and leptin, with higher levels in STEMI group than NSTEMI group. This pattern of results indicates a role for these markers in forming thrombosis, atherosclerosis, oxidative stress, and the occurrence and prognosis of myocardial infarction.

Keywords: Acute myocardial infarction, apelin, leptin, troponin I

Introduction

The clinical issue of acute myocardial infarction (AMI) refers to the acute death of heart muscle tissue caused by an imbalance between the demand for oxygen in the myocardium and the supply of oxygen-rich blood through the coronary arteries.[1] Typically, AMI results from the formation of a thrombus that obstructs blood flow to the heart due to rupture or erosion of an atherosclerotic plaque in the coronary artery. If left untreated, the artery affected by the infarction remains blocked in around 30% of patients.[2]To put it differently, AMI can be defined as the death of heart cells caused by a decrease in oxygen supply, which occurs when the coronary arteries become obstructed. As cardiac cells primarily rely on oxidative metabolism for energy production, they are highly sensitive to fluctuations in intracellular oxygen levels. When the coronary arteries become obstructed, oxygen concentration decreases, and anaerobic ATP synthesis is activated, leading to energy deprivation and the death of cardiac cells.[3]

AMI is more common among older adults, with an average age of 65.6 years for males and 72.0 years for females being the first incidence. Although the incidence of AMI has decreased in developed countries due to better healthcare systems and public health interventions, it is on the rise in developing regions such as South Asia, parts of Latin America, and Eastern Europe.[4]

The symptoms of AMI include chest pain, which may radiate to the left arm or left side of the neck, shortness of

Access this article online

Quick Response Code:

Website: https://journals.lww.com/mjby

DOI: 10.4103/MJBL.MJBL_621_23

Address for correspondence: Mr. Ahmed Faysel Falih Al-Baaj, Department of Chemistry and Clinical Biochemistry, College of Medicine, University of Babylon, Hillah, Iraq. E-mail: fesslahmed3@gmail.com

Submission: 25-May-2023 **Accepted:** 06-Jul-2023 **Published:** 23-Jan-2026

How to cite this article: Al-Baaj AFF, Ewadh MJ, Alsaad SF. Correlation of apelin and leptin with troponin I titer among patients with acute myocardial infarction in Babylon Province. Med J Babylon 2025;22:1149-54.

breath, sweating, nausea, vomiting, irregular heartbeats, anxiety, fatigue, and other factors.[5] Along with chest pain, warning signs of a heart attack include high blood pressure, tightness, squeezing, burning sensations, aching, and heaviness in the chest lasting for more than 10 minutes, pain in the left shoulder or left arm, extending up to the neck or along the jawline, shortness of breath, profuse sweating, dizziness, muscle weakness, nausea or vomiting, choking with smoke inhalation, anxiety or stress, feeling of impending doom, and depression.[6]

AMI can be classified into two types based on electrocardiogram (ECG) readings: ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI).[7] STEMI is characterized by an acute coronary thrombosis or persistent ST-segment elevation of at least 1 mm in at least two contiguous electrocardiographic leads. NSTEMI is characterized by ischemic symptoms at rest resulting from an acute coronary plaque rupture or erosion lasting at least 10 min and occurring within 24 h before hospital admission. NSTEMI also involves elevated cardiac biomarkers within 24 h after initial presentation.[8]

Apelin (APLN) is an endogenous peptide hormone composed of 35 amino acids and identified as a ligand of the G protein-coupled receptor APJ. APLN belongs to the family of adipokines, which are bioactive mediators released by adipose tissue.[9] Several cell types in the body can synthesize APLN protein, For example, the central nervous system (CNS), pituitary gland, and mammary glands, with lower values in kidneys and skeletal muscles. The highest APLN concentrations have been found in adipose tissue, gastric exocrine, and endocrine cells.[10]

APLN is considered to be a potent vasodilator by stimulating endothelial nitric oxide synthase (eNO); eNO stimulates soluble guanylate cyclase resulting in increased cyclic guanosine monophosphate [cGMP] that leads to vasodilation effect and regulation of blood pressure.[11] a previous study was done by Yümün *et al*.[12] concluded that APLN could be considered an independent risk factor for coronary heart disease.

