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Evaluation the serum level of Dehydroepiandrosterone sulfate associated with STEMI and NSTEMI in a sample of Iraqi male patients

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Abstract
Background: Acute coronary syndromes (ACS) are a group of conditions characterized by a sudden reduction in blood supply to the heart, including ST-segment elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unsTable angina. ACS affects over 7 million people globally each year, with over 1 million hospitalizations in the USA alone. Dehydroepiandrosterone Sulfate (DHEAS) is the most abundant steroid hormone in human plasma, synthesized in the adrenal cortex. High DHEAS levels are considered a marker of longevity, while decreased levels are linked to various conditions such as severe stress, anorexia, and adrenal insufficiency. The study aimed to determine DHEAS levels in men with ACS to explore the potential of DHEA supplementation in treatment regimens. Method: case-control study analyzed data from 150 male patients with ACS at Al-Emamin Al-Khadhemain City Hospital between April and October 2023. Participants, aged 40-69 years, were divided into three groups: 76 controls, 38 STEMI patients, and 38 NSTEMI patients. Serum DHEAS levels were measured using the enzyme-linked immunosorbent assay (ELISA) method.
Results: Showed a significant negative correlation between DHEAS levels and ACS in the STEMI and NSTEMI groups, with P-values of 0.002 and 0.048, respectively. The control group also showed a highly significant negative correlation ($P < 0.001$). Other parameters did not show a significant correlation with DHEAS. Conclusion: serum DHEAS levels are significantly negatively associated with STEMI and NSTEMI in middle-aged and older Iraqi men.

Keywords: STEMI, NSTEMI, Dehydroepiandrosterone sulfate, evaluation, serum level

Introduction
Acute coronary syndrome (ACS) is a collective term for conditions that indicate either unsTable angina or myocardial infarction, presenting with a variety of symptoms. Patients typically report chest pain, tightness, and breathlessness, which may or may not be accompanied by changes on a 12-lead electrocardiogram (ECG) and elevated cardiac troponin (cTn) levels [1]. These symptoms arise due to a severe reduction in coronary blood supply, often caused by a thrombus that partially or completely obstructs the coronary artery, leading to significant stenosis and myocardial ischemia [2]. The resultant myocardial cell loss is marked by an elevation in cardiac serum troponin levels [3]. There are three primary types of ACS: ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unsTable angina (UA) [2]. STEMI occurs when a thrombus fully occludes a coronary artery, completely blocking blood flow [3], while NSTEMI results from a partial or transient obstruction of blood flow at the site of stenosis [4]. UA, also referred to as pre-infarction angina, represents an unsTable clinical status that may progress to myocardial infarction, serious arrhythmias, or sudden death [4]. Epidemiologically, cardiovascular diseases, particularly ischemic heart disease (IHD), account for approximately one-third of all deaths globally, with about 7.5 million of these deaths attributed to IHD [5]. ACS and sudden death are responsible for about 1.8 million deaths annually. Although advancements have been made in the diagnosis and treatment of ACS, cardiovascular disease remains the leading cause of death worldwide, with ischemic heart disease accounting for about 12% of global mortality [6]. In high-income countries, the proportion of ACS cases involving STEMI has decreased due to factors such as declining smoking rates and the increased use of high-sensitivity troponin (hsTn) assays, which have improved the diagnosis of NSTEMI [7].

