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CASE REPORT

A Rare Case of Esotropia Induced by Short-Term Use of 0.01% Atropine for Myopia Control

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Abstract: This report a case of intermittent esotropia in an 8-year-old boy with high axial myopia, which manifested within just three months of initiating 0.01% atropine treatment, despite minimal axial elongation. After discontinuation of atropine and introduction of multifocal soft contact lenses, normal binocular visual function was restored. This case emphasizes the importance of monitoring binocular parameters during low-dose atropine treatment for myopia control.

Keywords: LDA, binocular vision, multifocal soft contact lenses, progressive myopia

Introduction

Myopia is a leading cause of visual impairment globally, with increasing prevalence, particularly among children.[1] Low dose atropine 0.01% (LDA), has emerged as an effective intervention to slow axial elongation in progressive myopia while minimizing side effects.[2] Despite its efficacy and safety profile, rare adverse effects such as accommodation dysfunction and binocular vision disturbances have been reported.[3,4] Understanding the full spectrum of LDA-associated complications is critical for clinicians managing pediatric myopia, especially in cases with pre-existing binocular dysfunction or high accommodative demand. We report a rare case of acquired esotropia in a child that was treated with short-term LDA treatment. The esotropia resolved, and fusion was restored in the child after discontinuation of LDA and the use of multifocal soft contact lenses.

Case Report

An 8-year-old boy presented to Metro Eye Care Lumbini with a primary complaint of gradually blurring distance vision in both eyes for the past three months. Upon examination, it was noted that both parents had high myopia. The patient's presenting visual acuity was 20/80 in the right eye (RE) and 20/60 in the left eye (LE) with his previous glasses, prescribed six months earlier (−8.00/-2.00×180; LE −7.00/-1.00×180) . Following both dry and cycloplegic retinoscopy, the new prescription was −10.00/−2.00×180 (20/25) in the right eye and −9.00/−1.00×180 (20/25) in the left eye. With the new prescription, a small exophoria of 2 prism diopters (PD) at near and orthophoria at distance were observed, with other binocular parameters within the normal range. The axial length was recorded as 26.46 mm in the right eye and 26.15 mm in the left eye, reflecting an increase of 0.56 mm in the right eye and 0.48 mm in the left eye compared to measurements taken six months earlier (Figure 1). Due to the significant increase in axial length, treatment with 0.01% atropine sulfate eye drops at night was initiated.

Following a three months of follow-up period, the patient occasionally complained of binocular double vision. The cover test revealed comitant intermittent esotropia in the right eye with poor control, measuring 18 prism diopters (PD) at near and 6 PD at a distance. The accommodative convergence to accommodation (AC/A) ratio, assessed via the gradient method, was high (7:1). Stereoacuity was absent, and uncrossed diplopia at both distance and near vision was observed on the Worth Four Dot test. Although Magnetic Resonance Imaging (MRI) of the brain and orbit was recommended,

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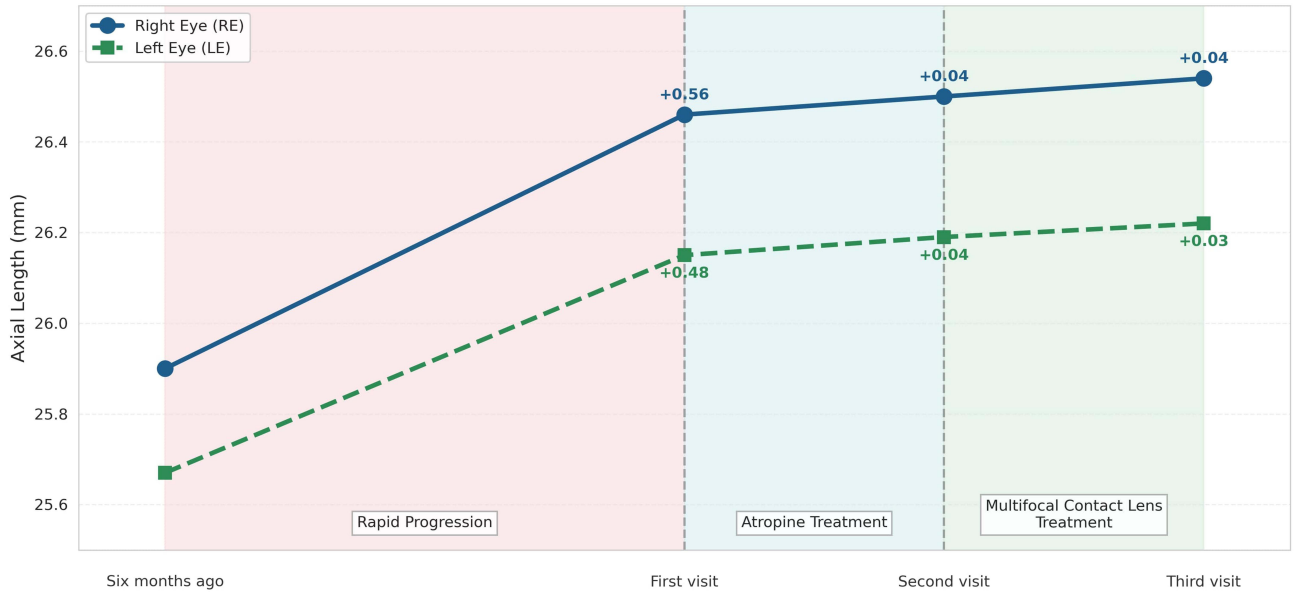

