International Journal of Clinical Science and Medical Research
ISSN(print): 2770-5803, ISSN(online): 2770-582X
Volume 05 Issue 05 May 2025
DOI: https://doi.org/10.55677/IJCSMR/V5I5-09/2025, Impact Factor: 8.005
Page No : 132-133

Olanzapine-Induced Systemic Lupus Erythematosus: A Case Report

B. Raouf[1], I. Adali[2], F. Manoudi[3]
[1,2,3]Ibn Nafis University Psychiatric Hospital of Marrakech
Faculty of Medicine and Pharmacy of Marrakech, Morocco

ABSTRACT

Olanzapine is a widely prescribed atypical antipsychotic, generally considered safe regarding hematological adverse effects compared to other antipsychotics. We report the case of a 19-year-old female treated for depression with fluoxetine 20 mg, who developed a clinical and biological picture suggestive of drug-induced systemic lupus erythematosus (SLE) following the introduction of olanzapine 5 mg for delusional symptoms. Thirty days after starting olanzapine, the patient presented with prolonged fever, fatigue, anorexia, polyarthralgia, and a scaly skin rash. Laboratory tests revealed anemia (hemoglobin 9 g/dL), severe leukopenia (1000/mm³), and positive antinuclear antibodies, while other specific antibodies remained negative. This case highlights the importance of considering drug-induced SLE in the presence of systemic manifestations in patients treated with olanzapine, even in the absence of classic specific SLE antibodies.

Published Online : May 31, 2025

KEYWORDS:
Olanzapine, drug-induced lupus, atypical antipsychotics, adverse effects, leukopenia

I- INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease that can be triggered by various environmental factors, including certain medications. Drug-induced lupus (DIL) accounts for approximately 10% of SLE cases, with over 80 drugs implicated, mainly anticonvulsants, antihypertensives, and some antibiotics.

Atypical antipsychotics are rarely associated with the development of SLE, unlike other psychotropic drugs. Olanzapine, a second-generation antipsychotic that antagonizes dopaminergic D2 and serotonergic 5-HT2 receptors, is generally considered to have a favorable safety profile regarding hematological and autoimmune complications compared to clozapine.

We present a rare case of SLE likely induced by olanzapine in a young patient initially treated for depression and subsequently for psychotic symptoms. This observation highlights an underreported adverse effect that deserves the attention of clinicians prescribing atypical antipsychotics.

Corresponding Author: B. Raouf

Cite this Article : B. Raouf, I. Adali, F. Manoudi (2025). Olanzapine-Induced Systemic Lupus Erythematosus: A Case Report. International Journal of Clinical Science and Medical Research, 5(5), 132-133

II- CASE REPORT

A 19-year-old female was followed at Mohamed VI University Hospital (CHU Marrakech) for a major depressive episode evolving over two years. She was treated with fluoxetine 20 mg/day with favorable clinical evolution. Her personal and family history was unremarkable, with no history of autoimmune disease.

The recent onset of psychotic symptoms, specifically delusional ideas, led to the introduction of olanzapine at a dose of 5 mg/day. Thirty days after starting this treatment, the patient presented with progressively worsening systemic symptoms: prolonged fever (38.5°C), debilitating fatigue, anorexia, polyarthralgia mainly affecting the wrists, knees, and interphalangeal joints, and a scaly skin rash predominantly on the face and chest. The family also reported persistence of delusional symptoms despite treatment.

Clinical examination confirmed the presence of a malar erythematous-squamous rash, tenderness of the affected joints without frank synovitis, and mild splenomegaly. The temperature was 38.2°C.

III- LABORATORY FINDINGS

Laboratory tests revealed:

- Hemoglobin: 9 g/dL (normochromic normocytic anemia)
- Leukocytes: 1000/mm³ (severe leukopenia)
- Platelets: 180,000/mm³ (normal)

- Erythrocyte sedimentation rate: 42 mm/h (elevated)
- C-reactive protein: 22 mg/L (elevated)
- Renal and hepatic function: normal
- Antinuclear antibodies (ANA): positive at a titer of 1/320, speckled pattern
- Anti-native DNA antibodies: negative
- Anti-Sm antibodies: negative
- Antiphospholipid antibodies: negative
- Complement CH50: normal

IV- MANAGEMENT AND OUTCOME

Given the clinical and biological picture suggestive of SLE possibly induced by olanzapine, the medication was immediately discontinued. Moderate-dose corticosteroid therapy (prednisolone 0.5 mg/kg/day) was initiated, along with hydroxychloroquine (200 mg twice daily).

