WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Coronary Artery Disease Difference by Age, Gender, Region

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ABSTRACT:

Background:

Coronary Artery Disease (CAD), a major manifestation of cardiovascular system (CVS) disease, remains a leading cause of morbidity and mortality worldwide. It is primarily driven by atherosclerosis, characterized by plaque buildup within coronary arteries, which restricts blood flow to the heart. Despite advances in healthcare, CAD persists as a global health challenge, underscoring the need for enhanced preventive, diagnostic, and therapeutic strategies.

Methods:

A comprehensive review of the pathophysiology, risk factors, clinical presentation, diagnostic methods, and treatment approaches was conducted. Epidemiological data were analyzed to underscore CAD's prevalence, while recent advancements in diagnostics (e.g., biomarkers, imaging) and therapeutic options (pharmacotherapy, interventional procedures) were explored. Preventive measures were also reviewed, including lifestyle modifications and public health strategies.

Results:

CAD is associated with both modifiable (e.g., lifestyle, diet, smoking) and non-modifiable (e.g., age, genetics) risk factors. Early diagnosis through imaging and biomarker testing significantly improves patient outcomes, while lifestyle interventions and pharmacotherapy remain essential for disease management. For advanced cases, interventional procedures like angioplasty and coronary artery bypass grafting (CABG) have proven effective. Preventive measuresMBBS) are crucial in reducing CAD incidence and related complications, particularly in high-risk populations.

Conclusions:

The findings reinforce the importance of early diagnosis, risk factor management, and adherence to preventive measures in CAD management. A comprehensive approach, integrating lifestyle changes, pharmacological treatment, and, where necessary, surgical interventions, can substantially reduce CAD's burden. Ongoing research and public health efforts are essential to further mitigate the impact of CAD on global health.

1.INTRODUCTION

The cardiovascular system (CVS) is fundamental to sustaining life by delivering oxygen and nutrients to tissues and removing waste products through blood circulation. It comprises the heart, blood vessels, and blood, working in unison to maintain homeostasis and support various physiological functions. A healthy CVS is vital for overall well-being, while any disruption within this system can lead to significant health consequences.

Coronary Artery Disease (CAD) is one of the most prevalent and severe forms of CVS disease. CAD occurs when the coronary arteries, responsible for supplying oxygen-rich blood to the heart muscle, become narrowed or blocked due to atherosclerosis—a condition where plaque made up of cholesterol, fat, and other substances accumulates on arterial walls. This narrowing restricts blood flow, which can lead to symptoms like chest pain (angina) and, in severe cases, myocardial infarction (heart attack).

As the leading cause of death worldwide, CAD poses a substantial global health challenge, impacting millions each year. Risk factors such as high cholesterol, hypertension, smoking, diabetes, obesity, and sedentary lifestyles contribute to its prevalence.

The progressive nature of CAD and its potential complications make early diagnosis, preventive measures, and effective management essential to reducing its impact on patients and healthcare systems alike. Understanding CAD's pathophysiology, risk factors, and available treatment options is crucial for developing comprehensive strategies to combat this widespread disease.

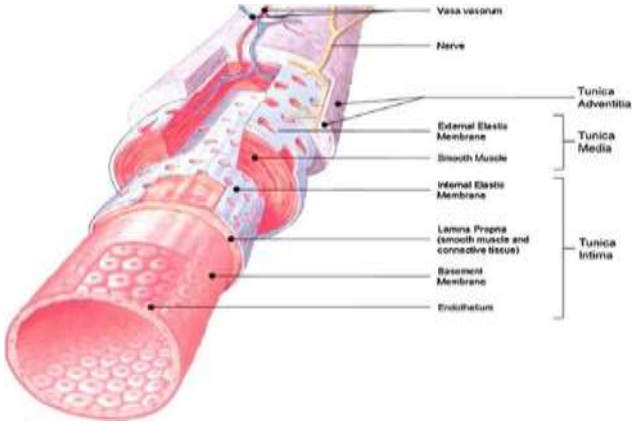
2. Coronary Artery disease

Coronary Artery Disease (CAD) is a condition where the coronary arteries, which supply oxygen-rich blood to the heart muscle, become narrowed or blocked. This occurs due to a buildup of plaque, a mixture of cholesterol, fats, calcium, and other substances, on the inner walls of the arteries—a process known as atherosclerosis.

