Case Report

Total Binocular Blindness Resulting from an Untreated Seizure: A Case Report

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Abstract

Cortical blindness is a rare but serious complication of seizure disorders, typically associated with occipital lobe involvement. We report a case of a 32-year-old female with a history of hydrocephalus who developed acute, irreversible bilateral blindness following an untreated generalized tonic-clonic seizure episode. Flash visual evoked potential (VEP) testing revealed cortical dysfunction, consistent with seizure-related occipital damage. This case suggests that untreated seizures may lead to permanent vision loss through cortical visual pathway disruption. It underscores the potential neurological risks of delayed seizure intervention and highlights the importance of timely management to mitigate such outcomes. The report is contextualized with a review of relevant literature on seizure-induced cortical visual impairment.

Keywords: Cortical Blindness; Seizures; Visual Pathways; Hydrocephalus; Status Epilepticus; Evoked Potentials, Visual.

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Introduction

Seizures are a frequent neurological emergency, particularly in pediatric populations, but with serious implications for adults as well [1]. Epilepsy incidence is estimated to be 50 to 60 per 100,000 individuals globally, with nearly 8% of population experiencing at least one seizure in their lifetime [2]. Initial seizure treatment is critical to prevent complications such as neuronal damage, cognitive impairment, or even death [3].

Though uncommon, cortical blindness has been reported as a seizure consequence [4]. For instance Zhao and Si [5] reported a case of posterior reversible encephalopathy syndrome in a patient with end-stage kidney disease, where seizures led to transient bilateral cortical blindness. Hydrocephalus, a known risk factor for visual pathway compromise via periventricular white matter injury, may potentiate seizure-related cortical damage [6].

The present case explores a similar, though irreversible, visual deficit linked to untreated seizures. This report aims to highlight the clinical consequences of delayed seizure intervention by presenting a case of permanent cortical blindness and contextualizing it within relevant neurological literature.

Case Presentation

A 32-year-old female presented at Basir Eye Clinic, Tehran, Iran, for flash visual evoked potential (VEP) examination due to complete loss of vision in both eyes. Medical history included hydrocephalus managed with a shunt when she was 5 months old. Six months prior to the present episode, she experienced a generalized tonic-clonic convulsive episode of about three minutes followed by a rapid decline in visual acuity, progressing to no light perception (NLP) bilaterally within 24 hours. No medical treatment was provided at the time of seizure onset. VEP testing revealed cortical dysfunction consistent with visual pathway disruption leading to probable irreversible blindness. No visual recovery occurred after repeat VEP after one month showing persistent cortical silence. Neurological exam confirmed absent light perception bilaterally with normal pupillary reflexes. Fundoscopy revealed no optic disc edema or atrophy. Shunt patency was not assessed.

Discussion

Our case highlights one of the rare and severe consequences of untreated seizures. Joseph and Louis [7] reviewed 44 cases of seizure-induced bilateral blindness, categorizing them by etiology, duration, and visual prognosis. Their findings confirmed that cortical blindness can arise during the ictal phase, post ictal phase (Todd's phenomenon), or as a lasting sequela of status epilepticus [7]. Their case of transient cortical blindness linked to left occipital seizure activity reinforces that even focal occipital seizures can produce bilateral visual loss [7]. This classification underscores the variability in clinical course and highlights the need for accurate diagnosis and early intervention to optimize visual outcomes.

Zhao and Si [5] described a patient who developed bilateral cortical blindness following seizures induced by posterior reversible encephalopathy syndrome. Imaging revealed occipital lobe involvement, areas critical for visual processing. Although their patient's vision recovered after prompt treatment, our patient experienced permanent loss, likely due to the absence of timely intervention.

Kornguth et al., [8] conducted a detailed histopathological study of a patient with cortical blindness and a history of seizures in

the context of untreated phenylketonuria. Using Golgi-Kopsch silver staining, they identified significant neuronal loss and architectural disruption in the calcarine cortex and lateral geniculate nucleus [8]. Although metabolic dysfunction contributed to the pathology, their findings underscore how prolonged excitotoxic stress, such as from recurrent or unmanaged seizures, can permanently injure visual processing centers [8]. This neuro- pathological evidence offers structural validation for the irreversible cortical blindness observed in our patient [8].

