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Education

Case Management for Coronary Artery Disease (CAD)

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Disclosures

Delbert Escher, M.D. has no relevant financial relationships to disclose.

Timothy Smith, M.D. has no relevant financial relationships to disclose.

Agenda

- Etiology of CAD
- Risk factors for CAD
- Clinical features
- Primary prevention
- Stable ischemic disease
- Unstable Angina / Myocardial Infraction (MI)
- Treatment
- Cardiac complications
- Revascularization
- Secondary prevention
- Mental health factors
- Clinical cases

Objectives

At the end of this activity, participants should be able to:

- Explore clinical features, risk factors, screening, prevention, evaluation and treatment of Coronary Artery Disease (CAD)
- State the relationship between physical and mental health and its impact on CAD
- Discuss the importance of a multidisciplinary approach when treating CAD
- Identify optimal clinical management strategies for CAD through case study examples

Etiology of CAD

Coronary Artery Disease (CAD) occurs when plaque builds up in the coronary arteries. These arteries supply oxygen-rich blood to your heart. Plaque narrows the coronary arteries and reduces blood flow to the myocardium (heart muscle).

Plaque may:

- Limit blood flow through the coronary artery causing angina, or
- Block flow through the coronary artery causing a myocardial infarction (MI), or
- Rupture – a blood clot may develop over the plaque occluding the coronary artery resulting in a myocardial infarction

https://www.nhlbi.nih.gov/health/health-topics/topics/atherosclerosis

CAD Risk Factors

Major cardiac risk factors:

- Hypertension (HTN)
- Smoking
- Diabetes
- Family history of CAD
- Elevated low-density lipoprotein (LDL)
- Earlier onset in males by 6-10 years as compared to females
- Obesity
- Increased risk with advancing age
- Physical inactivity

Presentation of CAD

- **Asymptomatic:** CAD with non-obstructing/flow limiting CAD
- **Angina (stable/unstable):** Cardiac demand exceeds oxygen supply, exertional chest pain/chest pressure/anginal equivalents
- **Sudden cardiac death:** Initial coronary event in 15% of patients with CAD
- **Myocardial infarction:** STEMI (ST segment elevation myocardial infarction) or NSTEMI (non-ST segment elevation myocardial infarction)
- **Silent myocardial infarction and ischemia:** Detected by stress test or ambulatory ECG monitoring

Primary Prevention of CAD

Cardiovascular disease is the leading cause of death in the United States.*

Addressing the major modifiable cardiac risk factors:

- Smoking: One of the most significant modifiable cardiac risk factors
- Obesity: Weight loss (obesity increases risk for HTN, dyslipidemia and DM)
- Physical inactivity: Physical activity decreases the risk of Coronary Artery Disease (CAD)
- Dyslipidemia: Exercise, prudent diet, weight loss as appropriate, potential Statin therapy
- HTN:
  - JNC8 BP goal is < 140/90 for ages 18-59 and < 150/90 for ages 60+; high risk patients may have a lower goal
  - 2017 ACHA guidelines: BP goal < 130/80
- Diet: Change to a healthy diet to include fruits and vegetables, fiber, low sodium; avoid saturated fats and trans fatty acids; increase omega-3 fatty acids
- Diabetes Mellitus: Glycemic control, individualized treatment plan, usually HbA1C goal (6-8%) < 65, diabetic action plan and if provider recommended SMBG goal range

*www.cdc.gov, Fast Stats leading-causes-of-death

Stable Ischemic Heart Disease

- Diagnosis is based upon:
  - Clinical history
  - Physical exam
  - Diagnostic testing (ECG and stress test)
  - Angiography may be recommended for atypical symptoms or if stress testing is non-diagnostic

- Predictable exertional chest pain/chest pressure/anginal equivalent

- Relieved by medication – Nitroglycerin

Stable Ischemic Heart Disease: Medical Therapy

Anti-anginal Therapy:

