# Conquering myopia: Have we hit pay dirt?

As school myopia attains epidemic proportions in large parts of urban India,[1] interventions to arrest the progression of myopia are in the spotlight. The causative factors for onset and progression of simple myopia have remained elusive in spite of being the subject of global investigation over the past few decades. Based on epidemiological distribution, it was thought that defects of the accommodation were responsible for the onset and progression. However, efforts to retard the progression of myopia by the use of addition lenses yielded only limited success.[2] From the time, it was discovered that atropine sulfate can effectively retard the progression of myopia; till very recently, it was thought that atropine acted by relaxing accommodation. This was one intervention that proved effective in almost every trial till date. However, the accompanying side effects such as need for additional lenses, photophobia, and high incidence of allergy to atropine prevented clinicians and parents from adopting it as a standard of treatment. Kothari and Rathod have filled the much needed gap by providing evidence that atropine is efficacious in darkly pigmented Indian eyes as well.[3] ATOM 2 conclusively established that atropine was effective in lower concentrations.[4] The efficacy of atropine in retarding axial elongation is dose dependent. Nevertheless, even concentration as low as 0.01%, the effect is clinically significant. At such low concentrations, there is no effect on accommodation, and only a very small number of participants develop a marginally larger pupil which hardly causes any significant intolerance to light. The risk of allergy, however, remains. This also challenged the belief that atropine acted by relaxing accommodation. The mechanism of action of atropine remains a matter of ongoing investigation. It is thought to act through some unknown receptors (possibly M2 subtype on the sclera) to ultimately inhibit scleral growth and hence retard the increase in axial length.[5] This has even been demonstrated in tissue culture studies.

The use of 0.01% atropine to help retard myopia marks a significant change in paradigm. It is quickly gaining popularity among clinicians as they now have an effective and safe intervention to offer. Several issues still need to be addressed. The timing of initiation of therapy is one such issue. While several authors suggest using it in cases where progression in more than 0.75 or at least 0.5 D in 1 year, it may be argued that since myopia does not follow a linear progression, such cutoffs may be all but arbitrary. Some may argue that therapy is instituted at the first diagnosis of myopia as the natural history of the disease suggests likely progression; more so in children who have a strong positive family history. There is a lack of consensus on the duration of therapy as well. ATOM 1 and ATOM 2 showed that myopia rebounds after discontinuation of therapy.[6] Although the risk of rebound is also dose dependent, it does mean that the therapy may need to be continued through childhood and adolescence. How does one, then, determine when to stop therapy? As of now, it is by trial method. Therapy is continued for a couple of years and then discontinued temporarily for a few months. It is restarted at the first hint of rebound. It would be prudent not to discontinue at a time when there is a growth spurt in the child. This may differ for each child, so treatment has to be individualized. The prolonged use of atropine may also have yet unknown side effects on the eye or the individual.

Another important issue is availability of low-dose atropine. Till the time of writing this article, 0.01% atropine eye drop is not available from any manufacturer. Clinicians all over India have to use either 1% atropine eye drops or injection atropine and dilute it to 0.01% concentration with artificial tears. This is a logistic hurdle in a treatment that requires good compliance over a significant period of time. The bioavailability of such formulation is questionable and may not replicate the result of ATOM 2 in spite of good adherence to treatment protocol. We hope this will change very quickly in the near future.

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