ACS is most commonly seen in patients with atherosclerosis, a condition where atheromatous plaques develop due to a gradual accumulation of cholesterol, macrophages, and fibrin in arterial walls [8]. These plaques can rupture or trigger thrombus formation, leading to partial or complete occlusion of the artery and, consequently, myocardial ischemia [3]. In cases of complete occlusion, STEMI occurs, while partial or transient occlusion results in NSTEMI [3]. Diagnosis of ACS involves a combination of patient history, physical examination, ECG, and measurement of serum cardiac biomarkers, particularly troponins [1]. Cardiac troponins, specifically troponin T (cTnT) and troponin I (cTnI), are structural proteins released during myocardial necrosis, making them key markers for diagnosing acute myocardial infarction [9]. Other biomarkers such as creatine kinase, high-sensitivity C-reactive protein (hsCRP), and lactate dehydrogenase (LDH) are also used to aid in the diagnosis [10]. Dehydroepiandrosterone sulfate (DHEAS) is a prohormone produced in the adrenal gland and serves as a precursor to sex hormones like testosterone and estradiol [11]. DHEAS has been implicated in various physiological processes, including neuroprotection, immune response, and modulation of endothelial function, making it a potential protective factor in cardiovascular diseases [12]. Elevated levels of DHEAS have been associated with improved endothelial cell survival and reduced inflammation, which could contribute to cardiovascular health [13]. Aim of the study to determine the level of DHEAS in the serum of men with ACS in order to predict the use of DHEA supplement in the ACS regimen.

Methods

This case-control study was conducted between April 2023 and October 2023 at Al-Imamin Al-Kadhimeen Teaching Hospital in Baghdad, involving 150 male patients aged 40-69 years. The diagnosis of acute coronary syndrome (ACS) was based on medical history, clinical signs and symptoms, and confirmed by cardiac enzyme tests (troponin) and electrocardiogram (ECG) findings. Patients were categorized into two groups: 38 patients with ST-elevation myocardial infarction (STEMI) and 38 patients with non-ST-elevation myocardial infarction (NSTEMI). A control group of 74 apparently healthy individuals was also included. Inclusion criteria followed the World Health Organization's definition of ACS, requiring clinical presentation with chest pain, elevated troponin levels, and ECG changes. Exclusion criteria included patients aged 70 years and older, those with pituitary or adrenal gland dysfunction, glucocorticoid use, and adrenal gland disorders such as Addison's disease. Venous blood samples were collected from all subjects in the morning after at least 10 hours of fasting. The samples were divided: one portion was used to analyze troponin levels using an enzyme-linked fluorescent assay (ELFA), and the other portion, containing an anticoagulant (EDTA), was used to measure DHEAS levels by enzyme-linked immunosorbent assay (ELISA). Body mass index (BMI) was calculated using the formula BMI = weight (kg) / height (m^2) and classified according to WHO criteria. Biochemical tests included serum total cholesterol, triglycerides, HDL-C, VLDL-C, and LDL-C levels, measured using spectrophotometry. HbA1c was determined by ion-exchange high-performance liquid chromatography (HPLC). DHEAS levels were measured using a competitive binding ELISA method, where the intensity of color developed was inversely proportional to the DHEAS concentration in the sample.

Table 1: Comparison of age, body mass index, and blood pressure between control, STEMI and NSTEMI patient group Comparison between control with STEMI and NSTEMI.

Parameter		Controls, N=74	STEMI, N=38	NSTEMI, N=38
Age (yr)	Mean ± SD	53.62±8.61	55.53±9.3	56.74±7.93
	Median (Range)	53.5 (40-69)	56 (40-69)	58 (41-68)
	P-Value *		0.275[a]	0.069[a]
	P-Value **			0.651[a]
BMI (kg/m^2)	Mean ± SD	26.6±2.29	31.25±2.97	30.64±3.35
	Median (Range)	26.35(22.3-32.6)	31.3(26.7-36.7)	30.5(24.4-36.9)
	P-Value *		<0.001[b]	<0.001[b]
	P-Value **			0.405[b]
Syst. BP (mmHg)	Mean ± SD	118.78±14.57	143.42±26.54	147.63±22.83
	Median (Range)	120 (90-150)	150 (100-190)	150 (110-190)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.441[a]
Diast. BP (mmHg)	Mean ± SD	75.61±12.02	88.16±11.36	91.84±10.1
	Median (Range)	80 (50-90)	90 (70-120)	90 (70-110)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.114[a]
Smoking	Yes [(N (%)]			
	No (N (%)]			

