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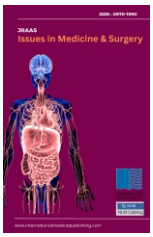

Case Report

Section: Medicine

Paradoxical Optochiasmatic Arachnoiditis with Acute Bilateral Visual Loss During Treatment of Tuberculous Meningitis: A Case Report

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HIGHLIGHTS

- Paradoxical OCA in TBM
- Acute bilateral visual loss
- Early diagnosis is crucial
- Steroids for inflammatory control
- Exclude ethambutol toxicity

Key Words:
Tuberculous meningitis
Paradoxical reaction
Optochiasmatic arachnoiditis
Bilateral visual loss
Tuberculoma
Corticosteroids.

ABSTRACT

Introduction: Tuberculous meningitis (TBM) is the most severe form of central nervous system tuberculosis and is associated with significant neurological morbidity and mortality. Paradoxical reactions, characterized by clinical or radiological deterioration despite appropriate antituberculous therapy (ATT), may complicate treatment. Optochiasmatic arachnoiditis (OCA) is a rare but devastating complication that can cause irreversible visual impairment. **Aim & Objective:** To highlight paradoxical optochiasmatic arachnoiditis as a rare complication of TBM and emphasize the importance of early diagnosis and aggressive anti-inflammatory management. **Case Presentation:** A 24-year-old woman presented with irrelevant speech, altered mental status, headache, and vomiting. Examination revealed impaired consciousness, meningeal signs, sluggish pupils, and abnormal plantar responses. CSF showed protein of 56 mg/dL, glucose of 52 mg/dL, and lymphocytic predominance. Cartridge-based nucleic acid amplification testing detected *Mycobacterium tuberculosis* without rifampicin resistance. MRI demonstrated acute infarcts in the bilateral basal ganglia, genu of corpus callosum, and left amygdala, with tuberculous granulomatous lesions in the right cerebellum and pons. She was diagnosed with TBM with tuberculomas and cerebral infarctions and initiated on ATT and corticosteroids. **Result:** After initial improvement, she developed paradoxical neurological deterioration with acute painless bilateral visual loss progressing to perception of light only. MRI findings were consistent with OCA. Ethambutol toxicity was considered and the drug was withheld. ATT was continued with high-dose dexamethasone and adjunctive thalidomide**. Conclusion:** Paradoxical OCA should be considered in TBM patients developing visual impairment after ATT. Early recognition and aggressive anti-inflammatory therapy are crucial to prevent irreversible visual disability and differentiate the condition from ethambutol toxicity, treatment failure, and drug resistance.

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INTRODUCTION

Tuberculous meningitis (TBM) is the most severe form of tuberculosis and is associated with significant mortality and neurological disability. Its complications include hydrocephalus, cerebral infarction, tuberculoma formation, vasculitis, cranial neuropathies and basal arachnoiditis [1,2].

A paradoxical reaction (PR) is characterized by clinical or radiological worsening of tuberculosis after initiation of appropriate ATT, usually after an initial clinical response and in the absence of another explanation such as treatment failure, drug resistance, or a new infection [1,2]. In a prospective cohort of 141 patients with TBM, 44 patients (31.2%) developed a paradoxical reaction; 12 developed OCA, representing 8.5% of the entire cohort and 27.3% of patients with paradoxical reactions [1]. OCA is a serious manifestation of basal tuberculous inflammation involving the optic nerves and chiasm. Inflammatory exudates in the basal cisterns may produce optic pathway compression, vascular compromise, adhesion, and fibrosis, resulting in severe visual impairment or blindness [3,4].

