The prevalence of cardiac valvular pathosis in patients with systemic lupus erythematosus

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The purpose of this study was to determine the prevalence of valvular pathosis in a population of patients with SLE, to assess the candidacy of such patients for antibiotic prophylaxis before dental treatment. The hospital records of 112 patients with SLE were reviewed and screened for endocarditis, heart murmurs, and other valvular pathosis. Two of the 112 patients had confirmed cases of bacterial endocarditis. This prevalence is comparable to endocarditis prevalence rates in patients with prosthetic valves and is also three times that in patients with rheumatic heart disease. The high prevalence of endocarditis in this population of patients with SLE suggests that according to present perspectives on patient management, patients with SLE should be considered for antibiotic prophylaxis before dental therapies associated with formation of a bacteremia.

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Systemic lupus erythematosus (SLE) is associated with multiple systemic complications including cutaneous, renal, pulmonary, and cardiac manifestations.[1] Cardiac manifestations of SLE include fibrinous pericarditis,[2,3] myocardial infarction,[4] acute myocarditis,[5] aortic[6] and mitral[7] insufficiency, and verrucous endocarditis.[8] Verrucous endocarditis, also known as Libman-Sacks endocarditis, results after a series of events initiated when antibodies to nuclear antigens combine with specific tissue antigens to form antigen-antibody complexes. Deposition of these complexes on cardiac valvular endothelium initiates the formation of fibrin-platelet deposits on the valves known as nonbacterial thrombotic endocarditis (NBTE) lesions. NBTE lesions can act as a nidus for colonization during transient bacteremias[9,10] and have been detected in 50% of patients with SLE at autopsy.[3]

Currently, signs and symptoms used in the diagnosis of cardiac involvement in patients with SLE have included the presence of heart murmurs, pericardial friction rubs, and echocardiographic evidence of valvular vegetations and pericardial fluid. Previous reports have shown little correlation between the clinical presence of a murmur and the finding of endocarditis at autopsy.[11-13] The use of echocardiography has also been of limited value in detecting valvular lesions. A recent study involving patients with SLE with clinically evident valvular abnormalities (excluding mitral valve prolapse) was able to detect valvular pathosis in only 21% of patients with the use of two-dimensional echocardiography.[14] Another study using echocardiography involving patients with SLE with undetermined valvular disease showed an 18% prevalence of detectable valvular disease.[15] The reported prevalence of bacterial endocarditis in patients with SLE is between 1% and 7%.[16] Compiling the results from nine studies, conducted after steroids came into common use in the 1940s, shows a prevalence rate of 1.3% in clinical patients with SLE and 4% in postmortem cases.[16]

The purpose of this retrospective study was to determine the prevalence of valvular pathosis in a group of patients with SLE, to assess their candidacy for antibiotic prophylaxis before dental procedures likely to produce a bacteremia.

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MATERIAL AND METHODS

The inpatient and outpatient records of 112 patients diagnosed with SLE were reviewed. All patients were being followed up in the Rheumatology Clinic of the Bexar County Hospital District, San Antonio, Texas. Patient records were reviewed for diagnostic criteria confirming the diagnosis of SLE, for confirmatory evidence of valvular pathosis including the presence of a heart murmur, and for an episode of bacterial endocarditis. A diagnosis of endocarditis was confirmed by the presence of a murmur, positive blood cultures, echocardiographic evidence of valvular vegetations, clinical observation at the time of valve replacement surgery, and pathologic study after valve replacement. Records were also screened for other possible sources of cardiac valvular pathosis such as the presence of prosthetic heart valves, rheumatic fever/rheumatic heart disease, congenital heart disease, and drug abuse. The diagnosis of SLE required that a patient historically manifest, either serially or simultaneously, four or more of the 11 diagnostic criteria set forth by the American Rheumatism Association in 1982[17] and was confirmed in each record reviewed (Table I).

Table I. Diagnostic criteria for SLE

1. Butterfly rash
2. Discoid lupus
3. Photosensitivity
4. Oral ulcers
5. Arthritis
6. Serositis
   a. Pleuritis
   b. Pericarditis
7. Renal disorder
   a. Persistent proteinuria >0.5 gm/day or >3+
   b. Cellular casts
8. Neurologic disorder
9. Hematologic disorder
   a. Hemolytic anemia
   b. Leukopenia
   c. Lymphopenia
   d. Thrombocytopenia
10. Immunologic disorder
    a. Positive LE cell preparation
    b. Anti-DNA antibody
    c. Anti-Sm
    d. False-positive STS (serologic test for syphilis)
11. Antinuclear antibody

RESULTS

For the 112 patients, age ranged from 12 to 78 years with an average age of 30 years. One hundred (89.3%) were women and 12 (10.7%) were men. All patients satisfied the criteria for diagnosis of SLE. Bacterial endocarditis developed in two of the 112 patients (1.8%), and these two patients required valve replacement surgery; however, one of those patients also had a history of rheumatic fever. Discarding this case, bacterial endocarditis developed in one in 112 (0.9%) with no other apparent risk factors other than SLE. In addition, of the 112 patients, 56 (50%) had a history of heart murmur detected on clinical examination.

DISCUSSION

Previous studies have reported 1% to 7% prevalence rates of endocarditis in patients with SLE.[1] This rate is approximated at 1.3% with the therapeutic use of corticosteroids in the treatment of SLE.[16] The reported prevalence rate of endocarditis in patients with rheumatic heart disease is 0.3%, which is one third that of the prevalence of endocarditis determined by this article.[16] (The only reported data available for patients with rheumatic heart disease were expressed as incidence[18, 19] and not as prevalence. However, this incidence rate can be converted to prevalence per patient population with the use of the average lifespan of American citizens.[16]) The prevalence of endocarditis in patients with prosthetic heart valves is reported at 1.1% (excluding early infections believed to result from the surgical implantation procedure), or just slightly greater than this study's reported prevalence.[20]

The American Heart Association recommends antibiotic prophylaxis before invasive dental procedures likely to induce gingival bleeding for patients with evidence of rheumatic heart disease and for patients with prosthetic heart valves.[21] The data from this study support conclusions from previous studies that identify patients with SLE as a population at an increased risk of development of endocarditis.[13, 22-24] Although these results support those of prior studies, this investigation has limitations. First, it is a retrospective chart review that relies on multiple observers to produce the data. This drawback is most glaring with respect to the detection of heart murmurs. Second, the study population is small and may not be repesentative of the SLE patient population. In contrast to the aforementioned deficits, the prevalence of endocarditis may be underestimated. For example, it was not determined whether patients relocated or were treated for endocarditis at another hospital.

Acceptance of this purported degree of endocarditis risk among patients with SLE illuminates the need to identify patients with SLE at risk. However, existing technology does not allow accurate clinical detection of Libman-Sacks lesions. The prevalence of these lesions in patients with SLE is 50% at autopsy, whereas studies on live patients have been unable to

report nearly the same percentage, even with the advent of two-dimensional echocardiography. Recent studies attempting to quantify valvular disease in patients with SLE have reported 18%[15] and 21%[14] of their patients as having valvular abnormalities. It is apparent that current clinical techniques cannot approach the accuracy achieved during examination at autopsy and that current methods do not allow a cardiologist to select out accurately the subpopulation of patients with SLE at increased risk.

Therefore, because patients with SLE appear to be at approximately equal risk of development of bacterial endocarditis as patients with prosthetic heart valves and because of the uncertainty in selecting a subpopulation of patients with SLE at risk, it is recommended that patients with SLE be considered for antibiotic prophylaxis. It is not our intent to defend or promote the concept of antibiotic prophylaxis inasmuch as significant controversy surrounds this practice today.

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