Section: Medicine

Case Report

SYSTEMIC LUPUS ERYTHEMATOSUS: A FEMALE CASE REPORT HIGHLIGHTING CHALLENGES AND MANAGEMENT OF DISEASE

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Abstract

SLE is an autoimmune disease that results from chronic activation of the immune system with the production of antibodies. The cause of SLE is not clear and is thought to have genetic and environmental factors. Hereby we present a 45-year-old female with complaints of right-sided weakness in her upper and lower limb for two days. After the investigations, the patient was diagnosed as a case of SLE with right-sided hemiparesis with Liebman Sack Endocarditis with lupus nephritis with hypothyroidism. The patient was started on Inj. Ceftriaxone 1mg. Inj. Linezolid 600 mg, Inj. Methylprednisolone 500 mg, Tab. Torsemide 10mg, Tab. Thyroxine 25mg, Tab. Levetiracetam 500mg. The patient showed improvement after the treatment and was discharged after 20 days of admission. SLE is a chronic autoimmune disease with significant multisystem involvement with significant morbidity and mortality if not diagnosed or treated on time.

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is an autoimmune disorder commonly known as lupus. Though the cause of the disease is unknown, the female gender is a major risk factor, and various genetic, hormonal, immunological, and environmental factors are also thought to play a significant role.[1,2] It is characterized by the presence of various autoantibodies; the most common among them is antinuclear antibody (ANA).[3] SLE most frequently involves the skin and joints, although serositis, nephritis, hematologic cytopenia, and neurologic manifestations may occur during the course of the disease. The heterogenicity of the disease and its ability to affect any organ makes the diagnosis difficult.

CASE REPORT

A 45-year-old female presented with complaints of right-sided upper and lower limb weakness for two days [Figure 1]. She has been a known case of seizure disorder for the last two years and is on a tablet of Levetiracetam 500mg. She is also a known case of Hypertension and ischemic heart disease, for which she is on a tablet of Amlodipine 5mg and a tablet of aspirin, respectively. There is no significant obstetric history.

On general examination, the patient was drowsy but responded to verbal stimuli. Pulse rate and blood pressure were within normal limits. A soft systolic murmur and bilateral basal crepitations were present on systemic examination. Per abdomen, examination showed hepatomegaly. CNS examination showed Babinski's sign positive and power of 2/5 in the right upper and lower limbs, and on the left side, upper and lower limb power were found to be normal.

Figure 1: A 45-year-old female presented with complaints of right-sided upper and lower limb weakness for two days

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Routine Investigations were done, which showed a significantly low hemoglobin level of 4.1 mg/ dl, and a blood smear showed severe microcytic hypochromic anemia with thrombocytopenia. Renal function tests showed a blood urea level of 87mg/dl and a serum creatinine level of 2.1, suggesting acute kidney injury. The thyroid profile was suggestive of hypothyroidism. CT Brain showed acute infarct involving the left frontal, parietal, and occipital regions. Echocardiogram showed hypokinesia of the anterior wall, anterior septal wall, lateral wall, apex akinetic, moderate MR and PAH, ejection fraction was 25-30%, and also showed healed vegetations at the atrial part of the mitral valve. Ultrasonography showed moderate hepatomegaly with right- sided pleural effusion with basal consolidation, and the left kidney was hazy. All the blood culture samples were sterile. The urine routine showed proteinuria and granular casts. All the other investigations were within normal limits.

Due to the multi-systemic nature of the illness, an autoimmune condition was suspected, and specific investigations were done. Out of which, ANA, Anti dsDNA, SSA, Ro 52kD was positive. Though APLA, and anti-histone antibodies were negative. [Table 1]. After the investigations, the patient was diagnosed as a case of SLE with right-sided hemiparesis with Liebman Sack Endocarditis with lupus nephritis with hypothyroidism. The patient was started on Inj. Ceftriaxone 1mg. Inj. Linezolid 600 mg, Inj. Methylprednisolone 500 mg, Tab. Torsemide 10mg, Tab. Thyroxine 25mg, Tab. Levetiracetam 500mg. The patient showed improvement after the treatment and was discharged after 20 days of admission. She was called for regular follow-up. She was maintained on tapering doses of prednisolone & mycophenolate mofetil. She showed significant improvement on follow up visits [Figure 2].

