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Coronary Artery Disease Prevention

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Continuing Education Activity

In the United States, cardiovascular **disease** is one of the leading causes of mortality, morbidity, and increased healthcare cost. According to the AHA, in 2018, 16.5 million people over 20 years of age have **coronary** artery **disease**. Mortality rates due to ischemic **heart** diseases are decreasing in developed countries like the United States and the United Kingdom. This decline is attributable to increased awareness of **disease** prevention. Guidelines are available for primary and secondary prevention and are actively implemented; however, there are still some barriers in different aspects of implementation — awareness among healthcare providers critical and part of an ongoing process to improve cardiovascular mortality and morbidity. This activity addresses the evaluation and treatment of **coronary** artery **disease** and highlights the role of the healthcare team in managing patients with this condition.

Objectives:

- Summarize the risk factors associated with **coronary** artery diseases.
- Outline the pathophysiology of **coronary** artery diseases.
- Explain the modifiable factors for **coronary** artery **disease**.
- Review the role of the interprofessional team in improving outcomes for patients either with or at risk for **coronary** artery **disease**.

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Introduction

Coronary artery **disease** (CAD) is the most common form of **heart disease**. It is the result of atheromatous changes in the vessels supplying the **heart**. CAD is used to describe a range of clinical disorders from asymptomatic atherosclerosis and stable angina to acute **coronary** syndrome (unstable angina, NSTEMI, STEMI). In the US, it is still one of the leading causes of mortality. Initial evaluation of risk factors is the first step in the prevention of **coronary** artery diseases.[1]

Etiology

Risk factors of coronary artery disease are as follows[2][3]:

Non-Modifiable

- Age
- Gender
- Race
- Family history

Modifiable

- Type 2 diabetes mellitus
- Hypertension
- Smoking
- Dyslipidemia
- Chronic kidney **disease**
- Obesity and metabolic syndrome

Risk enhancing factors

- Premature menopause
- Preeclampsia
- Chronic inflammatory conditions (for example rheumatoid arthritis, HIV, psoriasis)
- Persistently elevated triglycerides

Epidemiology

Coronary artery **disease** is a leading cause of death worldwide. The World Health Organization (WHO) reported that ischemic **heart disease** was responsible for approximately nine million deaths in 2016.[3] Developed and developing countries show opposite trends in mortality due to CAD. In developed countries like the U.S. and the UK, mortality rates due to ischemic **heart** diseases are decreasing. Nevertheless, according to AHA, 16.5 million people older than 20 in the U.S. had **coronary** artery **disease** in 2018, and 55% of them were males.[4] The status of CAD in developing countries is worse with increasing trends of mortality.[3] Increased implementation of primary and secondary prevention methods of cardiovascular is responsible for the decline in mortality in developed countries. Primary prevention methods are intended to prevent cardiovascular events with high risks but no previous history. Secondary prevention methods are therapies that prevent any further cardiac damage to those with a history of CAD.

Pathophysiology

The beginning of **coronary** artery **disease** is generally attributed to a chronic inflammatory process, from the earliest formation of the fatty streak to the final formation of a fibrous-atheroma. This process gets incited by endothelial dysfunction. It can result from one or a combination of multiple of the following factors – sheer stress, oxidation injury due to free radicals, genetic alterations, chronic infection, or hypercholesterolemia. These inciting factors are thought to be due to uncontrolled hypertension, diabetes, smoking, and certain genetic factors.

After initial insult, the endothelium releases inflammatory cytokines and becomes highly receptive to the leukocytes, especially monocytes, and platelets. The monocytes attracted to the endothelium, mature to become macrophages and engulf the oxidized LDL particles to form lipid-laden foamy macrophages. Due to continuous inflammation, there is also migration and proliferation of smooth muscles, which finally form a fibrous atheroma. The core of the fibrous atheroma consists of oxidized lipid with macrophages and necrotic tissue that is surrounded by collagen-rich matrix and proliferative smooth muscles.

