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SYSTEMIC LUPUS ERYTHEMATOUS IN A MALE PATIENT – A RARE INCIDENCE

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Abstract:

Systemic lupus erythematosus (SLE) is an auto immune connective tissue disease in which the body immune system mistakenly attacks healthy tissue in many parts of the body, where vascular lesions are one of the typical symptoms. The pathological progress often involves skin vessels, renal glomeruli, cardiovascular system, brain, lung alveoli, and gastrointestinal tract vessels. We report a case of 35 year, old male patient who admitted in our hospital with rashes, fever, vomiting, severe joint pains, hair loss and burning sensation in stomach and was diagnosed to have SLE by anti nuclear antibody (ANA)profile.

Key words: *Systemic lupus erythematous, anti nuclear antibody profile.*

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INTRODUCTION:

Systemic lupus erythematosus (SLE), a disease characterized by the production of autoantibodies, complement activation and multisystem damage often presents itself with involvement of the gastrointestinal tract of several causes, ranging from adverse drug effects and infections related to immunosuppression, to pancreatic diseases, lupoid hepatitis, peritoneal serositis associated disease (celiac disease, inflammatory bowel disease) and ischemia [1]. Gastrointestinal (GI) manifestations of SLE are protean. Abdominal pain is the most common symptom but with different etiologies in SLE. GI vasculitis is very important to be recognized and treated early. Studies suggest that the clinical manifestation and serological features of old age lupus differ from lupus in younger patients. The female: male sex ratio declines from a 10:1 ratio in the younger patient to a 4:1 ratio in the older patient. This reduction has been related to the absence of sex hormones [2].

CASE REPORT:

A male patient of 35yrs old presented to the clinic with the chief complaints of

- Rashes since 3days over face and limbs, over head with hair loss
- Fever since 1 week on and off associated with body pains
- Vomiting since 10days
- Swelling of face and limbs since 3days
- Severe joint pains
- Burning sensation in stomach.

His past medical history shows that he had butterfly rashes over the body associated with bleeding one month back and diagnosed to have SLE treated with hydroxychloroquine, methyprednisolone, methotrexate and topical corticosteroids. On the day of admission his vitals were temperature- 100.5F, pulse-78bpm, respiratory rate -22bpm, Blood pressure – 110/80 mm of Hg.

ANA profile

RNP/sm – positive++
Sm Antibody – Positive ++
SS-A(Ro-60) – Positive++
Ribosomal P protein – strong positive +++

Interpretation:

- ❖ RNP/sm : Antibodies(Ab) are characteristic marker for SHARP syndrome, a multi symptomatic and multimix connective tissue disease combining characteristics of RA,SLE, systemic sclerosis and polymiositis
- ❖ Sm Antibody: Ab against Sm are highly specific (>90%) for SLE and can be found in 5-40% of patients.
- ❖ SS-A(Ro-60): Ab to SSA most commonly occur in patients with Sjogrens syndrome but also in SLE and primary biliary cirrhosis and occasionally in HIV, viral hepatitis and 100% cases of neonatal lupus erythematosis.
- ❖ Ribosomal P protein: Ab against Ribosomal P protein is specific to SLE. These are also detected in SHARP syndrome.

Table 1: Diagnostic investigations:

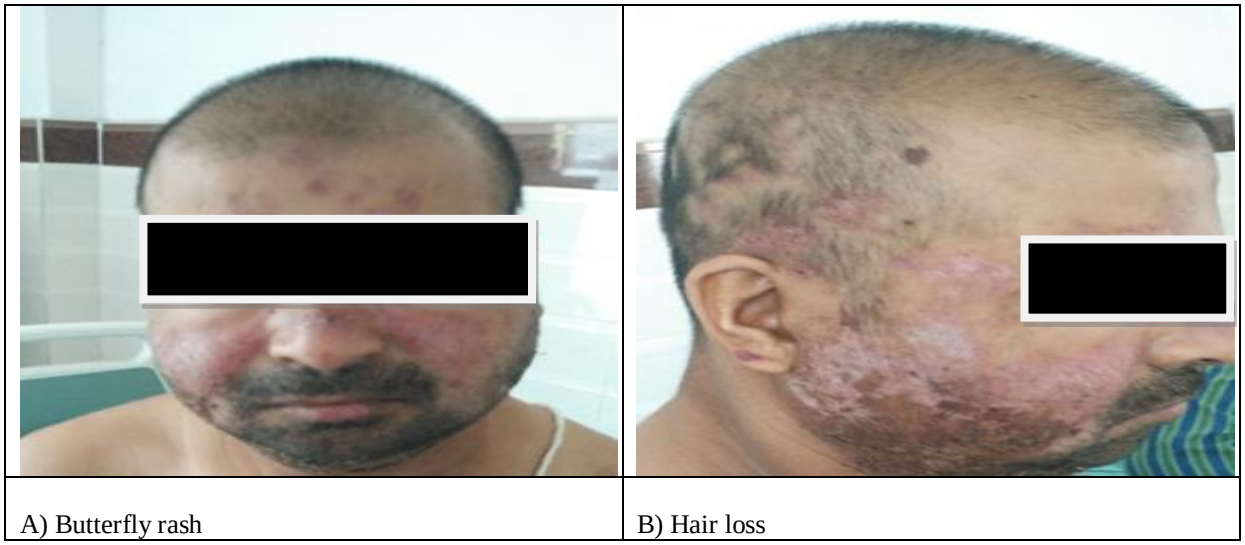
Lab reports:

