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CASE REPORT

A RARE CASE: SLE WITH LIBMAN-SACKS ENDOCARDITIS

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ABSTRACT: Libman-Sacks endocarditis is the characteristic cardiac manifestation of autoimmune disease Systemic Lupus Erythematosus. The condition most commonly involves the mitral and aortic valves, but all the four cardiac valves and the endocardial surfaces can be involved[1]. Persons with Libman-Sacks endocarditis are usually asymptomatic., but become symptomatic due to cardiac failure, cerebrovascular embolism, systemic thromboembolism and secondary infective endocarditis. 2D Echocardiography should be performed when it is suspected. With introduction of steroid therapy for SLE, improved longevity of patients appears to have changed the spectrum of valvular disease. Herewith, we are reporting a 35 year old female who presented at Department of General Medicine, GGH, Guntur with seven day history of anasarca, shortness of breath, cough and fever of long duration. ECHO was suggestive of Libman-Sachs endocarditis and further investigations revealed SLE. On addition of steroids, there was good response and the patient was improving, but she had sudden cardio-respiratory arrest 5 days later.

KEYWORDS: Libman-Sacks endocarditis, Systemic Lupus Erythematosus, 2D Echocardiography.

INTRODUCTION: Libman-Sacks endocarditis is a form of nonbacterial endocarditis that is seen in SLE.[2] It is one of the most common manifestations of SLE (Most common being pericarditis). It was first described by Emanuel Libman and Benjamin Sacks.[3] SLE occurs 5 to 9 times more common in women, therefore cardiac valvular lesions are more common in women. Current ECHO studies reveal valvular abnormalities in 28%-74% of patients, with valvular masses in 4%-43% of patients with SLE. We are reporting a case of 35 year old female who presented with longstanding fever which was diagnosed as Libman-Sacks endocarditis and on further investigation diagnosed as a part of SLE.

CASE REPORT: 35 years old female presented at Department of General Medicine, GGH, Guntur with generalized oedema, shortness of breath and decreased urine output for 10 days, fever with joint pains for 4 days which was associated with productive cough. She is non-diabetic, normotensive. No past history suggestive of pulmonary tuberculosis or rheumatic heart disease.

Clinical examination revealed anaemia with anasarca. There is tachycardia and tachypnoea with low oxygen saturation. On auscultation, there is a pansystolic murmur in the mitral area with bilateral diffuse wheeze and coarse crepitations. Initially, she was diagnosed to have LRTI with Acute LVF. She was treated with broad spectrum antibiotics and Diuretics. Her investigations revealed Bilateral basal opacities with the possibility of Bilateral pneumonitis Vs CCF. 2 D echo showed evidence of Nodules over AML & PML. Which suggested the possibility of Libman-Sacks Endocarditis. Simultaneously, patient developed Oral Ulcers. Investigations showed:

- CBC: Hb – 8 g/dL, TC- 9000 cells/cu.mm, platelet count- 1,30,000; ESR- 110 mm/1st hr.
- RFT- Blood urea- 83 mg/dl, S. Creatinine – 1.9 mg/dl.

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- S. Sodium- 144 mEq/L, S. Potassium- 4.1 mEq/L, S. Bicarbonate- 22 mEq/L, S. Chloride- 114 mEq/L.
- LFT- S. Bilirubin- 1.1 mg/dL, S. Total protein- 7.1 g/dL, S. Albumin- 4.2 g/dL
- Blood cultures- NO organism grown.
- Urine albumin 2+; 24 hour urine protein- 0.6 g.
- Sputum C/S- E.Coli isolated; Sputum for AFB- Negative.
- Ultrasound abdomen- mild ascites.
- CXR PA view - Bilateral pleural effusions & basal edema/ consolidation with mild cardiomegaly.
- ECG- Normal sinus rhythm, left axis deviation. No ST-T changes.
- 2D ECHO**-** NODULES over AML, PML.