- **Beta Blockers** (e.g. Propranolol, Metoprolol, Carvedilol, Atenolol)
  - Recommended as first-line therapy
    - Decreases heart rate and blood pressure
    - Prevents re-infarction and improves survival after MI
- **Calcium Channel Blockers** (e.g. Amlodipine, Felodipine, Diltiazem, Verapamil)
  - Combined with beta blockers, as needed
  - Recommended if intolerant or contraindication to beta blockers
- **Nitrates**
  - Short acting: Sublingual for treatment of acute angina (e.g. short acting sublingual Nitroglycerin, spray)
  - Long acting: Controls stable angina – improves time to onset of angina; exercise tolerance and ECG changes during exercise testing (e.g. Isosorbide Dinitrate, Isosorbide Mononitrate, transdermal patches)

    NOTE: *PDE5 inhibitors [e.g. Sildenafil (Viagra®), Tadalafil (Cialis®), Vardenafil (Levitra®, Staxyn®) and Avanafil (Stendra®)] are contraindicated with nitrates*
- **Ranolazine** (Ranexa®)
- **Antiplatelet Therapy**
  - Aspirin, Clopidogrel (Plavix®), Ticagrelor (Brilinta®), Prasugrel (Effient®)
- **Intensive Statin Therapy**
  - Atorvastatin (Lipitor®) 40–80 mg/day or Rosuvastatin (Crestor®) 20–40 mg/day

Stable Ischemic Heart Disease: Management

Revascularization:

- Percutaneous Coronary Intervention (PCI) vs Coronary Artery Bypass Grafting (CABG)

- Decision is based upon factors such as:
  - Coronary anatomy
  - Left ventricular function
  - Diabetes

Stable Ischemic Heart Disease: Coronary Angiography

Indications for Coronary Angiography:

- Angina that significantly interferes with patient's lifestyle despite maximal medical therapy
- Patients with high risk criteria based upon noninvasive testing
- Establish diagnosis for atypical symptoms and equivocal findings on stress test
- Depressed left ventricular ejection fraction and moderate risk criteria on noninvasive testing
- Inconclusive prognostic information after noninvasive testing or in patients for whom noninvasive testing is contraindicated or inadequate

Stable Ischemic Heart Disease: Revascularization

Indications for Revascularization:

- PCI
  - Meets criteria for CABG but is not a candidate for CABG
  - Relief of angina not adequately controlled by optimized medical therapy

- CABG
  - Left main coronary artery disease or equivalent
  - Triple vessel CAD, particularly with Ejection Fraction (EF) < 40 percent
  - Two vessel CAD with more than 75% stenosis in left anterior descending (LAD) proximal to first major septal artery

Unstable Angina/Acute Myocardial Infarction (MI)

