Ischemic heart disease conundrums

by Alba Kuqi, MD, CCS, CDIP, CCDS, CRCR, CICA, CSCM

Ischemic heart disease has a multifactorial etiology and can be prevented from developing in populations primordially, and in individuals at high risk by primary prevention. The primordial approach focuses on the social determinants of health in populations, such as:

- Political
- Economic
- Social factors, principally unplanned urbanization, illiteracy, poverty, and working and living conditions

Ischemic heart disease, also called coronary heart disease (CHD) or coronary artery disease (CAD), is the term given to heart problems caused by narrowed coronary arteries that supply blood to the heart muscle. Ischemia refers to a decrease in blood flow to an organ, and it’s usually due to atherosclerosis.

If you have atherosclerosis in the coronary artery, you will decrease the amount of blood flow going to the myocardium. Therefore, the risk factors for ischemic heart disease are going to be pretty similar to those of atherosclerosis, and it so happens that the incidence of ischemic heart disease increases with age just as the incidence of atherosclerosis does.

CDI professionals need to pay attention to the definitions of acute ischemic syndrome and the clinical indicators that make the difference while reviewing a medical record. Any discrepancy in the documentation may warrant a query.

Alba Kuqi, MD, CCS, CDIP, CCDS, CRCR, CICA, CSCM

Stable angina

Stable angina refers to reversible chest pain. The term “stable” implies that the patient does not have chest pain at rest but instead develops it with exertion or emotional stress. The idea here is that if atherosclerosis of the coronary artery is decreasing the blood flow to the myocardium, the patient will be fine at rest since in a resting state the myocardium will still be getting enough blood. If, however, the patient exerts themselves, not enough blood will be provided to the myocardium and hence there would be injury to the myocardium. The patient will be having reversible myocardial injury swelling.

Patients with atherosclerosis will only get angina when there is higher than 70% stenosis. If there’s less than 70% stenosis, the patients are relatively asymptomatic even with exertion. When patients present with stable angina, although they have chest pain that radiates to the left arm or jaw, note that this chest pain will last less than 20 minutes, which is about the amount of time that the myocardium can withstand the lack of blood flow.

After that, you get irreversible injury and cell death. As chest pain classically lasts less than 20 minutes,
patients may also have diaphoresis or sweating and shortness of breath. If an EKG is performed, it would show ST-segment depression.

Stable angina will be relieved by rest because this decreases the demand on the myocardium, which then will decrease the amount of blood flow that needs to be provided to the myocardium. Another way to relieve the chest pain is nitroglycerin, which acts as a vasodilator for both arteries and veins.

Furthermore, nitroglycerin’s major mechanism of action is the visible dilation of veins, which would then decrease the amount of blood that’s returning to the heart, thereby decreasing the preload on the heart and thus the stress on the myocardium.

**Unstable angina**

Unstable angina occurs at rest. It is due to the rupture of the atherosclerotic plaque with thrombosis and can result in complete occlusion of a coronary artery. This again would represent reversible injury to the cell. On EKG, you would see ST-segment depression because you would have predominantly subsegmental cardiac ischemia.

Nitroglycerin is used to treat unstable angina. As stated, nitroglycerin causes vasodilation and decreases preload, which then decreases the amount of work the myocardium has to do. Unstable angina has a very high risk of progression because it would be easy for this thrombus to occlude the vessel completely, resulting in myocardial infarction.

CDI professionals need to always look for clinical indicators of unstable angina. If signs/symptoms of acute myocardial infarction (AMI) are not present, acute coronary syndrome (ACS) is an umbrella term used to describe chest pain caused by either AMI or unstable angina. If angina and CAD are both documented, then the combination code is assigned. Treatment for unstable angina is as follows: supplemental oxygen, nitroglycerin, morphine, beta-blocker, antiplatelet, beta-blocker, anticoagulant, and statin therapy.

**Prinz Metal’s angina**

The third type of chest pain is called Prinzmetal’s angina. This is caused by a spasm of the coronary artery, which can completely cut the blood supply and result in episodes of chest pain that are unrelated to exertion. Whenever the vessel clamps down, that’s when the patient is going to have chest pain.

Again, it’s important to note that this still represents reversible injury to the myocyte. On the EKG, it would show as ST-segment elevation because the coronary artery is completely clamping for a short period of time and cutting blood supply to the entire wall. To decrease the preload on the heart, calcium channel blockers can be used, which would help to relieve the coronary artery vessels.

**Myocardial infarction**

Another ischemic heart disease syndrome is myocardial infarction (MI). This occurs when there is a rupture of an atherosclerotic plaque that would result in complete occlusion of the coronary artery. The idea here is that a complete thrombus blocks off the blood supply, resulting in a lack of flow into the myocardium, which will result in the death of the myocardium.

Different forms of acute myocardial ischemia include:

- Type 1: STEMI (ST-elevation MI), Q wave MI, and NSTEMI (Non-ST Elevation MI)
- Type 2: MI secondary to ischemic imbalance (supply/demand mismatch) as in coronary vasospasm, anemia, or hypotension.
- Type 3: MI resulting in death when biomarkers not available.
- Type 4: MI related to PCI and stent thrombosis.
- Type 5: MI related to CABG
- Demand Ischemia (supply/demand ischemia)
- Unstable angina (definite, probable, or possible)

Unstable angina is ischemia primarily due to CAD. Demand ischemia is due to supply/demand mismatch, but CK-MB and troponin levels do not rise above the
99th percentile of the lab test reference range (Troponin I = 0.04 mcg/L). Common causes of supply/demand mismatch (Type 2 MI and demand ischemia) include tachyarrhythmias, severe anemia, sepsis, shock states, prolonged hypertension, hypertensive crisis, and coronary spasm. Many causes of elevated troponin levels that do not necessarily indicate MI include heart failure, renal failure, arrhythmias, myocarditis, and pulmonary embolism.