** Comparison between STEMI and NSTEMI, a: P-Value by Mann Whitney test, b: P-Value by unpaired test, P-Value by Fisher exact test

Results

Age: The mean age was 53.62±8.61 years in the control group, 55.53±9.3 years in the STEMI group, and 56.74±7.93 years in the NSTEMI group. There was a significant difference in age between the patient groups and the control group (P=0.069), but no significant difference between the STEMI and NSTEMI groups (P=0.651). Body Mass Index (BMI): The mean BMI was 26.6±2.29 kg/m² in the control group, 31.25±2.97 kg/m² in the STEMI group, and 30.64±3.35 kg/m² in the NSTEMI group. There was a significant difference between the patient groups and the control group (p<0.001), but no significant difference between the STEMI and NSTEMI groups (P=0.405). Systolic Blood Pressure: The mean systolic blood pressure was 118.78±14.57 mmHg in the control group, 143.42±26.54 mmHg in the STEMI group, and

147.63±22.83 mmHg in the NSTEMI group. There was a significant difference between the patient groups and the control group (p<0.001), but no significant difference between the STEMI and NSTEMI groups (P=0.441). Diastolic Blood Pressure: The mean diastolic blood pressure was 75.61±12.02 mmHg in the control group, 88.16±11.36 mmHg in the STEMI group, and 91.84±10.1 mmHg in the NSTEMI group. There was a significant difference between the patient groups and the control group (p<0.001), but no significant difference between the STEMI and NSTEMI groups (P=0.114). Smoking. The median smoking rate was 28 (36.8%) in the control group, 23 (60.5%) in the STEMI group, and 22 (57.9%) in the NSTEMI group. There was a significant difference in smoking rates between the patient groups and the control group (P=0.027 for STEMI, P=0.045 for NSTEMI), but no significant difference between the STEMI and NSTEMI groups (P=0.114) as in Table 1.

The study found that high-sensitivity troponin I levels were significantly elevated in both STEMI (3531.08±1228.52 µg/dl) and NSTEMI (3977.02±1181.86 µg/dl) groups compared to the control group (21.27±26.84 µg/dl) (p<0.001) with no significant difference between the STEMI and NSTEMI groups (P=0.081). Similarly, DHEAS levels were significantly lower in both patient groups (STEMI: 61.71±28.07 µg/dl, NSTEMI: 82.08±59.63 µg/dl) compared to the control group (221.39±137.13 µg/dl) (p<0.001), with no significant difference between STEMI and NSTEMI groups (P=0.234) as in Table 2.

Table 2: Comparison of serum troponin and serum Dehydroepiandrosterone sulphate between control and two patient's groups.

Parameter		Controls, N=74	STEMI, N=38	NSTEMI, N=38
Troponin (ng/l)	Mean ± SD	21.27±26.84	3531.08±1228.52	3977.02±1181.86
	Median(Range)	5.95(0.2-84.1)	3546.1 (1249.8-6295.5)	4324.55 (1579.9-5936.2)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.081[a]
DHEAS (µg/dl)	Mean ± SD	221.39±137.13	61.71±28.07	82.08±59.63
	Median(Range)	176(58-522)	58.5 (29-159)	71.5 (19-249)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.234[a]

* Comparison between control with STEMI and NSTEMI, ** Comparison between STEMI and NSTEMI, a: P-Value by Mann Whitney test

The study found significant differences in total cholesterol (TC), triglycerides (TG), LDL-C, and HDL-C levels between the control and patient groups. TC, TG, and LDL-C levels were significantly higher in both STEMI and NSTEMI groups compared to controls (p<0.001), with a significant difference in TC between STEMI and NSTEMI (P=0.021). HDL-C levels were significantly lower in both patient groups compared to controls (p<0.001), with no significant difference between the STEMI and NSTEMI groups (P=0.560) as in Table 3.

