11 Steps Before Consulting Cardiology In The Patient With Chest Pain

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Introduction

Chest pain is a common presentation in general practice. Each year about 1% of the UK population visit their general practitioner with chest pain.[1] (US figures not easily found). The average GP will see, on average, four new cases of angina each year.[2] The Euro heart survey of newly diagnosed stable angina patients showed that the incidence of death and myocardial infarction (MI) was 2.3/100 patient-years. This is increased in patients with a previous MI, short history, more severe symptoms and with heart failure or other co-morbidities, such as diabetes.[3] The recognition of these patients as at high risk for cardiovascular events has led to the improvement of diagnosis and management of angina.

1. Take a Detailed History

A careful history remains the cornerstone of the diagnosis of angina pectoris.[4] There are some typical characteristics of chest pain that increase the likelihood that the pain is angina and caused by underlying coronary heart disease (CHD).[5]

Stable angina is characterized by the type of discomfort and location (Table 1):[6] it is elicited by physical exertion or emotion and relieved by rest or glyceryl trinitrate (GTN). Duration is short and it is worse in cold weather or after a meal (Table 2).

In unstable angina, the symptoms are more severe, more prolonged, more frequent, and may occur at lower thresholds or even at rest.

Table 1. Clinical Classification of Chest Pain[6]

Typical angina (definite) **Meets three of the following characteristics:**
• Substernal chest discomfort of characteristic quality and duration • Provoked by exertion or emotional stress • Relieved by rest and/or glyceryl trinitrate spray
Atypical angina (possible) **Meets two of these characteristics**
Non-cardiac chest pain **Meets one or none of these characteristics**

Table 2. Stable Angina Descriptions

• Type of discomfort, often tight, dull or heavy
• Location in left chest, can radiate to jaw, shoulder, back, arms
• Elicited by physical exertion or emotion and relieved by rest or glyceryl trinitrate (GTN) - although esophageal spasm may also respond to GTN
• Duration of several minutes after exertion or stress has stopped
• Associated factors, for example worse in cold weather or after a meal

2. Consider Non-cardiac Causes of Chest Pain

The most common causes of chest pain seen in the office or ER are non-cardiac (Table 3). They are usually differentiated by careful history taking. Differential diagnoses include esophageal disorders, such as gastro-esophageal reflux or esophageal dysmotility suggested by the character of the pain with epigastric burning, acid reflux, and relief with antacids.

Pleural pain may be caused by infection, pulmonary embolism or tumor. The character of the pain is important with elucidation of pulmonary symptoms. Musculoskeletal pain, such as with Tietze syndrome, is suggested by a pleuritic character and local tenderness. Referred pain from the thoracic spine can be suggested by previous history, trauma and local tenderness. Biliary pain with epigastric or right hypochondrial discomfort is worse with fatty foods and associated with nausea.

Psychological causes include anxiety, panic attacks, and depression. Somatic symptoms of psychological disorders are very common. However, it is also important to remember that the onset of angina itself may induce significant anxiety.

Anginal pain is not usually sharp or stabbing in nature, not usually influenced by respiration, not fleeting nor does it last all day. Scottish Intercollegiate Guidelines Network (SIGN) guidelines[5] recommend that if the diagnosis is uncertain then physicians should not give the impression that the patient has angina, which may lead to the development of adverse false beliefs. Early, appropriate specialist opinion may help to dispute an inaccurate diagnosis and reduce the risk of these false beliefs developing, but not if it automatically leads to cardiac catheterization.. Low-risk patients with atypical symptoms should be managed in primary care where possible.[5] It is important to explain symptoms and concerns, and provide reassurance where necessary. Correct management by a confident GP may both reduce morbidity and the need for referral.

Table 3. The Non-cardiac Causes of Chest Pain

- gastro-esophageal reflux
- esophageal dysmotility
- chest infection
- pulmonary embolism
- lung tumor
- musculoskeletal pain (Tietze syndrome)
- referred pain from the thoracic spine
- biliary pain
- psychological pain

3. Establish the Risk Factors: How Likely is it That Your Patient has CHD?

Risk factors can be modifiable or non-modifiable. Non-modifiable risk factors include increasing age and sex. About 85% of people who die of coronary artery disease are aged 65 years or older.[7] Men have a greater risk of premature heart disease than women.[7]

The risk of cardiovascular disease is higher in ethnic groups such as South Asians, and those of African-Caribbean origin. This increased risk is partly due to higher rates of high blood pressure, obesity and diabetes in these populations. Risk of cardiovascular disease increases if there is a positive family history of premature cardiovascular disease in a first-degree relative. This is defined as a history of premature CHD under age 55 in fathers, sons or brothers, or under age 65 in mothers, daughters or sisters.[8]

Modifiable risk factors include smoking, which is the most important preventable risk factor for cardiovascular disease and stroke. Increase in weight and lack of physical exercise contribute to cardiovascular risk, partially related to association with type 2 diabetes. Diabetes mellitus is considered by some to be a cardiovascular disease equivalent although this has been recently disputed.

