Is your patient's chest pain relatively benign or does it signal an impending cardiac arrest? Here's how to sort out the signs and relieve the heartache of angina.

A cry from the heart

LYLE TODD, 70, is in your emergency department with shortness of breath and chest pain that woke him from sleep. He describes the pain as a heavy, nonradiating, burning sensation that he rates as a 10 on a scale of 0 (no pain) to 10 (worst pain).

A month ago, Mr. Todd had angioplasty of the right coronary artery. You suspect that he's now experiencing angina pectoris—the distinctive chest pain caused by myocardial ischemia—possibly from restenosis of the right coronary artery. Because angina can cause the same pain as an impending myocardial infarction (MI), your next steps are crucial.

In this article, I’ll discuss how to assess a patient like Mr. Todd and initiate appropriate interventions. I’ll also review both conventional and investigational treatments for angina. But first, let’s examine the causes and types of angina.

Sending out an SOS

Although coronary artery disease (CAD) is the leading cause of angina, anything that decreases blood flow and oxygen delivery to the heart muscle—such as hypertension, tachycardia, and coronary artery spasms—can produce it. Angina may be the first sign of CAD or indicate a worsening of the disease and the potential for MI. Ninety percent of patients with recurrent angina have significant coronary artery stenosis or occlusion. (See Problems in the Pipes.)

Angina is grouped into three categories:

- **Stable angina** is chest pain that occurs in a predictable fashion (for example, following exercise) and that hasn’t changed in frequency, duration, or precipitating factors in the previous 60 days.
- **Unstable angina** is unpredictable chest pain lasting 5 to 30 minutes per episode; it typically occurs at rest and is of new onset. This type of angina often increases in frequency or duration and is more painful than other types of angina.
- **Prinzmetal’s, or variant, angina**, the least common type of angina, also can be severe and prolonged. The pain is sim-

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Taking control of angina

To prevent Mr. Todd's suspected myocardial ischemia from progressing to infarction, you'll need to intervene quickly. You escort him to a bed and elevate the head 30 to 45 degrees to make him more comfortable and promote cardiovascular function. Connect him to a cardiac monitor, administer oxygen at 4 liters/minute via nasal cannula, and start an intravenous (I.V.) line with 0.9% sodium chloride solution at a keep-vein-open rate. You take Mr. Todd's vital signs: temperature, 97.5° F (36.4° C); pulse, 64; respirations, 18 and nonlabored; and BP, 110/68.

Call for a stat 12-lead electrocardiogram (ECG) and notify the physician. Begin pulse oximetry and give Mr. Todd one sublingual nitroglycerin tablet, followed by two more tablets at 5-minute intervals if needed. After each dose of nitroglycerin, take his vital signs and monitor for hypotension. Draw blood for cardiac enzymes (to rule out MI), hemoglobin and hematocrit, and a chemistry profile.

While Mr. Todd has pain, monitor his BP, heart rate, and respiratory rate every 5 minutes. When you assess his heart tones, you hear S1 and S2 with no murmur or extra heart sounds. His lungs are clear bilaterally.

The 12-lead ECG shows normal sinus rhythm with inverted T waves in leads II, III, and aVF. Mr. Todd is pain-free after two nitroglycerin tablets taken 5 minutes apart. (If the maximum dose of three tablets hadn't relieved the pain, you'd have given morphine as ordered.)

As you treat Mr. Todd, you conduct a focused assessment:

- **Ask the patient to describe the pain and its location.** Is it sharp, dull, burning, or throbbing? Is it in the chest only, or does it radiate? If so, to which parts of the body? Is the pain related to activity? Does it worsen with deep breathing or when the patient is lying down? Ask him to rate the pain on a 0-to-10 scale.

- **Is he experiencing other signs and symptoms, such as nausea, dizziness, palpitations, or diaphoresis?**

- **Does he have any risk factors for angina?** Patients who are genetically predisposed to angina or have Type 1 diabetes, as well as those (like Mr. Todd) who are male, non-Hispanic, and between ages 65 and 74, are at higher risk for angina. Modifiable risk factors include hyperlipidemia, hypertension, smoking, Type 2 diabetes, obesity, sedentary lifestyle, stress, and heavy alcohol use.

- **Does he have a history of MI or angina?** Ask Mr. Todd if this pain is similar to past angina pain.

- **What medications is he taking?** Mr. Todd takes an enteric-coated aspirin daily and 50 mg of metoprolol b.i.d.

  Mr. Todd is admitted to the telemetry unit with a diagnosis of unstable angina.

