A 70-Year-Old Healthy Man With Chest Discomfort

Ronald N. Rubin, MD[1,2] —*Series Editor*

A 70-year-old man presented to your office with a history of chest "discomfort" of several months' duration. He is not precisely sure when it had started but knows it has been present for at least 3 to 4 months. His description of the discomfort is a "tightness and pressure sensation" rather than pain. It is located across his chest centrally and does not radiate. He has no associated shortness of breath and does not have discomfort or dyspnea at night while sleeping.

Initially, he had thought it was muscular since he is quite active in his metal working business, which requires lifting and exertion when operating his machine shops. When questioned in detail, he recognized that the pain might also be present during recreational activities. He had casually mentioned these symptoms to a friend, who had helped test the theory by jogging or running with the patient and then asking whether the discomfort was elicited. When the answer was indeed "yes," a prompt visit to your office was arranged.

Medical history

The patient is otherwise in good health. His only major medical diagnosis is a localized Gleason score 6 prostate carcinoma, which was diagnosed 3 years ago and treated with radiation therapy for curative intent. At his latest follow-up examination, his prostate-specific antigen level was 0.

He has never smoked, has no symptoms of chronic obstructive pulmonary disease or congestive heart failure, and does not have diabetes.

Physical examination

The patient was a thin, athletic, healthy-appearing man. His vital signs were within normal limits, including blood pressure (128/82 mm Hg). Examination findings of the head, eyes, ears, nose, and throat were normal. His chest was clear to percussion and auscultation. His cardiac rhythm was regular, and there were no murmurs or gallops. The remainder of the examination was noncontributory.

Diagnostic testing

Results of a complete blood cell count and metabolic panel were within normal limits, including his glucose (104 mg/dL) and hemoglobin A1C (5.0%) levels. His troponin levels were also examined and were not elevated. Lipid analysis was pending, but prior measurements had revealed only a slightly decreased level of high-density lipoprotein cholesterol.

An electrocardiogram showed normal sinus rhythm and no ST-T abnormalities, Q-waves, or acute injury current.

Which of the following is the optimal next step in the management of this patient?

A. He should immediately be taken to the cardiac catheterization suite for coronary angiography with intent for stenting or surgical bypass.

B. He should be scheduled for electrocardiography stress testing (treadmill testing).

C. He should be scheduled for a coronary computed tomographic angiography.

D. He should be scheduled for stress myocardial perfusion imaging.

Correct Answer: C. He should be scheduled for a coronary computed tomographic angiography

"But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it and the sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris. Those who are afflicted with it are seized while they are walking (more especially if it will be uphill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or continue but the moment they stand still, all this uneasiness vanishes...The pain is sometimes in the middle, sometimes the bottom of the os sterni and affected more

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inclined to the left than to the right side. It likewise every frequently extends from the breast to the middle of the left arm. Males are more liable to this disorder, especially such those past their fiftieth year..."

—William Heberden, MD, on Angina Pectoris, 17721

As will be discussed, the presented patient is manifesting angina pectoris, and I will introduce the topic with the second oldest reference in my repertoire, Sir William Heberden's original description delivered to the Royal Academy of Medicine in London in 1772.[1] Please invest the time to read it and note that he came to these observations by history and quite limited physical findings alone—no imaging, no laboratory studies, no electrocardiography (EKG). And yet, if any trainee in medical school could elicit and deliver such a stunningly accurate history for an ongoing patient, as well as grossly describe the natural history of angina pectoris as accurately and efficiently as Dr Heberden, then that trainee would have an honors grade to be sure. It is almost as though he was visualizing the coronary artery pathology and pathophysiology by using his powers of observation.

Returning to our era, we now know a lot, documented by many studies in large numbers of patients over long periods of time. We know that angina pectoris is brought about when myocardial ischemia occurs because of an imbalance of myocardial oxygen supply (related to inadequate coronary perfusion) in the setting of increased needs (related to effort, exertion, or emotional distress). By far, the most common pathophysiology that causes it is coronary artery atherosclerosis, wherein luminal plaque formation caused by a complex interaction of lipoprotein deposition and inflammatory processes narrows the lumen such that there develops a fixed stenosis that does not allow for increased coronary artery blood flow when required, hence the symptoms occur with effort and exertion.[2,3] As is typical for our ever more subclassifying every entity to small subspecific bits, there are modifiers used when describing angina. Two useful ones are "stable angina" and "unstable angina."

Stable angina clinically will demonstrate the typical chest pain features of angina—pain that is characterized as pressing constricting or dull; pain being brought on reproducibly by the aforementioned physical activity, exertion, or emotional distress; the pain-eliciting fear and anxiety as described by Dr Heberden yet abating within minutes once the trigger activity is gone or relaxed. The syndrome is stunningly reproducible in its behavior—what brings it on, what it feels like, and prompt resolution with rest.[2,3] I have had many patients with whom we could quantitate the numbers of subway steps required or household chores performed to elicit the angina. What this describes is stable angina pectoris and will be the entity discussed and for which the answer to the question is framed.

