The Egyptian Journal of Hospital Medicine (July 2023) Vol. 92, Page 5570-5676

Association between Vitamin D Deficiency and Electrolyte Level and Myocardial Infarction in Patients Presenting with Acute Coronary Syndrome

Nadia A. Abdalla[1], Aleya A. Mohamed[1], Fatma A. Ibrahim[2], Muhammad M. Hassan[3], Ramdan Ghaleb[4], Naggeh M.Mahmoud[*4]

[1]Department of Inorganic Chemistry, Faculty of Science, Aswan University, Egypt
[2]Department of Biochemistry, Biotechnology Research Institute, National Research Centre, Egypt
[3]Department of Chemist, Aswan Heart Centre, Magdy Yacoub Heart Foundation, Egypt
[4]Department of Cardiology, Faculty of Medicine, Aswan University, Egypt
***Corresponding author:** Naggeh M. Mahmoud, **Mobile:** (+20) 01127161616, **E-mail:** nagehmahmoud79@gmail.com

ABSTRACT

Background: Acute myocardial infarction had been related to greater rates of morbidity and mortality in hospitalized studied cases around the globe. It has been established that male-studied cases have AMI more frequently than female ones do.

Objectives: The present research sought to examine serum electrolytes, myocardial infarction, and levels of 25-hydroxyvitamin D in studied cases with acute coronary syndrome.

Subjects and techniques: This research had been a case-control study, where 150 participants (sixty STEMI cases, sixty NSTEMI cases and thirty controls) had been recruited from the Cardiology Department, Faculty of Medicine, Aswan University. **Results:** All Vit-D level was significantly lower among STEMI and NSTEMI cases linked to a healthy control population ($p < 0.001$). Serum Ca, K, and Na were significantly lower among STEMI and NSTEMI cases compared to healthy control population, while Mg was significantly higher among STEMI and NSTEMI cases compared to healthy control population ($p < 0.05$).

Conclusion: vitamin D and serum electrolytes were deficient in all studied cases with ACS compared to controls. Additionally, we determined that low vitamin D, high LDL, total cholesterol, impaired renal function and low HDL-C were significant ACS predictors.

Keywords: 25-Hydroxyvitamin D, Acute coronary syndrome, Myocardial infarction, Electrolyte.

INTRODUCTION

Cardiovascular disease is regarded as a serious issue in public health. Around the world, CVD had been responsible for ten percent of morbidity and around 3rd of mortality. Based on WHO, seventeen million of fifty-eight million fatalities that occurred in the world in 2005 could be directly linked to CVD. Total of 7.5 million of them had been victims of coronary heart disease. 1 of 5 primary indicators of coronary heart disease, along with stable and unstable angina pectoris, myocardial infarction, heart failure, and sudden death [(1)]. Acute myocardial infarction had been associated with higher rates of morbidity and mortality in hospitalized studied cases around the globe. It has been established that male-studied cases have AMI more frequently than female ones do. Myocardial necrosis in MI is described as occurring when there is insufficient coronary blood flow. As a result, its myocardial sustains damage, raising metabolic demand while reducing oxygen and nutrient supply via coronary flow. This came after the sudden narrowing of coronary arteries caused by atherosclerosis [(2)].

Inflammatory cells, smooth muscle, and vascular endothelial cells are all implicated in the creation and stabilization of atherosclerotic lesions as well as vascular homeostasis [(3-5)].

Additionally, it had been understood that vitamin-D receptors in the nucleus of these cells would facilitate the essential mechanistic role of vitamin D in the onset and progression of ischemia disorders [(6-9)]. Previous studies have consistently found evidence of a potential link between vitamin D levels and cardiovascular disease [(10-14)]. Previous research on humans and animals revealed that vitamin D might be hazardous to the arteries. Additionally, there is evidence to support the hypothesis that there is a link among higher incidence of IHD and excessive vitamin-D intake [(10)].

The present research wanted to explore levels of 25- hydroxyvitamin-D and serum electrolytes and myocardial infarction in studied cases presenting with acute coronary syndrome.