High total cholesterol and low-density lipoprotein (LDL) cholesterol with low high-density lipoprotein (HDL) cholesterol are modifiable risk factors. High salt consumption and a diet rich in saturated fat can contribute to cardiovascular disease. Elevated blood pressure is associated with increased heart attack and strokes. Excess alcohol consumption can lead to increased blood pressure, heart failure and stroke.

4. Perform a Physical Examination

This is an important step[9] before referral (Table 4) because it may identify conditions that can precipitate angina (such as anemia or hyperthyroidism) and conditions other than CHD that can present with angina (aortic stenosis/hypertrophic obstructive cardiomyopathy). There may also be findings that would make a treadmill test unsuitable, such as uncontrolled blood pressure or aortic stenosis.

A comprehensive examination should include weight and height to allow calculation of body mass index, and waist circumference to evaluate presence of the metabolic syndrome. Record the pulse rate, rhythm and blood pressure. Listen for the presence of murmurs, especially aortic stenosis. Look for evidence of hyperlipidemia with xanthelasma or tendon xanthomata. Examine for evidence of peripheral vascular disease with absent foot pulses, bruits, skin changes or hair loss. Look for evidence of anemia or thyroid disease.

Table 4. What to Include in Patient Examination

- Weight, height and calculation of body mass index (BMI)
- Waist circumference to evaluate presence of the metabolic syndrome
- Pulse rate and rhythm
- Blood pressure
- Presence of murmurs, especially aortic stenosis
- Evidence of hyperlipidemia with xanthelasma or tendon xanthomata
- Evidence of peripheral vascular disease with absent foot pulses, bruits, skin changes, hair loss
- Evidence of anemia or thyroid disease

5. Carry Out the Relevant Investigation

Where there is a low index of suspicion of CHD, then it is not recommended that the patient undergoes further tests.[9,10]

For patients considered at higher risk, the following tests are recommended: A full blood count, serum creatinine or estimated glomerular filtration rate (eGFR), a fasting lipid profile and blood glucose. Thyroid function tests should be carried out if there is clinical suspicion of thyroid disease.

A resting 12-lead electrocardiogram (EKG) should be recorded in anyone with suspected angina to provide information on rate and heart rhythm to check for atrial fibrillation or heart block, and to check for signs of myocardial ischemia, hypertrophy, or previous MI. Some EKG abnormalities may exclude patients from a diagnostic treadmill test such as left bundle branch block (LBBB) and atrial fibrillation. A normal EKG does not exclude angina as more than 50% of people with stable angina have a normal resting EKG.[11]

Chest X-ray is not useful in the initial evaluation of chest pain, unless there is a history suggestive of heart failure, pulmonary disease or valvular pathology.

6. Estimate the Cardiovascular Disease Risk

Using the information obtained by the recommended assessment, all patients should have their risk of cardiovascular disease estimated using a risk estimation tool such as the Framingham cardiovascular risk assessment. (http://hp2010.nhlbihin.net/atpiii/calculator.asp?usertype=prof)
Alternatives include the Joint British Societies (JBS2) risk estimation tool[12] and the ASSIGN tool in Scotland.

The use of these charts is not appropriate for patients with pre-existing cardiovascular disease, chronic kidney disease and proteinuria, familial hypercholesterolaemia or other inherited dyslipidaemia, or type 1 and type 2 diabetes mellitus, for whom the UK Prospective Diabetes Study (UKPDS) risk assessment tool may be used.

Cardiovascular risk is higher than indicated in the charts for those with a family history of premature cardiovascular disease or stroke (male first-degree relatives aged < 55 years and female first-degree relatives aged < 65 years), which increases the risk by a factor of 1.5,[8] and 2.0 times with two first-degree relatives. It is also higher in those of South Asian background where the increased risk for men is 1.4 times, in the presence of left ventricular hypertrophy (LVH) on the ECG, and in those with raised triglyceride levels. Special consideration is required for those with chronic kidney disease without proteinuria, where a correction factor is not yet identified, women with premature menopause and those who are not yet diabetic, but have an impaired fasting glucose.

7. Give Lifestyle Advice

Anyone with angina should be advised to stop smoking. Smoking increases the risk of small vessel disease and the risk of dying from it, and risk is increased as more cigarettes are smoked.[10] Risk of a cardiovascular event falls from the first day the patient stops. Patients may need referral to specialist smoking cessation services and nicotine replacement therapy.