The continuous process of chronic inflammation and atheroma formation causes the arterial wall to thicken. This thickened arterial wall leads to a compensatory dilation of the artery with no change in the lumen (also called “remodeling”). However, after a prolonged period of remodeling, the arterial wall can no longer dilate. Finally, the lesion alters the blood by intruding in the lumen and decreasing the area.[1][5][6][7][8]

Evolving plaque Complications[1][5][6][7][8]:

- Thin cap or large lipid atheroma (greater than 40% of plaque volume) increases the chances of **coronary** plaque disruption followed by thrombotic occlusion plaque
- Thrombus in the lumen caused by endothelial erosion

- Intra-plaque hemorrhage causing expansion of plaque volume
- Lumen thrombus caused by protruding calcific nodules from complex fibro–lipid–calcific architecture and calcified shell

History and Physical

Symptoms of **coronary** artery **disease** presentations can vary from asymptomatic, stable chest pain (stable angina), and acute **coronary** syndrome (unstable angina, NSTEMI, and STEMI) to sudden cardiac death. Chest pain seen with stable angina is often mid-sternal, squeezing in quality, associated with a feeling of constriction or anxiety, radiating to the arms, neck, jaw, back or upper abdomen. These symptoms worsen with exertion (physical or emotional) because of the increased oxygen demand and improve with rest because of the decreased oxygen demand. The level of physical activity at which the symptoms start to occur is called the "angina threshold."

Unstable angina is defined with similar chest pain as stable angina but is present even at rest with normal EKG, and normal cardiac enzymes. NSTEMI presents as chest pain at rest with elevated cardiac enzymes, and normal EKG or with ST-segment depressions. Similarly, STEMI presents with chest pain with nausea/vomiting, diaphoresis with ST-segment elevation in EKG, and elevated cardiac enzymes. Patients may present with absolutely no symptom of chest pain or angina, and the initial presentation could be an abnormal electrocardiogram or echocardiogram ordered for other reasons. It is necessary for proper evaluation of the history of present illness in these patients, including the past medical history, social and family history that includes the risk factors for **coronary** artery **disease**.[1][9]

Evaluation

History taking is the most valuable technique to differentiate among different causes of chest discomfort. A thorough history and physical exam is the hallmark for the diagnosis of **coronary** artery **disease**. For example, a history pertinent for typical anginal symptoms, decreased exercise tolerance, syncope, pre-syncope, orthopnea, or paroxysmal nocturnal dyspnea should prompt the clinician to get a detailed history and physical exam and diagnostic tests (e.g., electrocardiogram and cardiac enzymes for patients who present with chest pain).

Characteristic features of stable angina are chest pain that worsens with emotional and physical exertion, cold weather, and meals. The patient experiences pain relief from rest and nitrates. Features of unstable angina include pain at rest that lasts over 30 minutes and does not relieve with sublingual nitroglycerine. Pain associated with sweating, nausea, and vomiting may suggest myocardial infarction (MI). Patients may present with complications of MI such as **heart** failure, ventricular septal defect (VSD), harsh systolic murmur, or papillary muscle rupture.

EKG is a tool for assessing patients who are either stable or in emergent situations. The presence of specific EKG findings should prompt referral, when necessary, for thrombolysis. However, EKG is not absolute as ST-segment elevations are present in only 50% of MIs confirmed by cardiac enzymes. Echocardiogram, stress testing, cardiac CT, and angiography are also other available options. Patient’s age, risk factors, and the symptoms will determine whether if any additional testing is necessary.[10][11][12]

Treatment / Management

Treatment of **coronary** artery **disease** depends upon the symptoms and clinical presentation of the patient. It can range from medical management for controlling angina symptoms to acute intervention by **coronary** artery stenting.[13] Patients, who present with unstable angina and NSTEMI, require urgent evaluation - the clinician should initiate immediate pain control with morphine and nitroglycerin, as well as anticoagulation with heparin, and antiplatelet agents (aspirin or clopidogrel). Furthermore, if the patients present with STEMI, urgent revascularization is necessary, in addition to the initial stabilization. Also, for patients with unstable angina and NSTEMI with significant risk factors, the decision should be made on whether cardiac catheterization is warranted based on risk assessment.

On the other end of the spectrum, for patients who present as an outpatient with stable angina, the main goals of treatment are to help relieve the symptoms of the **disease** and prevent further complications associated with **coronary** artery **disease**. For pain, antianginals like nitroglycerin, beta-blockers, and calcium channel blockers are therapeutic options. Beta-blockers have negative chronotropic and ionotropic effects and reduce the **heart**'s workload by decreasing the oxygen demand and reducing the blood pressure. Further, antithrombotic therapy (aspirin or

clopidogrel) is used to decrease the aggregation of platelets, which is a key pathology of CAD. ACE inhibitors and angiotensin II receptor antagonists are blood-pressure-lowering drugs that relax the blood vessels and causes the blood pressure to drop. This drop reduces the pressure in the **heart**'s chambers. For a patient with CAD who have had **heart** failure, these drugs lower the risk of complications and thus, improves life expectancy.[14][15] Prevention plays a major role in the management of **coronary** artery **disease**.

