

Prevalence of Risk Factors in Egyptian Patients with Atherosclerotic Cardiovascular Diseases in Benha City: Hospital Based Cross Sectional Study

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Abstract:

Background: Atherosclerosis is a leading global cause of cardiovascular diseases (CVDs). Understanding the distribution of modifiable and non-modifiable risk factors across atherosclerotic conditions is essential for effective prevention. This study assessed the pattern of risk factors among Egyptian patients with coronary artery disease (CAD), cerebrovascular disease (CVD), and peripheral artery disease (PAD). **Methods:** This cross-sectional hospital-based study was conducted at Benha University Hospital over 12 months (August 2023–July 2024). A total of 1,000 adult patients with documented CAD (n=317), CVD (n=301), or PAD (n=382) were enrolled from inpatient and outpatient departments. Clinical, anthropometric, and laboratory data were collected. **Results:** There were no statistically significant differences among the groups regarding age, gender, residence, smoking status, BMI, or waist circumference. The prevalence of hypertension, diabetes mellitus, and dyslipidemia was high but evenly distributed across CAD, CVD, and PAD groups ($p > 0.05$). Significant overlap in vascular comorbidities was noted ($p < 0.001$). Laboratory parameters including fasting blood sugar (FBS), total cholesterol, triglycerides, LDL, and HDL levels showed no significant differences between groups. **Conclusion:** This study highlights a homogenous pattern of modifiable and non-modifiable atherosclerosis risk factors across CAD, CVD, and PAD among Egyptian patients. The high prevalence of shared risk factors underscores the need for integrated screening and preventive strategies targeting the entire cardiovascular continuum. **Keywords:** Atherosclerosis, Cardiovascular Disease, Coronary Artery Disease, Egypt, Risk Factors.

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Received:
Accepted:

Introduction

Cardiovascular disease (CVD) is the leading cause of mortality globally. Cardiovascular disease encompasses a diverse range of conditions, including ailments of the cardiac muscle and the vascular system that supplies the heart, brain, and other essential organs ⁽¹⁾.

CVD is the leading cause of mortality globally ⁽²⁾. CVD encompasses a diverse range of conditions, including ailments of the cardiac muscle and the vascular system that supplies the heart, brain, and other essential organs. This discussion focusses on the epidemiological transition that has rendered CVD the predominant cause of mortality globally, evaluating the transition's state by region and highlighting regional disparities in the burden of CVD. Also, the cost-effectiveness of diverse therapies targeting the principal drivers of CVD morbidity and mortality was evaluated ⁽³⁾.

Atherosclerosis is the primary aetiology of CVD, encompassing myocardial infarction (MI), heart failure, stroke, and claudication.

Atherosclerosis predominantly occurs in the intima of several medium and large arteries, particularly at bifurcation points. This is presumably controlled by blood flow characteristics, as regions subjected to normal shear stress appear to be safeguarded; in these areas, endothelial cells express atheroprotective genes. The adventitia may contribute to the development of atherosclerosis and is characterised by lymphocytic infiltrates ⁽⁴⁾.

The activation of endothelium, characterised by the production of adhesion molecules, is a first occurrence in atherosclerosis, facilitating the attachment and infiltration of mononuclear leukocytes, including monocytes and T-cells, into the intima. Dendritic cells, mast cells, and a limited number of neutrophils and B-cells may also be found in lesions, albeit less frequently than these cells. Another cell type found in lesions is smooth muscle cells (SMC), which undergo phenotypic

transformation into synthetic SMC and move from the media to the intima. The concept of atherosclerosis as an inflammatory illness is founded on the observation that immunological capable cells are prevalent in atherosclerotic lesions and are also synthesising cytokines, particularly proinflammatory cytokines ⁽⁵⁾.

Although atherosclerosis itself may reduce blood flow by stenosis and hence lead to CVD, the predominant mechanism seems to be atherothrombosis, typically occurring when plaques are compromised by proinflammatory cytokines and chemokines affecting the fibrous cap. When plaques are compromised and break, prothrombotic substances are revealed to the coagulation system, resulting in impaired blood flow and hence the onset of CVD. The primary modifiable risk factors for atherosclerosis and CVD include hypertension, smoking, diabetes, and dyslipidaemia. Furthermore, age and male gender are very important ⁽⁶⁾.

Implementing therapeutic lifestyle modifications alongside an intensive multidrug strategy aimed at normalising key cardiovascular risk factors will mitigate the atherogenic environment, diminish vascular inflammation, and significantly lower the likelihood of adverse cardiovascular events and the necessity for revascularisation procedures. Specific cardiovascular risk factors and optimum medications for primary and secondary prevention are examined ⁽⁷⁾.

This study aims to assess the pattern and incidence of risk factors for atherosclerosis, both modifiable and non-modifiable, in a cohort of Egyptian patients with atherosclerotic cardiovascular illnesses.

Patients and Method

Study design and population

This was a cross-sectional, hospital-based study done over a 12-month period from August 2023 to July 2024. This study included 1,000 patients [CAD (n=317),

CVD (n=301), or PAD (n=382)] recruited from individuals visiting the outpatient clinics and inpatient departments of Internal Medicine, Surgery, Neurology, and Cardiology, in addition to the Coronary Care Unit (CCU) and Intensive Care Unit (ICU) at Benha University Hospital. All individuals with verified atherosclerotic cardiovascular illnesses were incorporated into the study.

Eligibility criteria

This study included participants who were 18 years or older and had a documented diagnosis of atherosclerotic CVD. Patients diagnosed with acute coronary syndrome (ACS), including ST-segment elevation myocardial infarction (STEMI), non-ST segment elevation myocardial infarction (NSTEMI), or unstable angina (UA), were also eligible. Exclusion criteria were patients with non-atherosclerotic vascular disease (e.g., vasculitis, congenital vascular anomalies), recent acute infection or inflammatory disorders, severe systemic illness, or those unwilling to consent are excluded.

Atherosclerosis is characterized by endothelial dysfunction, lipid deposition, chronic inflammation, and plaque formation within the arterial wall, diagnosed by evidence of arterial plaque, stenosis, or calcification detected through imaging (e.g., ultrasound, CT angiography) or confirmed histopathologically^(8, 9).

Clinical manifestation and classification of atherosclerotic CVD

Coronary Artery Disease (CAD) Patients exhibiting symptoms including chest discomfort, angina, myocardial infarction, or a history of coronary revascularisation treatments (e.g., percutaneous coronary intervention or coronary artery bypass grafting) were categorised as having CAD. Cerebrovascular disease encompassed patients who had suffered ischaemic stroke or transient ischaemic attacks (TIAs), validated through clinical evaluation and neuroimaging examinations. This group also included patients with carotid artery

disease or a history of cerebrovascular intervention.

