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Acute Manic Episode and Psychosis as Early Manifestations of Systemic Lupus Erythematosus with Lupus Nephritis: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Author Oluwafunbi Opadola conceptualized the idea for the case report while author BS wrote the first manuscript draft. Authors MI, SO and Olaitan Oladele were involved in extracting medical information from the record archives and other investigatory reports from various records. Authors SA and AA edited and formatted the manuscript for final consideration. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Background: Neurological and psychiatric conditions seen in SLE in the absence of other causes are broadly termed Neuropsychiatric SLE (NPSLE).

Case Presentation: This case report was of a 37-year-old, separated female Banker who presented initially with symptoms of mania with psychosis at the Psychiatry Department. She was initially managed as such but represented again with similar symptoms in addition to fluffy brownish hair with hair loss, and hyper-pigmented patches along the bridge of her nose, glabella, and zygomatic regions, extending to the ears and retro auricular region. Annular hypo-pigmentation, atrophic patches were also observed on the extensor surface of both forearms. She developed fever, and epigastric tenderness with several episodes of vomiting and diarrhea which were initially non-bloody at 48hrs into admission on account of which she was placed on antibiotics. The patient improved significantly after which she discharged herself against medical advice however represented 2 days after with a worsened condition. Proactive management was ensured with some sessions of hemodialysis. An assessment of acute flare of systemic lupus erythematosus SLE, precipitated by sepsis was made and was managed as such. The patient recovered tremendously and was discharged to the clinic.

Conclusion: The need for comprehensive examinations and investigations in this regard to exclude other differentials cannot be jettisoned. In this case, it could be said that the psychiatric symptoms came before the obvious signs of SLE, however, it is still possible to think of these symptoms as early signs of SLE or SLE aggravating an underlying mental disorder.

Keywords: Manic episode; psychosis; systemic lupus erythematosus; lupus nephritis.

1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that results in an inflammatory process in multiple organ systems. This is mediated by the interplay of genetic susceptibility, and environmental, and immunological factors, leading to the production of autoantibodies against specific antigens and the deposition of immune complexes causing damage to organs and tissues. The incidence and prevalence are highest in people of black ethnicity and more prevalent in women than men across all age groups and populations [1].

In about 50% SLE cases, there is kidney involvement. Lupus nephritis (LN) is more prevalent among blacks, develops early, and runs an aggressive course with a worse outcome compared to other populations [2,3,4]. It accounts for a major risk factor for morbidity and mortality. In the face of highly effective therapies against the inflammatory and immunological processes, most cases of LN end in chronic kidney disease (CKD) [2].

Neuropsychiatric manifestation in SLE portends a poor prognosis [5]. Neurological and psychiatric conditions seen in SLE in the absence of other causes are broadly termed Neuropsychiatric SLE (NPSLE) [6]. The incidence ranges from 12.2% to 94.7% [5]. Its presentations include headache, seizure, delirium, psychosis, mood disorder, cognitive dysfunction, stroke, movement disorder, peripheral neuropathies, myelitis, or meningitis with 20% prevalence for a mood disorder [7,8].

This case report involved a patient who presented initially with symptoms of mania with psychosis and was later diagnosed as SLE with LN at the Department of Psychiatry, LAUTECH Teaching Hospital Ogbomoso Oyo State Nigeria.

2. CASE REPORT

The patient was a 37-year-old, separated female Banker who presented to our facility in the year 2020 on account of undue irritability, talkativeness, decreased need for sleep, undue familiarity, and belief that her family members were conspiring against her. These started two months prior presentation and had progressively worsened. She was a known patient with peptic ulcer disease but had no past psychiatric history. No family history of mental illness. She denied the use of alcohol or other psychoactive substances.

A mental state examination revealed an appropriately dressed and groomed lady, irritable with increased volume and tone of speech, flight

of ideas, persecutory delusion, intact cognition, and partial insight. The general physical and systemic examinations were not remarkable. A diagnosis of Acute Manic episode with psychotic symptoms was made. She declined admission and promised to present the following week. However, she was placed on oral olanzapine tablet 5mg and oral nitrazepam 5mg nocte (for 5 days). The baseline investigations were essentially normal.

