Case Report

Acute Acquired Comitant Esotropia in Adults; a Case Report

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Abstract

Purpose: The aim of this manuscript was to report the clinical characteristics of two patients suffering from acute acquired comitant esotropia presented in adulthood.

Cases Report: Both patients reported intermittent diplopia especially during car driving before their diplopia becoming constant. They had 20/20 uncorrected visual acuity and a cyclorefraction of + 0.50 D with normal extraocular function except very mild lateral rectus underaction (< 1) in their both eyes. In alternate prism cover test, the manifest esotropia of 25 - 30 Δ was measured at both far and near distances. They did not show any signs of neurological disorders such as marked extraocular underaction or different esotropia on lateral gazes compared with esotropia in primary position. No abnormality was found on electromyography, visual field measurements and magnetic resonance imaging for both cases.

Conclusion: Based on our clinical assessment, hereditary causes, mild lateral rectus underaction or inability to compensate esotropia due to decreased divergence amplitude and fusion could be considered as the probable risk factors for acute acquired comitant esotropia in adults, although the actual etiology of this disease has not been determined.

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Introuduction

Acute acquired comitant esotropia (AACE) in adults is a rare form of esotropia that presents with recent onset diplopia and can be intermittent at beginning and become constant at later phases [1]. Patient tries to eliminate diplopia with his/her face turn. These patients usually have low refractive errors with good visual acuity. Their version and duction ocular movements are within normal range with no A or V pattern. Also their far and near deviation show no significant difference [2]. They do not report any history of trauma, VI nerve palsy, age related esotropia, divergence palsy, decompensated accommodative esotropia and intracranial lesions, intracranial hypertension or systemic diseases such as thyroid ophthalmopathy, myasthenia gravis, multiple sclerosis, diabetes and high blood pressure, which are considered as the etiologic factors for esotropia in adults [3]. Prescription of prism glasses, botulinum toxin injection or surgical management are suggested for these patients [1]. In this manuscript, we report the characteristics of two patients with AACE who were referred to our clinic.

Case Report

Case I

A 28-year-old female was referred to our clinic due to the comitant esotropia of her left eye since 2 years ago accompanied with horizontal binocular diplopia. She did not report any history of recent head trauma and strabismus in her first degree family members. Cycloplegic refraction of both eyes showed mild hyperopia [+ 0. 50 dioptres (D)] and the presenting visual acuity of both eyes were 20/20. She had a small face turn to compensate her diplopia when fixated to the target especially at far, which resolved by wearing her glasses (Figures 1 A and B). Stereopsis was 40 seconds of arc when patient had her face turned. Furthermore, the function of each extraocular muscle was evaluated in 9 cardinal visual gazes, which was normal with exception of a mild limitation (< 1) for the left lateral rectus on abduction (Figure 2).

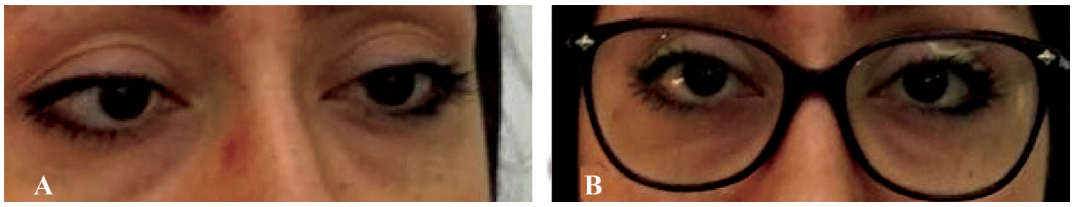

Figure 1. (A) Abnormal head posture of Case I. (B) This resolved completely after wearing glasses with base out prism.