PATIENTS AND METHODS

This research had been case-control study where 150 participants (sixty STEMI cases, sixty NSTEMI cases and thirty control) had been recruited from Cardiology Department, Faculty of Medicine, Aswan University.

Sample size calculation

Minimum sample size of eighteen studied cases had been determined to be necessary for research based on 1st type error of $\alpha = 0.05$, statistical power of ninety-five percent clinical study power of eighty percent,

Received: 05/01/2023
Accepted: 08/03/2023

and prevalence of vitamin D insufficiency in our nation (on average seventy-five percent).

Study Population

The research enrolled all willing participants who had been eighteen years of age or older and had diagnosis of acute MI, comprising both ST-segment elevation of MI and Non-ST-segment elevation of MI. Both STEMI and NSTEMI patients had been randomly chosen for research population. For sampling, computer-generated random numbers were used in systematic manner of randomization. Acute and chronic renal and hepatic failure, pregnancy, presence of autoimmune and inflammatory illnesses, and recent use of vitamin D supplements within previous month were among exclusion criteria.

Collection of Data

Clinical Data:

Were collected thorough history and physical examination as well as interview with studied cases. Particularly, history involved personal demographic data and traditional CVD risk factors containing years old, gender, smoking, dyslipidemia, HTN, and DM. Besides, complete cardiac examination, electrocardiogram and echocardiography were done on all patients. Ejection fraction had been measured by modified Simpson's technique using echocardiography device.

Diagnosis of ACS:

At least 2 of following—ischemia-related symptoms, diagnostic ECG, and increased cardiac biomarkers—must be present in order to diagnose acute myocardial infarction. New left bundle branch block and presumed new pathologic Q waves were among ECG features indicative of ACS. Additionally, ST-elevation was less than 0.1 mV in 2 or more adjacent limb leads and less than 0.2 mV in chest leads. Studied cases who presented with symptoms of ischemia but no rise in cardiac biomarkers were found to have unstable angina, and ischemia may be indicated by abnormal ECG [12].

Laboratory Data:

Research participants underwent laboratory examinations; blood samples had been taken from anti-cubital vein in supine position, using aseptic techniques, within thirty minutes of admission for measurement of 25-hydroxyvitamin D levels, liver function test, serum electrolytes such as calcium, magnesium, sodium, and potassium, random blood sugar levels, and haemoglobin AIC. The levels of vitamin D (25[OH] D) had been measured using radioimmunoassay method.

Ethical Considerations:

Written informed consent was given by all studied cases. Studied cases had been informed that their information would be kept private. This experiment was ethically approved by the Faculty of Medicine, Aswan University's. After being fully informed, all participants provided written consent. According to Helsinki Declaration of World Medical Association, the present research had been carried out.

Statistical analysis

Researcher checked data, coded information, and used IBM-SPSS 24.0 to analyse them. Means, standard deviations, medians, inter-quartile ranges, and percentages had been determined as descriptive statistics. Chi square analysis had been used to compare variations in frequency distribution amongst several groups. Continuous variables were subjected to Shapiro-Wilk/Kolmogorov Smirnoff test for normality. Means of data that follow normal distribution and independent sample were compared using ANOVA test. Post-hoc test had been calculated using Bonferroni adjustments. Kruskal-Wallis had been used to assess variation in medians among groups that don't match normal distribution. When it is <0.05, substantial p value had been considered.

RESULTS

Age, sex and left ventriclaur ejection fraction (EF) were significantly different in study groupes comparing with the control group (Table 1).

Table (1): Baseline socio-demographics and clinical data of studied groups

	Control (I) (n = 30)	STEMI (II) (n = 60)	NSTEMI (III) (n = 60)	P-value
Age/year	32.60 ± 5.8	55.23 ± 9.5	53.77 ± 9.6	**< 0.001***
• **P-value****	**I vs. II < 0.001**	II vs. III = 0.372	**I vs. III < 0.001**	
Sex				
• **Female**	17 (56.7%)	11 (18.3%)	11 (18.3%)	**< 0.001*****
• **Male**	13 (43.3%)	49 (81.7%)	49 (81.7%)	
DM	8 (26.7%)	20 (33.3%)	20 (33.3%)	= 0.783***
HTN	6 (20%)	24 (40%)	24 (40%)	= 0.125***
Smoking	8 (26.7%)	27 (45%)	27 (45%)	= 0.190***
EF%	64.90 ± 6.5	46.23 ± 5.8	48.57 ± 5.3	**< 0.001***
• **P-value****	**I vs. II < 0.001**	**II vs. III = 0.027**	**I vs. III < 0.001**	