Leptin is a 146-residue polypeptide hormone that is normally produced by adipocytes. It was discovered by studying genetically obese mice in the 1960s.[13] Leptin acts on cell receptors in the arcuate and ventromedial nuclei, as well as other parts of the hypothalamus and dopaminergic neurons of the ventral tegmental area. Leptin is thought to serve as an indicator of energy stores (lipostat), as well as a modulator of energy balance.[14] Circulating leptin serves to communicate the state of body energy repletion to the CNS to suppress food intake and permit energy expenditure.[15]

Recently MayerBruthans Jr *et al*.[16] concluded that high leptin concentration entails an increased risk of mortality in patients with acute coronary syndrome (ACS) as leptin connects directly to obesity and metabolic syndrome. Also, Attia *et al*. studied the prognostic value of leptin in the different types of CAD.[17]

Troponins are regulatory proteins that assist in cardiac and skeletal muscle contraction.[18] Skeletal and cardiac troponin isoforms differ in structure, with the cardiac troponin complex comprised of troponin C, troponin I (TnI), and troponin T (TnT). TnI binds to actin and inhibits the interaction of actin and myosin; TnI is an extremely sensitive biomarker for AMI and is the current biomarker of choice for the diagnosis, risk-stratification, and clinical management of AMI.[19] TnI appears in the plasma 4–8 h or earlier with high-sensitivity troponin assays after symptoms of AMI and is best measured 12 h after the start of chest pain.[20]

The present study aims to determine the levels of serum APLN and leptin in AMI patients and their correlation with TnI titer among different types of AMI (STEMI and NSTEMI).

Materials and Methods

Date and duration

The period extended from March 2022 until February 2023. This work was done in the Department of Biochemistry, College of Medicine University of Babylon and Marjan medical city in Hilla City, Iraq.

Study design

The study design was a case–control study.

Patients and control

A total of 60 patients with AMI were enrolled in this study, divided into two groups according to the type of AMI group 1 (G1) STEMI and group 2 (G2) NSTEMI, and 60 participants were apparently healthy control group (CG).

Inclusion criteria

All patients with AMI, STEMI, and NSTEMI were diagnosed by ECG and TnI (qualitative).

Exclusion criteria

1- Patients with congenital heart disease.
2- Patients with other ischemic heart disease.
3- Patients with all types of cancer.

Determination of serum APLN

Determination of serum APLN levels in patient and CG were done by using Human ELISA kits and according to the manufacturer manual.[9]

Determination of serum leptin

Determination of serum leptin was done by Human ELISA kits and according to the manufacturer manual.[10]

Determination of TnI titer

Determination of troponin titer was done by cobas e411 instrument from Roche diagnostic according to the manufacturer's instructions.[11]

Ethical Approval

The study was conducted in compliance with ethical principles based on the Declaration of Helsinki. Patients provided both verbal and written consent before the sample was taken. The study protocol, as well as the subject information and consent form, were reviewed and approved by a local Ethics Committee, as evidenced by document number fifth session, which includes the approval date of November 23, 2021.

Results

The mean age + SD of AMI patients and control were shown in Table 1.

In this study, serum APLN was significantly higher in AMI patient groups compared with normal subjects (CG) *P* value < 0.001. In this study, the level of leptin is highly elevated in STEMI group than non-STEMI group. Serum APLN was significantly higher in STEMI group when compared to non-STEMI group (*P* value < 0.001) [Table 2].

In this study, serum leptin was significantly higher in AMI patient groups compared with normal subjects (CG) *P* value < 0.001. Also, the level of leptin was more elevated in STEMI group than non-STEMI group. Serum leptin was significantly higher in STEMI group when compared to non-STEMI group (*P* value < 0.05) [Table 3].

The results of the current study were found toward strong positive correlations in STEMI and NSTEMI groups between TnI and APLN among AMI patients. Figures 1 and 2 show the correlations between TnI and APLN for patients groups (STEMI and NSTEMI), respectively.

The results of the current study were found toward moderate positive correlations between TnI and Leptin among AMI patients, *P* value < 0.05. Figures 3 and 4 show the correlations between TnI and Leptin for patient groups (STEMI and NSTEMI), respectively.

