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Psychiatric manifestations of systemic lupus erythematosus: A brief review with two case-reports

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Abstract

Neuropsychiatric systemic lupus erythematosus is a severe neurological and psychiatric manifestation following systemic lupus erythematosus. Neuropsychiatric systemic lupus erythematosus is a global concern with limited data on its impact on quality of life in Africa. Furthermore, there is a lack of published research on neuropsychiatric systemic lupus erythematosus in Ethiopia. In this article, we present two case reports of Ethiopian patients with systemic lupus erythematosus and neuropsychiatric systemic lupus erythematosus, highlighting the challenges of diagnosing neuropsychiatric systemic lupus erythematosus worldwide. Although the patients were treated with alternative pharmacological agents based on available medications, interdisciplinary collaboration between psychologists, psychiatrists, neurologists, and internists is necessary to decrease the burden of systemic lupus erythematosus patients with neuropsychiatric manifestations. Overall, symptomatic therapy for neuropsychiatric systemic lupus erythematosus in developing countries is a good approach until future evidence-based pharmacotherapy is developed.

Keywords

Antipsychotics, antidepressants, neuropsychiatric lupus, systemic lupus erythematosus, psychosis, depressive disorder

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Introduction

Neuropsychiatric systemic lupus erythematosus (NPSLE) is a severe neuropsychiatric manifestation following systemic lupus erythematosus (SLE) in which other causes are excluded by careful examinations; see.[1] Two decades ago, the American College of Rheumatology developed case definitions for 19 neuropsychiatric syndromes observed in SLE. The 19 NPSLE syndromes have two essential components: 12 central nervous system (CNS) and 7 peripheral nervous system (PNS) manifestations. The CNS complications are aseptic meningitis, cerebrovascular disease, demyelinating syndrome, headache, chorea, myelopathy, seizure disorders, acute confusional state, cognitive dysfunction, mood disorder, and psychosis. The PNS complications are Gullian-Barre Syndrome, autonomic disorder, mononeuropathy, myasthenia gravis, cranial neuropathy, plexopathy, and polyneuropathy.[2]

Because of the variations in the study designs, the exact figure of neuropsychiatric manifestations in patients with SLE across the globe is not well known and the existing reports show wide ranges (between 12% and 95%).[3] However, around two-thirds of the NPSLE cases are secondary and are not directly linked to SLE.[4]

Several epidemiological studies have identified that being female is an important risk factor for autoimmune disorders in general[5] and for SLE in particular.[6] A population-based brief report has reported that SLE is the major cause of death among females of reproductive age in the USA.[7] A recent study in Nigeria also showed that SLE is highly predominant in African females, with a female to male ratio of 8:1.[8] Multiethnic and multicentre studies also found that NPSLE is more common with less favorable outcomes in non-Whites than in White populations.[7,9,10] Although SLE, manifestations of SLE, and mortality in patients with SLE are a global concern,[6,11] most studies pay little attention to developing

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countries.[12–14] There are limited data on SLE and its impact on the quality of life in Africa.[15,16] However, the existing evidence shows that SLE is not rare in Africa[17] and it has negatively impacted the quality of life among Africans, causing functional disability and exacerbating psychological burdens.[12] Furthermore, the psychiatric manifestations of SLE are often undiagnosed and untreated.[18] To the best of our knowledge, there is no previous published research on SLE or NPSLE in Ethiopia.

Therefore, the current study aimed to report the psychiatric manifestations of SLE in Ethiopian patients, the interventions taken, and the treatment outcomes for the psychiatric symptoms. NPSLE cases are commonly referred to the psychiatry clinics, from medical and or other departments, and the diagnosis is done by psychiatrists or psychiatry residents. However, there is a paucity of NPSLE cases being referred to the psychiatry clinic, and identifying them independently in the retrospective charts documented over the past few years was a challenge. First, the number of SLE cases presented to health facilities in low- and middle-income countries (LMICs) are low.[19] Second, SLE patients with NPSLE in health facilities of LMICs might not commonly visit psychiatry clinics. Then, to minimize the potential effects of treatment or selection bias,[20] physicians in the psychiatry department were randomly asked whether they had patients with NPSLE. We retrospectively selected the cases of three patients who completed their treatments at the psychiatry clinics, and the outcome assessor was blinded during the process. The charts were reviewed and revised independently, with written informed consent obtained for the two cases presented below.

