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Original Research Paper

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SYSTEMIC LUPUS ERYTHEMATOSIS PRESENTING AS LUPUS ENCEPHALITIS IN A YOUNG FEMALE.

Dr. H.l. Haritha Resident, Department of General Medicine, Mahatma Gandhi Medical College And Research Institute, Puducherry,india

Dr. Arun Prakash.T Assistant Professor, Department of Neurology, Mahatma Gandhi Medical College And Research Institute, Puducherry, India.

ABSTRACT Systemic lupus erythematosis can cause a variety of neurological and psychiatric symptoms. It mostly affects females of reproductive age group(15-45 years). Hereby, we present a case of systemic lupus erythematosis presenting as lupus encephalitis in a young female. Patient had altered sensorium and seizures on presentation. Routine investigations, CT brain and Lumbar puncture was normal. Patient improved after steroids and IV immunoglobulin therapy.

KEYWORDS : Lupus encephalitis, seizures, steroids, iv immunoglobulins, neuropsychiatric manifestations.

INTRODUCTION:

CNS lupus manifestations are multifactorial(3). Pathophysiology can be due to vascular event or cytokine mediated cell damage (3). ACR/EULAR criteria defines 19 distinct clinical central(12) and peripheral(7) neuropsychiatric symptoms that can occur in SLE(2). Multiple triggers which damage blood brain barrier such as infection, stress(3). Seizures are common in SLE, both generalised and focal(3). Can be because of autoimmune epilepsy or a lowered seizure threshold(5). Early treatment is necessary for good prognosis.

CASE REPORT:

This 40 year old female, presented with five episodes of seizures and altered sensorium since one day. On arrival, patient was drowsy, not obeying commands, GCS-E2V2M5 (9/15). Her blood pressure 160/110 mmhg, Pulse-72/min, Spo2-95% at room air, RR-16/min. CNS examination showed decreased tone in all 4 limbs, B/L pupils 2mm ERTL, plantars were bilateral extensor. Other systemic examinations were normal. Routine investigations were normal. CT brain was normal. Patient was monitored in HDU care. Antiepileptics was started in the form of Inj.Levitiracetam. Lumbar puncture was done which was normal. ANA was done in view of suspicion of autoimmune etiology, which was strongly positive(+++). ENA blot was sent in view of ANA positivity, antibody to Sm, antibody to SS-A, antibody to U1-RNP, antibody to Rib-po, antibody to ds-DNA, antibody to histones, antibody to nucleosomes, antibodies to DFS70-all positive. Patient was diagnosed to have lupus encephalitis and was started on pulse steroid therapy followed by maintanence steroid therapy. Patient's sensorium dint improve, hence she was started on intravenous immunoglobulin x 5 days. Patient's sensorium and general clinical condition improved after 5 days of IV immunoglobulin.

DISCUSSION:

SLE can involve CNS in early stages, can present as neuropsychiatric manifestations in 39-50% of patients(1). In our patient also, the presenting symptom was CNS manifestations like seizures and altered sensorium x one day. Such an acute presentation can also occur in SLE, but quite rare.

SLE can present as both mild and severe potential life threatening manifestations like stroke, myelitis, psychosis, seizures etc. It poses a diagnostic challenge as radiographic presentation may vary(2).

Usually, aseptic meningitis is associated with SLE(5). In our patient also, we ruled out infective etiology and vascular etiology as evidenced by aseptic meningitis and routine investigations. We thought of autoimmune encephalitis as it is re-emerging. Aseptic meningitis is also common in CNS lupus. Mechanism of injury is by immune mediated cell damage or antibody mediated tissue damage(7).

This patient didn't fit into ACR/EULAR criteria of SLE. She had malar rash and arthritis which she ignored. It's quite interesting that this patient developed lupus encephalitis even without fulfilling the criteria.

MRI BRAIN can exclude other neurological conditions, but can be normal in more than 50 % of the patients(3). Infections play a major part in mortality of SLE(4). Prevention of associated CNS infections prolongs life and decreases hospital stay.

Treatment should be aimed at reducing antibody levels(6). Generally, steroids, immunosuppressants, anti CD20 receptor antibody rituximab, iv immunoglobulins and plasma exchange are being used. Treatment response generally vary based on various factors. In our patient, early initiation of steroids and IVIG was crucial in the improvement of the patient. Patient is now currently on treatment with immunosup pressants (cyclophosphamide) and hydroxy chloroquine.

CONCLUSION:

Neuropsychiatric manifestations are quite common in SLE. Young females with reproductive age group presenting with neuropsychiatric manifestations should always warrant the clinical suspicion of autoimmune encephalitis. Early diagnosis and prompt treatment can prevent significant morbidity and mortality.

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