Discussion

Age distributions of AMI patients in this study show a peak level in the age of 58–67 years and the least between 48 and 57 year's age groups. This finding is in agreement with a previous study done in Iraq by Amen *et al.*[1] which found the highest rate of AMI in the same age category. The high incidence of AMI in this age group

Table 1: Age and gender for patients and control

	Group	No.	Mean ± SD
Age (years)	STEMI	27	63.3 ± 10.5
	Non-STEMI	33	60.8 ± 8.0
	Control	60	62.4 ± 9.4
P value	STEMI versus control group (*P* > 0.05) Non-STEMI versus control group (*P* > 0.05) STEMI versus non-STEMI group (*P* > 0.05)		

Table 2: The Mean ± SD of apelin in AMI compared to control group

Parameter	Subjects	No.	Mean	Standard deviation
Apelin (pg/mL)	STEMI	27	389.3	±14.1
	Non-STEMI	33	220.6	±11.0
	Control	60	70.7	±8.2
P value	STEMI versus control group (*P* < 0.001) non-STEMI versus control group (*P* < 0.001) STEMI versus non-STEMI group (*P* < 0.001)			

Table 3: The mean ±SD of serum leptin in AMI compared to control group

Parameter	Subjects	No.	Mean	Standard deviation
Leptin (ng/mL)	STEMI	27	23.5	±2.4
	Non-STEMI	33	15.3	±3.1
	Control	60	6.8	±1.8
P value	STEMI versus control group (*P* < 0.001) non-STEMI versus control group (*P* < 0.001) STEMI versus non-STEMI group (*P* < 0.05)			

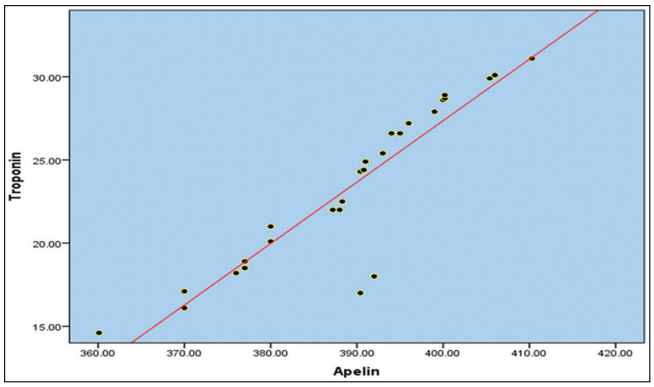

Figure 1: Correlation between troponin I and apelin in STEMI patients ($R = 0.910$, P value < 0.001)

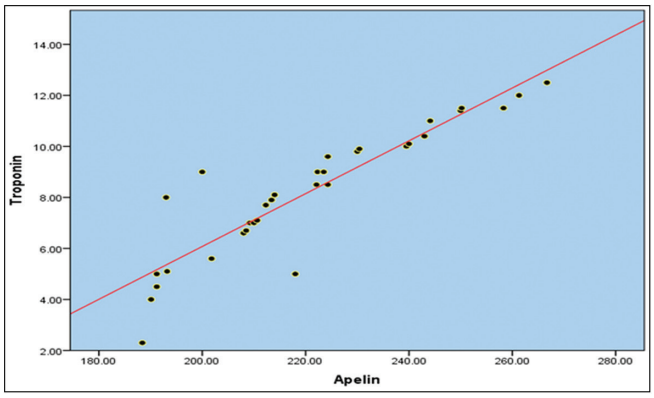

Figure 2: Correlation between troponin I and apelin in NSTEMI patients ($R = 0.902$, P value < 0.001)

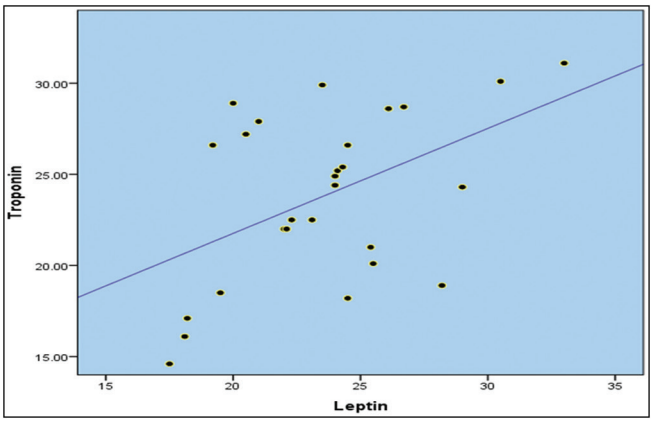

Figure 3: Correlation between troponin I and leptin in STEMI patients ($R = 0.476$, P value < 0.05)

category found in the current study is lower than the age group category concluded in previous studies, such as Erol *et al.*[21] in Turkey and Millett *et al.*[22] in the United Kingdom. This difference in a high prevalence of AMI with specific age categories in the present study when compared to previous studies in other countries is related to the difference in environmental and regional conditions such as air pollution, exposure to oxidant agents, fatty diets, and lack of physical activity.