As plaque accumulates, it restricts blood flow to the heart, which can cause chest pain (angina), shortness of breath, or, in severe cases, a heart attack if blood flow is completely blocked. Risk factors include high cholesterol, hypertension, smoking, diabetes, obesity, and a sedentary lifestyle. CAD is one of the leading causes of death worldwide

2.1. Normal Coronary Artery Anatomy :

- Intima: The innermost layer, composed of a thin layer of endothelial cells. This layer provides a smooth lining for blood flow and is sensitive to damage from factors like high cholesterol and hypertension, making it susceptible to atherosclerosis.
- Media: The middle layer, consisting of smooth muscle cells. It provides structural support and flexibility to the artery, allowing it to adjust to changes in blood flow and pressure.
- Adventitia: The outermost layer, made of connective tissue, nerve fibers, and small blood vessels (vasa vasorum). This layer supplies nutrients to the artery wall and anchors the artery within the surrounding tissues.



2.1.2. Normal Coronary Blood Flow and Oxygen Delivery:

- Coronary arteries originate from the base of the aorta and branch out across the heart, ensuring that all areas of the myocardium (heart muscle) receive oxygenated blood.
- These arteries deliver oxygen and nutrients required by the myocardium for continuous contraction and relaxation.
- In a healthy heart, this blood flow is unimpeded, allowing for optimal oxygen delivery, essential for maintaining normal heart function but can often be managed or prevented through lifestyle changes, medications, and medical interventions.

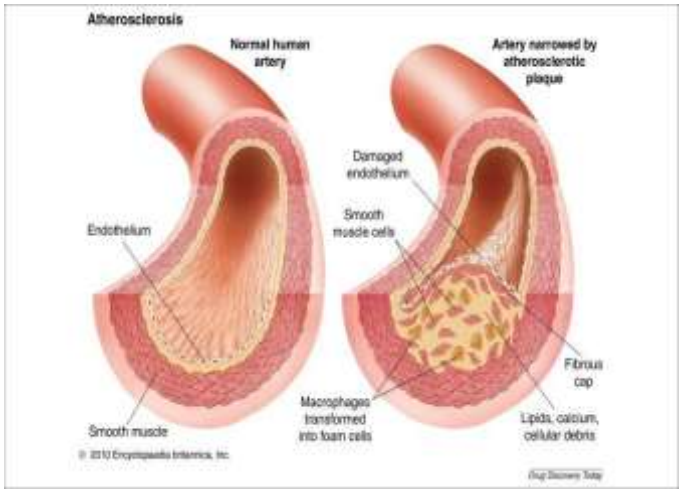
2.2.Causes of CAD

- High LDL Cholesterol: Contributes to plaque formation in arteries
- Low HDL Cholesterol: Inadequate “good” cholesterol to clear LDL, increasing risk.
- Hypertension (High Blood Pressure): Damages artery walls, facilitating plaque buildup.
- Smoking: Toxins and nicotine damage arteries, promote plaque, and raise blood pressure.
- Diabetes and Insulin Resistance: High blood sugar damages arteries, accelerating plaque buildup.
- Obesity: Raises LDL, blood pressure, and risk of insulin resistance.
- Sedentary Lifestyle: Leads to weight gain and low HDL, contributing to CAD risk.
- Unhealthy Diet: High in saturated/trans fats and sodium; increases cholesterol and blood pressure.
- Chronic Stress: Increases blood pressure, inflammation, and unhealthy coping behaviors.

- Family History and Genetics: Inherited factors influencing cholesterol and blood pressure.
- Age and Gender: CAD risk increases with age; men have higher risk earlier, women's risk rises post-menopause.

