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# Aseptic Vegetation in Pulmonary Artery Valve, Finding in a Patient with Systemic Lupus Erythematosus. Case report

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**ABSTRACT**

Systemic lupus erythematosus (SLE) is an entity that promotes the formation of autoantibodies that trigger immune complexes that damage various organs of the body. Worldwide, SLE has a prevalence of 13-7000 per 100,000 people and leads to a high mortality from cardiovascular diseases, as well as the risk of developing lupus nephritis (LN) in 60% of cases. We present the case of a 41-year-old patient with a history of recently diagnosed arterial hypertension and bronchial hyperreactivity of 20 years of evolution, admitted for 1 month of evolution with asthenia, myalgia, arthralgia and fever; she identifies malar erythematous dermatosis and systolic murmur in a pulmonary focus. When presenting with proteinuria, microhematuria and renal functional impairment, positive ANAs were performed, which is why it was classified as lupus nephropathy, and she needed to start hemodialysis. In the presence of the murmur, an echocardiogram was performed, which showed a pulmonary valve with a 1cm image of vegetation, causing moderate regurgitation.

With the diagnosis of Libman-Sacks endocarditis, anticoagulant treatment, steroids, and mycophenolate were started, evolving to normal renal function.

**KEY WORDS**: lupus erythematosus systemic, pulmonary artery, non-infective endocarditis

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## INTRODUCTION

Systemic lupus erythematosus (SLE) is an entity that promotes the formation of autoantibodies and triggers the formation of immune complexes that damage various organs in the body[1,2,3]. Worldwide, SLE has a prevalence of 13-7000 per 100,000 people, and leads to high mortality, of which more than a third is due to cardiovascular diseases[1,4].

## BACKGROUND

Among the cardiac manifestations, Libman-Sacks endocarditis can be observed, which was first described in 1888 by Ziegler, who observed sterile thrombi in cardiac valve structures and were later observed in 4 cases and described by Libman et al. Sacks, as Non-Bacterial Thrombotic Endocarditis (NBTE) in 1924. In 2022 Wang carried out a cohort study of patients with NBTE (with a total of 42 cases in 20 years)[4].

Libman-Sacks (LS) endocarditis is a non-bacterial thrombotic endocarditis, one of the cardiac manifestations of systemic lupus erythematosus (SLE)[5]. Previous studies have shown that LS endocarditis is associated with antiphospholipid antibodies. In addition, patients with primary APS commonly present with nonbacterial thrombotic endocarditis and heart valve dysfunction. The pathogenesis is thought to involve the formation of fibrin-platelet thrombi, which organize and lead to fibrosis and scarring with subsequent valvular dysfunction6.

Central nervous system embolization presents as cerebrovascular accident or delirium[6].

SLE has been associated with a 2- to 10-fold increase in the risk of cardiovascular disease, including acute myocardial infarction, cerebrovascular events, and atherosclerosis, and may increase the risk of heart attack 50-fold acute to the myocardium in women of reproductive age[1,2,5,6]. Evidencing

the rarity in the incidence of said alteration, this case report is presented.

## CLINICAL CASE

A 41-year-old female with a history of recently diagnosed arterial hypertension and bronchial hyperreactivity of 20 years of evolution, presents asthenia, adynamia, myalgias, arthralgias, intermittent fever up to 39.5 C and convulsive crises of 1 month of return. On physical examination with malar erythematous dermatosis and systolic murmur in the pulmonary focus, an echocardiogram was performed that revealed a pulmonary valve with a 1cm image of vegetation, causing moderate regurgitation (Fig. 1A), with no evidence of abscesses, pericardial effusion, or other complications. The laboratories show creatinine of 5.7 mg/dl, hemoglobin 8g/dl, protein in urine of 2,081mg/g, erythrocytes of 10-20 red blood cells/field, immunological with C3 of 47 mg/dL, C4 of 11 mg/dL , ANA positive 1/160 homogeneous pattern, anti-DNA 3.3 U/ml and ENA, SSA, SSB, U1-RNP, SM, SCL-70 negative, blood cultures without development. Starting rescue hemodialysis. A simple skull tomography was performed with the presence of an ischemic lesion (fig. 1B). Concluding in the diagnosis of Libman-Sacks endocarditis, treatment with anticoagulant in combination with steroids (1mg/kg/day) and mycophenolate mofetil was started until reaching a dose of 1g/12 hrs, improving clinically, as well as renal function discharged to continue with follow-up. for rheumatology.

