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RESEARCH ARTICLE

A SEVERE PRESENTATION OF DRUG INDUCED LUPUS ERYTHEMATOSUS WITH MULTI-ORGAN INVOLVEMENT: A RARE CASE REPORT

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ABSTRACT

Drug induced lupus erythematosus (DILE) is a subset of lupus, defined as a lupus-like syndrome that develops in temporal relation to exposure to a drug and resolves after cessation of the drug exposure. It can be induced by various medications. The presentation is often vague and needs a high index of suspicion to diagnose this condition which has a potential to cause significant morbidity[1]. It should be distinguished from multi systemic, auto-immune systemic lupus erythematosus (SLE). The clinical manifestations of DILE are usually mild to moderate and are rarely severe [2]. We present a 31-year-old female with severe multi-system dysfunction probably due to drug-induced lupus erythematosus that required immunosuppressive therapy. Specific classification criteria for drug-induced lupus have not been published [3].Based on the WHO-UMC [4] and Naranjo's causality assessment criteria[5], an association between the reaction and the offending drug was deemed possible. The reaction was severe (level 5) according to the Hartwig severity assessment scale [6].

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INTRODUCTION

The incidence of drug-induced lupus continues to rise as clinicians expand their diagnostic and therapeutic capabilities. The list of drugs that are associated with this disorder exceeds 100, including newer biologic agents, and the list continues to expand[7]. Although SLE has a striking female predominance, DILE has a more equal female-to-male distribution and an older age of onset[8]. Drug induced lupus eryethmatosus (DILE) results in clinical ,histological and immunological manifestations similar to SLE, but unlike SLE, DILE usually resolves with discontinuation of the inciting medication. The disease may occur days to years after the initiation of the drug and the onset is often gradual[2]. The mechanism of how drugs cause this autoimmune phenomenon is only partially understood. The complex disease pathophysiology includes such factors as the host's genetic and epigenetic susceptibility status, alterations in innate and adaptive immunity, certain drugs acting as haptogens and the generation of cytotoxic metabolites, which result in an autoimmune reaction, often causing detrimental effects on the target organs [9,10]. DILE commonly affects the liver but is unlikely to cause significant renal or CNS disease[2].

Multi-organ manifestations seen in idiopathic SLE are uncommon in drug-induced lupus[2]. DILE is characterized by a positive ANA, similar to idiopathic lupus . The ANA in DILE tends to have a homogeneous pattern, although a speckled pattern may also be seen. Other laboratory abnormalities include anti-histone antibodies, anti -SSA, anti -SSB, anti-dsDNA , ANCA, hematological abnormalities and normal complement levels although hypocomplementemia may be seen[11]. Anti–dsDNA antibodies are less likely to occur in drug-induced lupus than in SLE[12]. Presently there are no diagnostic criteria for DILE, the diagnosis is confirmed retrospectively. Treatment of drug-induced lupus involves withdrawal of offending medication and immunosuppressive therapy, if indicated[13]. Resolution of clinical findings may take months to years.

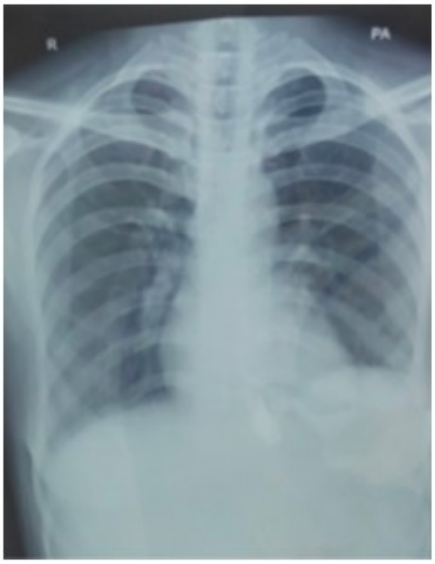
CASE REPORT

A 31-year old female patient had history of intermittent fever for last 10-12 days. She was already treated with some medications from local practitioner , the documents of which she could not provide.