Data are presented as mean ± standard deviation or as frequency (Percentage)
*ANOVA test had been used for comparing mean variation among groups
**Post-hoc test had been used for pairwise comparing with Bonferroni correction
***Chi-square test had been used for comparing proportion variation among groups

Vitamin D level was sinincantly lower in patients' groups than control group (Table 2).

Table (2): Laboratory findings of studied groups (A)

	Control (I) (n = 30)	STEMI (II) (n = 60)	NSTEMI (III) (n = 60)	P-value
Vit-D Level (ng/ml)	21.04 ± 2.3	13.95 ± 1.8	15.38 ± 1.9	**< 0.001***
• **P-value****	**I vs. II = 0.001**	II vs. III = 0.341	**I vs. III = 0.012**	
Serum Electrolytes				
Ca (mg/dl)	10.36 ± 0.7	9.37 ± 0.4	9.41 ± 0.4	**< 0.001***
• **P-value****	**I vs. II = 0.014**	II vs. III = 0.900	**I vs. III = 0.018**	
Mg (mg/dl)	2.09 ± 0.1	2.25 ± 0.3	2.25 ± 0.3	**= 0.018***
• **P-value****	**I vs. II = 0.017**	II vs. III = 1.000	**I vs. III = 0.017**	
Ph (mg/dl)	3.50 ± 0.6	3.44 ± 0.85	3.44 ± 0.9	= 0.953*
• **P-value****	I vs. II = 0.777	II vs. III = 1.000	I vs. III = 0.777	
Na (mg/dl)	142.93 ± 2.3	137.84 ± 3.9	137.84 ± 3.9	**< 0.001***
• **P-value****	**I vs. II < 0.001**	II vs. III = 1.000	**I vs. III < 0.001**	
K (mg/dl)	4.34 ± 0.4	4.01 ± 0.5	4.09 ± 0.5	**= 0.012***
• **P-value****	**I vs. II = 0.003**	II vs. III = 0.318	**I vs. III = 0.029**	
Cardiac Enzymes				
CK-T (U/L)	111.33 ± 9.9	823.50 ± 57.5	826.07 ± 57.3	**< 0.001***
• **P-value****	**I vs. II = 0.004**	II vs. III = 0.990	**I vs. III = 0.004**	
CK-MB (U/L)	20.24 ± 4.5	99.70 ± 19.1	102.71 ± 18.8	**< 0.001***
• **P-value****	**I vs. II = 0.008**	II vs. III = 0.900	**I vs. III = 0.006**	
LDH (U/L)	327.40 ± 52.6	786.08 ± 93.6	786.08 ± 93.6	**< 0.001***
• **P-value****	**I vs. II = 0.002**	II vs. III = 1.000	**I vs. III = 0.002**	
TRO (ng/ml)	< 0.1 (< 0.1)	0.4 (1)	0.4 (1)	**< 0.001*****
• **P-value****	**I vs. II < 0.001**	II vs. III = 0.990	**I vs. III < 0.001**	

Data are presented as mean ± standard deviation or as median (Inter-quartile range)
*ANOVA test had been used for comparing mean variation among groups
**Post-hoc test had been used for pairwise comparing with Bonferroni correction
***Kruskal Wallis test had been used for comparing median variation between groups

HbA1C, lipid profile, liver enzymes were significantly different in patients' groups comparing with control group (Table 3).