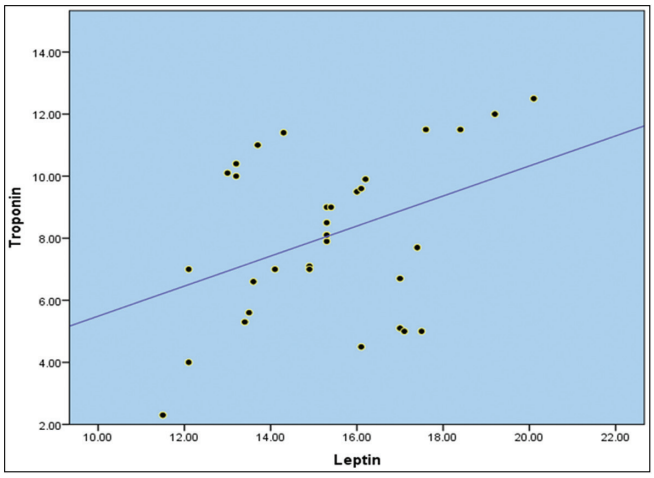

Figure 4: Correlation between troponin I and leptin in STEMI patients ($R = 0.389$, P value < 0.05)

There was no significant difference in this study in the mean age between AMI patients and CG (P value > 0.05). In the current study, extremely high levels of APLN were found in STEMI group higher than NSTEMI group; this is consistent with what was previously mentioned about the fact that an increase in APLN production is a normal response of the heart muscle during events that lead to infarction, as APLN inhibited thrombin-induced and collagen-induced platelet activation and aggregation, reduces both systolic and diastolic pressure.[23] The larger the infarction, the greater the response of the heart muscle and, consequently, the higher the levels of APLN.

Guzelburc *et al.*[24] also found higher APLN levels in patients STEMI than NSTEMI in a study, including different types of ACS in the University of Health Sciences Haydarpasa Numune, Turkey, suggested that higher APLN levels represent a reflection of complete occlusion of one of the coronary arteries, faster cell apoptosis and a higher degree of necrosis of cardiac muscle cells. This gives the APLN great diagnostic and categorical value.

A possible mechanism to explain the high leptin levels in AMI patients is the role of leptin in atherogenesis. Leptin transmits a signal by binding to its receptor LepR, which is mainly found in the hypothalamus but is also expressed in macrophage, endothelial cells, and smooth muscle cells, which enhance inflammation, dysfunction of endothelial cells, and migration of smooth muscles to the injured intima of the myocardiocyte and that considered an important event in the formation of plaque, thus contributing to AMI.[25]

The leptin result in this study is in agreement with Syed *et al.*[26] that also found high leptin results in AMI patients. Leptin, in addition to its angiogenic activity, leptin increases oxidative stress in endothelial cells by inducing reactive oxygen species formation in phagocytic as well as nonphagocytic cells and activation of NADPH oxidase.[27]

Table 4: The mean ±SD of troponin I in AMI patients of both types

Parameter	Subjects	No.	Mean	Standard deviation
Troponin I (ng/mL)	STEMI	27	23.8	±5.6
	Non-STEMI	33	8.1	±2.2
P value	STEMI versus non-STEMI group ($P < 0.001$)			

In the current study, there was a significant difference (P value < 0.05) in the level of leptin between STEMI and non-STEMI, suggesting that a higher leptin level is associated with complete and prolonged occlusion of a coronary blood vessel that causes STEMI. While moderate elevation level of leptin associated with NSTEMI usually results from severe coronary artery narrowing, transient occlusion, or microembolization of thrombus and/or atheromatous material.

These results are concordant with Belik *et al*.[28] which found similar associations while studying the degree of lesion of AMI as shown in Table 4.