Primary Prevention [13]

Early recognition of risk factors and primary prevention have significantly decreased the morbidity and mortality associated with CAD. The risk assessment and preventive therapy is a combined discussion and decision that should take place between the patient and their physician. ACC/AHA 2019 recommends risk stratification by calculating 10-year-ASCVD risk, using the Pooled Cohort Equation (PCE). In adults aged between 40 and 75 years without any significant CAD history, a 10-year risk should be part of every visit. For adults between 20 and 39 years, it is reasonable to asses 10-year ASCVD risk at least every four to six years. Based on ASCVD scores, the 10-year risk can classify as low risk (lower than 5%), borderline risk (5 to 7.5%), intermediate-risk (7.5 to 20%) and or high risk (greater than 20%). Lifestyle modification with diet, exercise, and smoking cessation is crucial to reduce cardiovascular risk factors. Further control of hypertension, diabetes, and hyperlipidemia is essential to reduce the risk of CAD.

In 2020 the American **Heart** Association developed a health prescription called "Life's Simple 7" designed to improve overall cardiovascular health and reduce the risk of cardiovascular **disease**. These recommendations are outlined below.

Diet

Diet is a significant contributing factor to reduce the risk of **coronary** artery **disease**. According to ACC/AHA 2019, the plant-based Mediterranean diet (high in vegetables, fruits, legumes, nuts, whole grains, and fish) is highly recommended. Replacing saturated fats with dietary monosaturated and polyunsaturated fats are found to be beneficial to reduce cardiovascular risks. Besides, dietary sodium reduction is found to have reduced BP and decreased risk for cardiovascular events, according to the DASH trial. On the other side, sugar-sweetened and artificial sweeteners have shown to increase the risk of diabetes, leading to an increased 10-year ASCVD risk. Moreover, increased intake of trans fat also correlates with increased ASCVD risk.

Exercise, physical activity, and weight loss

Physical activity is also equally beneficial for CAD risk reduction. At least 150 minutes per week of moderate-intensity activities and greater than 75 minutes a week of vigorous-intensity physical activities are helpful. Moderate activities include brisk walking (2.4 to 4 mph), biking (5 to 9 mph), active yoga, and recreational swimming, whereas vigorous activities include jogging/running, biking (greater than 10 mph), playing tennis, swimming, etc.

Individuals are diagnosed as overweight when body mass index (BMI) is between 25 to 29.8 kg/m^2 and obese when BMI greater than or equal to 30 kg/m^2. Conditions of both overweight and obesity increase the ASCVD risk compared to normal weight. Recommentations include annual calculation of BMI, and lifestyle modification, including calorie restriction and weight loss, based on the BMI values. Weight loss has consistently shown to improve the ASCVD risk profile. Strong recommendations include high levels of physical activities (200 to 300 minutes per week), low-calorie diet (800 to 1500 kcal/day), and if possible, weight-loss maintenance programs.

Tobacco Use

Using tobacco is among the leading causes of preventable deaths in the U.S. and also a significant risk factor of CAD. Tobacco usage requires assessment in all adults at every primary care visit. Three to ten minutes of status assessment with counseling to quit at each clinical encounter should take place. For anyone who uses tobacco, healthcare providers should provide assistance on readiness to quit, with behavioral and pharmacological interventions. Nicotine replacement therapy is available in patches, gums, lozenge, nasal spray, and oral inhalers. Other medications, such as bupropion and varenicline are also used to assist tobacco cessation.

Hypertension management

The current definition of hypertension is systolic blood pressure (BP) greater than or equal to 130 mm Hg and diastolic blood pressure (BP) greater than or equal to 80 mm Hg. Stage 1 hypertension defines as systolic BP between 130 and 139 and diastolic BP between 80 and 89. Similarly, stage 2 hypertension is when systolic BP is greater than or equal to 140 and diastolic BP is greater than or equal to 90.

The use of a 10-year ASCVD risk score for blood pressure is used to guide the therapy for hypertension management. Initially, non-pharmacological measures with diet and exercise are recommended in the adult with stage 1 hypertension (130 to 139/80 to 89) with a 10-year ASCVD risk under 10%. However, if the 10-year ASCVD risk is over 10% with stage-1 hypertension, pharmacological management along with non-pharmacological measures are the recommendaed approach. For stage 2 hypertension, the clinician should initiate pharmacological therapy, along with non-pharmacological interventions.

