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A REVIEW ON PREVALENCE OF ANGINA PECTORIS IN ASSOCIATION WITH CCF (CONGESTIVE CARDIAC FAILURE) IN GENDER AND AGE-BASED POPULATION

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Abstract

Angina pectoris, a hallmark symptom of ischemic heart disease (IHD), is a leading cause of morbidity and mortality worldwide. It typically presents as retrosternal chest pain radiating to the jaw, arms, or shoulders, often triggered by exertion or stress, and is caused by an imbalance between myocardial oxygen supply and demand due to atherosclerosis, endothelial dysfunction, or vasospasm. Persistent ischemia and recurrent angina can lead to or worsen congestive cardiac failure (CCF), characterized by the heart's inability to maintain adequate circulation. CCF further complicates angina, reducing quality of life and increasing cardiovascular risk. This study explores the prevalence of angina in relation to CCF across genders and age groups. Data show that prevalence rises with age; men are more affected at younger ages, while women show higher rates post-menopause. Risk factors like hypertension, diabetes, obesity, dyslipidemia, and smoking contribute to disease progression. Management includes lifestyle changes, medications such as beta-blockers, calcium channel blockers, nitrates, and newer agents, along with risk factor control. The study underscores the importance of early diagnosis, gender- and age-specific risk stratification, and targeted therapies to reduce the burden of angina and its progression to heart failure, ultimately improving outcomes and reducing mortality.

Introduction

Angina pectoris, commonly referred to as chest pain, is the most characteristic symptom of ischemic heart disease (IHD), a major contributor to morbidity and mortality worldwide. Chest pain may originate from both cardiac and non-cardiac causes, hence detailed history and physical examination are critical in identifying patients with acute coronary syndrome (ACS) [1]. Angina can be classified as stable or unstable. Stable angina typically appears with exertion or stress and is relieved by rest or nitrates, whereas unstable angina occurs at rest or with increasing severity and requires urgent evaluation [2]. The pain is usually described as heaviness, pressure, or tightness in the retrosternal area, often radiating to the neck, jaw, arms, or shoulders [3]. Dyspnea on exertion is also considered an anginal equivalent. In contrast, pain associated with posture, respiration, or trauma is generally non-cardiac [4]. Globally the prevalence of

chronic stable angina ranges from 30,000 to 40,000 cases per million people, with higher rates in elderly populations. Men are more commonly affected at younger ages, while women show an increased prevalence after menopause [5]. Risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, and sedentary lifestyle contribute significantly to the development and progression of angina [6]. Understanding the patophysiology, risk factors, and clinical profile of angina pectoris is essential for timely diagnosis and management, particularly as it remains as a major contributor to the global burden of cardiovascular disease [7].

Types of angina

1. Angina only with strenuous exertion.
2. Angina during ordinary daily activities.
3. Angina with normal walking or climbing one flight of stairs.
4. Angina with minimal activity or at rest [8].

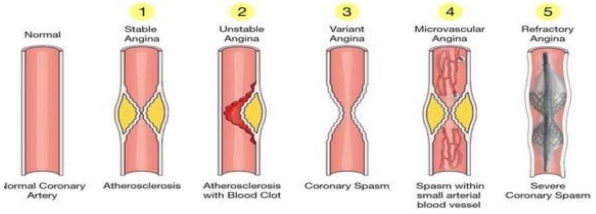

Fig 01: Image of types of angina pectoris

Stable Angina

Predictable, short-lasting chest pain occurring with exertion or stress, relieved by rest or nitrates. The pain is due to a temporary reduction in blood flow to the myocardium as a result of fixed atherosclerotic narrowing of the coronary arteries. It usually subsides within a few minutes after rest or administration nitrates. The underlying pathology involves a stable atheromatous plaque that limits coronary blood flow but does not rupture [9].

Common symptoms include retrosternal chest pain radiating to the left arm, neck, jaw or back, often described as a feeling of heaviness, pressure, or squeezing. Factors like smoking, hypertension, diabetes mellitus, and hyperlipidemia further aggravate the condition. Electrocardiogram (ECG) findings during an episode may show transient ST-segment depression or T-wave inversion, which return to normal at rest.