Several clinical and cerebrospinal fluid (CSF) characteristics have been associated with an increased risk of optochiasmatic arachnoiditis (OCA). In a cohort of 163 patients with tuberculous meningitis, female sex, age younger than 27 years, and CSF protein concentration >260 mg/dL were identified as independent predictors of OCA [4]. In the same cohort, 23 patients (14%) developed OCA, and 18 of these 23 patients (78%) developed OCA while already receiving antituberculous therapy [4]. The patient in the present case was a 24-year-old woman and therefore had two of the reported demographic risk factors; however, her CSF protein was 56 mg/dL, below the reported high-risk threshold. This highlights that OCA can occur even in the absence of markedly elevated CSF protein. Visual impairment itself is an important prognostic marker in TBM. In a cohort of 101 adults, OCA on MRI was a predictor of visual deterioration and blindness at six months [5]. Optochiasmatic arachnoiditis results from inflammatory exudation predominantly involving the basal cisterns around the optic nerves and optic chiasm. Subsequent inflammatory adhesion, fibrosis and compression of the visual pathways may result in severe visual impairment. It is particularly important because visual loss may occur despite appropriate ATT and corticosteroid therapy [6]. **Figure 1.** Schematic representation of paradoxical optochiasmatic arachnoiditis following initiation of antituberculous therapy, showing inflammatory exudates, fibrosis, and compression of the optic pathways.

The present case describes a young woman with microbiologically confirmed TBM who developed acute bilateral visual loss during treatment, with MRI findings interpreted in the clinical setting as optochiasmatic arachnoiditis. The case highlights the diagnostic challenge of distinguishing paradoxical OCA from treatment failure and antituberculous drug toxicity.

CASE PRESENTATION

A 24-year-old woman presented to the emergency with irrelevant speech, altered mental status, headache and vomiting from last 3 days. There was no reported history of seizures, visual disturbances or focal neurological deficits at presentation. On examination, her GCS score was E3V4M5. Meningeal signs were present. The right plantar response was extensor, while the left plantar response was non-elicitable. Both pupils were sluggishly reactive to light.

On investigation, initial complete blood count, renal function, liver function and serum electrolytes were reportedly within normal limits. Her HIV status was negative. CSF examination at the current presentation is shown in **Table 1** below.

Contrast enhanced MRI brain demonstrated acute infarcts involving the bilateral basal ganglia, genu of the corpus callosum