2.3. Development of CAD through Atherosclerosis :

1. Plaque Formation: CAD begins with atherosclerosis, where plaque (cholesterol, fats, calcium) starts building up on the inner walls of coronary arteries.
2. Endothelial Damage : High cholesterol, hypertension, smoking, and other factors damage the artery lining (endothelium), making it easier for plaque to accumulate.
3. LDL Cholesterol Deposition: LDL ("bad") cholesterol particles penetrate damaged areas of the endothelium, starting the buildup of fatty deposits.
4. Inflammation and Immune Response :The body responds to this damage with inflammation, where white blood cells (like macrophages) try to "clean up" LDL, but instead, they absorb it and form foam cells, thickening the arterial walls.
5. Plaque Growth: Over time, plaque hardens and enlarges, narrowing the artery and restricting blood flow. This process can continue over many years without symptoms.
6. Reduced Blood Flow : Narrowed arteries limit the oxygen-rich blood supply to the heart, causing symptoms like chest pain (angina) during physical exertion or stress.
7. Rupture and Clot Formation: Plaque can eventually rupture, triggering a blood clot at the site, which may fully block blood flow and lead to a heart attack.



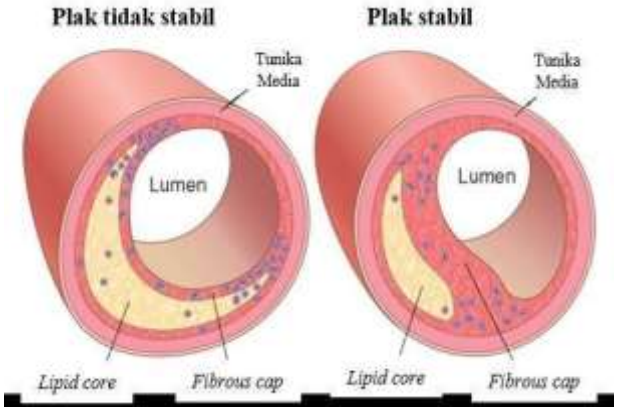
3. Plaque structure and composition

3.1.Types of Plaque

1. Stable Plaque:
   - Characteristics: Has a thick, robust fibrous cap and a smaller lipid (fat) core.
   - Behavior: Stable plaques are less likely to rupture and cause blood clots but still narrow the artery, restricting blood flow.
   - Symptoms: Typically causes angina (chest pain), especially during exertion when oxygen demand is higher.
2. Unstable Plaque:
   - Characteristics: Has a thin, fragile fibrous cap and a larger lipid core.
   - Behavior: More likely to rupture, exposing the core to the bloodstream and triggering blood clot formation.
   - Consequences: When rupture and clotting occur, this can suddenly block blood flow, leading to a heart attack (myocardial infarction).

3.2 Components of Plaques:

- Lipid Core: Composed of cholesterol and other fatty substances. The size and composition of the lipid core can influence plaque stability; a larger core increases the risk of rupture.
- Fibrous Cap: A layer of connective tissue covering the lipid core. It's made of collagen and proteins, which give it strength. The thickness of the cap determines the plaque's stability.
- Smooth Muscle Cells: These cells migrate to the plaque from the artery's middle layer and produce collagen for the fibrous cap. They play a role in stabilizing the plaque but can contribute to artery wall thickening.
- Foam Cells: Macrophages (immune cells) that have absorbed oxidized LDL cholesterol and become engorged. Foam cells accumulate in the plaque, contributing to its growth and instability.



3.3. Differences Between Plaques that Cause Angina vs. Heart Attacks :

3.3.1.Plaques Leading to Angina:

- Typically stable plaques with a thick fibrous cap and moderate narrowing of the artery.
- These plaques restrict blood flow without rupturing, causing chest pain (angina), especially during increased physical activity when oxygen demand rises.

3.3.2.Plaques Leading to Heart Attacks:

- Typically unstable plaques with a thin fibrous cap and large lipid core.
- These plaques are prone to rupture. When they rupture, a blood clot can form at the site, blocking blood flow entirely and resulting in a heart attack.

Understanding the differences in plaque composition helps in predicting the risk of CAD complications, such as angina or heart attack, based on plaque stability and likelihood of rupture.