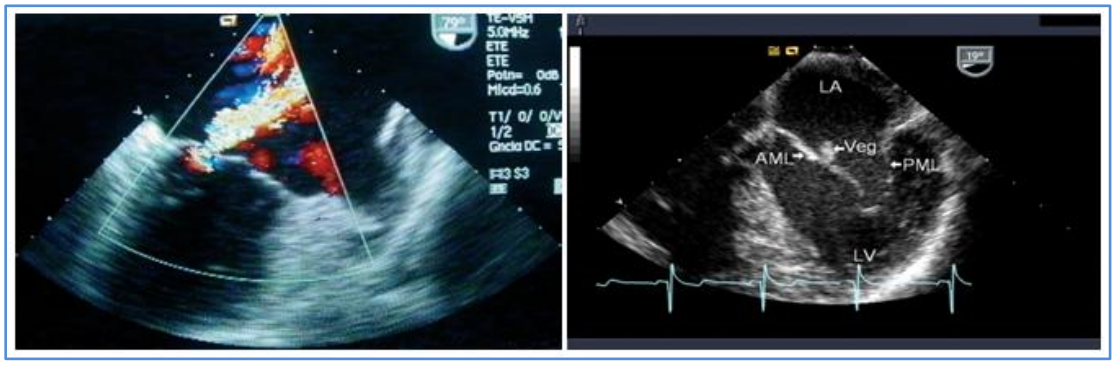

Figure 1

- Severe MR, TR, moderate PAH.
- No LV RWMA.
- Good LV, RV systolic function.
- ? Libmann Sacks Endocarditis.
- Further investigations revealed SLE.
- ANA- 3.37 (> 1.2 is positive).
- Anti-ds DNA- 44.88 IU/ml (>25 is positive).
- Anti Sm Antibody- 5.54 U/ml (> 10 is positive).
- APLA Ig M- 6.01 U/ml (> 15 is positive).
- APLA Ig G – 4.52 U/ml (>15 is positive).

She was initially treated with broad spectrum antibiotics, diuretics for which she showed no significant response. After diagnosing as SLE with Libman Sacks endocarditis sterois were added and there was good response and the patient was improving, but she had sudden cardiorespiratory arrest 5 days latter.

DISCUSSION: The possibities considered were:

- Infective endocarditis.
- Marantic endocarditis.
- Libman-sacks Endocarditis.

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Echocardiographic findings: In Infective endocarditis, the vegetations are large, irregular, mobile with rotatory or vibratory motion, distributed along the closure of leaflets and are homogeneous in echoreflectance. In contrast, Libman–Sacks vegetations are usually located at the basal, middle, or tip of leaflets, located on the atrial side of the mitral valve or vessel side of the aortic valve, are of variable sizes and shapes, and heterogeneous in echogenicity. Marantic endocarditis shows lesions located over the cusps and the patient usually has Hypercoagulable state like APLA syndrome.[4].

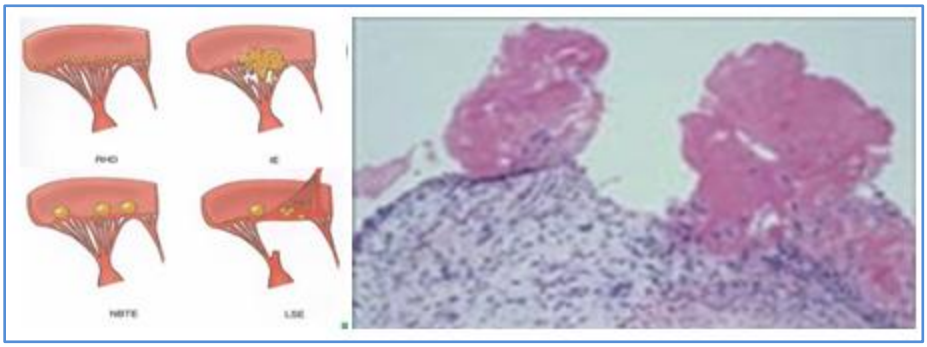

Figure 2

Final diagnosis: SLE with Libman-Sacks Endocarditis.

She was started on steroids with which her respiratory status improved but patient succumbed to sudden cardiorespiratory arrest five days later.

CONCLUSION: Libman-Sacks endocarditis is indeed described in literature as a postmortem diagnosis. The diagnosis was made antemortem in our patient by carefully considering the echocardiographic findings, negative blood cultures and improvement with steroids, but patient succumbed to sudden cardiorespiratory arrest probably due to cardiac arrhythmias common in Libman Sacks endocarditis.

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