Figure 2: Patient at follow up.

Table 1: Test conducted and their disease association.

Test	Disease association	Observation
Mi-2	Polymyositis and dermatomyositis	Negative
Ku	SLE, Sjogren's syndrome, Scleroderma, Myositis and MCTD	Negative
Sm/RNP	MCTD, Sharp syndrome	Negative
Sm	SLE	Negative
SSA	Sjogren's syndrome	Positive
Ro 52kD	Autoimmune (Sjogren's syndrome) & Infectious disease	Positive
SSB	Sjogren's syndrome, SLE	Negative
Scl-70	Systemic sclerosis	Negative
PM-Scl-100	Overlap syndrome (Polymyositis, Dermatomyositis and Systemic sclerosis	Negative
Jo-1	Polymyositis, interstitial lung fibrosis	Negative
CENP-A/B	CREST syndrome	Negative
PCNA	SLE	Negative
Anti dsDNA	SLE	Positive
Nucleosome	SLE	Negative
Histones	Drug induced lupus, Rheumatoid arthritis	Negative
Ribosome PO	SLE	Negative
AMA M2	Primary biliary cirrhosis	Negative

DISCUSSION

SLE is an autoimmune disease that results from chronic activation of the immune system leading to the production of autoantibodies and immune complexes.[3] Though the cause of SLE is not clear, it is thought to have various etiological factors. The biggest risk factor for SLE is female gender, with females being 7 to 15 times more prone to SLE.[1] Other than the female sex, various genetic, hormonal, environmental, and immunological factors play a role in the etiology of the disease.[2]

Our patient being a female and having multisystemic involvement raised the suspicion of a connective tissue disorder like SLE. Our patient fulfilled five criteria of the American College of Rheumatology for diagnosis of SLE, which are strong positive ANA, positive Anti dsDNA, seizures, right hemiparesis, casts in the urine, serositis-pleural effusion, and thrombocytopenia.[4] Additionally, she also showed healed vegetation at the atrial part of the mitral valve. These findings show valvular involvement and are consistent with non-bacterial thrombotic endocarditis occurring in SLE, known as Libman-Sacks endocarditis.[5]

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Libman-Sacks endocarditis is a form of nonbacterial endocarditis that is seen in association with SLE. The prevalence of Libman-Sacks endocarditis in SLE is 11-74%, with the mitral valve being the most common valve to be involved in SLE.[5-7] It typically appears as pea-sized, flat or raised, granular lesions that occur most commonly on the ventricular aspects and posterior leaflet of the mitral valve.

SLE has the potential to harm or complicate numerous bodily systems over time. Blood clots, blood vessel inflammation (vasculitis), pericarditis (inflammation of the heart), heart attack, stroke, memory loss, behavioral changes, seizures, lung inflammation (pleuritis), decreased kidney function, and kidney failure are some examples of potential complications.

As it is difficult to achieve prolonged remission,[8,9] the primary treatment of this disease is preventing flares and reducing their severity and duration when they occur.[10] Treatment includes corticosteroids and hydroxychloroquine.[11] Due to the variety of symptoms and organ system involvement with SLE, its severity in an individual must be assessed to treat SLE successfully. The mild or remittent disease may, sometimes, be safely left untreated. If required, nonsteroidal anti-inflammatory drugs and hydroxychloroquine may be used.[11] Medications such as prednisone, mycophenolic acid, and tacrolimus have been used in the past.

CONCLUSION

Our case report highlights the complexities in the management of SLE, a chronic autoimmune disease with significant multisystem involvement with significant morbidity and mortality if not diagnosed or treated on time. Our patient being a female and having multisystemic involvement raised the suspicion of a connective tissue disorder like SLE. The patient showed improvement after the treatment and was discharged after 20 days of admission. She showed significant improvement on follow up visits.

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