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# A CASE REPORT ON SYSTEMIC LUPUS ERYTHEMATOSUS

**Ajeena Anto[1*], Tasneem Abdul Gafoor[1*], Maheshwari V[1*], Syam Prakash KR[1*], Sulfath T.S[2*], Sheik Haja Sherief[3*]**

*1Pharm- D Interns Nandha College of Pharmacy, Erode, Tamil Nadu, India*
*[2]Department of Clinical pharmacy, IQRAA International hospital and research centre, Calicut, India*
*[3]Head Of Department ,Department Of Pharmacy Practice ,Nandha College of pharmacy, Erode, Tamil Nadu, India.*

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**ABSTRACT**

Systemic Lupus Erythematosus (SLE) is an autoimmune, chronic multisystem inflammatory condition that occurs when the body's tissues are attacked by its own immune system. This has a broad clinical appearance, and is primarily hard to diagnose across the emergency departments (EDs). Most of the SLE-stricken patients continue to experience "secondary heart disease" during their primary illness. Most patients experiences severe fatigue, joint pain, swelling, headache, butterfly rashes and other symptoms which depends on the area affected. A 29 year old female patient presented to emergency department with complaints of generalized tiredness, several on and off seizure episodes, numbness of upper and lower limbs and blackish discoloration of big toe. Examination revealed patient was ill, tired, febrile with fever 100.5°F,tachycardia and oxygen saturation 96%. The patient was diagnosed with probable SLE. The patient was treated with Hydrocortisone, hydroxychloroquine, antiepileptics, prednisone and antibiotics and noticed symptomatic improvement. Multisystemic and acute life-threatening conditions may be suspected for autoimmune diseases, predominantly SLE. The treatment shall be planned separately taking into account the use of "best-suited medication" for addressing the affected organ system. Lack of an explicit biological marker, the heterogeneity of the disease and the absence of a specific outcome measurement for improvement makes this procedure more difficult.

***Corresponding author***
**Ajeena Anto**
PHARM D Intern.
Department of Pharmacy Practice,
Nandha College of Pharmacy, Erode, TamilNadu, India.
jafs21995@gmail.com

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## INTRODUCTION

Systemic lupus erythematosus (SLE) is characterized by auto antibodies against self-antigens, resulting in inflammation mediated multiorgan damage. Infections, cardiovascular diseases and renal failure which accounts for the majority of mortality in these patients[1]. The Lupus foundation of America estimates prevalence to be atleast 1.5 million cases [2]. In 2008 National arthritis working group study reported the prevalence of 161000 cases of SLE identified and 322000 cases of definite or probable SLE[3]. The frequency of SLE varies by race and ethnicity, with higher rates reported in Blacks and Hispanics. The complications seen in the EDs should be treated in the normal way, the most often seen being pulmonary embolism, respiratory embolism, respiratory arrest, hemoptysis,acute myocardial infarction, and stroke[4,5]. Numerous other complications including cerebritis, renal dysfunction, pulmonary hemorrhage, and pericardial tamponade can be treated by proper consultations[6]. Congestive heart failure in patients with SLE is often multifactorial in origin[7]. Individuals with lupus have a considerably high risk of stroke, atherosclerosis or early "coronary heart disease" (CHD) and many other "cardiovascular-related disorders" as relative tothose without lupus[8]. Numerous clinical signs include proteinuria, renal involvement, muco-cutaneous involvement/ ulcers, malar rash, seizures, urinary cellular casts, fever, hemolytic anemia, thrombocytopenia, and lymphadenopathy[9]. People with SLE often experience a wide range of symptoms as well as have multiple combinations of the organs involvement, while no definitive test can establish the systemic lupus diagnosis[10]

To assist health care professionals to improve the SLE diagnosis accuracy, 11 such criteria were identified by the American Rheumatism Association[11]. These include discoid skin rash (redness in patches with hypopigmentation and hyperpigmentation tends to cause scarring), arthritis (more than two tender and swollen joints of the extremities), malar (observed on face mainly over the cheeks) "butterfly" rash, irritation of brain (manifested by psychosis or seizures referred to as "lupus cerebritis"), pericarditis or pleuritis, mucous membrane ulcers, antinuclear antibody, blood-count abnormalities, photosensitivity (rash observed in skin in reaction to exposure to ultraviolet light or sunlight), and abnormalities in kidney[7].

Pharmacological therapy varies depending on the severity of the disease condition and the organ systems involved. Patients with mild symptoms may only require the use of NSAIDS or antimalarials, while more severe disease may require corticosteroids or immunosuppressant[12]. Unfortunately there is a higher mortality risk due to a higher risk of complications, and adverse effects due to high doses can make interpretations of clinical presentations or clinical responses complicated[12]. It is therefore important that the management of SLE patients require a healthcare team of multiple disciplines and professions, with close monitoring and control to prevent the worsening of the disease or the development of complications. This case study presents a case of an SLE patient who has developed complications during her course of treatment. The study aims to emphasize the need for proper guidelines for management of SLE and its complications that could change the disease course.

