MINISTRY OF HIGHER EDUCATION AND SCIENTIFIC RESEARCH UNIVERSITY OF DIYALA COLLEGE OF MEDICINE



STUDY OF LIPID PROFILE, GLYCOSYLATED HEMOGLOBIN, AND INFLAMMATORY MARKER IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Submitted to the council of the College of Medicine, Diyala University, In Partial Fulfillment of Requirements for the Bachelor Degree in Medicine

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ABSTRACT

We measured fasting serum total cholesterol (TC), triglycerides (TG), and low- and high-density lipoprotein cholesterol (LDL-C and HDL-C) and HbA1C and CRP levels in 100 individuals in Baqubeh general hospital – CCU.

They was present in fourth groups, the first 25 was control and healthy people.

The second group was 25 patients diagnosed with AMI.

The third group was 25 patients diagnosed with AMI plus hypertension.

The fourth group was 25 patients diagnosed with AMI plus diabetes mellitus.

And there's an effect between variable levels of lipid profile

, HbA1C and CRP and acute myocardial infarction according to studies and statistics conducted in this research.

INTRODUTION

<u>Acute myocardial infarction</u> is myocardial necrosis resulting from acute obstruction of a coronary artery.

Symptoms include chest discomfort with or without dyspnea, nausea, and/or diaphoresis.

Diagnosis is by electrocardiography (ECG) and the presence or absence of serologic markers. Treatment is antiplatelet drugs, anticoagulants, nitrates, beta-blockers, statins, and reperfusion therapy. For ST-segmentelevation myocardial infarction, emergency reperfusion is via fibrinolytic drugs, percutaneous intervention, or, occasionally, coronary artery bypass graft surgery. For non-ST-segment-elevation myocardial infarction, reperfusion is via percutaneous intervention or coronary artery bypass graft surgery.

<u>Dyslipidemia</u> is *elevation* of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein cholesterol level that contributes to the development of <u>atherosclerosis</u>.

Causes may be primary (genetic) or secondary. Diagnosis is by measuring plasma levels of total cholesterol, TGs, and individual lipoproteins. Treatment involves dietary changes, exercise, and lipidlowering drugs.

<u>Hypertension</u> is sustained elevation of resting systolic blood pressure ($\geq 130 \text{ mm Hg}$), diastolic blood pressure ($\geq 80 \text{ mm Hg}$), or both. Hypertension with no known cause (primary; formerly, essential, hypertension) is most common.

Hypertension with an identified cause (secondary hypertension) is usually due to primary aldosteronism. Sleep apnea, chronic kidney disease, obesity, or renal artery stenosis are other causes of secondary hypertension. Usually, no symptoms develop unless hypertension is severe or long-standing.

Diagnosis is by sphygmomanometry.

Tests may be done to determine cause, assess organ damage, and identify other cardiovascular risk factors.

Treatment involves lifestyle changes and medications, including diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, and calcium channel blockers.

<u>Diabetes mellitus</u> is impaired insulin secretion and variable degrees of peripheral insulin resistance leading to hyperglycemia.

Early symptoms are related to hyperglycemia and include polydipsia, polyphagia, polyuria, and blurred vision.

Later complications include vascular disease, peripheral neuropathy, nephropathy, and predisposition to infection.

Diagnosis is by measuring plasma glucose.

Treatment is diet, exercise, and drugs that reduce glucose levels, including insulin, oral antihyperglycemic drugs, and non-insulin injectable drugs.

Complications can be delayed or prevented with adequate glycemic control; heart disease remains the leading cause of mortality in diabetes mellitus.

Elevated levels of serum total cholesterol (TC), triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C) and low levels of highdensity lipoprotein cholesterol (HDL-C)

And uncontrolled diabetes mellitus (elevated HbA1C), uncontrolled hypertension (elevated blood pressure) and elevated CRP.

Is a majors modifiable risk factors for cardiovascular disease, all components are associated with increased incidence of acute myocardial infarction (AMI).

Intensive treatment of Dyslipidaemia, HTN and DM. stabilizes atherosclerosis and promotes its regression, and reduces all-cause and cardiovascular mortality in patients with AMI.