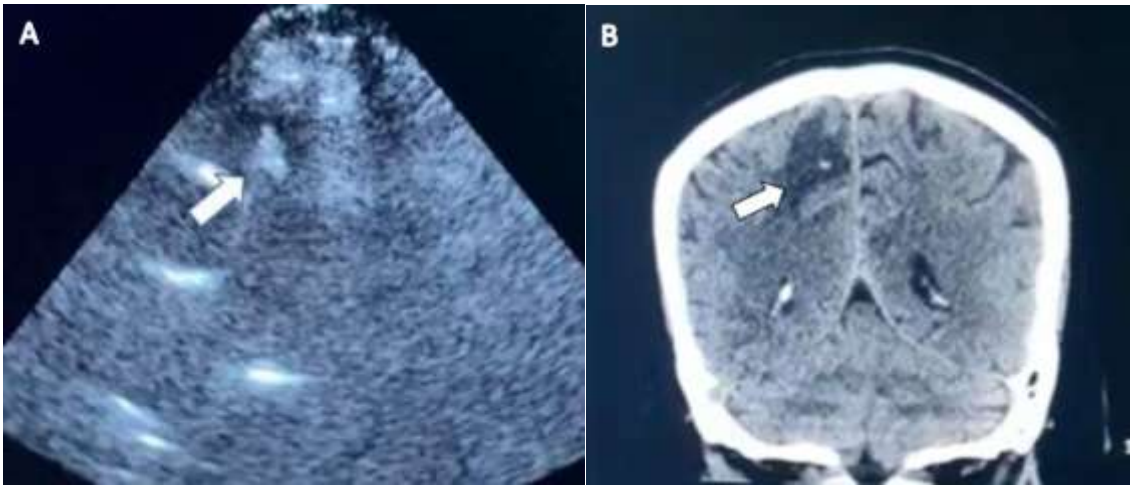


**Figure 1.- A) Echocardiogram showing 1 cm vegetation on the pulmonary valve.**
**B) Simple cranial tomography showing the presence of a right temporal ischemic lesion with perilesional edema.**

## DISCUSSION

Pulmonary valve infective endocarditis is a rare clinical entity whose incidence ranges from 0% to 1.3% in different series, which in many cases is associated with tricuspid endocarditis and mainly affects intravenous drug users where it occurs in 0.75-3.5% of cases[7,8]. In patients with SLE, up to 46% of patients with SLE present with symptoms of lupus nephritis and cardiovascular disease, with myocardial involvement such as pericarditis, myocarditis, and valvular involvement due to thickening, insufficiency, stenosis, and valvular vegetations, the mitral valve being the most affected, present in one of every 10 patients[9]. There are few publications on pulmonary valve involvement. Cardiovascular complications are 2 to 10 times more frequent, the most common being valvular dysfunction, thromboembolic complications and mainly cerebrovascular accident, reporting up to 30% mortality in those patients who have presented ischemic cerebral events[9,10].

Libman-Sacks endocarditis may be a common but underrecognized pathogenic factor for embolic cerebrovascular events in SLE. These findings support the idea that Libman-Sacks vegetations can generate emboli, presenting poor short-term evolution[11]. High prevalence rates of aseptic valvular involvement have been reported using transthoracic echocardiography with SLE with aPL12 antibodies. The current role of echocardiography is indisputable, not only in diagnosis, but also in recognizing the existence of complications and in evaluating the hemodynamic repercussions of valve lesions. The sensitivity of transthoracic echocardiography for the diagnosis of endocarditis is around 70%, depending on the characteristics of the population studied, the size of the vegetation and the presence of

the disease on the native valve or on the prosthetic valve[13,16]. Lesions occur less frequently in the aortic and pulmonary. However, the differential diagnosis of non-infectious lesions diagnosed in the general population includes primary tumors, as well as more typical organized thrombi in non-bacterial antiphospholipid syndrome and autoimmune diseases[14,16]. Although both adverse effects and benefits of anticoagulants and glucocorticosteroids on vegetations in Libman-Sacks endocarditis have been reported, they require further

investigation[15,16]. Sterile valvular vegetations form and embolize, clinically mimicking

infective endocarditis in many respects, with negative blood cultures[13,17]. In a study of patients with native valve infective endocarditis, the existence of a prolonged febrile episode prior to admission, as well as the embolization of large

vegetations were independent risk factors for the development of complications during hospitalization. In our case, the prognosis was favorable despite having presented both embolic and inflammatory events, since these conclusions may not be extrapolated to infective endocarditis of the right valves[17].