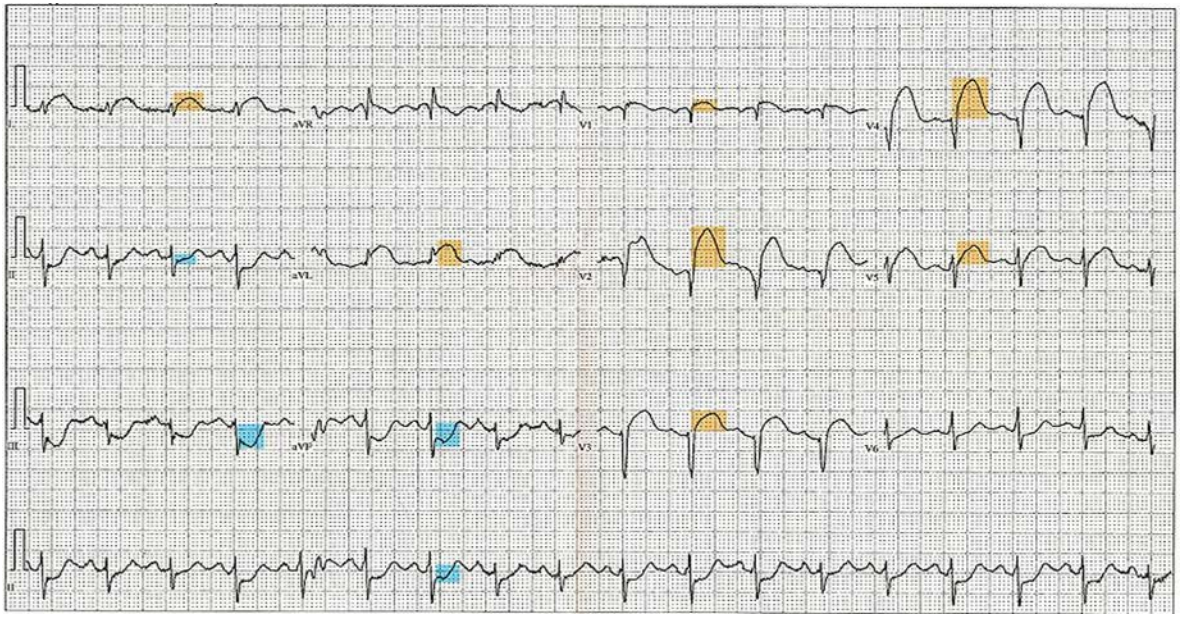
Angina that is occurring in a stable pattern and has been well controlled with medication may suddenly, without warning escalate in its clinical expression and morph into a much more serious clinical picture such as manifesting as unstable angina or sudden plaque rupture resulting in a MI.

Warning signs for this escalation include:

- Rest angina
- New onset angina limiting physical activity
- Increased angina frequency, duration or occurring with less exertion
- Angina unrelieved by nitroglycerin lasting more than 5 minutes
- Radiating pain (arm, jaw, back, shoulder, neck, upper abdomen)
- Atypical angina symptoms:
  - Breast pain (women)
  - Perceived indigestion
  - Fatigue, shortness of breath
  - Nausea/vomiting, weakness, sweating
  - Rapid/irregular heartbeat
  - Syncope

Acute Coronary Syndrome

- Unstable Angina, may also be called Intermediate Coronary Syndrome
- Non-ST-Elevation Myocardial Infarction
- ST-Elevation Myocardial Infarction (STEMI)



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ACS: Unstable Angina, NSTEMI and STEMI

- **Unstable Angina**: Cardiac biomarkers are not elevated as with NSTEMI and STEMI

- **NSTEMI**: ECG changes suggesting partial thickness damage of heart muscle
  - Short-term (in hospital/one month) mortality is lower in NSTEMI
  - Re-infarction rate (further heart attack) is higher in NSTEMI after hospital discharge compared to STEMI
  - Long-term mortality is similar or higher in NSTEMI compared to STEMI

- **STEMI**: ECG changes suggesting full thickness damage of heart muscle
  - Increased risk for complications like cardiogenic shock, left ventricular failure, severe mitral regurgitation due to papillary muscle rupture, cardiac tamponade due to ventricular wall rupture are more in STEMI (due to full thickness heart muscle damage) than NSTEMI

Treatment of NSTEMI

- **Medical therapy to reduce ischemic pain, control hypertension and tachycardia**
  - Beta Blockers, Nitrates, Antiplatelet therapy (aspirin and platelet inhibitors), Statins, parental anticoagulation, ACE Inhibitors (ACEi) and Calcium Channel Blockers (CCB)

- **Risk stratification to identify individuals at the highest risk for further cardiac problems**
  - Thrombolysis in Myocardial Infarction (TIMI) Score (incorporates age, number of cardiac risk factors, prior coronary stenosis of ≥ 50 percent, anginal frequency, elevated cardiac biomarkers and history of aspirin use)
  - Clinical symptoms (cardiogenic shock/hemodynamic instability, severe left ventricular dysfunction, HF, new VSD, new or worsening MR and sustained ventricular arrhythmias)