Figure 1 Line chart showing axial length changes in the right eye (RE) and left eye (LE) across three visits: prior to treatment (rapid progression phase), during 0.01% atropine treatment, and after switching to multifocal soft contact lenses.

financial constraints led to the acquisition of a Computed Tomography (CT) scan of the brain and orbit, which revealed no abnormalities. Axial length progression was minimal, measuring 26.50 mm in the right eye and 26.19 mm in the left eye three months after atropine treatment. With the multifocal contact lens (center-distance, +2.50 diopter), the prism bar cover test showed 2 PD esophoria at near and 4 PD exophoria at distance (Figure 2), with a normal AC/A ratio. The multifocal soft contact lenses effectively resolved diplopia and restored binocular fusion. Consequently, atropine treatment was discontinued, and multifocal soft contact lenses were prescribed as an alternative, alongside divergence therapy. Three months post-atropine cessation, axial lengths were 26.54 mm in the right eye and 26.22 mm in the left eye. While wearing multifocal soft contact lenses, prism bar cover testing revealed 2 PD esophoria at near and 4 PD exophoria at distance, with normal binocular vision. Given the stable binocular function and effective control of axial elongation, the multifocal soft contact lenses were continued as part of an ongoing myopia control strategy.

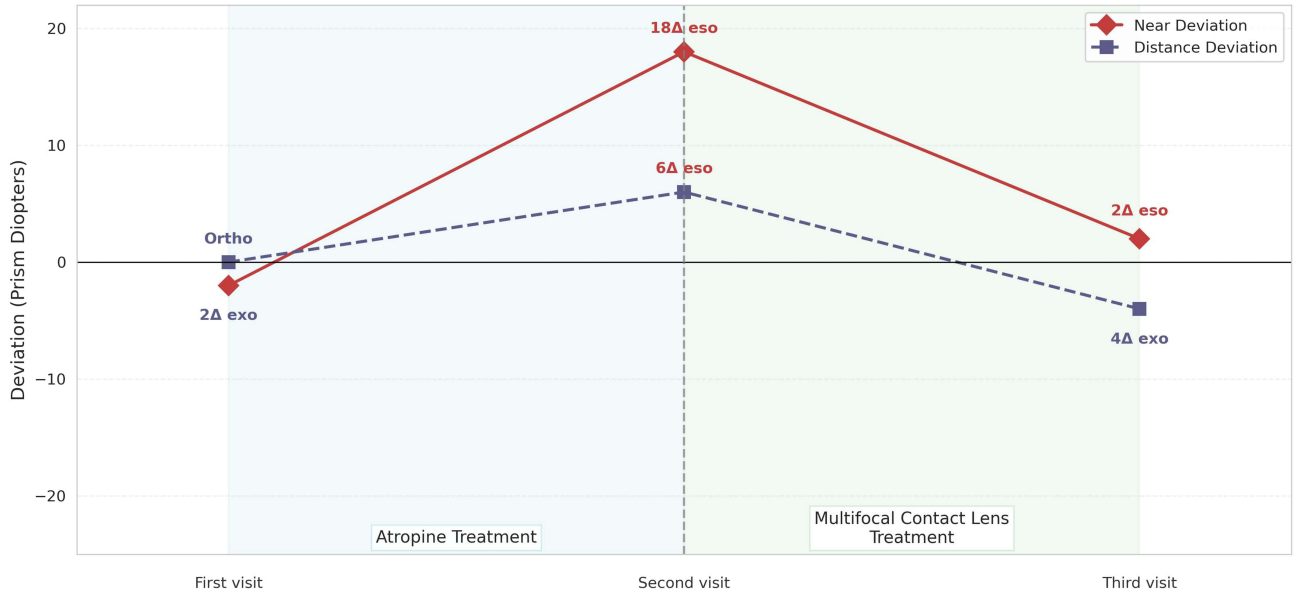

Figure 2 Line chart showing near and distance deviation (in prism diopters) across three visits: initial assessment (orthophoria at distance, small exophoria at near), during 0.01% atropine treatment (development of esodeviation), and after switching to multifocal soft contact lenses (improvement in deviation). Positive values indicate esodeviation; negative values indicate exodeviation.

