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unusual fungal pathogens or superinfection with resistant bacteria can cause additional complications. Computed tomography of the sinuses provides a quick and cost-efficient means of diagnosis. The recommended management is antibiotic therapy; patients who fail to respond may require endoscopic drainage of the sinuses.

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A MIMIC OF VASCULITIS

Among the many disorders that mimic vasculitis, atheromatous embolization (AE) syndrome is especially challenging to the diagnostician and produces significant patient morbidity and mortality. AE may appear in a variety of ways, and physicians in many different specialties see patients with it. Despite this, AE has received little attention in major medical textbooks and is frequently misdiagnosed or unrecognized by physicians.

PATHOPHYSIOLOGY

AE syndrome involves the noncardiac embolization of atheromatous plaques that contain red blood cells, fibrin, platelet aggregates, and cholesterol crystals, and is sometimes referred to as "cholesterol embolization." It is seen most often in patients with advanced atherosclerosis.

AE can occur spontaneously; however, three events are known to precipitate AE: abdominal surgery, anticoagulation, and arteriography. Abdominal surgery or any surgery involving manipulation of the aorta carries an obvious risk for AE in the presence of diffuse, severe atherosclerosis. Anticoagulation with heparin or coumadin apparently leads to AE by causing the release of atheromatous material from the aorta, although the precise mechanism is not known. Arteriography is the most common precipitator of AE; it is not uncommon for patients to undergo arteriography, then come back 3 to 4 weeks later with an elevated serum creatinine related to atheromatous emboli.

Interestingly, the incidence of AE is very low (approximately 0.7%) when coronary arteriography is done via the arm, whereas the incidence is much greater when done via the leg, no matter how flexible the guidewire.

DIAGNOSTIC CLUES

AE is a multisystem disorder involving the skin, kidneys, central nervous system, and gastrointestinal tract; it causes a host of constitutional symptoms such as fever, malaise, weight loss, and anorexia, simulating vasculitis or underlying malignancy.

Skin changes include livedo reticularis and purple or blue toes and are the most common manifestations of AE. Livedo reticularis is due to embolization to the dermal blood vessels. If these lesions are biopsied, typical cholesterol clefts are seen microscopically. Livedo reticularis may occur associated with various forms of vasculitis and may also be a normal finding in young, healthy women who have increased vascular reactivity.

Blue, cyanotic, or purple toes may be seen in AE. More often than not, physicians misdiagnose this as being due to emboli coming from the heart, whereas the emboli are often from a severely atherosclerotic aorta.

Livedo reticularis on the lateral aspect of the foot or on the heel, along with bluish or purplish toes, is highly suggestive of AE. The ability to demonstrate lesions on both feet isolates the atherosclerotic plaque to the area above the aortic bifurcation. If only one foot is affected, then the lesion can be in the iliac artery, femoral artery, superficial femoral artery, or other distal artery.

Ischemic-appearing ulcerations in unusual locations (ie, neither distally nor over bony prominences) are another feature of AE, but also may suggest arteriosclerosis obliterans with underlying trauma, leukocytoclastic vasculitis, or other vasculitides. Hollenhorst plaques or cholesterol deposits seen on fundus examination reinforce the diagnosis of AE in the presence of skin lesions.

AE frequently involves the kidneys and, when it does, can cause accelerated hypertension, ischemic atrophy of large segments of the kidney, and progressive renal failure, often leading to end-stage renal disease. Pathological evidence of AE in the kidneys has been found in as many as 77% of patients who underwent surgery for abdominal aortic aneurysm and subsequently died.

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Unfortunately, no single laboratory test makes the diagnosis. A high index of clinical suspicion is essential, since many of the nonspecific findings in AE can also occur in patients with necrotizing vasculitis. Eosinophilia can occur early in atheroembolic renal disease in as many as 80% of patients, and in some series hypocomplementemia (low C3 and C4) has occurred in patients with AE, again confusing the picture by suggesting necrotizing vasculitis. Therefore, in the elderly patient with diffuse and severe atherosclerosis but normal complement and immunologic tests, AE should be considered.

TREATMENT AND PROGNOSIS

Treatment of AE is threefold. First, the source of the atheromatous material must be removed. For example, if both feet are affected and the abdominal aorta is the source of the atherosclerotic plaque, the treatment of choice is replacement of the abdominal aorta. If one leg is affected and the source is the iliac or superficial femoral artery, percutaneous transluminal angioplasty, atherectomy, or surgery is required. Second, the affected end organ is treated. If the kidney is affected, the hypertension and renal failure are managed as effectively as possible. If the leg or foot is affected, the ischemic ulcerations are treated conservatively, a sympathectomy is performed if necessary, and amputation is a last resort. Third, because AE is a complication of atherosclerosis, aggressive risk factor modification should be undertaken to prevent the progression of atherosclerosis.

Because AE is a marker for diffuse atherosclerosis, the long-term outlook is very poor. In some series, the 1-year mortality is as high as 90%. Screening of the carotid and coronary circulation for atherosclerosis may be a way to improve this dismal mortality rate.

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ANTIMICROBIAL RESISTANCE IN *NEISSERIA GONORRHOEAE*

When penicillin was first introduced as antimicrobial therapy for gonorrhea in the 1940s, many believed that the disease would soon be totally eradicated. No one had anticipated that the causative agent, *Neisseria gonorrhoeae*, would have the unique ability to develop both plasmid-mediated and chromosomally mediated mechanisms for antimicrobial resistance. Today, gonorrhea remains the most frequently reported communicable disease in the United States, with over 700,000 cases per year. Although the overall incidence has decreased over the past 20 years from a peak of over 1 million cases per year, the incidence of infection due to antimicrobial-resistant strains has steadily increased, which contributes to the continuing high prevalence of the disease.

As the proportion of antimicrobial-resistant strains of *N gonorrhoeae* has risen, effective therapy has become increasingly expensive and complicated. Many of the strains that exist today have developed high levels of plasmid-mediated resistance to penicillin and tetracycline, the relatively inexpensive drugs that were advocated for primary treatment in earlier years. Chromosomally mediated resistance mechanisms have been identified for penicillin and other beta-lactams, tetracycline, erythromycin, spectinomycin, quinolones, and sulfonamides. Every class of new drugs introduced for the treatment of gonorrhea has met with the emergence of new resistant strains that render treatment ineffective for some cases.

REGIONAL RESISTANCE

Epidemiological studies have shown that resistant strains often originate in a particular region. For example, penicillinase-producing *N gonorrhoeae* (PPNG) can be caused by any of at least six different plasmids with unique patterns of resistance, and the emergence of these plasmid types has been traced to various geographic locations including Southeast Asia, West Africa, Rio de Janeiro, Toronto, Nimes (France), and New Zealand. Resistant strains also tend to proliferate more readily in some areas. Between 1981 and 1985, PPNG became endemic in the United States, especially in New York City, Miami, and Los Angeles. Recent surveillance data indicate that in large East Coast cities, including Philadelphia and Baltimore, the incidence of PPNG increased from 3% in 1988 to more than 15% in 1990. In Cincinnati and other areas, the percentage of

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