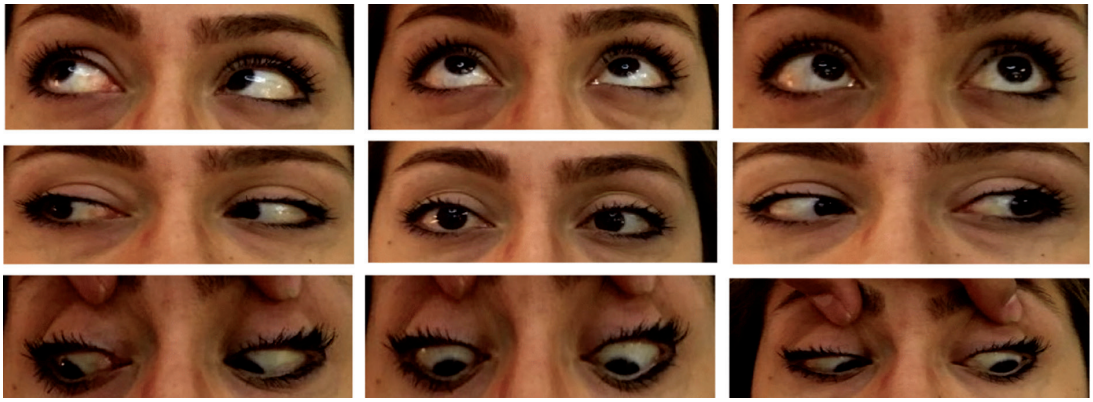

Figure 2. Function of extraocular muscles of case I in different visual gazes with no limitation.

The left esotropia was measured 25 Δ and 30 Δ with and without her presenting glasses, respectively. The patient did not report double vision when the total magnitude of deviation was placed in front of the deviating eye.

There was not any significant difference in measurement of deviation angle between the up and downward visual gazes (no pattern). There was not any pathologic lesion in examination of the anterior and posterior ocular segments and the intraocular pressure (IOP) was 10 mmHg in both eyes. In spite of comprehensive examinations, no specific cause was found to justify her recent onset esotropia. In addition, the patient did not complain of systemic diseases such as diabetes mellitus, hypertension, and general health problems. Visual field was recorded as normal for both eyes and measurement of ocular deviation on deviometry was the same as alternate prism cover test results.

She had a history of 10 sessions of orthoptic exercises for improvement of divergence fusional reserve, but finally she had achieved 18 Δ positive fusional vergence (PFV) and only 4 Δ negative fusional vergence (NFV).

There was no sign of pathologic lesions in her brain magnetic resonance imagination (MRI).

Her first neurological consultation suggested treatment of myasthenia gravis by neostigmine tablet for 6 months, but patient's signs and symptoms returned following the discontinuing of treatment.

Eventually it was suggested for patient to undergo strabismus surgery to resolve her ocular deviation using left medial rectus recession, since she was not eager to wear her prism glasses due to the cosmetic considerations. Seven months after the surgery, her deviation was left esotropia of 10 Δ and esophoria of 4 - 6 Δ, with no glasses at far and near fixation distances, respectively (Figure 3). She had no deviation when she wear her glasses with base out prism.

At last visit before her operation, her father accompanied her to the office and we saw that he had esotropia in his left eye and reported that he also has amblyopia, while his daughter had not mentioned it before. Therefore, we concluded she had a hereditary esophoria, which became manifest with increasing age since two years ago due to the disruption of the binocular fusion.

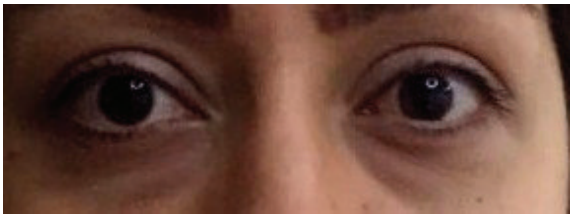

Figure 3. Ocular alignment with no strabismus at seven months after the surgery.

Case II

The patient was a 50-year-old man who was referred to our clinic in 2013 with the chief complains of diplopia and abnormal head posture since one year ago. He had no history of diabetes, high blood pressure, cardiovascular diseases, or ocular deviation among his family members. He was a smoker for 10 years.