Table 3: Comparison of lipid profile between control and two patient's groups

Parameter		Controls, N=74	STEMI, N=38	NSTEMI, N=38
HDL (mg/dl)	Mean ± SD	38.96±10.93	26.89±7.43	28.37±9.43
	Median (Range)	38 (20-89)	26 (15-47)	27.5(17-60)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.560[a]
LDL (mg/dl)	Mean ± SD	112.77±18.15	154.84±46.24	180.26±58.5
	Median (Range)	113.4(35.2-144.6)	142.5(89-255)	175.5(98-315)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.094[a]
TG (mg/dl)	Mean ± SD	91.08±20.68	218.95±72.24	240.18±66.48
	Median (Range)	88 (44-148)	201(104-390)	253.5(128-385)
	P-Value *		<0.001[b]	<0.001[b]
	P-Value **			0.186[b]
TC (mg/dl)	Mean ± SD	169.78±12.83	225.5±43.65	255.92±57.2
	Median (Range)	169.5(135-193)	210(159-312)	247(178-382)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.021[a]

*Comparison between control with STEMI and NSTEMI, **Comparison between STEMI and NSTEMI, a: P-Value by Mann Whitney test, b: P-Value by unpaired t-test

The study found significantly higher HbA1c and fasting blood sugar (FBS) levels in both STEMI and NSTEMI groups compared to the control group (P<0.001). The mean HbA1c was 7.45±1.55% in STEMI and 7.82±1.26% in NSTEMI, compared to 4.15±0.98% in controls. FBS levels were also elevated, with STEMI at 287.43±160.42 mg/dl and NSTEMI at 291.42±146.12 mg/dl, compared to 97.8±14.05 mg/dl in controls. No significant differences were observed between the STEMI and NSTEMI groups for either HbA1c (P=0.397) or FBS (P=0.571). As in Table 4.

Table 4: Comparison of fasting blood sugar and glycated hemoglobin between control and two patients groups.

Parameter		Controls, N=74	STEMI, N=38	NSTEMI, N=38
FBS (mg/dl)	Mean ± SD	97.8±14.05	287.43±160.42	291.42±146.12
	Median (Range)	98.5 (73-140)	245.75 (115-652)	254 (123-650)
	P-Value *		<0.001[a]	<0.001[a]
	P-Value **			0.571[a]
HbA1c (%)	Mean ± SD	4.15±0.98	7.54±1.55	7.82±1.26
	Median (Range)	4.2 (1.9-6.8)	7.3 (5.1-10)	7.55 (4.5-10.2)
	P-Value *		<0.001[b]	<0.001[b]
	P-Value **			0.397[b]

* Comparison between control with STEMI and NSTEMI, ** Comparison between STEMI and NSTEMI, a: P-Value by Mann Whitney test, b: P-Value by unpaired t-test.

The mean value for DHEAS in STEMI smoking group was (65, 91+ 32.15) ug/dl. The mean value for DHEAS STEMI nonsmoking patients was (55.27+19.62) ug/dl. There was no significant difference between the patient groups and the control group (P-Value 0.479). The mean value for DHEAS in NSTEMI smoking group was (80.23+53.62) ug/dl. The mean value for DHEAS NSTEMI nonsmoking patients was (84.63+68.8) ug/dl. There was no significant difference between the patient groups and the control group (P-Value 0.804). As in Table 5.

Table 5: Comparison of DHEAS according to smoking in STEMI and NSTEMI patients

Parameter	STEMI	Smoker, N=23	Non-Smoker, N=15	P-Value
DHEAS (µg/dl)	Mean ± SD	65.91±32.15	55.27±19.62	0.479[a]
	Median (Range)	65 (29-159)	50 (36-105)	
Parameter	**NSTEMI**	**Smoker, N=22**	**Non-Smoker, N=16**	**P-Value**
DHEAS (µg/dl)	Mean ± SD	80.23±53.62	84.63±68.8	0.804[a]
	Median (Range)	84.5 (20-230)	65.5 (19-249)	