4.Plaque Rupture and Thrombosis

Plaque rupture and thrombosis are critical events in the progression of Coronary Artery Disease (CAD) and can lead to severe complications, such as heart attacks or embolization. Here's an explanation of each component:

4.1. Mechanism of Plaque Rupture and Blood Clot Formation :

- Plaque Structure: Plaques consist of a lipid-rich core covered by a fibrous cap. In unstable plaques, this fibrous cap is thin and prone to rupture.
- Rupture Process: Factors like high blood pressure or inflammation can weaken the cap. When it ruptures, it exposes the lipid core to the bloodstream.
- Clot Formation : (Thrombosis): The body perceives this exposure as an injury and rapidly activates platelets and clotting factors, forming a thrombus (blood clot) at the rupture site. This clot can partially or completely block the artery, stopping blood flow.

4.2. Myocardial Infarction (Heart Attack)

- Sudden Blockage: When a clot fully blocks a coronary artery, oxygen-rich blood can no longer reach part of the myocardium (heart muscle).
- Ischemia and Cell Death: The deprived heart muscle becomes ischemic, leading to damage and eventual cell death. This process results in a myocardial infarction (heart attack).
- Symptoms: Common symptoms include severe chest pain, shortness of breath, and sweating. Rapid treatment is essential to restore blood flow and minimize heart damage.

4.3.Embolization :

- Clot Dislodgement: Sometimes, pieces of the clot can break off (embolize) and travel through the bloodstream.
- Effects on Other Areas: These emboli can lodge in smaller arteries within the heart or even in other organs, causing additional blockages. If they reach the brain, they may lead to a stroke.
- Systemic Impact: Embolization increases the risk of widespread complications, making plaque rupture a potentially systemic issue.

Plaque rupture and thrombosis highlight how CAD can quickly escalate from gradual artery narrowing to life-threatening events like heart attacks and embolic complications.

5.Microscopic and Gross pathological Findings in CAD :

The pathological examination of coronary arteries affected by Coronary Artery Disease (CAD) reveals significant changes at both microscopic (cellular) and gross (visible) levels, illustrating the progression and severity of atherosclerosis.

5.1.Microscopic Findings :

5.1.1 Foam Cells:

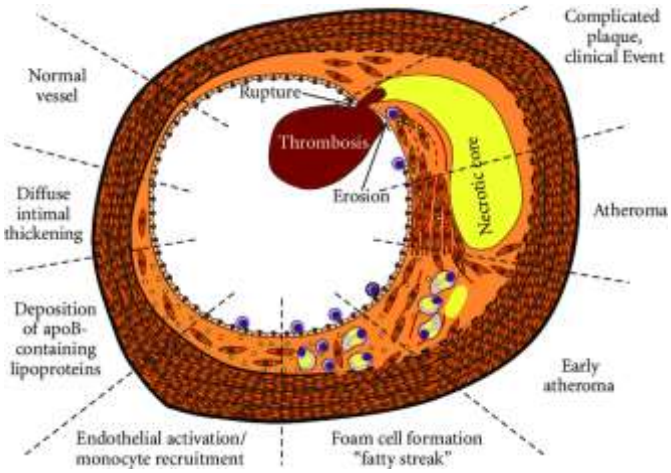
- Foam cells are one of the earliest microscopic changes in atherosclerosis. They form when macrophages (immune cells) engulf oxidized LDL (bad cholesterol) within the arterial wall.
- These lipid-laden macrophages appear “foamy” under a microscope due to their high fat content. They accumulate and initiate the formation of fatty streaks, which are the first visible signs of plaque development.

5.1.2 Macrophages:

- Macrophages are immune cells that migrate to areas of endothelial damage and inflammation, where they attempt to “clean up” oxidized LDL. However, they often end up becoming foam cells instead, contributing to plaque growth.
- These macrophages also release inflammatory chemicals that attract more immune cells, furthering the inflammatory response and accelerating plaque progression.

5.1.3 Necrotic Core:

- As plaques mature, areas within the plaque undergo cell death (necrosis), forming what is known as a necrotic core. This core is composed of dead cells, cellular debris, and cholesterol crystals.
- The necrotic core increases plaque instability. The larger the core, the more prone the plaque is to rupture, leading to potential complications like blood clot formation.