Unstable Angina

More severe, prolonged chest pain, often occurring at rest or with increasing intensity, requiring hospital admission due to high risk of myocardial infarction [10]. Unstable angina is a more severe and unpredictable form of angina pectoris, often considered as a medical emergency. It is marked by prolonged, recurrent, or severe chest pain that may occur even at rest or with minimal exertion. The discomfort trends to last longer than that seen instable angina and is less responsive to rest or nitrates.

The unstable angina results from rupture or erosion of an atherosclerotic plaque, leading to platelet aggregation and partial thrombus formation within the coronary artery. This causes sudden and significant reduction in myocardial blood flow, increasing the risk of myocardial infraction if left untreated. Patients of often experience increasing frequency or intensity of chest pain, sometimes accompanied by shortness of breath, sweating, or nausea. ECG changes my include ST-segment depression or transient elevation, and cardiac biomarkers (like troponins) may remain normal, distinguishing from myocardial infraction. Management requires immediate hospital admission, continuous ECG monitoring, and treatment with antiplatelet agents (aspirin, clopidogrel), anticoagulants (heparin), and anti-ischemic drugs. Lifestyle modifications along with early medical intervention play a vital role in preventing progression to myocardial infarction.

Pathophysiology of Angina

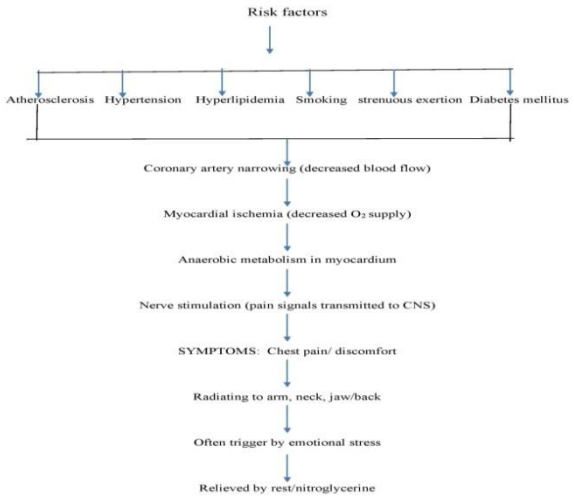

Fig 02: Patophysiology of angina pectoris

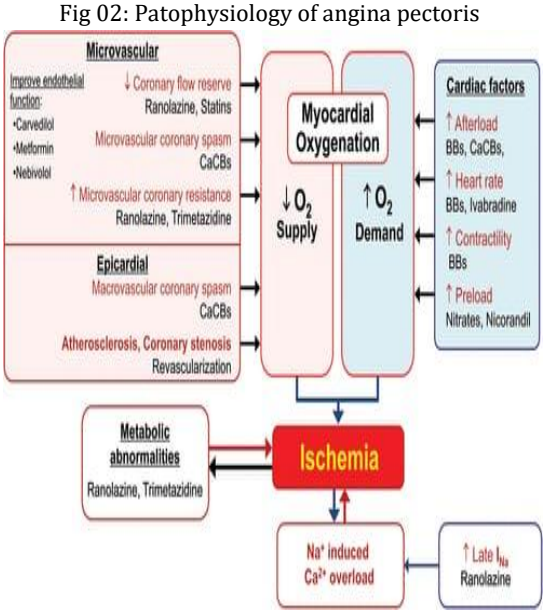

Fig 03: Mechanism action of angina pectoris

Microvascular Angina

Caused by impaired coronary flow reserve due to microvascular resistance or abnormal vasodilation [11].

Vasospastic (prinzmetal) Angina

Results from transient vasospasm of epicardial coronary arteries, leading to temporary ischemia, sometimes in the absence of fixed stenosis [12].

Congestive cardiac failure (CCF)

Heart failure occurs when the heart is unable to pump blood efficiently to meet the metabolic needs of the body [13].

Classification by Pumping Ability

- **Systolic failure (HFrEF):** Reduced ejection fraction due to weakened myocardium.
- **Diastolic failure (HFpEF):** Preserved contractility but impaired relaxation and filling [14].