Once the stable pattern breaks or changes (eg, more frequent attacks involving less strenuous activity), then one is dealing with unstable angina. And if and when the angina or chest symptoms are present at rest, then we are dealing with an acute coronary syndrome. Both unstable angina and acute coronary syndromes are much more dangerous situations, requiring far more urgent and aggressive measures and will not be discussed further here. The presented patient had 4 to 8 months of classic symptomology, very titratable and unchanging frequency, trigger activities, and prompt resolution. I did not fabricate the clinical vignette, as I have personally observed these symptoms, interviewed and elicited the full history, and referred the patient for further care. Results of good routine studies had excluded acute coronary syndromes or acute myocardial infarction (eg, normal EKG without Q-waves, ST-segment injury currents, or left bundle branch block and normal troponins). Thus, it is stable angina, and the next issue is how to confirm the diagnosis anatomically, assess the degree of pathology (coronary stenosis extent and location), and with that information, then proceed to optimal therapeutics.

There is now an enlarging array of confirmatory testing for the presence of coronary artery disease as the cause of the angina syndrome. The studies vary in their use of functional testing vs anatomical presence/confirmation of coronary artery disease. The former utilizes some form of "stress" (eg, increases myocardial needs) with a variety of physiologic indications' responses to the stress.[2] So, in classical exercise stress testing, the stress testing is a treadmill, and the effect is ischemic changes demonstrated on the EKG scan. Both stress echocardiography and stress myocardial imaging utilize either exercise or pharmacologic stress and monitor left ventricular dysfunction (ejection fraction decrements and their location in a coronary artery distribution). Coronary computed tomography angiography scanning and invasive cardiac catheterization allow us to directly visualize coronary artery anatomy and physiology.[2,4] They all have their advantages and disadvantages, and guidelines are ever evolving.

Based on raw sensitivity and comparable specificity, recent review favors coronary computed tomography angiography (Answer C) as a first-line approach, although US guidelines have not as of yet been updated.[2,5-7] Answer B, classic treadmill testing, remains useful but has the weakest specificity/sensitivity figures and prognostic value. Stress testing would surely confirm the diagnosis (which is already quite apparent), but most clinicians would want to know more about the underlying pathology (and thus prognosis). Therefore, Answer B is not the optimal choice here.

Answer D, stress myocardial perfusion imaging, has good sensitivity and specificity (although less than coronary computed tomography angiography) and can give prognostic data such as extent/location of regional wall perfusion defects and degree of left ventricular dysfunction

TAKE-HOME MESSAGE

Angina pectoris remains a commonly encountered condition in the United States today. The clinical presentation, first elegantly and accurately described by Heberden 250 years ago, entails the onset of dull or pressing sensation substernally, frequently radiating to the left arm and neck, which is brought on by effort/exertion or emotional stress lasting minutes. Discomfort is essentially always relieved within minutes by rest. The term "stable angina pectoris" implies predictable, reproducible frequency, severity, causation, and reversibility. We now know that the pathophysiology involves imbalance between myocardial oxygen demand and supply, which is overwhelmingly caused by luminal stenosis within the coronary arteries. Angina pectoris will not cause fixed EKG changes or troponin spillage, which are part of more-advanced acute coronary syndromes. Confirmatory studies involve either functional demonstration of reversible myocardial ischemia by EKG or imaging or anatomic demonstration of the coronary stenosis. Examples of the former are stress electrocardiography, stress echocardiography, and stress myocardial perfusion scanning. Examples of the latter are computerized coronary anatomy tomography and invasive coronary angiography. All have good experiential records. The newer modality of computerized coronary anatomy tomography has overall superior sensitivity and specificity compared with other noninvasive modalities with lesser radiation exposure and is emerging as the optimal choice for patients initially suspected of a stable angina pectoris syndrome.

but carries the highest radiation load. And a subtle but definite factor in the presented patient is the significant past radiation exposure for curative intent therapy for prostate cancer. The traditional "gold standard" for coronary artery disease diagnosis is coronary angiography (Answer A), which confirms or refutes the diagnosis and, by demonstrating and quantitating extent/locations of coronary artery lesions, provides prognostic data. Newer data seems to indicate that coronary computed tomography angiography essentially matches that data yield without any of the bleeding risks (to be sure less frequent with current techniques) and utilization issues of invasive coronary angiography.[5-7]

Over the years, I've come to love cardiac catheterization angiography, a study that in itself provides diagnosis, prognosis, and (in selected patients) therapeutics using angioplasty/stenting and all within an hour or two in the catheterization laboratory. But data is accumulating that, as an initial study in a patient with stable angina pectoris, coronary computed tomography angiography seems optimal.

Patient Follow-Up

The patient was referred to a cardiologist and underwent a coronary computed tomography angiography scan within days. Although the findings of coronary artery disease as the cause for his angina were not surprising, the severity was. He had 85% stenosis in the mid-right coronary artery and more than 90% stenosis in the proximal left anterior descending artery. There were no fixed regional wall abnormalities, and his left ventricular ejection fraction was normal at rest, 57%. Invasive cardiac catheterization had followed. He had 2 stent placements separated by 4 weeks, and after cardiac rehabilitation, he was entirely asymptomatic at full and vigorous activity. Of course, he had a regimen of aggressive lipid control with a target low-density lipoprotein cholesterol level of less than or equal to 70 mg/dL using statins, as well as a course of standard antiplatelet agents followed by daily aspirin. He is currently well and active without any cardiac symptoms.

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