Analyte	**Test value**	**Normal value**
RBS	114mg/dl	80-160
Blood urea	28mg/dl	10-50
Sr.creatinine	1.2mg/dl	0.6-1.5
Liver function test		
Total bilirubin	0.6gm/dl	0.2-1.2
Direct bilirubin	0.2mg/dl	0-0.4
SGOT	**153IU/L**	5-34
SGPT	**139IU/L**	Upto42
ALP	249IU/L	80-290
Total protein	6.8gm/dl	6.2-8
Albumin	3.5gm/dl	3.2-5
Globulin	3.3gm/dl	2.2-4.2
Electrolytes		
Sodium	127	135-155
Potassium	4.9	3.5-5.5
Chloride	99	90-110

Table 2: Antinuclear Antibody Test: Patterns, Antigens, and Specificities

Pattern	Antigen	Disease
Peripheral	DsDNA	SLE
Speckled	Acidic nuclear protein	Rheumatoid arthritis
	Ribonucleoprotein	SLE
	Extractable nuclear antigen	Scleroderma; mixed connective tissue disease
Homogeneous	dsDNA, ssDNA	Rheumatoid arthritis
	Histones	SLE; drug-induced lupus
Nucleolar	Nucleolar RNA	Progressive systemic sclerosis

Table 3 : US EXAMINATION OF ABDOMEN	
On day 2 of admission ✓ Grade-I right renal parenchymal disease ✓ Minimal ascities ✓ Minimal pleural effusion on right side	On day 6 of admission ✓ Grade I renal parenchymal changes ✓ Minimal ascities ✓ GB wall oedema ✓ Anterior abdominal wall oedema



A) Butterfly rash B) Hair loss

Fig 1: Patient with A) Butterfly rash B) Hair loss

The patient was treated with ceftriaxone 1gm, rabeprazole 20mg, paracetamol 650mg, ondansetron 4mg, intravenous methylprednisolone 1000mg daily with NS, semithecone 50mg+ megaldrate 540mg +oxetacine 10mg syrup, fluticasone 0.05%w/w cream.

After 4th day IV corticosteroid was stopped and was prescribed with azathioprine 50mg oral, deflazzacort 30mg oral. As the patient has experienced the withdrawal symptoms like insomnia, vertigo, confusion and anxiety he was treated with haloperidol 5mg and lorazepam 2mg the patients recovered with all the symptoms and get discharged after one week in good condition.

DISCUSSION:

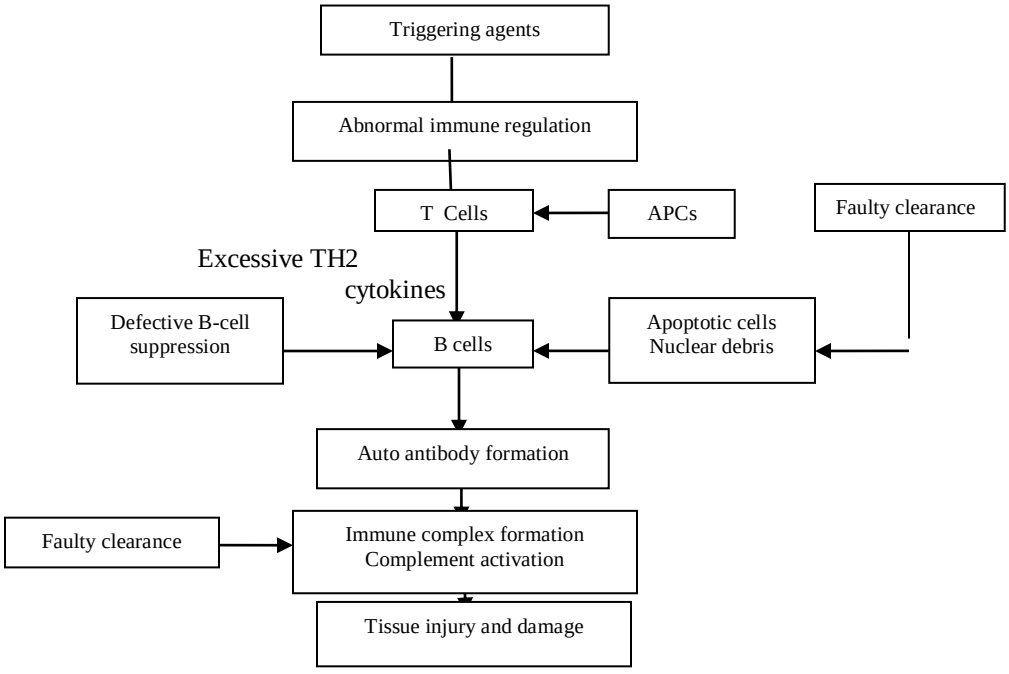
SLE is a fluctuating multisystem disease with a diversity of clinical presentations. Abnormal immunologic function and formation of antibodies against "self" antigens underlie the pathogenesis of SLE. The etiology of abnormal autoantibody production and development of SLE is still unknown. Genetic, environmental, and hormonal factors all may play a role in loss of "self" tolerance and expression of disease. A popular theory is that autoimmune disease such as SLE develops in genetically susceptible individuals after exposure to a triggering agent, possibly something in the environment [3-5].