Classification by Onset

- **Acute HF:** Sudden onset, often after myocardial infarction or arrhythmia.
- **Chronic HF:** Gradual onset, usually due to long-standing hypertension or CAD [15].

Classification by Heart Involvement

- **Left-sided HF:** Pulmonary congestion, dyspnea, and cough.
- **Right-sided HF:** Venous congestion, edema, ascites.
- **Biventricular HF:** Combination of left and right failure [16].

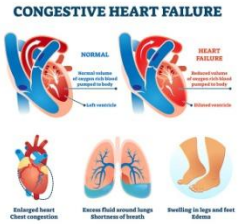

Fig 04: Image of Congestive Heart Failure.

How Patients of Angina pectoris leads to CCF

Angina pectoris occurs when the heart muscle does not get enough oxygen due to reduced blood flow, usually because of coronary artery disease. The coronary arteries become narrowed or blocked by atherosclerotic plaques, which decreases oxygen supply to the myocardium. When the oxygen supply is continuously reduced, the heart muscles become week and are unable to contract efficiently. This leads to a decrease in cardiac output (the amount of blood pumped by the heart per minute). Overtime the lack of oxygen causes damage and death of cardiac muscle cells, resulting in the form of scar tissue. The heart gradually loses its pumping ability, leading to left ventricular dysfunction.

Table 01: Stages of Angina Pectoris to Congestive Heart Failure.

Stage	Event	Effect
1st stage	Coronary artery narrowing reduced oxygen to heart muscle	Myocardial ischemia (angina)
2nd stage	Prolonged ischemia	Myocardial damage and weakened contraction
3rd stage	Decreased Cardiac output	Left ventricular failure
4th stage	Blood backs up into lungs and body	Pulmonary and systemic congestion

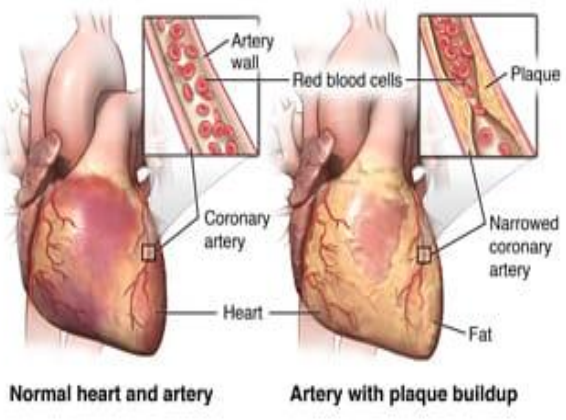

Fig 05: Image of Plaque Buildup.

Factors related to Angina and CCF based on gender and age-based population

Epidemiology

Chronic stable angina remains one of the most common clinical manifestations of ischemic heart disease (IHD) worldwide. In Western countries, its prevalence is estimated at approximately 30,000 to 40,000 cases per million populations. The burden of angina clearly increases with advancing age in both men and women. Epidemiological studies report that in individuals aged 45 to 64 years, the prevalence is around 4–7% in men and 5–7% in women. However, in the elderly group aged 65 to 84 years, the prevalence significantly rises to 14–15% in men and 10–12% in women [17].

Several risk factors have been strongly associated with the development of angina. Modifiable risk factors include hyperlipidemia, hypertension, tobacco smoking (current or past), diabetes mellitus, and obesity or metabolic syndrome. Notably, an increase in body mass index (BMI) has been identified as an independent risk factor for coronary artery disease (CAD) and consequently angina.[18] In contrast, non-modifiable risk factors consist

of advanced age, male sex, family history of CAD, and ethnicity, all of which play a crucial role in the susceptibility to angina [19].

Table 2: Prevalence of Congestive Heart Failure.

