



Pathophysiology

As a brief review, ischemic heart disease or coronary artery disease can be thought of as a spectrum that ranges from stable angina to unstable angina which may progress to myocardial infarction and sudden cardiac death.

As we know, stable angina is caused by luminal narrowing of coronary arteries whereas unstable angina is caused by plaque rupture and thrombus formation leading to stenosis.

Myocardial infarction results when you have occlusion of the coronary artery leading to ischemia and then infarction of the myocardium.

And within that, a STEMI is caused by complete occlusion and a N-Stemi is typically caused by a partial occlusion.

First Objective

- 1. Given a specific clinical scenario in the officer emergency setting diagnose presentations of ischemic heart disease that are:**
 - classic
 - atypical (eg. in women those, with diabetes, the young and those at no risk)

Now to address the first objective, we're going to go through the presentations of Ischemic heart disease. To do that we will first define Angina.

Angina, as per Rosen's, can be described as Chest discomfort, with a squeezing, pressure, tightness, fullness, heaviness or burning sensation. This may radiate to the right arm, both arms, jaw or even back. And it can be associated with things like SOB, presyncope/syncope, diaphoresis, nausea, vomiting and even worsening with exertion. (Rosen's).

Within that, there is stable angina: Relieved by rest and nitro and Unstable angina: which typically occurs at rest or is not relieved by rest or nitro. For those who are interested, the CCS has also provided definitions on how to grade angina. This will be provided in the show notes on the episode webpage.



Canadian Cardiovascular Society grading of angina pectoris

Grade	Description
Grade I	Ordinary physical activity does not cause angina, such as walking and climbing stairs. Angina with strenuous or rapid or prolonged exertion at work or recreation
Grade II	Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions
Grade III	Marked limitation of ordinary physical activity. Walking one or two blocks on the level and climbing one flight of stairs in normal conditions and at normal pace
Grade IV	Inability to carry on any physical activity without discomfort, anginal syndrome may be present at rest

References
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Available on the Canadian Cardiovascular Society Website at www.ccs.ca

https://ccs.ca/app/uploads/2020/12/Ang_Gui_1976.pdf

Now that is the typical presentation. However, it is very important to be cognizant of what are referred to as atypical presentations which because they are less characteristic, can cloud the clinical picture leading to delays in treatment or missed diagnosis.

Atypical presentations may include epigastric or back pain, described as burning, stabbing or even characteristic of indigestion. (AHA journals). Atypical Presentations of ACS are most commonly seen in women, those with diabetes, the elderly, the young and those at no risk.

Second Objective

Objective 2

In a patient with modifiable risk factors (ischemic heart disease, smoking, diabetes control, obesity), develop a plan in collaboration with the patient to reduce his or her risk of developing the disease.

In terms of risk stratifying, there are a number of risk factors for developing ischemic heart disease and optimizing management of these can improve outcomes. Some of the biggest contributors are things like dyslipidemia, diabetes, HTN and smoking. And we will briefly go through those now.

Diabetes is believed to contribute to ischemic heart disease through a number of different mechanisms. These include but are not limited to impaired blood flow, increased



vasoconstriction, inflammation, hypercoagulability, and increased susceptibility to development of atherosclerosis.

Now from a prevention perspective, it is generally recommended to use a fasting plasma glucose and HbA1C to screen patients every 3 years if they are 40 or older.

Diabetes Canada recommends maintaining a HbA1C < 7%. As there is an estimated 11 to 16% increase in cardiovascular events for every 1% increase in HbA1C.

Dyslipidemia is another contributor and as per the CCS 2021 guidelines, Lipid and lipoprotein screening should be performed for men and women older than 40 years of age or at any age with any of the following conditions including

- Clinical evidence of atherosclerosis
- AAA
- DM
- HTN
- Cigarette smoking
- Stigmata of dyslipidemia
- Family history of premature CardioVascularDisease
- Family History of dyslipidemia
- CKD
- Obesity (BMI>30)
- Inflammatory diseases
- HIV
- Erectile dysfunction
- COPD
- Hypertensive disorder of pregnancy

Specifically for routine screening, you will want to order a Total cholesterol, LDL-C, HDL-C, non HDL-C and Triglycerides. It is also indicated to order a Lipoprotein once in a patient's lifetime with initial screening.

