Dear Sir:

I would like to congratulate Drs Pareja-Esteban J et al for their article titled “Brown-McLean syndrome and refractive phakic anterior chamber intraocular lenses” (1).

The purpose of this letter is to clarify the concept of the Brown-McLean syndrome or circumferential peripheral corneal edema which appears as idiopathic due to a number of cases with anterior chamber refractive intra-ocular lens in phakic patients.

In the past five years we have observed ten eyes of seven phakic patients treated by means of refractive surgery with anterior chamber intraocular lens with a diagnostic of Brown-McLean syndrome which appeared between eighteen months and four and a half years after the implantation of the lenses. The surgery had been performed without complications, respecting in all cases the implantation criteria — anterior chamber depth over 3.2 mm, size of the selected haptics 0.5 mm greater than the white-white diameter—. The previous grades of the patients ranged between +3,5 y –18 diopters. All the phakic angular support lenses which were implanted were of the Baikoff model with three support points: 7 lenses of the GBR ® myopia model and 3 New-Life ® lenses for presbytia. Prior to the appearance of the syndrome, all cases had remained asymptomatic, with total response of the symptoms in 60% of cases, partial response in 30% and no response in 10%, mainly to low power corticoids (fluorometholone), and all the corneal finding of microedema localized in the limbar zone. Visual acuity remained without changes and the central cornea did not exhibit edema or other type of alterations such as keratinic or pigment precipitates). In no case did we observe alterations under Goldman biomicroscopy: the lenses are respected the safety distance to the endothelial without inclination or vibration of the lens with eye movements («tilt») or rotation of the haptics in the chamber angle or synechia of the peripheral iris or pupil deformations. However, it became evident in 100% of cases that the central endothelial counting was normal, with low values ranging from 1340 to 1620 cel/mm3 and with well maintained morphological structure (fig. 1), but positioning the patients to make a peripheral counting showed in all cases a pathological reduction in the number of cells— under 1000 cel/mm 3 — as well as alterations of their hexagonal shape (fig. 2). In nine out of the ten cases, we proceeded to remove the lens, which led to the disappearance of the symptoms without treatment as well as of the corneal epithelial bullous microcystic alterations in 100% of cases between one and three months, without relapses during follow up. In four cases it was possible to perform at a later date corner and refractive surgery — hypermetropic LASIK— without complications or abnormal behavior of the cornea. The only case in which the lens was not removed the edema involved to become a general corneal edema which required a corneal transplant.

It must be remembered that a review of PubMed showed that when the Brown-McLean syndrome was explored, a pathological reduction of the peripheral corneal endothelial cells was described even though the central cells were normal. In addition, in some cases the progress shown by these alterations have required corneal transplants due to general progressive corneal decompensation (2). Similarly, more cases of complete corneal decompensation were reported involving the need of a corneal transplant with anterior chamber phakic lenses (3). We conclude that the finding of a Brown-McLean syndrome in patients with an anterior chamber lens

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**Fig. 1:** Central endothelial microscopy of the Brown-McLean syndrome: a normal cellular structure, Lo physiological number.
must be considered as the first subclinical stage of a complete corneal decompensation which is likely to occur in a variable period of time, with immediate removal of the lens.

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REFERENCES


Reply

Dear Sir,

Please allow me to express my gratitude for the interest shown by Dr. Díaz-Llopis and his team in our publication. Without a doubt, his experience on the peripheral corneal decompensation in ten cases of anterior chamber refractive intraocular lens in phakic patients is of great interest. However, one of the characteristics which differentiates the cases presented by said author from the patient who is the focus of our publication (1) is the type of intraocular lens utilized. In our case, we presented an aphakic patient with IOL in the anterior chamber. This could partially explain a different form of endothelial trauma which would justify the lack of progression of the corneal edema in our case, leading to a diagnostic of Brown-Mclean syndrome.

We would like to emphasize the point of the ophthalmological clinical exploration which led to the diagnostic of Brown-Mclean syndrome, either in the presence of an anterior intraocular lens, in the absence thereof (aphakia) or even in the absence of any surgical process (1).

In the first place, the Brown-Mclean syndrome is characterized by the presence of a peripheral corneal edema which typically does not progress towards the centre of the cornea, therefore exhibiting abnormal central endothelial count. The circumferential peripheral progression pattern (from the corner region corresponding to six o’clock towards three and nine o’clock simultaneously) has been described in a similar manner.

On the other hand, the subject exhibits characteristic brownish precipitates in the endothelial region corresponding to the corneal decompensation, with the finding of cornea gutatta not been infrequent.

We consider that these three characteristics can be key for diagnosing the Brown-Mclean syndrome.

However, in the case of phakic patients with anterior chamber intraocular lens, the presentation of the syndrome could be overlapped with total corneal decompensation (or be the beginning thereof) as pointed out in the letter to the editor which precedes this one.

It would be necessary to have a larger series of cases to reach a well founded conclusion about this type of statements.
REFERENCES