The strong positive correlation between aplein and TnI can explain as follow: high levels of troponin express greater necrosis in the myocardium, which leads to the release of higher levels of APLN. APLN had protective rules for the coronary artery system, such as vasodilation and degradation of inflammation. Endothelial damage caused by ischemia leads to increased vascular permeability, which increases not only water permeability but also protein leakage and enhances inflammation. APLN was found to alleviate myocardial injury by inhibiting vascular permeability.[29]

The moderate positive correlation between leptin and TnI can explain as follows: higher leptin levels are associated with higher troponin levels as leptin increase the production of pro-inflammatory cytokine and different types of acute phase protein, triggering an inflammatory process and contributing to atherosclerosis and plaque formation, this increase in leptin levels may contribute to an exacerbation of necrosis volume and elevated troponin levels.[30] STEMI group had higher leptin and troponin levels, as observed in this study.

Conclusions

The current study concludes that patients with AMI in Babylon province have high levels of APLN and leptin with higher levels in STEMI group than NSTEMI group. This pattern of results indicate a role for these markers in the formation of thrombosis, atherosclerosis, oxidative stress and the occurrence and prognosis of myocardial infarction.

Acknowledgments

The authors would like to thank the staff of the Biochemistry Department at College of Medicine, University of Babylon, Iraq, for their efforts and facilities to complete the task of this study. Also, the authors would like to thank the staff of Marjan medical city Hospital, Hilla City, Iraq, for their assistance in the collection of samples.

Conflict of interest

No conflict of interest is present in the current study.

Financial support and sponsorship

Self-funding.

References

1. Amen SO, Baban ST, Yousif SH, Hawez AH, Baban ZT, Jalal DM. Prevalence of the most frequent risk factors in Iraqi patients with acute myocardial infarction. Med J Babylon 2020;17:6-18.
2. Mohammed SY, Rasool MT. The prognostic value of serum uric acid in ST-segment elevation myocardial infarction. Med J Babylon 2021;18:230.
3. Smit M, Coetzee AR, Lochner A. The pathophysiology of myocardial ischemia and perioperative myocardial infarction. J Cardiothorac Vasc Anesth 2020;34:2501-12.
4. Berg DD, Bohula EA, Morrow DA. Epidemiology and causes of cardiogenic shock. Curr Opin Crit Care 2021;27:401-8.
5. Tedeschi D, Rizzi A, Biscaglia S, Tumscitz C. Acute myocardial infarction and large coronary thrombosis in a patient with COVID-19. Catheter Cardiovasc Interv 2021;97:272-7.
6. Baloglu UB, Talo M, Yildirim O, San Tan R, Acharya UR. Classification of myocardial infarction with multi-lead ECG signals and deep CNN. Pattern Recognit Lett 2019;122:23-30.
7. Aslanger EK, Yıldırımtürk O, Şimşek B, Bozbeyoğlu E, Şimşek MA, Karabay CY, *et al*. Diagnostic accuracy oF electrocardiogram for acute coronary OCClUsion resuLTing in myocardial infarction (DIFOCCULT Study). IJC Heart Vasc 2020;30:100603.
8. Liu WC, Lin CS, Tsai CS, Tsao TP, Cheng CC, Liou JT, *et al*. A deep-learning algorithm for detecting acute myocardial infarction. EuroIntervention 2021;17:765-73.
9. Antushevich H, Wójcik M. Apelin in disease. Clin Chim Acta 2018;483:241-8.
10. Chen J, Li Z, Zhao Q, Chen L. Roles of apelin/APJ system in cancer: Biomarker, predictor, and emerging therapeutic target. J Cell Physiol 2022;237:3734-51.
11. Deniz R, Baykus Y, Ustebay S, Ugur K, Yavuzkir S, Aydin S. Evaluation of elabela, apelin and nitric oxide findings in maternal blood of normal pregnant women, pregnant women with pre-eclampsia, severe pre-eclampsia and umbilical arteries and venules of newborns. J Obstet Gynaecol 2019;39:907-12.
12. Yümün G, Gürkan S, Yilmaz A, Güzel S. The role of serum apelin levels in peripheral artery disease. Indian J Surg. 2022;21:1-5.
13. Gao N, Qi X, Dang Y, Li Y, Wang G, Liu X, *et al*. Establishment and validation of a risk model for prediction of in-hospital mortality in patients with acute ST-elevation myocardial infarction after primary PCI. BMC Cardiovasc Disord 2020;20:1-0.
14. Obradovic M, Sudar-Milovanovic E, Soskic S, Essack M, Arya S, Stewart AJ, *et al*. Leptin and obesity: Role and clinical implication. Front Endocrinol 2021;12:585887.
15. Friedman JM. Leptin and the endocrine control of energy balance. Nat Metab 2019;1:754-64.
16. MayerBruthans OJ Jr, Seidlerová J, Gelžinský J, Kučera R, Karnosová P, Mateřánková M, *et al*. High leptin status indicates

