International Journal of Medical Science and Clinical Research Studies

ISSN(print): 2767-8326, ISSN(online): 2767-8342

Volume 03 Issue 03 March 2023

Page No: 306-308

DOI: https://doi.org/10.47191/ijmscrs/v3-i3-05, Impact Factor: 6.597

Cardiovascular Alterations Present in Systemic Lupus Erythematosus: Review of the Literature

Omar Alejandro Avila Hernández[1], Manuel Enrique Vadillo Flores[2], Miguel Ángel Sánchez Dorantes[3], Diana Laura García Segovia[4]

[1,3]Universidad Autónoma de Querétaro. Santiago de Querétaro, México.

[2]Universidad Autónoma de Guadalajara. Guadalajara, México.

[4]Universidad Autónoma del Estado de Hidalgo. Pachuca,Hidalgo.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic inflammatory disease secondary to an autoimmune response that is characterized by the deposition of immune complexes that activate complement and cause tissue damage. The American College of Rheumatology (ACR, American College of Rheumatology) in 2012 established the new diagnostic criteria for SLE, identifying 17 criteria of which at least 4 must be met (at least one clinical, and one immunologic) or lupus nephritis as the sole criterion in the presence of antinuclear antibody (ANA) or double-stranded anti-DNA for diagnosis.[1]

The incidence of SLE varies from 1 to 25/100,000 persons in America and Europe. The incidence and prevalence of SLE in Latin American countries is not well known, but it would appear that the disease presents at younger ages, with greater frequency and severity in the mestizo population of Latin America, which is believed to be due to the genetic component due to the European and Amerindian ancestry of this population. Regarding the age of disease presentation, 60-70% of patients develop SLE between the ages of 16 and 55 years, approximately 15% before the age of 15 years and 15% after the age of 55 years.[1,2]

CARDIAC ALTERATIONS

Cardiovascular and cerebrovascular disease

The prevalence of cardiovascular pathologies in patients with a diagnosis of Systemic Lupus Erythematosus Erythematosus is estimated at 6-8%, cerebrovascular pathologies at 3-15%8 and approximately 40% of patients with SLE have sustained hypercholesterolemia within 3 years of diagnosis. SLE presents a bimodal pattern of mortality: initially, death due to the same pathology or sepsis and in a second stage, after 5 years, due to complications of atherosclerotic pathology as the main cause. [3,4]

Vascular disease

In SLE as in other rheumatologic or autoimmune diseases, it is well known that they present accelerated atherosclerosis, showing a progression rate more than double that of non-lupus patients, being directly related to advanced age at the time of diagnosis, time of disease evolution and homocysteine levels.[5,6]

In SLE there is an increase in cholesterol deposition in smooth muscle cells mediated by the presence of immune complexes loaded with low density lipoprotein (LDL) cholesterol, in addition to the fact that these immune complexes inhibit the enzyme cholesterol 27-hydroxylase, thus decreasing the elimination of cholesterol from the blood vessel wall. In addition, interleukin 6 (IL6) and anti-lipoprotein lipase (LPL) antibodies, which are increased in this disease, inhibit this enzyme, generating an atherogenic lipid profile, with elevated LDL cholesterol, very low density lipoprotein (VLDL), triglycerides and decreased high density lipoprotein (HDL) cholesterol.[7,8,9]

Patients with SLE generally have traditional risk factors for atherosclerosis such as: age, sex, family history, dyslipidemia, arterial hypertension, diabetes, smoking, sedentary lifestyle, among others; but present non-traditional factors specific to the disease such as: the use of corticosteroids, elevated levels of C-reactive protein (CRP), complement activation, antiphospholipid antibodies including beta 2-glycoprotein 1, antibodies against oxidized LDL, among others.[10,11]

PERICARDITIS AND PERICARDIAL EFFUSION

Pericarditis was the first known cardiac manifestation of SLE and has also been referred to as one of the most frequent complications. Let us not forget that one of the diagnostic criteria of SLE is serositis, so we must take this pathology into account when we are faced with a patient with pericardial effusion or pericarditis. The prevalence of pericarditis as a

manifestation of SLE is 12-48% in adults and 5-25% in children, and up to 70% may be accompanied by pleuritis.[11]

The clinical presentation usually coincides with an active phase of lupus, although pericardial manifestations have been observed in patients with SLE in an inactive phase, and it is observed as an initial manifestation of the disease in only 1% of patients. As with pericarditis due to other causes, the main symptom is pain, which improves with prone decubitus, of prolonged duration of hours to days, with general symptoms such as fever and malaise, presenting pericardial rub with effusion or pericardial thickening by echocardiography. Cardiac tamponade is an infrequent event, its incidence is estimated at less than 1%, although in small or selected series it may be higher.[12]