Case presentations

Case 1

A 23-year-old female with a history of SLE presented to the psychiatric department with a chief complaint of worsening change of behavior over the last 3 months. The patient reported that she had been relatively mentally well until she started to have hair loss 1 year ago. The hair loss was also accompanied by palmar and malar rash, with major joint pain and swelling for 2 months, and dry cough and anorexia for 5 days. Lately, the hair loss induced stress, leading to feelings of sadness, anxiety, and difficulty sleeping. She started to isolate herself and to say words that were not fully understood by her family. She had also persecutory and referential delusions, with no hallucinations. She had a belief that people talk about her or try to hurt her, leading to physical aggression towards others and herself. She had psychomotor agitation. She mentioned that, because of her irresistible anxiousness, she tried to strangulate herself with a scarf. Her affect was blunt and she appeared acutely sick-looking during her visit to our department.

The patient reported that she was diagnosed with lupus nephritis and hypertension a month before her presentation to our clinic and was being treated with oral administrations of prednisolone (60 mg/daily), Furosemide (40 mg/daily), and enalapril (5 mg/daily). There was no history of low mood or loss of interest, and no reported history of excessively and abnormally elated, expansive or irritable mood, or increased energy. There was no history of problematic substance use or no family history of mental illness.

On physical examination, her blood pressure was 120/80, her pulse rate was 105, her temperature was 36°C and her saturation was 99%. Her conjunctiva showed a slight paleness. She had a puffy face and it had a butterfly-shaped lesion around the cheeks. She had a pan-systolic murmur. There was also a purpuric lesion on her palms. Pertinent laboratory investigation includes complete blood count (CBC) with a white blood cell count of 7220 WBCs per microliter, a hemoglobin level of 9.2 mg/dl (i.e., moderate anemia), mean corpuscular volume (MCV) of 80 fL, urine protein at+2, urine blood at+2, and 24-h urine protein of 1789 mg. She had an unremarkable renal function test. Her antinuclear antibody (ANA) was positive, with a high amount of double-stranded DNA (147). Fasting blood sugar and electrolytes were normal. She received an immunosuppressive treatment, prednisolone 40 mg po daily, chloroquine (200 mg dose), and mycophenolate mofetil (MMF) 1 gm daily.

Based on her medical history and objective findings, the patient was diagnosed with psychosis resulting from SLE. Then, she received oral risperidone (1 mg, nocturnal) but, after a month of treatment, she reported that her pictures were being posted on Facebook and she was a subject of media attention. She also reported that her mother is being isolated from the society because of her. The patient claimed to have heard voices that others could not and also believed that visitors who came to see her were spies. Due to these symptoms, the dosage of risperidone was increased to 2 mg. After a follow-up visit, the patient reported an improvement in sleep and a decrease in hallucinations but continued to experience suspiciousness. The dosage of risperidone was increased to 3 mg. Two weeks later, she reported that her sleep had improved and the hallucinations had subsided, but she felt sad most of the time. She was not interested in taking care of herself. She had difficulty in concentrating. She felt hopeless and had suicidal ideation. She also had a dysphoric effect. On this visit, she was diagnosed with psychosis with depressive disorder due to SLE and she started taking sertraline 50 mg per os (PO) in the morning and risperidone 3 mg. After 3 weeks on sertraline, her low mood improved, and she had good sleep and appetite. She no longer had suicidal ideation and her suspiciousness decreased.

On the next follow-up (a month later), she reported that her symptoms had resolved. She had no low mood and anxiousness. She had no delusions or hallucinations. Her affect was euthymic. After the resolution of the above psychotic symptoms, a renal biopsy was done and revealed proliferative

lupus nephritis. Cyclophosphamide 500 mg monthly was initiated and she received it for a total of 6 cycles. There were no emotional ailments or behavioral changes reported during this period. She was on antidepressants for 10 months and on antipsychotics for 1 year. Furthermore, these medications were continued till the last date of the follow-up.

Case 2

A 19-year-old female first-year college-admitted student. She was a known SLE patient for 1 year, having follow-ups at the medical outpatient department (OPD). She was admitted to a medical emergency a year ago, with a diagnosis of severe heart failure – New York Heart Association class IV Congestive heart failure. She later developed acute renal failure due to diffuse lupus nephritis and began treatment with daily oral administrations of prednisone 40 mg, MMF 500 mg PO daily, chloroquine 250 mg, and furosemide 40 mg. She had monthly follow-up appointments at the renal clinic. Throughout treatment, prednisone was tapered to 5 mg/day, while the MMF and chloroquine doses remained unchanged. Although the patient was distressed by the renal failure, she experienced a rapid response to treatment and was informed by the physician that she had a good prognosis.

However, 3 weeks after a neighbor patient's death (i.e., 2 weeks before presenting to the Department of Psychiatry), the patient refused to take the medications prescribed by the renal clinic, believing they would not be effective. She presented to the Department of Psychiatry with a complaint of problematic, disorganized behavior of 2 weeks' duration. She had been relatively well 6 weeks before this presentation. Her behavioral changes began with progressive social withdrawal, failure to respond to communication, and decreased interest in spending time with her family. She became irritable when asked about her strange behavior. Her mother explained that the behavioral changes began after the death of a 19-year-old male neighbor who was on dialysis for unspecified kidney disease. The patient and neighbor did not have a close relationship, but the patient assumed that they had similar illnesses.