Peripheral arterial disease (PAD) Patients exhibiting intermittent claudication, rest discomfort, or possessing a history of peripheral artery revascularisation or amputation attributable to ischaemia were classified as having PAD. The diagnosis was corroborated by clinical manifestations and, when accessible, Doppler investigations or angiographic results.

The research examined both non-modifiable and modifiable risk factors linked to atherosclerosis such as non-modifiable risk factors (Age, gender, Family History). Alterable risk factors (Hypertension (HTN), Diabetes Mellitus (DM), Dyslipidaemia, Smoking, Sedentary Lifestyle, Alcohol Consumption, Overweight).

Cardiac assessments and terminologies

All patients had a 12-lead ECG, analysed by a cardiologist. Coronary angiography was conducted as warranted, with obstructive CAD defined as a diameter stenosis of $\geq 50\%$. An early invasive method is characterised by coronary angiography accompanied by potential intervention within 24 hours after presentation. Cardiac interventions prior to and during hospitalisation, including PCI or CABG, were documented.

Pharmacological evaluation: Medication consumption was recorded at two intervals. Upon admission (chronic medicines) and Upon discharge (in-hospital pharmacotherapy). The classes comprised anti-platelet medicines, statins, anti-ischemic medications, and therapy for heart failure.

Treatment decisions were determined by the attending cardiologist at each centre; the trial did not disrupt usual care protocols.

Data acquisition and quality assurance

All data were inputted into an online case report form (CRF) utilising secure credentials. The lead investigators routinely checked and validated data entry

to guarantee accuracy and consistency. Data were obtained with a standardised case report form during patient interviews and record inspections.

Ethical considerations

Informed consent was secured from all participants before their inclusion in the study. Approval was granted by the Research Ethics Committee of Benha Faculty of Medicine (Approval code: MS 40-8-2023).

Statistical methods

Data management and statistical analysis were conducted utilising SPSS version 25 (IBM, Armonk, New York, United States). Numerical data were presented as averages and standard deviations or medians and ranges. Categorical data was presented as numerical values and percentages. Comparisons were conducted using the independent t-test for normally distributed numerical data and the Mann-Whitney U test for non-normally distributed numerical data. Categorical data were analysed based on gender and residence via the Chi-square test. All P values were bilateral. P values below 0.05 were deemed significant⁽¹⁰⁾.

Results

Comparison between all studied groups according to disease type

An analysis of the ages of the three groups indicated no age disparity among them. The mean age showed no significant difference among the CAD group (56.06 ± 9.71 years), the CVD group (54.43 ± 9.42 years), and the PAD group (54.94 ± 9.55 years) ($F = 2.388$, $p = 0.092$). The three groups exhibited a nearly similar distribution of males and females, with male percentages of 59.3%, 61.1%, and 60.7% in the CAD, CVD, and PAD groups, respectively. The chi-square test indicated no significant difference between disease type and gender ($\chi^2 = 0.243$; $p = 0.885$).

A limited, non-significant inclination was observed towards a greater percentage of urban cases in the CVD group (56.1%) compared to the CAD (49.2%) and PAD

(47.4%) groups. The overall disparity between the two was not statistically significant ($\chi^2 = 5.531$, $p = 0.063$). The distribution of smoking status was uniform across all categories (CAD: 27.1%, CVD: 30.2%, and PAD: 31.7%) ($P = 0.417$), indicating no correlation between smoking behaviour and illness type in the examined cohort.

Among CAD patients, 31.86% exhibited concomitant CVD and 25.87% presented with peripheral artery disease (PAD), whereas 42.27% of CAD patients were devoid of associated vascular illness. In individuals with CVD, only 16.61% were free of other vascular conditions, whereas 55.48% had CAD and 27.91% had PAD. Approximately 67.80% of the PAD cohort had CAD, whereas 32.20% had CVD; there were no patients devoid of concomitant vascular illness. All relationships were extremely significant ($p < 0.001$), indicating a major overlap in vascular comorbidities among the various groups.

Anthropometric measurements, including weight, exhibited no significant differences across the groups, with mean body weights for CAD, CVD, and PAD patients recorded at 77.92 ± 10.18 kg, 78.22 ± 10.07 kg, and 78.97 ± 10.22 kg, respectively ($F = 0.996$, $p = 0.370$). The height was comparable across groups, with a median of 1.67 m in both the CAD and PAD groups, and 1.68 m in the CVD group. The interquartile ranges were comparable, and no significant difference was detected ($H = 0.078$, $p = 0.962$).

The Body Mass Index (BMI) did not exhibit statistically significant differences among the three groups. The BMI was 27.85 (IQR: 24.96–30.48) in the CAD group, 27.72 (IQR: 25.15–30.81) in the CVD group, and 28.14 (IQR: 25.39–30.86) in the PAD group, with the difference being non-significant as per the Kruskal-Wallis test ($H = 1.882$, $p = 0.390$). Waist circumference (WC), an indicator of abdominal obesity, was likewise similar among the three groups. The average waist

circumference (WC) for the CAD group was 102.16 ± 10.64 cm, for the CVD group was 101.17 ± 10.34 cm, and for the PAD group was 101.81 ± 10.46 cm ($F = 0.706$, $p = 0.494$). The gender-separate analysis of waist circumference was also non-significant (females: $p = 0.2552$; males: $p = 0.9568$). **Table 1**

Table 2 indicates that Hypertension (HTN) was prevalent across all three groups: 44.8% in CAD, 49.8% in CVD, and 44.2% in PAD. The CVD group had the highest ratio; nevertheless, there was no significant difference ($\chi^2 = 2.438$, $p = 0.296$) in the distribution of hypertension among illness classes. The mean systolic blood pressure (SBP) did not exhibit significant differences across the groups: CAD patients 127.54 ± 11.62 mmHg, CVD patients 127.77 ± 11.75 mmHg, and PAD patients 127.04 ± 11.22 mmHg ($F = 0.366$, $p = 0.694$), indicating comparable control or baseline levels of systolic pressure across the groups.

Diastolic blood pressure (DBP) was seen to be elevated in CVD at 76.01 ± 8.61 mmHg, relative to CAD at 74.98 ± 8.85 mmHg and PAD at 74.50 ± 8.67 mmHg; however, this difference did not achieve statistical significance ($F = 2.582$, $p = 0.076$). The prevalence of dyslipidaemia was comparable across all disease types: CAD (45.1%), CVD (45.2%), and PAD (47.4%). The homogeneity was confirmed by a non-significant finding ($\chi^2 = 0.476$, $p = 0.788$), demonstrating no disease-specific differences in lipid abnormalities. A 19.6% of patients with CAD, 17.6% of patients with cerebrovascular disease (CVD), and 18.1% of patients with PAD had a familial history of atherosclerotic CAD. No significant difference was observed ($\chi^2 = 0.438$, $p = 0.803$), indicating a uniform genetic risk across vascular characteristics. Diabetes mellitus (DM) was common across all patients, affecting 47.0% of those with CAD, 49.5% with cerebrovascular disease (CVD), and 46.1% with PAD. The prevalence of DM

did not substantially vary by disease type ($\chi^2 = 0.823$, $p = 0.663$).

The prevalence of Type 2 Diabetes Mellitus (T2DM) was 24.9%, 27.2%, and 24.6% in CAD, CVD, and PAD, respectively, while the prevalence of Type 1 Diabetes Mellitus (T1DM) was 22.1%, 22.6%, and 21.5%, respectively. No significant difference was seen between the distributions ($\chi^2 = 1.093$, $p = 0.895$), suggesting analogous patterns of diabetes subtypes among patients with vascular disorders.

The assessment of diabetic complications indicated that a minimal number of patients experienced multiple diabetes-related complications; 82.3% in the CAD group, 78.4% in the CVD group, and 83.0% in the PAD group reported none. No substantial difference existed between the two groups ($\chi^2 = 2.300$, $p = 0.317$), indicating that the burden of complications cannot be readily attributed to illness type. In **Table 3**, The fasting blood sugar (FBS) means among the three groups were comparable: 120.89 ± 17.05 mg/dL in CAD patients, 120.43 ± 17.67 mg/dL in CVD patients, and 120.09 ± 17.37 mg/dL in PAD patients. The absence of a substantial difference between the values in the two groups ($F = 0.186$, $p = 0.830$) indicates that the glycaemic profile, as evaluated by FBS, was comparable across the illness types.

No significant difference was seen among the three groups concerning total cholesterol levels (205.88 ± 42.10 mg/dL vs 208.54 ± 40.37 mg/dL vs 207.54 ± 42.70 mg/dL). No notable variations were seen in the lipid profiles of TC among the three vascular disorders ($F = 0.321$, $p = 0.726$), indicating that the lipid profiles of TC were comparable among the three conditions.

Triglyceride levels were comparable among groups (CAD: 223.29 ± 40.02 mg/dL, CVD: 224.04 ± 41.48 mg/dL, and PAD: 225.81 ± 41.14 mg/dL). The difference was non-significant ($F = 0.354$,

$p = 0.702$), suggesting the uniformity of lipid problems among the disorders.

No significant differences in LDL cholesterol were seen among CAD [mean, 125.85 ± 22.89 mg/dL], CVD [125.07 ± 22.93 mg/dL], and PAD [126.88 ± 23.67 mg/dL] ($F = 0.524$, $p = 0.592$). This indicates that LDL cholesterol, a primary atherogenic factor, is consistently distributed across all patient categories.

High-density lipoprotein cholesterol (HDL-C), typically protective against atherosclerosis, exhibited equivalent means across groups, specifically between CAD and PAD (both 46.30 mg/dL), and

closely approximated the mean value in CVD (46.34 mg/dL), resulting in a statistically non-significant difference ($F = 0.004$, $p = 0.996$). In a comparison between sexes, female HDL levels were marginally elevated across all groups: 47.77 ± 7.73 mg/dL in CAD, 47.00 ± 7.05 mg/dL in CVD, and 47.16 ± 7.16 mg/dL in PAD, with no statistically significant difference seen ($p = 0.686$). In males, HDL levels were comparable, measuring 45.29 ± 6.66 mg/dL in CAD, 45.92 ± 6.81 mg/dL in CVD, and 45.75 mg/dL in PAD ($p = 0.6937$).

Tables 1: Comparison between all studied groups regarding demographic data.

Parameter		Coronary artery disease (n=317)	Cerebrovascular disease (n=301)	Peripheral artery disease (n=382)	p-value
Age (years)	Mean $\pm$ SD	56.06 ± 9.71	54.43 ± 9.42	54.94 ± 9.55	$p=0.092$
Gender	Male	188 (59.3%)	184 (61.1%)	232 (60.7%)	$p=0.885$
	Female	129 (40.7%)	117 (38.9%)	150 (39.3%)	
Residence	Urban	156 (49.2%)	169 (56.1%)	181 (47.4%)	$p=0.063$
	Rural	161 (50.8%)	132 (43.9%)	201 (52.6%)	
Smoking	Yes	86 (27.1%)	91 (30.2%)	121 (31.7%)	$p=0.417$
	No	231 (72.9%)	210 (69.8%)	261 (68.3%)	
Associated Disease	Cerebrovascular disease	101 (31.86%)	0 (0.00%)	123 (32.20%)	$p<0.001$
	Coronary artery disease	0 (0.00%)	167 (55.48%)	259 (67.80%)	$p<0.001$
	Peripheral artery disease	82 (25.87%)	84 (27.91%)	0 (0.00%)	$p<0.001$
	No associated disease	134 (42.27%)	50 (16.61%)	0 (0.00%)	$p<0.001$
Weight (kg)	Mean $\pm$ SD	77.92 ± 10.18	78.22 ± 10.07	78.97 ± 10.22	$p=0.370$
Height (m)	Median (IQR)	1.67 (1.63–1.71)	1.68 (1.63–1.71)	1.67 (1.63–1.71)	$p=0.962$
BMI (kg/m ²)	Median (IQR)	27.85 (24.96–30.48)	27.72 (25.15–30.81)	28.14 (25.39–30.86)	$p=0.390$
	Mean $\pm$ SD	102.16 ± 10.64	101.17 ± 10.34	101.81 ± 10.46	$p=0.494$
WC (cm)	Female	102.30 ± 10.52	100.18 ± 10.69	101.32 ± 9.91	0.2552
	Male	102.06 ± 10.74	101.80 ± 10.08	102.13 ± 10.81	0.9568

SD: Standard Deviation, IQR: Interquartile Range, BMI: Body Mass Index, WC: Waist Circumference, kg: Kilogram, m: Meter, cm: Centimeter, $p \leq 0.05$ is regarded significant.

Tables 2: CVS risk factors in studied groups according to disease type.

Parameter	Category	Coronary artery disease (n=317)	Cerebrovascular disease (n=301)	Peripheral artery disease (n=382)	p-value
HTN	Yes	142 (44.8%)	150 (49.8%)	169 (44.2%)	p=0.296
	No	175 (55.2%)	151 (50.2%)	213 (55.8%)	
SBP (mmHg)	Mean $\pm$ SD	127.54 $\pm$ 11.62	127.77 $\pm$ 11.75	127.04 $\pm$ 11.22	p=0.694
DBP (mmHg)	Mean $\pm$ SD	74.98 $\pm$ 8.85	76.01 $\pm$ 8.61	74.50 $\pm$ 8.67	p=0.076
Dyslipidemia	Yes	143 (45.1%)	136 (45.2%)	181 (47.4%)	p=0.788
	No	174 (54.9%)	165 (54.8%)	201 (52.6%)	
Family history of atherosclerotic CAD	No	255 (80.4%)	248 (82.4%)	313 (81.9%)	p=0.803
	Yes	62 (19.6%)	53 (17.6%)	69 (18.1%)	
DM	Yes	149 (47.0%)	149 (49.5%)	176 (46.1%)	p=0.663
	No	168 (53.0%)	152 (50.5%)	206 (53.9%)	
Type of DM	T2DM	79 (24.9%)	82 (27.2%)	94 (24.6%)	p=0.895
	No	168 (53.0%)	151 (50.2%)	206 (53.9%)	
Other complications of DM	T1DM	70 (22.1%)	68 (22.6%)	82 (21.5%)	p=0.317
	No	261 (82.3%)	236 (78.4%)	317 (83.0%)	
	Yes	56 (17.7%)	64 (21.3%)	65 (17.0%)	
	nan	0 (0.0%)	0 (0.0%)	0 (0.0%)	

HTN: Hypertension, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, CAD: Coronary Artery Disease, DM: Diabetes Mellitus, T2DM: Type 2 Diabetes Mellitus, T1DM: Type 1 Diabetes Mellitus, SD: Standard Deviation, mmHg: Millimeters of Mercury, $p \leq 0.05$ is regarded significant.

Table 3: Laboratory Parameters in Studied Groups According to Disease Type

Parameter	Category	Coronary artery disease (n=317)	Cerebrovascular disease (n=301)	Peripheral artery disease (n=382)	p-value
FBS (mg/dL)	Mean $\pm$ SD	120.89 $\pm$ 17.05	120.43 $\pm$ 17.67	120.09 $\pm$ 17.37	p=0.830
Total Cholesterol (mg/dL)	Mean $\pm$ SD	205.88 $\pm$ 42.10	208.54 $\pm$ 40.37	207.54 $\pm$ 42.70	p=0.726
Triglycerides (mg/dL)	Mean $\pm$ SD	223.29 $\pm$ 40.02	224.04 $\pm$ 41.48	225.81 $\pm$ 41.14	p=0.702
LDL (mg/dL)	Mean $\pm$ SD	125.85 $\pm$ 22.89	125.07 $\pm$ 22.93	126.88 $\pm$ 23.67	p=0.592
	Mean $\pm$ SD	46.30 $\pm$ 7.21	46.34 $\pm$ 6.91	46.30 $\pm$ 7.09	p=0.996
HDL (mg/dL)	Female	47.77 $\pm$ 7.73	47.00 $\pm$ 7.05	47.16 $\pm$ 7.16	p=0.686
	Male	45.29 $\pm$ 6.66	45.92 $\pm$ 6.81	45.75 $\pm$ 7.00	p=0.693

FBS: Fasting Blood Sugar, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, mg/dL: Milligrams per Deciliter, SD: Standard Deviation, $p \leq 0.05$ is regarded significant.

Discussion

Cardiovascular disease (CVD) is the predominant global cause of mortality, contributing to substantial death and disability, especially from atherosclerosis-related illnesses such as coronary heart disease, stroke, and peripheral artery disease^(6, 11). Therefore, this study aims to assess the pattern and prevalence of risk factors for atherosclerosis in Egyptian patients with atherosclerotic cardiovascular illnesses.

The average age of patients with CAD was 56.06 ± 9.71 years, whereas it was 54.43 ± 9.42 years for cerebrovascular disease (CVD) and 54.94 ± 9.55 years for peripheral arterial disease (PAD), with no statistically significant difference ($p = 0.092$). These findings align with Reda et al.⁽¹²⁾, who documented a mean age of 55.8 years in the CardioRisk project. Khalfallah et al.⁽¹³⁾ indicated elevated mean ages in a Middle Delta cohort (59.3 ± 10.1 years), potentially signifying regional variations in healthcare accessibility and exposure to risk factors.

In CAD patients, the mean age is 56.06 ± 9.71 years. This age corresponds with the findings of Reda et al.⁽¹²⁾, Elkersh et al.⁽¹⁴⁾, and Reda et al.⁽¹⁵⁾, indicating that the majority of CAD patients in Egypt are aged between 50 and 60 years. Khalfallah et al.⁽¹³⁾ indicated a marginally elevated mean age of 58.9 years in the Middle Delta, potentially attributable to delayed diagnosis or more advanced disease in underprivileged areas. This contrasts with the findings of El-Moselhy et al.⁽¹⁶⁾, who documented a markedly elevated age-related prevalence of CAD in elderly Egyptian patients. The absence of a notable difference in this study may be ascribed to the coinciding age of onset and same pathophysiological mechanisms among atherosclerotic illnesses, as emphasised by Adhikary et al.⁽³⁾.

In patients with CVD, the mean age is 54.43 ± 9.42 years. These statistics indicate the onset of CVD in mid-life,

corroborated by Reda et al.⁽¹²⁾, who noted stroke incidents in the fifth and sixth decades of life. Elkersh et al.⁽¹⁴⁾ documented an analogous mean age of 55.2 years. Khedr et al.⁽¹⁷⁾ discovered a marginally older average age (≈ 58 years) among southern Egyptian stroke patients, potentially attributable to demographic and diagnostic variables.

In patients with PAD, the mean age is 54.94 ± 9.55 years. Patients with PAD in this study are also within the age range of 50 to 60 years. Reda et al.⁽¹²⁾ observed an increasing frequency of PAD in this demographic, particularly among individuals with concurrent CAD. Khalfallah et al.⁽¹³⁾ indicated an older group with PAD (mean age 59.7 years), presumably attributable to delayed diagnosis in rural regions. The younger PAD cohort may indicate enhanced screening or an earlier manifestation of the illness.

Male preponderance was noted in all three groups as follows: CAD: 59.3%, CVD: 61.1%, PAD: 60.7% (Table 1). The differences were not statistically significant ($p = 0.885$), but they indicate a consistent pattern observed in Egyptian cardiovascular literature.

Elkersh et al.⁽¹⁴⁾ noted that male representation surpassed 62% in acute coronary syndrome hospitalisations, whereas Reda et al.⁽¹⁵⁾ highlighted male gender as a significant risk factor in phase II of the CardioRisk trial. This trend is frequently ascribed to heightened exposure among men to modifiable risk factors such as smoking, occupational stress, and insufficient participation in preventive healthcare⁽¹⁸⁾. However, El Sayed et al.⁽¹⁹⁾ indicated that women—particularly those who are diabetic and postmenopausal—may encounter similar risks, implying that comorbidities and hormonal alterations can diminish the conventional gender disparity.

El Sayed et al.⁽¹⁹⁾ highlighted that disparities in cardiovascular problems related to sex and gender are diminishing,

attributed to the rising frequency of risk factors among females, particularly within diabetes cohorts.

CAD: males represented 59.3%, consistent with the findings of Kamal et al. ⁽¹⁸⁾ and Reda et al. ⁽¹⁵⁾, who ascribed the male predominance in CAD to increased exposure to risk factors. However, El Sayed et al. ⁽¹⁹⁾ discovered that diabetic postmenopausal women exhibit a comparable CAD risk, underscoring the shifting gender dynamics in the context of comorbidities.

CVD exhibits a male predominance of 61.1%, consistent with the findings of Kamal et al. ⁽¹⁸⁾, Reda et al. ⁽¹⁵⁾, and Elhfnawy et al. ⁽²⁰⁾, which documented analogous trends in CVD populations. The gap is largely attributable to elevated smoking rates, occupational stress, and insufficient utilisation of preventive treatment among males. Nevertheless, El Sayed et al. ⁽¹⁹⁾ highlighted that postmenopausal diabetic women are more impacted, especially regarding metabolic management.

PAD exhibits a male preponderance of 60.7%. This male predominance is attributed to lifestyle variables, as seen by Elkersh et al. ⁽¹⁴⁾ and Kamal et al. ⁽¹⁸⁾, in contrast to the findings of Reda et al. ⁽¹⁵⁾ and El.

El Sayed et al. ⁽¹⁹⁾ revealed that the risk of PAD in females escalates with age and the presence of concomitant diseases, particularly diabetes and hypertension. All comparisons revealed no statistically significant changes, affirming the persistent male preponderance throughout vascular diseases. The findings correspond with national trends outlined by Kamal et al. ⁽¹⁸⁾ and Elkersh et al. ⁽¹⁴⁾; however, recent studies—particularly El Sayed et al. ⁽¹⁹⁾—underscore the necessity of monitoring increasing vascular risk in diabetic and postmenopausal women, indicating a potential convergence of the historical gender gap within these populations.

Nevertheless, research conducted by El Sayed et al. ⁽¹⁹⁾ identified an escalating prevalence of PAD among Egyptian women attributed to heightened rates of diabetes and smoking.

The identified gender disparity in this cohort may indicate variations in traditional risk exposure, especially for occupational activities and the postponement of diagnosis in females.

In terms of residence, urban residency was more prevalent among: CVD patients at 56.1%, CAD patients at 49.2%, and PAD patients at 47.4%. Despite lacking overall statistical significance ($p = 0.063$), this trend indicates changing patterns of atherosclerotic disease distribution in Egypt. The research revealed no substantial disparity in disease prevalence between urban and rural populations ($p = 0.063$). Despite metropolitan areas exhibiting marginally elevated percentages within the CVD category, this difference lacked statistical significance. In contrast, Reda et al. ⁽¹²⁾ identified a correlation between urban residency and elevated cardiovascular risk in Egypt, presumably attributable to sedentary lifestyles and eating habits. The present data may indicate a diminishing disparity between urban and rural lifestyles and healthcare availability, as shown by Francis et al. ⁽²¹⁾. This contrasts with Hussein et al. ⁽²²⁾, who indicated elevated cardiovascular risk in urban settings due to lifestyle and dietary practices; conversely, Khalfallah et al. ⁽¹³⁾ identified increased CAD incidence in rural Middle Delta regions, ascribed to inadequate healthcare access and delayed diagnosis.

The almost identical urban-rural distribution in Table 3 may indicate a nationwide trend towards uniform risk, possibly attributable to the proliferation of sedentary lifestyles and high-calorie diets across both urban and rural populations.

Concerning smoking, it was documented among: Patients with PAD: 31.7%, Patients with CVD: 30.2%, Patients with CAD: 27.1%. The difference was not

statistically significant ($p = 0.417$), indicating that smoking continues to be a prevalent modifiable risk factor across all types of atherosclerotic diseases. This contradicts research by Ellaien et al. ⁽²³⁾ and Elhfnawy et al. ⁽²⁰⁾, which identified smoking as a major predictor of CAD and stroke. The insignificance in this dataset may arise from underreporting or regional discrepancies in smoking behaviours, despite the recognised correlation between smoking and vascular disease.

Conversely, Ellaien et al. ⁽²³⁾ discovered that in young CAD patients, familial hypercholesterolaemia surpassed smoking as the primary risk factor, indicating that genetic and lifestyle risk factors may interact variably across different age cohorts.

Hussein et al. ⁽²²⁾ documented elevated smoking rates among rural diabetics, further suggesting that comorbidities and geographic location affect smoking behaviours.

Concerning anthropometric characteristics of patients across all groups, CAD, cerebrovascular disease (CVD), and peripheral arterial disease (PAD)—demonstrated comparable anthropometric profiles, with no statistically significant variations in weight, height, BMI, or waist circumference (WC). The average weight exhibited minimal variation: 77.92 kg in CAD, 78.22 kg in CVD, and 78.97 kg in PAD ($p = 0.370$). The median results were consistent (77–79 kg, IQR: 70–86), closely aligning with Sobhy et al. ⁽²⁴⁾, who documented a mean weight of approximately 78.3 kg in Egyptian patients with acute coronary syndrome. This finding contrasts with the conclusions of Abd El-Gawad et al. ⁽²⁾ and Hussein et al. ⁽²²⁾, who identified obesity as a significant predictor of CAD and PAD in Egypt. A rationale could be that weight alone may inadequately represent risk without accounting for fat distribution or metabolic condition.

The BMI values among the three groups demonstrated a uniform tendency of

overweight status: 27.85 in CAD, 27.72 in CVD, and 28.14 kg/m² in PAD ($p = 0.390$). These results correspond with Reda et al. ⁽¹²⁾, who reported average BMIs of 28.3 ± 5.1 among Egyptian vascular patients. Arafa et al. ⁽⁴⁾ and Sobhy et al. ⁽²⁴⁾ reported analogous trends. Conversely, elevated BMI levels (>30 kg/m²) were noted in diabetic vascular populations, as indicated by Laimoud et al. ⁽²⁵⁾ and Khalfallah et al. ⁽¹³⁾, implying that concomitant diabetes may exacerbate obesity in particular subgroups. This contradicts with the findings of Azab et al. ⁽⁶⁾, which indicated that BMI significantly affected CAD risk in obese compared to non-obese persons. The comparable BMI across groups indicates that being overweight is a prevalent background risk factor, irrespective of the kind of vascular disease.

Waist circumference (WC) was increased in all patients, signifying pervasive central obesity. The mean waist circumference values varied from 101.17 cm in CVD to 102.16 cm in CAD, with no statistically significant variations between genders or groups ($p > 0.1$). These measurements surpass the 102 cm level deemed a risk indicator for metabolic syndrome and cardiovascular incidents. This corresponds with the findings of Ibrahim et al. ⁽²⁶⁾, who determined that a waist circumference over 102 cm is a significant predictor of early CAD and PAD in Egyptians. Arafa et al. ⁽⁴⁾ correlated increasing waist circumference with genetic variants related to heightened coronary and cerebrovascular risk. This contradicts research by El Faramawy et al. ⁽²⁷⁾ and Kamal et al. ⁽¹⁸⁾, which demonstrated a robust correlation between higher waist circumference and cardiovascular risk as well as disease severity. The insignificant result may indicate the prevalence of abdominal obesity across various types of vascular disease in Egypt.

Regionally, Shehab et al. ⁽²⁸⁾ indicated elevated waist circumference averages (>106 cm) among UAE patients, implying

regional or lifestyle-related variability in central obesity patterns. Nonetheless, the WC findings in this study suggest a high metabolic load shared across all vascular disease groups.

Concerning blood pressure and hypertension, Hypertension (HTN) was common throughout all vascular disease categories, exhibiting no statistically significant differences: 44.8% in CAD, 49.8% in cerebrovascular disease (CVD), and 44.2% in PAD ($p = 0.296$). These percentages align with national statistics from the CardioRisk project ⁽¹²⁾, which identified hypertension in approximately 47% of acute coronary syndrome (ACS) patients throughout Egypt. This finding contrasts with the conclusions of Elkersh et al. ⁽¹⁴⁾ and Francis et al. ⁽²¹⁾, who identified a more pronounced correlation between hypertension and stroke or PAD. The minimal variance observed may indicate the elevated baseline prevalence of hypertension among all vascular disease categories in Egypt, as shown by the Egyptian Hypertension Clinics Registry ⁽²⁷⁾.

The prevalence of CAD (44.8%) corresponds with the findings of Elkersh et al. ⁽¹⁴⁾ in Menoufia (45%), although PAD patients had comparable values at 44.2%. A marginally elevated prevalence (49.8%) was seen in CVD, consistent with the results of Elkersh et al. ⁽¹⁴⁾ and Reda et al. ⁽¹⁵⁾. Khedr et al. ⁽¹⁷⁾ showed markedly elevated hypertension rates (>65%) in ischaemic stroke patients, potentially indicative of demographic, regional, or clinical disparities, especially in more severe or uncontrolled instances.

Concerning dyslipidaemia and lipid profile, Dyslipidaemia was identified in around 45% of patients—45.1% in CAD, 45.2% in CVD, and 47.4% in PAD—without statistically significant differences ($p=0.788$), highlighting its common involvement in atherosclerotic disorders. These findings correspond with Sobhy et al. ⁽²⁴⁾ and Reda et al. ⁽¹²⁾, who documented comparable frequencies (44–48%) of lipid

abnormalities in Egyptian patients with ACS and stroke. Elhfnawy et al. ⁽²⁰⁾ observed elevated dyslipidaemia rates above 60% in patients with carotid atherosclerosis, indicating possible differences based on vascular region or disease severity. The mean total cholesterol levels varied between 205.88 and 208.54 mg/dL ($p=0.726$), beyond the recommended limit (<200 mg/dL) and aligning with the national averages documented by Sobhy et al. ⁽²⁴⁾. Triglyceride levels were uniformly increased across all groups (mean ≈ 223 –226 mg/dL, $p=0.702$), significantly beyond the optimum threshold of 150 mg/dL, corroborating findings by Assem et al. ⁽⁵⁾ and regional investigations by Shehab et al. ⁽²⁸⁾. LDL-C readings were consistently elevated (≈ 125 –127 mg/dL, $p=0.592$), above secondary prevention objectives (<100 mg/dL), corroborating findings by Reda et al. ⁽¹²⁾ and Sobhy et al. ⁽²⁴⁾, who emphasised ongoing deficiencies in lipid management across the nation. HDL-C levels were similar across groups (mean ≈ 46.3 mg/dL, $p=0.996$), exhibiting anticipated gender disparities—females exhibited greater HDL levels than males, albeit frequently below optimal sex-specific thresholds. These findings jointly underscore the systemic characteristics of dyslipidaemia throughout vascular regions and emphasise the necessity for more assertive lipid management approaches in high-risk Egyptian populations. This homogeneity stands in contrast to Sobhy et al. ⁽²⁴⁾, who identified elevated frequencies of lipid abnormalities in patients with CAD. The observed similarity may indicate prevalent dietary risk factors and the underdiagnosis or undertreatment of lipid diseases in Egypt, as highlighted by Reda et al. ⁽¹²⁾.

Diabetes mellitus (DM) exhibited similar prevalence across the three vascular categories, impacting 47.0% of CAD patients, 49.5% of cerebrovascular patients, and 46.1% of PAD patients, with no statistically significant difference ($p =$

0.663). These percentages correspond with earlier regional research, like Hussein et al. ⁽²²⁾, which documented a 48% prevalence of diabetes among cardiovascular patients in Upper Egypt. Reda et al. ⁽¹²⁾ and Laimoud et al. ⁽²⁵⁾ also reported that over fifty percent of Egyptian patients with vascular disease have diabetes. Khalfallah et al. ⁽¹³⁾ observed a diabetes prevalence over 60% in PAD patients, possibly indicating a greater atherosclerotic burden or inequities in diabetes management and healthcare accessibility. This corresponds with Assem et al. ⁽⁵⁾, who identified diabetes as a significant common determinant of both macrovascular and microvascular problems in Egyptian patients.

Fasting blood sugar (FBS) levels were comparable among the three groups (CAD: 120.89 ± 17.05 mg/dL, CVD: 120.43 ± 17.67 mg/dL, PAD: 120.09 ± 17.37 mg/dL), exhibiting no statistically significant difference ($p = 0.830$). This resemblance underscores the shared metabolic foundation of atherosclerotic illnesses. These findings differ marginally from those of Hussein et al. ⁽²²⁾, who indicated that FBS levels were considerably elevated in PAD relative to CVD and CAD among Egyptian diabetic patients. The existing consistency may result from comparable diabetes prevalence and glycaemic regulation among the study groups.

Concerning diabetes subtypes, type 2 diabetes mellitus (T2DM) was predominant in all groups, with no significant intergroup variance ($p = 0.895$). Type 2 diabetes mellitus (T2DM) was observed in 24.9% of CAD patients, 27.2% of CVD patients, and 24.6% of PAD patients. These findings align with El-Moselhy et al. ⁽¹⁶⁾, who established T2DM as the prevalent subtype among senior CAD patients. The prevalence of T2DM is corroborated by Assem et al. ⁽⁵⁾, who identified T2DM in more than 50% of PAD patients with macrovascular problems, and by Laimoud et al. ⁽²⁵⁾, who

connected T2DM to CAD and stroke due to its significant association with insulin resistance and dyslipidaemia.

The prevalence of type 1 diabetes mellitus (T1DM) in this cohort was notably elevated—22.1% in CAD, 22.6% in CVD, and 21.5% in PAD—statistics that surpass those documented in the majority of national investigations. Assem et al. ⁽⁵⁾ and Khalfallah et al. ⁽¹³⁾ indicated that T1DM was present in less than 10% of patients with PAD and CAD. The increased frequency of T1DM may indicate a younger patient demographic, early-onset vascular problems, or possible referral and selection biases. El-Amin et al. ⁽²⁹⁾ have shown a correlation between autoimmune or inflammatory disorders and heightened vascular risk in Type 1 Diabetes Mellitus (T1DM), indicating that comorbidities may affect diabetes subtypes and vascular outcomes.

