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Comparison between eyelid indices of ptotic eye and normal fellow eye in patients with unilateral congenital ptosis



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KEYWORDS

Ptosis;
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Summary Purpose: To evaluate the relationship between levator muscle function (LMF) and other eyelid parameters in the normal and affected eyes of patients with unilateral congenital ptosis.

Methods: This study includes subjects with unilateral congenital upper lid ptosis who were referred for operation over a 2-year period. Patients with other eyelid abnormalities and previous eyelid surgery were excluded. Eyelid parameters including LMF, lid fissure height (LFH) and margin reflex distance (MRD) were measured in both eyes and analyzed.

Results: A total of 77 patients with mean age of 26.4 ± 16.4 years were enrolled in the study. Mean LMF was 8.3 ± 4.6 mm in the ptotic and 13.1 ± 3.6 mm in the normal fellow eyes. Each millimeter of difference in LMF was associated with 0.30 mm of difference in LFH (95% CI: 0.25–0.35, $P < 0.001$) and 0.11 mm of difference in MRD of the ptotic eyes (95% CI: 0.08–0.12, $P < 0.001$) in the same direction. In addition, each millimeter of difference in LMF of ptotic eyes was associated with 0.48 mm of difference (95% CI: 0.33–0.62, $P < 0.001$) in LMF of non-ptotic eyes in the same direction.

Conclusion: A direct correlation was observed between LMF, and LFH and MRD in ptotic eyes which confirms the role of levator muscle dysfunction in the development of congenital ptosis and its severity. Furthermore, a direct correlation was also present between LMF of ptotic and non-ptotic eyes suggesting possible bilateral involvement in apparently unilateral congenital ptosis.

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Introduction

Congenital ptosis is generally recognized to be the consequence of myopathic defects in the levator muscle, however there are controversies about the nature of these defects.^{1,2} Measurement of eyelid parameters is an essential part for evaluation of patients with blepharoptosis. These parameters are not confined to, but consist lid fissure height (LFH), upper lid margin to corneal light reflex distance (MRD) and levator muscle function (LMF). LFH and MRD are considered as indicators of the severity of ptosis while LMF is a measure of maximal lid excursion and crucial for selecting the best surgical approach.

Structural abnormalities in the levator muscle give rise to reduced LMF and eventually clinical upper lid ptosis. However, no relationship has been reported between LMF and other eyelid ptosis indices in congenital ptosis.^{3,4}

This study aims to define a quantitative correlation between eyelid parameters and LMF in unilateral congenital ptosis. In addition we compared these parameters between ptotic and contralateral normal eyelids in this particular group of patients.

Material & methods

Over a two year period, (from April 2009 to April 2011) patients with unilateral congenital ptosis who were referred for surgery to our clinic were enrolled in this study. Subjects who were at least 4 years of age and cooperative for eyelid evaluation tests were enrolled. Patients with systemic conditions such as diabetes mellitus, thyroid disease and rheumatologic disorders, and subjects with previous eye surgery including eyelid surgery were excluded from the study. Patients with any eyelid disorder other than ptosis (e.g. lid deformity, ectropion, entropion and trichiasis) were also excluded.

The research adhered to the tenets of the Declaration of Helsinki. All patients were informed about the nature of the study and associated procedures. The study was approved by the Ethics Committee of the Ophthalmic Research Center, Shahid Beheshti University of Medical Sciences.

All subjects underwent a full ophthalmic examination. Eyelid parameters including LFH, MRD and LMF as well as presence and intensity of Bell's reflex and tear film status were documented for each eye. All measurements were performed by one of the authors (HN) while the frontalis power were neutralized by headband of the slit lamp.

Data analysis was performed with SPSS software employing the Wilcoxon signed rank test and the generalized estimating equation (GEE) method. P values less than 0.05 were considered statistically significant.

Results

Seventy-seven patients including 41 (53.2%) male and 36 (46.8%) female subjects with mean age of 26.4 ± 16.4 (range 4–66) years were enrolled for the purpose of the study. The left eye was involved in 45 (58.4%) patients. All represented data are measured before any surgical intervention.

The mean LMF was 8.3 ± 4.6 mm in the ptotic eyelids versus 13.1 ± 3.6 mm in the normal fellow eyes ($P < 0.001$, Wilcoxon signed rank test, Figure 1). Linear regression analysis demonstrated that each millimeter of difference in LMF in ptotic eyes was associated with a 0.48 mm difference in LMF of non-ptotic eyes in the same direction (95% CI: 0.33–0.62 mm, $P < 0.001$, Figure 2). The mean ratio of LMF in non-ptotic to ptotic eyes was 2.23 ± 1.8 (median: 1.54, range: 1.0–13.0).

The mean LFH was 7.4 ± 1.5 mm (range 4–13) in ptotic and 10.8 ± 1.6 mm (range 6–15) in normal fellow eyes. A direct relationship was observed between LMF of the ptotic eyes and LFH of both affected and normal fellow eyes. GEE analysis showed that each millimeter of difference in LMF was associated with 0.1 mm of difference in LFH in the ptotic eye (95% CI: 0.06–0.20 mm, $P < 0.001$) and 0.2 mm of difference in LFH in normal fellow eye in the same direction (95% CI 0.13–0.30 mm, $P < 0.001$) (Figure 3).

The mean MRD was 1.2 ± 1.0 (range 0–4) versus 4.5 ± 0.9 (range 2–7) millimeters in ptotic and normal fellow eyes, respectively. A direct relationship was observed between LMF of the ptotic eyes and MRD of both affected and normal fellow eyes. GEE analysis revealed that each millimeter of difference in LMF was associated with 0.06 mm of difference in preoperative MRD in the ptotic eyes (95% CI: 0.04–0.14 mm, $P = 0.001$) and 0.09 mm of difference in MRD in the normal fellow eyes in the same direction (95% CI: 0.04–0.14 mm, $P = 0.008$, Figure 4).

Discussion

Based on the results of the current study, LMF is directly proportionate with MRD and LFH in ptotic eyes. This relationship confirms the role of levator muscle dysfunction in the severity of eyelid drooping in patients with congenital ptosis. Several histopathologic studies have demonstrated abnormalities of levator muscle in congenital ptosis which have been described as dystrophy versus degeneration by different authors.^{5–12} Most studies advocating muscle abnormalities in congenital ptosis to be a kind of dystrophy were published back in the 1960s and 1970s. Dystrophic changes were detected by light and electron microscopes and included severe atrophy of muscular fibers, irregularities of residual fibers, loss of cross striations and sarcolemmal retraction.^{5–8} Nevertheless, true muscle dystrophies (e.g. Duchene dystrophy) usually demonstrate an inheritance pattern and a progressive course, both of which are absent in congenital ptosis.^{9,10} Later theories believed that a developmental defect might result in structural abnormalities of the levator muscle.^{11,12} Iljin and colleagues showed that the severity of histopathological changes is correlated with levator muscle function and clinical severity of blepharoptosis.¹³ In patients with minimal ptosis, subtle pathologic findings were detected including proliferation of collagen fibers while in more severe clinical ptosis, extensive histopathological changes consisting of mitochondrial loss, cytoplasmic thinning, and areas of homogenous fibers were reported.^{11,13} Preira and colleagues demonstrated an association between levator muscle function and severity of blepharoptosis in involutional ptosis, a similar finding to the present study on

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