Following the death news of patient's neighbor, the patient became worried about her illness and feared that she would also die from renal failure. Despite taking medications and receiving reassurance from her physicians, the fear persisted. After discontinuing her medication, the behavioral changes worsened for 2 weeks. She began shouting and yelling, claiming that "people are coming after her." Her presentation also included talking alone, saying words that did not make sense to the listener, refusing food and drink suspecting that her mother and sister would poison her, being restless and physically and verbally aggressive, having difficulty falling asleep, spending the night sitting, walking in the house and expressing a lack of reason to live. However, she had no suicidal thoughts or attempts. She also had no history of elated mood, increased goal-directed activity, or substance use.

She was uncooperative and was found to have persecutory delusion, and negativism. She displayed psychomotor agitation with adysphoric affect. She uttered a few words and shouted at times.

On physical examination, her blood pressure was 100/70 mmHg, pulse rate was 108 beats/min, respiratory rate 22/min and she had a normal body temperature.

Pertinent laboratory investigations include a CBC (WBC = 3900, hemoglobin = 12.9, MCV = 87.3 and Platelet = 225,000), a urine analysis (blood = +3, protein = +3, and 24-h urine protein = 980 mg/24 h), an echo at presentation (moderate left ventricular systolic dysfunction, and moderate pericardial effusion), a positive ANA, creatinine = 1.07, urea = 43.9, serum albumin = 3.5 g/dl, triglyceride = 238, total cholesterol = 172.8, HDL (high-density lipoprotein) = 18.1, LDL (low-density lipoprotein) = 109.3, and normal serum electrolytes, and a kidney biopsy showing diffuse lupus nephritis, class 4.

She was admitted to the psychiatry ward with a diagnosis of psychosis due to SLE with comorbid major depressive disorder (MDD) with a moderate risk of suicide and aggression plus severe functional impairment and lupus nephritis. She received oral olanzapine (5 mg, nocturnal) and her previous medications were also reinitiated. However, she remained suspicious of her family even after a week of olanzapine, and refused to communicate with her physicians due to her persecutory delusion. Olanzapine was escalated to 10 mg but she had a prominent low mood and suicidal ideation at the end of the second week. Olanzapine was then escalated to 15 mg nocturnal, and oral fluoxetine 20 mg (morning) was started. After 3 weeks of admission, her sleep and appetite had improved, but her suspiciousness and suicidal ideation persisted. Then, olanzapine was escalated to 10 mg twice daily (BID), and fluoxetine was escalated to 40 mg morning. Oral lorazepam 1 mg BID was added. At the end of the fourth week, she developed rigidity, masked face, hand tremors, and mild drooling side effects. During the fifth week of treatment, the olanzapine dosage was tapered to 10 mg and oral haloperidol was initiated at a dose of 1.5 mg/day that was gradually escalated to 5 mg BID. Lorazepam was tapered down to 1 mg. By the eighth week, she was taking haloperidol 7.5 mg nocturnal and oral propranolol 20 mg/day. Oral Trihexyphenidy 2 mg/day was also introduced due to the extrapyramidal side effects.

The patient was discharged after 10 weeks, and was prescribed oral diazepam 2.5 mg nocturnal, which was to be discontinued after a week. She was also taking fluoxetine 40 mg/day, and Trihexyphenidy 5 mg/day. She continued to have follow-ups at the psychiatry OPD and renal clinic and showed improvement in her depressive and psychotic symptoms. Five months after discharge, in the subsequent year, the patient reported that she had started going to school to earn her degree. She is still on follow-up on fluoxetine treatment.

Discussion

We presented the psychiatric manifestations of two patients with SLE. The diagnosis of NPSLE has been a challenge because of several confounding factors,[4,21] such as nonspecific symptoms, few specific examination methods, presence of the symptoms in the general population, presence of symptoms in other comorbid conditions, adverse effects of SLE medications, simple co-occurrences of neuropsychiatric disorders with SLE and psychological burdens of having any severe illness.[3] Likewise, in our current cases, related concerns and challenges regarding the diagnosis and treatment of NPSLE were observed. Importantly, patients' awareness, disease characteristics, and the country's healthcare system pose significant challenges in the management of NPSLE in Ethiopia. Firstly, patients with SLE often do not seek medical attention at the onset of symptoms, which leads to delayed diagnosis and treatment. Secondly, healthcare professionals lack advanced knowledge of the diagnosis and treatment of SLE and NPSLE.[1,18] Lastly, the economic conditions of the country restrict access to first-line medication for SLE and NPSLE, resulting in reliance on alternative psychopharmacological agents.

