patients at a dermatology research institution reviewed the outcomes of 25 survivors aged 10 months to 25 years (survival rate of 56%). Ophthalmological problems included persistent ectropion in 64% of survivors, epiphora in 48%, and exposure keratitis in 12%. Sequela noted included corneal scarring and corneal perforation. Cataract formation was noted secondary to corticosteroid therapy in one patient, and retinoid therapy in another patient.[4]

Cicatricial ectropion eyelid surgery and skin grafting have been described in the management of the ocular manifestations of ARCI4B to preserve corneal clarity.[6-8] Risks of surgical intervention include increased likelihood of infection and risk factors associated with sedation in the setting of systemic comorbidities.[9] Skin grafting is limited by the availability of healthy skin, and ectropion often recurs.[4] Management of ARCI4B requires a multidisciplinary approach. The use of retinoids such as acitretin as well as proper humidification and sterilization have improved outcomes of newborns with ARCI4B, reducing both morbidity and mortality.[10]

This case series of 2 newborns with ARCI4B demonstrates that medical management alone can provide good ocular outcomes without the need for skin-releasing surgery, skin grafting, or alternative surgical management. This approach was effective in reducing the ectropion and preventing ocular surface sequela in both of our patients. Aggressive lubrication with close follow-up is necessary for preservation of the ocular surface. Additionally, erythromycin may be used for antimicrobial prophylaxis. Nonsurgical management of ARCI4B in a newborn requires collaboration of care with multidisciplinary specialties to prevent sequelae of severe exposure keratopathy.

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Corneal ectasia and high ametropia in an infant with microcephaly associated with presumed Zika virus congenital infection: new ocular findings

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We report the first case of a corneal ocular ectasia in an infant with Zika virus congenital infection (CZS). We suspect that the ocular embryology and neurotropism of the Zika virus could account for the corneal involvement.

According to the World Health Organization, the number of cases of microcephaly associated with presumed Zika virus congenital infection (CZS) is increasing worldwide.[1] The first human case in Brazil was reported in the state of Bahia in 2015,[2] although cases have also been reported in the northeastern states of Pernambuco and Paraiba.[2] Children with microcephaly have also been identified in southeast Brazil.[1,2] Several ocular manifestations may occur in infants with CZS, including abnormalities of the retina, choroid, and optic nerve.[2] Anterior segment findings, such as iris coloboma, lens subluxation, and congenital glaucoma, have been described with a lower prevalence.[2,3] We report presumed corneal ocular ectasia in an infant with CZS.

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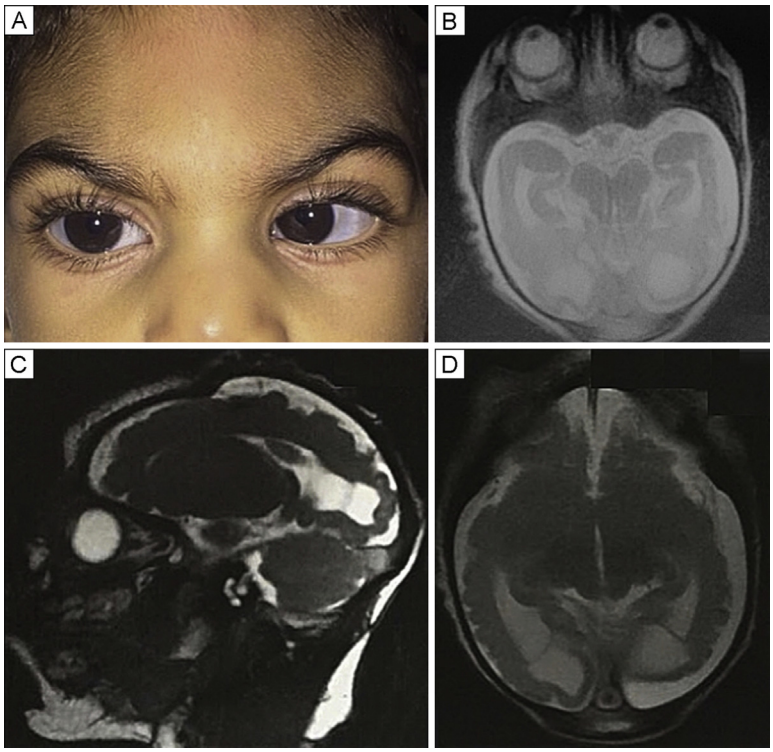

FIG 1. A, Patient with clear corneas and esotropia. B, T2-weighted magnetic resonance (MR) image (axial slice showing pachygyria. C, T2-weighted MR image (sagittal slice) showing pachygyria, agenesia of the mild- posterior portion of the corpus callosum, colpocephaly and pseudocysts. D, T2-weighted MR image (axial slice) showing colpocephaly, polymicrogyria, and pseudocysts.