AIM OF STUDY

To evaluate the effect of lipid profile, HbA1C and CRP on patients with acute myocardial infarction.

PATIENT AND METHOD

The study was conducted during the period August- 2022 to the March - 2023

Individuals ≥ 18 years of age were enrolled in the study on a consecutive basis as they presented for check-up or for management of acute myocardial infarction-related admission until the predetermined number (n = 100) was met .

Patients with acute illnesses admitted to the CCU and diagnosed with AMI all of them was tokens samples of venous blood to measure lipid profile and HbA1C and CRP as part of management of them.

All participants agreed to have their lipid profile measured by the study investigator as part of their management and for the data to be used in the study.

There were no refusals to participate. The study was approved by the ethics committees in the participating hospital: Baqubeh general hospital at Baqubeh - Diyala

Venous blood levels of TC, TG, LDL-C, HDL-C, HbA1C, and CRP were measured.

Measurements were made on the day of collection after 8–12 hours of fasting or upon admission of patients with AMI.

Serum levels of TC, HDL-C and TG were measured using fully automated methods,

The ARCHITECT *c***4000 clinical chemistry analyzer** demonstrates high-quality testing results and rapid STAT turnaround time.

The ARCHITECT *c*4000 enhances laboratory productivity and provides users high confidence in clinical results.

The ARCHITECT *c*4000 offers a maximum throughput of up to 800 tests per hour. Featuring a load-up capacity of 100 samples with 35 priority positions, the ARCHITECT *c*4000 allows for up to 90 refrigerated reagent positions on board, plus Integrated Chip Technology (Na+, K+ and Cl-).

For *in vitro* diagnostic use only.

Methodology for assays:

TC, by enzymatic

TG, by glycerol phosphate oxidase

HDL, by accelerator selective detergent

And unfortunately the rest of assays isn't available and the technical team of laboratory don't tell us about its methods.

Reference values for optimal and high levels of serum lipids were based on the National Cholesterol Education Program

As follows: optimal TC < 200 mg/dL, borderline 200–239 mg/dL, high \geq 240 mg/dL

Optimal TG 199 mg/dL for HDL-C, we used 40 mg/dL as the cut-off for both sexes

Regarding LDL-C, for patients with cardiovascular disease or diabetes optimal level was < 100 mg/dL

For individuals with ≥ 2 risk factors this was < 130 mg/dL and for individuals with ≤ 1 risk factors it was < 160 mg/dL

 $CRP < 5.0 \mbox{ mg}\label{eq:cress}$ MbA1C 4.2 -6.2 %

Acute myocardial infarction was diagnosed based on the symptoms of infarction is deep, substernal, visceral pain, described as aching or pressure, often radiating to the back, jaw, left arm, right arm, shoulders, or all of these areas and electrocardiographic, echocardiographic, troponin test and coronary angiographic studies or prior percutaneous or surgical coronary revascularization .

Definition of risk factors was similar to criteria set in the Euro Heart Survey for patients with acute coronary syndromes

Current smoking was defined as smoking up to 1 month before enrolment. Hypertension was defined as prior diagnosis by a physician, current use of blood pressure lowering medications

Or known blood pressure values of ≥ 140 mmHg systolic or ≥ 90 mmHg diastolic on ≥ 2 occasions.

Diabetes was defined as prior diagnosis by a physician or current use of hypoglycaemic medications.

STATISTICAL METHODS

Data are presented as mean plus standard deviation (SD) or median values for continuous variables and as absolute and relative frequencies for categorical variables.

Calculations of the mean values excluded the extremely high values of TC (> 350 mg/dL), TG (> 500 mg/dL), or LDL-C (> 280 mg/dL) or HbA1C (> 15 %) or CRP (> 55 mg\l).

No exclusions were made when the median values were determined.

Comparisons between groups were performed using the P-value to compare results of 4 groups.

Multiple regression analyses were applied for serum lipoproteins and inflammatory marker as dependent variables and age, sex and smoking as independent variables.