5.2.Gross Pathology :

5.2.1. Narrowing of Coronary Arteries:

- Atherosclerosis causes visible narrowing (stenosis) of the coronary artery lumen (the blood-flowing space within the artery). In severe cases, this narrowing can significantly restrict blood flow, especially during physical exertion.
- This narrowing appears as a reduction in the diameter of the artery and can be observed during angiography or in post-mortem examinations of the heart.

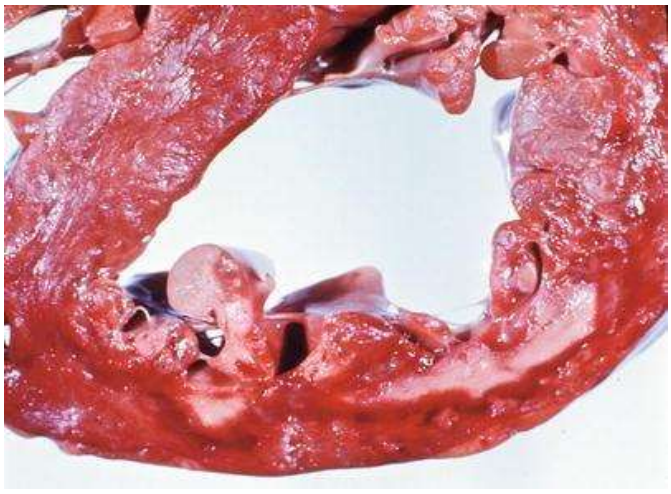
5.2.2. Plaque Formation and Appearance:

- On gross examination, plaques appear as raised, yellowish-white areas along the inner wall of the coronary artery.
- Advanced plaques are thicker and protrude into the artery's lumen, sometimes visibly blocking blood flow. Over time, plaques may become calcified, making them appear hard and rigid.

5.2.3. Calcifications:

- In later stages of CAD, calcium deposits may accumulate within plaques. This process, known as calcification, gives the artery walls a hardened, "bone-like" appearance and decreases their flexibility.
- Calcified plaques are visible on imaging tests, such as CT scans, and indicate advanced atherosclerosis. Although calcified plaques tend to be more stable, they contribute to artery stiffening, which can reduce the artery's ability to expand and contract with blood flow.

In summary, microscopic findings in CAD reveal cellular changes that lead to plaque development, while gross pathological changes illustrate the visible consequences of plaque buildup and calcification in coronary arteries. Both levels of findings help to provide a comprehensive view of CAD progression, from early cellular changes to advanced structural modifications of the arteries.



6.Age Group characteristics in CAD Development with prevalence Estimates:

Age Group	Type of plaque	Key Condition	Detection and Management	Prevalence of CAD
Less than 20	Uncommon at this age	Familial Hypercholesterolemia (FH): A genetic mutation causing severely elevated LDL cholesterol levels, significantly increasing early CAD risk.	Early Screening: Lipid testing for those with family history can identify at-risk youth early, enabling preventive care like statins if needed.	<1%
			Preventive Lifestyle Guidance: Educating young individuals on diet and exercise helps delay or prevent early plaque formation.	
21-30	Unstable plaques	Smoking and Lifestyle Choices: Smoking accelerates endothelial damage, initiating plaque formation early in life.	Early Counseling: Advising on smoking cessation, dietary changes, and physical activity is essential to reduce CAD onset in this group.	1-5%
			Subclinical Atherosclerosis Testing: Coronary calcium scoring may be beneficial for at-risk individuals showing early signs of CAD.	
31-45	stable and unstable plaques	Hypertension and Hyperlipidemia: Persistent high blood pressure and cholesterol accelerate plaque growth and arterial damage.	Routine Screenings: Regular checks on blood pressure, lipid profile, and lifestyle can help detect early CAD for timely intervention.	5-10%
			Medication and Lifestyle Modifications: Prescribing statins and antihypertensives, along with dietary and exercise changes, to control risk.	
46-65	stable can lead to angina, unstable can trigger heart attacks	Plaque Maturation and Inflammation: Chronic inflammation and plaque growth lead to stable or unstable plaques, increasing CAD risk.	Stress Testing and Management: Exercise stress testing is crucial to identify symptoms; statins, lifestyle modifications, and rehabilitation are recommended.	20-40%
			Dual Approach to Therapy: Combining medication with lifestyle interventions improves	