  He's continued on metoprolol and aspirin, and heparin is started with an 80-units/kg bolus and an 18-units/kg/hour infusion.

  That evening, the telemetry alarm sounds, alerting the staff to ventricular bigeminy with a rate of 38.

  The telemetry nurse calls for a stat ECG. Mr. Todd is ashen, severely diaphoretic, and slightly nauseated. His vital signs are: pulse, 77 and slightly irregular; respirations, 22 and labored; and BP, 180/98. The stat ECG shows no change from his admission ECG. His pain is relieved with three sublingual nitroglycerin tablets, and supplemental oxygen continues. The bigeminy resolves on its own.

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Atherectomy. In this procedure, a rotating shaver on the catheter tip is used to remove plaque. Atherectomy is typically used for well-defined lesions that can be easily reached without excessive risk, for new lesions, and for restenosis of a previous coronary artery bypass graft.

CABG surgery. This is often the choice for treating multivessel disease and occlusions of 75% or more.

Minimally invasive direct CABG. This new technique is used to treat uncomplicated single- and double-vessel disease. Performed via small (5-cm [2-inch]) incisions in the thoracic, sternum, or epigastric area, this technique avoids the standard mid sternotomy incision and use of cardiopulmonary bypass and cardioplegia.

Percutaneous transluminal coronary angioplasty (PTCA), with or without stent insertion, is typically used in patients with single- or double-vessel disease where the blockage is less than 50% or the patient has adequate collateral blood flow to the left side of the heart. To reduce the 25% to 30% risk of restenosis, physicians at the facility where I work routinely place a stent in patients having PTCA.

Drugs in the new glycoprotein IIb-IIIa inhibitor class are used to improve the results of PTCA and stenting by preventing early restenosis (within the first 6 months), coronary dissection, or stent thrombosis. Abciximab (ReoPro) prevents early restenosis and stent thrombosis by inhibiting platelet aggregation. Abciximab is used routinely at most centers with patients who receive PTCA with stenting because of its ability to improve vascular patency and reduce residual stenosis.

Eptifibatide (Integrilin), a synthetic heptapeptide inhibitor, is also used to reduce the thrombotic complications of angioplasty. In one trial, eptifibatide provided benefits for patients undergoing PTCA without raising the risk of major bleeding or other adverse reactions.

The facility where I work is researching tirofiban (Aggrastat), a novel nonpeptide antagonist, to determine whether it's effective for preventing thrombi and inhibiting platelet function in patients with unstable angina and non-Q-wave myocardial infarctions.

Transmyocardial revascularization (TMR). Indicated for intractable angina, this procedure may keep a patient from needing heart transplantation. It can also be used for patients with severe ischemia, previous bypass, or occluded vessels too small to bypass. In TMR, a high-powered carbon dioxide laser creates transmural channels that allow blood to flow to the ischemic myocardium. Early results have been promising, but TMR is still too new to be used as a primary treatment.

Nitroglycerin, a vasodilator, is usually the first line of defense. It relaxes peripheral veins, which decreases preload and (to a lesser extent) afterload, and relaxes the coronary arteries, which increases coronary blood supply.

Beta-blockers (including metoprolol and atenolol) reduce the heart's workload and oxygen demand by decreasing heart rate and reducing peripheral resistance to blood flow.

Aspirin, an antiplatelet medication, is included in the regimen to reduce the risk of thrombus formation and coronary artery occlusion.

Other drugs that may be prescribed include heparin, to prevent clots and reduce the risk of MI, and nitrates, which dilate coronary arteries and increase coronary blood flow. Calcium channel blockers, such as diltiazem, nifedipine, and verapamil, can prevent coronary artery spasms associated with variant angina. These drugs may be used in combination with nitrates and aspirin to reduce the cyclic pain of variant angina.

Medical management is the first choice for all types of angina, and stable angina generally responds to drugs. Unstable angina, however, may not respond to medical therapy, so further treatment, including invasive procedures, may be warranted. For more on traditional treatments and some new approaches, see Opening the Blockage.

Caring for Mr. Todd
Once stabilized, Mr. Todd is taken to the catheterization lab, where, after the restenosis of his right coronary artery is confirmed, he undergoes percutaneous transluminal coronary angioplasty (PTCA). During the PTCA, a stent is inserted and he receives abciximab (ReoPro). After an uncomplicated 3-day hospital stay, he's discharged home on aspirin and ticlopidine (another antiplatelet medication) to prevent stent thrombosis.

By knowing about the many options available for managing angina, you can help patients and their families select the most appropriate treatment.

SELECTED REFERENCES

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