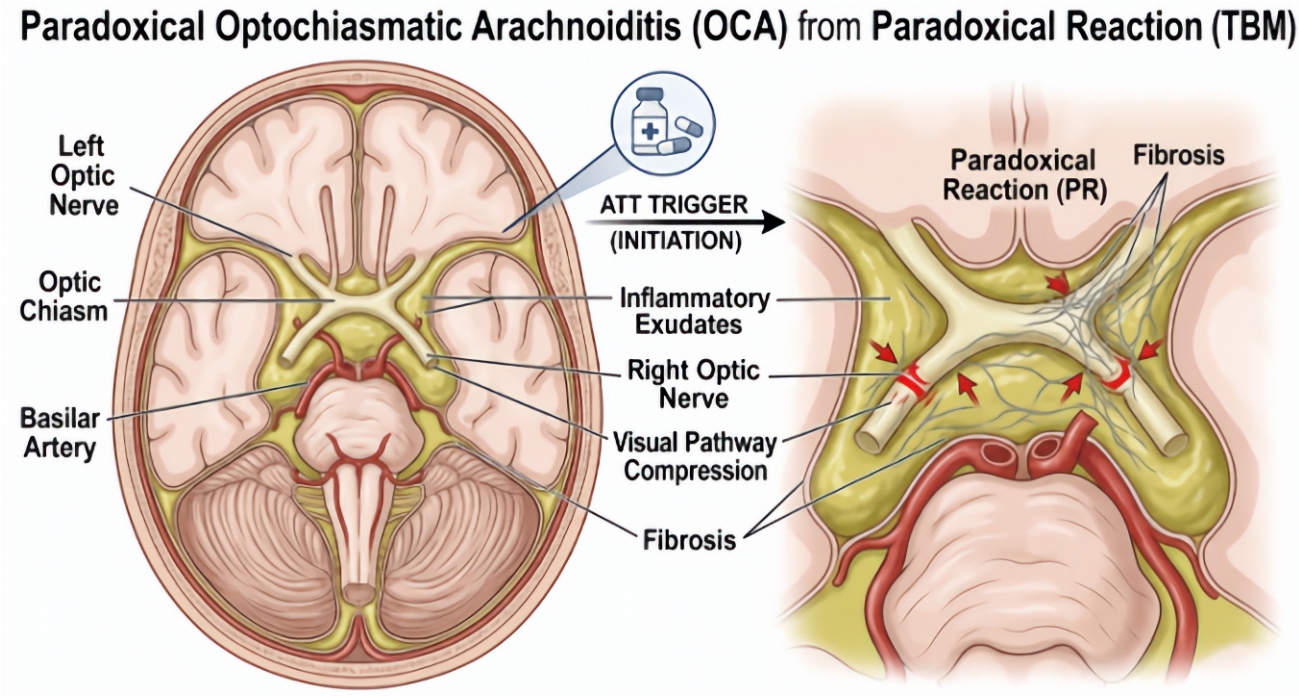

Figure 1: Paradoxical optochiasmatic arachnoiditis showing optic pathway compression and fibrosis..

and left amygdala. Nodular enhancing lesions were present in the right cerebellar hemisphere and right pons. The lesions were considered radiologically compatible with infectious granulomatous lesions, with tuberculosis favored in the clinical and microbiological context. These findings, together with microbiological confirmation of *M. tuberculosis* in CSF, supported a diagnosis of tuberculous meningitis with intracranial tuberculomas and multiple acute cerebral infarctions.

The patient was started on standard antituberculous therapy (HRZE) with adjunctive corticosteroids. She initially demonstrated clinical improvement and was discharged in a stable condition. The subsequent clinical deterioration occurred after 3 days of discharge and 6 days of initiation of ATT, raising the possibility of a paradoxical reaction. Paradoxical worsening during treatment of TBM is well described and should not automatically be interpreted as treatment failure or drug resistance.

The patient was subsequently readmitted with altered sensorium. Hyponatremia was detected and corrected. Repeat CSF examination did not demonstrate evidence of a new central nervous system infection. During the hospital course, the patient developed acute, painless bilateral visual loss, which progressed to such severity that there is only perception of light present.

This deterioration prompted consideration of several possible causes, including:

- Paradoxical optochiasmatic arachnoiditis;
- Optochiasmatic tuberculoma;
- Ethambutol-associated optic neuropathy;
- Raised intracranial pressure;
- Infectious or inflammatory optic neuropathy;
- Ischemic involvement of the visual pathways.

Fundoscopic examination at that time showed no papilledema. There were no fundoscopic and colour contrast study findings specifically supporting established ethambutol toxic optic neuropathy. Nevertheless, because of the acute severe visual deterioration while receiving ethambutol, the drug was withheld. The absence of papilledema was clinically relevant because visual impairment in TBM can result from several mechanisms, including raised intracranial pressure, optic pathway involvement and basal arachnoiditis.

On radiological reassessment, the MRI findings were considered consistent with inflammatory involvement of the optochiasmatic region **(Figure 2 a–c)**, supporting the diagnosis of optochiasmatic arachnoiditis in the setting of TBM and new severe bilateral visual loss.Because paradoxical worsening does not necessarily represent treatment failure, antituberculous therapy was continued. The regimen was modified to Isoniazid, Rifampicin, Pyrazinamide and Streptomycin (HRZS). Ethambutol was withheld because drug-associated optic toxicity remained a possible contributor to the acute visual deterioration.

Because of rapidly progressive bilateral visual loss attributed to severe inflammatory involvement of the optic pathways, high-dose corticosteroid therapy was administered. Dexamethasone 8 mg intravenously three times daily was administered for four weeks, followed by a planned gradual taper.

Prolonged corticosteroid therapy was planned for at least 12 weeks because of the severity of the paradoxical inflammatory complication. Standard WHO guidance recommends adjunctive dexametha-sone or prednisolone for TBM, generally tapered over 6-8 weeks. However, the optimal duration and intensity of corticosteroid therapy specifically for severe paradoxical OCA are uncertain, and prolonged high-dose therapy has been reported in individual cases and small series.