Table (3): Laboratory results of studied groups (B)

	Control (I) (n = 30)	STEMI (II) (n = 60)	NSTEMI (III) (n = 60)	P-value
HbA1C (mmol/L)	5.98 ± 1.4	7.65 ± 1.9	7.65 ± 1.9	**< 0.001***
• **P-value****	**I vs. II = 0.006**	II vs. III = 1.000	**I vs. III = 0.006**	
Liver Function Test Parameters				
Cholesterol (mg/dl)	174.10 ± 42.2	199.58 ± 47.2	199.58 ± 47.2	**= 0.029***
• **P-value****	**I vs. II = 0.015**	II vs. III = 1.000	**I vs. III = 0.015**	
TGD (mg/dl)	68.31 ± 12.5	116.96 ± 15.1	116.96 ± 15.1	**< 0.001***
• **P-value****	I vs. II = 0.063	II vs. III = 1.000	I vs. III = 0.063	
HDL (mg/dl)	9.90 ± 1.8	9.21 ± 1.2	9.21 ± 1.2	**= 0.044***
• **P-value****	**I vs. II = 0.009**	II vs. III = 1.000	**I vs. III = 0.009**	
LDL (mg/dl)	103.13 ± 16.8	124.43 ± 22.1	124.43 ± 22.1	**< 0.001***
• **P-value****	**I vs. II = 0.022**	II vs. III = 1.000	**I vs. III = 0.022**	
T. Bilirubin (mg/dl)	0.45 ± 0.1	0.54 ± 0.12	0.54 ± 0.12	= 0.001*
• **P-value****	I vs. II = 0.252	II vs. III = 1.000	I vs. III = 0.252	
D. Bilirubin (mg/dl)	0.15 ± 0.03	0.20 ± 0.04	0.20 ± 0.04	**< 0.001***
• **P-value****	I vs. II = 0.112	II vs. III = 1.000	I vs. III = 0.112	
ALT (U/L)	19.03 ± 2.1	31.47 ± 2.7	31.47 ± 2.7	**< 0.001***
• **P-value****	**I vs. II = 0.004**	II vs. III = 1.000	**I vs. III = 0.004**	
AST (U/L)	17.80 ± 4.4	76.67 ± 11.3	76.67 ± 11.3	**= 0.001***
• **P-value****	**I vs. II = 0.001**	II vs. III = 1.000	**I vs. III = 0.001**	
Albumin (g/dl)	4.48 ± 0.3	4.24 ± 0.4	4.24 ± 0.4	**= 0.015***
• **P-value****	**I vs. II = 0.008**	II vs. III = 1.000	**I vs. III = 0.008**	

Data are presented as mean ± standard deviation

*ANOVA test had been used for comparing mean variation among groups

**Post-hoc test had been used for pairwise comparing with Bonferroni correction

Serum creatinin and blooud urea were singnifcantly higher in patients' groups comparing with control group (Table 4).

Table (4): Laboratory findings of studied groups (C)

	Control (I) (n = 30)	STEMI (II) (n = 60)	NSTEMI (III) (n = 60)	P-value
Renal Function Test Parameters				
S. Creatinine (mg/dl)	0.73 ± 0.18	1.05 ± 0.2	1.05 ± 0.2	**< 0.001***
• **P-value****	**I vs. II < 0.001**	II vs. III = 1.000	**I vs. III < 0.001**	
B. Urea (mg/dl)	21.18 ± 3.2	32.09 ± 5.5	32.09 ± 5.5	**< 0.001***
• **P-value****	**I vs. II < 0.001**	II vs. III = 1.000	**I vs. III < 0.001**	
CBC Parameters				
Hgb (g/dl)	13.87 ± 2.1	14.24 ± 1.6	14.24 ± 1.6	= 0.588*
• **P-value****	I vs. II = 0.347	II vs. III = 1.000	I vs. III = 0.347	
RBCs	4.99 ± 0.7	5.05 ± 0.6	5.05 ± 0.6	= 0.889*
• **P-value****	I vs. II = 0.658	II vs. III = 1.000	I vs. III = 0.658	
HCT%	42.29 ± 6.1	42.25 ± 5.1	42.25 ± 5.1	= 0.994*
• **P-value****	I vs. II = 0.973	II vs. III = 1.000	I vs. III = 0.973	
WBCs *10^9	7.47 ± 1.8	12.63 ± 3.1	12.63 ± 3.1	**< 0.001***
• **P-value****	**I vs. II < 0.001**	II vs. III = 1.000	**I vs. III < 0.001**	
Platelet *10^9	245.93 ± 47.5	218.63 ± 38.1	218.63 ± 38.1	= 0.005*
• **P-value****	I vs. II = 0.083	II vs. III = 0.900	I vs. III = 0.083	

