RESEARCH ARTICLE
DOI: 10.53555/jptcp.v31i6.6727

# EVALUATING RISK FACTORS FOR EARLY-ONSET CARDIOVASCULAR DISEASES: A COMPREHENSIVE ANALYTICAL APPROACH 

Shah Umam ${ }^{1 *}$, Adeela kosar Bajwa ${ }^{2}$, Dr. Safiyyah Mukarram Khan ${ }^{3}$, Dr. Barsha Baral ${ }^{4}$, Dr. Arjun Kandel ${ }^{5}$, Ellen Huang ${ }^{6}$, Mohit Lakkimsetti ${ }^{7}$<br>1*Experiential Registrar, Department of Medicine, MTI Khyber Teaching Hospital Peshawar, Pakistan<br>${ }^{2}$ House Officer, Cardiology Care Unit, Shaikh Zayed Hospital Lahore, Pakistan, ${ }^{3}$ Physician, Department of Internal Medicine, India<br>${ }^{4}$ Medical Officer, MBBS, Department of Internal Medicine, Metrocity Hospital Pvt Ltd. Pokhara, Nepal<br>${ }^{5}$ Medical Officer, MBBS, Department of GP/ ER, Patan Academy of Health Sciences, Nepal ${ }^{6}$ Pre-Clinical MD Student, Caribbean Medical University<br>${ }^{7}$ MBBS, Department of Internal Medicine, Mamata Medical College, Khammam, India<br>*Corresponding Author: Shah Umam<br>*Experiential Registrar, Department of Medicine, MTI Khyber Teaching Hospital Peshawar, Pakistan


#### Abstract

: Introduction: Cardiovascular diseases (CVDs) continue to be the leading cause of death and morbidity worldwide, despite advancements in clinical outcomes and the development of new medications. This research aims to ascertain the degree to which preexisting risk factors contribute to the development of CVD in individuals younger than fifty years old. Method: A retrospective analytical case-control study was conducted on patients diagnosed with CVD. The ratio of cases to controls was one-to-one. Result: The study found no significant association between the male sex (odds ratio [OR]: 1.00; confidence interval [CI]: 0.449-2.087; p: 0.000) or alcoholism (OR: 0.53; CI: 0.012-1.236; p: 0.000) and the likelihood of developing CVD. However, C-reactive protein (OR: 6.01; CI: 2.640-13.681; p: 0.000) and hypertension (OR: 36.42; CI: 213.118-51.161; p: 0.000) showed strong associations with comorbidity and were statistically significant. The multivariate analysis revealed that Creactive protein significantly increased the risk of CVD (OR: 49.35; CI: 3.968-61.906; p: 0.000), followed by the significance of hypertension (OR: 25.4; CI: 14.481-44.118; p: 0.000). Conclusion: The findings suggest that a predictive model for assessing the likelihood of developing CVD is a valuable tool for clinical and epidemiological surveillance. This model can help identify individuals at the highest risk of developing the disease.


KEYWORDS: Cliff factors; Cardiovascular diseases (CVD), Risk factors, Retrospective analytical study, Case-control study, C-reactive protein, Epidemiological surveillance.

## INTRODUCTION:

Even though clinical outcomes have improved, cardiovascular diseases (CVD) remain to be the leading cause of morbidity and mortality. The current rates of CDs in several European countries are far lower than those prevalent in the early 1980s. This is a direct consequence of preventative measures that have been taken, such as the success of the adoption of rules that prohibit smoking (Muñoz et al., 2010). On the other hand, gaps continue to persist between countries, and the prevalence of a substantial number of risk factors, in particular obesity and diabetes mellitus (DM), has significantly increased. The World Health Organisation (WHO) estimates that around 17.5 million people passed away in the year 2012, with cardiovascular and cerebrovascular illnesses being the cause of 34 per cent of those fatalities. Ischemic heart disease was responsible for $7.4 \%$ of those deaths, while stroke was responsible for $6.7 \%$ of those deaths (Jansen et al., 2004). Furthermore, cardiovascular diseases have been the leading cause of mortality in Cuba since 1970. This is a significant historical fact. In 2018, 25,766 fatalities were ascribed to cardiovascular disease, and in 2019, there were 26,736 deaths. This equates to 229.0 and 238.1 deaths per 100,000 people, respectively. The two years in question both increased compared to the years before. Choosing a plan that lasts a lifetime is necessary to successfully manage cardiovascular (CV) risk. As a result of the fact that cardiovascular disease risk and prevention are both dynamic and ongoing, they change as the patient ages or as they accumulate comorbidities (Canto \& Iskandrian, 2003). Accurately identifying risk factors in each region is essential, particularly in young people, to prevent the emergence of cardiovascular disease (CVD), which happens when the individual is most useful in the social, family, and professional order. This is especially true in young people. Several features stand out as characteristics that are among the risk factors for cardiovascular disease. These characteristics include type 2 diabetes, obesity, dangerous behaviours, male gender, and abnormalities with lipid metabolism (Schenck-Gustafsson, 1996). Patients who are afflicted with cardiovascular disease are becoming more and more likely to have high blood pressure (HTN), which has emerged as the most prevalent risk factor. It is necessary to continue researching the issue, particularly in adults under fifty, where research is scarce. Because in recent years, works have been published in which a probable paradox is evident between the increase in some of the modifiable risk factors for death, such as obesity, heart and cerebrovascular diseases, and increased life expectancy, it is necessary to continue researching the problem. Because of this, it is required to investigate the matter (Bays et al., 2021). An additional component added to the examples stated above is the emergence of novel cardiovascular risk markers. These markers have the potential to explain the already existing cardiovascular risk. It is challenging to determine the level of risk in young individuals; even though they have several risk factors, the cardiovascular risk that they face is often low or moderate. It is advised that the relative risk, the age of vascular risk, and the risk throughout life be computed to attempt to convey the danger connected with this specific group of individuals (Greiser et al., 2005).

