

Case Report

SENSORY DEPRIVATION AND POST-ICTAL VULNERABILITY? PSYCHOSIS FOLLOWING PHENYTOIN-INDUCED TOXIC EPIDERMAL NECROLYSIS - A CASE REPORT

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Abstract

Background: Acute cutaneous and mucosal manifestations of drug-induced toxic epidermal necrolysis are well recognized. Ocular complications lead to visual impairment and sensory deprivation and may predispose to psychosis. **Case Report:** A 59-year-old woman with epilepsy, previously stable on anti-seizure medications, in whom phenytoin was introduced for breakthrough seizures. Following this, she developed toxic epidermal necrolysis (TEN), resulting in bilateral vision loss and subsequently full-blown psychosis. While she had shown mild irritability earlier, it was only after the seizure and abrupt sensory deprivation that she developed vivid hallucinations, delusions, and poor insight. The clear temporal sequence highlights the role of both postictal vulnerability and sensory deprivation in precipitating organic psychosis. **Conclusion:** This case emphasizes the need to evaluate organic causes when elderly patients present with new-onset psychotic symptoms.

Keywords: Anti-seizure medications, Toxic Epidermal Necrolysis, Visual Impairment, Sensory Deprivation, Psychosis

INTRODUCTION

Most anti-seizure medications (ASM) are associated with some level of cognitive, behavioural, or psychiatric side effects; however, certain ASM, such as topiramate, tiagabine, clobazam, levetiracetam, vigabatrin, and perampanel, are more commonly linked to aggression-related behaviours.¹ In addition to neuropsychiatric effects, some ASMs are also among the most common triggers for toxic epidermal necrolysis (TEN), a rare but life-threatening dermatologic emergency.² While

the acute cutaneous and mucosal manifestations of TEN are well recognized, long-term ocular complications can lead to lifelong visual impairment and discomfort.³ On the other hand, evidence also suggest that poor vision, particularly when it remains uncorrected despite using glasses, has been associated with an increased risk of developing psychosis.⁴

We report a case of behavioural disturbances in a patient on ASM, potentially reflecting poorly controlled epilepsy with features of postictal or interictal psychosis, which



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progressed to full-blown psychosis following sensory deprivation due to vision loss.

CASE REPORT

A 59-year-old woman with a medical history of seizure disorder, systemic hypertension, and mild hearing loss. She was on long-term treatment with Levetiracetam 1gm and Clobazam 5mg for seizures for the last 10 years. According to her son and husband, she started showing irritability for trivial reasons after being started on anti-seizure medication. However, she was still functioning well in her daily life, so they did not take any psychiatric treatment.

In August 2024, following a hospitalization for bronchopneumonia, she developed an acute confusional state with clouding of consciousness and difficulty with attention and awareness, triggered by hyponatremia and sepsis as per medical records. After discharge, the above symptoms resolved within 1 week, but she later had a seizure episode 1 week later while being on Levetiracetam and Clobazam, and was started on Phenytoin 300mg.

After starting phenytoin, the patient developed peeling and blistering skin over much of her body, including the mouth, eyes, and genitals. Despite these symptoms, she continued the medication for three more days, unaware of the potential side effects. This led to severe ocular complications. In the left eye, she developed a sterile corneal melt with perforation, while the right eye showed leucomatous corneal opacity. Both conditions resulted in significant vision loss, with the patient having hand movement (HM) vision in the right eye and a visual acuity of 2/60 in the left eye. She was admitted to Intensive care and managed with intravenous (IV)

dexamethasone, which was later switched to oral and stopped, along with IV antibiotics and topical ointments and creams. Due to ocular complications, the patient underwent left-eye lamellar keratoplasty to address a corneal melt with perforation.