Data are presented as mean ± standard deviation

*ANOVA test had been used for comparing mean variation among groups

**Post-hoc test had been used for pairwise comparing with Bonferroni correction

DISCUSSION

It was proposed that low blood levels of vitamin D are connected to how well ACS patient would fare. Based on long-term findings and follow-up, numerous researches have related low serum vitamin D levels to higher hospitalisation rates and requirement for intensive care in ACS studied cases. Yet, research on specific effects of vitamin D deficiency or insufficiency on ACS case fatality rates is scarce [15].

In this research we showed that age had been greater between STEMI and NSTEMI cases compared to healthy control population and there was significantly high prevalence of male among STEMI and NSTEMI cases compared to healthy control population (p <0.001). **Elsaughier *et al.*** [16] found that when compared to healthy control patients, STEMI group's age was substantially greater (55.2 ± 9.6 vs. 32.6 ± 5.8 years; P< 0.001). It had been significant (P< 0.001) that majority of STEMI patient's eighty-two percent were males and majority of controls (56.7 percent) were females.

According to Health Professionals Follow-up Study, that involved 18,225 males and included ten-year follow-up, men with low vitamin D levels had greater risk of developing MI than men with adequate levels (relative ratio: 2.42; 95% CI: 1.53-3.84; p< 0.001), that has been in line with our results [17].

Hopkins *et al.* [18] had 24.4% women and 75.6% men. prevalence of ACS in men is revealed by this result.

In this study we demonstrated that EF% was significantly lower among STEMI cases compared to NSTEMI cases and healthy control (p=0.027 and p<0.001). **Bashandy *et al.*** [19] found that the prevalence of impaired ejection fraction <fifty percent had been substantially higher in STEMI patients (43.8%) compared to NSTEMI (20.1%) and UA (3.7%). **Chen *et al.*** [20] showed that LVEF had been significantly reduced in STEMI group compared to healthy control group (42.4±9.3 vs. 57.1±7.8, P<0.01). Furthermore, **Elsaughier *et al.*** [16] found that studied cases in STEMI group who had lower left ventricular ejection fraction likened to those who had higher LVEF had lower vitamin-D levels, with p-value of< 0.001.

In this study we illustrated that all Vit-D level was significantly lower among STEMI and NSTEMI cases compared to healthy control population (p <0.001). **Elsaughier *et al.*** [16] discovered that STEMI group's vitamin-D levels were considerably lower than those of control group (201.7 ± 9.7vs. 25.3 ± 10.7mg/dl; P = 0.02). **Mohammed *et al.*** [21] found that mean ± SD of 25(OH)D serum level inSTEMI group was 25.13 ± 5.42, while it had been 33.23 ± 1.99 ng/ml in control group. In STEMI group, amount of vitamin D was considerably lower (P< 0.001).

According to earlier research, vitamin D is important negative regulator of renin-angiotensin-aldosterone system and regulates turnover of extracellular matrix in myocardium. Increasing RAAS activity, that causes hypertension, cardiac hypertrophy, increased water intake, and sodium retention, as well as improved metalloproteinase activity, that encourages destruction of myocardial tissue and causes ventricular remodelling, were all observed in vitamin-D receptor knockout mice. Thus, vitamin-D deficit could cause heart function to worsen and myocardial remodelling to proceed more quickly [22].