## CONCLUSION

SLE can cause multiple systemic effects, both in its debut and in its evolution; In this spectrum the cardiovascular system can be compromised. Diagnostic suspicion is often low, however, the multidisciplinary approach to these patients is highly favorable in their diagnosis and treatment.

## REFERENCES


I. Watana kunakorn C, Burket T. Infective endocarditis at a large community teaching hospital, 1980-1990. A review of 210 episodes. Medicine (Baltimore) 1993; 72: 90-102.
II. Anders HJ, Saxena R, Zhao M-H, Parodis I, Salmon JE, Mohan C. Lupus nephritis. Nat Rev Dis Primers. 2020;6(1):7.
III. Yafasova A, Fosbøl EL, Schou M, Baslund B, Faurschou M, Docherty KF, et al. Long-term cardiovascular outcomes in systemic lupus erythematosus. J Am Coll Cardiol. 2021;77(14):1717–27.
IV. Todolí-Parra JA, Tung-Chen Y, Micó L, Gutiérrez J, Hernández-Jaras J, Ruiz-Cerda JL. Lupus nephritis with preserved kidney function associated with poorer cardiovascular risk control: A call for more awareness. Hipertens Riesgo Vasc. 2018;35(3):110–8.
V. Huang S, Huang F, Mei C, Tian F, Fan Y, Bao J. Systemic lupus erythematosus and the risk of cardiovascular diseases: A two-sample Mendelian randomization study. Front Cardiovasc Med. 2022;9: 896499.
VI. Giannelou M, Mavragani CP. Cardiovascular disease in systemic lupus erythematosus: A comprehensive update. J Autoimmun. 2017; 82: 1–12.
VII. Watanakunakorn C, Burket T. Infective endocarditis at a large community teaching hospital, 1980-1990. A review of 210 episodes. Medicine (Baltimore) 1993; 72: 90-102.
VIII. Hecht SR, Berger M. Right-sided endocarditis in intravenous drugs users. Pronostic features in 102 episodes. Ann Intern Med 1992;117: 560-566
IX. Ibrahim AM, Siddique MS. Libman Sacks Endocarditis. StatPearls Publishing; 2022.
X. Fanouriakis A, Tziolos N, Bertsias G, Boumpas DT. Update on the diagnosis and management of systemic lupus erythematosus. Ann Rheum Dis 2021;80(1):14–25.
XI. Fanouriakis A, Kostopoulou M, Cheema K, et al. 2019 update of the joint European League against rheumatism and European renal Association-European dialysis and transplant association (EULAR/ERA-EDTA) recommendations for the management of lupus nephritis. Ann Rheum Dis 2020; 79:713–23.
XII. Czub P, Żbikowska K, Arendarczyk A, Budnik M. Aortic valve Libman-Sacks endocarditis mimicking papillary fibroelastoma: therapeutic possibilities. Pol Arch Intern Med. 2022; 132(7–8).
XIII. Choi JH, Park JE, Kim JY, Kang T. Non-bacterial thrombotic endocarditis in a patient with rheumatoid arthritis. Korean Circ J. 2016;46(3):425–8.
XIV. Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Tleyjeh IM, Rybak MJ, et al. Infective endocarditis in adults: Diagnosis, antimicrobial therapy, and management of complications: A scientific statement for healthcare professionals from the American Heart Association: A scientific statement for healthcare professionals from the American Heart Association. Circulation [Internet]. 2015;132(15): 1435–86.
XV. Chalvon NB, Pennaforte JL, Servettaz A, Boulagnon C, Gavand PE, Lekieffre M, et al. Les valvulopathies sévères associées au LES et/ou au SAPL (dont l'endocardite de Libman-Sachs) sont une complication du syndrome des anti-phospholipides : analyse rétrospective de 23 patients opérés. Rev Med Interne [Internet]. 2021;42: A84–5.
XVI. Sanfilippo AJ, Picard MH, Newell JB, Rosas E, Davidoff R, Thomas JD, et al. Echocardiographic assesment of patients with infectious endocarditis. Prediction of risk for complications. J Am Coll Cardiol 1991; 18: 1191-1199
XVII. Goldman ME, Fisher EA, Winters S, Reichstein R, Stavile K, Gorlin R, et al. Early identification of patients with native valve infectious endocarditis at risk for major complications by initial clinical presentation and baseline echocardiography. Int J Cardiol 1995; 52: 257-264.