INTRODUCTION

Brown-McLean syndrome is a clinical condition with corneal oedema involving the peripheral 2-3 mm of cornea, first described in 1969. The oedema typically starts inferiorly and progresses circumferentially, but spares the central portion of the cornea. In most cases, fine pigment is deposited on endothelium in the involved areas. Endothelial cell density may be decreased. The cause of Brown-McLean syndrome is still unexplained. It is possible that it could develop in eyes with a genetic predisposition when these eyes are exposed to certain conditions, such as intracapsular cataract extraction. It may also occur after extracapsular cataract extraction, phacoemulsification or pars plana lensectomy and vitrectomy. The onset of this condition since the time of procedure usually ranges from 6 to 16 years, having been described even 34 years later. Surgical complications and multiple intraocular procedures are frequently observed in these patients. Less frequently, Brown-McLean syndrome can occur in eyes that have not had surgery. The present article describes a case of Brown-McLean syndrome with keratoconus.

CASE REPORT

A 30 years old lady presented to the outpatient department with reduced vision in her right eye. It was gradual in onset. The problem was persistent and worsening over the last 2 years. There was no pain or any discharge from the eye. There was no history of surgery or trauma to the eye. The patient was advised to use glasses by some optician but after few months she felt no improvement with spectacles. Patient consulted a local doctor for her problem and she was given some eye drops. The treatment did not work and patient continued having reduction in vision. She was otherwise healthy. She had no previous history of ocular or any systemic illness. She was a married woman with 2 daughters. There was no history of ocular diseases in her family. The patient belonged to the middle-income class.

On examination, she was a young lady of average built who was well oriented. Her visual acuity was 6/60 in the right eye and 6/9 in the left eye. Her right eye best corrected visual acuity was 6/18 with +4.00 diopters sphere. Her left eye showed no improvement on refraction. Her pericentral and adnexal examination was normal. Extraocular muscles were unrestricted in all gazes. Pupils were equal in size, round, reacting to the light and accommodation, with no relative afferent pupillary defect. Irides were deep brown in color. On slit lamp examination there was mild conjunctival injection of bulbar conjunctiva in her right eye. The peripheral corneal of the right eye was hazy. There was peripheral 360 degrees corneal oedema extending 2.5-3.5 mm from the limbus (Figure 1). The central cornea was spared. Fine microcysts were noted in the epithelium of the involved segment. There was thickening of the stoma and there were prominent descemet's folds (Figure 2). Fine pigmentation was noted on the endothelium of the involved area. The central cornea was transparent and normal. The left limbus was normal. Both eyes’ anterior chamber were deep and quite. Both eyes were phakic with clear crystalline lenses. Intraocular pressures were 12 mmHg in both eyes. Both eyes had normal vitreous and retina.

The differential diagnoses considered in that case include marginal keratopathy related to collagen vascular disease, Endotheliatis and Fuchs endothelial dystrophy. The laboratory workup for collagen vascular disease was negative. Endotheliatis was ruled out by the
peripheral concentric pattern and absence of anterior chamber reaction. Fuchs endothelial dystrophy was ruled out by the young age, unilaterality and sparing of the central cornea.

Right central corneal thickness on pachymetry was 510µ while peripheral corneal thickness was 650µ. Left central corneal thickness was 478µ while peripheral corneal thickness was 500µ. Right corneal endothelial specular microscopy showed count of 2015/mm² with 51% hexagonality, while left eye specular microscopic endothelial cell count was 2610/mm² with 56% hexagonality. Corneal topography was performed and it revealed bilateral keratoconus (Figure 3).

The patient was advised to use hypertonic saline eye drops 4 times in a day. For best visual functions she was advised to use rigid gas permeable contact lenses.

It is planned to follow-up the patient for progression of corneal oedema and keratoconus.

**DISCUSSION**

Brown-McLean syndrome is a rare pathology described at the end of 1960’s, progressing with a corneal oedema that extends itself to the corneoscleral limbus (2 to 3 mm) and typically not involving the central visual axis. It may be associated with orange or brownish endothelial pigmentation with iridodonesis, appearing more frequently in eyes with intraocular lens in anterior chamber. When the symptoms appear, a foreign body feeling and epithelial defects usually accompany them.

Its origin is uncertain. It has been linked to several types of lens surgery such as the extracapsular cataract extraction, phacoemulsification, pars plana vitrectomy, the extracapsular cataract extraction being the most frequent of all, although it has been also described in the absence of such surgical procedures. Brown-McLean syndrome has been described in cases of spontaneous re-absorption of the lens, lens subluxation, glaucoma due to angle closure and in one case of myotonic dystrophy. Generally, when symptoms appear, they are treated with hypertonic saline, lubrication and in some cases by stromal puncture.

In the present case, the patient had all the clinical features of Brown-McLean syndrome without any history of trauma or surgery. The etiologies of both the keratoconus and Brown-McLean syndrome remain uncertain; it seems reasonable to assume that both may share the same genetic sequences.

**REFERENCES**


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