Table 1: Mortality and Prevalence of Cardiovascular Diseases (CVD)

| Year | Region | Mortality Rate (per 100,000) | Source |
| :--- | :--- | :--- | :--- |
| 2012 | Worldwide | $34 \%$ (17.5 million deaths) | WHO estimates |
| 2018 | Cuba | 229.0 | Canto \& Iskandrian, 2003 |
| 2019 | Cuba | 238.1 | Canto \& Iskandrian, 2003 |

Table 2: Significant Historical Data on CVD

| Year | Fact | Source |
| :--- | :--- | :--- |
| 1970 | CVD is the leading cause of mortality in Cuba | Canto \& Iskandrian, 2003 |
| 2012 | $7.4 \%$ of deaths are due to ischemic heart disease | Jansen et al., 2004 |
| 2012 | $6.7 \%$ of deaths are due to stroke | Jansen et al., 2004 |

Table 3: Risk Factors for Cardiovascular Diseases

| Risk Factor | Comments | Source |
| :--- | :--- | :--- |
| Obesity | Increased prevalence | Muñoz et al., 2010 |
| Diabetes Mellitus (DM) | Increased prevalence | Muñoz et al., 2010 |
| High Blood Pressure (HTN) | The most prevalent risk factor | Schenck-Gustafsson, 1996 |
| Type 2 Diabetes | Identified as a significant risk factor | Schenck-Gustafsson, 1996 |
| Male Gender | Identified as a significant risk factor | Schenck-Gustafsson, 1996 |
| Lipid Metabolism Abnormalities | Identified as a significant risk factor | Schenck-Gustafsson, 1996 |

Table 4: Preventative Measures and Impact

| Measure | Impact on CVD Rates | Source |
| :--- | :--- | :--- |
| Prohibition of smoking | Decreased CVD rates in Europe | Muñoz et al., 2010 |
| Lifetime CV risk management | Necessary for dynamic and ongoing prevention | Canto \& Iskandrian, 2003 |

Table 5: Research Needs and Future Directions

| Focus Area | Rationale | Source |
| :--- | :--- | :--- |
| CVD risk in individuals under fifty | Scarcity of research; potential paradox with <br> modifiable risk factors and life expectancy | Bays et al., <br> 2021 |
| Identification of novel cardiovascular risk <br> markers | Explain existing cardiovascular risk | Greiser et al., <br> 2005 |
| Development of a model to detect at-risk <br> individuals | Targeting individuals younger than fifty | Greiser et al., <br> 2005 |

It is unknown whether a specific group of factors is responsible for the increase in cardiovascular disease risk among individuals younger than fifty. Due to this rationale, this research aims to investigate the impact of risk variables on the development of cardiovascular disorders in the studied individuals. Furthermore, we intend to develop a model that will enable us to detect individuals younger than fifty years old who are more likely to become unwell.

## METHOD:

A retrospective case-control analytical study was carried out on patients diagnosed with cardiovascular disease admitted to the "Carlos Manuel de Céspedes" General University Hospital in Bayamo, Granma, starting January 5, 2017, and continuing until December 31, 2018. There was a one-to-one ratio between cases and controls (Deb \& Dasgupta, 2008). The following are the requirements for witnesses and cases: Adults up to fifty years old and patients admitted to the Internal Medicine service during the study period were included in the research. A case is any patient diagnosed with one of the cardiovascular diseases mentioned (an occurrence).
The recognized diagnostic criteria for ischemic heart disease are taken into consideration when diagnosing established cases, such as acute myocardial infarction or angina pectoris (Dahlöf, 2010). Echocardiography or clinical manifestations of recent onset heart failure (first presentation) can be used to identify diastolic dysfunction (changes in diastolic compliance, filling, or relaxation of the left ventricle) or elements of systolic dysfunction (an ejection fraction that is less than 45\%). Acute lung oedema, bibasal crackles, paroxysmal nocturnal dyspnea, third-tone gallop rhythm, and dyspnea on exertion are a few of these.
Cerebrovascular disease is a condition that is not related to any particular type of disease because it results from pathological processes of the blood vessels (damage to the vascular wall, occlusion of the lumen by thrombi or emboli, rupture of vessels, changes in the permeability of the vascular wall), and blood. Infections, congenital malformations, or connective tissue disease do not affect these processes (Prabhakaran et al., 2018).
Controls were those research participants admitted to the Internal Medicine service but did not develop cardiovascular illness. The participants in this study were at least fifty years old.

