THERAPEUTIC EFFECT OF ATROPINE 1% IN CHILDREN WITH LOW MYOPIA

To the Editor: We congratulate Yi and colleagues[1] on their successful use of atropine 1% to control myopia, which is rare in mainland China. However, there are several questions to address. First, the authors indicate that 6 patients (8%) withdrew from the treatment group and 2 (3%) from the control group, but Table 1 indicates that 68 subjects are in the treatment group and 64 in normal group withdrew. Second, there was a reduction of myopia by -0.32 ± 0.22 D in the atropine group; in addition, the axial length was -0.03 ± 0.07 mm compared with baseline after 1 year. Atropine has been much discussed recently and has been reported to effectively control myopia[2]; however, this report suggests that it can even reverse myopia, as indicated by the authors' Figures 2 and 3. These results are surprising, and we wonder how and why this was possible? Third, atropine has been reported to have dose-related side effects, although none were reported in this research: 1% atropine is a relatively high dose, and adverse effects might be expected. Therefore, data on accommodation amplitude, near vision and, low-dose atropine treatment would be helpful.[3] Finally, the authors assert in their discussion that uncorrected distance visual acuity in 35 of 68 patients in the atropine group was ≤0.3 (20/40). These 35 patients should be able to see clearly without glasses for most tasks. However, for children 7-12 years of age, visual acuity ≤0.3 is below normal and glasses may be important. Photophobia could also be a problem requiring the use of photochromatic glasses.

Qianwen Gong, PhD
Longqian Liu, MD, PhD
Department of Optometry
West China Hospital Sichuan University
China

References

1. Yi S, Huang Y, Yu SZ, Chen XJ, Yi H, Zeng XL. Therapeutic effect of atropine 1% in children with low myopia. J AAPOS 2015;19:426-9.
2. Shih KC, Chan TC, Ng AL, et al. Use of atropine for prevention of childhood myopia progression in clinical practice. Eye Contact Lens 2016;42:16-23.
3. Chia A, Lu QS, Tan D. Five-Year clinical trial on atropine for the treatment of myopia 2: myopia control with atropine 0.01% eyedrops. Ophthalmology 2016;123:391-9.

http://dx.doi.org/10.1016/j.jaapos.2016.03.005
J AAPOS 2016;20:379.

1091-8531/$36.00

DENSITY OF THE CRYSTALLINE LENS IN OBESE AND NONOBESE CHILDREN

To the Editor: Acer and colleauges[1] evaluated crystalline lens density in obese and nonobese children by Pentacam HR and found that lens density was higher in obese children than in normal controls in 7- to 14-year-olds but no different in 15- to 18-year-olds. If obesity causes an increase in lens density, we would expect density to increase with age; the absence of difference between obese and normal controls in the older group is interesting.

A strong link between obesity and cataract has been reported in several studies.[2] Osmotic stress, oxidative stress, and nonenzymatic glycation of lens proteins are accepted mechanisms for the development of cataract. Obesity may influence all of these pathologic pathways.[3] Difference in glucose metabolism between obese and nonobese children may be the cause of difference in lens densities. Lenses of obese children are exposed to more glucose than those of nonobese children. This greater exposure may explain the difference between obese children and controls in 7- to 14-year-olds. During puberty, increase in hormone levels may affect glucose metabolism. Growth hormone levels rise during puberty and increase blood glucose levels. Hyperinsulinemia is a characteristic finding of obesity. The effects of growth hormone may counterbalanced by insulin in obese chilren.[4] As a result of this situation, both lenses of obese and nonobese children may be exposed to the same levels of glucose during puberty. While 15-18 years does not exactly correspond to puberty, the relationship between growth hormone and insulin may explain the absence of difference in the older group.

