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# Corneal hydrops induced by Bell's paralysis in a case of corneal ectasia

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#### ABSTRACT

An 18-year-old male patient presented with suddenly decreased vision, itching, corneal edema and an inability to close the left eye. He had left Bell's paralysis for two weeks and used high diopter glasses for five years. The best corrected visual acuity was 0.4 in his right eye and counting fingers in the left eye. Biomicroscopic examination revealed thinning and steepening of the cornea in the right eye and anterior protrusion of the cornea, stromal edema and punctate disruption of the epithelium in the left eye. Topographic image of the right eye was consistent with keratoconus. Six months later, stromal edema gradually regressed and a corneal scar ensued. This case presentation emphasizes that Bell's palsy may induce disease progression in a patient with preexisting corneal ectasia and results in corneal hydrops.

Key words: Corneal ectasia, facial paralysis, corneal hydrops

## Introduction

Keratoconus is a bilateral, progressive condition and presentation may be asymmetric [1]. In 3% of keratoconus patients, acute corneal hydrops may occur where an abrupt rupture of Descemet's membrane results in acute overhydration of the cornea and accumulation of fluid lakes within the corneal stroma. Over time, endothelial cells spread over the posterior stromal defect to lay down new Descemet's membrane and recompensate the cornea [1,2]. Unilateral corneal ectasia after Bell's palsy has been reported with a case report in the literature [3]. In the present case, we diagnosed left corneal hydrops following Bell's palsy with pre-existing corneal ectasia. The aim of this case presentation was to emphasize that Bell's palsy can cause the progression of pre-existing ectasia and may result in corneal hydrops.

### **Case Presentation**

An 18-year-old male patient presented with suddenly decreased vision, itching, corneal edema and inability to close the left eye (Figure 1). In terms of medical history, he had left Bell's paralysis for two weeks and used high diopter glasses for five years. Upon ophthalmological examination, the best corrected visual acuity was 0.4 in his right eye and counting fingers in the left eye. Biomicroscopic examination revealed thinning and steepening of the cornea in the right eye and anterior protrusion of the cornea, stromal edema and punctate disruption of the epithelium in the left eye (Figure 2). Topographic image (Pentacam High Resolution, Oculus, Wetzlar, Germany) was provided for strictly the right eye and it showed thinning and steepening of the corneal apex marked inferiorly, consistent with

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Figure 1. Left facial paralysis.



Figure 2. Corneal hydrops in the left eye.



Figure 3. Topographic image of the right eye.

keratoconus (Figure 3). The patient developed corneal hydrops in the left eye following Bell's paralysis. Six months later, stromal edema gradually regressed and a corneal scar ensued.

#### Corneal hydrops in Bell's paralysis

#### Discussion

The etiology of the keratoconus is multifactorial with both genetic and environmental factors playing a role, but initiating the structural disruption and the underlying biochemical processes are not clear [1]. In addition, whether the onset mechanism triggering the disease and the mechanism playing a role in its progression is the same or not remains unknown. Moreover, it is known that irritation, such as eye rubbing, wearing contact lenses, dryness, ultraviolet radiation and atopy, accelerate keratoconus progression [2]. Several studies have emphasized that chronic habit of abnormal eye rubbing cause both corneal curvature asymmetry in the normal cornea and unilateral ectatic progression that brings about hydrops [2,4]. The enzyme and proteinase inhibitor abnormalities responsible for the occurrence of the disease are most prominent in the epithelial layer of the cornea and the basic defect in keratoconus is suggested that may reside in the epithelium [4]. In the present case, mechanical epithelial trauma, triggering a woundhealing response and its interaction with the stroma, may have led to progression of the ectasia, as in eye rubbing.

Unilateral peripheral corneal ectasia was developed after Bell's palsy in a case without prior ectasia [3]. The author noted that the patient worked in an environment where there was abundant dust and particles, and irritation and severe rubbing resulted in unilateral ectasia. Bell's palsy may be associated with initiation and progression of ectasia in certain regards. An injured 7<sup>th</sup> nerve cause may lagophthalmos that causes an inability to close the eye. Another possible effect was nerve paralysis of the decreased lacrimation as parasympathetic fibers are carried by that nerve. Both conditions may contribute to the development of dry eye and exposure keratopathy. Fifth nerve paralysis was also associated with keratoconus progression, a cause of neurotrophic keratopathy, a situation similar to exposure keratopathy in a number of ways [5-7]. Such examples emphasize the importance of epithelial trauma in the pathophysiology and progression of the keratoconus. The exposure keratopathy induced by Bell's palsy in our case may be responsible for the progression of the disease and corneal hydrops.

## Conclusion

Certain structural and environmental factors are responsible for the progression of keratoconus. The structural impairment triggering ectasia and the mechanism leading to progression of the disease may be different. Bell's palsy may not only cause ectasia in an intact eye but may also accelerate disease progression in an eye with keratoconus and result in corneal hydrops.

## **Conflict of interest statement**

The authors have no conflicts of interest to declare. **References** 

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