Also considering, the severe inflammatory nature of the OCA and the potential for irreversible visual loss, adjunctive thalidomide was considered as an immunomodulatory therapy. Thalidomide 50 mg once daily was initiated and increased to 50 mg twice daily after two weeks, as the patient tolerated the drug well and contraceptive counselling was provided given its teratogenic potential.

At presentation, visual acuity was approximately 6/12 bilaterally. During the subsequent course, she developed acute painless bilateral visual loss progressing to perception of light only.

Following treatment with high-dose intravenous dexamethasone and adjunctive thalidomide, visual acuity improved to 6/24 bilaterally at two weeks, but remained unchanged at 6/24 at six-month follow-up, without further recovery **(Table 2)**. **Table 3:** Clinical time-line of the patient.

Table 1: CSF parameters of the patient at presentation.

CSF parameter	Result
Protein	56 mg/dL
Glucose	52 mg/dL
Total cells	2 cells/µL
Differential	Lymphocytic predominance
CBNAAT	M. tuberculosis detected
Rifampicin resistance	Not detected

Table 2: Visual acuity of the patient during treatment.

Clinical stage	Visual acuity
Initial presentation	Approximately 6/12 OU
At severe visual deterioration	Perception of light only OU
Two weeks after dexamethasone + thalidomide	6/24 OU
Six-month follow -up	6/24 OU (unchanged)

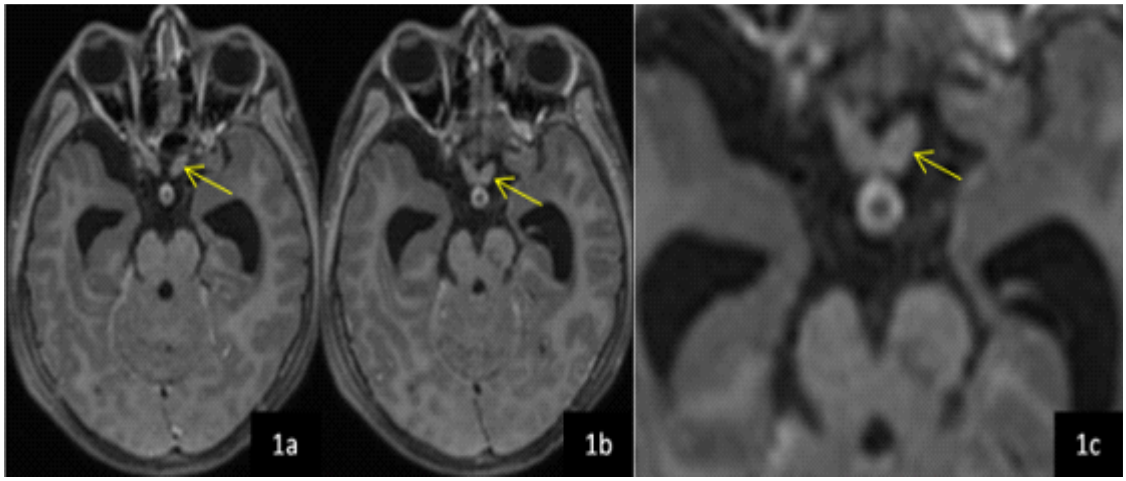

Figure 2: MRI findings in optochiasmatic arachnoiditis. (1a, 1b) Axial contrast-enhanced T1-weighted MRI images showing abnormal enhancement involving the basal/perichiasmatic region (arrows), interpreted in the clinical context as optochiasmatic arachnoiditis. (1c) Magnified view of the perichiasmatic region, demonstrating the enhancing lesion (arrow) in greater detail.

Table 3: Clinical timeline of the patient.

Time point	Clinical event
Day 0	Presentation with altered mental status, headache & vomiting; CSF CBNAAT positive for *M. tuberculosis* .
Day 0	HRZE and adjunctive corticosteroids initiated.
Early course	Initial neurological improvement; discharged in stable condition.
Day 6 after ATT initiation	Readmitted with altered sensorium; hyponatremia detected and corrected.
Subsequent hospital course	Acute painless bilateral visual loss progressing to perception of light only.
After visual deterioration	MRI reassessed; findings supported optochiasmatic arachnoiditis. Ethambutol withheld.
Treatment escalation	Dexamethasone 8 mg IV three times daily initiated; ATT continued as HRZS.
Same treatment period	Thalidomide 50 mg once daily initiated and increased to 50 mg twice daily after 2 weeks.
2 weeks after intensified anti-inflammatory therapy	Visual acuity improved to 6/24 bilaterally.

