

EPV0442

Dual Challenges: Managing Schizophrenia Relapse in a Patient with Sickle Cell Anemia

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Introduction: Sickle cell anemia (SCA) is a genetic disorder resulting in chronic hemolysis and vaso-occlusive crises, which can lead to severe systemic complications. Schizophrenia is a major psychiatric disorder characterized by persistent psychotic symptoms, including delusions and hallucinations. The intersection of these two conditions presents unique clinical challenges, as the management of SCA may complicate the treatment of schizophrenia and vice versa. Despite the prevalence of such comorbid cases, there is a significant gap in research addressing the complexities of managing these conditions concurrently.

Objectives: This case report aims to describe a rare and complex case of schizophrenia in a patient with sickle cell anemia, including the clinical presentation, diagnosis, and management. Additionally, it seeks to highlight the challenges of managing these co-occurring conditions and to promote further research into integrated treatment strategies for such comorbidities.

Methods: We present a 32-year-old male with a history of sickle cell anemia and schizophrenia who was admitted to the psychiatric department at Razi Hospital, LaManouba. The patient was admitted following a suicide attempt driven by acute psychotic symptoms, including delusions and auditory hallucinations commanding self-harm. Upon admission, he was started on a regimen of benzodiazepines to manage severe anxiety and antipsychotic medication to address the hallucinations and delusions. The initial physical examination revealed no significant abnormalities. On the third day of hospitalization, the patient began exhibiting respiratory symptoms, including chest pain and difficulty breathing, which prompted further diagnostic evaluation.

Results: On the third day of hospitalization, the patient developed respiratory symptoms including chest pain, fever, and dyspnea. Evaluation, including lab tests and imaging, revealed acute chest syndrome. Given the risk of benzodiazepines worsening respiratory issues, their use was discontinued, and Haloperidol was introduced at 5 mg to manage psychotic symptoms, with close monitoring for suicidal ideation. The hematology and pneumology departments initiated a treatment protocol involving oxygen therapy, intravenous hydration, paracetamol for pain management, and antibiotics (ceftriaxone and clarithromycin). Within a week, both psychiatric and respiratory symptoms significantly improved, leading to a reduction in supplemental oxygen and discharge with a comprehensive follow-up plan.

Conclusions: This case underscores the complexity of managing co-occurring schizophrenia and sickle cell anemia, highlighting the need for a multidisciplinary approach. Integrated treatment strategies are crucial for effectively addressing both psychiatric and medical complications in such comorbid conditions

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Systemic Inflammation from Periodontal Disease and its Neuropsychiatric Implications

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Introduction: Chronic oral health issues, particularly periodontal disease, are significant contributors to systemic inflammation. Emerging evidence indicates that this inflammation can influence neurophysiological processes and contribute to psychiatric disorders, including depression and anxiety. Recent literature review has revealed that periodontal disease and depression share common biomarkers, such as lipopolysaccharide (LPS), C-reactive protein (CRP), interleukin-6 (IL-6), and cortisol.

Objectives: To elucidate the connection between poor oral health and psychiatric disorders through the lens of systemic inflammation and its neurophysiological impacts.

Methods: A comprehensive literature review was conducted using PubMed, PsycINFO, and Google Scholar, focusing on studies from the past decade that examine the relationship between periodontal disease, systemic inflammation, and psychiatric disorders. Key findings regarding the involvement of pro-inflammatory cytokines and their neurophysiological effects were synthesized.

Results: The review highlighted that periodontal disease leads to elevated levels of pro-inflammatory cytokines. Such cytokines as CRP, IL-6, and tumor necrosis factor-alpha (TNF- α) can cross the blood-brain barrier, contributing to neuroinflammation and dysregulation of neurotransmitter systems. One significant effect is on serotonin metabolism, which is critical in the pathophysiology of depression. Pro-inflammatory cytokines can lead to reduced serotonin availability by upregulating genetic expression of serotonin transporters and increased serotonin metabolism. Given the interplay between inflammatory cytokines and neurotransmitter metabolism, the studies indicate that individuals with periodontal disease are more likely to exhibit depressive and anxiety symptoms, with neuroinflammation serving as a mediating factor.

Table 1: Impact of Pro-inflammatory Cytokines on Neurophysiological Processes

Cytokine	Effect on Neurophysiology
CRP	Contributes to neuroinflammation and affects processing skills, motor function, and working memory.
IL-6	Promotes neuroinflammatory responses via impacting neurotransmitter regulation and gut-microbiota-brain axis
TNF- α	Crosses BBB, leading to neuroinflammation and activates hypothalamus-pituitary axis leading to serotonin and tryptophan dysregulation.

Conclusions: Systemic inflammation resulting from poor oral health significantly impacts neurophysiological processes associated with psychiatric disorders. These findings underscore the need for integrated healthcare approaches that consider both oral and mental health to mitigate the adverse effects of neuroinflammation on psychiatric well-being.

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