Six weeks later, she presented again with similar symptoms. She was said to have stopped the prescribed medication from her last visit. She was still irritated, speech was increased in volume and tone with racing thoughts, persecutory delusion, and lack of insight. Additionally, she had fluffy brownish hair on her scalp with some areas of hair loss (Image a). There were hyper-pigmented patches distributed along the bridge of the nose, glabella, and zygomatic regions, extending to the ears and retro auricular region (Image b). Annular hypo-pigmented, atrophic patches were also observed on the extensor surface of both forearms. Systemic Lupus Erythematosus (SLE) was suspected by the Dermatologist who was consulted to review on the Psychiatry ward. She was recommenced on oral Olanzapine tablet 5mg nocte and sedated with IV diazepam 20mg slowly and IM haloperidol 5mg stat.

Two days into admission, she developed fever, vomiting (several episodes), diarrhea (2 episodes), and epigastric pain. Vomitus contains recently ingested food, non-bilious, and not blood-stained. The stool was loose, non-mucoid, and not blood-stained. No abdominal swelling. There was tenderness on the epigastrium and right renal angle. The pulse rate was 96b/m, full volume and regular, temperature 39^0C, and blood pressure was 104/68mmHg. Immediately, she was transferred to the Accident and Emergency section.

The patient was commenced on antibiotics (IV ciprofloxacin 200mg 12 hourly and IV metronidazole 500mg 8 hourly), IV omeprazole 20mg 12 hourly, IV metoclopramide 10mg stat, intravenous fluid 0.9% normal saline alternating with 5% dextrose saline 1L 8 hourly) and strict fluid input/output and vital signs monitoring were instituted. The following investigations were requested: Full blood count (FBC), electrolytes, urea, creatinine (E.U.Cr), blood film for malaria parasite (MP), abdominopelvic scan, urinalysis, and urine microscopy, culture and sensitivity (m/c/s). The E.U.Cr was not remarkable. MP was negative, PCV, 26% with neutrophilia and lymphopenia. Abdominopelvic scan showed bilateral grade II renal parenchyma disease, mild-moderate ascites, bi-basal pleural effusion and mild hepatomegaly. Urinalysis was normal, while urine m/c/s was not done.

There was a significant resolution in symptoms until 72 hours later when the patient suddenly developed a surge in the number of episodes of diarrhea, stool became blood-stained and there was an episode of breathlessness, lasting 10 minutes. Findings revealed a patient in respiratory distress, dehydrated, pigmented patch and ulcers on the lips, PR 85b/m, regular, small volume, and BP 113/83mmHg. On the chest, RR was 26c/m, SPO_2 98%, percussion note was dull on the right and left lung zones, reduced breath sound in the right lower long zone laterally with fine crepitations. An assessment of acute flare of SLE, precipitated by sepsis? lung focus was made. Urgent chest X-ray, erythrocyte sedimentation rate (ESR), E.U.Cr., anti-nuclei antibody (ANA), retroviral screening (RVS), hepatitis $(HB(S)_{Ag}$ and anti-HCV), liver function test (LFT) and urinalysis were requested. Chest X-ray could not be done immediately due to logistic, however, other results are as shown in Table 1. The patient was adequately rehydrated and monitored, intravenous steroid was administered for 3 days while the antibiotics were maintained. She was also considered for hemodialysis.

After gaining some significant improvement, she decided to be discharged against medical advice. All efforts to counsel her against this decision proved abortive. She left but was brought back to the emergency room after 72 hours with a deteriorating clinical state. There was facial puffiness, whitish patches on the tongue extending to the throat, and bilateral pedal edema up to the mid-leg. Vital signs were within the normal range. She was recommenced on intravenous antibiotics (Rocephin 1g 12 hourly and Flagyl 500mg 8 hourly), lasix 20mg 12 hourly, omeprazole 20mg 12 hourly, methylprednisolone 500mg daily (as an infusion with 250mls N/S over 1 hour, cap selenium 300mg daily, IVF 0.9% normal saline alternating with 5%D/S 500mls 8 hourly and high protein diet.