The outcome was favorable, with resolution of fever within 48 hours, progressive improvement of arthralgia and skin rash over two weeks. One-month follow-up labs showed normalization of leukocyte count (4500/mm³) and improvement in hemoglobin (10.5 g/dL).

For management of psychotic symptoms, risperidone was cautiously introduced at a low dose, with no recurrence of lupus signs or symptoms after six months of follow-up.

V- DISCUSSION

Our case illustrates a probable instance of SLE induced by olanzapine in a young woman initially treated for depression. Several arguments support this hypothesis: the chronology of events, with symptom onset 30 days after olanzapine introduction; the typical clinical and biological presentation with fever, arthralgia, skin rash, anemia, leukopenia, and positive ANA; and the resolution after discontinuation of the suspected medication.

The diagnosis of drug-induced lupus (DIL) is based on several criteria: absence of prior SLE before taking the implicated medication; presence of at least one clinical sign of SLE; presence of serological markers (typically ANA); and regression of manifestations after drug withdrawal.

The severe leukopenia observed in our patient (1000/mm³) is particularly notable. Hematological side effects of olanzapine, though described, are generally considered rare and less frequent than with clozapine. In the literature, a few cases of olanzapine-induced agranulocytosis have been reported in patients with pre-existing SLE, suggesting a particular susceptibility in these patients to the hematological effects of this drug.

Our case presents certain peculiarities: negativity for anti-native DNA, anti-Sm, and antiphospholipid antibodies despite positive ANA. This presentation is compatible with DIL, which generally shows positive anti-histone antibodies (not tested in our case), while anti-native DNA antibodies are more often negative, unlike idiopathic SLE.

The mechanism by which olanzapine may induce SLE remains hypothetical. Some drugs can bind to nuclear proteins and become immunogenic. Others may alter tolerance to autoantigens or interfere with DNA methylation. Drug metabolism by acetylation is also implicated, with slow acetylators being more susceptible to developing DIL.

The treatment of DIL is primarily based on discontinuation of the implicated drug, along with nonsteroidal anti-inflammatory drugs or corticosteroids depending on the severity of manifestations. Hydroxychloroquine is also used for its immunomodulatory effect. In our case, the severity of leukopenia and symptom intensity justified moderate-dose corticosteroid therapy.

VI- CONCLUSION

This case suggests that olanzapine can, in rare instances, induce SLE with significant clinical and biological manifestations. Psychiatrists and physicians prescribing olanzapine should be aware of this potential complication and maintain a high index of suspicion when systemic signs such as unexplained fever, arthralgia, or skin manifestations appear in their patients.

A complete blood count and ANA testing should be considered in the presence of such symptoms. Further studies are needed to better characterize the link between olanzapine and drug-induced SLE, and to identify potential predisposing factors.

REFERENCES

1. Yaseen K. Systemic lupus erythematosus. MSD Manuals Professional Edition. 2024.
2. Chang C, Gershwin ME. Drug-induced lupus erythematosus: incidence, management and prevention. Drug Saf. 2023;34(5):357-374.
3. Cleynen I, Goyvaerts A, De Rijcke L, et al. Side effects of antipsychotic medications: hematologic effects and drug-induced autoimmunity. CNS Drugs. 2022;36(8):673-688.
4. Sarkar P, Aga P, Banerjee A. The Association of Psychiatric Medications and Systemic Lupus Erythematosus: A Comprehensive Review. Cureus. 2024;13(2):e13572.
5. Antonov D, Kazandjieva J, Etugov D, Gospodinov D, Tsankov N. Drug-induced lupus erythematosus. Clin Dermatol. 2022;30(2):195-201.
6. Jain R, Thakkar N, Feldman G. Olanzapine-induced agranulocytosis in systemic lupus erythematosus. Pharmacotherapy. 2006;26(10):1511-1513.