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Assessment of Risk Factors for Coronary Artery Disease in Women Over 40 Years of Age

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Abstract: ***Background**: Coronary artery disease (CAD) remains a leading cause of morbidity and mortality in women globally, particularly post - menopause. Despite historical underrepresentation in cardiovascular research, emerging evidence highlights distinct gender - specific risk factors influencing the onset and progression of CAD in women. **Objective**: This study aimed to assess the risk factors associated with CAD in women aged 40 years and above, focusing on both traditional and sex - specific contributors, and to raise awareness for early detection and prevention. **Methods**: A retrospective analytical case - control study was conducted using medical records of 200 women diagnosed with CAD over a ten - year period. Risk factors assessed included diabetes, hypertension, stress, early menopause, smoking, lifestyle, and hereditary predisposition. Statistical analysis was performed to determine associations and odds ratios. **Results**: The study identified psychosocial stress (94.5%), hypertension (87.5%), and diabetes mellitus (81%) as the most significant risk factors. Early menopause (69%), sedentary lifestyle (70%), and hereditary factors (71%) also showed significant associations. Passive smoking, though present in 54% of participants, was not statistically significant. **Conclusion**: The findings underscore the multifactorial etiology of CAD in women, with a particularly high burden of psychosocial and metabolic risks. These results advocate for gender - specific screening protocols, preventive strategies, and heightened clinical awareness to reduce cardiovascular morbidity and mortality in women over 40 years of age.*

Keywords: Coronary artery disease (CAD), Women's cardiovascular health, Postmenopausal risk, Psychosocial stress, Hypertension, Diabetes mellitus, Early menopause, Lifestyle risk factors

1. Introduction

Cardiovascular disease (CVD), including coronary artery disease (CAD), is one among those leading etiologies of illness in both men and women globally. While traditionally considered a predominantly male condition, CVD in women presents unique challenges due to differences in symptomatology, risk factors, and disease progression. However, emerging evidences highlight significant gender - based differences in the risk profile, clinical presentation, and outcomes of coronary artery disease (CAD). While women generally exhibit a more favorable cardiovascular risk profile at younger ages, this advantage wanes with age, particularly after menopause. Among Indian women, with the changing life styles, specific risk factors like elevated triglycerides, low - density lipoprotein (LDL), lipoprotein (a) [Lp (a)], low high - density lipoprotein (HDL), and polycystic ovary syndrome (PCOS) have shown strong associations with CAD.[1]

Notably, despite a lower prevalence of traditional risk factors such as hypertension, smoking, and obesity compared to other populations, Indian men and women display higher rates of central obesity, glucose intolerance, and dyslipidemia. Urban populations in India, although exhibiting lower tobacco use, face significantly higher CAD rates—a phenomenon termed the "Tobacco Paradox"—emphasizing the detrimental impact of urbanization - related lifestyle factors.[2]

Furthermore, younger Indian patients often present with advanced and diffuse atherosclerotic disease, including premenopausal women with multi - vessel involvement. Global data, such as the INTERHEART study, have shown that women typically experience their first cardiac symptoms nearly a decade later than men. Nonetheless, their outcomes are often worse, due to delayed diagnosis, older age at presentation, and multiple coexisting conditions. Anatomical and physiological differences—such as smaller coronary vessels, denser plaques, and higher coronary artery calcium scores—further contribute to adverse clinical outcomes in women.[3]

The prognosis for women with coronary artery disease (CAD) varies based on age, the manner in which the disease presents, the accuracy of diagnosis, and the number of associated risk factors. In general, outcomes tend to be poorer in women. This is partly due to a historical lack of aggressive treatment compared to men, although this difference is decreasing significantly in present day medical practice.[2 6]

Given these complexities, this study aims to assess the risk factors for coronary artery disease (CAD) in women, focusing on identifying gender - specific factors that contribute to the development of CAD. The goal is to enhance understanding of these factors to improve prevention, early detection, and treatment strategies tailored to women.

2. Aim and Objectives of the Study

Aim:
The aim of this study is to assess the risk factors of coronary artery disease in women and to create awareness about various risk factors of coronary artery disease in women.

Objectives:
1) To evaluate the risk factors associated with Coronary artery disease.
2) To assess if Diabetes is associated with increased risk of Coronary artery disease.
3) To assess if Stress is associated with increased risk of Coronary artery disease.

4) To assess if Smoking is associated with increased risk of Coronary artery disease.
5) To assess if Early Menopause is associated with increased risk of Coronary artery disease.
6) To assess if Lifestyle is associated with increased risk of Coronary artery disease.
7) To assess if Hereditary Factors are associated with increased risk of Coronary artery disease.
8) To create awareness about various risk factors of Coronary artery disease in women.

Method of Collection of Data

This is a retrospective analytical type of case control study utilizing an observational approach based on hospital records of patients admitted for treatment of coronary artery disease.

Selection Criteria for Study Group

Inclusion criteria:

- Female patients of ≥ 40 age groups.
- Confirmed diagnosis of CAD.
- Complete medical records available including relevant co - morbidity and family history data.

Exclusion criteria:

- Incomplete or missing medical records.
- Patients with uncertain or non - CAD - related cause of death.

3. Methodology

This study is a retrospective analytical type of case control study conducted in a hospital - based setting. It involves the analysis of patient records of women ≥ 40 years who were diagnosed with coronary artery disease (CAD). Data was collected from hospital archives and electronic medical records spanning a ten - year period, from 2015 to 2024. Relevant records from past 10 years who were diagnosed with CAD and the co - morbidities was recorded, family history was also be retrieved. Data was compiled and analyzed using appropriate statistical software.