The study found the following correlations between serum DHEAS levels and various parameters:

- **Age:** There was a highly significant negative correlation between serum DHEAS and age in the control (r=-0.523, *p*<0.001), STEMI (r=-0.322, P<0.005), and NSTEMI groups (r=-0.484, *p*<0.048).
- **BMI:** There was a positive correlation with BMI in the control group (r=0.232, P=0.047), but negative, non-significant correlations in the STEMI (r=-0.081, P=0.629) and NSTEMI groups (r=-0.110, P=0.511).
- **Blood Pressure:** Correlations with systolic and diastolic blood pressure were weak and not statistically significant across all groups.
- **High-Sensitivity Troponin I:** There was no significant correlation between serum DHEAS and troponin I levels in any group.
- **Lipid Profile:** Correlations between DHEAS and lipid profile components (TC, TG, LDL-C, and HDL-C) were weak and not statistically significant across all groups.
- **HbA1c and Fasting Blood Sugar:** There were non-significant negative correlations between DHEAS and HbA1c and fasting blood sugar in the STEMI and NSTEMI groups. As in Table 6.

Table 6: Correlation of DHEAS with other parameters in patients and controls.

Parameters		DHEAS			
		Control	All patients	STEMI	NSTEMI
Age (yr)	r	-0.523	-0.322	-0.484	-0.323
	p	<0.001	0.005	0.002	0.048
BMI (kg/m^2)	r	0.232	0.029	-0.081	0.110
	p	0.047	0.802	0.629	0.511
Syst. BP (mmHg)	r	-0.148	0.071	0.162	0.002
	p	0.208	0.545	0.330	0.992
Diast. BP (mmHg)	r	0.020	0.070	0.050	0.031
	p	0.865	0.546	0.766	0.853
Troponin (ng/l)	r	-0.034	0.092	0.227	-0.025
	p	0.772	0.431	0.171	0.882
HDL (mg/dl)	r	0.020	-0.017	-0.128	-0.004
	p	0.868	0.887	0.445	0.981
LDL (mg/dl)	r	-0.181	0.099	0.169	0.007
	p	0.123	0.396	0.310	0.965
TG (mg/dl)	r	0.065	-0.144	0.007	-0.304
	p	0.584	0.213	0.965	0.063
TC (mg/dl)	r	-0.088	0.068	0.162	-0.050
	p	0.455	0.558	0.331	0.766
FBS (mg/dl)	r	-0.080	-0.107	-0.137	-0.114
	p	0.496	0.358	0.412	0.497
HbA1c (%)	r	-0.022	-0.037	-0.112	-0.040
	p	0.854	0.753	0.502	0.813

