I Felt a Funeral in my Brain: An Unusual Presentation of Psychosis with Cotard's Delusion due to Lupus Cerebritis and Glucocorticoid Use complicated by Catatonia

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Introduction

- Neuropsychiatric manifestations of systemic lupus erythematosus (NPSLE) have been well-documented in the literature (Carrión-Barberà, 2021).
- This case of psychosis with Cotard's delusion as a neuropsychiatric manifestation of SLE and possible steroid-induced effect followed by onset of catatonia contributes to the small body of literature about this unique pattern of symptoms in SLE.

Case Report

Initial History:

- A 30-year-old female with PMHx of SLE presented to the ED after being found at home unresponsive and in respiratory distress. The patient had a 1-2 month history of paranoid delusions and hallucinations prior to admission.
- SLE had been diagnosed approximately one month before admission. The patient had been taking prednisone 10 mg daily since that time.

Hospital Course:

- **In the ED,** the patient was febrile, tachycardiac, tachypneic, and hypoxic, with SpO2 63% on room air.
- Admitted to ICU with with acute hypoxic respiratory failure, sepsis secondary to unclear etiology, and acute metabolic encephalopathy.
- **In the ICU,** the patient had further cognitive decline and hypoxia, requiring intubation. On extensive medical workup, patient's presentation and findings appeared consistent with **encephalopathy due to lupus cerebritis**.

- **Notable Labs and Imaging:**

1. MRI: Mildly increased signal on the diffusion-weighted images in the medial left frontal cortex and left insular cortex, with equivocal findings elsewhere.

2. Positive anti-RNP antibody with a titer of over 8 and a positive **double-stranded DNA antibody** with a titer of over 300.

- **Medications:**

1. IV methylprednisolone was administered for 3 days before transitioning to IV hydrocortisone.

2. Hydroxychloroquine and cyclophosphamide were initiated.

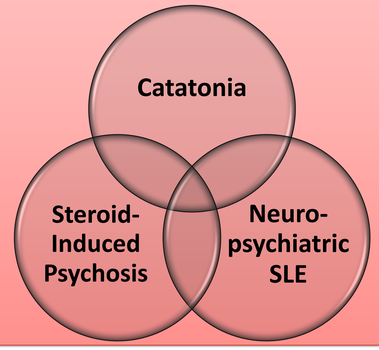
- After stabilization and transfer to the medical floor, the patient refused to eat or swallow, and a nasogastric tube was placed.

Key Points

•The differentiation of neuropsychiatric manifestations of systemic lupus erythematosus (NPSLE) from steroid-induced psychosis often depends on the timeline of symptoms and treatment; however, when the onset of symptoms of NPSLE overlaps with the administration of steroids for acute treatment, distinguishing these two entities poses a diagnostic challenge.

•In this case, there was a unique pattern of neuropsychiatric symptoms. Catatonic symptoms emerged after treatment with and discontinuation of antipsychotics and resolved after zolpidem challenge and clonazepam treatment.

•After the resolution of catatonia, the persistence of paranoia and Cotard's delusion in this case was likely due to the multifactorial effect of continued steroid treatment as well as symptoms of NPSLE.



Case Report, continued:

Psychiatry Consult on day 9 of hospitalization.

Reason for consult: Patient continued to exhibit **paranoia and refusal to eat**.

During initial evaluation, the patient endorsed unspecified auditory and visual hallucinations and Cotard's delusion, believing that she had died and was missing organs. She endorsed paranoid delusions that she was being poisoned. Past psychiatric history included ADHD; the patient had not recently taken psychotropic medications.

Assessment: The patient's presentation appeared consistent with **delirium secondary to multifactorial etiology, including lupus cerebritis (NPSLE) and corticosteroid use**.

Management: Steroids were continued and olanzapine 2.5 mg po bid was started and titrated to 7.5 mg BID, 2 days later. The patient developed an acute dystonic reaction with torticollis. QTc increased to 482 ms. Antipsychotics were discontinued. Benztropine 1 mg daily, was started, after which the dystonia resolved.

Course and complications: On day 16, she developed **catatonia** with Bush-Francis Catatonia Rating Scale (BFCRS) score of 7. **Because of lorazepam shortage, zolpidem 10 mg challenge was utilized.** Within 1 hour of administration, BFCRS score was 1. Oral intake improved significantly. Treatment with Clonazepam 0.5 mg three times daily initiated and continued for 11 days. BFCRS score decreased to 0 during hospitalization. At the end of hospitalization, the patient had persistent Cotard's delusion and residual paranoia. Antipsychotics were held at that time due to the prolonged QTc interval. Psychiatric hospitalization was recommended. Due to socioeconomic barriers, psychiatric placement was not possible.

Case Outcome: The patient was discharged home with family. Upon discharge, oral prednisone 40 mg daily was continued and was tapered over several weeks. Psychosis improved after several months of continued cyclophosphamide treatment and initiation of aripiprazole in the outpatient setting.

Discussion

- Onset of psychosis was several weeks before SLE diagnosis; however, psychosis persisted after glucocorticoid treatment.
- While the timeline supports the NPSLE diagnosis, the persistence and worsening of symptoms with glucocorticoids suggests a multifactorial etiology of the neuropsychiatric symptoms.
- Catatonic symptoms emerged after antipsychotics were discontinued. After successful treatment with zolpidem and benzodiazepines, the patient had persistent Cotard's delusion and paranoia, likely because of the multifactorial effect of continued steroid treatment and NPSLE.

Conclusion

1. This case of psychosis due to SLE and glucocorticoid use highlights a unique pattern of neuropsychiatric symptoms, including psychosis with Cotard's delusion complicated by catatonia.

2. This rare presentation of NPSLE underscores the challenge of differentiating glucocorticoid-induced psychosis from NPSLE, especially when the timeline of the possible etiologies overlaps.

3. This case highlights alternative approaches to treatment of catatonia with zolpidem and clonazepam in patients with SLE.

4. Further investigation is required into the pathophysiology and treatment of this pattern of neuropsychiatric symptoms.

References

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