It has been suggested that the low prevalence of SLE in Africa[22] might not represent the actual figure as there is the possibility of poor reporting or under-diagnosis due to poor health access.[13–15,17] Importantly, SLE complications and physical disabilities following SLE are associated with poor financial and psychosocial support,[23–25] indicating that the poor health access in Africa might worsen the suffering of patients with SLE. In addition, the lack of full-description of SLE (and its manifestations) and population prevalence is a challenge in Africa.[26] Psychiatric manifestations are found to be important determinants of mortality among patients with SLE in Africa.[13,26] For example, a recent meta-analysis showed that SLE is predominant in female natives of sub-Saharan Africa, with a pooled prevalence of 1.7%, and morbidity was commonly associated with infections, renal diseases and NPSLE.[26]

The current two patients had moderate to severe forms of psychiatric manifestations (depressive disorder and/or psychosis), which are consistent with previous reports that showed that mood disorders and psychosis are two of the five most common NPSLE.[18,27] Further studies also showed that SLE activity is strongly associated with MDD, and patients with active SLE have a chance of developing MDD or depressive symptoms, which can also explain the current NPSLE in the two patients.[25] However, it has to be noted that although NPSLE commonly occurs following SLE, mild forms of depression, anxiety, or cognitive impairments, and headache could be also common in non-SLE patients[4] or psychiatric symptoms might occur prior to the onset of SLE diagnosis.[27] Studies showed that a point or lifetime prevalence of psychiatric manifestations, particularly mood disorders,[28] following SLE is very high (around 40%–50%).[18,29,30] While psychosis has a relatively low prevalence (ranging from 1% to 3%), it is important to note that cognitive impairment is much more prevalent, affecting up to 80% of individuals with SLE.[18,30] In particular, patients taking corticosteroids must be closely monitored for signs of psychosis, as it may exacerbate or contribute to psychosis development.[18]

The majority of NPSLE in patients with SLE are not primarily attributed to SLE, although the "primary NPSLE" have more encouraging outcomes than the "secondary NPSLE".[29,31] The current patients had received different treatment combinations based on the patients' conditions, the time of onset of the psychiatric manifestations, the availability of the medications, and the prescribers' preference, as pharmacotherapy of SLE in Africa is limited by the availability and affordability of medications.[15] However, regardless of the medication limitations, treatment with antipsychotics or antidepressants have been found to be effective in reducing mortality and suffering from psychiatric complications.[32] Furthermore, the individual patient factors play a key role in the selection of medications and management of SLE complications, as the clinical manifestations of the diseases are diverse.[33]

Overall, based on the type of manifestations and the degree of the symptoms, individualized pharmacotherapeutic approaches have been recommended.[34] Pharmacotherapeutic management of NPSLE has two important strategies: treatment of the underlying condition or aggravating factors such as medication adverse reactions, metabolic disturbances, infection, or hypertension; and symptomatic management. Symptomatic management depends on the type of neuropsychiatric manifestations, while treatment of the triggering or aggravating factors includes immunosuppressive therapy and anticoagulation/antiplatelet therapy based on the feasibility.[3,4] However, treating the underlying condition should be the primary step.[4] In the present cases, the patients' underlying SLE condition or aggravating factors (such as hypertension) were managed with immunosuppressive agents, corticosteroids, antimalarial drugs or antihypertensive agents. It has to be noted that symptomatic therapy of NPSLE in developing countries is a good approach until future evidence-based SLE pharmacotherapy is developed.

Conclusion

Even though SLE is not uncommon in developing countries, the magnitude assessment and management of NPSLE have received poor attention. To decrease the suffering and burden of SLE patients due to the neuropsychiatric manifestations, the interdisciplinary collaborations of psychologists, psychiatrists, neurologists, and internists are indispensable. Until future evidence-based pharmacotherapy of NPSLE develops, empirical therapy based on the available medications is a good approach in developing countries.

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Authors' contribution

F.K. and D.W. contributed to the conceptualization. F.K. searched the literature and wrote the first draft of the manuscript. M.B., D.W., S.M., and S.A. reviewed the chart of the patients and revised the manuscript. S.M. clerked the first patient and D.W. clerked the second patient. All authors sufficiently contributed to the case report preparation and reviewed the manuscript draft. All authors read and approved the final manuscript.

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Ethics approval

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Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article. The consent was obtained after psychotic symptoms resolved and the patients had decisional capacity to provide the written consent. A copy of the written consent is available with authors and will be provided upon request from Editors.

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