Global Prevalence	Prevalence by age group	Age factor	Risk factor	Conclusion
In Western countries, prevalence is estimated at approximately 30,000-40,000 cases per million population.	45-6 Figure 4: Image of Congestive Heart Failure.4 years: around 7-8% in men and 4-7% in women. 65-84 years: Rises to 14 -15% in men and 10-12% in women.	The burden of angina increases with advancing age in both men and women.	Modifiable: Hyperlipidemia, Hypertension, smoking Non modifiable: Age, Male sex, CAD	Advancing age and presence of multiple risk factors increasing susceptibility to angina, often leading to complications like CCF.

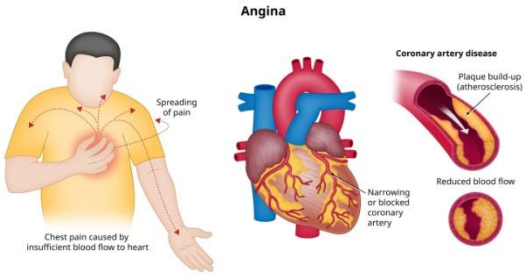

Fig 06: Image of Angina Pectoris.

Etiology

The etiology of chest pain is multifactorial and may originate from non-cardiac, non-ischemic cardiac, or ischemic cardiac causes.

- Non-cardiac causes include conditions such as gastroesophageal reflux disease (GERD), pulmonary disorders (e.g., asthma, chronic obstructive pulmonary disease), musculoskeletal abnormalities, and psychiatric conditions like anxiety and panic attacks [20].
- Non-ischemic cardiac causes most frequently involve pericardial diseases including pericarditis and pericardial effusion [21].
- Ischemic cardiac causes, which form the major category, are primarily due to atherosclerosis of coronary arteries and coronary vasospasm [22].

The pathophysiological mechanism underlying ischemic angina involves a mismatch between myocardial oxygen supply and demand. unstable angina, ischemia occurs predominantly during exertion due to an increase in myocardial oxygen demand—caused by elevated heart rate, however, in the presence of atherosclerosis, this mechanism is impaired, leading to reduced perfusion and chest discomfort [23]. In contrast, unstable angina results from ischemia that can also occur at rest, often associated with plaque rupture and thrombosis.

Another important form, vasospastic (Prinzmetal or variant) angina, occurs due to transient spasm of the coronary arteries. Unlike stable angina, this condition can develop at rest and is not necessarily linked to significant atherosclerotic disease [24].

Management:

1. Non-pharmacological/Life style measures:

These are always used in conjunction with drugs:

Smoking cessation:

(Including active smoking, passive exposure, and smokeless tobacco). Smoking increases vasoconstriction, inflammation, and promotes atherosclerosis.

Alcohol moderation:

- Excessive alcohol may occur hypertension, dyslipidemia, etc.

Dietary modification:

- Low saturated fat, increased fiber, fruits/vegetables, limiting cholesterol;
- Mediterranean-style diets are favourable.

Exercise

Regular moderate aerobic exercise improves endothelial function, reduces symptoms and improves cardiovascular fitness.

Weight control

Obesity contributes to hypertension, dyslipidemia, diabetes.

Stress reduction

Avoiding cold exposure, heavy metals etc. which may precipitate angina by increasing myocardial oxygen demand [25, 26].

2. Prevention and Risk Factor Control

Since angina is usually a manifestation of coronary artery disease (CAD), controlling risk factors is critical.

Lipid control

- Statins are recommended to lower LDL cholesterol. Many guidelines aim for LDL-C < 70 mg/dL in high risk (existing CAD) patients [27,28].

Blood pressure control: Target systolic/diastolic BP often < 140/90 mm Hg (or lower depending on comorbidities) [29].

Glycemic control if diabetic

- HbA1c target close to 7% (or tailored as per patient tolerance).
- Antiplatelet therapy, usually aspirin, for secondary prevention of event (MI, stroke).
- ACE inhibitors / ARBs in appropriate patients (e.g. diabetes, hypertension, reduced LV function) for both blood pressure control and potential prognostic benefit.

These measures aim to reduce risk of myocardial Infarction, death, need for revascularization.

3. Pharmacological Therapy

Pharmacologic treatment of angina has two overlapping goals:

1. Symptom relief: reduce frequency/severity of angina attacks, improve exercise tolerance and quality of life.
2. Prognosis improvement / prevention of adverse cardiovascular events: prevent MI, reduce mortality, prevent progression of CAD.

