Analysis of selected risk factors associated with cardiovascular diseases : A study on patients living in Tébessa region (Algeria)

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ABSTRACT

Cardiovascular diseases (CVDs) represent a major public health concern due to their increasing prevalence and significant impact on health outcomes. To prevent the development of such complications, we focused on studying modifiable and non-modifiable factors involved in their progression. This study was conducted on 73 patients diagnosed with various cardiovascular pathologies, living in the Tébessa region (Algeria). Data are collected through a structured questionnaire designed to document behavioral and physiological risk factors. Among these participants, 25 provided blood samples, which are analyzed to perform a biochemical profile and measure the concentrations of reduced glutathione (GSH) and malondialdehyde (MDA). Our findings reveal that men are at a higher risk of CVDs compared to women. Hypertension, diabetes, smoking, and obesity are more prevalent, accompanied by statistically significant disturbances in biochemical metabolism and redox status. In conclusion, our results indicate that cardiovascular diseases are associated with both biochemical and behavioral disruptions. Early screening, regular health monitoring through blood tests, weight management, physical activity, and smoking cessation are critical recommendations to prevent cardiovascular risk.

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1. INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of global morbidity and mortality, representing a critical public health challenge. In 2017, an estimated 17.7 million of the 55 million global deaths were attributed to CVDs, with projections suggesting this figure could rise to 23.6 million by 2030 [1]. This alarming trend underscores the urgent need for effective strategies to mitigate the growing burden of these diseases [2].

CVDs encompass a spectrum of conditions, including coronary artery disease, stroke, hypertension, and heart failure, each with distinct pathophysiological mechanisms. Despite this diversity, these diseases share common risk factors that necessitate a comprehensive approach to prevention and management [3]. These risk factors are typically categorized as non-modifiable (e.g., age, sex, race, and genetic predisposition) and modifiable (e.g., diabetes, hypertension, dyslipidemia, smoking, physical inactivity, poor dietary habits, and obesity). Behavioral, environmental, and social determinants further contribute to the complexity of CVD etiology, amplifying the challenge of risk reduction [4].

Among the modifiable factors, elevated blood pressure imposes significant strain on the cardiovascular system, increasing the risk of myocardial infarction and stroke [5]. Similarly, dyslipidemia, marked by elevated low-density lipoprotein (LDL) cholesterol and reduced high-density lipoprotein (HDL) cholesterol levels, accelerates atherosclerosis, a key driver of CVD progression [6]. Lifestyle behaviors, such as smoking, unhealthy diets, and physical inactivity, exacerbate these risks by promoting oxidative stress, inflammation, and endothelial dysfunction [7].

This study aims to analyze the demographic, behavioral, and biochemical characteristics of patients diagnosed with cardiovascular diseases in Tébessa, Algeria. By identifying prevalent patterns and associations, the research seeks to enhance understanding of these factors and their implications for targeted prevention and management strategies.

2. RESEARCH METHOD

2.1. Population

Our study was conducted in two parts. The first part involved 73 patients (both sexes included) suffering from various cardiovascular pathologies. This phase was carried out in the biochemistry laboratories of Bouguera Boulaares Hospital (Bakaria – Tébessa) and Mohammed Chbouki Hospital (Chéria – Tébessa). During a training internship and with the participants' consent, a questionnaire was distributed to gather information on the following factors : age, sex, weight, height, health issues, tobacco and alcohol consumption, and frequency of

physical activity. Exclusion criteria included individuals who declined to participate, young subjects, and those with mental retardation.

2.2. Biochemical and redox status analysis

Out of the 73 patients, 25 volunteers agreed to provide blood samples for the analysis of the following biological parameters : glucose [8], urea [9], creatinine [10], uric acid [11], triglycerides [12], cholesterol [13], LDH and HDL-cholesterol [14,15], GSH [16], and MDA [17]. For accurate interpretation of the obtained results, we recruited a healthy population of 25 volunteers to serve as the control group in our study.

2.3. Statistical Analysis

Descriptive data were presented using Excel (2016). Logistic regression analysis was performed using Python/Statsmodels with a significance threshold of $p<0.05$. The results of the biochemical analyses and redox status were expressed as mean ± standard deviation (M ± SD). These means were compared to the control values using a Student's t-test, performed with the MINITAB Software (ver. 13.31).

3. RESULTS AND ANALYSIS

3.1. Demographic and clinical Characteristics

The sample consisted of 73 patients with various cardiovascular diseases, of which 30 (41.1%) were female and 43 (58.9%) were male (Table 1). The patients were distributed across three age groups: 5 (6.8%) patients aged 20 to 40 years, 25 (34.2%) aged 41 to 60 years, and 43 (58.9%) aged over 60 years. Regarding lifestyle factors, 32 (43.8%) were non-smokers and 41 (56.2%) were smokers, while 50 (68.5%) were non-drinkers, and 23 (31.5%) consumed alcohol. In terms of physical activity, 28 (38.4%) patients were physically active, and 45 (61.6%) were inactive

Table 1. Distribution of patients based on demographic and clinical characteristics

Sex	Women		Men			
N	30		43			
%	41.1%		58.9%			
Age groups	20-40	41-60		> 60		
N	5	25		43		
%	6.8	34.2		58.9		
Tabacco consumption	Non smoker		Smokers			
N	32		56.2			
%	43.8		41			
Alcohol consumption	Non-drinkers		Drinkers			
N	50		23			
%	68.5		31.5			
Physical activity	Inactive		Active			
N	45		28			
%	61.6		38.4			
Existing health conditions						
	HTN	Diabetes	Anemia	Stroke	CHD	DLD	HT
N	31	22	4	2	2	10	2
%	42.5	30.1	5.5	2.7	2.7	13.7	2.7
	anorexia	underweight	Normal wt.	Over wt.	MO	SO	MBO
BMI	<16	16.5-18.5	18.5-25	25-30	30-35	35-40	> 40
N	0	0	35	26	10	1	1
%	0	0	47.9	35.6	13.7	1.4	1.4

HTN: Hypertension; CHD: Coronary Heart Disease; DLD: Dyslipidemia;
Ht: Hyperthyroidism; MO: Moderate obesity; SO: Sever obesity and MBO: Morbid obesity

The demographic and clinical characteristics of the study population provide a foundational understanding of the factors contributing to cardiovascular disease (CVD) risk. The predominance of male patients (58.9%) is consistent with studies suggesting a higher prevalence of CVD among men, potentially due to hormonal, genetic, and behavioral differences, such as higher rates of smoking and alcohol consumption [18]. The age distribution reveals that a majority of patients (58.9%) are over 60 years old, underscoring the well-established relationship between aging and cardiovascular risk. Age-related changes, such as arterial stiffening and endothelial dysfunction, have been extensively documented as critical contributors to CVD progression [19]. Patients lifestyle habits are among further highlight modifiable risk factors. Smoking prevalence (56.2%) is significantly high, emphasizing its role as a major preventable cause of CVD through mechanisms like oxidative stress and inflammation [20]. Combustion of tobacco products generates two primary forms of smoke: mainstream and sidestream. Mainstream smoke is inhaled and subsequently exhaled by the smoker, whereas sidestream smoke, emitted from the burning tip of a cigarette, is even more hazardous due to its higher concentration of toxic constituents [21]. Among the over 7,000 chemicals identified in cigarette smoke, numerous compounds are implicated in the pathophysiology of cardiovascular diseases (CVD) [22]. Toxic agents such as carbon monoxide, polycyclic aromatic hydrocarbons, nicotine, and heavy metals, along with their

oxides, exert detrimental effects on the vascular endothelium, blood lipids, and coagulation pathways [22]. These disruptions contribute to the development of atherosclerosis and significantly increase the risk of adverse cardiovascular events, including myocardial infarction, stroke, and aortic dissection [23]. Conversely, alcohol consumption was reported in 31.5% of patients, a finding that warrants nuanced interpretation. While excessive alcohol is detrimental, moderate consumption has been linked to a paradoxical protective effect in certain populations [24]. Physical inactivity was prevalent in 61.6% of the patients, reflecting a sedentary lifestyle that significantly exacerbates cardiovascular risk by contributing to obesity, insulin resistance, and hypertension. Encouragingly, evidence suggests that even modest increases in physical activity can lead to substantial reductions in CVD risk [25]. The BMI distribution highlights critical issues related to weight management. A significant proportion of patients (50.7%) were overweight or moderately obese, while 15.1% were classified as severely or morbidly obese. Obesity is a well-documented driver of hypertension, dyslipidemia, and systemic inflammation, which collectively increase CVD risk [26]. Notably, the absence of underweight individuals in the cohort reflects the nutritional transition seen in many regions, characterized by a shift toward high-calorie, low-nutrient diets.

3.2. Logistic regression model

A logistic regression analysis was performed to examine the predictors of hypertension (HTN) among the study population. The results are shown in Table 2.

Table 2: Logistic regression results for predicting hypertnsion (HTN)

Variable	Coefficient	p-value	Odds Ratio
Sex (male)	0.5	0.10	1.65
Age (per year)	0.02	0.001	1.02
Smoking (yes)	1.5	0.03	4.48
Alcohol (yes)	-0.8	0.15	0.45
Physical Activity	-0.3	0.05	0.74
BMI (per unit)	0.2	0.01	1.22

The logistic regression results are presented below:

Sex: Sex did not show a statistically significant relationship with the probability of having HTN (coefficient = 0.5, p = 0.10), suggesting that sex is not a major predictor of HTN in this population.

Age: Age showed a significant association with the likelihood of having HTN (coefficient = 0.02, p = 0.001). For each additional year of age, the probability of having HTN increased by 2% in log-odds terms. This indicates that older patients have a higher risk of HTN.

Smoking: Smoking was identified as a significant risk factor for HTN (coefficient = 1.5, p = 0.03). Smokers were about 4.48 times more likely to have HTN compared to non-smokers.

Alcohol Consumption: Alcohol consumption did not show a significant relationship with HTN (coefficient = -0.8, p = 0.15). Although the negative coefficient suggests a potential reduction in risk, this association was not statistically significant.

Physical Activity: Physical activity showed a significant association with the probability of HTN (coefficient = -0.3, p = 0.05). Physically active patients had a reduced risk of HTN compared to inactive patients, although this effect was relatively modest.

BMI: BMI was also found to be a significant predictor for HTN (coefficient = 0.2, p = 0.01). Each unit increase in BMI was associated with a 20% increase in the probability of having HTN in log-odds terms, highlighting the significant impact of obesity on HTN risk.

The Forest Plot of the logistic regression results for hypertension (HTN) visualizes the odds ratios (OR) for each variable along with their 95% confidence intervals (CI). The red dashed line represents an odds ratio of 1, which would indicate no effect. Variables such as Sex, Smoking, and BMI show significant effects, as their confidence intervals do not cross 1. The Alcohol variable does not show a significant effect, as its confidence interval includes 1.

The logistic regression analysis underscores the role of modifiable and non-modifiable risk factors. Age emerged as a robust predictor, with a 2% increase in HTN risk per year of age, aligning with prior evidence that aging elevates vascular stiffness and blood pressure [27]. Smoking showed a strong association, with smokers being 4.48 times more likely to develop HTN, corroborating its known vasoconstrictive effects [20]. Conversely, alcohol consumption did not significantly affect HTN, reflecting heterogeneity in alcohol-related HTN mechanisms observed across populations [24]. Physical activity demonstrated a protective effect, reducing HTN risk by 26%, which aligns with studies emphasizing regular exercise in mitigating cardiovascular risks [25]. BMI, another significant predictor, reinforces the critical role of obesity management in HTN prevention, as every unit increase in BMI heightened the odds of HTN by 20% [26].

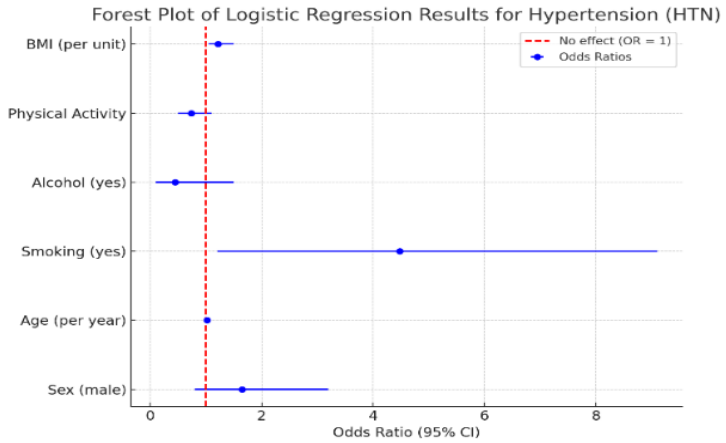

Figure 1. Forest plot of logistic regression results for hypertension (HTN)

3.3. Biochemical study results and redox status

The study findings reveal significant differences between patients with cardiovascular diseases (CVDs) and the control group. A marked increase in fasting blood glucose levels was observed, alongside notable elevations in renal function markers, including urea, uric acid, and creatinine. Lipid profile analysis demonstrated a significant rise in lipid levels among CVD patients. Additionally, the data indicated a significant reduction in GSH levels, accompanied by a highly significant increase in plasma MDA levels in the patient group.

