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Case Report

Phenytoin-induced cerebellar atrophy: A case for reversibility of neurological decline ☆☆☆★

Edwin Mogere^{a,*}, Davis Cheruiyot^b, Manakhe Nassiuma^a^a Section of Neurosurgery, Department of Surgery, Aga Khan University Hospital, Nairobi, Kenya^b Department of Neurosurgery, Moi Teaching and Referral Hospital, Eldoret, Kenya

ARTICLE INFO

Article history:

Received 14 September 2023

Revised 22 October 2023

Accepted 26 October 2023

Keywords:

Anticonvulsant

Ataxia

Apoptosis

Drug-Related side effects and adverse reactions

Nystagmus

Pathologic

ABSTRACT

This case serves as a reminder of the infrequent, yet consequential occurrence of cerebellar degeneration linked to phenytoin usage. Whilst emphasizes the importance of monitoring patients on long-term phenytoin therapy, and it further suggests considering employing bedside imaging tools such as Ultrasound fusion imaging for follow-up of patients at risk of this type of disorder. We present a case study involving a 23-year-old woman who experienced significant neurological impairment resulting in severe cerebellar atrophy while undergoing phenytoin treatment. On cessation of phenytoin, the patient exhibited improvement with enhanced cerebellar function.

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Introduction

Phenytoin, an often-used medication for epilepsy in Kenya, has been associated with various neurological side effects, including rare cases of cerebellar atrophy [1,2]. We present a case study of a woman who experienced severe cerebellar atrophy while receiving phenytoin treatment for post-traumatic seizures following a car accident.

Case presentation

A 23-year-old woman weighing 62 kg was referred due to neurological deterioration over 18 months after starting phenytoin therapy. The medication was prescribed daily at 300 mg to manage post-traumatic seizures (2 episodes) resulting from a motor vehicle accident. Before the accident, she had been in good health with no medications or known allergies.

☆ The objective of the case is to highlight the rare but serious complication of cerebellar atrophy that can be mitigated with phenytoin cessation.

☆☆ Acknowledgments: There was no funding for this study.

* Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.

E-mail address: edwin.mogere@aku.edu (E. Mogere).

<https://doi.org/10.1016/j.radcr.2023.10.064>

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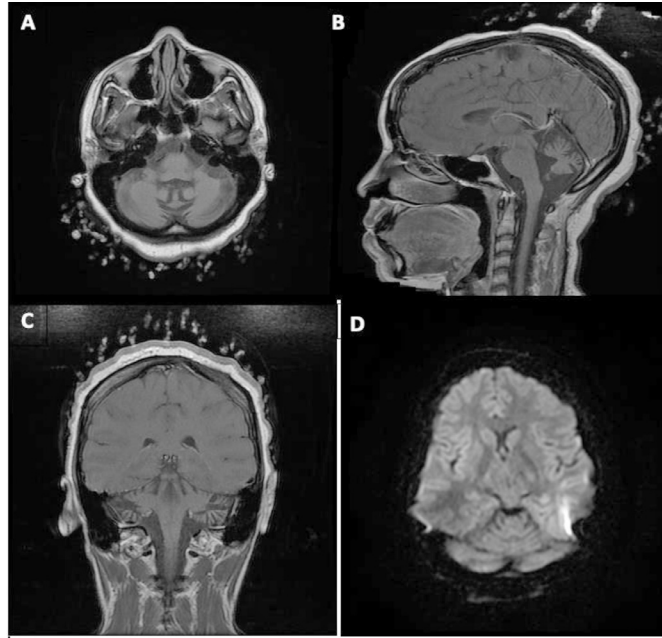


Fig. 1 – T1 weighted MRI images with gadolinium. (A) Axial T1 precontracted. (B) Sagittal T1 post-contrast. (C) Coronal T1 post-contrast. (D) Diffusion-weighted image.

Her condition progressively worsened, leading to difficulties with balance and coordination (appendicular ataxia), involuntary eye movements (nystagmus), and resting and intention tremors. These symptoms significantly impacted her life as she became dependent on a wheelchair and unable to continue her education or work. Magnetic resonance imaging (MRI) revealed a cerebellar volume loss consistent with at-

rophy without any other observable structural abnormalities (Figs. 1 and 2).