Non-pharmacological interventions are lifestyle modifications that include changes in diet and exercise. A **heart**-healthy diet like the DASH diet pattern that is rich in fruits, vegetables, whole grains, and low-fat dairy products with reduced content of saturated fat would lower the systolic BP by approximately 11 mm Hg. Similarly, reducing dietary sodium by 1000 to 1500 mg/day and increasing dietary potassium to 3500 to 5000 mg/dl by consuming potassium-rich food can lower the systolic BP by approximately 5 points, respectively.

Weight loss also has a positive impact on lowering blood pressure. Reduction of one kg in body weight for overweight adults can reduce the systolic BP by one mm Hg. Furthermore, physical activities such as aerobic exercises of 90 to 150 minutes per week and increasing **heart** rate reserve to 65% to 75% would lower the systolic BP by 5 to 8 mmHg. For those who prefer dynamic resistance training, a weekly total of 90 to 150 minutes of six exercises, three sets and 10 repetitions per exercises, would lower the systolic BP by approximately 5 to 8 mm Hg. Other forms of exercises like isometric resistance (e.g., four repetitions of 2-minute handgrip with 1-minute rest in between), 30% to 40% maximum voluntary contraction and three sessions per week for 8 to 10 weeks would lower the systolic BP by approximately 4 mmHg.

Reducing alcohol consumption also has blood-pressure-lowering effects. Current recommendations are for men to drink no more than two drinks per day and for women to drink no more than one drink per day. This would help lower the systolic BP by approximately 4 mm Hg.

Type 2 Diabetes mellitus (DM)

Type 2 diabetes mellitus is categorized when hemoglobin A1c (HbA1c) is greater than 6.5 %. Type 2 diabetes mellitus is strongly related to a sedentary lifestyle, dietary habits, physical activity, and body weight. Among 12 % of the U.S. adults having diabetes, 90 to 95 % have type 2 diabetes mellitus. It is one of the major cardiovascular risk factors.

Initially, dietary modifications using a **heart**-healthy diet (like Mediterranean and DASH diet as mentioned above) and physical activities (at least 150 minutes/week of moderate to vigorous) are encouraged. Additionally, weight loss is recommended if the individual is overweight or obese. Metformin can also be considered as first-line therapy for type 2 DM to improve the glycemic index and reduce cardiovascular risk. If the HbA1c remains over 7% with lifestyle modification and metformin, then the addition of SGLT–2 inhibitors or GLP-1 agonists can be considered as they have shown to reduce the ASCVD risk.

Statin use

A moderate-intensity statin is recommended to any patient aged between 40 to 75 years with type 2 DM, regardless of cholesterol levels and ASCVD risk. In this age group, for patients with low-density lipoprotein (LDL) exceeding 190, high or maximum tolerable intensity statin is recommended. In addition to diabetes and LDL values, the 10-year ASCVD risk should guide the use of statins. If the 10-year ASCVD risk is high (greater than 20%), the maximum tolerated statin should be used to reduce LDL by more than 50%. For intermediate-risk (7.5% to 20%), a moderate-intensity statin is recommended to reduce LDL by 30% or more. Discussion about initiating statin should begin with patients who are at borderline risk (5% to 7.5%), and there are risk enhancing factors.

Coronary artery calcium (CAC) scoring should be used to further guide the decision, in case a decision cannot be reached based on a 10-year ASCVD risk assessment, especially for patients at borderline or intermediate risk. If the CAC score is less than 0, with no risk conditions, then holding statin therapy is reasonable and if the CAC score is

above 100, then starting statin therapy is reasonable. A CAC score 1 to 99 favors the use of statin, especially if the patient is aged 55 or above.

For patients aged between 20 to 39 years, ACC/AHA recommends establishing lifetime risk of CAD to encourage lifestyle modification. Treatment with a statin should be a consideration if there is a significant family history of premature ASCVD and LDL is greater than or equal to 160. For patients older than 75 years, discussions between patient and physician, assessment of risk factors, and side effects should be all looked at for initiation or continuation of statin therapy.

Aspirin

Aspirin is anti-thrombotic and reduces the risk of cardiovascular **disease** by irreversibly binding with the platelets. However, the use of low-dose aspirin (75 to 100 mg orally) for primary prevention is getting more controversial recently. Previous U.S. guidelines recommended aspirin for primary prevention in the settings of significant ASCVD risk factors. However, according to the recent ACC/AHA 2019 guidelines, the use of aspirin may be considered in patients (40 to 70 years old) with significant risk factors for cardiovascular **disease** and no risk of bleeding. The strength of recommendation is comparatively weaker, and a thorough evaluation with risk versus benefit assessment is necessary. These guidelines for primary preventions should undergo evaluation based on the individual patient basis, and risk versus analysis should always be based on the physician's best clinical judgments.[13]

Secondary Prevention[16]

Secondary prevention is the therapy to prevent further damage and progression of the **disease** after the patient has a diagnosis of cardiovascular **disease**, including **coronary** artery, cerebrovascular, or peripheral arterial **disease**. The guidelines are somewhat similar to that of primary prevention, including diet, exercises, and smoking cessation as discussed above.