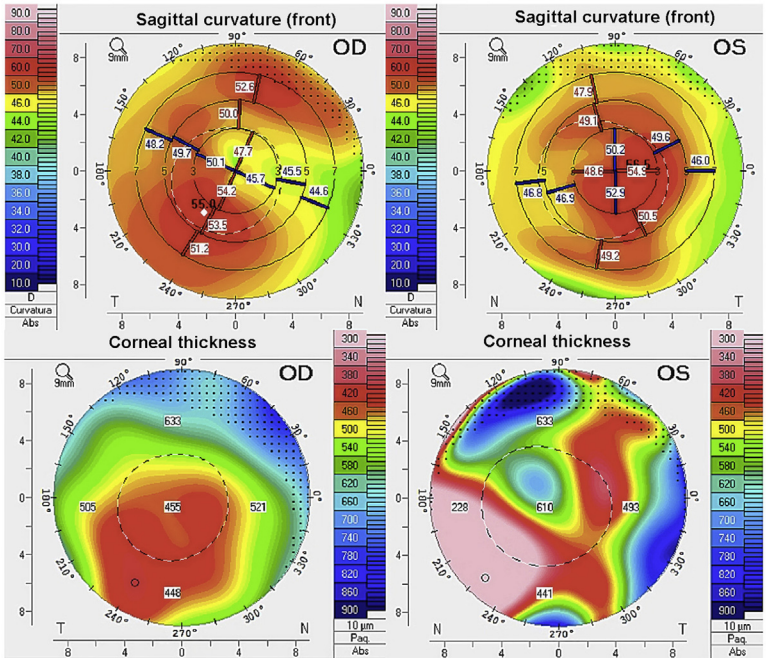

FIG 2. Anterior sagittal curvature and corneal thickness of the right eye and left eye showing increased anterior corneal curvature and decreased corneal thickness in both eyes (Pentacam; Oculus, Wetzlar, Germany).

Case Report

A 1.5-year-old boy presented at the Pediatric Low Vision clinic at the Federal University of Minas Gerais Hospital, Belo Horizonte, Brazil, for evaluation. The child had a history of presumed Zika virus infection at 11 weeks' gestational age, with the mother exhibiting classical signs of infection, including IgG MAC-ELISA positive serology

for Zika virus in the third trimester. Microcephaly was confirmed via intrauterine ultrasonography at 7 months' gestation. Serology for syphilis, toxoplasmosis, HIV, and hepatitis B was negative throughout the gestational period. Serology for rubella and cytomegalovirus was IgG positive and IgM negative at 9 weeks' gestation.

On examination at our clinic, uncorrected visual acuity was 20/470 in the right eye and 20/1900 in the left eye using Teller Acuity Cards. Intraocular pressure was 10.5 mm Hg in both eyes (Tono-Pen; Reichert, Buffalo, NY). Biomicroscopy showed clear corneas, lenses with no opacities, normal irides, and a normal anterior chamber (Figure 1). The horizontal corneal diameter was approximately 10 mm in the right eye and 11 mm in the left eye. An indirect binocular fundus examination revealed attached retinas, normal maculae, well-delimited optic nerves with normal nerve cupping and normal axial length, mild vascular tortuosity, and no choroidal lesions. Assessment of alignment revealed an esotropia of 30$^{\Delta}$ by Hirschberg testing (Figure 1) with a preference for the right eye and normal versions.

Results of dynamic retinoscopy were $-6.00 + 6.00 \times 90^{\circ}$ in the right eye and $-5.50 + 6.50 \times 90^{\circ}$ in the left eye. Cycloplegic retinoscopy examination showed $-4.50 + 7.00 \times 90^{\circ}$ in the right eye and $-5.00 + 7.00 \times 90^{\circ}$ in the left eye. The Lea Screen Hiding Heidi test revealed binocularly reduced contrast sensitivity.

Corneal keratometry was measured using the Pentacam HR (Oculus, Wetzlar, Germany). See Figure 2. The boy had irregular and asymmetrical astigmatism with increased sagittal curvature in both eyes associated with a corresponding decrease in the corneal thickness. The rather symmetric refractive error might suggest lower asymmetry of the left cornea, but the patient's inability to fix the deviated eye as well as his impaired cognition affected the quality of the examination.

The optic nerve appeared normal. Brain magnetic resonance imaging revealed structural changes related to malformations induced by the Zika virus (Figure 1).

Optical lenses for ametropia correction were prescribed. The patient continues to be followed for early visual intervention.

Discussion

Ocular embryology and the neurotropism of the Zika virus could account for the possible corneal involvement in our patient by as-yet-unknown mechanisms.[4] Studies in mice have revealed Zika RNA in the cornea, neurosensory retina, and optic nerve.[5] These findings are consistent with the origin of the present ectasia. The strong right eye preference might not be associated with the current ectasia but could be associated with the esotropia. Because of examination limitations, it was not possible to determine which eye was most affected.

For patient rehabilitation, it is important to emphasize ametropia identification and correction, considering the hypoaccommodation already described in this group of patients.[6] Because identified cases of intrauterine infection by Zika virus with ocular involvement are still very recent, and this is, to our knowledge, the first report of corneal ectasia associated with CZS, the evolution of the ectasia remains unknown.

Literature Search

PubMed was searched on July 17, 2019, without language restriction, using the following terms: *CZS*, *Zika*, *cornea*, *children*, *visual deficiency*, and *strabismus*.

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Extraocular muscle biopsy during surgery for strabismus of unknown etiology

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