Discussion

In the present study, the findings demonstrate a significant correlation between hypertension, obesity, smoking, dyslipidemia, hyperglycemia, and low DHEAS levels with the incidence of acute coronary syndrome (ACS), including STEMI and NSTEMI. These results are consistent with a large body of research, reinforcing the understanding that modifiable risk factors play a critical role in the development of cardiovascular diseases [14]. Hypertension was a key finding, with STEMI and NSTEMI patients showing significantly higher systolic and diastolic blood pressure compared to the control group. This result is consistent with a study by Tevestin *et al.* [15], which indicated that increased blood pressure was a significant risk factor for ACS across both genders. The association between hypertension and coronary artery disease (CAD) can be attributed to the increased mechanical stress on blood vessels, leading to endothelial dysfunction and subsequent atherosclerosis [16]. Elevated blood pressure increases left ventricular afterload and peripheral vascular resistance, resulting in structural remodeling of the left ventricle and, over time, heart failure due to increased stiffness [17]. Obesity, measured by body mass index (BMI), was also significantly higher in ACS patients than in controls. This is consistent with the findings of Khan *et al.* [18], who highlighted the increased cardiovascular mortality associated with obesity. Studies by Mirza *et al.* [19] and Unamuno *et al.* [20] further confirmed that obesity is a risk factor for atherosclerosis, and a study by Demirci *et al.* [21] demonstrated that obese individuals experience ACS at a younger age. The pathophysiological mechanisms underlying this include metabolic disturbances and dysfunction of white adipose tissue, which lead to chronic inflammation and activation of the renin-angiotensin system, resulting in elevated blood pressure and increased cardiovascular risk [22]. Smoking was another major factor, with a significant portion of ACS patients being smokers compared to the control group. This finding is supported by the work of Kondo *et al.* [23], who demonstrated that even light smoking increases the risk of cardiovascular events. The oxidative stress and inflammation caused by smoking lead to endothelial dysfunction, promoting atherosclerosis and increasing the risk of ACS [24]. Additionally, reducing smoking intensity has been shown to lower the incidence of cardiovascular disease, as observed in the study by Joanne T Chang *et al.* [25]. Dyslipidemia, characterized by elevated total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C), along with decreased high-density lipoprotein cholesterol (HDL-C), was prevalent among ACS patients. These findings align with studies by Georgoulis *et al.* [26 and Kaneko *et al.* [27], which identified dyslipidemia as a critical predictor of cardiovascular disease, especially in younger populations. The pathophysiology involves excessive lipid deposition in the arterial walls, leading to endothelial damage and foam cell formation, which are central to atherosclerotic plaque development and subsequent cardiovascular events [28, 29]. Moreover, elevated hs-Troponin-i levels in ACS patients were consistent with previous research by Brophy *et al.* [30] and Hamaya *et al.* [31], confirming its role as a gold-standard biomarker for diagnosing ACS. The release of cardiac troponins following myocardial injury is a well-established mechanism in the pathophysiology of ACS [32]. Diabetes mellitus (DM), as reflected by elevated HbA1c and fasting blood sugar (FBS) levels in ACS patients, was another significant finding. This agrees with studies by Rosenger *et al.* [33] and Ma *et al.* [34], which linked DM, particularly type 2 diabetes, with an increased risk of cardiovascular diseases. The contribution of metabolic syndrome, characterized by insulin resistance, hypertension, and dyslipidemia, to the development of atherosclerotic changes further underscores the connection between diabetes and ACS [35]. Finally, decreased serum levels of DHEAS were observed in ACS patients. This finding aligns with the studies of Zhang *et al.* [36] and Varma *et al.* [37], which suggested that higher levels of androgens, including DHEAS, provide some protection against cardiovascular disease. However, not all studies agreed; for example, the study by Jia *et al.* [38] did not find a significant association between low DHEAS levels and cardiovascular disease. These discrepancies might be explained by differences in study populations and methodologies [39].

Conclusion

Men with middle and old age have been shown to have a significantly unfavorable connection between their blood level of Dehydroepiandrosterone sulphate and Acute Coronal Syndrome. Although these results may guide future studies, the function of DHEAS in determining the occurrence of ACS may be via affecting the underlying the burden of cardiovascular illnesses.

Conflict of Interest

Not available

Financial Support

Not available

References

1. Camaro C, *et al*. Rule-out of non-ST-segment elevation acute coronary syndrome by a single, pre-hospital troponin measurement: A randomized trial. Eur Heart J. 2023;44(19):1705-1714. DOI: 10.1093/eurheartj/ehac601.
2. Amsterdam EA, *et al*. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: Executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;130(25):2354-23594. DOI: 10.1161/CIR.0000000000000133.
3. Pouralijan Amiri M, *et al*. Metabolomics in early detection and prognosis of acute coronary syndrome. Clin Chim Acta. 2019;495:43-53. DOI: 10.1016/j.cca.2019.03.045.
4. Khan AA, *et al*. The biochemical and clinical perspectives of lactate dehydrogenase: An enzyme of active metabolism. Endocr Metab Immune Disord Drug Targets. 2020;20(6):855-868. DOI: 10.2174/1871530320666200327110729.
5. Haider A, *et al*. Sex and gender in cardiovascular medicine: presentation and outcomes of acute coronary syndrome. Eur Heart J. 2020;41(13):1328-1336. DOI: 10.1093/eurheartj/ehz898.
6. Omer MA, *et al*. Clinical characteristics and outcomes of STEMI patients with cardiogenic shock and cardiac