			long-term outcomes in managing CAD symptoms.	
More than 65	**Predominantly Stable plaques**	**Age-Related Arterial Stiffness: Reduced arterial flexibility leads to stable plaques, causing gradual artery narrowing and frequent angina.**	**Comprehensive Cardiac Care: Involves cardiology, lifestyle adjustments, medication (statins, antiplatelets), and cardiac rehab for functional health.**	**50 -70%**
			Medication Optimization: Using a combination of statins, antiplatelets, and anticoagulants to manage severe cases and prevent complications.	

- Coronary artery disease (CAD) follows a distinct trajectory across age groups, influenced by genetic, lifestyle, and physiological factors. In those under 20, CAD is exceedingly rare and almost exclusively linked to genetic conditions, such as familial hypercholesterolemia, necessitating early lipid screening and preventive measures for those with significant family history.
- In early adulthood (21-30), risk begins to rise as lifestyle choices like smoking, poor diet, and stress initiate endothelial damage.While clinical symptoms are usually absent in this age range, early intervention through lifestyle counseling and risk-factor modification can play a crucial role in delaying disease onset.
- As adults reach their 30s and early 40s, the prevalence of CAD escalates, influenced by systemic factors such as hypertension, hyperlipidemia, and metabolic syndrome. This age group often experiences initial symptoms like angina, signaling the need for routine cardiovascular monitoring and the potential introduction of medications, such as statins and antihypertensives, to control plaque progression.
- In middle age (46-65), CAD prevalence increases sharply, with years of cumulative inflammation and endothelial stress contributing to both stable and unstable plaque formation, which heightens the risk of acute coronary events.
- In older adults over 65, CAD becomes most common, as aging-related arterial stiffness and the gradual narrowing of vessels create a predisposition to stable plaque accumulation, chronic angina, and complications like heart failure. Multidisciplinary care, including medication optimization, lifestyle modifications, and regular cardiac monitoring, becomes essential to manage symptoms and maintain cardiovascular health.
- This age-stratified approach to CAD highlights the importance of early lifestyle interventions, vigilant monitoring, and comprehensive management to mitigate the disease's progression across the lifespan.

7.Gender characteristics in CAD Development with prevalence Estimates:

characteristic	**Males**	**Females**
plaque	**Unstable plaques more common in younger males, increasing the risk of sudden events like heart attacks.**	**Predominantly stable plaques, especially pre-menopause, leading to gradual narrowing and chronic symptoms later.**
Plaque composition	**More prone to unstable plaques in younger males, which are at higher risk for rupture, leading to sudden heart attacks.**	**More likely to develop stable plaques, especially pre-menopause, leading to gradual artery narrowing and angina in later years.**
symptomatic vs Asymptomatic	**More likely to experience symptomatic CAD at an earlier age;**	**Higher likelihood of asymptomatic or atypical symptoms, like fatigue or nausea, especially pre-menopause.**

	chest pain (angina) is the common symptom.	
symptom onset	**Symptoms (e.g., angina) often start earlier, with common onset around 45-55 years.**	**Symptoms often present later, usually post-menopause (55-65 years), when risk significantly increases.**
Types of symptoms	**Typical symptoms include chest pain (angina), pressure, or discomfort in the chest, often triggered by physical exertion.**	**Symptoms are often atypical, such as fatigue, shortness of breath, nausea, or jaw pain, making CAD harder to diagnose.**
Role of Estrogen	**Estrogen has minimal impact on CAD risk in males.**	**Pre-menopausal estrogen offers a protective effect, reducing risk. Post-menopause, decreased estrogen accelerates CAD risk.**
Response to stress	**Psychological and work-related stress strongly contribute to early CAD in males.**	**Stress often impacts females differently; social, family-related stress, and anxiety can increase CAD risk, especially post-menopause.**
Complications	**Higher risk of sudden cardiac events like myocardial infarction due to unstable plaque rupture.**	**More prone to chronic CAD complications like heart failure due to gradual narrowing, especially in older age.**
Prevalence	**Higher prevalence overall, with earlier onset of symptoms, often starting around 45-55 years.**	**Lower prevalence in early years; catches up to males post-menopause, around 55-65 years.**
Prevalence of asymptomatic CAD	**Lower percentage of asymptomatic cases, with symptoms presenting earlier.**	**Higher prevalence of asymptomatic CAD, especially pre-menopause, increasing risk of delayed diagnosis.**
Primary risk factors	**Smoking, high cholesterol, high blood pressure, and stress are more direct contributors to early CAD onset.**	**Hypertension, diabetes, and family history play a strong role post-menopause.** **Lifestyle factors like inactivity and stress are impactful.**
Diagnosis and detection	**Typically diagnosed earlier due to more classic symptoms and regular screenings.**	**Often diagnosed later due to atypical symptoms; risk of delayed detection increases with age and post-menopausal changes.**
Treatment response and Tolerance	**Generally respond well to standard treatments (e.g., statins, beta-blockers); fewer medication side effects.**	**May experience higher sensitivity or side effects to certain medications; post-menopausal hormone therapy impacts treatment response.**

Who is Most Affected by CAD?

Based on the insights, CAD impacts both genders significantly but in different ways and timelines:

1. Males are affected earlier in life due to lifestyle factors (like smoking and stress) and the absence of protective estrogen, leading to symptomatic CAD, often around age 45-55.

2. Post-menopausal females become increasingly affected by CAD as estrogen levels drop, which previously provided a protective effect. This shift makes women over 55-65 years more susceptible, especially with chronic conditions like hypertension and diabetes.

3. Individuals with high-risk factors, such as a family history of CAD, smoking, high cholesterol, and diabetes, are at greater risk for early onset and more severe CAD, regardless of gender.

In summary: Younger males tend to experience earlier onset with higher risks of sudden events (like heart attacks due to unstable plaques), while post-menopausal females have a high prevalence with a progression toward chronic complications (like heart failure due to stable plaques).

8.Region characteristics in CAD Development with prevalence Estimates:

Characteristic	**Tamil Nadu**	**Osh**
Primary Risk Factors	High rates of hypertension, diabetes, obesity, and stress, with dietary habits high in saturated fats and carbohydrates.	Smoking and high cholesterol are more common risk factors; traditional diets are lower in fat but high in carbohydrates.
Lifestyle and Physical Activity	Sedentary urban lifestyle with low physical activity levels among adults; limited time for exercise due to work stress.	Rural areas have higher physical activity, but urban lifestyles in Osh are shifting towards more sedentary habits.
Dietary Influence	High intake of rice, fried foods, and sugary beverages in urban regions; southern cuisine rich in fats and carbs.	Traditional diet includes bread, meat, and dairy; rising consumption of processed foods in urban settings contributes to risk.
Smoking and Alcohol Consumption	Smoking rates are comparatively lower but rising in certain demographics; alcohol consumption is moderate.	Higher smoking rates, especially among males, significantly contributing to CAD risk; alcohol consumption also notable.
Symptom Presentation	Symptoms like angina and shortness of breath are common, often detected early due to awareness and screenings.	CAD often remains undiagnosed until later stages, with symptoms like chest pain presenting later due to limited screening.
Awareness and Preventive Measures	Increased awareness and focus on preventive	Limited awareness; fewer preventive campaigns, and

