and control of other risk factors.

However, diet and life-style modification often may not be sufficient to attain the goals mentioned above, and pharmacotherapy may be required. The following are four scenarios for the use of lipid-altering drug therapy.

1. *Elevated LDL-C, triglyceride levels <200 mg/dL.* Use a hydroxymethylglutaryl coenzyme A (HMG CoA) reductase inhibitor ("statin" drug), equivalent to 20 to 40 mg of lovastatin or pravastatin or 10 to 20 mg of simvastatin). Then consider combination therapy: add a bile-acid sequestrant to lower the LDL-C and consider niacin to increase the HDL-C.

2. *Elevated LDL-C, triglyceride levels 200 to 400 mg/dL.* Use niacin, but with the understanding that patients frequently do not tolerate an adequate dose. Statin drugs may not control HDL-C and triglycerides in these cases. Combination therapy is generally indicated: eg, a statin drug plus niacin, or a statin drug plus gemfibrozil. These regimens carry an increased risk of myopathy, especially when a statin is combined with gemfibrozil. Pravastatin may be less likely to cause myopathy. These patients are at very high risk, so referral may be appropriate if you do not feel comfortable with combination therapy.

3. *Triglyceride levels >400 mg/dL.* Use gemfibrozil or niacin. After triglycerides are lowered you may need to control the LDL-C.

4. *HDL-C <35 mg/dL, LDL-C <130 mg/dL, triglycerides <200 mg/dL.* Use niacin or statin. Gemfibrozil produces about the same increase in HDL-C as the statins. Statins are more effective in lowering LDL-C.

Most patients with established atherosclerotic vascular disease are candidates for drug therapy. Future studies will define the need for additional drugs, such as antioxidants. The role of hormone replacement therapy is also being defined. Many postmenopausal women with established disease may be candidates for this treatment.

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SUGGESTED READING

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. JAMA 1993; **269**:3015—3023.

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RHEUMATIC MANIFESTATIONS OF HIV INFECTION

In patients infected with the human immunodeficiency virus (HIV), the development of rheumatic conditions—including Reiter's syndrome, psoriatic arthritis, HIV-associated arthritis, myopathy, and Sjögren's syndrome—show that, besides inducing a state of immune deficiency, HIV also leads to a state of profound immunodysregulation. Treatment of these diseases is problematic, given that many of the standard therapies are themselves immunosuppressive. But the occurrence of rheumatic diseases among HIV-infected patients may provide added insight into the pathogenesis of clinical rheumatic diseases.

The association of rheumatic disease with HIV infection means that physicians need to take candid, nonjudgmental histories for HIV-associated risk behavior in all new patients. Physicians, especially those whose practice does not include many HIV patients, need to be aware of the signs and symptoms of HIV infection, including unexplained weight loss, diarrhea, fatigue, cutaneous herpes zoster, and lymphadenopathy. Physical signs that may appear include thrush, hairy leukoplakia, and cutaneous fungal infections.

For physicians who treat a large number of HIV patients, recognition of rheumatic conditions is both challenging and important. For example, the aches and pains of spondyloarthropathy may be confused with peripheral neuropathy. Conditions such as HIV-associated myopathy may be difficult to recognize. The design of aggressive therapies for both the underlying HIV infection and the clinical rheumatic state needs further development.

Interestingly, not a single case of classic rheumatoid arthritis has been reported to develop during an

established HIV infection. In patients who have rheumatoid arthritis and become infected with HIV, the arthritis has gone into high-grade remission. Similar findings have been seen among some patients with systemic lupus erythematosus, suggesting that CD4 cells may play an important role in the pathogenesis of rheumatoid arthritis and systemic lupus erythematosus. Because other conditions such as Reiter's syndrome and the spondyloarthropathies can occur in conjunction with HIV infection, CD4 cells probably play a lesser role in their pathogenesis.

Occurrence of rheumatic diseases in association with HIV infection poses an interesting therapeutic dilemma. Most conventional therapies are immunosuppresive, an obvious problem for HIV patients. For instance, in Reiter's syndrome methotrexate may be less desirable and may lead to a rapid progression of the underlying HIV infection. Low-dose cyclosporine has met with some success, but only in a small study. Other disease-modifying drugs like gold, penicillamine, and hydroxychloroquine are untested in controlled studies.

The problem presents itself in other diseases, too. Zidovudine may have some effect in controlling the skin disease of psoriatic arthritis, but rarely is it effective for controlling the articular manifestations. The difficulty in treating rheumatoid disease clearly indicates the need for more creative protocols.

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