arrest. JACC Cardiovasc Interv. 2020;13(10):1211-1219. DOI: 10.1016/j.jcin.2020.02.007.
7. Bhatt DL, *et al*. Diagnosis and treatment of acute coronary syndromes: A review. JAMA. 2022;327(7):662-675. DOI: 10.1001/jama.2022.0390.
8. Elkholi IE, *et al*. Myoglobin: From physiological roles to potential implications in cancer. Biochim Biophys Acta Rev Cancer. 2022;1877(3):188693. DOI: 10.1016/j.bbcan.2022.188693.
9. Lorubbio M, *et al*. Macro-aspartate aminotransferase in a healthy woman. Clin Mol Hepatol. 2020;26(3):378-381. DOI: 10.3350/cmh.2020.0037.
10. James S, Bueno H. Epidemiology of acute coronary syndromes. In: The ESC Textbook of Cardiovascular Medicine, 3rd Ed. ESC Publications, Oxford; c2018. DOI: 10.1093/med/9780198784906.003.0045.
11. Wierman ME, Vassiliades KK. Should Dehydroepiandrosterone be administered to women?. J Clin Endocrinol Metab. 2022;107(6):1679-1685. DOI: 10.1210/clinem/dgab726.
12. Hong SJ, Hong MK. Drug-eluting stents for the treatment of coronary artery disease: A review of recent advances. Expert Opin Drug Deliv. 2022;19(3):269-280. DOI: 10.1080/17425247.2022.2030245.
13. Savineau JP, Marthan R, de la Roque ED. Role of DHEA in cardiovascular diseases. Biochem Pharmacol. 2013;85(6):718-726. DOI: 10.1016/j.bcp.2012.11.020.
14. Tivesten Å, *et al*. Dehydroepiandrosterone and its sulfate predict the 5-year risk of coronary heart disease events in elderly men. J Am Coll Cardiol. 2014;64(17):1801-1810.
DOI: 10.1016/j.jacc.2014.07.969.
15. Kjeldsen SE. Hypertension and cardiovascular risk: General aspects. Pharmacol Res. 2018;129:95-99. DOI:10.1016/j.phrs.2017.11.003.
16. Mehlum MH, *et al*. Blood pressure variability and risk of cardiovascular events and death in patients with hypertension and different baseline risks. Eur Heart J. 2018;39(24):2243-2251.
DOI: 10.1093/eurheartj/ehy175.
17. Khan SS, *et al*. Association of body mass index with lifetime risk of cardiovascular disease and compression of morbidity. JAMA Cardiol. 2018;3(4):280-287. DOI: 10.1001/jamacardio.2018.0003.
18. Mirza AJ, *et al*. Risk factors for acute coronary syndrome in patients below the age of 40 years. Egypt Heart J. 2018;70(4):233-235.
DOI: 10.1016/j.ehj.2018.05.004.
19. Unamuno X, *et al*. Adipokine dysregulation and adipose tissue inflammation in human obesity. Eur J Clin Invest. 2018;48(9). DOI:10.1111/eci.12997.
20. Demirci D, *et al*. Association between obesity grade and the age of the first acute coronary syndrome: Prospective observational study. Int J Cardiol. 2022;351:93-99. DOI:10.1016/j.ijcard.2022.07.021.
21. Cuevas GJ, *et al*. Pathophysiological molecular mechanisms of obesity: A link between MAFLD and NASH with cardiovascular diseases. Int J Mol Sci. 2022;22(21):11629. DOI: 10.3390/ijms222111629.
22. Kondo T, *et al*. Effects of tobacco smoking on cardiovascular disease. Circ J. 2019;83(10):1980-1985. DOI: 10.1253/circj.CJ-19-0324.
23. Münzel T, *et al*. Effects of tobacco cigarettes, e-cigarettes, and waterpipe smoking on endothelial function and clinical outcomes. Eur Heart J. 2020;41(41):4057-4070.