Upon examination of diabetes-related sequelae, including nephropathy and neuropathy, the rates were moderate and statistically insignificant among the groups ($p = 0.317$). Complications were observed in 17.7% of patients with CAD, 21.3% of individuals with CVD, and 17.0% of patients with PAD. The data align with El-Amin et al. ⁽²⁹⁾, who reported a 20–25% prevalence of microvascular problems in diabetic patients with subclinical CVD. Kamal et al. ⁽¹⁸⁾ highlighted the significance of poor glycaemic control in worsening consequences, especially in CAD patients, where an increase in epicardial fat thickness was noted. The marginally elevated complication incidence in CVD patients may indicate the cerebral vasculature's susceptibility to metabolic damage or a postponed diabetes diagnosis. This finding contradicts Laimoud et al. ⁽²⁵⁾, who reported a greater incidence of sequelae such as nephropathy and retinopathy in individuals with CAD and PAD. This may indicate either early-stage diabetes mellitus in participants or insufficient screening for end-organ damage.

In patients with PAD, merely 17% demonstrated additional diabetes-related problems, a figure inferior to the 26% documented by El-Amin et al. ⁽²⁹⁾ in diabetic individuals with inflammatory bowel disease and preclinical cardiovascular impairment. This may suggest early identification of PAD or underreporting of complications in outpatient environments.

A positive family history of CAD was observed in 19.6% of cases, a figure marginally lower than the 23% documented by Ellaïen et al. ⁽²³⁾, who concentrated on individuals fulfilling criteria for familial hypercholesterolaemia. This slight disparity likely indicates the larger and more diverse characteristics of the current cohort, as research concentrating on early-onset or genetically susceptible populations typically reveals greater familial clustering. Arafa et al. ⁽⁴⁾ similarly shown that genetic polymorphisms, including ApoE variations, are more prevalent in younger CAD patients with a significant family history, indicating that age and genetic profile may affect the reported prevalence. This is unexpected considering findings from Arafa et al. ⁽⁴⁾ and Ellaïen et al. ⁽²³⁾ indicating that a positive family history markedly elevates CAD risk. The outcome may be partially attributed to underreporting resulting from inadequate family health documentation in certain places.

In cerebrovascular disease (CVD) patients, a positive family history was noted in 17.6%, somewhat lower than the 19.6% seen in CAD patients and less than the 23% reported by Ellaïen et al. ⁽²³⁾. This disparity may indicate the more complex aetiology of stroke, wherein conventional family history has a comparatively diminished influence relative to coronary occurrences. Arafa et al. ⁽⁴⁾ emphasised that although genetic polymorphisms like eNOS or ApoE may influence stroke vulnerability, their impacts are frequently

moderated by environmental and metabolic variables.

Likewise, 18.1% of patients with PAD indicated a positive family history, akin to the rates observed in both CAD and cerebrovascular disease (CVD) cohorts ($p = 0.957$). This parity strengthens the common genetic predisposition that underlies all variants of atherosclerotic CVD. According to Ellaïen et al. ⁽²³⁾, family history prevalence generally varies from 18% to 23% in vascular disease populations, while Arafa et al. ⁽⁴⁾ corroborated the significance of polymorphic markers in predisposing individuals to coronary and cerebrovascular atherosclerosis.

The found familial prevalence rates among CAD, CVD, and PAD groups indicate a constant albeit mild influence of genetic variables. Although genetic predisposition plays a role in the development of vascular disease, particularly among younger individuals or those with hereditary lipid disorders, its impact may diminish in older and more diverse groups where metabolic, behavioural, and environmental factors predominantly influence disease manifestation.

Conclusion

This study emphasises the significant burden and interrelated characteristics of atherosclerotic cardiovascular diseases—coronary artery disease, cerebrovascular disease, and peripheral artery disease—among the study participants. Notwithstanding varied clinical presentations, the three groups exhibited very analogous profiles for demographic data, cardiovascular risk factors, and laboratory markers. Prevalent modifiable risk factors, including hypertension, diabetes, smoking, and dyslipidaemia, were observed across all groups, highlighting the systemic effects of atherosclerosis and the necessity for cohesive, risk-oriented prevention and management approaches. The significant comorbidity of illness types underscores

the necessity for coordinated screening and early management to mitigate morbidity and mortality.

Study limitations

Being a single-center, hospital-based study, the generalizability of the findings to the wider national population may be restricted. In addition, its cross-sectional nature precludes establishing causal relationships between risk factors and disease occurrence or progression. Finally, reliance on standard hospital records and basic laboratory investigations may have underestimated subclinical or early atherosclerotic changes, thereby limiting the depth of diagnostic precision.

Study recommendations

The findings of this study emphasize the need for integrated prevention programs that address the common risk factors of hypertension, diabetes, dyslipidemia, and smoking across all atherosclerotic conditions, rather than focusing on each disease entity in isolation. Routine comprehensive screening should be encouraged for high-risk individuals, as the significant overlap in vascular comorbidities highlights the importance of early detection of coronary, cerebrovascular, and peripheral artery diseases together. Moreover, public health interventions promoting lifestyle modification, including dietary improvement, regular physical activity, and smoking cessation, remain essential strategies to reduce the shared burden of atherosclerosis within the Egyptian population.

Conflict of interest: None to be declared.

Funding: None to be declared.

References

1. Di Cesare M, Perel P, Taylor S, Kabudula C, Bixby H, Gaziano TA, et al. The Heart of the World. *Glob Heart*. 2024;19:11.
2. Abd El-Gawad HN, El-Baz M, El-Deeb AF, El-Shenawy SI. Risk factors profile of coronary artery disease among medical students at Al-Azhar University, Cairo. *Journal of Recent Advances in Medicine*. 2022;3:119-30.
3. Adhikary D, Barman S, Ranjan R, Stone H. A Systematic Review of Major Cardiovascular Risk

Factors: A Growing Global Health Concern. *Cureus*. 2022;14:e30119.

4. Arafa S, Abdelsalam S, El-Gilany AH, Mosaad YM, Abdel-Ghaffar A. Endothelial nitric oxide synthase Glu 298 Asp (G894T) and Apolipoprotein E gene polymorphism as possible risk factors for coronary heart disease among Egyptians. *Egypt Heart J*. 2018;70:393-401.

5. Assem M, Mousa S, Abdelhamid A, Amin S, Elsamadony A, El-Sebaee E, et al. The risk of micro and macrovascular disease in Egyptian patients with diabetes and peripheral arterial disease. *J Community Hosp Intern Med Perspect*. 2021;11:216-9.

6. Azab M, Al-Shudifat AE, Johannessen A, Al-Shdaifat A, Agraib LM, Tayyem RF. Are Risk Factors for Coronary Artery Disease Different in Persons With and Without Obesity? *Metab Syndr Relat Disord*. 2018;16:440-5.