In order to determine the frequency and clinical correlations of asymptomatic pericardial effusion in lupus patients with echocardiography and electrocardiography, a study was published in 2009 in the journal Lupus in which a series of 50 patients with SLE was evaluated. It was found that 24% had pericardial effusion and 34% had hypoalbuminemia. Patients with pericardial effusion had lower serum albumin level ($p<0.001$), higher incidence of proteinuria ($p=0.003$), higher C-reactive protein (CRP) level ($p=0.036$) and higher pulmonary artery systolic pressure ($p=0.011$), tending to have a higher incidence of PR segment depression ($p=0.082$) compared to those without pericardial effusion.[13]

MYOCARDITIS

Clinically, myocardial involvement is not as frequent as pericardial disease, although autopsy studies show inflammatory changes in the myocardium in up to 57% of cases. Myocardial involvement by lupus can evolve acutely or chronically to dilated cardiomyopathy, with its respective complications such as heart failure, low cardiac output, conduction disturbances and atrial or ventricular arrhythmias. It should be noted that in patients with lupus there may be myocarditis secondary to cardiotoxicity due to hydroxychloroquine or to a viral etiology due to the immunosuppression of these patients, making the differential diagnosis difficult. In symptomatic patients (7-10%), it may manifest with sinus tachycardia, fever, palpitations, chest pain and findings typical of heart failure. [14]

In the electrocardiogram it is common to find repolarization alterations, with nonspecific ST segment and T wave changes, heart block and, if there is associated pericardial effusion, a low voltage electrocardiographic trace can be seen. In the echocardiogram, abnormalities in the systolic or diastolic motion of the cardiac walls may be found diffusely, with decreased ejection fraction and dilatation of cavities. Histological findings consist of interstitial and perivascular mononuclear infiltrate, fibrosis and myocyte degeneration, nonspecific findings that can also be found in viral myocarditis. Biopsy, being quite nonspecific in most cases, is meaningless to perform. When myocardial involvement is severe and decompensates the patient, putting his life at risk, treatment with high-dose corticosteroids together with immunosuppressants is justified.[15]

VALVULAR INVOLVEMENT

Valvular involvement in SLE is one of the most frequent and clinically important forms of cardiac manifestations. 40-60% of SLE patients have cardiac valvular alterations demonstrated by echocardiography, while in autopsy studies the frequency varies between 13-74%. Valvular lesions are frequently reported in patients with antiphospholipid syndrome, independent of the presence or absence of SLE. Valvular lesions most frequently affect the aortic and mitral valves, and the most frequent is thickening, leading to stenosis and insufficiency, as well as the formation of vegetations of various sizes that contribute to the functional alteration of the valvular apparatus.

Libman-Sacks endocarditis, described in 1924, is the most characteristic lesion at the valvular level and corresponds to the formation of large sterile vegetations. The pathophysiology consists of the sterile deposit of immune complexes in the left valves (aortic and mitral), which generates insufficiency or, less frequently, stenosis.[16]

Libman-Sacks vegetations can be flat or coliform, sessile or pedunculated, and can usually measure between 1 and 4 mm. They preferentially adhere to the ventricular side of the mitral valve, either at the commissures, leaflets, chordae tendineae or papillary muscles. These lesions are generally asymptomatic and rarely generate audible murmurs; however, due to their location, they present an increased risk of generating embolisms to the central nervous system.[17]

The most frequent findings in these patients are: valvular thickening (51%), vegetations (43%), valvular regurgitation (25%) and stenosis (4%). The combined incidence of stroke, embolism, heart failure, infective endocarditis and the need for valve replacement was 22% in patients with abnormal TEE versus 8% with negative TEE. As this "aseptic" endocarditis favors the superimposition of infective endocarditis, antibiotic prophylaxis is suggested in predisposing situations.[18]

Treatment of valvular and endocardial lesions may require surgery in case of significant hemodynamic compromise, which occurs in less than 3% of individuals.

It is important to mention that prolonged treatment with corticosteroids and the immunosuppressive treatment that patients with SLE often receive to control the disease generate a state of immunosuppression that predisposes to infections, facilitating bacteremia and increasing the probability of suffering infective endocarditis.[19]

CONCLUSIONS

SLE can cause damage at any level of the cardiovascular system. Pericarditis is the classic element of the disease and constitutes a diagnostic criterion of SLE. Heart disease can

occur in patients with an already established diagnosis, it may be the initial manifestation in some of them or the adverse effect of any of the drugs used for their treatment. Therefore, most SLE patients develop some type of heart condition during their lives. An adequate knowledge of this pathology and its cardiovascular affections is essential to ensure early and accurate care in patients with this disease.

REFERENCES

I. Duarte C, Couto M, Ines L, Liang MH. Epidemiology of systemic lupus erythematosus. In: Lahita RG, Tsokos G, Buyon J, Koike T. Systemic Lupus Erythematosus. (2011, 5ta edición, 673-96) London: Elsevier.