Risk Factors of Coronary Artery Disease and their Pathophysiology:

The risk profile for coronary artery disease (CAD) in women includes both traditional and sex - specific factors that contribute to its development and progression. Traditional risk factors such as hypertension, dyslipidemia, diabetes mellitus, obesity, and smoking remain significant contributors, with diabetes conferring a disproportionately higher risk in women compared to men (Maas & Appelman, 2010). Moreover, women often exhibit a clustering of risk factors, including metabolic syndrome and physical inactivity, especially post - menopause. Psychosocial factors—such as depression, anxiety, and chronic stress—also play a more prominent role in women and are associated with increased cardiovascular risk (Mehta et al., 2016). Additionally, sex - specific risk enhancers such as pregnancy - related complications (e. g., preeclampsia, gestational diabetes), early menopause, and polycystic ovary syndrome (PCOS) further elevate the risk of CAD in women (Sharma et al., 2020). These findings underscore the importance of adopting a tailored, sex - specific approach to cardiovascular risk assessment and prevention in women.[21, 22, 23]

Diabetes as a risk factor for CAD in women:

Pathophysiology:

Diabetes mellitus (DM), particularly type 2, is a major and disproportionately potent risk factor for coronary artery disease (CAD) in women. Epidemiological studies indicate that diabetic women have a significantly higher relative risk of CAD compared to diabetic men, and often suffer from worse outcomes following myocardial infarction (Maas & Appelman, 2010; Peters et al., 2014).[24] This elevated risk is attributed to a combination of pathophysiological mechanisms that are often more pronounced or differently manifested in women.

Hyperglycemia leads to endothelial dysfunction, increased oxidative stress, and low - grade chronic inflammation, all of which promote atherogenesis. Additionally, insulin resistance—a hallmark of type 2 DM—results in dyslipidemia characterized by elevated triglycerides, low HDL cholesterol, and small, dense LDL particles, which are highly atherogenic. Women with diabetes also exhibit a greater tendency toward visceral adiposity, compounding metabolic disturbances that exacerbate cardiovascular risk (Mehta et al., 2016).[25]

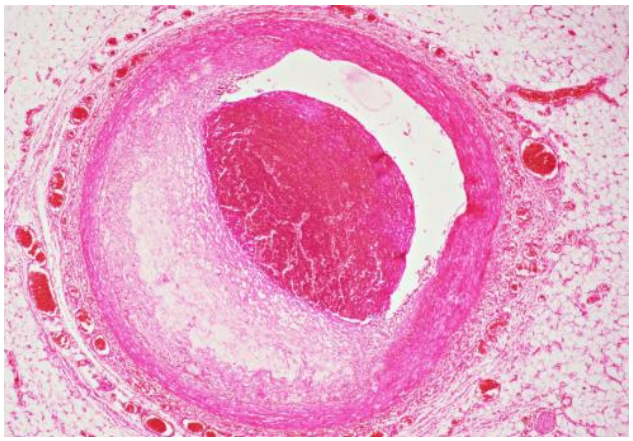
Furthermore, diabetic women are more likely to have coexisting hypertension, obesity, and metabolic syndrome, creating a synergistic effect that accelerates atherosclerotic plaque development and vascular damage. Microvascular dysfunction, more prevalent in women, is another critical component, contributing to ischemic symptoms even in the absence of obstructive coronary lesions. These complex pathophysiological interactions emphasize the need for aggressive cardiovascular risk management in diabetic women, including strict glycemic control and modification of coexisting risk factors.[26]

Hypertension as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:

Hypertension is a major modifiable risk factor for coronary artery disease (CAD) in women and contributes significantly to cardiovascular morbidity and mortality. The prevalence of hypertension increases with age in women, particularly after

menopause, when the loss of estrogen's vasoprotective effects exacerbates blood pressure dysregulation. The pathophysiological impact of hypertension on the cardiovascular system is multifactorial. Chronic elevation of blood pressure leads to endothelial dysfunction, characterized by impaired nitric oxide (NO) production, increased oxidative stress, and reduced vasodilatory capacity. These changes promote a pro - inflammatory and pro - thrombotic state within the arterial wall, setting the stage for atherogenesis (Reckelhoff, 2001).[27]



Sustained high arterial pressure causes mechanical injury to the endothelium, which in turn stimulates the migration and proliferation of vascular smooth muscle cells, enhances the deposition of lipids, and accelerates the formation of atherosclerotic plaques. In women, hormonal factors influence the renin - angiotensin - aldosterone system (RAAS), which regulates blood pressure and fluid balance.[28] Estrogen deficiency post - menopause up - regulates RAAS activity, leading to vasoconstriction, sodium retention, and increased vascular resistance. Additionally, hypertension is frequently associated with left ventricular hypertrophy and diastolic dysfunction, which further strain the myocardium and increase the risk of ischemic events. Importantly, hypertension in women may be under diagnosed or undertreated due to sex - related differences in symptom presentation and healthcare access, despite its clear association with increased CAD risk. Effective blood pressure management, particularly in midlife and postmenopausal women, is therefore a critical strategy in the prevention of CAD.[29]

