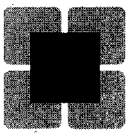

WILLIAM S. WILKE, MD, EDITOR

DIAGNOSIS AND TREATMENT OF LOWER-EXTREMITY ARTERIOSCLEROSIS

Patients with lower-extremity arteriosclerosis obliterans (ASO) should receive aggressive medical treatment, which should include management of cardiovascular risk factors. Complications of atherosclerosis (stroke, myocardial infarction) are the leading causes of morbidity and mortality in the U.S., and most physicians are familiar with diagnosis and management of these disorders. However, lower-extremity ASO—typically a marker for generalized atherosclerosis—is often misdiagnosed and not managed optimally.

CLAUDICATION AND OTHER SIGNS AND SYMPTOMS

Patients with lower-extremity ASO usually present with intermittent claudication, ischemic rest pain, or ischemic ulcerations. They may describe the pain of claudication as aching, tightness, cramping, tiredness or soreness in the legs brought on by exercise and relieved by a short period of rest.

A common misconception is that "a little" claudication is inconsequential and requires no treatment. Intermittent claudication in the lower extremities is analogous to angina pectoris in the heart; it is not a benign disease. The Framingham study demonstrated an average annual mortality of 39/1000 for men with intermittent claudication compared to 10/1000 for men without the disease.

Seventy-five percent of these deaths are due to cardiovascular disease. Life expectancy is decreased 10 years overall, and only 26% of patients with intermittent claudication are still alive at 15 years. The incidence of coronary artery disease proven by coronary arteriography is 30%–90% in persons with intermittent claudication, and many of these individuals have no cardiac symptoms.

VALUE OF NONINVASIVE VASCULAR LAB TESTS

The noninvasive vascular laboratory (pulse volume recordings and Doppler examination) is the initial diagnostic test of choice for lower-extremity ASO. It is important to perform an examination at rest and after exercise on the treadmill to uncover mild arterial narrowing. Arterial waveforms and systolic blood pressures are measured before and after exercise. These pulse volume recordings help answer several questions:

1. Determining whether arterial insufficiency is present.
2. Predicting the level at which there is major occlusive disease (iliac, superficial femoral, below knee, small vessel).
3. Providing a *functional* assessment of vascular insufficiency.

Arteriography, on the other hand, should be performed only when considering some form of interventional therapy such as surgery, percutaneous transluminal angioplasty, atherectomy, or laser angioplasty. Arteriography provides only an anatomical assessment of the atherosclerosis, not a functional assessment.

TREATMENT

A thorough treatment program for lower-extremity ASO includes:

1. Modifying risk factors. Lifestyle changes are perhaps the most effective means of reducing symptoms: discontinuation of cigarette smoking and tobacco in all forms, meticulous control of blood pressure, normalization of the blood lipid profile, and attaining ideal body weight.
2. A walking program. Patients should walk for 45–60 minutes a day. They should be instructed to walk until they get discomfort in their leg(s), stand and wait 3–5 minutes until the discomfort goes away, and then continue walking. This helps by increasing their walking distance (i.e., training effect) and it helps to develop good collateral circulation around the area of arterial obstruction. Patients who follow a walking program can often double, triple, or even quadruple their walking distance.
3. Medications. Anti-platelet agents and direct-acting vasodilators are uniformly ineffective in patients with lower-extremity ASO. Pentoxifylline (Trental)

may be helpful in some patients with intermittent claudication.

Patients should be considered for some type of intervention if they develop rest pain, ischemic ulcerations that will not heal, or have intermittent claudication that interferes with their livelihood or lifestyle.

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AGGRESSIVE PREVENTION IS KEY TO OSTEOPOROSIS CONTROL

The diagnosis and treatment of osteoporosis are controversial. Although forearm densitometry is widely used, its value is questionable; the currently approved treatment modalities—calcium, vitamin D, and estrogen supplementation—all stabilize existing bone density, but do very little to stimulate new growth. Sodium fluoride appears to cause a linear increase in skeletal density, but there are unresolved questions about the effects of long-term use. Given this somewhat grim outlook, prevention remains the best way to control the disease.

DIAGNOSIS

Bone densitometry of the forearm, which measures mainly cortical bone density, was advocated at one time as a screening method for osteoporosis. However, measurement of trabecular bone by dual photon absorptiometry (DPA) or quantitative computed tomography (CT) is more sensitive, because trabecular bone is lost earlier and at a faster rate. The primary value of densitometry is to monitor treatment designed to stimulate bone growth and identify patients at risk. It is superfluous for diagnosis in older patients who already have radiographic evidence of osteoporosis, but it has great value in high-risk middle-aged asymptomatic patients. In this population group, bone loss is better measured in the lumbar region, where trabecular bone is lost earliest.

CT is diagnostic, but it is expensive and impractical for routine office practice. DPA machinery can be acquired for a fraction of the cost of CT equipment.

THERAPY

Most therapy in use today stabilizes existing bone density. A few experimental modalities stimulate new bone growth, but these do not correct the destruction of osteoporotic bone. Rather, they stimulate the growth of new bone over the existing framework which, in the case of osteoporosis, is weak.

Calcium

The usual calcium intake for women age 35 and older is 500 mg/d; yet the postmenopausal woman needs 1,500 mg/d, and men and premenopausal women should consume 1,000 mg/d. If dietary calcium is inadequate, then hormonal systems draw on the bone to meet daily calcium requirements. Adequate dietary calcium will block this hormonal "robbing" of the skeleton.

Calcium alone will not protect the skeleton of a woman in early menopause. Although calcium is somewhat protective of cortical bone, it has little effect on trabecular bone, which is estrogen sensitive.

Estrogen

Estrogen is the best modality for maintaining skeletal integrity in the early menopausal years. Women who are not on estrogen supplementation have a decrease in bone density. Estrogen therapy will help stabilize bone mass at its existing level, but it must be continued indefinitely. Upon discontinuation, bone mass deteriorates rapidly. Estrogen therapy has little protective value when begun in late menopause because so much bone may have already been lost. But excessive bone loss may not have occurred; density measurements can determine this.

Sodium fluoride

Long-term sodium fluoride therapy causes a linear increase in bone density at dosages ranging from 40 to 80 mg/day. Gastrointestinal side effects—primarily nausea and vomiting—are largely eliminated by a new formulation that is now being studied. However, we do not have all the answers regarding the consequences of long-term use. To minimize the risk, serum fluoride levels should be maintained at 90–195 ng/ml. Sodium fluoride appears to have little efficacy in the prevention of hip fractures, but it may help to prevent spinal fractures.