7. Rippe JM. Lifestyle Strategies for Risk Factor Reduction, Prevention, and Treatment of Cardiovascular Disease. *Am J Lifestyle Med*. 2019;13:204-12.

8. Teramoto T, Sasaki J, Ishibashi S, Birou S, Daida H, Dohi S, et al. Executive summary of the Japan Atherosclerosis Society (JAS) guidelines for the diagnosis and prevention of atherosclerotic cardiovascular diseases in Japan -2012 version. *J Atheroscler Thromb*. 2013;20:517-23.

9. Teramoto T, Sasaki J, Ueshima H, Egusa G, Kinoshita M, Shimamoto K, et al. Diagnostic criteria for dyslipidemia. Executive summary of Japan Atherosclerosis Society (JAS) guideline for diagnosis and prevention of atherosclerotic cardiovascular diseases for Japanese. *J Atheroscler Thromb*. 2007;14:155-8.

10. Jasrai L. An Introduction to Data Analysis Using IBM SPSS: Routledge India; 2024.

11. Turk-Adawi K, Sarrafzadegan N, Fadhil I, Taubert K, Sadeghi M, Wenger NK, et al. Cardiovascular disease in the Eastern Mediterranean region: epidemiology and risk factor burden. *Nat Rev Cardiol*. 2018;15:106-19.

12. Reda A, Bendary A, Elbahry A, Farag E, Mostafa T, Khamis H, et al. Prevalence of atherosclerosis risk factors in Egyptian patients with acute coronary syndrome: final data of the nationwide cross-sectional 'CardioRisk' project. *J Public Health Afr*. 2020;11:1368.

13. Khalfallah M, Habib M, Kishk AM, Saeed B, Hemdan S, Eissa A, et al. Atherosclerotic Cardiovascular Diseases in Middle Delta of Egypt: A Systematic Analysis of Risk Factors Associated with the Rising Burden of the Disease. *Glob Heart*. 2025;20:11.

14. Elkersh AA, Samir A, Reda A. The risk factor profile in Egyptian patients with acute coronary syndrome: an observational study. *Menoufia Medical Journal*. 2022;35:359-63.

15. Reda A, Ashraf M, Soliman M, Ragy H, El Kersh A, Abdou W, et al. The pattern of risk-factor profile in Egyptian patients with acute coronary syndrome: phase II of the Egyptian cross-sectional CardioRisk project. *Cardiovasc J Afr.* 2019;30:87-94.
16. El-Moselhy E, Mohammed A, Abd El-Aziz A, Sadek I, Hagrass S, Farag G. Coronary artery disease among elderly Egyptian patients: I. socio-demographic, lifestyle, psychosocial, medical, and biochemical risk factors. *Am J Gerontol Geriatr.* 2018;1:1006.
17. Khedr E, Tony AA, Habeel M, Nasreldein A. Frequency and risk factors of carotid artery disease among ischemic stroke patients in the south Egypt: hospital-based study. *The Egyptian Journal of Neurology, Psychiatry and Neurosurgery.* 2021;57:128.
18. Kamal D, Abd ElMoteleb AM, Samir R, Saeed M. Epicardial fat thickness can predict severity and multivessel distribution in Egyptian patients with atherosclerotic coronary artery stenosis. *Egypt Heart J.* 2018;70:323-7.
19. M El Sayed A, M Ibrahim W, M Elbadawy A, H Mohammed S, O Abd ElMoneim R. Impact of Sex and Gender Differences on Cardiovascular Risk Factors and Cardiovascular Complications in Diabetic Patients in Benha City, Egypt: A Hospital-Based Cross-Sectional Study. *Benha Medical Journal.* 2024;41:130-40.
20. Elhfnawy A, Galeel AA, Abdelkhalek H. Carotid atherosclerosis in a sample of Egyptian patients with or without ischemic vascular events. *The Egyptian Journal of Neurology, Psychiatry and Neurosurgery.* 2023;59:144.
21. Francis V, Elshamy S, Mahgoub A, Shelbaya K, Zarif B. Distribution of cardiovascular risk severity among Egyptian patients with established cardiovascular diseases. *European Journal of Preventive Cardiology.* 2024;31:zwae175. 298.
22. Hussein A, Mahmoud SED, Awad MS, Mahmoud HEM. Assessment of Cardiovascular Risk Factors in Patients with Type 2 Diabetes in Upper Egypt Villages. *Diabetes Metab Syndr Obes.* 2020;13:4737-46.
23. Ellaïen A, Reda A, Elkersh A. The Pattern of Risk Factor Profile and Dutch Lipid Clinic Network Criteria of Familial Hypercholesterolemia in Egyptian Patients with Premature Coronary Heart Disease. *Cardiology and Cardiovascular Research.* 2020;4:203-9.
24. Sobhy M, El Etriby A, El Nashar A, Wajih S, Horack M, Brudi P, et al. Prevalence of lipid abnormalities and cholesterol target value attainment in Egyptian patients presenting with an acute coronary syndrome. *Egypt Heart J.* 2018;70:129-34.
25. Laimoud M, Faris F, Elghawaby H. Intravascular evaluation of coronary atherosclerotic lesions among Egyptian diabetic patients with acute coronary syndromes. *Egypt Heart J.* 2018;70:237-41.
26. Ibrahim RR, Ghoneim KT, Ghoneim SM, Farag ESM, Salama AE. Non-Traditional Predictors for Occurrence and Severity of Premature Atherosclerosis in Acute Coronary Syndrome. *The Egyptian Journal of Hospital Medicine.* 2022;88:2948-54.
27. El Faramawy A, Youssef G, El Aroussy W, El Remisy D, El Deeb H, Abdel Aal A, et al. Registry of the Egyptian specialized hypertension clinics: patient risk profiles and geographical differences. *J Hum Hypertens.* 2020;34:520-7.
28. Shehab A, Bakir S, Sabbour H, Elnour AA, Mahmeed WA, Salam AM, et al. Prevalence of Cardiovascular Risk Factors and 10-Years Risk for Coronary Heart Disease in the United Arab Emirates. *Curr Diabetes Rev.* 2023;19:e210422203892.
29. El-Amin H, Shehata KMA, Ammar AA, Ali WA, Abdel Aal AM, Abokresha MM, et al. Predictors of subclinical atherosclerotic cardiovascular disease in inflammatory bowel disease patients. *The Egyptian Journal of Hospital Medicine.* 2022;87:1666-73.

To cite this article: Alaa M. Shahin , Mohamed S. Saleh, Ayman M. Elbadawy, Walaa M. Ibrahim, Rasha O. Abdelmoniem. Prevalence of Risk Factors in Egyptian Patients with Atherosclerotic Cardiovascular Diseases in Benha City: Hospital Based Cross Sectional Study. *BMFJ XXX*, DOI: 10.21608/bmfj.2025.407136.2572.