Psychosocial Stress as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:
Psychosocial stress, including chronic emotional stress, depression, and anxiety, is a significant and often underappreciated risk factor for coronary artery disease (CAD), particularly in women. Women report higher levels of psychological stress than men and exhibit a stronger physiological response to such stressors, which contributes to their cardiovascular vulnerability (Vaccarino et al., 2014).[30] The pathophysiology linking stress to CAD involves complex neuroendocrine and autonomic dysregulation. Chronic stress activates the hypothalamic - pituitary - adrenal (HPA) axis and the sympathetic nervous system, leading to sustained elevations in cortisol and catecholamines. These hormonal changes contribute to endothelial dysfunction, increased arterial stiffness, and pro - inflammatory cytokine release, all of which accelerate atherosclerosis (Steptoe & Kivimäki, 2012). In addition, stress - induced autonomic imbalance promotes vasoconstriction, increased heart rate, and elevated blood pressure—factors that strain the cardiovascular system over time.[31]

Women are also more susceptible to stress - related cardiac conditions such as **Takotsubo cardiomyopathy**, a transient but severe form of left ventricular dysfunction often triggered by emotional or physical stress. Moreover, stress contributes to unhealthy behaviors—such as poor diet, physical inactivity, smoking, and nonadherence to medication—that indirectly increase CAD risk. Social and cultural roles, caregiving responsibilities, and socioeconomic pressures may further intensify chronic stress in women, compounding its physiological impact. The interplay of these biological and behavioral mechanisms underscores the critical importance of integrating psychosocial assessment and mental health support into cardiovascular risk management strategies for women.[32]

Passive Smoking as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:
Smoking is one of the most potent and modifiable risk factors for coronary artery disease (CAD) in both sexes, but its impact appears to be more pronounced in women. Studies have demonstrated that women who smoke have a significantly higher relative risk of developing CAD compared to men who smoke, with the risk being particularly elevated in younger women (Huxley & Woodward, 2011). The pathophysiology of smoking - induced CAD involves several interrelated mechanisms. Tobacco smoke contains thousands of harmful chemicals, including nicotine and carbon monoxide, which contribute to endothelial dysfunction—a critical early event in atherogenesis. Nicotine stimulates sympathetic nervous system activity, leading to increased heart rate, vasoconstriction, and elevated blood pressure, while carbon monoxide reduces oxygen delivery by binding to hemoglobin, exacerbating myocardial ischemia.[32]

Smoking promotes oxidative stress and systemic inflammation, increasing the levels of circulating inflammatory cytokines such as interleukin - 6 and C - reactive protein. These processes accelerate the development of atherosclerotic plaques and increase their vulnerability to rupture. In women, smoking also has unique interactions with sex hormones; it reduces circulating estrogen levels and may blunt the protective cardiovascular effects of endogenous and exogenous estrogen, especially in premenopausal women and those using oral contraceptives. This hormonal interaction likely contributes to the disproportionately higher cardiovascular risk observed in female smokers. Furthermore, smoking induces a prothrombotic state by increasing platelet aggregation and fibrinogen levels, which raises the risk of acute coronary events. Given these multifactorial effects, smoking cessation

is an essential component of CAD prevention strategies, particularly in women.[33]

Hormonal influence on Coronary artery disease in women:

Early Menopause as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:
Menopause is a significant sex - specific risk factor for coronary artery disease (CAD) in women, primarily due to the sharp decline in endogenous estrogen levels that occurs during this transition. Prior to menopause, women generally have a lower risk of CAD compared to men, a difference attributed in part to the protective vascular effects of estrogen. Estrogen modulates lipid metabolism, promotes vasodilation via nitric oxide synthesis, inhibits vascular smooth muscle cell proliferation, and exerts anti - inflammatory and antioxidant effects (Mendelsohn & Karas, 2005).[34] With the onset of menopause, these protective mechanisms diminish, resulting in an unfavorable shift in cardiovascular risk factors. Postmenopausal women often experience increased low - density lipoprotein (LDL) cholesterol, decreased high - density lipoprotein (HDL) cholesterol, insulin resistance, central adiposity, and elevated blood pressure, all of which contribute to accelerated atherosclerosis and endothelial dysfunction (Maas & Appelman, 2010).[35]

Furthermore, menopause is associated with enhanced sympathetic nervous system activity and a pro - inflammatory state, which can exacerbate vascular injury and plaque instability. The loss of estrogen's regulatory influence on vascular tone and lipid homeostasis also contributes to increased arterial stiffness and impaired coronary microvascular function, which are particularly relevant in women who may present with non - obstructive CAD. While hormone replacement therapy (HRT) was initially hypothesized to mitigate this risk, large clinical trials such as the Women's Health Initiative (WHI) have shown that HRT does not reduce cardiovascular events and may pose additional risks if initiated later in life. Thus, the menopausal transition marks a critical period for cardiovascular risk assessment and intervention in women.3[6]

Polycystic Ovary Syndrome (PCOS) as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:
Polycystic ovary syndrome (PCOS) is a common endocrine disorder that affects women of reproductive age and is increasingly recognized as a significant risk factor for coronary artery disease (CAD). Women with PCOS are at an elevated risk of developing CAD due to a combination of metabolic, hormonal, and inflammatory abnormalities that contribute to the acceleration of atherosclerosis. Pathophysiologically, PCOS is characterized by hyperandrogenism, insulin resistance, and chronic low - grade inflammation, all of which play crucial roles in the development of cardiovascular disease. Insulin resistance, a hallmark of PCOS, leads to hyperinsulinemia, which not only promotes the accumulation of visceral fat but also exacerbates dyslipidemia. This dyslipidemic state is marked by elevated levels of low - density lipoprotein (LDL) cholesterol, triglycerides, and a decrease in high - density lipoprotein (HDL) cholesterol, all of which are strongly associated with the development of atherosclerotic plaques (Moran et al., 2010). Additionally, insulin resistance in PCOS results in endothelial dysfunction, a precursor to the development of CAD, as it impairs the production of nitric oxide, leading to reduced vasodilation and increased vascular stiffness.