Results were classified according to 5 sheets.

RESULT

In (Table 1-1) shows non- significant difference of the triglycerides (TG) level between all groups of healthy individuals and patients with myocardial infarction (MI), myocardial infarction association with hypertension (HT), and myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
169.3396 ± 127.177095	137 ± 123.6406787	N.S
Control	MI+HT	P-value
169.3396 ± 127.177095	151 ± 124.0202537	N.S
Control	MI+DM	P-value
169.3396 ± 127.177095	165 ± 118.7181115	N.S

Table 1-1 shows the comparison of TG level between health individuals and patients with myocardial infarction (MI), myocardial infarction association with hypertension (HT), myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value. <0.05. *P<0.05, **P<0.001, ***P<0.0001

In (Table 1-2) shows significant decrease of Cholesterol level between groups of healthy individuals and patients with myocardial infarction (MI), myocardial infarction association with hypertension (HT) and Non- significant difference of myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
173.2576 ± 42.79575176	145± 47.64806391	*
Control	MI+HT	P-value
173.2576 ± 42.79575176	138± 41.57815933	*
Control	MI+DM	P-value
173.2576 ± 42.79575176	173± 43.49616841	N.S

Table 1-2 shows the comparison of Cholesterol level between health individuals and patients with Myocardial infarction (MI), myocardial infarction association with hypertension (HT), and myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value <0.05. *P<0.05, **P<0.001, ***P<0.0001

 In (Table 1-3) shows highly significant decrease of high -density lipoprotein (HDL) level between groups of healthy individuals and patients with myocardial infarction association with hypertension (HT) myocardial infarction (MI), and non- significant difference of Myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
39.5276 ±	32.6±	N.S
14.08387709	15.56052052	
Control	MI+HT	P-value
39.5276 ±	29.6 ±	**
14.08387709	11.01459184	
Control	MI+DM	P-value
39.5276 ±	29.8 ±	N.S
14.08387709	14.42809759	

Table 1-3 shows the comparison of HDL level between health individuals and patients with myocardial infarction (MI), Myocardial infarction association with hypertension (HT) and Myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value <0.05. *P<0.05, **P<0.001, ***P<0.0001

In (Table 1-4) shows highly significant decrease of low-density lipoprotein (LDL) level between groups of healthy individuals and patients with myocardial infarction (MI) and significant decrease of (LDL) level with myocardial infarction (MI) association with hypertension (HT) and Non- significant difference of myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
101.6 ± 40.29444306	68.8± 35.03335704	**
Control	MI+HT	P-value
101.6 ± 40.29444306	87.5± 36.76884007	*
Control	MI+DM	P-value
101.6 ± 40.29444306	120± 39.19535814	N.S

Table 1-4 shows the comparison of LDL level between health individuals and patients with myocardial infarction (MI), myocardial infarction association with hypertension (HT), and Myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value. <0.05. *P<0.05, **P<0.001, ***P<0.0001

In (Table 1-5) shows Non- significant difference of glycosylated hemoglobin (HbA1C) level between all groups of healthy individuals and patients with Myocardial infarction (MI), myocardial infarction association with hypertension (HT), and highly significant increasing of (HbA1C) level with myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
5.7 ± 3.784887563	5.48± 0.672012649	N.S
Control	MI+HT	P-value
5.7 ± 3.784887563	6.19± 3.747023797	N.S
Control	MI+DM	P-value
5.7 ± 3.784887563	8.45± 3.322628076	* * *

Table 1-5 shows the comparison of HbA1C level between health individuals and patients with myocardial infarction (MI), Myocardial infarction association with hypertension (HT) and Myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value. <0.05. *P<0.05, **P<0.001, ***P<0.0001

In (Table 1-6) shows non- significant difference of C-reactive protein (CRP) level between all groups of healthy individuals and patients with Myocardial infarction (MI), myocardial infarction association with hypertension (HT), Myocardial infarction association with diabetes mellitus (DM).