Postoperatively, vision in the left eye improved to 12/60. Although vision improved slightly, it remains severely impaired. The patient became increasingly worried and anxious due to the sudden loss of vision and was deeply disturbed by the severity of the skin lesions. After the patient lost her vision, she began reporting seeing things that others couldn't. She described seeing dust falling on her, water falling from the sky, and fire burning, even keeping a cloth on her face to protect herself. She also claimed to see and feel a metre-long lizard crawling on her body, which she believed was planted by the doctors during her hospitalization. She named one of the lizards "Cicily," although she reported that it did not bite her. These visions were clear, well-formed, and frequent in nature. Additionally, the patient stated that a small child began walking with her after her illness. A female bystander was arranged to assist her during hospitalisation. Son reports this led to the patient becoming suspicious, believing her husband was having an affair with the woman. Whenever her husband went out or received a phone call, she would question him and accuse him of talking to the female bystander. She also believed that the female bystander would accompany her without her knowledge. She blamed her for the loss of her eyesight, claiming it was due to the eye drops administered by the female bystander. Following vision loss, the patient experienced added distress due to the worry that she may never regain her eyesight. She became increasingly preoccupied with these thoughts, growing more suspicious and increasingly

angry. Her recent laboratory reports, including Complete Blood Count (CBC), Liver Function Test (LFT), and Renal Function Test (RFT), showed no abnormalities, and her brain MRI was normal.

On mental status examination, the patient was well oriented and had normal psychomotor activity. Her memory was normal. Thought content included delusions of infidelity and persecution. Additionally, she experienced visual and tactile hallucinations, with high conviction in her beliefs and acting on these delusions. Her insight was graded as 1 (no insight), as she had no awareness of the nature of her condition. She presented to psychiatry with the above symptoms, and a diagnosis of organic psychosis was made. She was started on Amisulpride 50 mg. At this dose, the patient stopped reporting of visual and tactile hallucinations of a lizard crawling. However, her delusions of infidelity persisted. Therefore, at follow-up, the dose of Amisulpride was increased to 100 mg. At the 1-month follow-up, all her psychotic symptoms had subsided.

DISCUSSION

This case illustrates a rare organic psychosis that possibly began after a seizure episode and was exacerbated by sudden bilateral visual loss due to Phenytoin-induced TEN. The patient, on long-term Levetiracetam and Clobazam, had prior behavioural changes like irritability but no psychotic symptoms. Following the seizure, Phenytoin triggered TEN and profound vision loss, after which she developed vivid hallucinations and delusions, suggesting sensory deprivation played a key role in precipitating psychosis. One differential diagnosis considered was Charles Bonnet Syndrome (CBS), which is characterized by

complex visual hallucinations in individuals with visual impairment, typically without delusions or psychiatric symptoms and with preserved insight.⁵ However, in this case, the patient exhibited delusions, hallucinations, and impaired insight, indicating a broader organic psychosis, rather than a benign perceptual phenomenon.

Numerous case reports have documented psychosis following anti-seizure medications (ASMs), particularly Phenytoin and Levetiracetam, often associated with postictal states or medication side effects.⁶ However, in our literature research, we could not find any case report defining psychosis that is postictal and worsened by sensory deprivation, such as visual loss resulting from Phenytoin-induced toxic epidermal necrolysis (TEN). This unique combination of factors, in our case, highlights an under-recognized clinical scenario. While congenital blindness has been shown to offer some protection against the development of schizophrenia, sudden vision loss in later life, as seen in this patient, appears to predispose individuals to the onset of psychosis. The abrupt sensory deprivation following visual loss, compounded by the psychological trauma and dependence on others, contributed significantly to the development of psychosis in this case.⁷ This case underscores the critical role of sensory deprivation, particularly vision loss, in the onset of psychosis, especially in older adults. It emphasizes the need for heightened awareness and vigilant monitoring for psychiatric symptoms in patients experiencing sudden sensory loss, particularly when compounded by neurological or medication-related factors. Clinicians should consider the interplay between sensory deprivation, medication side effects, and preexisting neurological conditions in the development of psychosis.

In conclusion, in elderly patients, psychosis may often be misdiagnosed as a primary psychiatric illness; however, when closely examining the temporal sequence of events, such as seizures followed by abrupt sensory deprivation, it becomes evident that organic factors may be the primary cause. The argument that sudden loss of sensory input can precipitate psychosis in vulnerable populations is particularly thought-provoking. It highlights the importance of thorough medical evaluation in elderly patients presenting with new-onset psychosis.

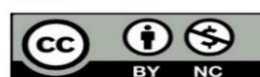
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