## CASE PRESENTATION:

A 29 year old female patient was admitted to the emergency department with complaints of generalized tiredness, fever with joint pain, swelling, and few episodes of seizure on and off and blackish discoloration of right prig toe. The patient had numbness on left upper and lower limb. On local examination she had butterfly rashes over the cheeks and gangrenous blackish discoloration over the right big toe and her vital signs were noted for tachycardia 143 beats per minute, temperature 100.5°F and BP 100/60 mm Hg.
Initial laboratory tests demonstrated hypothyroidism and severe inflammatory condition: hemoglobin of 11.2 g/dL (12.0 - 16.0 g/dL), platelets 380 × 109/mL, white blood cells (WBC) count of 25.3 × 103/μL, CRP of 78.8010 mg/L (< 10mg/L) ,TSH of 10.61 mU/L (0.4 – 4mU/L). Other investigations include beta 2 glycoprotein IgG:Negative, C3-81mg/dl, C4-50mg/dl, lupus anticoagulant: Negative and AntidsDNA:68. The CT Brain report shows large left MCA territory infract with mass effect and multiple other infarcts in left capsuloganglionic region left thalamus mid brain and pons which indicating CVA. The ECHO reports show LV dysfunction indicating myocarditis secondary to SLE. The HRCT of lungs revealed patchy ground glass opacity in the posterior segment of right upper lobe, few subtle ground glass opacities scattered in the basal segments of left lower lobe and few fibro atelectatic bands in the posterior basal segment of right lower lobe.Following days the patient had complaints of hypoxia, aspiration pneumonia, on and off fever and hypotension.The culture test was performed and report revealed Staphylococcus haemolyticus in blood ,klebsiella species in Tissue and Bronchoalveolar lavage.

Based on all these findings she was diagnosed with SLE.She was treated with injection Hydrocortisone 50mg and later changed to oral prednisolone 25mg, Hydroxychloroquine200mg, Digoxin0.5mg, Aspirin 75,Eltroxin25mcg, antiepileptic such as Inj.Levetiracetam 500mg, Inj.Fosphenytoin 150mg and antibiotics such as Inj.Meropenem 2g and Inj.Colistin 3mu.The patient showed symptomatic improvement through the course of treatment.

## DISCUSSION:

SLE carries the highly variable prognosis for individual patients. SLE's evolutionary history varies from fairly benign to fast growing and often lethal illness. The disease characteristics often vary between the individuals.

SLE is an autoimmune disorder characterized by multisystem inflammation, clinical manifestation, relapsing and remitting course. 90% of the SLE cases occur in women, particularly at child bearing age. The nature of the disease is milder and the chances of survival are higher in persons with isolated skin and musculoskeletal involvement than for those with the renal diseases[13] and CNS diorders[14].Congestive heart failure in patients with SLE is often multifactorial in origin[15]. Individuals having lupus possess a significantly high risk for stroke, atherosclerosis or premature "coronary heart disease" (CHD) and many other "cardiovascular-related conditions" as compared to those deprived of lupus[16] .

The diagnosis of the SLE is based on combination of clinical results and laboratory data. In 2008, The American College Of Rheumatology (ACR) and The European League Against Rheumatism (EULAR) published new standards for the classification of SLE[17]. Prognosis factors from the EULAR recommendation[18].

- Clinical findings: Skin lesions, arthritis, serositis, neurologic manifestations such as seizures, psychosis and renal involvement
- Diagnostic study results: Anemia, thrombocytopenia, leukopenia, increased serum creatinine levels
- Immunologic test results: Serum C3 and C4 concentration (which may be low), as well as the presence of anti–double-stranded DNA (anti-dsDNA), and antiphospholipid (aPL), and anti-ribonucleoprotein (anti-RNP).

Management of systemic lupus erythematosus (SLE) often depends on disease severity and disease manifestations, [19] although hydroxychloroquine has a key role for long-term treatment in all SLE patients. Cutaneous symptoms, musculoskeletal symptoms, and serositistypically represent a milder disease, that may wax and wane with the progression of the disease. These are often regulated with nonsteroidal anti-inflammatory drugs (NSAIDS) or low-potency immunosuppression drugs beyond hydroxychloroquine and/or short courses of corticosteroids. More prolonged steroid use is generally intended for cases which involvesvital organs[20].In addition to thenon-pharmacologic and pharmacological therapies, the primary concern for the individuals with SLE requires medical and educational resources. Drug therapy for SLE are designed to suppress the inflammation and immune responses[7].

## CONCLUSION

The current case report implies that SLE should be considered during refractory heart failure to substantial conventional therapy, exclusively in young females. Furthermore, early corticosteroids treatment, either with or without immunosuppressive agents, might result in improved and better outcomes. SLE treatments need to be planned on individual basis with primary consideration in order to use the best suited therapy to target the affected organ systems. Lack of certain biological markers, heterogeneity of the disease as well as absence of single outcome measurement for improvement often makes it a critical process. It is therefore important to involve professionals from various disciplines, to form a multidisciplinary team, depending on the organ systems affected. Fast and reliable flow of information is crucial among clinicians working together to provide a positive prognosis for the patients. There needs to be constant monitoring and evaluation of therapeutic regimens of the patient along with their response, due to the complicated and severe nature of the disease.