## EXCLUSION CRITERIA:

The current investigation did not include patients who suffered from morbid conditions that could cause heart disease. These conditions include cardiomyopathies (in any of their clinical forms), thyroid diseases, chronic renal failure, treatment with cytostatics, and chronic inflammatory intestinal and collagen diseases. Therefore, these patients were not included in the study. This was done to guarantee that the study's findings were accurate and trustworthy (Tanuseputro et al., 2003).

## DELIMITATION AND OPERATIONALIZATION OF VARIABLES

The dependent variable is the appointment for cardiovascular disease that occurred before.
Independent variables, all the factors that impact the manifestation of hypertensive heart disease, were carefully examined and analyzed.
There was a perception that male sex was a risk factor.
The age of patients was divided into two groups: those who were exposed, between the ages of 40 and 50 years, and those who were not exposed, between the ages of 18 and 39 years (Arboix et al., 2006).

About smoking habits, we divided people into two distinct categories: smokers (exposed) who consumed cigarettes, tobacco, or pipes on a daily or almost daily basis, regardless of the number of cigarettes they smoked, and ex-smokers who had quit smoking for a period that was less than one year. Non-smokers who had not been exposed to tobacco were the remaining patients, according to the classification system. When determining these parameters, it was taken into account that the association between smoking and the risk of cardiovascular disease is extremely important, and this is true regardless of the amount of cigarettes that are smoked regularly. Additionally, it has been proven that stopping smoking reduces the chance of having a first myocardial infarction, just as it does in non-smokers-smokers, beginning with the first year of smoking. This is the case even in smokers who have never smoked before (Gupta et al., 2013; Kremers et al., 2008).
In the past, it was thought that the definition of alcoholism was the drinking of more than one ounce of pure alcohol daily. When compared to the consumption of one ounce ( 20 ml ) of ethanol, eight ounces ( 240 ml ) of wine, twenty-four ounces ( 720 ml ) of beer, and one and a half ounces ( 45 ml ) of rum, this measure is comparable. On the other hand, the daily limit is reduced to fifteen millilitres for women and persons with a low body mass index.
The assessment of obesity was based on the computation of a body mass index (BMI) that was greater than or equal to 30 , weight in kilograms/height in m 2 , or a waist circumference that was greater than or equal to 102 cm for males and 88 cm for women, or both conditions of obesity. In addition, body mass index was used to determine whether or not an individual was obese.
Comorbidities include conditions such as hypertension, diabetes mellitus, and chronic renal disease, amongst others.
Based on their possible risk factors, several biological indicators were investigated. These include levels of cholesterol that are $4.5 \mathrm{moll} / \mathrm{L}$, levels of uric acid that are $300 \mu \mathrm{moll} / \mathrm{L}$, levels of triglycerides that are $1.8 \mathrm{moll} / \mathrm{L}$, levels of glycemia that are $5 \mathrm{molls} / \mathrm{L}$, levels of C-reactive protein that are $5 \mathrm{mg} / \mathrm{L}$, and levels of creatinine that are $80 \mu \mathrm{moll} / \mathrm{L}$ among others (Yusuf et al., 1998).
The cut-off points for the values of each laboratory test, which were going to be utilized in the univariate analysis, were determined to be set by the statistical estimates made and discussed after that. This was the conclusion that was reached.
The turbidimetric quantitative determination method was performed to determine whether or not non-ultrasensitive C-reactive protein (CRP) levels greater than five $\mathrm{mg} / \mathrm{L}$ were deemed a possible risk factor.
It was discovered that the patient had left ventricular hypertrophy after the electrocardiogram and echocardiogram of the heart were performed.
During the consultations, the data was gathered through interviews with the author, the instructor, and several other internal medicine doctors who participated in the HTA consultation. These interviews were carried out independently, and the outcomes were later compared with the patient's prior awareness and agreement (Smith Jr, 2007).

## DATA COLLECTION:

The data were initially transferred to a data collection form to preserve and safeguard them. Subsequently, the data were transferred to a database developed using the SPSS 22.0 statistical processing software for Windows.