The Mediterranean diet is recommended with five portions of fruit and vegetables, and monounsaturated fats from olive and rapeseed oils. People at high risk of cardiovascular disease are recommended to consume at least two portions of fish per week, including one portion of oily fish. Omega-3 fatty acids are also found in flaxseed, almonds and walnuts.

Weight loss is recommended for all those who are obese or overweight. Moderate exercise for 30 minutes a day, on most days, within the limits set by their symptoms, should be advised.[10] Patients with cardiovascular disease will be offered a screening questionnaire for anxiety and depression, and may need advice to manage stress, relaxation techniques and setting realistic goals.

Holders of commercial drivers' licenses with new onset of angina should notify the Driver and Vehicle Licensing Agency (DVLA) and stop driving their vehicle. Holders of an ordinary driving license may still drive providing that symptoms are controlled, but must inform their motor vehicle insurance company. People who operate heavy machinery may also be affected.[13]

It is important to reassure that angina does not damage the heart, however, patients should be advised about the risk and symptoms of heart attack and when to call 911.[10]

8. Treatment to Control Symptoms and Reduce Cardiovascular Risk

There are two arms to treatment, which should be commenced if there is a high suspicion of cardiovascular disease.

Sub-lingual GTN should be prescribed to abort attacks, or to provide a short period of prophylaxis while doing activities likely to precipitate angina. If angina is not relieved by GTN treatment they should be advised to seek urgent medical advice.

A beta blocker should be commenced as first-line treatment for long-term symptom control.

A calcium channel blocker, Diltiazem, is usually the first choice where a beta blocker is either contraindicated or not tolerated.

Aspirin 75 mg should be started if cardiovascular risk > 20%, or if there is a high suspicion of CHD and blood pressure < 150/90 mmHg. If there is a high risk of gastrointestinal adverse effects it can be used in combination with a proton-pump inhibitor. Clopidogrel 75 mg is an alternative option.

Statins should be offered depending on the cardiovascular risk for primary prevention and for all patients who fit into secondary prevention. It is recommended to start with Simvastatin 40 mg.[8]

9. Consider exercise electrocardiography

An exercise EKG may provide valuable prognostic information. The appropriate use of exercise and isotope stress tests should be understood, particularly as it applies to “routine follow-up” or “annual” stress tests. (See joint paper from ACC/AHA).

People should not be referred for an exercise ECG if they are on maximal medical treatment and still have symptoms, the diagnosis of CHD is unlikely, or if they are physically incapable of taking the test. Patients who may have aortic stenosis or cardiomyopathy are not suitable. There is no point in referring patients in whom the results of an exercise ECG would not affect management[10] or, alternatively, who do not wish further investigation of their pain.

10. Consider Referral to a Cardiologist

Many patients need not be referred, and some may not wish further investigation. However, those in the following groups should be considered for early referral:
- those with new onset of chest pain;
- those with a history of previous MI;
- those with a history of coronary artery bypass grafting, or percutaneous transluminal coronary angioplasty;
- those who fail to respond to medical treatment;
- those with an ejection systolic murmur suggestive of aortic stenosis.

Some symptoms may represent an acute coronary syndrome and should be considered for hospital admission. These include:
- pain on minimal exertion;
- pain at rest (which may occur at night);
- angina that seems to be progressing rapidly despite increasing medical treatment.

Evidence of an MI in the ECG or dynamic changes suggestive of ischemia in the acute setting should be managed urgently.

11. Real Life - The Emergency Room Call

First, it is unlikely that you will be called to admit a patient who is pre-infarctional or actually infarcting or who has acute EKG changes. Such cases are likely to have been referred directly to cardiology. In this case, you have no role and should avoid implicating your supervising attending physician. However, should you find yourself asked to assess such a patient, it is worth stressing that the diagnosis is clinical, based on your examination and assessment, perhaps supplemented, but only supplemented, by laboratory findings. An elevated Troponin, for example, indicates probable infarction and waiting on such evidence represents an opportunity lost. As Dr. Halvorson has said repeatedly, “How do you revascularize a scar?”. Consequently, should intervention be indicated, it should be rapid and not delayed until a “convenient time”. However, before the subsequent cascade of events takes place, it is worth pausing briefly to consider whether or not intervention is justified. Age, co-morbidity and patient wishes are paramount and if doubt exists, and it is thought possible that the patient's personal physician may have special knowledge, then contacting him/her is worthwhile. Any of your faculty would be happy to advise on a personal patient, whether officially on call or not.

A much more likely situation is to be called to a patient with some form of symptomatology between the sternal notch and xiphoid. Risk factors will have been casually “cobbled together” to support the need for admission. Almost inevitably, normal bio-mechanical markers and EKG will already have been obtained. The patient may or may not have been started on a nitroglycerin drip, and may or may not have been anti-coagulated. Almost always, you will be told that the patient's symptoms improved after the administration of oxygen or nitroglycerin, although this does not usually represent a cause and effect relationship. Even then, cardiology may have been consulted but declined to admit the patient, suggesting instead that the “attending physician” (actually no-one at this stage), do so. There then arises two scenarios. In the first, the more preferable, the cardiologist contacts the primary physician on call and discusses the case leading to joint agreement on management and division of responsibilities. On the other hand, if the discussion has taken place only between emergency room staff and cardiology, who together have decided that a third party should undertake the responsibility for admission, no form of consultation, or consultant relationship sanctified by hospital by-laws, has taken place, and you should proceed as if no such communication had taken place as outlined next.

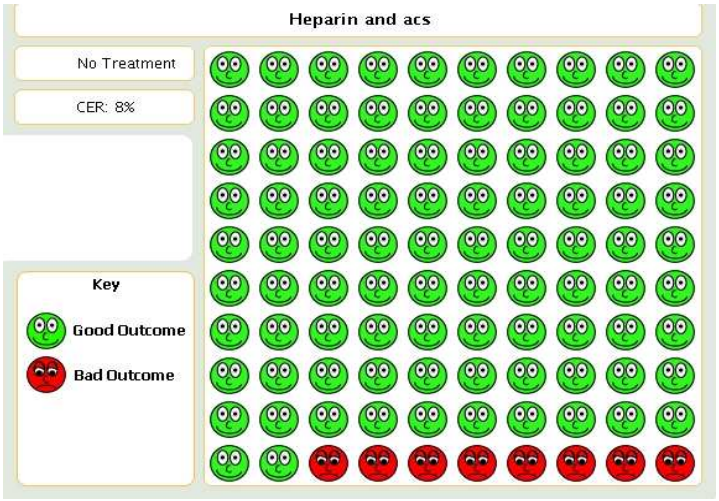
All of these are examples of **system failures –** breaks in the natural flow of care around a designated physician who then accepts responsibility for the direction of future care, even if not providing all aspects of that care himself/herself. Often innocent-looking or unrecognized, they are potent causes of sub-optimal care, unnecessary expense, and sometimes patient harm. Such breaches should be recognized together with the fact that all medical care takes place within systems. These situations are excellent teaching opportunities, and should be noted in sufficient detail to allow discussion at morbidity/mortality conferences.

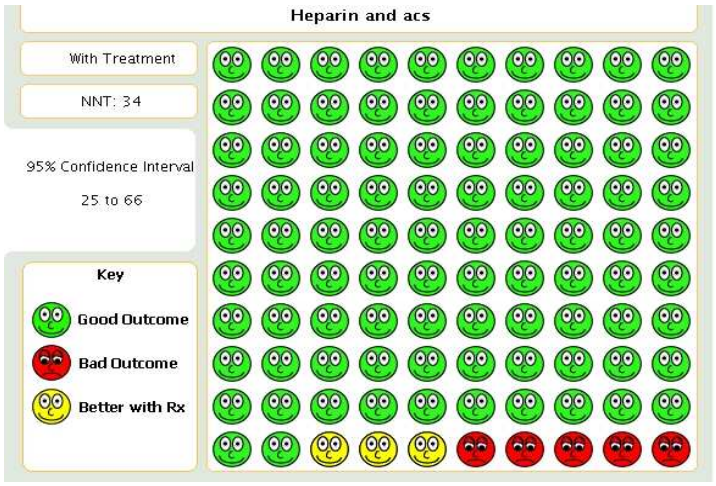
So what should you do when called to the patient with vague substernal symptomatology, negative laboratory and EKG findings, on a nitroglycerin and heparin drip?

First and foremost, begin afresh forming your own conclusions based on 1, 2, 3, 4, 5, 6 and 8.

You may conclude that this is non-cardiac. In such case, you should inform your supervising attending of your views, not simply repeating the opinions of others. Your attending physician may decide to interrupt the admission until he/she has had a chance to review the patient, with the possibility that the patient may be sent home and further diagnostic testing be carried out on an office basis.

You may decide that it is not possible to confidently exclude an anginal syndrome, but that the patient is neither infarcting nor pre-infarctional but that admission will be a matter of course. Should the patient receive nitroglycerin? Should the patient be anti-coagulated? The answer to both depends on, first, whether there is continuing pain, and second, where on the spectrum of illness severity you believe the patient lies. There is no such thing as 'standard of care' in this situation, unless you believe that all illness is binary. As regards anti-coagulation, the following pictograms may alleviate some anxiety and add some perspective concerning the use of heparin.





Finally, does nitroglycerin have any function other than the relief of pain? Other than some lowering of blood pressure due to reduced pre-load, for which there are probably preferable drugs, not that I know of. Other than the known pharmacologic effects, venodilation, reduced pre-load, diminished cardiac output with reduced myocardial oxygen demand, there has only been one randomized trial of 40 patients.

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