- **High risk individuals should undergo immediate angiography and revascularization**

Treatment of STEMI

- **Prompt restoration of myocardial blood flow**
  - Fibrinolytic therapy if PCI is not available within 120 minutes
  - PCI (Percutaneous Coronary Intervention)
    - DES (drug eluting stent)
      - Reduced risk of restenosis
      - Increased risk of stent thrombosis and requires **longer period of dual Antiplatelet therapy**
    - Bare metal stent
      - DES cannot be implanted for technical reasons
      - Patient will not be compliant with 12 months of dual Antiplatelet therapy , scheduled non-cardiac surgery within 12 months of stenting
      - High risk of G.I. bleeding including taking oral Anticoagulant

- **CABG**
  - Infrequently performed with acute MI; indications include failure of fibrinolysis or PCI, or hemodynamically important mechanical complications

Cardiac Complications That Can Occur with MI

- **Mechanical Complications**
  - Left ventricular dysfunction: “ischemic cardiomyopathy”, reduced EF ≤ 35-40%, and CHF, wearable cardioverter-defibrillator
  - Rupture of the free wall of the ventricle
  - Rupture of the interventricular septum
  - Acute mitral regurgitation: ischemic/ruptured papillary muscle displacement, left ventricular dilation or left ventricular aneurysm

- **Pericardial Complications**
  - Acute pericarditis
  - Pericardial effusion, with or without tamponade
  - Post-cardiac injury syndrome, “Dressler's syndrome” occurs late after MI; clinical diagnosis based on symptoms: pleuritic chest pain, pericardial friction rub, malaise, fever occurs frequently and elevated sed rate or higher in NSTEMI compared to STEMI

- **Arrhythmias**
  - Atrial arrhythmias: Sinus tachycardia, atrial fibrillation or flutter, SVT, sinus bradycardia
  - Ventricular arrhythmias: VBPs, ventricular tachycardia and ventricular fibrillation

Secondary Prevention

Same as primary prevention plus:

- Intensive Statin Therapy, age < 75: Atorvastatin (Lipitor®) 40-80 mg/day or Rosuvastatin (Crestor®) 20-40mg/day
- HTN treated with Beta-Blocker and Angiotensin Converting Enzyme Inhibitor/Angiotensin Receptor Blocker (ACEi/ARB), especially if DM or metabolic syndrome
- Beta-blocker after MI: In general everyone should be treated with Beta-Blocker unless contraindicated
- ACEi/ARB especially if decreased LV function, DM
- Nitrates
- Cardiac rehabilitation program
- Avoid heavy alcohol intake
- Antiplatelet therapy: Aspirin, Clopidogrel
- Stents, dual antiplatelet therapy for 1 year: Aspirin + another Antiplatelet agent, e.g. Clopidogrel, Ticagrelor (Brilinta®) or Prasugrel (Effient®)
- Anticoagulant (triple therapy): Apixaban (Eliquis®), Dabigatran (Pradaxa®), Rivaroxaban (Xarelto®) or Warfarin (Coumadin®) for non-valvular chronic atrial fibrillation, DVT/PE; only Warfarin should be used with prosthetic mechanical heart valves

Mental Health and CAD

- 30% of post-MI patients develop depression often with co-occurring anxiety
- Studies suggest that pre-morbid mental health conditions are a risk factor for later development of CAD
- Other mental health conditions also increase risk of CAD but confounding factors lead to inconsistent findings
- Mental health conditions if untreated = worse outcomes
- Cannot firmly establish direct causation

How Depression and Anxiety Effect Heart Disease

- Increase rate of atherosclerosis
- Increase risk of unhealthy lifestyle (smoking, diet, decreased treatment compliance, decreased exercise)
- Increased cortisol levels (increase in BP and blood glucose)
- Increase in other hormones which increase resting heart rate
- Increased platelet adhesiveness (serotonin)
- Increased neuro-immunologic response

Clinical Case

TCM case: Previously healthy 54 year old male discharged after an acute MI; the patient was in the hospital for one day and discharged home

- Review essential care elements for CAD angina
- Assess discharge instructions/providers plan of care
- Value Pillars: right provider, right care, right medication, right lifestyle
- SPA (Stop, Pursue and Act)

Which of the following elements of the discharge plan are important to assess for this consumer?

