

CASE STUDY ON SYSTEMIC LUPUS ERYTHEMATOUS WITH COMPLICATIONS

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ABSTRACT

Systemic lupus erythematosus is an autoimmune disease in which organ and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complexes. Etiology of SLE is still unknown. Each patient presents with different sign and symptom or cause of SLE disease. A 27 yr old female patient was admitted in medicine ward at district general hospital, Amravati. This report refers to a patient presented with disorganized behavior, lack of restraint, difficulty in walking, edema on both feet and general weakness. Laboratory investigation showed her to be anemic, kidney function test showed a mild increase in serum blood urea level, liver function tests reports was normal and ultrasonography of abdomen and pelvis was normal. We report a case on SLE with CNS flare with iron deficiency

anemia with lupus nephritis, effectively treated with corticosteroid and antipsychotics. A specification in laboratory tests should be there. Corticosteroid and other agent' is use with their monitoring of adverse events. Regular laboratory test performs for rule out prognosis of a disease.

KEYWORDS: Systemic Lupus Erythematosus, Corticosteroids, CNS flare, Anemia, Psychosis, Lupus Nephritis.

INTRODUCTION

Systemic lupus erythematosus (SLE) is affected by the multisystem of the body.^[1] SLE is an autoimmune disease in which organ and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complexes. In most patients, autoantibodies are present for a few years before the first clinical symptom appears.^[4] The prevalence rate is more

common in female than male in age group 15-45yr childbearing age and also in the black race.^[2]

Etiology of SLE is still unknown. Each patient presents different sign and symptom or cause of disease. So the disease is highly unpredictable. Clinical manifestations of SLE are musculoskeletal: arthritis, arthralgia; constitutional: fatigue, fever, weight loss; mucocutaneous: butterfly rash, photosensitivity, Raynaud's phenomenon, discoid lesion; central nervous system (CNS): psychosis, seizures; pulmonary: pleuritis, pleural effusion; cardiovascular: pericarditis, myocarditis, heart murmur, hypertension; renal; gastrointestinal: nausea, abdominal pain, bowel hemorrhage; Hematologic: anemia, leukopenia, thrombocytopenia, lymphadenopathy.^[5]

Epidemiologic characteristic, clinical sign, and symptoms and common laboratory abnormalities all are used in the diagnosis of SLE. Antinuclear antibody (ANA) test is used to diagnose the SLE, but these test not specific only for SLE. Antibody to native DNA and to Sm antigen are quite specific consider for SLE diagnosis.^[5] Nonsteroid anti-inflammatory drug (NSAID), antimalarial, corticosteroid cytotoxic drug is used to treat SLE.^[2]

CASE REPORT

A 27 yr old female patient was admitted in medicine ward at district general hospital, Amravati. A patient has a history of the known case of systematic lupus erythematosus with iron deficiency anemia. There was no social history and family history of the patient. A patient was history of taken tab FS/FA 1 twice a day (bd), tab Calcium 500mg once a day (qd), tab Prednisolone 40mg qd. Chief complains of the patient were a general weakness, difficulty in walking and edema on both feet, disorganized behavior, lack of restraint. Physical examination was general condition moderate, temperature normal, pulse 82bpm, bp 110/70mmHg, pallor positive, repertory system air entry reduced by the right side, cardiovascular system tachycardia, central nervous system conscious oriented, per abdomen soft.

Complete blood count was WBC $5.36 \times 10^3/\mu\text{L}$, RBC $3.41 \times 10^6/\mu\text{L}$, PLT $75 \times 10^3/\mu\text{L}$, lymphocyte 16.3%, mid cells 8.2%, neutrophils 75.5%, Hb 7.9g/dl, MCV 60.20fL, HCT 27.3%, MCH 23.2pg, MCHC 28.9g/dl, RDW CV 18.2%, RDW SD 58fL. Kidney function test (KFT) was serum blood urea 24.5mg/dl, blood urea nitrogen 11.44mg/dl, serum creatinine 0.99mg/dl. Liver function test was serum bilirubin total 0.47mg/dl, serum bilirubin