	IDIOPATHIC SLE	DILE
AGE	20-45 YRS	GENERALLY OLDER
FEMALE :MALE	9:1	1:1
COURSE	CHRONIC, RELAPSING	REMITS WITH DISCONTINUATION OF DRUG
SEVERITY	MILD TO SEVERE	GENERALLY MILD
MAJOR ORGAN INVOLVEMENT	COMMON	RARE
CUTANEOUS MANIFESTATION	50-70%	5-25%
ANA +VE	>99%	>99%
ANTI-HISTONE AB	50-60%	>95%
ANTI -DS DNA	50-70%	<5%
HYPOCOMPLEMENTEMIA	50-55%	<1%

Before Treatment

After Treatment



Chest Xray

Following the intake of the unknown medications for a few days , she developed a rash over her face , generalized weakness and significant hair fall. She presented to our casualty with high grade fever, facial rash and in shock. On admission her vitals were as follows, BP - 84/54mmHg, HR-116/min, RR- 22/min , Temp - 102°F, SpO2- 98% on RA, RBS-93mg/dL and GCS - 14/15. She was admitted in the ICU under the provisional diagnosis of septic shock and was started on empirical antibiotics and vasopressor support . Drug reaction was also considered in the differential diagnosis and a call was given to the Dermatology department to evaluate the case.

Routine investigations were conducted which gave the following findings

- CBC
  - Normocytic, normochromic anemia - 8.2gm%
  - Leucopenia - 2000/Cumm, N46L46
  - Platelet - 2.25L
- Malarial Parasite Dual Antigen card test - negative
- Typhidot IgM and IgG - negative
- Covid 19 RTPCR - not detected
- HIV/ HbsAg/ Anti- HCV - negative
- KFT- WNL
- LFT
  - SGOT/SGPT- 136/120, ALP- WNL
  - GGT- 110
- CXR PA - Mild pleural effusion of left side.
- ECG- WNL, 2D-Echo - WNL
- USG (W/A) - Splenomegaly (14cm). Rest NAD.

After her first day of receiving supportive treatment , she remained in vasopressor support , with no improvement in her facial rash and fever incompletely responding to antipyretics. She further complained of mild-moderate large joint pains without any objective clinical evidence arthritis. On the second day her blood reports revealed further anemia (6.3gm%) and leukopenia (2700), liver enzymes increased further. Her clinical condition remained same, without any improvement or worsening. IV steroids were started in consideration of auto-immune etiology/ drug reaction and empirical Doxycycline started to cover for atypical organisms and Scrub typhus. Investigations of ANA and scrub typhus

serology were sent. On her third day, she showed dramatic improvement in her clinical condition , her rash had reduced significantly, she was afebrile without antipyretics and her vasopressor requirements decreased but not completely. Her blood investigation revealed improvement in anemia (8.4gm%) and leukopenia (4700), but liver enzymes were still elevated to the same levels. On her fourth day, she showed further improvement in her clinical condition. She was afebrile and blood pressure remained stable as she was slowly tapered off vasopressor support completely. Her ANA report was positive with homogenous pattern at 1:1000 titre. Scrub typhus serology negative. She fulfilled EULAR 2019 criteria for classification of SLE[14]. She was transferred to the general ward the next day and was started on oral steroids. Antibiotics were stopped. She remained clinically stable and was discharged the next day with the diagnosis of Drug Induced SLE, on oral steroids ,planned to be tapered off over the next few weeks. She has remained asymptomatic in her follow up visits while the steroids were gradually tapered off.

DISCUSSION

Drug induced SLE (DILE) commonly presents with mild to moderate severity but severe manifestations occur rarely. The number of offending agents associated with this entity continues to expand. With the ever increasing population in our country, the demand for professional medical attention far outstrips the available supply. This huge gap is partially met with the help of unqualified medical practitioners (quacks). Quacks often prescribe medications without proper documentations, hence drug intake history should be thoroughly inquired about and drug reactions and adverse effects need to be kept in mind while attending patients, especially in rural areas of our country.

CONCLUSION

Establishment of the diagnosis of DILE requires a high level of suspicion, frequently leading to a delay in the diagnosis. Drug induced reactions should always be considered when the etiological cause is not easily perceived. A temporal association between the initiation of the suspected drug and the onset of the symptoms might be difficult in some cases as DILE may occur years after drug initiation. In our country, due to quack practitioners , the drug history should be actively looked into, even when there is no documentation. In the absence of any classification criteria , clinical improvement of symptoms after the withdrawal of the drug confirms the diagnosis.

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