Women with PCOS also exhibit chronic inflammation, evidenced by elevated levels of pro - inflammatory cytokines such as C - reactive protein (CRP), interleukin - 6 (IL - 6), and tumor necrosis factor - alpha (TNF - α). These inflammatory markers promote endothelial injury and accelerate the formation of atherosclerotic plaques, increasing the risk of cardiovascular events. Moreover, the elevated androgen levels seen in PCOS contribute to the development of metabolic disturbances, including abdominal obesity, which further enhances the risk of CAD through its effects on lipid metabolism, blood pressure regulation, and insulin sensitivity. The combination of these factors—insulin resistance, dyslipidemia, inflammation, and visceral adiposity—creates a multifaceted pathophysiological environment that significantly raises the risk of CAD in women with PCOS. Although PCOS itself does not directly cause CAD, it significantly increases the risk for cardiovascular events later in life, making early detection, regular monitoring, and lifestyle intervention crucial for managing cardiovascular risk in these women.

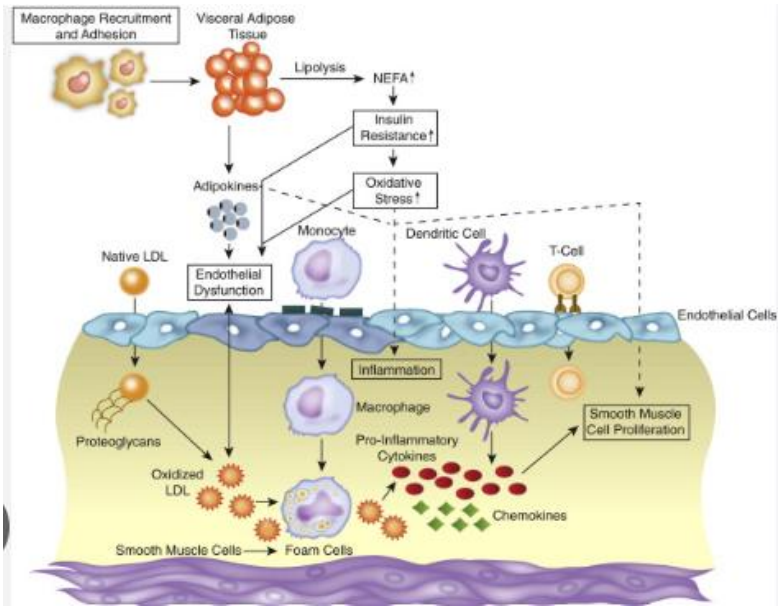
Lifestyle as a Risk Factor for Coronary Artery Disease in Women:

Pathophysiology:
Lifestyle behaviors play a critical role in the development and progression of coronary artery disease (CAD) in women, with sedentary behavior, poor diet, smoking, excessive alcohol consumption, and chronic stress constituting major modifiable risk factors. These behaviors often act synergistically, exacerbating metabolic and cardiovascular dysfunction. A sedentary lifestyle contributes to obesity, insulin resistance, and dyslipidemia, all of which are key drivers of atherosclerosis. Poor dietary habits—characterized by high intake of saturated fats, refined sugars, and sodium, and low intake of fruits, vegetables, and whole grains—promote systemic inflammation, endothelial dysfunction, and lipid abnormalities (Mozaffarian et al., 2016).[37] In women, these effects are often compounded by hormonal fluctuations, particularly during menopause, which amplify the metabolic impact of an unhealthy lifestyle.

Pathophysiologically, an adverse lifestyle fosters a pro - inflammatory, pro - thrombotic state marked by increased levels of C - reactive protein (CRP), interleukins, and tumor necrosis factor - alpha (TNF - α), all of which contribute to endothelial injury and plaque formation. Additionally, inactivity and poor nutrition can impair endothelial nitric oxide production, leading to reduced vasodilation and increased arterial stiffness. Visceral adiposity, more prevalent in women after menopause, further drives

metabolic syndrome—a constellation of risk factors that markedly heightens cardiovascular risk. Moreover, lifestyle factors influence autonomic balance, favoring sympathetic overactivity and increased heart rate and blood pressure, which impose additional strain on the cardiovascular system. Behavioral and psychosocial components, such as chronic stress and poor sleep, also activate the hypothalamic - pituitary - adrenal (HPA) axis and sympathetic nervous system, exacerbating inflammatory and metabolic disturbances. Given that many of these lifestyle factors are modifiable, early intervention and sustained behavioral change remain central to CAD prevention strategies in women.[38] obesity is one such example of a lifestyle risk factor.

Obesity is a well - established and potent risk factor for coronary artery disease (CAD) in women, contributing to the development and progression of atherosclerosis through multiple interrelated mechanisms. In women, obesity is particularly concerning as it often leads to a higher prevalence of other cardiovascular risk factors such as hypertension, dyslipidemia, type 2 diabetes, and metabolic syndrome. Pathophysiologically, excess adiposity, particularly visceral fat, leads to a state of chronic low - grade inflammation. Adipocytes release pro - inflammatory cytokines such as tumor necrosis factor - alpha (TNF - α), interleukin - 6 (IL - 6), and resistin, which promote endothelial dysfunction, increase the production of reactive oxygen species (ROS), and impair nitric oxide (NO) bioavailability, all of which contribute to atherosclerosis (Liu et al., 2016).[42] These inflammatory markers also promote the formation of atherogenic lipoproteins, such as small dense LDL particles, and contribute to an imbalance in the lipid profile, with elevated triglycerides and reduced HDL cholesterol.



Obesity also results in insulin resistance, a hallmark of metabolic syndrome, which exacerbates the risk for CAD. Insulin resistance leads to elevated circulating insulin levels, promoting endothelial dysfunction, smooth muscle cell proliferation, and an increased tendency for thrombosis. In women, the hormonal changes associated with obesity, including dysregulation of sex hormones such as estrogen, further influence cardiovascular risk.[43] Visceral fat also plays a crucial role in the development of hypertension by increasing circulating angiotensinogen levels, contributing to the activation of the renin - angiotensin - aldosterone system (RAAS), which raises blood pressure and exacerbates the strain on the cardiovascular system. Additionally, the mechanical burden placed on the heart in obese individuals leads to structural changes, including left ventricular hypertrophy and diastolic dysfunction, which further predispose to CAD. These multifaceted pathophysiological mechanisms highlight the importance of addressing obesity as a central target for CAD prevention and management in women.[44]

Hereditary Factors as a Risk for Coronary Artery Disease in Women:

Pathophysiology:

Genetic predisposition plays a substantial role in the development of coronary artery disease (CAD), and this influence is equally relevant in women, though often underrecognized. A positive family history of premature CAD—typically defined as myocardial infarction or sudden cardiac death before age 55 in a male relative or before age 65 in a female relative—is a well - established independent risk factor for cardiovascular events in women (Nasir et al., 2004).[39] The heritable component of CAD is thought to result from the interplay of multiple genetic variants that affect lipid metabolism, blood pressure regulation, endothelial function, and inflammatory responses. Specific gene loci, such as those on chromosome 9p21, have been consistently associated with increased CAD risk in genome - wide association studies (GWAS), and these genetic effects appear to be similar across sexes (Helgadottir et al., 2007).[40]

Pathophysiologically, inherited abnormalities can lead to early and more aggressive atherogenesis through mechanisms such as familial hypercholesterolemia (FH),

which results from mutations in genes involved in LDL receptor function (e. g., *LDLR*, *APOB*, or *PCSK9*), leading to markedly elevated LDL cholesterol levels from a young age. Women with FH may experience delayed diagnosis due to lower early - life risk perceptions, yet they remain at significantly elevated risk for early CAD. Other heritable conditions—such as hypertension, type 2 diabetes, and thrombophilic disorders—also have genetic components and contribute cumulatively to CAD pathogenesis. Inflammation - related genetic polymorphisms (e. g., in the *IL - 6* or *CRP* genes) may further predispose women to endothelial dysfunction and plaque instability. Importantly, the impact of genetic predisposition can be amplified or mitigated by environmental and lifestyle factors, making early screening and personalized prevention strategies vital, especially for women with a strong family history of cardiovascular disease.[41]

4. Results

Using Sampling method 200 case sheets of women with CAD were obtained. The age was distributed as follows.

Table 1: The age distribution of participants is detailed below

Age (in years)	No of patients	%
41 to 50	19	9.5
51 to 60	39	19.5
61 to 70	67	33.5
71 to 80	32	16
81 to 90	29	14.5
>90	14	7

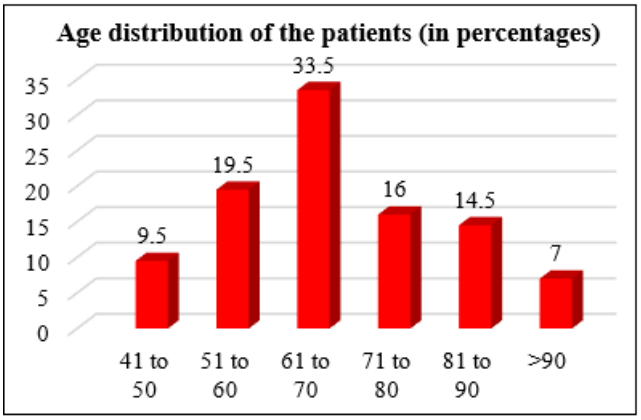


Figure 1: The age distribution of participants is detailed below

1) Diabetes as a risk factor

Table 2: Prevalence and association of Diabetes with CAD

Diabetes	No of patients	%
Present	162	81
Absent	38	13
Total	200	

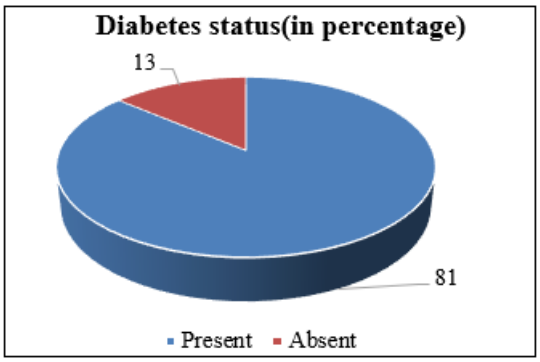


Figure 2: Pie chart showing diabetic vs non - diabetic distribution

Of the total 200 patients taken for the study 81% of them had diabetes and 19% of them did not had diabetes. Using the chi square test it was observed that diabetic is one of the risk factors for CAD in women. The result was found to be significant. ($p<0.05$), with a risk of 81% and odds ratio of 5.

2) Hypertension as a risk factor

Table 3: Prevalence and association of Hypertension with CAD

Hypertension	No of patients	%
Present	175	87, 5
Absent	25	12.5
Total	200	

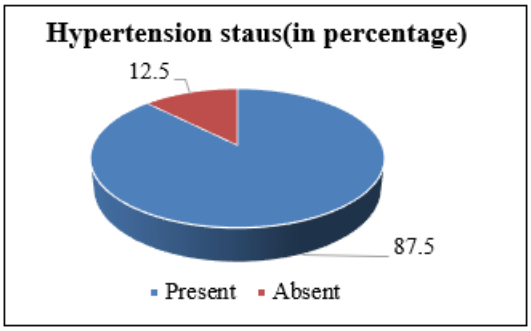


Figure 3: Pie chart of hypertensive vs normotensive participants

Data Analysis revealed that Hypertension is one of the risk factors for CAD in women, totally 200 records were analyzed and 87.5% of the patients had hypertension and the difference was significant ($p<0.05$) with a risk of 88%, with odds ratio of 7.

3) Stress as a risk factor

Table 4: Prevalence and association of Stress with CAD

Stress	No of patients	%
Present	189	94.5
absent	25	5.5
Total	200	

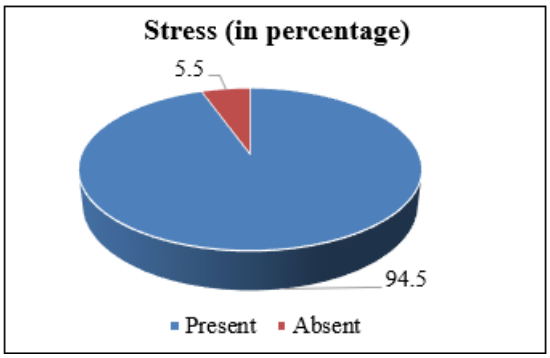


Figure 4: Pie chart of participants with stress vs without stress

Totally 200 records were analyzed and result was found to be significant ($p<0.05$) showing stress is a risk factor in CAD in females.94.5% of ladies had stress and only 5.5% of the records showed no stress. With a risk of 95% with odds ratio of 7.6.

4) Early menopause as a risk factor

Table 5: Prevalence and association of Early Menopause with CAD

Early menopause	No of patients	%
Present	137	69
11	43	31
Total	200	

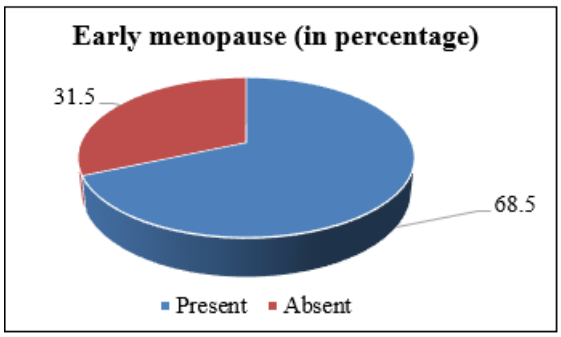


Figure 5: Pie Chart of Early Menopause vs. Normal/Late Menopause

Study revealed Early menopause is one of the risk factors for women who had CAD, about 137 (69%) of females had early menopause and the result was found to be significant using chi square test ($p<0.05$). with a risk of 69% with odds ratio of 3.

5) Passive smoking as a risk factor

Table 6: Prevalence and association of Passive Smoking with CAD

Smoking	No of patients	%
Present	108	54
Absent	92	46
Total	200	

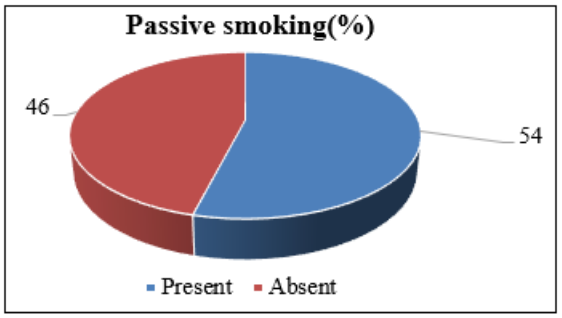


Figure 6: Pie chart of Passive Smoking vs. Passive Non - Smoking Participants

Passive smoking was not found to be significant in our study with a risk 54% of the patients have passive smoking but remaining did not. And the result was not significant ($p>0.05$).

