JIHS The Journal of Integrated Health Sciences

Available online at www.jihs.in

Case Report

Neuropsychiatric manifstation in patient having SLE

Lakhani JD[1], Santosh Kumar[2], Shah KP*[3], Rathod M[4], Shory N[5], Yadav L[6]

[1]Professor & HOD, [2]Assistant Professor, [3-6]Resident; Department of Medicine, SBKS MI & RC, Sumandeep Vidyapeeth, Piparia, Waghodia, Vadodara, Gujarat, India

ABSTRACT

A 30 year old woman residing at Khargaon, M.P., presented with complaints of abnormal behavior, decreased sleep, unable to perform routine activity and headache in psychiatry OPD of Dhiraj General Hospital. She was referred to medicine OPD for the complaints of fever and skin rash. On examination, she had terminal neck rigidity and brisk deep reflexes but normal plantar reflexes. On investigation she had pancytopenia with normal CT brain and CSF examination. ANA was positive. She had four positive criteria as per American college of Rheumatology Classification of SLE (updated in 1997) and thus diagnosis of SLE was made. On higher mental state examination she had poor affect, mood swings, delusions and poor judgment. Her orientation was normal. Memory and cognition appeared normal however it was difficult to test it as she did not co-operate. Patient was treated with steroid and immunosuppressive therapy and showed improvement. This suggests the importance of neurological and psychiatric examination in case of SLE.

Keywords: neuropsychiatric manifestation, SLE

INTRODUCTION

SLE is an autoimmune disease that predominantly affects women of child-bearing age. In the United States, SLE is more prevalent among African Americans, Hispanics, and Asians compared to Non-Hispanic Caucasians.[1] Neuropsychiatric lupus (NPSLE) is the least understood yet it is perhaps the most prevalent manifestation of lupus.[2] NPSLE is associated with increased morbidity and mortality.[3] In 1999, the American college of rheumatology (ACR) established case definitions for 19 specific neuropsychiatric syndromes, dividing them into two broad categories: central and peripheral as shown in Table 1.[4]

Case:

A 30 year old female residing at Khargaon M. P, presented in psychiatry OPD with the complaints of abnormal behavior, decreased sleep, unable to perform her routine activity and headache since seven days. Patient was admitted in psychiatry ward for cognitive disturbance and to rule out organic cause patient was referred to medicine deptt. Patient was she was transferred to medicine ward as she had skin rash and intermittent fever. On examination, temperature was 100.5^0 F, Pulse was 112/min, regular and BP was 116/70 mm Hg in supine position. On systemic examination respiratory, cardiovascular and cardiovascular examination were normal. On CNS examination patient was conscious and oriented to time, place and person, terminal neck rigidity was present with brisk deep tendon reflexes but Babinski's sign was negative. On MMSE (Minimal mental score examination) patient looked older than stated age, bald, wearing disheveled clothes, unkept with poor personal hygiene, lying on floor, maintaining one posture for long time, not initiating and maintaining eye to eye contact with the doctor. So, further MMSE could not be done as the patient couldn't communicate.

On investigation she was found to have pancytopenia (Hb: 6.7 gm%, TLC- 600/cu.mm, platelets - 36,000/cu mm. ANA was positive. CSF examination and CT brain was normal.

***Correspondence:**
E-mail: kunjanshah111@yahoo.co.in

Diagnosis:

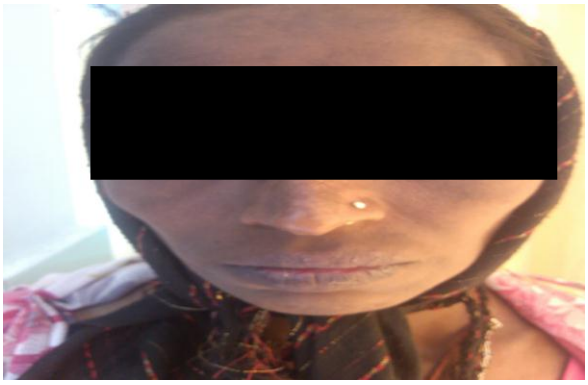
According to American college of Rheumatology 1999 patient was diagnosed to have NPSLE. She had four criteria out of 11. She was treated with glucocorticoids, oral antipsychotics, immunosuppressive therapy, blood transfusion and injectable antibiotics. After that patient showed response to the treatment and recovered

Table 1

Central nervous system
Aseptic meningitis
Cerebrovascular disease
Cognitive dysfunction
Headache
Movement disorder (chorea)
Seizures
Acute confusional state
Anxiety disorder
Mood disorder
Psychosis
Demyelinating syndrome
Myelopathy (transverse myelitis)
Peripheral nervous system
Autonomic disorder
Mononeuropathy
Cranial neuropathy
Plexopathy
Polyneuropathy
Acute inflammatory demyelinating polyradiculoneuropathy (guillain-barré syndrome)
Myasthenia gravis

DISCUSSION

In NPSLE, cognitive impairment is one of the most common manifestations with a varying prevalence of 15-66%.[5] It is estimated that 28% to 40% of adult NPSLE manifestations develop before or around the time of the diagnosis of SLE and 63% occur within the first year after diagnosis.[6] The pathogenesis of NPSLE is multifactorial and includes various inflammatory cytokines, autoantibodies and immune complexes resulting in vasculopathic, cytotoxic and autoantibody mediated neuronal injury. Disruption of the blood-brain barrier is integral to the neuropathology of SLE.[7]



In particular, it was mentioned that anti-nr2 glutamate receptor antibodies are seen in 25% to 30% of patients with SLE and may also play a role in cognitive dysfunction and psychiatric disease.[8] The importance of autoantibodies is still under active investigation and many of the observations are based only on association. So we have to be watchful for these symptoms and diagnose as early as possible.

CONCLUSION

The most prevalent neuropsychiatric manifestations of SLE are headache, mood disorders and cognitive dysfunction which may be associated with many other entities. Hence high degree of suspicion for SLE is required to identify and diagnose so that proper treatment can be started in time.

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