an increased risk of mortality and heart failure in stable coronary artery disease. Nutr Metab Cardiovasc Dis 2022;32:2137-46.
17. Attia S, Abdulla B Saeed K. Role of leptin as a risk factor and prognostic marker in patients with acute coronary syndrome. Egypt J Hosp Med 2022;87:1322-27.
18. Mazloomi S, Khodadadi I, Alizadeh N, Shafiee G. Association of glutamate cystein ligase (GCL) activity peroxiredoxin 4 (prxR4) and apelin levels in women with preeclampsia. Pregnancy Hypertens 2021;23:163-8.
19. Abdel Fatah MF, Elshafie MM, Essam Eldin ZM, Youssef AM. Assessment of serum leptin levels in pediatric patients of beta thalassemia. Egypt J Hosp Med 2022;87:2091-5.
20. Fitzgerald RL, Hollander JE, Peacock WF, Limkakeng AT, Breitenbeck N, Blechschmidt K, *et al*. Analytical performance evaluation of the Elecsys® Troponin I Gen 5 STAT assay. Clin Chim Acta 2019;495:522-8.
21. Erol MK, Kayıkçıoğlu M, Kılıçkap M, Arın CB, Kurt IH, Aktaş I, *et al*. Baseline clinical characteristics and patient profile of the TURKMI registry: Results of a nation-wide acute myocardial infarction registry in Turkey. Anatolian J Cardiol 2020;24:43-53.
22. Millett ER, Peters SA, Woodward M. Sex differences in risk factors for myocardial infarction: Cohort study of UK Biobank participants. BMJ 2018;363:363-4.
23. Ying Z, Jiansong Y, Yong W, Shubin Q. Serum apelin predicts spontaneous reperfusion of infarct-related artery in patients with ST-segment elevation myocardial infarction. Coron Artery Dis 2019;30:103-8.
24. Guzelburc O, Demirtunc R, Altay S, Kemaloglu OT, Tayyareci G. Plasma apelin level in acute myocardial infarction and its relation with prognosis: A prospective study. JRSM Cardiovasc Dis 2021;10:2048004020963970.
25. Baig M, Alghalayini KW, Gazzaz ZJ, Atta H. Association of serum omentin-1, chemerin, and leptin with acute myocardial infarction and its risk factors. Pak J Med Sci. 2020;36: 1183-8.
26. Syed AH, Lohana S, Aung NH, Memon MK, Shaikh A, Memon S, *et al*. Correlation of leptin with acute myocardial infarction: A case–control study. Cureus 2020;12:121-4.
27. Gradinaru D, Khaddour H, Margina D, Ungurianu A, Borsa C, Ionescu C, *et al*. Insulin-leptin axis, cardiometabolic risk and oxidative stress in elderly with metabolic syndrome. Exp Clin Endocrinol Diab 2018;126:445-52.
28. Belik E, Gruzdeva OV, Dyleva YU, Naumov DY, Barbarash OL. Relationship of the degree of coronary lesion and serum adipokine levels in patients with myocardial infarction. Eur Heart J 2021;42:ehab724-1166.
29. Zhang P, Fu R, Liu H, Li W, Zhang Y, Song N, Liu H, Yao N, Xue J, Yin L, Chao C. Apelin-13 alleviates hexavalent chromium-induced myocardial injury by inhibiting ros-mediated oxidative damage and regulating multiple signals.
30. Vijayalakshmi TN, Kavitha S, Manohar G, Priyadharshini M. Serum leptin level and its correlation with angiographic severity of coronary artery disease. Int J Sci Study 2021;9:125-9.