Shushtarian and Pour Mazar [9] detailed case where abrupt withdrawal of antiepileptic medication resulted in irreversible blindness. Moreover, Carroll et al., [10] presented a complex case involving seizures, fever, hallucinations, and vision loss later attributed to overlapping autoimmune disorders, anti N-methyl-D-aspartic acid receptor encephalitis and myelin oligodendrocyte glycoprotein antibody disease. Their patient's visual loss stemmed from optic neuritis and cortical lesions associated with an autoimmune process post-seizure. While our patient did not undergo comprehensive immunologic evaluation, this highlights the importance of considering autoimmune encephalitis in cases of post-seizure vision loss, particularly when standard imaging or clinical presentation is atypical.

Additionally, Aldrich et al., [11] reported a case of a patient who developed permanent visual deficits, including hemianopia and dyschromatopsia, following repeated occipital lobe seizures. This reinforces the concept that prolonged or clustered partial seizures, particularly in the occipital region, can result in irreversible cortical injury and blindness, even in the absence of generalized convulsive episodes or structural abnormalities at onset.

Further extending this understanding, Badran et al., [12] described a case of transient postictal blindness following a focal seizure originating from the posterior cingulate gyrus. Although the visual loss in that case resolved within 48 hours, it underscores that seizures originating outside the occipital lobe can also propagate to visual processing areas and cause temporary blindness [12].

In another notable case, Hadjikoutis and Sawhney [13] described a 61-year-old man who developed bilateral visual loss secondary to occipital seizures, confirmed by EEG showing bi-occipital spike discharges. His vision returned to baseline following carbamazepine therapy [13]. This case underscores the potential for visual recovery when seizures are promptly identified and treated [13]. It also emphasizes the necessity of considering occipital seizures in the differential diagnosis of sudden bilateral visual loss, especially in the absence of fundoscopic abnormalities [13].

Complementing this perspective, Yang et al., [14] reported a case where nonconvulsive status epilepticus manifested primarily as acute visual impairment, with magnetic resonance imaging (MRI) revealing transient peri-ictal abnormalities in the temporoparietal and occipital lobes. Electroencephalogram (EEG) confirmed continuous epileptic discharges in those regions. Following antiepileptic therapy, the patient's vision and imaging abnormalities resolved. This case illustrates that even in the absence of convulsive symptoms, seizure activity can significantly affect cortical visual processing, and emphasizes the diagnostic value of EEG and MRI in patients presenting with sudden vision loss.

The findings in our case are consistent with these reports, emphasizing that cortical blindness can result from occipital or propagation-involved seizure activity, especially when management is delayed or

absent. Prompt neurological intervention, including imaging, immunologic testing, EEG, and anti-epileptic therapy, may prevent such outcomes. While in our case abnormal VEP results support cortical dysfunction, hydrocephalus, a known risk factor for visual pathway injury, raises the possibility of multifactorial pathology. Undetected shunt malfunction could precipitate seizures and independently cause blindness, necessitating future cases to clarify interactions.

This report has several noteworthy limitations. Most critically, the absence of neuroimaging (MRI or CT) precludes definitive exclusion of structural etiologies, such as acute ischemic stroke, shunt malfunction, or occipital lobe pathology unrelated to seizure activity. Additionally, EEG was not performed to characterize ictal/postictal activity or identify subclinical seizures. The lack of immunological and metabolic testing (e.g., autoimmune encephalitis panels, metabolic screens) further limits our ability to rule out confounding contributors to cortical blindness, particularly given the patient's history of hydrocephalus, a potential independent risk factor for visual pathway compromise. These diagnostic gaps constrain the certainty of attributing blindness solely to untreated seizures and highlight the need for comprehensive evaluation in similar presentations.

Future studies should investigate the pathophysiological thresholds at which seizure activity causes irreversible visual cortical damage, with particular focus on the role of early intervention and biomarkers predictive of visual recovery.

Conclusion

This case underscores that untreated seizures can potentially lead to severe neurological sequelae, including permanent visual impairment. While the irreversible cortical blindness observed here aligns with prior reports of seizure-related occipital dysfunction, comprehensive neuroimaging and electrophysiological assessment would have strengthened etiological certainty. Nevertheless, this outcome reinforces the importance of prompt recognition and management of seizures to mitigate risks of significant neurological injury. Further research is needed to clarify pathophysiological thresholds for irreversible visual cortical damage and identify biomarkers to guide prognostication.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript.