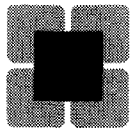

WILLIAM S. WILKE, MD, EDITOR

AGGRESSIVE INTERVENTION IN MI REDUCES MORTALITY

Current treatment of the acute phase of myocardial infarction (MI) is aggressive intervention aimed at recanalization of the artery, to salvage myocardium and improve survival. Both thrombolytic therapy and percutaneous transluminal coronary angioplasty (PTCA) have demonstrated efficacy in the treatment of acute MI.

THROMBOLYTIC THERAPY

The rationale for thrombolytic therapy with streptokinase, urokinase, or tissue plasminogen activator (tPA) is related to the pathophysiologic mechanism of acute MI. The formation of a new thrombus, superimposed on a pre-existing atheroma, is triggered by the cascade of events that leads to occlusion of the vessel with myocardial anoxia and infarction. In every case, there is a "window of time" during which the myocardium is viable and can be salvaged with reperfusion. To be effective, thrombolytic therapy must be administered during this period—within 6 to 8 hours of the onset of symptoms. The amount of myocardium with reversible damage diminishes rapidly, so thrombolytic therapy is a race against time.

Mortality during the acute phase of MI drops by nearly half when streptokinase is started within 2 hours of the onset of acute MI; when streptokinase therapy is delayed until 9 to 12 hours after onset, the mortality rate is comparable to that observed with conventional therapy. The addition of aspirin to the streptokinase regimen during the acute treatment has been shown to further reduce mortality.

PTCA

Thrombolytic therapy has limitations, including bleeding complications. Furthermore, there is a 15% to 35% likelihood of reocclusion, particularly in the presence of a large atheroma and significant narrowing.

TABLE
CLEVELAND CLINIC FOUNDATION APPROACH TO ACUTE MI

1. Thrombolytic therapy administered in the emergency room within 6 to 8 hours of onset of MI.
2. Transfer as soon as possible for cardiac catheterization to establish anatomy.
3. PTCA if artery is occluded or if pain continues (occurs in 45% of patients).
4. Elective PTCA or coronary artery bypass surgery after 7 days, if indicated by anatomy.

The reocclusion rate is significantly lower when PTCA is performed after thrombolytic therapy. PTCA prevents rethrombosis by reducing the size of the atheroma. Ongoing clinical trials, which include the Cleveland Clinic Foundation, have achieved greater than 95% successful recanalization with PTCA.

Bleeding complications are more likely among patients who undergo PTCA in the acute phase of treatment than among those who undergo the procedure electively. Given this limitation, the current recommendation is to administer thrombolysis within 6 to 8 hours of the onset of acute MI. If the patient's condition remains stable, then elective catheterization and PTCA are performed after one week. If pain continues or the occlusion remains despite thrombolytic therapy, PTCA may be indicated during the acute phase.

PTCA has proved to be particularly beneficial in the treatment of cardiogenic shock, which has a mortality rate of 90% to 95% with conventional therapy. Treatment with the intra-aortic balloon and dopamine reduces mortality to 70% to 75%. Acute intervention with PTCA has reduced the mortality rate to 30%.

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HIV MAY BE A CAUSE OF MUSCULOSKELETAL SYNDROMES

Musculoskeletal and connective tissue disease syndromes are developing in increasing numbers of patients

with human immunodeficiency virus (HIV) infection. These HIV-associated syndromes may mimic the idiopathic varieties (e.g. Sjogren's syndrome, polymyositis, and systemic lupus erythematosis [SLE]), presenting difficult diagnostic and treatment dilemmas, and the implications of misdiagnosis are profound. Although the HIV-associated syndromes mimic idiopathic connective tissue disease, they develop from immunopathogenic mechanisms. The idiopathic varieties generally reflect overactivity of the immune system and respond to immunosuppressive treatment such as steroid therapy—an approach that would be disastrous in the setting of HIV infection.

To minimize the likelihood of misdiagnosis and inappropriate treatment, HIV detection tests are warranted for any patient with apparent connective tissue disease who is at risk for HIV, or who does not fit the characteristic patient profile.

The frequency of musculoskeletal symptoms in patients with HIV infection ranges from 40% to 72%, according to studies in New York, Cleveland, and Tampa. Most of these patients are in the final stages of HIV infection, and the musculoskeletal symptoms may be manifestations of chronic opportunistic infection. But certain syndromes warrant further evaluation.

ARTHRITIS

The triad of psoriasis, arthritis, and HIV has been observed in as many as 20% of patients seen in HIV clinics in certain areas. In some cases, this group of symptoms may have been the first manifestation of HIV infection.

It has been suggested, but not proven, that Reiter's syndrome is represented disproportionately in HIV-infected individuals, with a frequency of 10% to 20%. The syndrome is generally characterized by arthritis, ocular inflammation, and urethritis, but it encompasses a broad spectrum of disease, including spondylitis in HLA B27-positive individuals.

A mono- or oligoarticular arthritis has been reported. The arthritis is inflammatory and sterile, lasts from days to weeks, and affected patients are HLA B27-negative and have no distinguishing features other than HIV infection. Because there have been reports of opportunistic infections in joints, monoarticular arthritis that is inflammatory should be considered infectious until proven otherwise.

SJÖGREN'S SYNDROME

A syndrome that mimics idiopathic Sjögren's syndrome is being reported increasingly in HIV patients. A recent report from New York University noted that biopsies of minor salivary glands from patients with HIV-associated Sjögren's syndrome were indistinguishable by light microscopic analysis from idiopathic Sjögren's syndrome. Despite the histologic similarities, there are immunopathologic and clinical differences. Idiopathic Sjögren's syndrome is characterized by a hyperactive humoral limb and an infiltrate rich in CD4 cells. The infiltrate in HIV-associated Sjögren's syndrome is characterized by CD8 cells or cytotoxic T-suppressor cells. Idiopathic Sjögren's syndrome is predominantly a disease of women; HIV-associated Sjögren's syndrome affects primarily men. Autoantibodies are an infrequent finding in HIV-associated Sjögren's syndrome, compared to the idiopathic variety. Because treatment for the two conditions differs, HIV should be considered in all men with clinical Sjögren's syndrome and a low antibody profile.

LUPUS SYNDROMES

It is difficult to distinguish SLE from HIV-associated lupus-like syndromes. The HIV-infected individual commonly presents with a history of fever, malaise, modest weight loss, and an erythematous, scaly facial rash. Examination is likely to reveal arthralgias, lymphadenopathy, thrombocytopenia, modest proteinuria, polyclonal hypergammaglobulinemia, and a low positive ANA. The same constellation of findings is characteristic of SLE. Because of the similarities in laboratory and clinical findings, suspicion of HIV depends on the natural history and target organ disease, as well as recognition of the patient in a high-risk population. As noted, HIV detection testing is warranted if the etiology is doubtful.

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C *PYLORIS*: ROLE IN GASTRIC MUCOSAL DISEASE

The organism *Campylobacter pyloris* is found in an increasing percentage of patients with chronic gastritis