

Q&A Summary:

Coronary Artery Disease: Prevention, Complications and Management

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Please define what you mean by a “rupture” of coronary plaque? Do you mean that it breaks off?

In patients with acute coronary syndrome (unstable angina and myocardial infarction), the plaque is commonly full of cholesterol and other lipids. The plaque ruptures into the lumen of the artery (just like a pustule or a pimple on the skin “pops”). The plaque itself doesn’t break off, but the content is spilled into the artery. This attracts platelets, ultimately resulting in a platelet-rich clot. This is the reason why lipid-reducing and antiplatelet therapy are recommended in patients with coronary artery disease.

Are there any evidenced-based recommendations for patients with multiple and significant risk factors such as diabetes, advanced age (70s or greater), previous coronary artery bypass graft (CABG), etc. as to when things like a stress test no longer make sense to continue to pursue?

Although diabetes, advanced age and previous coronary artery bypass surgery all increase the risk of developing coronary artery disease (CAD), none of these individually or collectively is a contraindication to treatment of myocardial ischemia or infarction. Some conditions — such as severe dementia or advanced cancer may be reasons to not pursue treatment of CAD. However, intervention must be individualized based on the clinical setting. In a patient with severe angina that is refractory to medication therapy, relief of pain may only be achieved by a procedure such as angioplasty with stenting. This is reasonable for symptom management even in patients with very limited longevity.

Stress testing is used to identify myocardial ischemia — inadequate blood supply to the heart muscle. It may be used for diagnostic purpose in patients who do not have known CAD. It is also useful in management of patients with known CAD as it detects residual or recurrent ischemia after previous procedures and/or despite appropriate medication therapy. Diabetes, advanced age and previous surgery are not contraindications to stress testing. Patients with limited longevity because of non-cardiac disease may not be suitable for stress testing.

Why are you limited to Warfarin if you have a mechanical heart valve?

Warfarin is the only oral medication approved for therapeutic anticoagulation in patients with mechanical valves.

non-Warfarin oral anticoagulant medications (also called novel oral anticoagulants, or NOACs) are not used in patients with mechanical heart valves because the incidence/risk of

thromboembolic events is higher with these medications than it is with Warfarin. Thus, Eliquis (Apixaban), Xarelto (rivaroxaban) and Pradaxa (dabigatran) are not approved for use in patients with mechanical heart valves.

Intravenous heparin can be used in patients with mechanical heart valves who are unable to take oral medications. Heparin may also be used as a “bridge” for patients who are just starting Warfarin or those who must interrupt Warfarin therapy for surgery, etc. Subcutaneous heparin may be used in certain circumstances (such as during pregnancy in women with mechanical heart valves).

If an individual lives in a rural area and is having active chest pain and it will be a while until they can get to a hospital, is it still advised to chew an adult aspirin or could this cause problems if a percutaneous coronary intervention (PCI) was ordered? Are there any other recommendations until they can get to hospital?

Coronary artery disease is only one cause of chest pain. Other causes of chest pain include pulmonary emboli, pneumothorax, pleurisy, pericardial disease, esophageal rupture, aortic dissection, etc. Aspirin may be contraindicated in some of these conditions. Suggesting medical therapy is out of scope for case management. When engaging with a member with a potential life-threatening illness, follow the Emergency Medical Services Contact Process Job Aid.

If you have stable angina, does that place you at higher risk of for myocardial infarction (MI) and/or sudden cardiac death?

A patient with stable angina has coronary artery disease. Therefore, the risk of MI is generally greater than it is for one with normal coronary arteries. However, the magnitude of this increased risk depends on many factors including the specific coronary artery issue that causes the angina. (For instance one with a single occluded branch vessel has different risk than one with multivessel disease.). Also, current medical therapy such as use of statins and risk modification such as abstinence from tobacco use impact risk.

The risk of MI for patients with stable angina — while above that of healthy people — is lower than it is for patients with unstable angina or recent myocardial infarction.

The risk for sudden cardiac death is influenced by many issues. Severely reduced left ventricular function — as measured by ejection fraction (EF) — is an important risk factor for sudden cardiac death. Previous myocardial infarction and myocardial ischemia are also risk factors, particularly if associated with poor left ventricular function.

A patient went for stenting last week, whereupon two attempts were made to stent right coronary artery, but these were unsuccessful, as she has 97 percent blockage. She was told there is nothing else they can do but treat her condition medically (which means to continue to take meds she is already on). What can she expect going forward? It seems like a ticking time bomb before she has an MI.

This member should discuss her specific situation with her cardiologist. Only the cardiologist can advise what she can expect and what her risks are.

It is not possible to comment on outcome and risk without full knowledge of the coronary artery anatomy. The size of the right coronary artery and the exact location are important factors that one can only know from viewing the actual catheterization images. The situation is different if

this is a small right coronary artery vs. a large dominant vessel. It matters if the stenosis is in the first part of the vessel or if it is distal (beyond key branches). Other important considerations include the size and location of other coronary arteries, and whether these have narrowing or are healthy. These are among the many factors that the cardiologist will consider in recommending treatment. If the cardiologist thought this member was a “ticking time bomb,” this member would likely have been referred for cardiac surgical evaluation.

Is coronary artery disease reversible with diet?

The Lifestyle Heart Trial (1978, Ornish, et.al.) demonstrated reversal of coronary artery disease in patients who were adherent to a plant-based, low-fat, vegetarian diet, in conjunction with other lifestyle changes, for five years.

<https://jamanetwork.com/journals/jama/fullarticle/188274>

There have been other trials with similar results. The challenge with this approach is long-term adherence to a very restrictive diet. This approach only works if one follows it diligently, exclusively and permanently. This approach is not feasible for the majority of patients.

Less-restrictive diets — such as the Mediterranean diet — are likely more practical and applicable to a larger number of patients.

How do you define “long-term mortality” as relates to its comparison in Non-ST-Elevation Myocardial Infarction (NSTEMI) vs. ST-Elevation Myocardial Infarction (STEMI)? Why is it higher in NSTEMI than in STEMI? (It seems counterintuitive.)

For this purpose “long-term mortality” refers to mortality at 10 years.

The second part of the question is complicated because it involves the concept of statistical adjustment.

Although the unadjusted mortality rate for NSTEMI is higher, careful analysis indicates that patients who present with NSTEMI are older and have more comorbidities that contribute to the higher death rate. In other words they are a ‘sicker’ group of patients in general. There are more deaths in 10 years, but many of the deaths may be related to factors other than the MI.

However, after statistical adjustment (which takes age and comorbidities into consideration), the mortality rate for STEMI is indeed higher. So the mortality rate of STEMI is higher when risk adjustment “levels the field.”

The following link is one study that addressed this issue:

<https://www.ncbi.nlm.nih.gov/pubmed/16644337>

For 66 year old male with no significant medical history but who has very high alcohol intake (approx. 750 ml/d liquor); taking atenolol, olmesartan, and transdermal clonidine; without significant decrease in blood pressure with average diastolic pressure of high 90s-110, who is unwilling/unable to decrease alcohol significantly, what is a good next step? Which class of drug should be increased, added, etc., if alcohol intake stays unchanged?

This is clearly a very difficult case. The member needs to work closely with his physicians to manage his hypertension and other problems.

The first question to address is the quality of blood pressure monitoring. Errors in measurement of blood pressure are very common. Who is checking blood pressure? Are these values determined in the physician's office? Is the member self-monitoring at home? Is he using the device correctly? Is the correct size cuff being utilized? If the cuff is too narrow for a large (fat or muscular) arm, the blood pressure will be artificially increased. For home use, electronic devices are best. If the consumer has a home blood pressure machine, suggest the consumer take the machine to providers visits to verify accuracy. The kiosks found in pharmacies and similar stores are commonly inaccurate because of calibration problems.

The second thing to be considered is medication adherence. Is this member really taking all of his medications as prescribed? If he is missing one of the agents ... or using medications sporadically ... then he is not on the regimen that his physician prescribed.

Atenolol is a beta blocker, Olmesartan is an angiotensin receptor blocker (ARB) and Clonidine is a centrally acting alpha-2 blocker. Only the treating physician can decide if dosage can/should be increased or if an additional agent may be appropriate. Medication dosage will commonly be limited by side effects.

If it is determined that the BP measurements are accurate and that the member is taking medications as prescribed, then it may be appropriate for him to see a hypertension specialist. It is important to evaluate for potential causes of hypertension such as renal etiology, renovascular disease and endocrine causes. There are specialists in hypertension at many academic/tertiary medical centers. Many Nephrologists have particular interest in evaluation and management of difficult hypertension.

Is there a known diagnosis of hepatic cirrhosis? It is clearly desirable for the member to reduce (or eliminate) alcohol use. This may or may not be realistic for this member at this time.

Would race be a factor in determining which antihypertensive to use first? If I recall, people from African American decent respond better to calcium channel blockers (CCBs)?

The guideline for management of hypertension currently approved by UnitedHealthcare is:

2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report from the Panel Members Appointed to the Eighth Joint National Committee (JNC 8).

This guideline was published in *JAMA* in February 2014:

<https://jamanetwork.com/journals/jama/fullarticle/1791497>

In regard to the above questions, the JNC8 Guideline states the following:

“In the general nonblack population, including those with diabetes, initial antihypertensive treatment should include a thiazide-type diuretic, calcium channel blocker (CCB), angiotensin-converting enzyme inhibitor (ACEI), or angiotensin receptor blocker (ARB).”

AND

“In the general black population, including those with diabetes, initial antihypertensive treatment should include a thiazide-type diuretic or calcium channel blocker (CCB).”