In the outpatient setting in terms of calculating total cardiovascular risk, you should screen men and women aged 40-75 every 5 years. For this, you can use risk assessment tools such as Framingham Risk Score (FRS) or the cardiovascular life expectancy model (CLEM).



Now there are a number of situations in which you will want to initiate a statin.

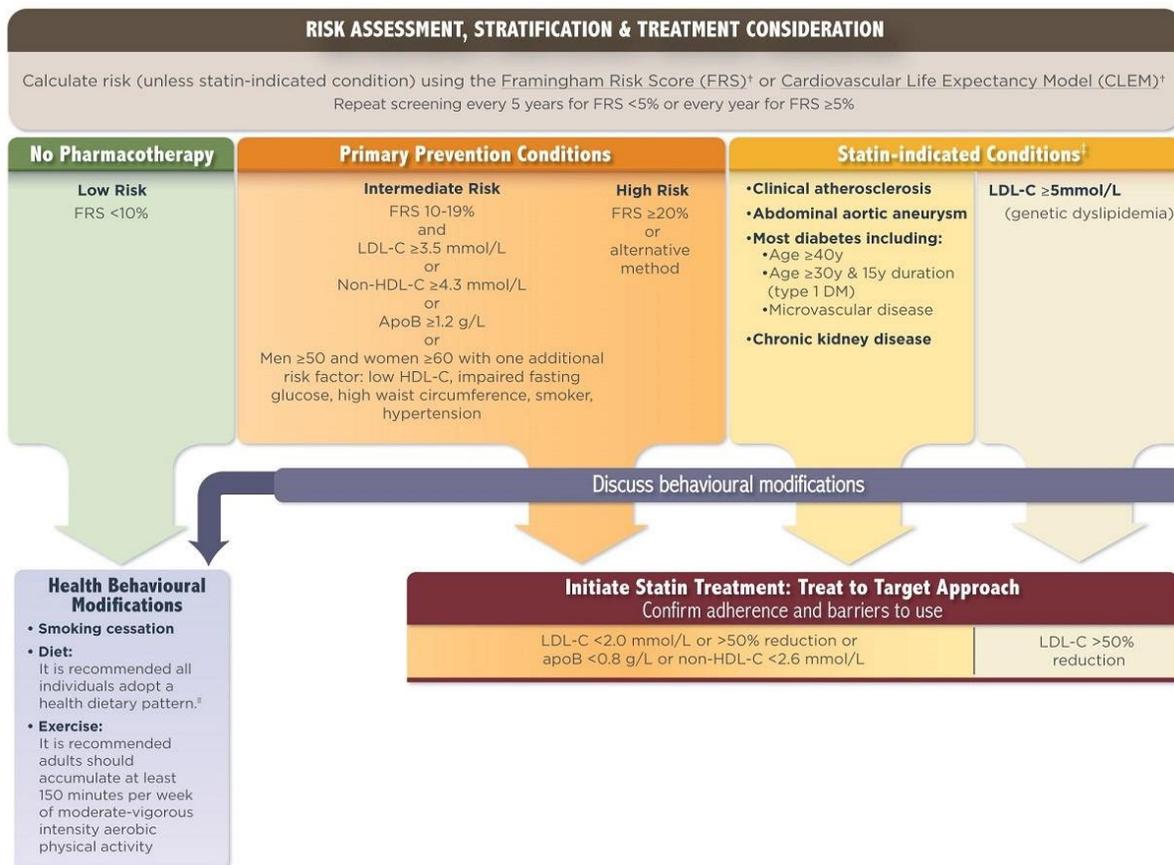
- Clinical atherosclerosis which includes Stable angina, ACS, stroke and TIA among others.
- AAA
- Diabetes
 - In patients who are age 40 or older
 - OR a person with type 1 diabetes who is Age 30 or older and has had the disease for at least 15 years
 - OR if there is evidence of microvascular disease
- Chronic Kidney disease
- LDL >5mmol/L

Beyond this.