Treatment is stratified into first-line and second-line (or alternative) agents; combinations are used if monotherapy is insufficient. Guidance suggests choice based on comorbidities, contraindications, side-effect profiles, cost, patient preference [30].

3a. First-line Antianginal Agents

These are typically used first unless contraindicated:

β-blockers

- Reduce heart rate, contractility, hence reduce myocardial oxygen demand. Especially helpful if the heart rate is elevated, or in patients with post-infarct situation or reduced left ventricular function.

Calcium-channel blockers (CCBs)

- Both dihydropyridine types (e.g. amlodipine, nifedipine) and non-dihydropyridine types (e.g. verapamil, diltiazem). These reduce afterload, coronary vascular resistance, and some reduce heart rate (non-DHP types).

Short-acting nitrates (e.g. sublingual nitroglycerin)

- For acute relief of angina attacks. They act quickly to dilate coronary vessels and reduce preload.

3b. Second-line / Alternative Agents

Used when first-line agents are not tolerated, contraindicated, or insufficient symptom control:

Long-Acting Nitrates

Ivabradine: reduces heart rate via sinus node modulation; useful if β-blockers are not enough or in specific heart rate targets.

- **Nicorandil:** has both nitrate-like and potassium channel opener effects.
- **Ranolazine:** works by altering the late inward sodium current, reducing ischemia; may improve symptoms without large changes in heart rate or blood pressure.
- **Trimetazidine:** a metabolic modulator; shifts myocardial energy substrate to more efficient ones so that under ischemia less damage / symptoms. Does not primarily act by hemodynamic changes [31].

3c. Combination Therapy

- If one first-line agent alone does not give acceptable symptom relief, combining a β-blocker + CCB or β-blocker + long-acting nitrate or other suitable combinations is often used.
- When two drugs are not enough, in more severe cases or when revascularization is not feasible or pending, triple therapy (adding a second-line agent) may be considered.

3d. Acute Symptom Treatment

- **Immediate relief:** short-acting nitrates (sublingual) at onset of angina.
- If pain persists after a few minutes, repeat dose, call for emergency help if still not relieved.

4. Tailoring Treatment to Co morbidities and Patient Factors

Choice among agents depends heavily on patients other medical conditions:

- **If hypertension present:** use agents that also lower BP (β-blockers, dihydropyridine CCBs) first.
- **If high heart rate:** β-blockers, non-DHP CCBs, ivabradine are preferred. Avoid agents that increase HR via reflex.
- **If low blood pressure:** choose agents with minimal BP-lowering effect (e.g. ranolazine, trimetazidine) or careful titration.
- **Left ventricular dysfunction / heart failure:** β-blockers are strongly beneficial; careful CCB use; avoid non-DHP types if LV function severely reduced.
- **If asthma or COPD:** cardio-selective β-blockers or avoid β-blockers; prefer CCBs or other agents.
- **Cost, availability, patient preferences** (side effects, pill burden) also play a role [32].

Diagnosis

- **Electrocardiography (ECG)**
  Helps in identifying ischemic alterations in the heart's electrical activity.
- **Exercise (Treadmill) Stress Test**

Assesses cardiac performance and oxygen demand during physical exertion.

- **Laboratory Investigations**
  Includes cardiac biomarkers, complete blood count (CBC), and metabolic profile to rule out contributing factors.
- **Imaging Studies**
  Chest X-ray, echocardiogram, CT scan, or MRI provide structural and functional details of the heart.
- **Coronary Angiography**
  Consider the gold standard to detect and measure the extent of coronary artery narrowing.
- **Clinical Examination**
  Physical assessment aids in supporting the diagnostic impression [33].