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Discussion

Atropine sulfate 0.01% has been demonstrated to have minimal side effects in children initiating treatment for progressive myopia.[5] We report a rare complication associated with the short-term use of LDA, wherein an 8-year-old boy developed acquired esotropia during the course of treatment.

Aman and Guyton[4] reported esotropia onset following a gradual increase in atropine concentrations over a six-year period to manage progressive myopia. Similarly, Kothari et al[3] described three patients who developed convergence excess esotropia following the use of 0.01% atropine after undergoing surgery for intermittent exotropia; in two of these cases, esotropia persisted despite discontinuation of atropine, with only partial recovery in accommodation and fusion. Unlike previous cases, our patient had no history of strabismus surgery or an increasing atropine dosage.

A recent report by Englisch et al[6] described a case of reversible esophoria decompensation in a child following an increase in atropine concentration from 0.01% to 0.025%, despite initial tolerance at the lower dose. Their case supports a dose-dependent effect of atropine on binocular function, particularly in patients with convergence excess. Although our case involved esotropia even at 0.01%, both cases highlight the importance of monitoring binocular vision when initiating or adjusting atropine therapy.

Several factors could explain the development of acquired esotropia in our patient. Initially, a child with high myopia developed esotropia, likely due to the combined effects of excessive convergence demand and atropine-induced accommodative lag. With spectacle correction, the high-minus lenses increased the strain by making near objects appear closer.[7] Additionally, the patient's working distance was notably shorter, further contributing to binocular stress. These factors likely triggered the onset of esotropia.

Furthermore, atropine, is a cycloplegic agent, interferes with accommodation. However, LDA may induce only partial cycloplegia, allowing for residual accommodation. This retained accommodative effort can trigger excessive convergence through the AC/A ratio relationship,[8,9] thereby increasing esodeviation.[10] In contrast, full and sustained cycloplegia, such as that achieved with 1% atropine, completely suspends accommodation, eliminating the accommodative component of esotropia, as observed in patients with fully refractive accommodative esotropia.[11]

Multifocal soft contact lenses present an effective alternative to atropine by creating peripheral myopic defocus, which slows axial elongation.[12] Unlike atropine, these lenses preserve normal accommodation and reduce the risk of inducing binocular vision disorders.[13] In our case, the multifocal soft contact lenses restored fusion and reduced esodeviation, highlighting their effectiveness in children with binocular dysfunction or atropine intolerance.

Finally, the anticholinergic effect of atropine partially inhibits peripheral accommodation, which may lead to enhanced central accommodative effort and, consequently, increased accommodative convergence, potentially contributing to the development of esotropia. Esotropia has also been documented with the use of systemic anticholinergics, including scopolamine patches for motion sickness,[14] medications like amitriptyline and oxybutynin for nocturnal enuresis, and haloperidol with benztropine mesylate for Tourette syndrome.[15,16] In most reported cases, patients had underlying neurological conditions or preexisting esophoria, and the esotropia typically resolved after discontinuing the medication.

Conclusion

While LDA is widely regarded as an effective and safe option for controlling progressive myopia, it may trigger esotropia and binocular disruptions. If esodeviation occurs, discontinuing atropine and introducing alternative approaches for myopia control may be necessary to maintain normal binocular function. Close monitoring and assessment of binocular vision parameters are essential to balance effective myopia control and preserve binocular function. Further research is needed to determine the effect of LDA on accommodative convergence in cases of high myopia and to evaluate the impact of alternative approaches, such as multifocal soft contact lenses, bifocal glasses, or progressive addition lenses, in such scenarios.

Ethics and Consent Statement

Formal institutional review board approval was not required to publish the case details at our institution. However, written informed consent was obtained from the patient's parents for the clinical examination, treatment, and publication

of this case report. All clinical procedures were conducted in accordance with institutional ethical standards and the principles outlined in the Declaration of Helsinki. The patient's identity has been anonymized to ensure confidentiality.

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Disclosure

The authors report no conflicts of interest in this work.

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