In this study we found that Ca (mg/dl), K (mg/dl) and Na (mg/dl) were significantly lower among STEMI and NSTEMI cases compared to healthy control population, while Mg (mg/dl) was significantly higher among STEMI and NSTEMI cases compared to healthy control population (p <0.05). **Elsaughier *et al.*** [16] showed that compared to control group, STEMI group exhibited substantially lower levels of calcium (9.37 ± 0.4 vs. 10.36 ± 3.9mg/dl; P=0.04), sodium (137.84 ± 3.9 vs. 142.93 ±2.3 mol/l; P<0.001), potassium (4.03 ± 0.5 vs. 4.33 ± 0.4; P<0.001), and magnesium (2.08 ± 0.2vs.2.40 ± 0.4) for comparing to control group; p-value = 0.01).

These results had been in line with results of **Hariprasad and Basavaraj** [23] who decided that in contrast to healthy controls, AMI cases' serum sodium levels were significantly lower in both age groups, but there had been reduction in potassium, which goes against our findings about potassium levels. Our finding was in line with **Vamne *et al.*** [24] who discovered that compared to controls, AMI sufferers had greater incidence of hyponatremia. According to report, hypoxia and ischemia may be to blame for drop in sodium levels, which would increase sodium permeability of sarcolemma.

This was in keeping with findings of **Kughapriya and Evangeline** [25], that found that studied cases with IHD had lower magnesium levels than studied cases in control group. Magnesium had been discovered to be crucial to process governing cardiovascular homeostasis. function of magnesium in pathophysiology of IHD has been explained by number of different ways. Severe ischemia may result in hypomagnesaemia.

In this study we illustrated that all cardiac enzymes were significantly lower among STEMI and NSTEMI cases compared to healthy control population (p <0.001). **Kamal *et al.*** [26] found that among ACS and serum CKMP levels, there was significant correlation (P< 0.05). **Abd Elrahman *et al.*** [27] showed that at admission, twenty-four hours after admission, and forty-eight hours after admission to ICU, mean serum levels of CKMB for STEMI and NSTEMI groups were substantially greater than those of control group (P< 0.01). Additionally, it had been discovered that control group's mean serum level of cTnI was considerably lower on admission, twenty-four hours later, and forty-

eight hours later than that of STEMI and NSTEMI groups (P <0.01).

In this study we cleared that HbA1C level was significantly higher among STEMI cases compared to NSTEMI cases and healthy control (p<0.001). **Kim *et al.*** [28] found that the HbA1c values had been greater in STEMI studied cases. At time of MI, studied cases with STEMI may have had more severe decompensated diabetes. Recently prospective cohort research by **Selvin *et al.*** [29] showed that increased HbA1c levels, independent of fasting glucose, are risk factor for development of cardiovascular events in general non-diabetic population. Cut-off of 6.5 percent was suggested by worldwide expert group for use of HbA1c in diagnosis of diabetes. In cohort of European men, **Khaw *et al.*** [30] found linear correlation between HbA1c and ensuing cardiovascular morbidity. **Preis *et al.*** [31] has showed that in non-diabetics, long-term risk of CAD can be predicted more accurately by glycosylated haemoglobin than by fasting blood glucose.

In this study we found that HDL was significantly lower among STEMI cases compared to NSTEMI cases and healthy control (p<0.001), while cholesterol and LDL were significantly higher among STEMI and NSTEMI cases compared to healthy control population (p <0.05). **Ismail *et al.*** [32] found that High TC, HDL-C, and systolic BP all substantially predicted the development of ACS.

In this study we demonstrated that S. creatinine and B. urea were significantly higher among STEMI cases compared to NSTEMI cases and healthy control (p<0.001). **Hassanien *et al.*** [33] found that in the group (STEMI), the mean ± standard deviation of creatinine were 0.904 ± 0.213; in the group (NSTEMI), they were 1.454 ± 0.336; and in the group (unstable), they were 1.212 ± 0.291 (P value = 0.004; statistically significant).

In this study we illustrated that WBCs count was significantly higher among STEMI cases compared to NSTEMI cases and healthy control (p<0.001). **Hassanien *et al.*** [33] found that the mean ± standard deviation of WBC in the group with STMI were 14.637 ± 4.861, in the group with NSTEMI had been 8.658 ± 1.721, and in the group with instability were 8.577 ± 2.890 (P value = 0.001; statistically significant).