6) Lifestyle as a risk factor

Table 7: Prevalence and association of Sedentary Lifestyle with CAD

Lifestyle	No of patients	%
sedentary	140	70
active	60	30
Total	200	

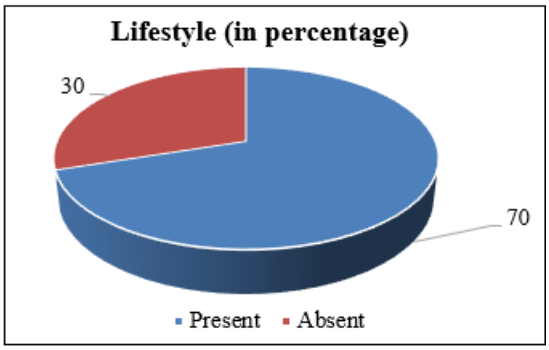


Figure 7: Pie chart of Sedentary vs. Active Lifestyle Participants

Only few ladies were found to be active in their lifestyle (30%) where as maximum no of ladies (70%) was found to be following sedentary lifestyle, and the result found to be significant with sedentary lifestyle is also one of the risk factors for CAD in women ($p<0.05$) with a risk of 70% with odds ratio of 2.3.

7) Hereditary as a risk factor

Table 8: Prevalence and association of Hereditary History with CAD

Hereditary factors	No of patients	%
Present	142	71
Absent	58	29
Total	200	

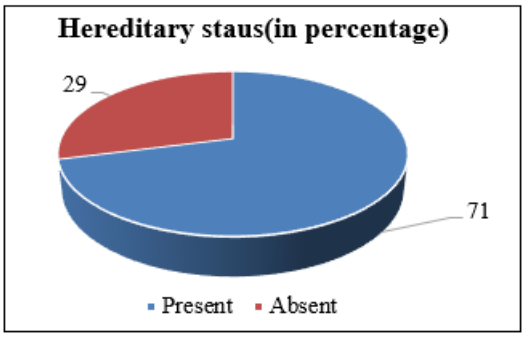


Figure 8: Pie chart of participants with vs without Hereditary History

Using the chi square test found that Hereditary was a risk factor for CAD in women, of the 200 records studied 142 patients (71%) had hereditary CAD remaining did not had, The result was found to be significant ($p<0.05$) with a risk of 71% with odds ratio of 2.4.

8) Risk factors

Table 9

Risk factors	(%)
Diabetes	81
Hypertension	88
Stress	95
Early menopause	69
Passive Smoking	54
Lifestyle	70
Hereditary	71

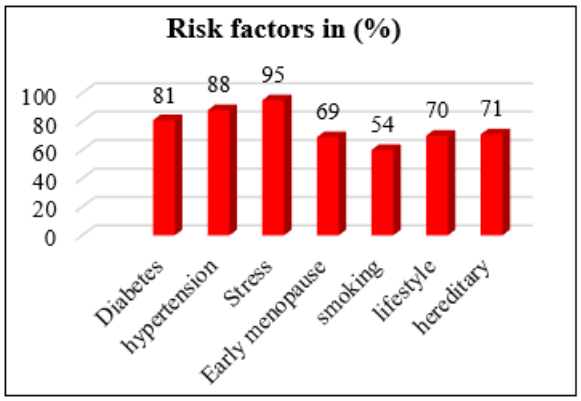


Figure 9

The present study found that the majority of women had multiple significant risk factors associated with CAD, with stress, hypertension, and diabetes being the most prevalent.

All major modifiable risk factors except passive smoking showed statistically significant associations. These findings underscore the cumulative impact of lifestyle, metabolic, and hereditary factors on coronary artery disease in women over 40. **(Table 9, Fig.9)**

5. Discussion

Cardiovascular disease remains the leading cause of mortality among women globally, and coronary artery disease (CAD) in particular accounts for a substantial share of these deaths. It has surpassed the combined mortality rates of all cancers in women over 40, a demographic especially vulnerable due to the convergence of traditional and sex - specific risk factors. Despite this, CAD in women remains underrecognized, underdiagnosed, and undertreated, largely due to persistent sex - based disparities in clinical research and medical practice.[13]

In the present study involving 200 women with documented CAD, we observed a high prevalence of modifiable risk factors. Diabetes mellitus was present in 81% of patients and showed a strong statistical association with CAD ($p < 0.05$, odds ratio = 5). This finding aligns with the INTERHEART study by Yusuf et al., which identified diabetes as one of the strongest independent predictors of acute myocardial infarction, particularly among women.[14] Peters et al. also reported that diabetes can amplify cardiovascular risk in women by fivefold, compared to a threefold increase in men.[15] This is partly due to the fact that diabetes neutralizes the cardioprotective effects of estrogen, contributing to endothelial dysfunction, lipid abnormalities, and vascular inflammation. Moreover, diabetic women tend to experience atypical symptoms, leading to delays in diagnosis and worse clinical outcomes.[16]

Hypertension was the most prevalent risk factor in our case control, seen in 87.5% of the women. The association was statistically significant ($p < 0.05$) with an odds ratio of 7. This reflects existing literature which positions hypertension as the most significant contributor to cardiovascular mortality in postmenopausal women. According to the Framingham Heart Study, elevated systolic blood pressure accounts for more than 60% of heart failure cases in women above 45.[17] The menopausal transition leads to a decline in estrogen, causing increased arterial stiffness, higher peripheral resistance, and subsequent hypertension, all of which exacerbate cardiovascular risk.[18]

Psychosocial stress was reported by 94.5% of women in our study—making it the most common risk factor. The association was highly significant ($p < 0.05$; odds ratio 7.6), reinforcing the critical role of mental health in cardiovascular disease. Vaccarino et al.[15] and Joynt et al.[20] have demonstrated that stress, anxiety, and depression independently increase the risk of myocardial infarction and poor post - MI outcomes, particularly in younger women. The American Heart Association's scientific statement on mental health and cardiovascular disease in women emphasized that psychological distress induces systemic inflammation, autonomic dysfunction, and hormonal imbalance, all of which contribute to atherogenesis. Stress also leads to poor health behaviors such as smoking, sedentary lifestyle, and poor diet, which further elevate CAD risk.[52]

Early menopause was present in 69% of our participants and showed a significant association with CAD ($p < 0.05$, odds ratio 3). The loss of estrogen before the age of 45 is a known risk enhancer for cardiovascular disease. Manson et al., [53] in the Nurses' Health Study, found that women with early menopause had a 50% higher risk of CAD. Estrogen exerts vasodilatory, anti - inflammatory, and lipid - regulating effects; its premature loss accelerates vascular aging and atherogenesis. El Khoudary et al.2[3] have further noted that menopause is associated with increased total cholesterol,

LDL, and central adiposity, all of which heighten cardiovascular risk.

Passive smoking, although present in 54% of the women, was not statistically significant in our analysis, suggesting that duration and intensity of exposure might play a role and merit further investigation.

A sedentary lifestyle was reported in 70% of patients and was found to be a statistically significant contributor to CAD ($p < 0.05$, odds ratio 2.3). This corroborates findings from the Harvard Nurses' Health Study[25] and the work of Mora et al., [54]both of which concluded that physical inactivity significantly increases the risk of CAD. Regular moderate - intensity exercise has been shown to improve insulin sensitivity, elevate HDL levels, reduce inflammation, and control blood pressure—all essential to cardiovascular health in women.

Hereditary factors were present in 71% of the study participants and significantly associated with CAD ($p < 0.05$, odds ratio 2.4). Family history plays a pivotal role in the early onset and severity of CAD. According to Khera et al., [55] individuals with high genetic risk who also have unhealthy lifestyles are significantly more likely to develop cardiovascular events, emphasizing the need for early screening and intervention in genetically predisposed women.

Age - wise, the burden of CAD peaked in the 61–70 age group, consistent with the well - documented increase in cardiovascular risk post - menopause. This trend aligns with data from multiple epidemiological studies, including the Women's Health Initiative, which highlights the increasing impact of metabolic syndrome, lipid abnormalities, and inflammation in this age group.[56]

Recent data from Arora et al.[57] also suggest that younger women are increasingly being hospitalized for myocardial infarction. This concerning trend has been linked to rising rates of obesity, stress, smoking, and diabetes in younger female populations. Additionally, autoimmune diseases such as systemic lupus erythematosus and rheumatoid arthritis, which are more common in women, accelerate atherosclerosis and are associated with increased cardiovascular mortality, as noted by Manzi et al.[58] These non - traditional risk factors further complicate the risk landscape for women and are often overlooked in standard cardiovascular screening.

Social determinants of health—including socioeconomic status, access to healthcare, education, and racial or ethnic disparities—play a crucial role in CAD prevalence and outcomes among women. The World Health Organization and authors like Gulati et al.[59] have emphasized that women from marginalized backgrounds face a disproportionate burden of cardiovascular risk due to systemic inequities. These factors not only affect access to timely diagnosis and treatment but also intersect with lifestyle - related risk factors such as diet, physical activity, and stress.

Finally, a persistent gap in cardiovascular research is the underrepresentation of women in clinical trials. As Regitz - Zagrosek[60]and colleagues have pointed out, this leads to incomplete understanding of sex - specific responses to treatment, drug metabolism, and outcomes. Beale et al.[51] have also explored neurobiological pathways linking psychological stress and cardiovascular risk, which may differ by sex, offering opportunities for more targeted therapeutic strategies.

In conclusion, our study confirms that CAD in women over 40 is multifactorial, with significant contributions from both traditional (diabetes, hypertension, smoking, sedentary lifestyle) and sex - specific (early menopause, stress, hereditary predisposition) risk factors.

These findings emphasize the importance of a gender - sensitive approach in both clinical practice and public health strategy. Enhanced screening, early intervention, and personalized care models that account for hormonal, psychosocial, and lifestyle dimensions are essential to curb the rising burden of CAD in this population. Bridging the gender gap in research, improving mental health integration, and empowering women through education and preventive care are vital steps in reducing cardiovascular mortality and improving outcomes.

6. Conclusion

Through our study, it was evident that stress emerged as the most significant risk factor for coronary artery disease (CAD) in women, followed by hypertension, diabetes, hereditary factors, unhealthy lifestyle habits, early menopause, and smoking, respectively. These findings highlight the multifactorial nature of CAD in women and underscore the unique interplay between biological, behavioral, and psychosocial determinants. Unlike traditional perceptions that primarily emphasized lifestyle and smoking, the prominence of stress and hypertension in this study indicates the need to broaden the scope of preventive strategies.

In conclusion, given the significant burden of CAD among women and the tendency for atypical symptom presentation, raising awareness is critically important. Women must be educated about the diverse risk factors—particularly stress and hormonal changes—that uniquely affect them. Early identification, regular screening, and management of modifiable risk factors can substantially reduce the incidence and severity of CAD. Public health initiatives, targeted educational programs, and personalized preventive strategies are essential to empower women to recognize early signs, adopt healthier lifestyles, and seek timely medical care. Furthermore, healthcare providers should be sensitized to the gender - specific aspects of CAD to ensure early diagnosis and appropriate intervention. Strengthening awareness, prevention, and early management can ultimately contribute to reducing the overall cardiovascular disease burden in women and improving long - term outcomes.

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