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Flat Cornea and Diplopia

QUESTION

A 50-year-old black man enters with a chief complaint of monocular diplopia and blurred vision (20/100) of the right eye that was noted when he got up this morning. He denies prior injury, disease, or surgery of the eye; denies working with chemicals; and denies the use of ocular or systemic medication. Examination reveals normal eye examination OD, except a 2.25-diopter hyperopic shift and corneal topography that shows a small flattened area of the central cornea. Refraction improves vision to 20/25 blurred, but does not eliminate the diplopia. With a plano hard contact lens over the eye, and the eye refracted again, vision of 20/15 is obtained and monocular diplopia is eliminated. What is your diagnosis? What do you believe the etiology is? What further tests would you consider? What treatment would you advise?

ANSWER

The topography provided (Fig 1) is an axial power map, which is the least valuable of all the maps currently available. A refractive power map uses Snell's law and allows correlation with refraction, whereas axial power maps are unreliable. Local radius maps (tangential maps) are not helpful in correlating refraction, but give much greater detail in the actual location on the cornea, making abnormalities "light up" because of the difference from normal. Distortion maps are valuable because the irregularities in the cornea can be correlated with the quality of vision.

Nevertheless, using only the axial power map, we can still make some observations and conclusions. The map shows a localized flat spot centered slightly temporal to the optical center of the cornea (0.2 mm) and about 0.6 mm temporal to the pupillary center in this right eye. One can see a commensurate steepening on the nasal side of the cornea, which is steepest at about 1 o'clock at the 3-mm diameter, and forms an incomplete, asymmetric ring centered around the flat spot. There is also an induced, irregular with-the-rule astigmatism of 0.75 D. The pupillary margin is irregular, but this is usually an artifact of the pupillary detection system through an irregular cornea.

Because the slit-lamp exam appears normal, the history indicates that all this occurred during the previous night, there is no other history of eye disease, and everything is eliminated by the rigid contact lens, we know the symptoms and signs are confined to the anterior cornea. The diagnosis and etiology are clear. The patient was sleeping on the right side of his face for at least enough time for a fairly sharp object, usually a ring but possibly a knuckle or fingernail, pushing through the lid at the center of the blue spot on the

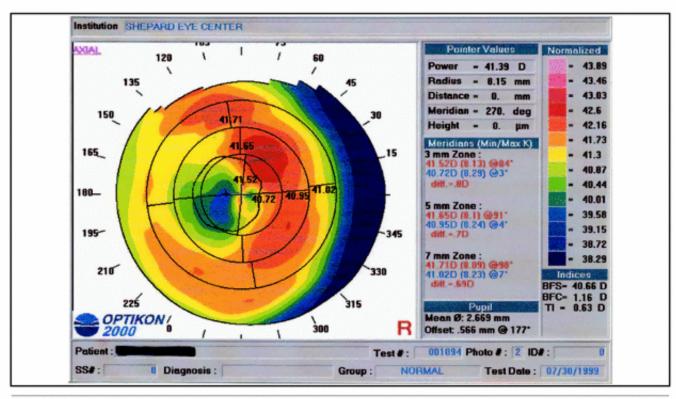


Fig 1.—Initial topography OD.

topography. The blue spot is slightly temporal and inferior, as would be expected from a moderate Bell's phenomenon and the fact the temporal aspect of the cornea is more accessible because of the orbital configuration (the same reason that traumatic retinal detachments are located more commonly temporally). Because of Euler's law of conservation of curvature with semi-elastic membranes, a commensurate steepening is in the form of a ring around the center of the flat spot (the center of the compression). Because the point of compression was slightly temporal, the result is an incomplete ring, resulting in an asymmetric steepening over the pupil resulting in 0.75 D of with-the-rule astigmatism and significant (2 to 3 D) of central flattening.

Because the gentleman is 50 years old, he is unable to overcome the +2.25 D of hyperopia or the with-the-rule astigmatism, but with refraction can get to 20/25 with most likely a $+2.00 +0.75 \times 91$; however, he cannot reach 20/15 because of the additional irregular astigmatism. The rigid hard contact eliminates the irregular component in the cornea, allowing him to return to 20/15 with the rigid contact.

The diagnosis is therefore "Factitious corneal flattening and induced irregular astigmatism during sleep." The etiology is explained above and the treatment is observation and explanation to the patient. The cornea will return to normal within 5 to 10 days in most patients, but like orthokeratology, the physics of each person's cornea is unique, so it is possible to last up to 6 weeks. The only other form of treatment is preventative and is to suggest the patient wear protective shields at night, because this compression of the eye during sleep can lead to far more severe permanent problems, such as ptosis, central retinal artery occlusion, central retinal vein occlusion, etc.

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ANSWER

This patient has monocular diplopia, blurred vision, and an irregular topographical profile. His vision is not fully correctable with spectacles, but it is fully restored with a rigid contact lens. By definition, the patient has irregular astigmatism. Although the patient has just noted the symptoms, it is possible that they were present for some time and just recently discovered.

His topography shows a paracentral zone of relative flattening with a large, 4-diopter drop-off over the pupillary region—likely accounting for the symptoms of monocular diplopia or image ghosting.

It would be useful to know the patient's previous and current refraction in both eyes. The corneal topography of the fellow eye may also be helpful to determine whether there is a congenital component or other suspicious patterns. Pachymetry mapping of both corneas with attention to the central as well as paracentral zones in the affected eye would also be beneficial. I would also question the patient about prior us of cosmetic, soft, hard, or orthokeratology contact lenses.

The etiology of irregular astigmatism may be congenital, traumatic, postsurgical, or secondary to a kerato-ectasia. The patient denies any trauma or any type of laser or refractive surgery. Keratoconus, keratoglobus, and pellucid marginal degeneration usually present with central or inferior regions of excessive

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