Table 3: Variation of biochemical parameters and oxidative stress biomarkers

Parameters	Control group n=25	CVDs group n=25
Blood glucose (g/L)	1.09 ± 0.43	1.43 ± 0.74 *
Urea (g/L)	0.25 ± 0.04	1.17 ± 0.6 *
Uric acid (mg/L)	56.6 ± 19.3	68.9 ± 32.5 *
Creatinine (Mg/L)	10.71 ± 7.98	15.5 ± 10.6 *
Total Cholesterol (g/L)	1.43 ± 0.328	2.06 ± 0.458***
Triglycerides (g/L)	0.82 ± 0.280	2.05 ± 0.300***
HDL-Cholesterol (g/L)	0.52 ± 0.042	0.44 ± 0.065*
LDL-Cholesterol (g/L)	0.95 ± 0.320	1.64 ± 0.395***
GSH (nmol.10^6/mL)	31.04 ± 5.26	21.78 ± 6.55 *
MDA (nmol/mL)	10.59±6.01	24.63±6.31 **

The study revealed significant disparities in biochemical parameters between patients with cardiovascular disease (CVD) and control subjects. Elevated levels of blood glucose, renal markers, and lipid profiles reflect pronounced metabolic dysregulation and an increased risk of cardiovascular events, consistent with findings reported by [28]. The observed rise in low-density lipoprotein (LDL) cholesterol and triglycerides, coupled with a reduction in high-density lipoprotein (HDL) cholesterol, underscores the critical role of dyslipidemia in atherosclerosis development, aligning with evidence presented by [29]. These findings are further supported by the work of [30], which also documented similar patterns of dyslipidemia in CVD patients.

Numerous studies have consistently shown that diabetes significantly elevates the risk of CVD in diabetic populations compared to non-diabetic individuals. Type 2 diabetes, in particular, disrupts lipid metabolism, leading to atherogenic dyslipidemia characterized by alterations in lipids and lipoproteins [31]. Atherogenic dyslipidemia not only promotes the oxidative modification of lipids, especially LDL, but also facilitates the oxidative modification of proteins. This, in turn, triggers both localized and systemic inflammatory responses, further exacerbating atherosclerotic processes [32].

The findings related to oxidative stress are particularly significant. The observed reduction in glutathione (GSH) levels, coupled with elevated malondialdehyde (MDA) levels in patients with cardiovascular disease (CVD), indicates a disrupted redox balance. This imbalance exacerbates vascular inflammation and endothelial dysfunction, consistent with the mechanisms described by Vekic et al. [33].

Under normal physiological conditions, reactive oxygen species (ROS) production is tightly regulated and balanced by detoxification mechanisms, serving essential roles in cellular signaling and function. However, in pathological states such as atherosclerosis or hypertension, ROS production surpasses the capacity of endogenous antioxidant defenses, leading to oxidative damage and cellular death. At the cardiovascular level, oxidative stress plays a pivotal role in the pathophysiology of conditions such as myocardial infarction, ischemia/reperfusion injury, and heart failure.

These findings align with existing evidence that links oxidative stress markers to both the initiation and progression of cardiovascular diseases, underscoring the critical role of redox homeostasis in maintaining vascular health [33].

4. CONCLUSION

The study highlights that several demographic and lifestyle factors are significantly linked to the risk of hypertension among patients with cardiovascular diseases. Specifically, age, smoking, and body mass index (BMI) emerged as independent risk factors for hypertension in this population. Interestingly, alcohol consumption did not demonstrate a significant association with hypertension, suggesting it may not play a major role as a risk factor in this specific cohort. While physical activity exhibited a protective effect against hypertension, the observed impact was modest, warranting further research to clarify its magnitude and implications.

These findings underscore the necessity for targeted public health strategies that prioritize smoking cessation, promotion of physical activity, and effective obesity management. Additionally, regular screening for comorbid conditions, such as diabetes and hypertension, should be emphasized to mitigate cumulative cardiovascular risks. Tailored health education campaigns focusing on modifiable risk factors are essential to reduce the disease burden in similar populations.

While the study provides valuable insights, its cross-sectional design limits causal inference. Future longitudinal studies should explore the temporal relationships between these factors and cardiovascular outcomes. Additionally, investigating the role of dietary antioxidants in modulating oxidative stress biomarkers could offer novel therapeutic avenues.

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