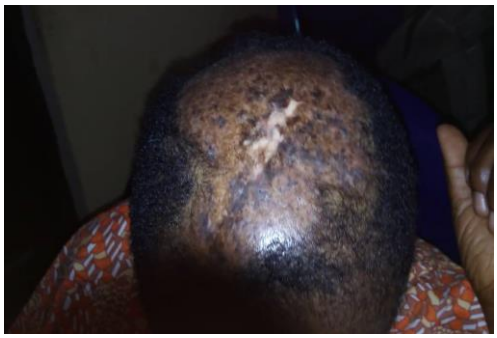

Image a. Fluffy brownish hair on with areas of hair loss scalp

Image b. Hyper-pigmented patches distributed on ears and retro auricular region

Table 1. Laboratory investigations results

SN	Investigations	Results
1.	Packed cell volume	21%
2.	White blood cells	8,900/cmm
3.	Neutrophil count	83%
4.	Eosinophil	05%
5.	Lymphocyte	08%
6.	Monocyte	04%
7.	Serum urea	24mmol/l
8.	Serum creatinine	792umol/l
9.	Bicarbonate	12mmol/l
10.	Sodium	114mmol/l
11.	Urine	protein^{+++}blood^{+++}
12.	Erythrocyte Sedimentation Rate	72mm/hr westergreen
13.	Serum protein	67g/l
14.	Serum albumin	27g/l
15.	Bilirubin(total)	10umol/l
16.	Bilirubin(conjugated)	06umol/l
17.	Aspartate Transaminase	10umol/l
18.	Alanine Transaminase	02umol/l
19.	Alkaline phosphatase	23
20.	Anti-Nuclear Antibodies	1:320
21.	RVS	non-reactive.
22.	$HB(S)_{Ag}$	negative
23.	Anti-HCV	negative

RVS: retroviral screening; $HB(S)_{Ag}$: hepatitis B surface antigen; Anti-HCV: antibodies against hepatitis c virus

The fever was not subsiding despite regular intravenous antibiotics. Therefore, urgent urea, creatinine, urine m/c/s, blood culture and renal biopsy were ordered. Urea and creatinine levels came out to be 30.1mmol/L and 934umol/L respectively. Urine m/c/s showed yellow and turbid urine, numerous white blood cells, red cell count of 1-2hpf, epithelial cells^{++}, and white-cellular cast^{++} while culture yielded growth of Escherichia coli which was sensitive to Nitrofurantoin. Blood culture yielded profuse growth of Pseudomonas aeruginosa, sensitive to ceftazidime and ciprofloxacin. Additional investigations such as echocardiography, pleural and ascitic fluid analysis were not done because of financial burden on the patient and family.

Renal biopsy result report: section shows renal tissue composed of three glomeruli with the expansion of mesangial matrix and proliferation of mesangial cells. There was endocapillary obliteration of lumens. There were lymphocytic infiltrates. There were focal areas of fibrinoid necrosis. There was tubular atrophy and interstitial fibrosis—features in keeping with Diffuse Lupus Nephritis (class IV).

A diagnosis of Systemic Lupus Erythematosus with Lupus nephritis and neuropsychiatric manifestation was made. Having established this, the patient was commenced on Tabs Hydroxychloroquine 200mg b.d, tabs prednisolone 40mg daily, tabs MMF 1g b.d, tabs

rabeprazole 20mg b.d, tab frusemide 20mg daily, and other drugs as needed. Antibiotic treatment was adjusted based on blood culture and urine m/c/s results. She was transfused a couple of times and had 6 sessions of hemodialysis.

She was discharged after 2 months, although, defaulted nephrology follow-up clinic, only to appear 9 months later with features suggestive of urinary tract infection which was treated appropriately. Subsequently, she attended 2 consecutive follow-up clinics and was stable clinically. She is currently stable on medications while still attending a nephrology clinic at a Teaching Hospital in another state where she works.

