CASE REPORTS

Refer to: Lawrence EC, Mills J: Bacterial endocarditis mimicking vasculitis with steroid-induced remission. West J Med 124:333-334, Apr 1976

Bacterial Endocarditis Mimicking Vasculitis with Steroid-Induced Remission

E. CLINTON LAWRENCE, MD
JOHN MILLS, MD
San Francisco

INFECTIVE ENDOCARDITIS AND VASCULITIS may be difficult to differentiate clinically.[1] Although a favorable response to steroid administration is often interpreted as presumptive evidence for vasculitis,[2,3] we recently encountered two patients with documented infective endocarditis who improved clinically with corticosteroid therapy alone.

Reports of Cases

CASE 1. A 78-year-old woman was referred to Moffitt Hospital on April 10, 1971 because of sudden loss of vision in the right eye and an ophthalmologist's diagnosis of temporal arteritis with thrombosis of the retinal artery.

The temperature was 38.8°C (101.8°F), a grade 2/6 mitral regurgitation murmur was heard, but no splenomegaly, splinter hemorrhages, Osler's nodes or Janeway lesions were present. Vision was 20/800 in the right eye and 20/100 in the left. The right retina was pale with extremely thin vessels. Hematocrit reading was 34 percent, leukocyte count was 11,200 per cu mm, erythrocyte sedimentation rate was 50 mm per hour and no abnormalities were found on analysis of the urine. Two blood specimens were drawn for cultures and, because of a presumptive diagnosis of temporal arteritis, therapy with orally given prednisone (60 mg per day) was started. The patient became afebrile and noted improvement in her vision. Findings from a right temporal artery biopsy carried out the day after admission showed no abnormalities. On day five, growth of an organism was noted in one of two blood cultures. Three additional blood specimens were obtained and cultured; all five cultures subsequently grew Streptococcus viridans. Prednisone therapy was discontinued and intravenous administration of penicillin, 12 million units per day, was begun. The patient recovered without complication and is well four years later.

CASE 2. A 50-year-old woman was referred to Moffitt Hospital on February 3, 1975 because of sudden loss of vision in the right eye and a diagnosis of retinal vasculitis. Five months previously, the patient had had fever with a flu-like syndrome and diarrhea. Results of sigmoidoscopy were consistent with ulcerative colitis although contrast studies showed no abnormalities; salicylazosulfapyridine was prescribed and symptoms resolved. Three months before admission, generalized myalgia and a purplish petechial lesion on the right great toe developed. Hematocrit reading was 38 percent, erythrocyte sedimentation rate was 56 mm per hour and on analysis of the urine there were four to eight red blood cells per high power field. Administration of salicylazosulfapyridine was discontinued and therapy with prednisone (40 mg per day given orally) was begun, with symptomatic improvement. Whenever this dosage was reduced, fever, arthralgia, myalgia and migratory tender nodules in the calves, thighs and palms developed. Two days before admission, sudden loss of vision occurred in the right eye, retinal vasculitis was diagnosed by an ophthalmologist and the prednisone dosage was increased to 60 mg per day.

On admission, vital signs were found to be normal; a right homonomous quadrantanopia and a grade 2/6 mitral regurgitation murmur were noted to be present. Roth spots, splinter hemorrhages, Osler's nodes and Janeway lesions were not found. Hematocrit reading was 38 percent, leukocyte count was 15,000 per cu mm, erythrocyte sedimentation rate was 60 mm per hour and findings on analysis of the urine showed two to five red blood cells per high power field. Tests

From the Medical Service, San Francisco General Hospital, and the Departments of Medicine and Microbiology, University of California, San Francisco.

Dr. Lawrence is now at the National Cancer Institute, National Institutes of Health, Bethesda, Maryland.

Submitted June 4, 1975.

Reprint requests to: John Mills, MD, Medical Service, San Francisco General Hospital, 1001 Potrero Avenue, San Francisco, CA 94110.

for fluorescent antinuclear antibody, lupus erythematosus preparation and rheumatoid factor were negative. On an echocardiogram, thickening of the mitral valve was noted. Streptococcus bovis grew in each of six blood cultures and penicillin therapy, 12 million units per day, given intravenously, was started. The dosage of prednisone was rapidly tapered and discontinued. The patient was well after a four week course of penicillin therapy.

Discussion

To our knowledge, this is the first documentation of corticosteroid suppression of the clinical manifestations of subacute bacterial endocarditis. Although corticosteroids have been used before in the successful management of endocarditis, the steroids were administered simultaneously with antibiotics or the diagnosis of endocarditis was suspect[2,4-6] making interpretation of steroid effect extremely difficult. The only instance in which an unequivocal steroid remission occurred was the case reported by Numainville and Scarpellino,[7] although the authors believed that the patient had coexistent rheumatic fever. Both of our patients had definite steroid-induced clinical remissions, one lasting nearly a week and the other lasting three months. Fortunately, findings on cultures of blood specimens—drawn as part of a routine workup for patients with fever—and on careful physical examinations led to the diagnosis of endocarditis in both cases.[8]

It was formerly thought that the peripheral manifestations of infective endocarditis were due to embolic phenomena. It is now appreciated that some of these manifestations, such as glomerulitis and Osler's nodes, are due to immune-complex deposition.[4,9,10] In the presence of an avirulent organism such as Streptococcus viridans, corticosteroid treatment might diminish immune-complex deposition (by decreasing antibody production) or reduce the inflammatory reaction at the site of immune-complex deposition, resulting in apparent clinical improvement despite continued infection.[11-14] We are not aware of any case of endocarditis in which remission of nephritis occurred with steroid therapy. The lack of severe nephritis in case 2 despite a prolonged disease course suggests that steroids might be able to prevent nephritis in these patients, but this remains speculative. The pathogenesis of many types of "autoimmune" vasculitis also involves antigen-antibody complex deposition, and response to steroids frequently occurs in patients with these disorders as well.[15-17]

Before steroid therapy is started in any patient with "vasculitis" (with or without fever), blood specimens should be drawn for culture lest the clinician be falsely reassured by a patient's favorable clinical response to these agents and not recognize the infectious nature of the disease process. When patients have an illness compatible with either vasculitis or "culture-negative" endocarditis (that is, endocarditis due to microorganisms not recoverable with standard blood culture media), the response to antibiotics (such as penicillin and an aminoglycoside) should be tested before starting steroid therapy.[9]

Summary

In two patients with infective endocarditis, a misdiagnosis of vasculitis was made at first and treatment with corticosteroids was begun, resulting in clinical improvement. Some of the manifestations of infective endocarditis are caused by antigen-antibody complex deposition similar to that seen in some "autoimmune" disorders. Therefore, response to corticosteroids cannot be used to differentiate between these two diseases.

REFERENCES

1. Case Records of Massachusetts General Hospital. Case 30-1974—Fever of unknown origin with severe thrombocytopenia. N Engl J Med 291:242-249, 1974

2. Kerr A: Subacute Bacterial Endocarditis. Springfield, Ill, Charles C Thomas, 1955, pp 173-174

3. Hurwitz MM: When coexisting conditions confuse the issue. Geriatrics 28:64-68, 1973

4. Gutman RA, Striker GE, Gilliland BC, et al: The immune complex glomerulonephritis of bacterial endocarditis. Medicine 51:1-25, 1972

5. Suzman MM: The clinical application of corticotropin and cortisone therapy, a report of 247 cases. S Afr Med J 27:195-212, 1953

6. Brunson JG: Coronary embolism in bacterial endocarditis. Am J Pathol 29:689-701, 1953

7. Numainville LJ, Scarpellino CJ: Coexistent rheumatic fever and subacute bacterial endocarditis treated with ACTH and antibiotics. Am Heart J 43:468-473, 1952

8. Manor RS, Sachs W: Visible retinal emboli in a case of subacute bacterial endocarditis. Ophthalmologica 166:10-15, 1973

9. Howard EJ: Osler's nodes. Am Heart J 59:633-634, 1960

10. Weinstein L, Rubin RH: Infective endocarditis. Prog Cardiovasc Dis 16:239-274, 1973

11. Germuth FG Jr, Valdes AJ, Senterfit LB, et al: A unique influence of cortisone on the transit of specific macromolecules across vascular walls in immune complex disease. Johns Hopkins Med J 122:137-153, 1968

12. Claman HN: Corticosteroids and lymphoid cells. N Engl J Med 287:388-397, 1972

13. Dale DC, Fauci AS, Wolff SM: Alternate-day prednisone—Leukocyte kinetics and susceptibility to infections. N Engl J Med 291:1154-1158, 1974

14. Butler WT, Rossen RD: Effects of corticosteroids on immunity in man—II. Alterations in serum protein components after methyl prednisolone. Transplant Proc 5:1215-1219, 1973

15. Keeffe EB, Bardana EJ Jr, Harbeck RJ, et al: Lupus meningitis—Antibody to deoxyribonucleic acid (DNA) and DNA:anti-DNA complexes in cerebrospinal fluid. Ann Intern Med 80:58-60, 1974

16. Atkins CJ, Kondon JJ Jr, Quismorio FP, et al: The choroid plexus in systemic lupus erythematosus. Ann Intern Med 76:65-72, 1972

17. Lampert PW, Oldstone MBA: Host immunoglobulin G and complement deposits in the choroid plexus during spontaneous immune complex disease. Science 180:408-410, 1973