If an EKG were to be done at the beginning of this event, it would show ST-segment depression. With continued ischemia, the necrosis will progress and involve the entire wall, which will result in a transmural infarction transmitter. At this point, you would see ST-segment elevation on the EKG.

**CDI professionals need to review the entire medical record to try to find the right clinical indicators before posing a query to a provider. The query must be non-leading and provide the physician with all the information needed to come up with a good answer. Whenever there’s a discrepancy in the documentation, we should seek clarification.**

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When you want to make a diagnosis of MI, look for cardiac enzymes in the blood. Again, you’re looking to see whether there has been irreversible damage to the myocardium. With the cardiac cell membrane damage, the enzymes will leak out into the blood, so if you can find them in the blood, that tells you that the patient had a MI.

The most sensitive and specific marker is troponin I. This rises in the four hours after the MI, peaks in 24 hours, and stays high for seven to 10 days. Another useful marker can be CK-MB because it rises quickly; however, it also goes down relatively quickly.

For example, let’s say the patient has an MI, and then five days later developed another MI. If you were looking to prove that there was a second MI, you wouldn’t be able to do so with troponin I because troponin I would be elevated from the first MI and then stay up for seven to 10 days. As a result, one potential useful marker would be CK-MB. Again, it would have gone down after 72 hours after the first MI, and if there’s reinfarction, it will then rise again. So CK-MB is particularly useful in detecting reinfarction a few days after an initial MI.

**CDI professionals need to have a clear understanding of the definition of acute MI (AMI) because this will help differentiate between various types of MI.**

**MI treatments**

The treatment for MI is aspirin and heparin. The goal here is to limit additional thrombosis, since it was the complete occlusion of the coronary artery by thrombosis that led to the MI. Supplemental oxygen is given to minimize ischemia, and nitrates are given to vasodilate both arteries and veins. The major mechanism would be vasodilation of the veins, which would decrease the preload on the heart, which would then decrease the stress on the heart.

Beta-blockers are useful because they slow the heart rate, which decreases the demand for oxygen and also decreases the risk of arrhythmia. Arrhythmias are feared complications very early after an MI occurs.

ACE inhibitors can be used. They decrease the left ventricular dilatation because they block the production of angiotensin II. Angiotensin II causes constriction of the peripheral artery so an ACE inhibitor blocks that constriction, thus decreasing the afterload on the heart.

Additionally, remember that angiotensin II goes to the adrenals and causes a release of aldosterone. Angiotensin II increases the blood volume, so by giving an ACE inhibitor, the blood volume will not increase. All this acts to decrease the risk of left ventricular dilatation. A more definitive treatment would be fibrinolysis or angioplasty.

**CDI professionals need to review the entire medical record to try to find the right clinical indicators before posing a query to a provider. The query must be non-leading and provide the physician with all the information needed to come up with a good answer. Whenever there’s a discrepancy in the documentation, we should seek clarification from the attending and try**
to pull information from different notes, most importantly from the cardiology consult note.

**Clinical documentation concepts**

ACS is a nonspecific term meaning a condition created by sudden, reduced blood flow to the heart. It can indicate a range of conditions, from unstable angina to AMI. ACS is only a provisional diagnosis encompassing clinical symptoms consistent with acute myocardial ischemia due to coronary artery disease. CDI professionals need to look for clinical evidence of AMI:

- Substernal or epigastric chest pain.
- Jaw pain
- Pain radiating down the left arm
- Diaphoresis
- Nausea and vomiting (especially in women)

We need to look for treatment as well: morphine, nitrates, beta-blockers, anticoagulation, thrombolytics (tPA), antiplatelets (aspirin, Plavix®), and percutaneous coronary intervention (PCI) when appropriate. CDI professionals need to seek clarification through querying if the patient demonstrates clinical evidence of an AMI.

The **Fourth Universal Definition of MI** separates myocardial infarction from myocardial injury. The new definitions were updated to accommodate the increased use of high-sensitivity cardiac troponins (hs-cTn). We should always remember that code assignment is based on provider documentation. If documentation of MI is conflicting or ambiguous, or if there is a concern for clinical validity, a clarification would be appropriate.

Per the new definitions, patients with elevated blood troponin levels but without clinical evidence of ischemia are said to have a myocardial injury. To have a myocardial infarction requires both an elevated troponin blood test along with at least one of the following:

- Symptoms of acute myocardial ischemia.
- Clinical evidence of ischemia, as evidenced in an EKG showing new ischemic changes.
- Development of pathological Q-waves.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy (Type 1 MI only).
- Angiographic findings consistent with procedural flow-limiting complications such as coronary dissection, occlusion of a major epicardial artery or graft, side-branch occlusion thrombus, or disruption of collateral flow or distal embolization (Type 4 or Type 5 MI only).

CDI professionals need to look for frequent secondary diagnoses that impact severity of illness/risk of mortality, such as chronic kidney disease, end-stage renal disease, hyponatremia, pancreatitis, pneumonia, sepsis, and urinary tract infection.

**Editor’s note:** Kuqi is the CDI supervisor at Prime Healthcare in Philadelphia. Contact her at albakuqi88@gmail.com. Opinions expressed are those of the author and do not necessarily reflect those of ACDIS, HCPro, or any of its subsidiaries.

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