3. DISCUSSION

The American College of Rheumatologists (ACR) expert committee identified 19 neuropsychiatric manifestations known as 'case definitions' in a patient with NPSLE which includes 12 central nervous system (CNS) and 7 peripheral nervous system (PNS) manifestations [6]. Despite this effort, some studies did not find it effective in distinguishing NPSLE patients from those with neuropsychiatric conditions due to other causes. Other studies however reported that NPSLE could be among the earliest presentations in 40% of patients with SLE within the first year of diagnosis [9].

NPSLE may stem from the disease process or as a result of treatment, especially with steroids, or by aggravating an underlying mental health condition [10]. The use of steroids in SLE can induce psychosis in 4.8% of patients however it is difficult to differentiate lupus psychosis from steroid-induced psychosis [11,12]. Plausible pathogenesis of NPSLE includes disruption of the blood-brain barrier, autoantibodies, and cytokine-mediated neuronal damage, production of phospholipid proteins, and inflammatory markers [13].

According to the 2019 European League Against Rheumatism/American College of Rheumatology Classification Criteria for SLE (EULAR/ACR), our patient met the diagnosis of SLE [14]. There was fever, oral ulcers, skin manifestations, pleural effusion, psychosis, ANA titer 1:320, and diffused lupus nephritis class IV. However, the anti-double-stranded DNA (Anti-dsDNA), Anti-Smith antibody, antiphospholipid antibodies and complement proteins (C3 and C4) were not done.

Serum Anti-dsDNA antibodies are said to be strongly associated with SLE and lupus nephritis, having a specificity of 96% and sensitivity (52-70%) [15]. It has been reported that cerebrospinal fluid (CSF) antiphospholipid antibodies such as lupus anticoagulant and anti-cardiolipin are well represented in SLE, especially in NPSLE [16]. Although all these were not done for financial reasons, ANA has been reported as an essential biomarker for screening, classification, diagnosis, prognosis and staging of the disease with a sensitivity of 90-95% [17].

Here, the patient presented with symptoms of acute mania with psychosis which had been on for two months before presentation. Considering the temporal sequence, it may not be wrong to say that this patient had an underlying mental disorder before the diagnosis of SLE. Also, it could be said that both conditions co-existed. At the same time, although the neuropsychiatric manifestation appeared first, the possibility of a behavioural change being part of the earliest signs in the spectrum of SLE or other organic factors cannot be completely excluded. However, cases presenting initially with neuropsychiatric symptoms which later became SLE have also been reported [18,19,20].

While it has been reported that female gender, high steroid use, and lupus nephritis are risk factors for steroid-induced psychosis [21], our patient is also a female, had lupus nephritis class IV but had no previous history of steroid use at presentation. However, she was noticed to have an exacerbation of psychotic symptoms when steroid was instituted and this necessitated the adjustment of Tabs olanzapine dosage to 10mg nocte. The subsequent increase in steroid dose did not in any way aggravate the neuropsychiatry symptoms.

We understand that some investigations such as neuroimaging, serum markers, CSF studies, and others which are important in the diagnosis and management of this case were not done either due to financial constraints or a dearth of facilities. Nevertheless, it was ensured that the collaborative effort of the Physicians, dermatologists, nephrologists, pulmonologists, and neuropsychiatrists was well coordinated.

4. CONCLUSION

Psychiatrists need a high index of suspicion of an organic template whenever patients present with

behavioural problems even with subtle physical signs. The need for proper examinations and investigations in this regard to exclude other differentials cannot be jettisoned. In this case, it could be said that the psychiatric symptoms came before the obvious signs of SLE, however, it is still possible to think of these symptoms as early signs of SLE or SLE aggravating an underlying mental disorder. An effective collaboration among specialists is pivotal to the management of patients with similar presentations.

CONSENT

Written informed consent was obtained from the patient for the publication of this case report and accompanying data.

ETHICAL APPROVAL

Approval was obtained from the ethical review committee of the LAUTECH Teaching Hospital, Ogbomoso, Nigeria.

AVAILABILITY OF DATA AND MATERIALS

Data and materials used for the study are available in the patient case note folder in the hospital record department. Since the institution still utilizes local or non-digitalized data storage systems, patient information can only be accessed from the medical record data.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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