In this patient the major event in the development of SLE is excessive and abnormal autoantibody production against multiple nuclear, cytoplasmic, and surface components of multiple types of cells in various organ systems and the formation of immune complexes.

Pathogenesis

A major event in the development of SLE is excessive and abnormal autoantibody production and the formation of immune complexes. Patients may develop autoantibodies against multiple nuclear, cytoplasmic, and surface components of multiple types of cells in various organ systems in addition to soluble markers such as immunoglobulin G and coagulation factors; these autoantibodies account for the multiple-organ system involvement of the disease. Excessive autoantibody production results from hyperactive B lymphocytes. Multiple mechanisms likely lead to B-cell hyperactivity, including loss of immune "self" tolerance and high antigenic load consisting of environmental and self antigens presented to B cells by other B cells or specific antigen-presenting cells, a shift of T-helper type 1 cells to T-helper type 2 cells that further enhance B-cell antibody production, and defective B-cell suppression. Impairment in other immune regulatory processes involving T lymphocytes (suppressor T cells), cytokines (e.g., interleukins, interferon-γ tumor necrosis factor-α, transforming growth factor-β), and natural killer cells also may be involved.

Many autoantibodies are directed against nuclear constituents of the cell; collectively, they are called antinuclear antibodies. Several antinuclear antibodies are important because their presence or absence may aid in the diagnostic and clinical evaluation of patients with SLE. The SLE patient may have more than one antigen-specific antinuclear antibody in the patient's serum and tissues. These are antibodies against such nuclear constituents as double-stranded, or native, DNA (dsDNA); single-stranded, or denatured, DNA (ssDNA); and RNA. Four RNA-associated antigens frequently occurring in SLE are the Smith (Sm) antigen, the small nuclear ribonucleoprotein (snRNP), the Ro (SS-A) antigen, and the La (SS-B) antigen. Histone, a basic component of chromatin and nucleosomes, is another important nuclear component against which antinuclear antibodies are formed in lupus patients. Antibodies to Ro, La, Sm, or RNP antigens plus antibodies to dsDNA will detect most patients with SLE. Antibodies also may be directed against the phospholipid moiety of the prothrombin activator complex (lupus anticoagulant) and against cardiolipin. The lupus anticoagulant and anticardiolipin antibodies constitute the two main types in a group of autoantibodies called antiphospholipid antibodies [6,7].



Flow chart 1: Pathogenesis of systemic lupus erythematosus.

Signs & Symptoms

- Achy joints / arthralgia
- Fever of more than 38 ° C
- Arthritis / swollen joints
- Prolonged or extreme fatigue
- Skin Rashes
- Anaemia
- Kidney Involvement
- Pain in the chest on deep breathing / pleurisy
- Butterfly-shaped rash across the cheeks and nose
- Sun or light sensitivity / photosensitivity
- Hair loss / Alopecia
- Abnormal blood clotting problems
- Fingers turning white and/or blue in the cold
- Mouth or nose ulcers

Achy joints, fever, skin rashes, butterfly rash across cheeks and nose, hair loss, light sensitivity were experienced by this patient

Treatment options [8, 9]

- Non Steroidal Anti Inflammatory Drugs (NSAIDS) :
  - NSAIDS have analgesic, antipyretic, and anti-inflammatory properties.
  - Drugs include:-ibuprofen, naproxen
- Antimalarial :
  - Hydroxychloroquine used as an adjunct to corticosteroid therapy.
  - Plaquenil can be used alone or with other drugs.
- Corticosteroids suppress the immune system and reduce inflammation caused by lupus.-prednisone
- Immunosuppressive drugs for patients whose kidneys and CNS is affected by lupus.
  - Cytoxan – restrain the overactive immune system by blocking the production of immune cells.
- Other therapies include:
  - Plasma exchange
  - Intravenous immunoglobin
  - Stem cell transplantation
  - Immune therapy

CONCLUSION:

Clinicians can prevent morbidity and mortality of SLE related GI vasculitis if they can recognize and treat it as early as possible. Abdominal computed tomography is a useful tool for detecting some abnormalities in abdominal pain of patients with SLE. SLE vasculitis is rarely confirmed with histology.

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