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## ABBREVIATIONS

| | |
|---|---|
| SLE | -Systemic Lupus Erythematosus, |
| ED | -Emergency Department, |
| CHD | -Coronary Heart Disease, |
| NSAIDS | -Non Steroidal Anti Inflammatory Drugs, |
| CT | -Computed Tomography, |
| ECHO | -Echocardiography, |
| HRCT | -High Resolution Computed Tomography. |

## CONFLICT OF INTEREST

The authors declare that they have no competing interests.

## REFERENCES

1. Ward MM, Pyun E, Studenski S. Causes of death in systemic lupus erythematosus: long-term followup of an inception cohort. *Arthritis Rheum 1995*;38:1492–9
2. Lupus Foundation of America. Understanding lupus. Available at https://www.lupus.org/understanding-lupus-0?utm_expid=.7AO1HbsrR9KO2Z8jF1HYTw.1&utm_referrer=. 2020; Accessed: March 11, 2020.
3. Helmick CG, Felson DT, Lawrence RC, Gabriel S, Hirsch R, Kwoh CK, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. *Arthritis Rheum*. 2008 Jan. 58(1):15-25.
4. Nossent J, Kiss E, Rozman B, Pokorny G, Vlachoyiannopoulos P, Olesinska M, Marchesoni A, et al. Disease activity and damage accrual during the early disease course in a multinational inception cohort of patients with systemic lupus erythematosus. Lupus. 2010;19(8):949-956.
5. Shah MA, Shah AM, Krishnan E. Poor outcomes after acute myocardial infarction in systemic lupus erythematosus. J Rheumatol. 2009;36(3):570-575.
6. Rosenbaum E, Krebs E, Cohen M, Tiliakos A, Derk CT. The spectrum of clinical manifestations, outcome and treatment of pericardial tamponade in patients with systemic lupus erythematosus: a retrospective study and literature review. *Lupus.* 2009;18(7):608-612.
7. Mohammed Huneif. Diagnosis and Management of Systemic Lupus Erythematosus: A Case Report.*Elmer press J Med Cases*. 2017;8(5):163-166

8. Manzi S, Meilahn EN, Rairie JE, Conte CG, Medsger TA, Jr., Jansen-McWilliams L, D'Agostino RB, et al. Agespecific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. *Am J Epidemiol*. 1997;145(5):408-415
9. Livingston B, Bonner A, Pope J. Differences in clinical manifestations between childhood-onset lupus and adultonset lupus: a meta-analysis. *Lupus*. 2011;20(13):1345- 1355.
10. Edworthy SM. Clinical Manifestations of Systemic Lupus Erythematosus. In: Harris ED, et al, eds. Kelley's Textbook of Rheumatology. 7th ed. Philadelphia, *Pa: WB Saunders*; 2005. p. 1201-1224.
11. Shiel WC, Jr. Systemic Lupus Erythematosus (SLE or Lupus), Medicine net; 2015; retrieved from http://www. medicinenet.com/systemic_lupus/page4.htm.
12. Salman Azeem1 , Syed Wasif Gillani2 *, Vinci Poh3 and Syed Azhar Syed Sulaiman4. Systemic lupus erythematosus (SLE) and its complications: A case study.*Oat open access.* ISSN: 2059-0393
13. Faurschou M, Dreyer L, Kamper AL, Starklint H, Jacobsen S. Long-term mortality and renal outcome in a cohort of 100 patients with lupus nephritis. *Arthritis Care Res (Hoboken)*. 2010 Jun. 62(6):873-80
14. Kasitanon N, Magder LS, Petri M. Predictors of survival in systemic lupus erythematosus. *Medicine (Baltimore)*. 2006 May. 85(3):147-56.
15. Ansari A, Larson PH. Heart disease in systemic lupus erythematosus: diagnosis and management. *Tex Heart Inst J*. 1985;12(1):9-21.
16. Manzi S, Meilahn EN, Rairie JE, Conte CG, Medsger TA, Jr., Jansen-McWilliams L, D'Agostino RB, et al. Agespecific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. *Am J Epidemiol*. 1997;145(5):408-415
17. https://emedicine.medscape.com/article/332244-overview
18. Bertsias G, Ioannidis JP, Boletis J, et al. EULAR recommendations for the management of systemic lupus erythematosus. Report of a Task Force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics. *Ann Rheum Dis*. 2008 Feb. 67(2):195-205
19. Bertsias G, Fanouriakis A, Boumpas DT. Treatment of Systemic Lupus Erythematosus. Firestein GS, Budd RC, Gabriel SE, MacInnes IB, O'Dell JR, eds. *Kelley and Firestein's Textbook of Rheumatology*. 10th ed. Philadelphia, Pa: Elsevier Saunders; 2017. 1368-88.
20. Broder A, Khattri S, Patel R, Putterman C. Undertreatment of Disease Activity in Systemic Lupus Erythematosus Patients with Endstage Renal Failure Is Associated with Increased All-cause Mortality. *J Rheumatol*. 2011 Nov. 38(11):2382-9.