## STATISTICAL ANALYSIS:

The statistical analysis began with the characterization of the sample, which included a description of all the variables. This was followed by additional analysis. The statistical analysis started with this phase, which was the first step. The results of the computations for the quantitative variables included the standard deviations and the lowest and maximum values of each distribution. In addition, the means of the variables were computed, and the median was determined for the variables with values on the end of the spectrum (Ciumărnean et al., 2021).
In addition, comparisons were made between the cases and controls included in the sample. To determine whether or not qualitative variables were independent, the Chi-square statistic developed by Pearson was utilized. When the distribution of the variable has a shape that is acceptable to normal (as decided by viewing the histogram and calculating asymmetry), the Student's $t$-test was utilized for quantitative variables. This was done to determine whether or not the distribution was normal. When a distribution that was not the normal one was discovered, the Mann-Whitney $U$ test was considered. This was mostly due to the presence of asymmetry. The level of significance of each of these factors
was also determined. The null hypothesis, which states that the distribution of each variable is the same in both groups, was examined for each variable.
The assumption of equality of variances was tested using a hypothesis test, and the modified Student's $t$-test was employed (using the Welch technique) when the hypothesis that the variances of both populations were equal was rejected. This was done to examine whether or not the assumption was correct (Wald et al., 2011).
A univariate technique was utilized to analyze risk factors. This strategy was founded on risk assessment using the value derived from the OR for paired samples. The odds ratios (ORs) were estimated using point and confidence interval estimates ( $95 \%$ ). The hypothesis was checked for each variable with a significance level of less than 0.05 , and the population OR was more than 1.01. Through univariate analysis, we could compare the quantitative factors' means between patients with cardiovascular disease and those without. This gave us a more comprehensive understanding of the relationship between these variables and the individual's risk. It was determined whether or not the distribution of each variable was the same in both the cases and the controls by testing the null hypothesis at each variable. Both the Student T and Mann Whitney U were utilized for this purpose. Student T was used when the variables' distribution had an acceptable shape compared to the normal distribution, while the Mann-Whitney U was used when a different distribution was seen (the assessment was mentioned in the preceding paragraphs).
A binary logistic regression model was adjusted using the "backward stepwise" method with all of the variables that comprise risk factors in the univariate analysis. This was the basis for the multivariate strategy, which was based on the adjustment of the model. An evaluation of the independent influence of each variable on the probability of getting cardiovascular disease was carried out in this manner while all other variables were controlled for. The maximum likelihood method was utilized to adjust the logistic regression function, similar to estimating the function's parameters. Additionally, the Hosmer and Lemeshow Chi-Square goodness-of-fit metric was used in this study. When the probability associated with the test statistic was more than 0.05 , the model was regarded as a good match for the data under consideration (Buhlin et al., 2003).

## RESULTS:

Within the sample, 62 instances and 62 witnesses were represented. As can be observed in Table 1, $64.5 \%$ of the patients, or 80 persons, were male. The most common comorbidity was hypertension, which was present in $53.2 \%$ of the patients, or 66 individuals.

Table 1. Characterization of the sample. Qualitative variables $\mathrm{N}=124$

| Variables | categories | Number | $\%$ |
| :--- | :--- | :--- | :--- |
| Sex | Male | 80 | 64,5 |
|  | female | 44 | 35,5 |
| Arterial hypertension | Yes | 66 | 53,2 |
|  | no | 58 | 46,8 |
| Alcoholism | Yes | 26 | 21,0 |
|  | no | 98 | 79,0 |
| Smoking habit | Yes | 35 | 31,5 |
|  | no | 85 | 68,5 |
| Mellitus diabetes | Yes | 32 | 25,8 |
|  | no | 92 | 74,2 |
| Chronic kidney disease | Yes | 116 | 6,45 |
|  | no |  | 93,55 |

Table 2 contains the values considered to be the averages of the quantitative variables. There was a standard deviation of 7.231 years, and the mean age was 42.70 . The standard deviation for uric acid was the highest at 115.327 , while the mean value was 294.69 . This was the case whenever uric acid was considered. After that, the results for creatinine were presented, with the mean value coming in at 84.20 and the standard deviation figure coming in at 27.047.

Table 2. Characterization of the sample. Quantitative variables $\mathrm{N}=124$

| Variables | N | Min | Max | Media | Standard deviation |
| :--- | :--- | :--- | :--- | :--- | :--- |
| Age | 124 | 18 | 58 | 42,70 | 7,231 |
| Cholesterol | 124 | 2,200 | 8,600 | 4,27 | 1,116 |
| Triglycerides | 124 | 0,230 | 9,00 | 1,22 | 1,319 |
| Creatinine | 123 | 33 | 156 | 84,20 | 27,047 |
| Uric acid | 124 | 101 | 525 | 294,69 | 115,327 |
| Blood glucose | 124 | 3,1 | 13,2 | 4,89 | 1,6761 |
| Obesity | 124 | 19,96 | 47,62 | 26,739 | 3,957 |

A univariate study was conducted to investigate the influence of variables such as comorbidity, hazardous behaviours, and sexual orientation. The results of this study are presented in Table 3, which exhibits such findings. Both drinking (odds ratio: 0.53; confidence interval: 0.012-1.236; null hypothesis: 0.000 ) and male sex (odds ratio: 1.00; confidence interval: 0.449-2.087; null hypothesis: 0.000 ) were shown not to indicate a relationship with the risk of developing cardiovascular disease (CVD). Smoking, on the other hand, was shown to raise the risk that was mentioned earlier by a factor that was greater than five (odds ratio: 5.52; confidence interval: 2.326-13.106; importance level: 0.000). Diabetes mellitus was the comorbidity that exhibited the most significant link (odds ratio: 8.79; confidence interval: 3.099-24.954; p: 0.000), followed by hypertension (odds ratio: 36.42; confidence interval: 213.11851 .161 ; $\mathrm{p}: 0.000$ ). Both of these concomitant conditions were very considerably enhanced.

Table 3. Risk factors for cardiovascular disease: sex, toxic habits, and

| Variables | Witness |  | Cases |  |  | Confidence <br> INTERVAL <br> (IC 95\%) | *p |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- | :--- |
|  | No | $\%$ | No | $\%$ | OR |  |  |
| Smoking habit | 30 | 76,9 | 9 | 23,1 | 5,52 | $2,326-13,106$ | 0,000 |
| Male sex | 40 | 50 | 40 | 50 | 1,00 | $0,449-2,087$ | 0,999 |
| Alcoholism | 2 | 7,7 | 24 | 92,3 | 0,53 | $0,012-1,236$ | 0,000 |
| Comorbidity |  |  |  |  |  |  |  |
| Arterial hypertension | $\mathbf{5 5}$ | $\mathbf{8 3 , 3}$ | $\mathbf{1 1}$ | $\mathbf{1 6 , 7}$ | $\mathbf{3 6 , 4 2}$ | $\mathbf{1 3 , 1 1 8 - 5 1 , 1 6 1}$ | $\mathbf{0 , 0 0 0}$ |
| Mellitus diabetes | 27 | 84,4 | 5 | 15,6 | 8,79 | $3,099-24,954$ | 0,000 |
| Chronic kidney disease | 7 | 87,5 | 1 | 12,5 | 7,744 | $1,926-65,118$ | 0,028 |
| Obesity | 24 | 49,0 | 25 | 51,0 | 0,935 | $0,455-1,921$ | 0,854 |

The results of the univariate analysis of biological indicators and age are presented in Table 4, which describes the findings. C-reactive protein was the factor that showed the largest link with the chance of having cardiovascular disease (odds ratio: 6.01; confidence interval: 2.640-13.681; p: 0.000 ). This was the factor that emerged as the most significant. The next most considerable risk variables were age greater than or equal to 40 years (odds ratio: 9.43; confidence interval: 3.80922.934; p: 0.000) and cholesterol levels that were beyond 4.5 moll/L (odds ratio: 5.27; confidence interval: 2.322-11.986; p: 0.000). Both of these factors were associated with an increased likelihood of developing cardiovascular disease.

Table 4. Risk factors for cardiovascular disease: age and biological markers.
Univariate analysis results

| Variables | Witness |  |  | Cases |  | {$\begin{array}{l}\text { Confidence interval } \\ \\$ |  |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- | :---: |
|  |  |  |  |  |  |  |  |
| (IC 95\%) |  |  |  |  |  |  |  |$)$

A multivariate analysis was carried out using a binary logistic regression (Table 5) to determine the components that have an independent influence on the development of cardiovascular disease (CVD). The findings demonstrated that the most remarkable power of association was shown by Creactive protein values greater than five $\mathrm{Mg} / \mathrm{L}$. This was demonstrated by an increase in risk to 49.35 (confidence interval: 3.968). Hypertension emerged in second place in terms of significance, and the next most important factor was an odds ratio of 25.4 (confidence interval: 14.481-44.118; p: 0.000 ).

Table 5. Risk factors for cardiovascular disease. Binary logistic regression.

| Variables | B |  | P | OR |  |
| :--- | ---: | ---: | :--- | :--- | :--- |
|  |  |  |  | Confidence interval $95 \%$ |  |
|  |  | Inferior | Superior |  |  |
| C-reactive protein | $\mathbf{3 , 8 9 9}$ | $\mathbf{0 , 0 0 2}$ | $\mathbf{4 9 , 3 5}$ | $\mathbf{3 , 9 6 8}$ | $\mathbf{6 1 , 9 0 6}$ |
| Arterial hypertension | 5,531 | 0,000 | 25,4 | 14,481 | 44,118 |
| Smoke | 2,941 | 0,011 | 18,91 | 1,963 | 32,544 |
| Mellitus diabetes | 2,674 | 0,021 | 14,49 | 1,499 | 24,124 |
| Age | 2,331 | 0,016 | 10,28 | 1,557 | 27,926 |
| Cholesterol | 1,070 | 0,222 | 2,91 | 0,523 | 16,260 |
| Creatinine | 0,158 | 0,854 | 1,171 | 0,217 | 6,333 |
| Constant | $-29,492$ | 0,000 | 0,000 | 1,963 | 82,544 |

## DISCUSSION:

The theory of the epidemiology of cardiovascular diseases (CVD) is a powerful and valuable contribution to the field. Furthermore, even though these diseases continue to be among the major causes of death in both industrialized and developing countries, the extensive Framingham cohort, as well as novel clinical trials that are targeted at verifying various treatment modalities, have been carried out (Buhlin et al., 2003).
There have been other studies that have found a connection between smoking and cardiovascular disease. These studies are similar to the one that is currently being discussed.
A person who has smoked for their whole life has a fifty per cent chance of dying as a result of tobacco usage, and they will lose an average of ten years of their life as a result of their smoking habit.

