Systemic lupus erythematosus in a male patient

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Abstract. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder with a broad spectrum of clinical presentations.\textsuperscript{1} Female to male ratio is approximately 9:1. A 20 years old male was admitted to HAM Hospital 3 months ago with chief complaint pain in both knees joint. After anamneses, physical examination and laboratory test the patient was diagnosed with systemic lupus erythematosus. The patient tested positive for ANA and anti-ds-DNA antibody test. The patient was with giving non-biologic DMARDS @myfortic 360mg, methylprednisolone, chloroquine and other symptomatic drugs.

1. Introduction
Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder with a broad spectrum of clinical presentations.\textsuperscript{1} Lupus can be a mild disease, a severe and life-threatening illness, or anything in between.\textsuperscript{2–5} Although the specific cause of SLE is unknown, there have been identifying multiple genetic predispositions and gene-environment interactions. There are genetic-susceptibility factors, environmental triggers, B-cell and T-cell interactions, antigen-antibody (Ab) responses, and immune clearance processes interact to generate and perpetuate autoimmunity in systemic lupus erythematosus (SLE). HLA = human leukocyte antigen; UV = ultraviolet light.\textsuperscript{6}

Prevalence: 2–140/100,000 worldwide but as high as 207/100,000. Incidence: 1–10/100,000 worldwide. The population at highest risk: women in their reproductive years. Female to male ratio is approximately 9:1 post-puberty and premenopausal. Environmental and exposure-related causes of SLE are less clear. Possible early-life risk factors include the following: low birth weight (<2,500 g), preterm birth (≥1 month early), childhood exposure to agricultural pesticides.\textsuperscript{7–10}

The pathogenesis of lupus remains unclear although the concept of apoptosis goes some way to explaining how the immune system may recognize predominantly intracellular antigens. Autoantigens are released by necrotic as well as apoptotic cells. Defects in the clearance of apoptotic cells have been described in SLE which may lead to aberrant uptake by macrophages which then present the previously intracellular antigens to T and B cells thus driving the autoimmune process.\textsuperscript{11–15}

Table 1. Classification criteria for SLE.

| Classification criteria | Definition |
|-------------------------|------------|
| Malar rash              | flat or raised, fixed erythema, tending to spare the nasolabial folds, over the malar eminences |
| Discoid rash            | Adherent keratotic scaling and follicular plugging is raised by erythematos; atrophic scarring may occur in |
Photosensitivity  
Skin rash is a result of unusual reaction to sunlight, by physician observation or patient history

Oral ulcer  
nasopharyngeal or oral ulceration is not usually pain, observed by a physician

Arthritis  
Non-erosive arthritis involving 2 or more peripheral joints, characterized by tenderness, swelling or effusion

Serositis  
a) Pleuritis  
heard by a physician or evidence of pleural effusion

b) Pericarditis  
documented by ECG or rub or evidence of pericardial effusion

Renal disorder  
a) Persistent proteinuria  
Proteinuria: more than 0.5 grams per day or greater than +++ if quantification not performed

b) Cellular casts  
Casts: may be red cell, hemoglobin, granular, tubular or mixed

Neurologic disorder  
See ACR definitions of 19 separate syndromes

Hematologic disorder  
With reticulocytosis

a) Hemolytic anemia  
Less than 4000/mm3 total on 2 or more occasions

b) Leukopenia  
Less than 1500/mm3 total on 2 or more occasions

c) Lymphopenia  
Less than 100,000/mm3 in the absence of offending drugs

d) Thrombocytopenia  
Antibody to native DNA in abnormal titer

Antinuclear antibody  
Abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay are at any point in time and there are no drugs known to associate with ‘drug-induced lupus’ syndrome

| Glucocorticoids/steroids: oral Prednisone 0.5-1 mg/kg/day for severe disease | Hydroxychloroquine: oral 200-400 mg/day |
|-----------------------------|--------------------------------------|
| Methylprednisolone: iv. lupus nephritis 1000 mg iv. for 3 days | Mycophenolamofetil: oral 2-3 g/day |
| Cyclophosphamide: iv. 7-25 mg/kg/month x6 or oral 1.5-3 mg/kg/day | Methotrexate: oral 10-25 mg once week |
| Azathioprine: oral 2-3 mg/kg/day | Topical glucocorticoids |
|                           | NSAIDs |

Table 2. Treatment of SLE.

2. Case Report
A male 20 years old, admitted to HAM Hospital 3 months ago with main complain pain in both knees joint. The patient stated pain, in the beginning, mild but in 1 month gradually increased. He could not walk along distance and easy to feel weak. He also felt redness on face following itch and sensitivity to sunlight on a half month ago. He was consulted to the general practitioner and was given analgetic to reduce the pain. After a month pain not decreased by consuming that tablet.

After anamneses, physical examination and laboratory test the patient was diagnosed with systemic lupus erythematosus. Laboratory test, Hb/WBC/Plt: 9.9 / 12080 / 281,000, Neutrophil / Lymphocyte /
Monocyte / Eosinophil / Basophil: 85/11 / 3.6 / 0.1 / 0.2%. Ureum / Creatinine: 21/ 0.5 mg/dl. ElectrolyteNatrium / Kalium / Chloride: 140/2.5/107 mEq/L. The patient tested positive for ANA: 204 and anti-ds-DNA antibody test: 742. Radiology imaging showed the normal result. The patient was hospitalized and given non-biologic DMARDS @myfortic 360mg (Mycophenolatemofetil) 2 times a day, methylprednisolone 12mg three times a day, chloroquine and other symptomatic drugs. After three months of laboratory finding back to normal, treatment continued, and the dose is tapered off. The complaint knee joint pain almost disappears, an itch on the face decreased, and now the patient can start to do this activity.

3. Discussion
Based on criteria ARA patient had six sign and symptoms: malar rash, arthritis, photosensitivity, anemia, immunologic disorder, antinuclear antibody. Patient categorized as mild SLE because there is no sign life-threatening and vital sign is considerable good. Prevalence SLE population at highest risk: women in their reproductive years with a female: male ratio is approximately 9:1 in this case patient was male and had no familial autoimmune disease. The patient was a farmer and often contact with agricultural pesticides, worked under the sun and consumed cigarettesand instant noodle. Immunological test ANA: 204 references strong, anti-ds-DNA: 742 references moderate confirm as Systemic lupus erythematosus.

Although the specific cause of SLE is unknown, multiple factors associated with the development of the disease, including genetic, epigenetic, ethnic, immunoregulatory, hormonal, and environmental factors. Patients with SLE must know to prevent triggers for flare.

People with SLE must prevent the ultraviolet light and sun exposure for minimizing worse symptoms from photosensitivity. Diet modification must base on the disease activity. A balanced diet has a lot of nutrition like vitamin B, C to prevent anemia, iron deficiency, and now check out the laboratory finding back to normal, threatening and vital sign vital.

Prognosis of the disease course is milder and the survival rate is higher in persons with isolated skin and musculoskeletal involvement than in those with renal disease and CNS disease.

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