Phenytoin was subsequently discontinued, and an impressive improvement in her clinical state was observed. By the 1-year follow-up, her truncal ataxia and nystagmus had resolved, and she could resume assisted ambulation. Additionally, she continued schooling and work. A follow-up MRI

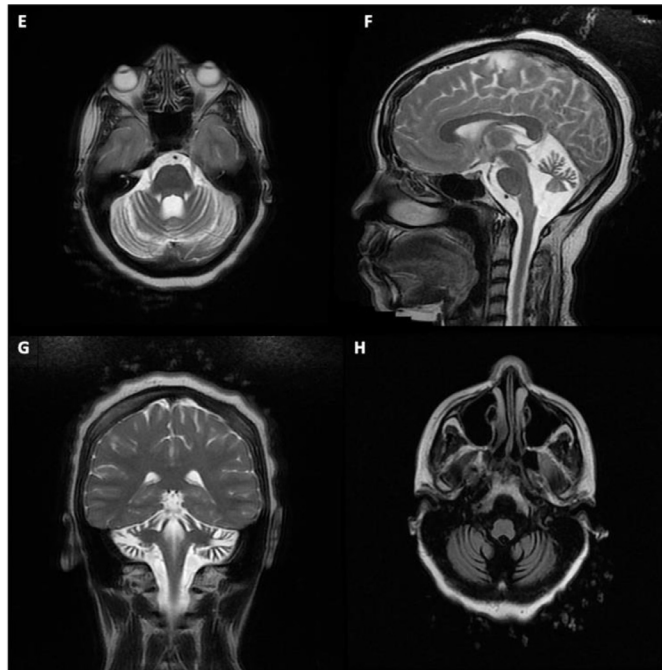


Fig. 2 – T2 Weighted (A) Axial T2. (B) Sagittal T2. (C) Coronal 2 (D) T2-FLAIR.

indicated a stable cerebellar volume. Differential diagnoses considered in this case include Post-traumatic brain injury, postinfectious cerebellitis, and idiopathic cerebellar degeneration/cerebellar ataxias (spinocerebellar ataxia, sporadic adult-onset ataxia). After negative systemic tumor surveillance, a Paraneoplastic syndrome was also excluded.

Discussion

Despite its effectiveness, phenytoin has been associated with adverse effects, including the occasional occurrence of cerebellar atrophy. Cerebellar atrophy caused by phenytoin, whilst uncommon, is a described side effect of the medication [3]. The exact mechanism behind this remains unclear. There is a hypothesis that phenytoin-induced neuronal apoptosis may be involved [4]. Phenytoin works by regulating voltage-gated sodium channels to control the firing of neurons. Current theories suggest that several factors contribute to induced neuronal apoptosis, such as increased production of reactive oxygen species (ROS) leading to oxidative stress, dysfunction in mitochondria as observed by Shin et al., [4] activation of apoptotic pathways, and potential disruptions in neural plasticity. These mechanisms are complex and continuously evolving.

This case emphasizes the importance of monitoring patients on long-term phenytoin therapy, especially those who experience new or worsening ataxia [5]. Early diagnosis and discontinuation of phenytoin can result in recovery, as demonstrated in our case and supported by other reports in medical literature [6,7]. This case also highlights the potential utility of employing bedside imaging tools such as Ultrasound fusion imaging to detect or follow up patients with suspected or confirmed anatomical lesions such as cerebellar degeneration [8].

Conclusion

Our case highlights the potential for reversible cerebellar atrophy caused by phenytoin, highlighting the need for careful attention among patients receiving this medication.

Healthcare providers should be aware of the possibility of induced cerebellar atrophy in patients with progressive neurological decline.

Clinicians should be aware of this side effect and have a structured plan to monitor patients on long-term phenytoin therapy. It might be wise to discuss alternatives to phenytoin for these patients and weigh the advantages and disadvantages of each option.

Patient consent

We would like to state that we have obtained written informed consent from the patient before submitting the manuscript entitled “Phenytoin-induced Cerebellar Atrophy: A Case for Reversibility of Neurological Decline” for consideration of publication in *Radiology Case Reports*.

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