For primary prevention purposes, it is recommended to use a statin if

- FRS > 20
- FRS 10-19% and LDL-C greater than or equal to 3.5.
- Non HDL-C greater than or equal to 4.3 mmol/l
- Apo B greater than or equal to 1.2 g/l
- Men ≥ 50 or women $60 \Rightarrow$ with one additional CVD risk factor

Again because this is a lot to remember, an infographic will be included in the show notes.



<https://ccs.ca/eguidelines/Content/Topics/Dyslipidemia/4.1%20Statin%20Therapy.htm>

The pathogenesis of smoking is thought to be due to multiple mechanisms including inflammation, vascular wall damage and vasoconstriction. Because of this, patients should be counselled on smoking cessation.

Exercise has similarly been found to reduce mortality rate in patients with CAD. It is recommended that a patient do 30 – 60 minutes of moderate intensity exercise 5 days a week.

Objective 3

In a patient presenting with symptoms suggestive of ischemic heart disease but in whom the diagnosis may not be obvious, do not eliminate the diagnosis



solely because of tasks with limited specificity and sensitivity (eg. Electrocardiography, exercise stress test, normal enzyme results).

Generally speaking, in the emergency medicine setting, to diagnose an acute MI you require at least 2 of the following

- Ischemic symptoms
- Diagnostic ECG changes
- Serum cardiac marker elevation

In the outpatient setting, a common investigation that is used to assess for CAD is the stress test of which there are many types which we will not go into today.

While these tests offer great utility, it is important to not eliminate the diagnosis solely based on the findings of normal ECG, cardiac enzymes or stress test because the specificity and sensitivity of these are limited.

As a quick review, sensitivity can be thought of as the proportion of test samples that will have a positive result. If the sensitivity of a test is high, then it will be able to identify a high proportion of patients who have the disease.

Specificity can be thought of as the proportion of tests that will have a negative result. If the specificity of a test is high, the more likely you'll be able to exclude the disease.

In patients with suspected ACS without sepsis or renal failure, the sensitivity and specificity of troponin is roughly 95% and 80%, respectively. However, it is important to recognize that troponin can be elevated for a number of reasons. Eg. HF, sepsis, renal failure.

Further, if a patient has had prolonged CP, one troponin may be sufficient. However, if the chest pain is new onset, you should do two and look at the delta (change between the two). ECGs are also very helpful in diagnosing acute MI. However, it is important to remember that you do not require ECG changes to have ischemia. For example in a non ST elevation MI, you do not require ST changes. And in fact, despite having a troponin elevation you may not see any ECG changes.

Some ECG changes may be much more subtle. For example, the presence of biphasic T waves or deep T wave inversions in V2 and V3, may be suggestive of a Wellen's Syndrome (aka widow maker) which refers to a total occlusion of the left anterior descending artery (LAD).

If left untreated, patients can have a massive anterior wall MI within a few weeks despite having no marked ischemic changes at the time.



Stress tests of which there are many types, while useful, have considerably limited specificity and sensitivity. Treadmill stress tests for example have a reported sensitivity and specificity of 68% and 77% respectively. Additionally, there a number of problems including referral bias. Further, there is a reported event rate of approximately 1% of MI or cardiac death after normal stress test.

Alternatively, while CT angiography can help to identify burden and presence of CAD, it may not necessarily provide functional information.

Chris: That was a lot of talking. Time for a dad joke. Hey Caleb, what is the name of the band whose frontman had a STEMI?

Caleb: Well Chris. I don't know.

Chris: The Jammers. Teehee.

Objective 4

In a patient with stable ischemic heart disease manage changes in symptoms with self -initiated adjustments of medication (eg. Nitroglycerin) and appropriate physician contact (eg. office visits, phone calls, emergency department visits) depending on the nature and severity of symptoms.

In the prehospital setting, a patient with stable angina who has developed changing symptoms can trial

- 1-2 sprays of sublingual nitroglycerin q 5 minutes. To a max of 3 doses in 15 minutes. If symptoms do not resolve or worsen, then they should go to the emergency department.

If this is occurring, you would also counsel the patient to administer

- ASA 325 mg, as long as they do not have allergies to this.

Objective 6

6. In a person with diagnosed acute coronary syndrome (eg. cardiogenic shock, arrhythmia, pulmonary edema, acute myocardial infarction, unstable angina) manage the condition in an appropriate and timely manner.



If a patient has not yet been diagnosed with an acute MI in the field and is not being sent directly to the cath lab, commonly patients with CP will present to the ER.

Back to our case, if you are the attending in this Everest base camp ER, you will want to remember the mnemonic “MOVIE” which stands for placing the patients on

- Monitors
- Oxygen (If saturating less than 90% on RA).
- Vital signs
- IV access
- 12 lead ECG

And

- Put on defibrillation pads

If we have diagnosed an Acute MI, reperfusion therapy is key and there are two ways in which we can do this. Percutaneous coronary intervention (PCI) and Fibrinolysis. While both are efficacious, unless there are contraindications, PCI is generally preferred.

Advantages include lower risk of intracranial bleed and higher initial reperfusion. Particularly in the setting of shock, PCI is preferred because fibrinolytics won't be able to appropriately circulate.

However, the one that we pick is based on the time we have, because the window for PCI is much smaller than the window for fibrinolysis. As you may have heard it described before, the door to balloon time, or time from first medical contact (FMC) to PCI is 120 minutes and ideally this would occur in less than 90 minutes. If you can perform PCI in this window, great.

Fibrinolysis

- If you cannot, in the absence of contraindications, you will opt for fibrinolysis.
- The therapeutic window for fibrinolytics is 12 hours from symptom onset with the best result being achieved within 6 hours from symptom onset.
- Fibrinolytics typically include tissue plasminogen activators such as Tenecteplase.

Prior to PCI or fibrinolysis, you will want to start the patient on 1. dual antiplatelet therapy (which consists of ASA and a P2Y12 inhibitor such as clopidogrel) and 2. anticoagulation with an antithrombin such as enoxaparin.

However, the ones that you choose largely depend on whether the patient is undergoing a STEMI or an NSTEMI. The next little bit can be slightly confusing so bare with me.



If your patient is having a STEMI and you are able to send them for PCI, you will want to initiate DAPT which consists of ASA and a P2Y12 inhibitor (either clopidogrel or ticagrelor). Additionally, you will want to start anticoagulation with either enoxaparin or unfractionated heparin. You should not use Fondaparinux.

On the other hand, if you are sending your STEMI patient for fibrinolysis, you will initiate an almost identical regime. However, in the setting of lysis, you should never use ticagrelor as your P2Y12 inhibitor because there is a higher incidence of intracranial bleeds. So use clopidogrel instead.

In the setting of N-STEMI, you will only send your patient for PCI. Not fibrinolysis. And prior, you will initiate dual antiplatelet therapy consisting of again ASA and a P2Y12 inhibitor (either clopidogrel or ticagrelor). However, for anticoagulation, you can use enoxaparin or fondaparinux.

In addition to acute MI, antithrombins such as Unfractionated heparin or Enoxaparin can also be indicated in the setting of recurrent angina, significantly elevated trop and dynamic ECG changes. If the patient is for example still experiencing CP and is having ECG changes, consider initiating heparin.

In addition to dual antiplatelet therapy and anticoagulants, in the setting of ACS, we should also initiate:

A Beta blocker, Ace inhibitor and a statin (which are to be administered within 24 hours)

- Unless contraindicated

Now, historically the MONA (Morphine, oxygen, nitroglycerin and aspirin) mnemonic was a crucial component of ACS care.

However, as per the new CCS guidelines, it has been recommended that we

- Try and avoid routine use of morphine. But we can use it if the patient is in severe pain. (CCS)
- It is recommended that we only administer O2 if $SPO_2 < 90$. Not recommended if 90 or greater (CCS).
- Additionally, nitroglycerin although helpful with reducing symptoms, does not improve mortality. And specifically in the setting of inferior MI you should not give nitro, as the RV is preload dependent.

The rest of this objective should be covered primarily in the ACLS objective. So I will instead provide a very brief overview of cardiogenic shock.

Cardiogenic Shock



- Which can be defined as a disorder of cardiac function in the form of a critical reduction of the heart's pumping capacity

Pathophysiology

- It is caused by systolic or diastolic dysfunction leading to a reduced ejection fraction or impaired ventricular filling.

Etiology

There are a number of different etiologies including

- Myocardial eg. ACS
- Rhythmologic eg. tachycardia, bradycardia
- Mechanical: eg. cardiomyopathy

Dx

- One useful tool in examining this in the acute setting is bedside echo. To assess for example pericardial effusion, tamponade, wall motion abnormalities.

Tx

- And ultimately, you're going to have to identify and treat the underlying cause

So for our patient currently at base camp, we have confirmed he is having a STEMI and there is no way that we can get him to the cath lab in Kathmandu in less than two hours, we are going to initiate our DAPT and anticoagulants and will be forced to lyse him.

He is medical evacuated to Kathmandu, where he is initiated on a beta blocker, ace inhibitor and statin, stays as an inpatient prior to return to Canada.

Objective 5

5. In the regular follow-up care of patients with established ischemic heart disease, specifically verify the following to detect complications and suboptimal control:

- **Symptom control**
 - **Episodes of chest pain. Frequency, severity, pattern (up to date)**
- **Impact on daily activities**
 - **Any impact on their physical activity? Exercise tolerance? (Up to date)**
- **Lifestyle modification**
- **Clinical screening (i.e. symptoms and signs of complications)**

Your patient has now returned home from Nepal and you are seeing them in clinic at a 6 month follow up appointment. Particularly, in the outpatient setting there are a number of things you will want to assess for to monitor disease progression and control.



Number 1: Symptoms

- Are they having chest pain? If so, when? and how bad is it? Are there any triggers such as exertion?
- If they are symptomatic, Is it interfering with their daily activities or limiting exercise tolerance?

Number 2: Medication compliance

- Are they on the appropriate medication? And if so, are they taking it?
As a brief review again, unless contraindicated its recommended that patients with CAD are on
 - Daily low dose aspirin: 81 mg po daily. Indefinitely. (CCS)
 - A statin
Generally trying to target an
 - LDL-C < 2.0 mmol/L or over 50% reduction
 - OR APO B < 0.8g/l
 - OR non HDL-C < 2.6 mmol/L
 - If targets have not been reached with max dose statins, you can consider Ezetimibe to lower LDL.
 - Have Diabetic control: to maintain HBA1C < 7.
 - Be on anti HTN therapy:
As per the SPRINT Trial, in patients at risk for cardiovascular disease, it was found that maintaining systolic BP < 120 mmhg reduced incidence of major cardiovascular events and death.
It is recommended that first line for antihypertensives are ACE inhibitors (if HTN, diabetes, LVEF< 40 or CKD. Stable CAD).
However, you can do an ARB if intolerant of ACE inhibitors (CCS)

From an anti anginal perspective,

- Betablockers are first line therapy for chronic stable angina if the patient has had an MI, reduced EF or HF. Or just generally for chronic stable angina (CCS).
- And Nitroglycerin may be considered for immediate relief of symptoms. However, it has not been found to improve mortality.

[https://www.onlinecjc.ca/article/S0828-282X\(14\)00356-0/fulltext#secsectitle0030](https://www.onlinecjc.ca/article/S0828-282X(14)00356-0/fulltext#secsectitle0030)

<https://www.aafp.org/pubs/afp/issues/2018/0315/p376.html#afp20180315p376-t3>

Number 3: Signs and symptoms of complications

So watching out for dysrhythmias and heart failure: Palpitations, SOB, decreased exercise tolerance, pedal edema and alike.



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