CONCLUSIONS

According to this research, all studied cases with ACS had statistically significant vitamin D and serum electrolyte deficiencies as compared to controls. Also shown to be statistically significant ACS predictors had been low HDL-C, low HDL-C, high LDL, total cholesterol, poor renal function, and low vitamin D levels.

Sponsoring financially: Nil.
Competing interests: Nil.

REFERENCES

1. **Harrison R, Aggarwal A, Ou F *et al.* (2013):** Incidence and outcomes of no-reflow phenomenon during percutaneous coronary intervention among patients with acute myocardial infarction. Am J Cardiol., 111(2):178–184.
2. **Bouleti C, Mewton N, Germain S *et al.* (2015):** The no-reflow phenomenon: state of the art. Arch Cardiovasc Dis., 108(12):661–674.
3. **Jaffe R, Charron T, Puley G *et al.* (2008):** Microvascular obstruction and the no-reflow phenomenon after percutaneous coronary intervention. Circulation, 117(24):3152–3156.
4. **Lee J, O'Keefe J, Bell D *et al.* (2008):** Vitamin D deficiency: an important, common, and easily treatable cardiovascular risk factor. J Am Coll Cardiol., 52(24):1949–1956.
5. **Kheiri B, Abdalla A, Osman M *et al.* (2018):** Vitamin D deficiency and risk of cardiovascular diseases: a narrative review. Clin Hypertens., 24(1):1–9.
6. **Bax J, Baumgartner H, Ceconi C *et al.* (2012):** Third universal definition of myocardial infarction. J Am Coll Cardiol., 60(16):1581–1598.
7. **American Diabetes Association (2019):** Classification and diagnosis of diabetes: standards of medical care in diabetes-2019. Diabetes Care, 42: 13–28.
8. **Ference B, Graham I, Tokgozoglu L *et al.* (2018):** Reprint of: Impact of lipids on cardiovascular health: JACC Health Promotion Series. J Am Coll Cardiol., 72(23 Part B):2980–2995.
9. **Lurbe E, Agabiti-Rosei E, Cruickshank J *et al.* (2016):** European Society of Hypertension guidelines for the management of high blood pressure in children and adolescents. J Hypertens., 34: 1887–1920.
10. **Schulz S, Richardt G, Laugwitz K *et al.* (2014):** Prasugrel plus bivalirudin vs. clopidogrel plus heparin in patients with ST-segment elevation myocardial infarction. Eur Heart J., 35(34):2285–2294.
11. **Gaspar A, Lourenço A, Pereira M *et al.* (2018):** Randomized controlled trial of remote ischaemic conditioning in ST-elevation myocardial infarction as adjuvant to primary angioplasty (RIC-STEMI). Basic Res Cardiol., 113(3):14. doi: 10.1007/s00395-018-0672-3.
12. **Gibson C, de Lemos J, Murphy S *et al.* (2001):** Combination therapy with abciximab reduces angiographically evident thrombus in acute myocardial infarction: a TIMI 14 substudy. Circulation, 103(21):2550–2554.
13. **Chesebro J, Knatterud G, Roberts R *et al.* (1987):** Thrombolysis in Myocardial Infarction (TIMI): Trial, phase I: a comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. Circulation, 76(1):142–154.
14. **Henriques J, Zijlstra F, Hof A *et al.* (2003):** Angiographic assessment of reperfusion in acute myocardial infarction by myocardial blush grade. Circulation, 107(16):2115–2119.
15. **Aleksova A, Belfiore R, Carriere C *et al.* (2015):** Vitamin D deficiency in patients with acute myocardial infarction: an Italian single-center study. Int J Vitam Nutr Res., 85(1-2): 23-30.