A large part of secondary prevention also includes pharmacological therapy. In contrast to primary prevention, anti-thrombotic therapy (low dose aspirin) is strongly recommended unless contraindicated. The daily strength of 75 mg of clopidogrel is recommended for people who are intolerant or allergic to aspirin. Blood pressure should be lowered in all patients with **coronary** artery **disease** and stage 1 hypertension using both non- pharmacological and pharmacological therapies. Metformin remains the first-line therapy in diabetic patients for secondary prevention. High intensity or maximally tolerated statin is part of secondary prevention, independent of the lipid levels in as long as the patient can tolerate, and the goal is to achieve LDL less than 70.[16]

Differential Diagnosis

Differential diagnosis is made based on the presenting signs and symptoms. Other conditions presenting as chest pain and mimicking CAD could be musculoskeletal pains, pleural inflammation, diaphragmatic symptoms, GERD, dysphagia, panic attacks, and neuralgia from neck and shoulder. A careful initial evaluation should be done with these differentials in mind as other non-cardiac causes, as mentioned above, could be the reason for the patient's presentation.

Prognosis

Prognosis depends on adherence to the prevention program. Prevention of **coronary** artery **disease** best starts with the initial evaluation of risk factors to prevent or halt the **disease** progression. Prognosis of **coronary** artery **disease** is discussed in detail other topics.

Complications

Prevention is intended to prevent a cardiovascular complication due to the chronic reduction of blood flood to the **heart** muscle cells. Arrhythmias, chest pain, **heart** attack, related arterial diseases, sudden death, **heart** failure are complications of **coronary** artery **disease** discussed in detail in other topics.

Deterrence and Patient Education

Patient education programs are fundamental to the prevention of cardiovascular diseases and their complications. They are designed to allow people with chronic conditions to actively participate in managing their condition, promoting self-care behavior, and modifying risk factors. The goals are to improve health outcomes and decrease the incidence of complications for patients by supporting, not replacing, medical care. Educational interventions in cardiac care have been shown to increase physical activity, healthier dietary habits, and smoking cessation. The delivery of patient education programs can vary substantially: locations can be in clinics, classrooms, or homes; the target could be an individual or groups, and content could be tailored or generic. Common topics include nutrition, exercise, risk factor modification, psychosocial well-being, and medications.[17]

Enhancing Healthcare Team Outcomes

Coronary artery **disease** is a multifaceted health problem. It comprises of modifiable and non-modifiable risk factors, physical and emotional imbalances, and private and social relationships. Therefore, it needs an interprofessional approach, mainly an efficacious interprofessional team. Prevention of cardiovascular diseases might provide a general framework for improving follow-up for patients with chronic diseases by targeting many domains for quality improvement: e.g., self-management support, decision support, clinical information systems, community resources and policies, and organization of health care. Interactive approaches have to be used to engage clinicians in simultaneously developing and implementing the interventions.

The optimal approach to managing **coronary** artery **disease** utilizes an interprofessional healthcare team approach. Doctors, nurses, physiotherapists, dietitians, physical trainers, psychologists, patients, and family members should all have involvement.[18] The family doctor and/or cardiologist will direct the overall plan, but other members of the team must make their unique contributions. Cardiac specialty pharmacists will be the experts on the medications used to address **coronary** artery **disease**, and its underlying conditions are comorbidities. They will monitor agent selection, dosing, and perform medication reconciliation to preclude drug interactions, reporting any adverse findings to the healthcare team. They are also involved in patient counseling. Nurses are crucial to this process, particularly those with cardiovascular specialty training. They can monitor patient compliance, treatment progression, provide counseling, and answer questions from the patient and their family, as well as administering medication for inpatients. Nursing also acts as a direct extension of the treating clinician on a properly functioning healthcare team. As mentioned above, dieticians or nutritionists, as well as exercise trainers may also play a role in the management of **coronary** artery **disease** and associated comorbidities, and when they are involved, they also need to be plugged in to the healthcare team structure, so they can operate from the same paradigm for the patient's case. Only through a fully collaborative, interprofessional team approach can **coronary** artery **disease** cases achieve optimal results for the patient. [Level 5]

Review Questions

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