·Letter to the Editor·

Abruptly developed large esotropia after full time occlusion therapy in anisometropic amblyopia

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Dear Sir,

I am Dr. Huijae Lee, from the Seoul Ire Eye Clinic, Seoul, Korea. I write to present a case report of esotropia after occlusion therapy.

Amblyopia can be caused by deprivation of vision early in life by vision-obstructing disorders such as congenital cataracts, strabismus, or anisometropia. If amblyopia is diagnosed in one eye, occlusion treatment in the other eye should be commenced without delay. Although occlusion therapy is an essential method for treatment of amblyopia, occlusion therapy can disrupt certain peripheral fusion, which may control a latent component of the existing strabismus [1]. Although some authors have contended that occlusion therapy might have an effect on strabismus, others contend that occlusion therapy might not have an effect on strabismus [2-5].

Most deteriorated esotropia cases are related with accommodative esotropia or partially accommodative esotropia, however, deteriorated esotropia without accommodative component has not been reported in the English literature. We report on one case of abruptly developed a large esotropia combined with superior oblique muscle palsy after full time occlusion therapy in anisometropic amblyopia.

An 8-year-old girl complained of abruptly developed esodeviation and diplopia after occlusion therapy. She had received full time occlusion therapy for anisometropic amblyopia in the left eye about 13 months ago. A large esodeviation developed suddenly at 5 months after full time occlusion. She was then referred for esotropia and anisometropic amblyopia. She had no history of trauma, abnormal head turn, or facial asymmetry, and her old photograph, which was taken before occlusion therapy, showed an orthotropic figure.

A full ophthalmologic examination was performed. Her best corrected visual acuity was 20/25 in the right eye and 20/20 in the left eye, and cycloplegic refraction was -2.00+3.25 95° in the right eye and +1.00 +0.50 85° in the left eye. Alternative cover test revealed 30 prism diopters of esotropia combined with 6 prism diopters of hypertropia in the left eye at distance and 40 prism diopters of esotropia was found at near. In the both lateral gazes, the 30 prism diopter of esotropia was noted (Figure 1). The stereopsis tests including Worth 4 dot test and Titmus test showed no stereopsis.

Imaging studies and brain magnetic resonance imaging showed no abnormal findings. At 6 months after the first visit, 40 prism diopters of esotropia was not changed. Under the general anesthesia, the forced duction test showed no restriction and laxity of the extraocular muscles. We performed the bilateral 4.5mm medial recuts recession. At 8 months after surgery, 4 prism diopter esophoria was showed. She had no stereopsis using by Titmus test, however, Worth 4 dot test showed normal finding without suppression.

Occlusion treatment was useful in amblyopia and strabismus. However, full time occlusion therapy might disrupt peripheral fusion and induce strabismus and have an influence on the amount of the preexisting deviation angle.

Holbach *et al* [6] have indicated that change of more than 5 prism diopters is 57%-65% in esotropia patients. Charney and Morris [2] reported increased esodeviation of 10 to 43 prism diopters after full time occlusion therapy. Kim *et al*[1] reported on 2 cases of suddenly deteriorated accommodative esotropia with amblyopia during part-time occlusion therapy; 21% of their patients under occlusion therapy showed changes of five or more prism diopters in the esodeviation at distance fixation [3]. Although most of deteriorated esotropia was accompanied with accommodative factor, the present case did not have hyperopia more than +2 diopter.

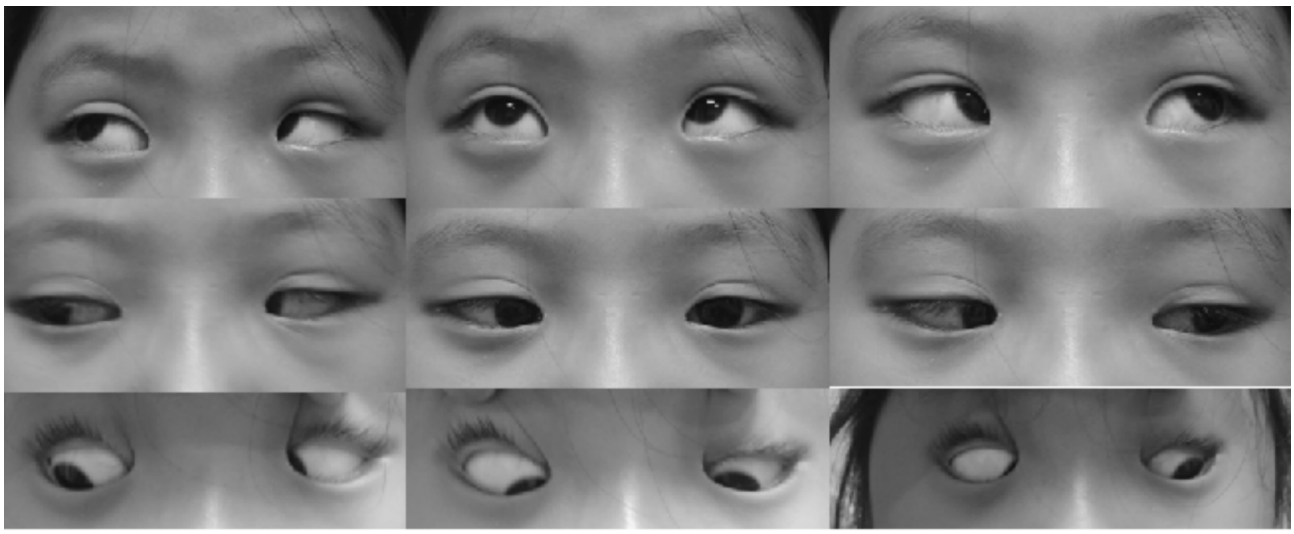

Figure 1 Nine gaze photograph The patient shows large esotropia at primary position and superior oblique muscle underaction in the left eye (lower left).

In our case, the patient had nearly orthotropia prior to occlusion treatment; however, she showed large angle esodeviation after full time occlusion therapy, which seems to presume that occlusion therapy might disrupt peripheral fusion, and her large esotropia was revealed. In postoperative state, she revealed a peripheral stereopsis because there are normal findings in the Worth 4 dot test. Thus, there might be a large esotropia with peripheral fusion before the occlusion therapy. In conclusion, we should evaluate the stereopsis and fusional ability before a full time occlusion that could result in a disruption of fusion.

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