DISCUSSION

This case illustrates a clinically important paradoxical complication of TBM in which severe visual loss developed after initiation of appropriate antituberculous therapy. Paradoxical reactions are relatively common during treatment of TBM. In a prospective cohort of 141 patients, 44 (31.2%) developed a paradoxical reaction. Among these patients, 12 (8.5%) developed optochiasmatic arachnoiditis [1]. A review by Garg et al. similarly emphasizes that paradoxical worsening may be mistaken for treatment failure, drug resistance, or drug toxicity [2].

Recognition of patients at increased risk may facilitate closer neurological and ophthalmological monitoring during treatment of tuberculous meningitis. In the 163-patient cohort reported by Anupriya et al., female sex, age <27 years, and CSF protein >260 mg/dL were independently associated with the development of OCA [4]. The present patient was a 24-year-old woman and therefore had two of these reported demographic risk factors, although her CSF protein was only 56 mg/dL. These factors should be considered associations rather than diagnostic criteria, and the absence of elevated CSF protein does not exclude OCA. In addition, the prospective cohort reported by Singh et al. identified female sex, HIV positivity, and shorter duration of illness as predictors of paradoxical reactions overall [1]. Thus, the young age, female sex and shorter duration of illness of our patient may have represented a predisposition to paradoxical inflammation and OCA, although causality cannot be inferred from a single case.

The pathogenesis is thought to involve an exaggerated host inflammatory response to mycobacterial antigens following initiation of effective therapy. In TBM, the inflammatory process can produce increased basal exudates, tuberculoma formation, vasculitis and arachnoiditis [2]. OCA is particularly important because of its potential to produce profound visual disability. Basal inflammatory exudates can involve the optic nerves and chiasm, with subsequent inflammation, adhesion and fibrosis producing visual pathway dysfunction. MRI typically demonstrates abnormalities in the basal cisternal and perichiasmatic regions [3,4].

OCA has important prognostic implications. In the cohort of 163 patients reported by Anupriya et al., 23 (14%) developed OCA, with 18 of 23 cases developing while patients were already receiving ATT. At six months, only 17% had improvement in visual acuity, while 52% had no further deterioration [4]. In another cohort of 101 adults with TBM, OCA on MRI was a

predictor of visual deterioration and blindness at six months [5]. These findings support the generally guarded visual prognosis of OCA.

In the present case, the temporal relationship between ATT initiation and subsequent neurological deterioration, followed by acute bilateral visual loss, raised strong suspicion of a paradoxical inflammatory process. The absence of papilledema made severe visual loss due solely to raised intracranial pressure less likely. Ethambutol toxicity was an important competing diagnosis and was addressed by discontinuing ethambutol. The distinction between paradoxical OCA and treatment failure is particularly important. Paradoxical worsening should not automatically lead to discontinuation of effective ATT or labeling the disease as drug resistant. Published reviews emphasize that paradoxical reactions represent an exaggerated host response rather than failure of antituberculous therapy. The timing of deterioration is noteworthy. Neurological worsening occurred six days after initiation of ATT, substantially earlier than the mean interval of 41 days reported in a series of paradoxical optochiasmatic tuberculoma [6]. This difference does not exclude a paradoxical reaction because the timing of paradoxical responses is variable, but it represents an unusual feature of the present case and should be highlighted rather than generalized.

Because the patient developed paradoxical neurological deterioration, repeat CSF examination was performed. Repeat CSF CBNAAT was not performed in this patient. The decision not to repeat microbiological testing was based on the absence of clinical features suggesting a new infection or treatment failure, and on the recognition that paradoxical reactions are primarily clinicoradiological diagnoses rather than microbiologically defined events. However, a negative or unchanged CSF profile would not by itself have excluded a paradoxical reaction, and in the prospective TBM cohort cited above, one patient with a paradoxical reaction had normal CSF findings [1].