	measures, with government campaigns tarneting lifestyle changes.	lifestyle changes are less commonly promoted.
Healthcare Access	Accessible healthcare infrastructure, with numerous specialized heart care centers and routine cardiac screenings.	Limited access to specialized cardiac care and advanced screenings; healthcare mainly in general practice settings.
Preventive Health Measures	Government and NGO-led initiatives promoting lifestyle changes, diet modification, and routine screenings are widely implemented and accepted.	Limited preventive campaigns; health-seeking behavior for routine checkups is lower, especially in rural areas, but awareness is gradually rising.
Response to Treatment	Good response to lifestyle interventions and medications; high compliance in urban centers with easier access to ongoing care and follow-ups.	Smoking culturally accepted, particularly among men; preventive health-seeking behavior lower, with less emphasis on lifestyle changes.
Treatment Options and Accessibility	Access to a variety of treatment options, including medications, interventions, and surgical procedures.	Limited availability of advanced treatments; cardiac interventions and surgery may require travel to larger cities.
Cultural Attitudes	High health awareness and willingness to pursue preventive and early treatment measures; social stigma around smoking and obesity in certain demographics.	Cultural acceptance of smoking among males and high-fat diets; lower preventive health-seeking behavior.
CAD Prevalence	CAD rates are notably high, with urban areas seeing 10-20% prevalence in middle-aged adults; rural areas also increasing due to lifestyle shifts.	Historically lower CAD prevalence, but rates are climbing with urbanization and dietary shifts; estimated around 5-15% in urban populations.
Physical Activity Levels	Sedentary urban lifestyle with low activity rates; rural areas more active, but transition to sedentary jobs is increasing across the region.	Physical activity remains relatively high in rural settings, but urban populations are adopting more sedentary habits, mirroring lifestyle in Tamil Nadu.

Based on the comparison between Tamil Nadu and Osh:

1. Middle-aged and Urban Populations are most affected by CAD in both regions, but particularly in Tamil Nadu, where urbanization has led to higher rates of hypertension, diabetes, and obesity. Urban residents in Tamil Nadu face a 10-20% CAD prevalence in middle-aged adults, driven by lifestyle changes, high-fat diets, and stress.

2. Men and Smokers are at higher risk in Osh, where smoking rates are significantly high, especially among men. This increases CAD risk, compounded by a diet rich in meat and dairy. Smoking prevalence is a major contributing factor to CAD in Osh, often more pronounced than in Tamil Nadu.

3. Individuals with Limited Access to Healthcare are more vulnerable in Osh, where specialized cardiac care is limited. This impacts early diagnosis and preventive care, causing CAD cases to remain undiagnosed until advanced stages.

4. People with High-Risk Lifestyles and Diets: In Tamil Nadu, high rates of physical inactivity, processed food consumption, and high-carbohydrate diets are major contributors to CAD prevalence. This particularly affects those in urban settings who adopt sedentary lifestyles.

5. Older Adults are more affected in both regions due to cumulative exposure to risk factors like hypertension and high cholesterol over time, with a greater likelihood of developing stable plaques that contribute to CAD.

Summary

Key Findings: Coronary artery disease (CAD) risk in Tamil Nadu and Osh is shaped by unique regional factors. In Tamil Nadu, CAD prevalence is higher due to urbanization, lifestyle shifts, and dietary habits that contribute to hypertension, diabetes, and obesity. Osh faces significant CAD risk from high smoking rates, particularly among men, coupled with limited healthcare access that delays diagnosis and treatment.

Public Health Implications: Effective CAD prevention strategies require region-specific approaches. In Tamil Nadu, promoting active lifestyles, dietary changes, and stress management in urban areas could mitigate rising CAD rates. In Osh, reducing smoking prevalence and improving healthcare infrastructure for early CAD detection are essential.

Gender and Age-Specific Strategies: Males experience earlier CAD onset due to lifestyle factors, while post-menopausal women face rising risk from declining estrogen levels. Age-targeted interventions, such as lipid screenings for younger adults with family history and enhanced cardiac care for seniors, are crucial for managing risk across the lifespan.

Community Awareness and Preventive Care Raising CAD awareness and enhancing preventive care efforts in both regions can lead to earlier intervention and better health outcomes. Community-focused education on lifestyle changes and regular screenings can substantially lower CAD rates and improve long-term cardiovascular health.

Future Research Directions: Further studies are recommended to investigate genetic, lifestyle, and environmental contributors to CAD in these regions, supporting the development of tailored interventions to address the unique needs of each population.

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