
Problems in Diagnosing Acute Mitral Regurgitation Due to Coronary Artery Disease

Chronic mitral regurgitation in patients with coronary artery disease is common. One recent series employing coronary arteriography and left ventriculography in 120 patients reported 39 (31 percent) with this finding. As expected, these 39 patients had a high frequency of prior myocardial infarction and left ventricular asynergy, factors known to contribute to papillary muscle dysfunction. Mitral regurgitation due to coronary artery disease can also occur on an acute basis during transient myocardial ischemia or acute myocardial infarction. In the latter setting, it may at times be severe. In both settings, the diagnosis of acute mitral regurgitation may present problems, though for different reasons.

Transient ischemia: It is not unusual for a patient with a history of angina to have a new holosystolic or late systolic murmur during an episode of chest pain. When the papillary muscle dysfunction is due to severe papillary muscle ischemia and/or left ventricular asynergy, the regurgitation can be pronounced, leading to sudden signs and symptoms of frank left-sided failure. However, if the regurgitation is not severe enough to cause such signs or symptoms (as it only rarely is), and auscultation is not performed, the diagnosis will not be made. In some patients, there may be no symptoms during acute ischemia, anginal or otherwise. (This phenomenon of silent myocardial ischemia is more common than realized and may be considered as a manifestation of a defective anginal warning system.) When there are no symptoms of either failure or angina, the index of suspicion for acute mitral regurgitation due to coronary artery disease is low, and many instances of mild to moderate mitral regurgitation will be undetected. This diagnostic problem is confounded by the variability of the murmur, since some ischemic events will precipitate it while others will not.

Acute infarction: In this setting, the patient is routinely examined and the prevalence of acute mitral regurgitation clinically has been reported to be as high as 56 percent. The acute mitral regurgitation is occasionally severe, since the base or head of the papillary muscle may become partly—or occasionally completely—necrotic. The murmur can be silent or barely audible, however, when there is enough impairment of left ventricular function to markedly depress cardiac output. Because of this feature, Friedman and Stein have stressed in this issue of Chest (see page 436) that the bedside diagnosis of severe acute mitral regurgitation may be difficult in very ill patients with both ischemic and nonischemic heart disease. A high index of suspicion for this lesion is required to obtain the necessary bedside hemodynamic measurements that will confirm the diagnosis. It should be noted, however, that the hemodynamic profile is not always clear cut. For example, it is often stated that since the left atrium is small and relatively noncompliant, large V waves in the pulmonary capillary wedge pressure tracing are the expected finding, but if severe acute mitral regurgitation is superimposed on chronic moderate regurgitation, left atrial size and compliance may be increased, effectively dampening out the V waves. Unless the physician knew of the presence of the prior lesion and obtained a left ventricular angiogram, he might wrongly conclude from the presence of a silent murmur and absence of giant V waves that severe acute regurgitation was not the cause of the deteriorating condition. The correct diagnosis in this situation could also be made by the echocardiographic demonstration of increased left atrial size and a flail mitral leaflet. (A similar sequence of events may occur in nonischemic disease when a ruptured chorda tendineae is superimposed on chronic mitral regurgitation due to mitral valve prolapse.) There is also the situation in acute myocardial infarction when large V waves in the presence of a systolic murmur do not indicate severe mitral regurgitation, but rather a ruptured ventricular septum. The physical findings of a ruptured ventricular septum are usually sufficiently different from those of mitral regurgitation to make the diagnosis, but at times, the location and quality of the murmurs can be perplexingly similar. Because the two lesions may be confused both clinically and hemodynamically, oxygen samples from both right atrium and pulmonary artery should be obtained as the balloon catheter is floated into place. This is simple enough to do, and absence of a step-up in oxygen saturation effectively rules out a ventricular septal defect.

In conclusion, it is apparent from these comments that acute mitral regurgitation due to coronary artery disease is not uncommon, can range from mild...
to severe, and may represent a challenging diagnostic problem to the clinician.

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REFERENCES

A Doctor’s Dilemma—1980

Physicians in the coming decade will be faced with an increasing population of patients who have impaired host defense mechanisms. This will occur as a result of continued development of new therapies for malignant diseases and their hemorrhagic and infectious complications. It is therefore important that we develop a rational approach to the occurrence of lung infiltrates in such patients to use efficiently the effective diagnostic and potential therapeutic measures currently available.

Tenholder and Hooper in this issue (see page 468) show how this can be accomplished for an important subset of compromised hosts, those with leukemias. What are the common etiologies of infiltrates in leukemic patients? Which of these etiologies are treatable? In which group of patients is the risk of an invasive diagnostic procedure justified by potential improvements in therapy? The authors review 139 leukemic patients in an attempt to answer these questions. They evaluate the radiologic characteristics of disease before and during chemotherapy and examine the risk of common invasive diagnostic procedures. They conclude that biopsies are of substantial value only during antineoplastic therapy and are more useful in patients with diffuse than in those with local disease.

The authors demonstrate that a careful analysis of each patient’s problem correlated with the natural history of their disease, their current clinical and roentgenologic findings, and the application of medical logic allows for rapid, appropriate decisions regarding management of a majority of cases. This avoids the empiric approach, all too frequently practiced at the present time, regarding both diagnosis and treatment.

A review of the literature on this topic emphasizes several caveats regarding such patients:

1. Even after lung biopsy or autopsy, many infiltrates (up to 35 percent in some series) in immunocompromised hosts will not have a specific diagnosis.

2. Differential diagnosis for each of these patients must include:
   a) complications of chemotherapy or radiation;
   b) progression of the primary disease process;
   c) hemorrhage or edema; and
   d) infection, opportunistic or not, with a spectrum of etiologies that includes the viral, bacterial, granulomatous, ricketsial-like, or protozoal.

3. To manage these patients’ illnesses, the nature of the basic derangements of their defense mechanisms and their projected duration must be appreciated. For example, an underlying cancer may have long-term effects on the humoral immunity, while chemotherapy may transiently compound the problem by decreasing granulocyte and lymphocyte defenses.

4. A team approach is certainly indicated, owing to the enormous gravity and complexity of many such problems and the need for full diagnostic facilities and surgical skills. This includes both technical (for example, fiberoptic bronchoscopic techniques to obtain secretions) and laboratory serologic and microbiologic diagnostic capabilities. If these are not available, transfer of the patient should be considered.

A most difficult aspect of management is determination of the role of invasive procedures, and each proposed intervention deserves careful risk-benefit analysis. The risks include the morbidity and mortality of the procedure, as well as the emotional distress resulting from an uninformative diagnostic attempt. The potential benefits include certainty of diagnosis, with its implied advantages of more precisely directed and less toxic therapy, greater hope for recovery without side effects, and potential for scientific contribution. In the severely compromised host, the inability to provide meaningful treatment for the underlying disease may, from a practical standpoint, lessen to insignificant proportions the value of any procedure to define