The treatment of paradoxical OCA remains poorly standardized. Corticosteroids are the main anti-inflammatory therapy, but the optimal dose and duration for severe OCA have not been established. WHO recommends adjunctive corticosteroids for TBM, generally tapered over 6-8 weeks [7]. More prolonged or intensified corticosteroid treatment has been used in patients with severe paradoxical visual complications. In a series of eight patients with paradoxical optochiasmatic tuberculoma and visual deterioration, an extended six-week course of dexamethasone was administered with ATT; several surviving patients demonstrated partial or complete visual recovery [6].

Thalidomide represents a potential adjunctive immunomodulatory approach in severe refractory CNS tuberculosis. Small case series have reported improvement in patients with TBM-associated optic neuropathy and OCA following thalidomide therapy [8,9,10]. However, these data remain limited to case reports, small series and observational cohorts, and therefore thalidomide should be regarded as an individualized adjunct rather than established standard therapy. Thalidomide has been investigated as adjunctive immunomodulation in severe CNS tuberculosis because of its ability to modulate TNF-α and other inflammatory pathways. A published case series described recovery of vision in four consecutive patients with TBM-related optic neuropathy treated with thalidomide, with corresponding radiological improvement [8]. A separate report of four patients with intractable intracranial tuberculosis also described clinical and neuroradiological improvement after addition of thalidomide, including a patient with chronic basal arachnoiditis and progressive visual loss [9]. More recently, an observational cohort of 38 children treated with adjunctive thalidomide for complicated CNS tuberculosis included four patients with blindness secondary to OCA, supporting further interest in targeted immunomodulation, although controlled trial evidence remains lacking [10]. Because thalidomide is highly teratogenic, pregnancy risk must be assessed before treatment, & appropriate contraception and pregnancy-prevention counseling must be documented in a reproductive-age woman.

The visual prognosis in optochiasmatic arachnoiditis is generally guarded. Published data demonstrate that OCA is strongly associated with visual deterioration and blindness in TBM. In a cohort of 101 patients, optochiasmatic arachnoiditis was a predictor of blindness at six months. The partial but incomplete visual recovery observed in our patient, followed by stabilization rather than continued improvement, is consistent with the outcomes reported by Anupriya et al., in which only 17% of patients with OCA had improvement in visual acuity at six months while 52% had no further deterioration [4].

A further limitation of this case is the availability of only one MRI examination demonstrating the relevant visual pathway abnormality. Consequently, serial radiological progression or resolution cannot be demonstrated. The diagnosis rests on the combination of the clinical course, microbiologically confirmed TBM, acute visual deterioration after initiation of ATT, and the MRI findings interpreted as OCA. A further limitation is that the individual contributions of corticosteroids, thalidomide, and modification of ATT cannot be separated.

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CONCLUSIONS

Optochiasmatic arachnoiditis is a rare but devastating complication of tuberculous meningitis that can occur paradoxically after initiation of antituberculous therapy. Acute bilateral visual deterioration during treatment should prompt immediate consideration of OCA alongside other causes of optic neuropathy, particularly ethambutol toxicity & raised intracranial pressure. Although the visual prognosis is generally guarded, early recognition and aggressive anti-inflammatory treatment may preserve or restore vision in selected patients. Effective antituberculous therapy should generally be maintained while the inflammatory reaction is treated. Thalidomide may have a role in selected severe cases, but current evidence remains limited to small observational studies and case series.

This case emphasizes the importance of recognizing paradoxical OCA early, particularly when severe visual symptoms develop despite apparently appropriate treatment of TBM.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-.care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

TBM: Tuberculous Meningitis
OCA: Optochiasmatic Arachnoiditis
ATT: Antituberculous Therapy
CSF: Cerebrospinal Fluid
CBNAAT: Cartridge-Based Nucleic Acid Amplification Test
TB: Tuberculosis
IV: Intravenous

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research diretions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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