Control	MI	P-value
7.2 ± 13.12970297	13.7± 18.66543753	N.S
Control	MI+HT	P-value
7.2 ± 13.12970297	14.7± 20.26042695	N.S
Control	MI+DM	P-value
7.2 ± 13.12970297	15.3± 20.50995693	N.S

Table 1-6 shows the comparison of CRP level between health individuals and patients with myocardial infarction (MI), myocardial infarction association with hypertension (HT) and myocardial infarction association with diabetes mellitus (DM).

The data represent as Mean ±SD.

The different consider statistically significant when the P-value <0.05. *P<0.05, **P<0.001, ***P<0.0001

DISCUSSION

The discussion will be based on three axes.

<u>The first axis</u> represents the results in the 1-1 table, and there is no statistical benefit or variable in the level of triglycerides in all blood samples taken (if it was high or low or the result is within the normal level) from all patients who suffer from diabetes mellitus or high blood pressure with acute myocardial infarction.

In the table 1-2 there's relationships between higher levels of cholesterol and patients diagnosed with acute myocardial infarction and AMI plus hypertension and there's no statistical significance with AMI plus diabetes mellitus.

In the table1-3 there's no statistical significance of HDL in patients diagnosed with acute myocardial infarction and AMI plus diabetes mellitus and statistical significance of HDL in patients with myocardial infarction plus hypertension.

In the table1-4 there's relationships between higher levels of LDL in patients diagnosed with acute myocardial infarction and AMI plus hypertension and no significance value in patients with AMI plus DM.

All this according to the pathophysiology of lipid and their metabolism and the incidence of dyslipidemia continuously increases.

Dyslipidemia, inflammation and immune reaction induced by abnormal lipid metabolism are the basis of the occurrence and development of CHD.

A previous study revealed that the incidence of AMI in an obese population was significantly higher than that in a normal population, and obesity was a risk factor for cardiovascular events, which increased the risk of death induced by AMI.

The second axis

In the table1-5 there's significance value of elevated HbA1C level in patients with myocardial infarction plus diabetes mellitus and no significance value on patients diagnosed with AMI or AMI plus hypertension.

All this according to the pathophysiology of glucose metabolism is a common risk factor for MI, and has an important influence on its prognosis.

This suggests that hyperglycemia is correlated to the occurrence of MI. In recent years, a number of studies have suggested that serum UA is closely correlated to CVD, and is one of the independent risk factors for CVD.

The third axis

In the table1-6 there's no statistical significance value of CRP level in patients with myocardial infarction and AMI plus hypertension and AMI plus diabetes mellitus.

Because the CRP elevation or normal values in all results of all groups considered non-specific inflammatory marker in AMI and can be elevated in another diseases such as (arthritis, infection of respiratory tract, GIT, UTI ... etc.)

CONCLUSION

In this study, six potentially modifiable variables (TG, Ch, LDL, HDL, HbA1C, and CRP) accounted for the risk of first or second AMI in women and men regardless of age, sex, smoking and drinking, obesity and hereditary hyperlipidemia.

Significant differences in risk factor profiles and risk factor associations were found in the tables presented in the outcome and AMI groups.

These findings indicate the need for easy-to-follow strategies and novel treatment protocols for the rapid diagnosis and prevention of AMI in young adults and the elderly.

The obtained results represent the risk of excess weight, hereditary fat diseases, old age, smoking and diabetes mellitus, and these factors can be controlled except for age, but the patients from whom blood samples were taken for laboratory tests (lipid profile, HbA1C, CRP) It was found that they had a previous disease history with (acute myocardial infarction) and that they were fully aware of their disease and some of them went to private clinics and were prescribed medications such as (aspirin, clopidogrel, statins, diuretics, ACEI, ARBS, anti-diabetic drugs, insulin) And that some of them led him to this disease, either because he is not committed to the treatment, he was exposed to psychological pressure or high blood pressure, he is not committed to the diet, because of the wrong food habits and traditions, lack of commitment or exercise are all overlapping causes that lead to heart attack, and some of them can be controlled or Some are being processed

Important finding of the present study was the additive effects of hypertension, diabetes, and dyslipidemia on AMI. Overall, dyslipidemia, hypertension had stronger effects on MI.

REFERENCES

1. <u>Thygesen K, Alpert JS, Jaffe AS, et al</u>, the Writing Group on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction: ESC/ACCF/AHA/WHF Expert Consensus Document Third Universal Definition of Myocardial Infarction. *Circulation* 126:2020–2035, 2012. doi: 10.1161/CIR.0b013e31826e1058

2. <u>Tamis-Holland JE</u>, Jneid H, Reynolds HR, et al: Contemporary diagnosis and management of patients with myocardial infarction in the absence of obstructive coronary artery disease: A scientific statement from the American Heart Association. *Circulation* 139:e891–e908, 2019. doi.org/10.1161/CIR.00000000000670

3. <u>Goff DC Jr, Lloyd-Jones DM, Bennett G, et al</u>: 2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk. J Am Coll Cardiol 63:2935–2959, 2014.

4. Grundy SM, Stone NJ, Bailey AL, et al: 2018

AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NL A/PCNA Guideline on the Management of Blood Cholesterol A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 139: e1082–e1143, 2019. doi: 10.1161/CIR.00000000000625

5. Grundy SM, Stone NJ, Bailey AL, et al: 2018

AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NL A/PCNA Guideline on the Management of Blood Cholesterol A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 139: e1082–e1143, 2019. doi: 10.1161/CIR.00000000000625

6. <u>Sabatine MS, Giugliano RP, Keech AC, et al</u>: Evolocumab and clinical outcomes in patients with cardiovascular disease. N Engl J Med 376:1713–1722, 2017. 15

7. <u>Million Hearts</u>: Estimated Hypertension Prevalence, Treatment, and Control Among U.S. Adults. https://millionhearts.hhs.gov/datareports/hypertension-prevalence.html March 21, 2021. Accessed September 8, 2022. https://millionhearts.hhs.gov/datareports/hypertension-prevalence.html

8. <u>Ostchega Y, Fryar CD, Nwankwo T, Nguyen DT</u>: Hypertension Prevalence Among Adults Aged 18 and Over: United States, 2017–2018. NCHS Data Brief No. 364, April 2020.

9. <u>Flynn J.T, Kaelber DC, Baker-Smith CM, et al; Subcommittee on</u> <u>Screening and Management of High Blood Pressure in Children</u>: Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics 140(3):e20171904, 2017.

Carey RM, Calhoun DA, Bakris GL, et al: Resistant hypertension: Detection, evaluation, and management: A Scientific Statement From the American Heart Association. *Hypertension* 72:e53–e90, 2018. doi: 10.1161/HYP.000000000000084

2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults.

10. <u>Brown JM, Siddiqui M, Calhoun DA, et al</u>: The unrecognized prevalence of primary aldosteronism: A cross-sectional study. *Ann Intern Med* 173(1):10–20, 2020. doi:10.7326/M20-0065

11. <u>Carey RM, Calhoun DA, Bakris GL, et al</u>: Resistant hypertension: Detection, evaluation, and management: A Scientific Statement From the American Heart Association. *Hypertension* 72:e53-e90, 2018. doi: 10.1161/HYP.00000000000084

12. <u>Muntner P, Shimbo D, Carey RM, et al</u>: Measurement of blood pressure in humans: A scientific statement from the American Heart Association. *Hypertension* 73:e35–e66, 2019.

13. <u>Williams B, Mancia G, Spiering W, et al</u>: 2018 Practice Guidelines for the management of arterial hypertension of the European Society of Hypertension and the European Society of Cardiology: ESH/ESC Task Force for the Management of Arterial Hypertension [published correction appears in J Hypertens. 2019 Feb;37(2):456]. *J Hypertens* 2018;36(12):2284-2309. doi:10.1097/HJH.000000000001961

14. <u>American Diabetes Association</u>: Standards of Medical Care in Diabetes. Diabetes Care 44 (Supplement 1): 1-259, 2022.

15. Lu S, Bao MY, Miao SM, Zhang X, Jia QQ, Jing SQ, Shan T, Wu XH, Liu Y. Prevalence of hypertension, diabetes, and dyslipidemia, and their additive effects on myocardial infarction and stroke: a cross-sectional study in Nanjing, China. Ann Transl Med. 2019 Sep;7(18):436. doi: 10.21037/atm.2019.09.04. PMID: 31700872; PMCID: PMC6803213.

16.

https://www.bing.com/ck/a?!&&p=4947873064a6ab0aJmltdHM9MTY3 OTg3NTIwMCZpZ3VpZD0xOTEzYmQ1OS1iY2Q3LTZIYzktMTkyYS 1iMmE3YmQ1YzZmMTYmaW5zaWQ9NTE2OQ&ptn=3&hsh=3&fclid =1913bd59-bcd7-6ec9-192ab2a7bd5c6f16&psq=ARCHITECT+c+SYSTEM+ASSAY+PARAMETE RS&u=a1aHR0cHM6Ly93d3cuY29yZWxhYm9yYXRvcnkuYWJib3R0 L3VzL2VuL29mZmVyaW5ncy9icmFuZHMvYXJjaGl0ZWN0L2FyY2h pdGVjdC1jNDAwMC5odG1s&ntb=1.

17. Executive summary of the third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Journal of the American Medical Association, 2001, 285(19):2486–97.

18. Hasdai D et al. A prospective survey of characteristics, treatments and outcomes of patients with acute coronary syndromes in Europe and the Mediterranean basin. The Euro Heart Survey of Acute Coronary Syndromes (Euro Heart Survey ACS). European heart journal, 2002, 23(15):1190–201.

19. Yusuf S et al. Effects of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case–control study. Lancet, 2004, 364(9438):937–42.

20. Shilbayeh S. Risk factors of coronary heart disease among Jordanians. Saudi medical journal, 2004, 25:1532–4.

21. Rafiei M, Boshtam M, Sarraf-Zadegan N. Lipid profiles in the Isfahan population: an Isfahan cardiovascular disease risk factor assay, 1994. Eastern Mediterranean health journal, 1999, 5(4):766–77.

22. Sempos CT et al. Prevalence of high blood cholesterol among US adults. Journal of the American Medical Association, 1993, 269:3009–14

23. Janszky I, Romundstad P, Laugsand LE, et al. Weight and weight change and risk of acute myocardial infarction and heart failure - the HUNT Study. *J Intern Med* 2016;280:312-22. 10.1111/joim.12494 [PubMed] [CrossRef] [Google Scholar]

24. Zhang JX, Dong HZ, Chen BW, et al. Characteristics of coronary arterial lesions in patients with coronary heart disease and hypertension. *Springerplus* 2016;5:1208. 10.1186/s40064-016-2828-7 [PMC free article] [PubMed] [CrossRef] [Google Scholar]

25. Tam WC, Hsieh MH, Lin YK, et al. Silent and Malignant Early Repolarization Syndrome Mimicking Hyper-Acute ST Elevation Myocardial Infarction. *Acta Cardiol Sin* 2016,32:506-10. [PMC free article] [PubMed] [Google Scholar]

26. Koracevic GP. Proposal of a New Approach to Study and Categorize Stress Hyperglycemia in Acute Myocardial Infarction. *J Emerg Med* 2016;51:31-6. 10.1016/j.jemermed.2015.03.047 [PubMed] [CrossRef] [Google Scholar]

27. Hajizadeh R, Ghaffari S, Salehi R, et al. Association of serum uric acid level with mortality and morbidity of patients with acute ST-elevation myocardial infarction. *J Cardiovasc Thorac Res* 2016;8:56-60. 10.15171/jcvtr.2016.11 [PMC free article] [PubMed] [CrossRef] [Google Scholar]

28. Shacham Y, Gal-Oz A, Flint N, et al. Serum Uric Acid Levels and Renal Impairment among ST-Segment Elevation Myocardial Infarction Patients Undergoing Primary Percutaneous Intervention. *Cardiorenal Med* 2016;6:191-7. 10.1159/000444100 [PMC free article] [PubMed] [CrossRef] [Google Scholar]