Treatment: Pharmacological Approaches

A. Beta-Blockers (BBs)

Beta-adrenergic blocking agents remain the cornerstone of therapy for patients with stable ischemic heart disease (SIHD) presenting with angina. They act by antagonizing β-adrenergic receptors, thereby reducing heart rate, myocardial contractility, and blood pressure. This ultimately decreases myocardial oxygen demand, while improving diastolic perfusion time and coronary blood flow to ischemic regions. Clinical practice guidelines strongly recommend β-blockers as the first-line treatment in patients with angina pectoris, particularly those with a prior history of myocardial infarction or left ventricular dysfunction [34].

B. Sinus Node Inhibitors

When β-blockers are contraindicated or not well tolerated, sinus node inhibitors such as ivabradine are considered. Ivabradine selectively inhibits the If (funny) current in the sinoatrial node, which slows heart rate without significantly affecting blood pressure, myocardial contractility, or atrioventricular conduction. This targeted mechanism allows for a reduction in myocardial oxygen consumption while preserving ventricular function. Current guidelines endorse ivabradine in patients with symptomatic angina who remain inadequately controlled on optimal β-blocker therapy or are intolerant to them.[35]

C. Amiodarone and Dronedarone

Amiodarone, a multi-channel blocker with class III antiarrhythmic properties, exerts anti-anginal effects primarily through heart rate reduction and vasodilatory actions. Its derivative, dronedarone, shares similar electrophysiological characteristics but lacks the iodine component, thereby reducing thyroid- and pulmonary-related side effects. Evidence from randomized, double-blind, placebo-controlled studies has shown that amiodarone can significantly improve exercise tolerance and symptoms in patients with advanced New York Heart Association (NYHA) class III angina. Although dronedarone is primarily used in atrial fibrillation, some studies suggest that it may reduce hospitalization rates and cardiovascular events in elderly patients with coexisting ischemic heart disease. However, its role as an anti-anginal agent remains less well established compared to β-blockers and ivabradine [36].

Table 03: Lifestyle Modifications of Cardiovascular Diseases.

Aspect	Life style modifications	Explanation
1. Diet control	Adopt a heart-healthy diet	Include fruits, vegetables, low fat, lean proteins (fish, chicken), and avoid saturated fats, salt and fried foods.
2. Regular Physical activity	Engage in moderate exercise (30min/day, 5days/week)	Walking, cycling, or swimming improves heart function and prevents further blockage.
3. Smoking cessation	Avoid smoking or tobacco in any form	Smoking narrows arteries, increases angina attacks, and worsen CCF.
4. Alcohol Limitation	Limit or avoid alcohol intake	Excess alcohol can damage heart muscles and worsen heart failure symptoms.
5. Stress Management	Practice yoga, meditation, or deep breathing	Stress increases heart rate and blood pressure, leading to angina episodes.
6. Weight Management	Maintain a healthy body weight	Reduces strain on the heart, improves blood flow, and decreases risk of heart failure progression.
7. Medication Adherence	Take Prescribed medicines regularly	Follow the doctor's instructions carefully for angina or CCF drugs (like beta-blockers, nitrates, diuretics etc.).
8. Gender & Age Considerations	Women: Focus on hormonal balance, weight, and stress control. Older adults: Light physical activity and regular monitoring.	Tailored approach helps manage risk better based on gender and age group.

Conclusion

The present study highlights the significant relationship between angina pectoris and congestive cardiac failure, particularly when analyzed across different gender and age groups. Findings indicate that the prevalence of angina increases with advancing age, with men showing higher susceptibility at younger ages and women experiencing greater burden post-menopause. The co-existence of angina and CCF not only worsens clinical outcomes but also increases the risk of hospitalization, reduced functional capacity, and mortality. These observations emphasize the importance of early screening, accurate diagnosis, and timely management of angina to prevent its progression into cardiac failure. Lifestyle modification, pharmacological interventions, and present education remain key strategies in reducing disease burden. Moreover, age and gender specific risk profiling may provide valuable insights for clinicians to design more effective preventive and therapeutic measures. Overall, this project underlines the need for continuous research and awareness regarding the association of angina pectoris with CCF, which will ultimately contribute to improved patient outcomes and better public health strategies.

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Conflicts of Interest

The authors declare no conflicts of interest.

Author Contribution

Both are contributed equally

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Ethical Considerations and Inform Consent

Not Applicable

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