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

chromosomally mediated resistant gonorrhea cases increased from less than 5% in 1988 to greater than 10% in 1990. Tetracycline-resistant *N gonorrhoeae* increased dramatically in Atlanta, Baltimore, and Boston.

UPDATED TREATMENT RECOMMENDATIONS

An understanding of the mechanisms of resistance and regional trends in resistance is very important to the effective clinical management and control of gonorrhea. Studies have indicated that up to 40% of private clinicians and 3% to 5% of clinics still prescribe penicillin and tetracycline for gonorrhea and its complications, even though these drugs are now contraindicated.

In 1989 the CDC updated recommendations for detection, treatment, and management of antimicrobial-resistant gonorrhea. The recommended treatment is summarized below.

The primary regimen for uncomplicated gonorrhea in adults should consist of ceftriaxone 250 mg intramuscularly once daily, accompanied by doxycycline 100 mg orally twice daily for 7 days for presumptive treatment of coexisting chlamydial infection. Further, gonococcal isolates should be routinely screened for beta-lactamase and, if available, for in vitro susceptibility to prescribed antimicrobial agents by disk agar diffusion. The standard in vitro susceptibility procedure is agar dilution, but this is not widely available and can be expensive for routine use and patient management.

Successful management in the future depends on careful surveillance and analysis of resistant strains to facilitate selection of effective therapy. An ongoing surveillance program is necessary to monitor the evolving resistance patterns of the disease and assist in development of control strategies.

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