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A Case of SLE with Seizure Disorder with Encephalitis (Systemic Lupus Erythematosus)

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I. INTRODUCTION :-

SLE (Systemic Lupus Erythematosus) is a chronic autoimmune disorder known to affect the body's various organs, such as skin joints, lungs, brain, kidney and blood vessels

The American college of Rheumatology develops the criteria which are used for the diagnosis of lupus SLE diagnosis is made depending upon both the laboratory results and clinical manifestations (3)

SLE is an autoimmune disease, and its symptoms very widely depending upon the organs involved. Due to its multisystem involvement, it sometimes poses a challenges for the physician to diagnose SLE patients (I-3)

Neuropsychiatric SLE refers to the presence of various neurological manifestations that developed after the involvement of CNS. It can either present as a diffuse from the is coma, psychosis, depression, or encephalopathy or in a complex from that is a psychiatric presentation with seizure stroke and encephalopathy with seizure or stroke (4)

Neuropsychiatric symptoms can be presentabout 10% to 80% of patients before diagnosing SLE or during this disease (5). The symptoms may include mood disorders, headaches, seizures, and cognitive decline.

The mechanisms of damage to the CNS tissue have not been formulated yet, but almost half to SLE seizures are either due to infections or metabolic disorders.

The cytokines IL6,IL1 and TNF alpha formed during the active disease are found to activate the HPA (hypothalamic pituitary axis) thereby decreasing the seizure threshold (6)
Seizures usually caused by SLE are usually generalized tonic-clonic lending to its bad prognosis.(7)

Case Presentation :-

Here we report a case of 17 year old female presented with involuntary movements of limbs from morning (convulsions) which is associated with 25-30 min of loss of consciousness, up rolling of eyeball , headache , vomiting (1-2 watery episode) , frothing from mouth , no h/o tongue bite , post episode confusion Complain of bilateral calf pain and fever for 2 days, there is black to brown coloredstaining of b/l calf area which is not painful or reddish.

Female with SLE ((Systemic Lupus Erythematosus) has 5 episodes of GTCS after headache and serum ANA & ANA Profile is positive and EEG showing seizure activity and MRI brain suggests encephalopathy in left high frontal lobe & parasagittal region. Patient symptoms improved after anti-epileptic, methyl prednisolone medication therapy.

Her previous diagnostic studies were within normal range and no precipitating cause for the seizure was found.

Lab Investigations :-

Hemoglobin (10.4mg/dl), platelets (45000/mm3) and WBC count (4720/ mm3) was recorded, Crp (3.78), Rbs (120mg/dl), Creatinine (1.23), Sgpt (85.2), Na+ (143.1), K+ (3.94), Upt (negative).
Antibody panel and antiepileptic drug blood level.
The patient's ANA (1.320-positive) with Speckled pattern.
2DEcho (55% LVEF, concentric LVH).
RAfactor, Anti-SM antibodies, Antiphospholipid antibody were negative.

Although the patient was asymptomatic there was a slight reduction in her WBC and platelet count compressed to the previous report. Her renal functions test, liver function tests, thyroid function tests , serum electrolytes, toxicological screening of urine, blood, Lumbar puncture were within normal limits

Brain imaging studies :Mri brain with venogram Possibility of mitochondrial encephalopathy involving b/lparasagital region and left high frontal lobe and right frontal lobe

EEG :8-10 hz , b/l symmetrical , moderate in amplitude nd few spikes and wave on photic stimulation.

Criteria for diagnosis :
1.Clinical criteria
2. Immunological criteria

A neurologic disorder withseizures in the absence of any other cause and hematologic disorder with pancytopenia makes up to clinical criteria. The immunological criteria show that anti-dsDNA and the ANA were strongly positive (9)

Treatment :-

Once the patient was diagnosed with autoimmuneepilepsy we started treatment with IV methylprednisolone lg/day for three days with supportive treatment following which she was discharged after careful evaluation with no seizure episodes.

We continued the patient on oral levetiracetam and oral prednisolone 45mg tapering itgradually for SLE and the patient is on follow up for five months since then the patient has had no seizure episodes or any sings of SLE.

II. DISCUSSION :-

The epidemiological data for SLE illustrates that it is most prevalent in southeast Asain ethnicity than Caucasians, the SLE being more prevalent amongst females (7-9)

The disease pathology is still not identified, but it is found that it is due to autoantibodies, inflammatory cytokines nonspecific which ab and ag, or specific complexes which causes neuronal damage.

The seizures in neuropsychiatric SLE arise from the medical temporal to be but in our patient we did not find any changes in the EEG post statues epilepticus (12)

Usually the patients with NPSLE responds to initial treatment with prednisolone and cyclophosphamide, but our patient responded to treatment with prednisolone and showed improvement in symptoms of SLE and is under remission (14)

There are reported cases that patients with SLE also presents with cardiomyopathy as its secondary complication, but out patient's cardiac status was normal (13). This case emphasizes the need for neurological evaluation in every SLE patient(10)

III. CONCLUSION :-

Seizures are common among patients with SLE but Status epilepticus is one of the rare phenomena at the presentation in Neuropsychiatric SLE (11)

CNS manifestations is strongly associated with hematologic manifestations (11)

This report highlights the importance of studyingautoimmune epilepsy associated with SLE. We encourage other physicians to report such a rare presentation.

The autoimmune etiology of the seizures should always be considered it patient is having recurrent episodes of seizures without any precipitating factors and despite being on a regular medication.