16. **Elsaughier S, Mahmoud N, Ghaleb R (2020):** Assessment of 25-hydroxy vitamin D and serum electrolytes in myocardial infarction patients with acute ST-segment elevation. J Cardiovasc Disease Res., 11(2): 237-240.
17. **Giovannucci E, Liu Y, Hollis B *et al.* (2008):** 25-hydroxyvitamin D and risk of myocardial infarction in men: a prospective study. Archives of Internal Medicine, 168(11): 1174-1180.
18. **Hopkins P, Wu L, Hunt S *et al.* (1996):** Higher serum bilirubin is associated with decreased risk for early familial coronary artery disease. Arteriosclerosis, Thrombosis, and Vascular Biology, 16(2): 250-255.
19. **Bashandy M, Abd Elgalil H, Abou Elhassan H (2019):** Epidemiological and clinical profile of acute coronary syndrome of Egyptian patients admitted to the Coronary Care Unit, Al-Azhar University Hospital, New Damietta. The Scientific Journal of Al-Azhar Medical Faculty, Girls, 3(3): 625-634.
20. **Chen Z, Yu Z, Yang H *et al.* (2016):** Rapid predictors for the occurrence of reduced left ventricular ejection fraction between LAD and non-LAD related ST-elevation myocardial infarction. BMC Cardiovascular Disorders, 163: 3. doi: 10.1186/s12872-015-0178-y.
21. **Mohammed H, El-Ashmawy H, Mohamed E *et al.* (2015):** The prognostic value of serum 25-hydroxyvitamin D level in patients with ST-segment elevation myocardial infarction. Egyptian Journal of Obesity, Diabetes and Endocrinology, 1(1): 49-52.
22. **Tomaschitz A, Pilz S, Ritz E *et al.* (2010):** Independent association between 1, 25-dihydroxyvitamin D, 25-hydroxyvitamin D and the renin–angiotensin system: the Ludwigshafen Risk and Cardiovascular Health (LURIC): study. Clinica Chimica Acta, 411(17-18): 1354-1360.
23. **Hariprasad S, Basavaraj M (2018):** Electrolyte dysfunction in myocardial infarction patients. Int J Adv Med., 5(5):1172-1176.
24. **Vamne A, Pathak C, Thanna R *et al.* (2015):** Electrolyte changes in patients of acute myocardial infarction. International Journal of Advances in Medicine, 5(1):78-80.
25. **Kughapriya P, Evangeline J (2016):** Evaluation of serum electrolytes in ischemic heart disease patients. National Journal of Basic Medical Sciences, 6(4): 1-14.
26. **Kamal Y, Hasanin H, Abdel-All Z (2022):** Study of vitamin D level in acute coronary syndrome. SVU-International Journal of Medical Sciences, 5(2): 470-483.
27. **Abd Elrahman B, Shaban G, Mahdy K *et al.* (2006):** The value of cardiac troponin-i as a biochemical markers of acute coronary syndromes compared with other traditional markers. Bulletin of Egyptian Society for Physiological Sciences, 26(2): 133-146.
28. **Kim Y, Her A, Rha S *et al.* (2022):** Comparison of clinical outcomes after non-ST-segment and ST-segment elevation myocardial infarction in diabetic and nondiabetic populations. Journal of Clinical Medicine, 11(17): 5079. doi: 10.3390/jcm11175079
29. **Selvin E, Streffes M, Zhu H *et al.* (2010):** Glycated hemoglobin, diabetes, and cardiovascular risk in non-diabetic adults. N Engl J Med., 362(9): 800-811.
30. **Khaw K, Wareham N, Luben R *et al.* (2001):** Glycated haemoglobine , diabetes and mortality in men in Norfolk cohort of European prospective investigation of Cancer and Nutrition (EPIC-Norfolk). BMJ., 322(7277): 15-18.
31. **Preis S, Hwang S, Coady S *et al.* (2009):** Trends in all-cause and cardiovascular disease mortality among women and men with and without diabetes mellitus in the Framingham Heart study, 1950 to 2005. Circulation, 119(13): 1728-1735.
32. **Ismail H, Algrafi A, Amoudi O *et al.* (2021):** Vitamin D and its metabolites deficiency in acute coronary syndrome patients undergoing coronary angiography: A case–control study. Vascular Health and Risk Management, 17: 471-480.
33. **Hassanien H, Kamal Y, Ahmed M *et al.* (2021):** Relationship of serum total bilirubin with acute coronary syndromes at Sohag University Hospitals. The Egyptian Journal of Hospital Medicine, 84(1): 2329-2336.