direct 0.21mg/dl, serum bilirubin indirect 0.26mg/dl, SGOT 15.4U/L, SGPT 20.7U/L, albumin 3.9g/dl. Ultrasonography (USG) was normal. On the basis of history, chief complaints and diagnostic test, treatment advice was pack cell volume (PCV) 1 unit, inj Iron Sucrose 100mg iv qd, inj Taxim 1gm iv bd, inj Ranitidine 50mg iv three times a day (tds), inj Lasix 20mg, tab Dyfor plus 1 qd, tab Risperidone 1mg qd, tab Prednisolone 40mg qd for 7 days. Patient diagnosed as SLE with CNS flare with iron deficiency anemia with lupus nephritis. Patient was feel better after 7 days of treatment but symptoms remain the same. Patient discharge with treatment tab Prednisolone 30mg qd for 7 days then 20mg for 7 days then 10mg for 7 days then 5mg for 7 days, tab Pan D 1 qd, tab FS/FA 1 qd, tab Calcimax 1 qd, tab Risperidone 1mg bd. After 1 month, iron deficiency anemia was correct and improve psychosis symptoms.

DISCUSSION

Before making a diagnosis of systemic lupus erythematosus was ruling out drugs as the cause of the condition important. Many pharmacologic agents have been associated with lupus-like syndrome. A specific test for SLE is ANA, dsDNA, smith (Sm) antigen test but it was not performed in the patient. Diagnosis of SLE may be done on the basis of sign and symptoms and another supportive laboratory test. Corticosteroids are first-line therapy for SLE. Corticosteroid act on SLE by anti-inflammatory and immunocompromise action. In SLE patients advice to avoid sun exposure due to verse the disease condition. Also drugs like Corticosteroid, Antimalarial, Anticonvulsants and Calcineurin inhibitor affect the metabolism of vitamin D. Corticosteroid uses in disease cause osteoporosis. Calcium and Vitamin D supplement reduced the event of hypocalcemia in SLE.

Anemia found in many patients with SLE. It is usually anemia of chronic inflammation, with mild normochromic, normocytic smear and low serum iron concentration but adequate iron stores. She was suffered from iron deficiency anemia with the lower value of hemoglobin (Hb 7.9g/dl) and MCV 60.20fL. PCV 1 unit, Iron Sucrose, and tab FS/FA were given to treat the iron deficiency anemia. Iron sucrose is dissociated into iron and sucrose and the iron is transported as a complex with transferrin to target cells including erythroid precursor cells. The iron in the precursor cells is incorporated into hemoglobin as the cells mature into red blood cells.

Psychosis can be a dominant manifestation of SLE; it must be distinguished from glucocorticosteroid induce psychosis. She was suffered from psychosis cause due to

Corticosteroid agent. Treatment for steroids induces psychosis is decrease dose of corticosteroid or antipsychotic agents. Risperidone was given for recovered psychosis symptoms of the patient.^[3] The exact mode of action is unknown. It is said to produce anti-psychotic effect via its antagonist action at various dopamine D2 and serotonin 5HT2 receptors in the brain.

Nephritis is usually the most serious manifestation of SLE. A urine analysis should order in any person suspected of having SLE. She had symptoms as edema on both feet and so that difficult to walk. The KFT of the patient was indicated normal value only serum blood urea mild increase as 24.5mg/dl. Laxis and Dyfor plus (Torsemide and Spironolactone) were given for nephropathy.^[2] Spironolactone is a potassium-sparing diuretic that acts by antagonism of aldosterone in the distal renal tubules. It is used mainly in the treatment of refractory edema in a patient with nephrotic syndrome.

CONCLUSION

We report a case on SLE with CNS flare with iron deficiency anemia with lupus nephritis, effectively treated with corticosteroid, iron supplement, diuretic and antipsychotics. A specification in laboratory tests should be there. Corticosteroid and other agent' is use with their monitoring of adverse events. Regular laboratory test performs for rule out prognosis of a disease.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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