- Providers
- Medication list
- Signs and symptoms to monitor for/report to provider, restrictions/limitations
- All of the above

Which of the following biometrics/labs should be documented in trackers?

- Blood pressure and pulse
- Cholesterol
- Weight, Height & BMI
- Smoking status
- All of the above

Clinical Case

Follow-up call one week later: The patient had recent hospital discharge for atrial fibrillation with rapid ventricular response and transient ischemic attack (TIA).

- Review essential care elements on Cardiac Arrhythmias and CVA / TIA
- Assess discharge instructions, providers and plan of care
- Value Pillars: right provider, right care, right medication, right lifestyle
- SPA (Stop, Pursue and Act)

Which of the following elements of the discharge plan are important to assess for this consumer?

- Providers
- Medication list
- Signs and symptoms to monitor for/report to provider, restrictions/limitations
- All of the above

Which lab should be added to the trackers?

- iPTH
- INR
- Hemoglobin A1c
- None of the above
- All of the above

Last updated 12-2017 Kelly Darlak, Pharm.D, BCPS

- These medication regimen checklists were derived from evidence-based medicine guidelines.
- Use the appropriate checklist to search for potential gaps in care. If found, please contact your Pharmacist or Medical Director for review.
- This sheet also contains high-level education for members on how their medications work, to help encourage compliance.

√ Angiotensin Converting Enzyme Inhibitor (ACEI) or Angiotensin Receptor Blocker (ARB)
√ Antiplatelet Agent(s) [2 after stenting, 1 of which is usually aspirin]
√ Beta-blocker
√ Statin
[Note: patients may also be on a nitrate]

HF with EF >40%

√ Control blood pressure: may use ACEI or ARB, &/or beta-blockers
√ May use diuretic for relief of symptoms due to volume overload.

Heart Failure (HF) with Ejection Fraction (EF) ≤40%

√ ACEI or ARB
√ Beta-Blocker
√ Diuretic
[Note patients may be on additional therapy, such as an aldosterone antagonist, hydralazine/isosorbide dinitrate, ivabradine, & digoxin, depending on clinical conditions. The provider may also use sacubitril/valsartan in place of ACEI or ARB monotherapy.]

Member Medication Education

- ACEI or ARB - relaxes blood vessels & lower blood pressure, which can benefit the heart and kidney
- Aldosterone Antagonist - lower blood pressure; may decrease impairment of the muscle cells in the heart & blood vessels
- Antiplatelet Agent - prevent coronary artery clots from forming and impeding blood flow, which can contribute to heart attack & stroke
- Beta-blocker - lower blood pressure & heart rate, increase blood flow to your heart
- Digoxin - helps the heart beat, and regulate heart rhythm
- Diuretic - rid the body of excess fluid, lowering blood pressure & making it easier for the heart to function
- Hydralazine/isosorbide dinitrate - lower blood pressure and increase blood flow
- Ivabradine - lowers heart rate
- Nitrate - relaxes blood vessels, which may improve blood flow and relieve chest pain/pressure
- Sacubitril/valsartan - relaxes blood vessels & lowers blood pressure, which can benefit the heart and kidney; also rids the body of excess sodium & fluid
- Statin - lowers cholesterol, which may decrease risk of stroke & heart attack

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Thank You.

Questions? Contact OptumHealth Education at moreinfo@optumhealtheducation.com

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