DOI: 10.1093/eurheartj/ehaa485.
24. Chang JT, *et al*. Cigarette smoking reduction and health risks: A systematic review and meta-analysis. Nicotine Tob Res. 2021;23(4):635-642.
DOI: 10.1093/ntr/ntaa186.
25. Georgoulis M, *et al*. Long-term prognostic value of LDL-C, HDL-C, lp(a) and TG levels on cardiovascular disease incidence, by body weight status, dietary habits and lipid-lowering treatment: The ATTICA epidemiological cohort study (2002-2012). Lipids Health Dis. 2022;21(1):141.
DOI: 10.1186/s12944-022-01685-3.
26. Kaneko H, *et al*. Lipid profile and subsequent cardiovascular disease among young adults aged < 50 years. Am J Cardiol. 2021;142:59-65. DOI: 10.1016/j.amjcard.2020.12.047.
27. Berberich AJ, Hegele RA. A modern approach to dyslipidemia. Endocr Rev. 2022;43(4):611-653. DOI: 10.1210/endrev/bnaa036.
28. Xiang Q, *et al*. New insight into dyslipidemia-induced cellular senescence in atherosclerosis. Biol Rev Camb Philos Soc. 2022;97(5):1844-1867.
DOI: 10.1111/brv.12848.
29. Brophy JM, *et al*. Variability in high-sensitivity cardiac troponin T testing in sTable patients with and without coronary artery disease. Can J Cardiol. 2019;35(11):1505-1512.
DOI: 10.1016/j.cjca.2019.05.036.
30. Hamaya R, *et al*. High-sensitivity cardiac troponin decrease after percutaneous coronary intervention in patients with sTable coronary artery disease. Heart Vessels. 2019;34(6):948-956.
DOI: 10.1007/s00380-019-01356-7.
31. Wang XY, *et al*. The biomarkers for acute myocardial infarction and heart failure. Biomed Res Int. 2020;2020:1-14. DOI:10.1155/2020/4510343.
32. Rosengren A, Dikaiou P. Cardiovascular outcomes in type 1 and type 2 diabetes. Diabetologia. 2023;66(3): 425-437. DOI: 10.1007/s00125-022-05757-w.
33. Ma X, *et al*. GLP-1 receptor agonists (GLP-1RAs): Cardiovascular actions and therapeutic potential. Int J Biol Sci. 2021;17(8):2050-2068.
DOI: 10.7150/ijbs.58177.
34. Brannick B, Jack DS. Prediabetes and cardiovascular disease: Pathophysiology and interventions for prevention and risk reduction. Endocrinol Metab Clin North Am. 2018;47(1):33-50.
DOI: 10.1016/j.ecl.2017.10.003.
35. Zhang S, *et al*. Effect of dehydroepiandrosterone on atherosclerosis in postmenopausal women. Biosci Trends. 2022;15(6):353-364.
DOI: 10.5582/bst.2021.01349.
36. Varma B, *et al*. Associations between endogenous sex hormone levels and adipokine levels in the Multi-Ethnic Study of Atherosclerosis. Front Cardiovasc Med. 2023;9:1062460. DOI: 10.3389/fcvm.2023.1062460.
37. Jia X, *et al*. Plasma Dehydroepiandrosterone sulfate and cardiovascular disease risk in older men and women. J Clin Endocrinol Metab. 2020;105(12). DOI: 10.1210/clinem/dgaa535.

38. Jiménez MC, *et al*. Cardiovascular risk factors and Dehydroepiandrosterone sulfate among Latinos in the Boston Puerto Rican Health Study. J Endocr Soc. 2018;3(1):291-303. DOI:10.1210/js.2018-00323.

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