Abdullah Kaya
Department of Ophthalmology
Anıttepe Military Dispensary
Ankara, Turkey

References

1. Acer S, Ağladıoğlu SY, Pekel G, Özhan B, Çetin EN, Yağcı R, Yıldırım C. Density of the crystalline lens in obese and nonobese children. J AAPOS 2016;20:54-7.
2. Cheung N, Wong TY. Obesity and eye diseases. Surv Ophthalmol 2007;52:180-95.
3. Serrano Rios M. Relationship between obesity and the increased risk of major complications in non-insulin-dependent diabetes mellitus. Eur J Clin Invest 1998;28(Suppl 2):14-7.
4. Møller N, Jørgensen JO, Abildgård N, Orskov L, Schmitz O, Christiansen JS. Effects of growth hormone on glucose metabolism. Horm Res 1991;36(Suppl 1):32-5.

http://dx.doi.org/10.1016/j.jaapos.2016.03.011
J AAPOS 2016;20:379.

1091-8531/$36.00

REPLY

We would like to thank Dr. Kaya for his interest in our article. He points out that obesity may affect several pathologic pathways of cataract formation. We presented increased lens density levels in obese children compared with nonobese children.[1] There was a positive correlation

between lens density and BMI, age, and the pubertal stage. However, in our subgroup analysis according to age, although the lens density levels were significantly higher in children under the age of 15 years, the difference was not significant in children over the age of 15 in the obese subgroups compared with the controls.[1] This outcome might be occurred due to the low number of participants in the subgroups.

Dr. Kaya suggests that the increased lens density in obese children younger than 15 years may be the result of high glucose exposure of the lenses. Nevertheless, increased insulin and growth hormone levels during puberty may balance blood glucose levels, resulting in normal lens density in older children. We do not know whether the lenses of obese children are exposed to more glucose than those of nonobese children younger than 15 years. We excluded diabetic children, and the blood glucose levels of all of the children were within normal limits in our study groups.[1] It is true that growth hormone levels increase during puberty and that the growth hormone increases blood glucose levels[2-3]; however, the blood glucose levels are within normal limits in non diabetic children during puberty.[4] Hyperinsulinemia is an often finding of childhood obesity.[5] We reanalyzed some of our findings according to Dr. Kaya's suggestions. When we compared all the children in terms of hyperinsulinemia, there was no significant difference in lens density measurements between the children with and without hyperinsulinemia ($P > 0.05$). We agree that levels of growth hormone or some other hormones, such as sex hormones, may affect the lens density in obese children. In addition, many other factors such as nutritional status, prematurity, dehydration, and accommodation problems may affect lens density of children. However, we have no data about hormone levels other than for insulin in our study; therefore, we are not able to explain the changes in lens density in children in our study with the association between insulin levels and the growth hormone or other hormones.

Semra Acer, MD
Gökhan Pekel, MD
Ebru Nevin Çetin, MD
Ramazan Yağcı, MD
Cem Yıldırım, MD
Department of Ophthalmology
Pamukkale University
Kinikli Kampusu, Denizli, Turkey

Sebahat Yılmaz Ağladıoğlu, MD
Bayram Özhan, MD
Department of Pediatric Endrocrinology
Pamukkale University, Kinikli Kampusu
Denizli, Turkey

References

1. Acer S, Ağladıoğlu SY, Pekel G, et al. Density of the crystalline lens in obese and nonobese children. J AAPOS 2016;20:54-7.
2. Rose SR, Municchi G, Barnes KM, et al. Spontaneous growth hormone secretion ıncreases during puberty in normal girls and boys. J Clin Endocrinol Metab 1991;73:428-35.
3. Møller N, Jørgensen JO. Effects of growth hormone on glucose, lipid, and protein metabolism in human subjects. Endocr Rev 2009;30:152-77.
4. Caprio S, Plewe G, Diamond MP, et al. Increased insulin secretion in puberty: a compensatory response to reductions in insulin sensitivity. J Pediatr 1989;114:963-7.
5. Felszeghy E, Juhasz E, Kaposzta R, Ilyes I. Alterations of glucoregulation in childhood obesity—association with insulin resistance and hyperinsulinemia. J Pediatr Endocrinol Metab 2008;21:847-53.

http://dx.doi.org/10.1016/j.jaapos.2016.07.216
J AAPOS 2016;20:379-380.

1091-8531/$36.00