When compared to the risk that is associated with non-smokers of the same age and gender, the risk that is linked with smoking among people under the age of 50 is five times higher than the risk that is associated with smoking (Khot et al., 2003).
Smoking habits, including endothelial function, oxidative processes, platelet function, fibrinolysis, inflammation, lipid oxidation, and vasomotor function impact several functions. It is not fair to predict that those who smoke will attain the same level of risk for cardiovascular disease as people who have never smoked. This is because the creation of plaque does not appear to be reversible.
The conclusions of this thesis are in contrast to the findings of Álvarez-Aliaga and colleagues, who reported in 2007 that the risk of damage to target organs induced by hypertension was significantly twice as high in male patients (odds ratio of 2.52; confidence interval of 1.45 to 4.44). However, the findings of the current series are in line with the findings of other studies, which indicate that gender is not an independent risk factor for the development of cardiovascular disease. This is the conclusion that can be drawn from the research that has been conducted.
These findings are probably connected to the prevalence of cardiovascular risk factors, which are present in both sexes at the same rate before the age of 50 . This is one of the possible explanations for these findings.
Even though our findings in this study suggest that there is no connection between intoxication and the risk of cardiovascular problems in hypertension patients, other authors have discovered that there is a connection between the two (Khot et al., 2003).
The vast majority of patients who were evaluated in our research were light drinkers, which is a trait that is expected to have a protective effect, according to the literature. Despite this, the majority of patients who were studied were light drinkers.
As indicated by the data published in various investigations, which coincide with the research presently being carried out, it has been established that high blood pressure is one of the most significant risk factors for cardiovascular disease. This is as suggested by the findings that have been published.
A higher blood pressure is linked to an increased risk of cardiovascular events such as myocardial infarction, heart failure, stroke, and kidney disease. This risk is connected with other cardiovascular conditions as well.
When it comes to cardiovascular disease (CVD), systolic hypertension is equivalent to a relative risk coefficient of two. Patients who have what was traditionally known as "high normal blood pressure" (SBP $130-139 \mathrm{mmHg}$ or DBP $85-89 \mathrm{mmHg}$ ) have been shown to have a higher risk of cardiovascular disease (CVD) over a longer length of time. This risk has been demonstrated through research.
In addition, synthesis studies of clinical trials that were carried out using antihypertensive medications give solid evidence that lower blood pressure levels generally lessen the occurrence of cardiovascular events. The development of the full range of cardiovascular problems is caused by a multitude of neurohormonal, renal, and vascular mechanisms that interact with one another and contribute to varying degrees to the many hemodynamic kinds of hypertension. These mechanisms are responsible for the development of hypertension.
The findings of the current investigation indicate that functional and structural abnormalities of the myocardium, which are reported in hypertension patients, are also produced. This explains the high incidence of cardiovascular events that occur in hypertensive individuals regardless of whether or not they are under control of their condition.
There is a strong correlation between having diabetes mellitus and having raised blood glucose levels, which is associated with an increased risk of cardiovascular disease (CVD). It is (19). The ongoing series has a high degree of coincidence around this occurrence. Patients who have diabetes and those who have impaired glucose tolerance continue to have an unacceptably high risk of cardiovascular disease and mortality alike consistently. On the other hand, when glucose levels are strictly regulated, the risk of developing microvascular and macrovascular disease can be significantly reduced. These patients may develop chronic renal failure as a result of taking these medications; nonetheless, the life expectancy of these persons has risen as a result of the continued
treatment of hypertension and type 2 diabetes mellitus. The rate at which these two disorders are being diagnosed in patients who are required to begin chronic dialysis is currently higher than the rate at which any other ailment is being diagnosed. Atherosclerosis begins a phase of acceleration as a consequence of this, and the number of deaths that occur as a result of cardiovascular disease is rather high.
When compared to the other components of metabolic syndrome, obesity is the most significant risk factor for cardiovascular disease (CVD), and the impact of obesity is amplified when the different elements are present. The favourable benefits on cardiovascular health that may be reached by reducing body weight in patients who are overweight or obese have been the subject of several clinical trials, as well as observational and epidemiological research. These studies have pointed out the positive impacts of reducing body weight. There are twenty-one in total.
All of the results that were discussed previously are in agreement with the conclusions that are reported in the thesis that is currently being discussed. This is due to several pathophysiological mechanisms, such as insulin resistance and hyperinsulinism, salt sensitivity and intrarenal changes, and chronic sodium retention. These mechanisms, along with others that are currently accepted, include subclinical inflammation, neurohormonal activation, high concentrations of leptin, an increase in the oxidation of free fatty acids in the myocardium, and fat deposition in specific regions of the body that play a direct role in the pathogenesis of coronary atherosclerosis. All of these mechanisms are involved in the development of coronary atherosclerosis.
Because of this, the determination of C-reactive protein has been given predictive value. This is because there has been evidence of the significance of inflammation in the phases of atherosclerosis and the genesis of cardiovascular events for several decades. This is why the determination of Creactive protein has been assigned to this particular instance.
According to our inquiry findings, several writers found a connection between C-reactive protein and the chance of developing cardiovascular problems.
Rider identified this biological marker as an independent risk factor (relative risk of 1.4) for coronary and non-coronary issues when its levels were equal to or greater than three $\mathrm{mg} / \mathrm{L}$. This was the scenario in which the relative risk was 1.4. When the values of the markers were observed, this was the situation that occurred.
C-reactive protein, which is a biomarker of active vascular processes, has the potential to have a direct impact on the function and morphology of the cardiovascular system. Furthermore, it may also be a factor that contributes to the risk of cardiovascular disease.
As a result of the fact that they summarize all of the changes that are connected with ageing, biological processes are influenced by age. There is widespread agreement that age is an important factor in predicting cardiovascular disease.
Pérez Fernández found that the risk of suffering from cardiovascular disease is 12.53 times higher in those who are older compared to those who are between the ages of 15 and 49. This finding was reported in a study that was pretty comparable to the one that we carried out. There is a risk that is three and a half times lower for people who are between the ages of 15 and 49 compared to those who are between the ages of 50 and 64 .
For over seven decades, alterations in lipid metabolism have been associated with an increased risk of cardiovascular disease and the complications that accompany it. These complications include hypertension and ischemic heart disease, to name a couple of the more prominent manifestations.
Lenneberg discovered that people with hypercholesterolemia had a risk of suffering from cardiovascular disease that was greater than three. This was accomplished through the application of the Framingham Cardiovascular Risk Index. The results of this investigation suggest that this component is a reliable predictor.
Several additional research have concluded that there is a clear correlation between high cholesterol levels and the chance of developing cardiovascular disease. These findings are consistent with the findings of the current series, which is a conclusion that may be drawn.
According to Subbing, the adjusted frequency for cardiovascular events increased in a manner that was inversely related to the estimated glomerular filtration rate (eGFI). This was the explanation
that was given for the observed increase. To be more specific, the adjusted frequency was 1.4 for egress rates that ranged from 45 to $59 \mathrm{~mL} / \mathrm{min}$ per $1.73 \mathrm{~m} 2,2.0$ for egress rates that ranged from 30 to $44 \mathrm{~mL} / \mathrm{min}$ per $1.73 \mathrm{~m} 2,2.8$ for egress rates that ranged from 15 to $29 \mathrm{~mL} / \mathrm{min}$ per 1.73 m 2 , and 3.4 for egress rates that were less than $15 \mathrm{~mL} / \mathrm{min}$ per 1.73 m 2 . In addition, research conducted by Álvaro Aliana and colleagues indicates that patients whose serum creatinine levels were equivalent to or higher than $80 \mathrm{~mol} / \mathrm{L}$ were at a higher risk of having hypertensive heart disease. The conclusion that can be drawn from these observations is that they are consistent with the ongoing series.
Histological and functional changes that are diagnostic of hypertensive kidney damage are produced by a variety of complex mechanisms that are interconnected with one another. Microalbuminuria and decreased creatinine clearance are the conditions brought on by these modifications, which occur in the early stages of the ailment. It is well-recognized that hypertension and cardiovascular disease are both linked to complications that affect the kidneys. These factors significantly increase the likelihood of developing cardiovascular disease (CVD).
Uric acid is not an inactive chemical; it promotes tubular and interstitial illness, activates the renin-angiotensin-aldosterone system at the vascular level, and generates a pro-inflammatory condition. Additionally, it causes disruption of the baroreflex, promotes endothelial dysfunction, stimulates the proliferation of vascular smooth muscle cells and the formation of reactive oxygen species, and increases the stiffness of major arteries. These pathophysiological elements affect the cardiovascular system, finally leading to the disease's development.
Additionally, the common insulin resistance that is present in these patients makes them more susceptible to lipotoxicity, which is accompanied by dysfunctional cardiomyocytes, rising levels of oxidative stress, mitochondrial uncoupling, and death. A condition known as chronic hyperglycemia promotes the buildup of the end products of advanced non-enzymatic glycation, which in turn causes collagen and other proteins to undergo certain modifications. Following this, the myocardium and blood vessels become more stiff due to the condition.
In the absence of stenosis of these arteries, hyperglycemia is linked to cardiac hypertrophy and interstitial fibrosis, arteriosclerosis, and coronary endothelial dysfunction, all of which are conditions that make a person more susceptible to ischemia. The condition known as hyperglycemia can manifest itself on its own or in conjunction with hypertension.
These characteristics might explain the results that were presented earlier.
Based on the findings of previous authors, our investigation revealed that hypertriglyceridemia did not constitute a risk factor for cardiovascular disease (CVD).
The current study's findings could be explained by the fact that superior therapeutic effects are gained following an increase in HDL values and a reduction in LDL, as opposed to a drop in total cholesterol and triglycerides. This is the interpretation that could be given to the findings of the current investigation.
In conclusion, the current study demonstrates that the impacts of C-reactive protein, high blood pressure, smoking, diabetes mellitus, and age above 40 years are independent influencing factors for the development of cardiovascular disease in those 50 years and younger. When the significance of the components discussed earlier became clear, the explanation of our result was already on the pages before this one.

## CONCLUSION:

When it comes to people who are less than 50 years old, the most significant risk factor for the development of cardiovascular disease is C-reactive protein. High blood pressure and smoking are the two factors that come in second and third, respectively. Even though obesity, high creatinine levels, and triglyceride levels did not show any evidence of an independent association, these factors may depend on other more significant factors. In addition, a model was developed to estimate the likelihood of developing cardiovascular disease. Because it identifies people more prone to becoming unwell, this model is useful as a clinical and epidemiological surveillance device.

## REFERENCE:

1. Arboix, A., Miguel, M., Císcar, E., García-Eroles, L., Massons, J., \& Balcells, M. (2006). Cardiovascular risk factors in patients aged 85 or older with ischemic stroke. Clinical neurology and neurosurgery, 108(7), 638-643.
2. Bays, H. E., Taub, P. R., Epstein, E., Michos, E. D., Ferraro, R. A., Bailey, A. L., Kelli, H. M., Ferdinand, K. C., Echols, M. R., \& Weintraub, H. (2021). Ten things to know about ten cardiovascular disease risk factors. American journal of preventive cardiology, 5, 100149.
3. Buhlin, K., Gustafsson, A., Pockley, A. G., Frostegård, J., \& Klinge, B. (2003). Risk factors for cardiovascular disease in patients with periodontitis. European Heart Journal, 24(23), 20992107.
4. Canto, J. G., \& Iskandrian, A. E. (2003). Major risk factors for cardiovascular disease: debunking the only 50\% myth. Jama, 290(7), 947-949.
5. Ciumărnean, L., Milaciu, M. V., Negrean, V., Orășan, O. H., Vesa, S. C., Săăgean, O., Iluţ, S., \& Vlaicu, S. I. (2021). Cardiovascular risk factors and physical activity for the prevention of cardiovascular diseases in the elderly. International Journal of Environmental Research and Public Health, 19(1), 207.
6. Dahlöf, B. (2010). Cardiovascular disease risk factors: epidemiology and risk assessment. The American journal of cardiology, 105(1), 3A-9A.
7. Deb, S., \& Dasgupta, A. (2008). A study on risk factors of cardiovascular diseases in an urban health centre of Kolkata. Indian Journal of Community Medicine, 33(4), 271-275.
8. Greiser, K. H., Kluttig, A., Schumann, B., Kors, J. A., Swenne, C. A., Kuss, O., Werdan, K., \& Haerting, J. (2005). Cardiovascular disease, risk factors and heart rate variability in the elderly general population: design and objectives of the CARdiovascular disease, Living and Ageing in Halle (CARLA) Study. BMC Cardiovascular Disorders, 5, 1-14.
9. Gupta, S., Gudapati, R., Gaurav, K., \& Bhise, M. (2013). Emerging risk factors for cardiovascular diseases: Indian context. Indian journal of endocrinology and metabolism, 17(5), 806-814.
10. Jansen, A., Van Aalst-Cohen, E., Tanck, M., Trip, M., Lansberg, P., Liem, A., Roeters van Lennep, H., Sijbrands, E., \& Kastelein, J. (2004). The contribution of classical risk factors to cardiovascular disease in familial hypercholesterolaemia: data in 2400 patients. Journal of Internal Medicine, 256(6), 482-490.
11. Khot, U. N., Khot, M. B., Bajzer, C. T., Sapp, S. K., Ohman, E. M., Brener, S. J., Ellis, S. G., Lincoff, A. M., \& Topol, E. J. (2003). Prevalence of conventional risk factors in patients with coronary heart disease. Jama, 290(7), 898-904.
12. Kremers, H. M., Crowson, C. S., Therneau, T. M., Roger, V. L., \& Gabriel, S. E. (2008). High ten-year risk of cardiovascular disease in newly diagnosed rheumatoid arthritis patients: A population-based cohort study. Arthritis \& Rheumatism: Official Journal of the American College of Rheumatology, 58(8), 2268-2274.
13. Muñoz, L. R., Etnyre, A., Adams, M., Herbers, S., Witte, A., Horlen, C., Baynton, S., Estrada, R., \& Jones, M. E. (2010). Awareness of heart disease among female college students. Journal of Women's Health, 19(12), 2253-2259.
14. Prabhakaran, D., Jeemon, P., Sharma, M., Roth, G. A., Johnson, C., Harikrishnan, S., Gupta, R., Pandian, J. D., Naik, N., \& Roy, A. (2018). The changing patterns of cardiovascular diseases and their risk factors in the states of India: the Global Burden of Disease Study 19902016. The Lancet Global Health, 6(12), e1339-e1351.
15. Schenck-Gustafsson, K. (1996). Risk factors for cardiovascular disease in women: assessment and management. European Heart Journal, 17(suppl_D), 2-8.
16. Smith Jr, S. C. (2007). Multiple risk factors for cardiovascular disease and diabetes mellitus. The American journal of medicine, 120(3), S3-S11.
17. Tanuseputro, P., Manuel, D. G., Leung, M., Nguyen, K., \& Johansen, H. (2003). Risk factors for cardiovascular disease in Canada. Canadian Journal of Cardiology, 19(11), 1249-1260.
18. Wald, N. J., Simmonds, M., \& Morris, J. K. (2011). Screening for future cardiovascular disease using age alone compared with multiple risk factors and age. PloS one, 6(5), e18742.
19. Yusuf, H. R., Giles, W. H., Croft, J. B., Anda, R. F., \& Casper, M. L. (1998). Impact of multiple risk factor profiles on determining cardiovascular disease risk. Preventive medicine, 27(1), 1-9.