BCVA was 20/20 with a cyclorefraction of + 0.50 D in both eyes. His diplopia was resolved when he put on a 12 Δ base out prism on each eye (total of 24 Δ base out (BO)). Extraocular muscle examination showed left medial rectus overaction (+ 1) and bilateral mild underaction of lateral rectus (< 1). Alternative prism cover test revealed esotropia of 22 Δ and 25 Δ in his left eye at far and near fixation distances, respectively with no pattern.

Anterior segment, IOP and posterior segment exams were within normal limits, except for tortuous fundus vessels. He reported a diplopic response (2 red and 3 green targets) in Worth 4 dot test assessment.

All laboratory findings were within normal limits except high hemoglobin and cholesterol levels

(Hb: 16.7 g/dl, cholesterol: 262 mg/dl). His neurologic consultation suggested taking a course of oral neostigmine and prednisolone to rule out the probable diagnosis of myasthenia gravis. It was not curative, therefore the patient discontinued this treatment. Furthermore, visual field, electromyography and MRI were within normal limits. He underwent left medial rectus recession of 5 mm on July, 2014. Left esotropia was measured as 6 Δ and 12 Δ at follow-ups of 2 and 5 months after strabismus surgery, respectively. His complain of diplopia and right face turn were significantly reduced, but we prescribed him a total prism of 6 Δ BO (3 Δ BO for each eye) to eliminate the remained diplopia and head posture at the last visit. His ocular deviation was measured as left esotropia of 4 Δ with prism glasses without any symptom of diplopia at his last visit.

Discussion

AACE is a rare condition with no extraocular dysfunction, difference between far and near esotropia, and any pattern or neurological disorders. Visual acuity is usually good with low refractive error of hyperopia or myopia. It begins with acute diplopia or after some period of intermittent diplopia before it becomes permanent [1]. Our patients experienced intermittent diplopia especially during car driving before their constant diplopia. They had normal extraocular function except very mild lateral rectus underaction (< 1) in their both eyes. They did not show any signs of neurological disorders such as marked extraocular underaction or different esotropia on lateral gazes compared with esotropia in primary position. Both cases had good visual acuity bilaterally and no abnormality was found on electromyography, visual field exam and MRI. Different etiologies have been suggested for AACE. It may appear after patching of one eye, especially among children who are treated for amblyopia, or it may be a result of decompensated accommodative esotropia or monofixation syndrome [4]. Sometimes it may be observed in myasthenia gravis, tumours and idiopathic intracranial hypertension, diabetes mellitus, demyelinating diseases and high blood pressure [1-5]. In addition, Lee et al., believe that the excessive use of smart phone might be causative in development of AACE in adolescence [6]. Although different possible risk factors are being considred for AACE, investigation of the central nervous system is highly recommended due to the possible association of the underlying neural disorders with AACE [7]. In our cases, none of these etiologies were present, and they might have decompensated monofixation syndrome. The father of our first case had esotropia and his left eye was amblyopic, which may lead us to hereditary origin of her esotropia. Both cases showed mild lateral rectus underaction on their both sides and intermittent beginning of their diplopia, which reveals their ability to increase divergence amplitude and fusion to resist with ocular deviation, before breaking and fatigue of fusion, and appearance of tropia and diplopia. The question is what factors cause this breaking of fusion. It might be due to older age, constant need to accommodate for low hyperopia, divergence palsy, stress or systemic diseases; which should be addressed in future studies.

Conclusion

Based on our clinical assessment, hereditary causes, mild lateral rectus underaction or inability to compensate esotropia due to decreased divergence amplitude and fusion could be considered as the probable risk factors for acute acquired comitant esotropia in adults, although the actual etiology of this disease has not been determined.

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Footnotes and Financial Disclosures

Conflict of Interest:

The authors declare no conflict of interest with the subject matter of the present manuscript.