II. Petri M, Orbai AM, Alarcón GS, Gordon C, Merrill JT, Fortin PR, et al. Derivation and Validation of the Systemic Lupus International Collaborating Clinics Classification Criteria for Systemic Lupus Erythematosus. Arthritis Rheum. 2012;64(8):2677-86.

III. Pons-Estel GJ, Alarcon GS, Scotfield L, Reinlib L, Cooper GS. Understanding the epidemiology and progression of systemic lupus erythematosus. Semin Arthritis Rheum. 2010;39(4):257-68.

IV. Pons-Estel BA, Catoggio LJ, Cardiel MH, Soriano ER, Gentiletti S, Villa AR, et al. The GLADEL multinational Latin American prospective inception cohort of 1,214 patients with systemic lupus erythematosus: ethnic and disease heterogeneity among "Hispanics". Medicine (Baltimore). 2004;83(1):1-17.

V. Battagliotti CA, Gentiletti AA, Pons Estel BA, Berbotto GA, Pons Estel GJ. Epidemiología en el lupus eritematoso sistémico. En "Lupus Eritematoso Sistémico, aspectos clínicos y terapéuticos" (2013, primera edición, 53-60), Buenos Aires: GB Editores Asociados.

VI. Rus V, Maury EE, Hochberg MC. The epidemiology of systemic lupus erythematosus. En: Wallace DJ, Hahn BH. Dubois lupus erythematosus. (2007, 7th ed, 34-44) Philadelphia: Lippincott Williams & Wilkins.

VII. Urowitz MB, Gladman DD, Ibanez D, Fortin PR, Bae SC, Gordon C, et al. Evolution of disease burden over five year in multicenter inception systemic lupus erythematosus cohort. Arthritis Care Res (Hoboken). 2012;64(1):132-7.

VIII. Battagliotti CA, Gentiletti AA, Pons Estel BA, Berbotto GA, Pons Estel GJ. Morbimortalidad y daño crónico en lupus eritematoso sistémico. En "Lupus Eritematoso Sistémico, aspectos clínicos y terapéuticos" (2013, primera edición, pág. 169-181), Buenos Aires: GB Editores Asociados.

IX. Bruce IN, Unrowitz MB, Glandman DD, Hallett DC. Natural history of hypercholesterolemia in systemic lupus erythematosus, J Rhematol. 1999;26(10):2137-43.

X. Asanuma Y, Oeser A, Shintani AK, Turner E, Olsen N, Fazio S, et al. Premature coronary artery atherosclerosis in systemic lupus erythematosus. N Engl J Med. 2003;349(25):2407-15.

XI. Roman MJ, Crow MK, Lockshin MD, Devereux RB, Paget SA, Sammaritano L, et al. Rate and determinants of progression of atherosclerosis in systemic lupus erythematosus. Arthritis Rheum. 2007;56(10):3412-19

XII. Petri MA, Kiani AN, Post W, Christopher-Stine L, Magder LS. Lupus Atherosclerosis Prevention Study (LAPS). Ann Rheum Dis 2011;70(5):760-5. Roman MJ, Salmon JE. Cardiovascular Manifestations of Rheumatologic Diseases. Circulation. 2007;116(20):2346-55. Moder KG, Miller TD, Tazelaar HD. Cardiac involvement in systemic lupus erythematosus. Mayo Clin Proc. 1999;74(3):275-84.

XIII. Sugiura T, Kumon Y, Kataoka H, Matsumura Y, Takeuchi H, Doi YL. Asymptomatic pericardial effusion in patients with systemic lupus erythematosus. Lupus. 2009;18(2):128-32.

XIV. Babini, SM. Lupus Eritematoso Sistémico. En: Maldonado Coco JA, Citera G. Reumatología. (2010, primera edición, Pág 208-219). Buenos Aires: Azurras

XV. Sagar S, Liu PP, Cooper LT. Myocarditis. Lancet. 2012;379:738-47.

XVI. Nussinovitch U, Shoenfeld Y. Anti-troponin autoantibodies and the cardiovascular system. Heart. 2010;96(19):1518-24.

XVII. Owlia MB, Mostafavi Pour Manshadi S, Naderi N. Cardiac manifestations of rheumatological conditions: a narrative review. ISRN Rheumatol. 2012;2012:463-620.

XVIII. Badsha H, Edwards CJ. Intravenous pulses of methylprednisolone for systemic lupus erythematosus. Semin Arthritis Rheum. 2003;32(6):370-7

XIX. Moder KG, Miller TD, Tazelaar HD. Cardiac involvement in systemic lupus erythematosus. Mayo Clin Proc. 1999;74(3):275-84.