full examination. Of the 16 people with refractive error, only 4 had spectacles (25% spectacle correction coverage).

Our finding that uncorrected refractive error (URE) is the main form of visual impairment is consistent with the major cause of visual impairment globally in children aged 5–15 years.[4] Early correction of refractive error is crucial, as it may lead to reduced education and employment activities and harm quality of life. School eye-health programmes are useful for screening for refractive error in Africa. Cost of spectacles is a major barrier to spectacle use, and students are more likely to wear spectacles if they have myopia and if the spectacles are free.[5] In Africa, where the spectacle correction coverage is low, adequate supply of cost-effective spectacles is required to reduce the burden of URE.

Conflict of interest
The authors declare no conflict of interest.

References

1 World Health Organization. *The Global Initiative for Elimination of Avoidable Blindness*. WHO: Geneva, 1997, (WHO/PBL/97.61 Rev 1).
2 World Health Organization. *Preventing Blindness in Children. Report of a WHO/IAPB Scientific Meeting*. WHO: Geneva, 2000, (WHO/PBL/00.77).
3 Gogate P, Kalua K, Courtright P. Blindness in childhood in developing countries: time for a reassessment? *PLoS Med* 2009; **6**: e1000177.
4 World Health Organization. *Elimination of Avoidable Visual Disability due to Refractive Error*. WHO: Geneva, 2000, (WHO/PBL/00.79).
5 Wedner S, Masanja H, Bowman R, Todd J, Gilbert C. Two strategies for correcting refractive errors in school students in Tanzania: randomised comparison, with implications for screening programmes. *Br J Ophthalmol* 2008; **92**: 19–24.

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Sir,

Retinopathy of prematurity in an infant with Aicardi's syndrome

Here, we present the first case of an infant with Aicardi's syndrome (OMIM 304050), morning-glory disc abnormality, and stage III retinopathy of prematurity (ROP).

Case report

An infant girl born at $30\frac{1}{7}$ weeks who had received supplemental oxygen (birth weight 1220 g) was examined at $37\frac{4}{7}$ weeks post-menstrual age. Fundus examination disclosed optic nerve colobomas OU with a morning glory disc anomaly OS. Chorioretinal lacunae were present in a peripapillary distribution OU (Figure 1). The right eye had 3 clock hours of zone II, stage I ROP temporally and the left eye had 3 clock hours of zone II, stage II ROP temporally (Figure 2). B-scan echography confirmed the presence of optic nerve colobomas and was negative for subretinal fluid. Magnetic resonance imaging was significant for agenesis of the corpus callosum. Skeletal survey X-rays were negative for costovertebral abnormalities. Over the next 2 weeks, the child went on to develop type 1 early treatment retinopathy of prematurity prethreshold disease and underwent laser photocoagulation to the avascular retina OU with resultant regression.

Comment

The pathognomonic chorioretinal lacunae in Aicardi's syndrome have been described as 'pseudotoxoplasmosis'[1] in a peripapillary distribution.[2] Histologically, they are defects in the choroid, choriocapillaris, and RPE and usually occur in the first trimester.[3,4] This time period is also when the embryonic fissures close and the corpus callosum develops. Thus, developmental defects during this period could explain the findings in Aicardi's syndrome, but not in ROP.

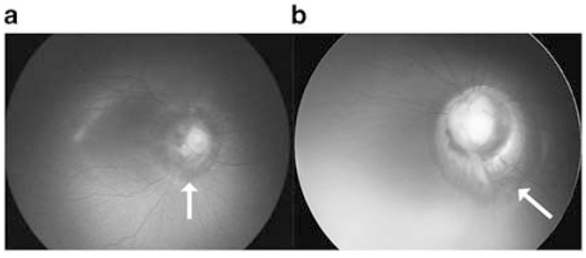

Figure 1 Fundus photographs of the right (a) and left (b) eyes of a premature infant with Aicardi's syndrome. Optic nerve colobomas were present in both eyes and a morning-glory disc anomaly was present in the left eye (b). Characteristic chorioretinal lacunae were present in a peripapillary location in both eyes (arrows).

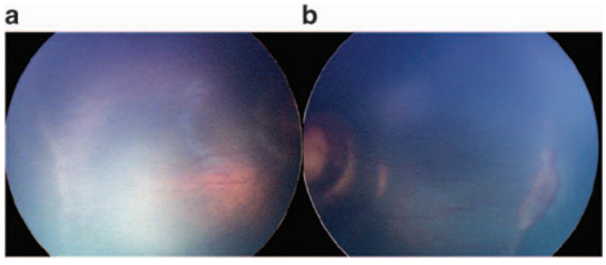

Figure 2 Colour fundus photographs of the temporal periphery of both eyes depicting zone II, stage I ROP in the right eye (a) and zone II, stage II ROP in the left eye (b).

No teratogens or infections have been consistently implicated in Aicardi's syndrome; however, malformations are likely due to failure of apoptosis and the abnormal synthesis of a peptide growth factor that directs the migration, differentiation, and proliferation of embryonic cells.[5] This case highlights the fact that Aicardi's syndrome neither worsens nor reduces the severity of ROP, which was likely present in this child because of prematurity, a low birth weight, and the use of oxygen during the postnatal period.

Conflict of interest
The authors declare no conflict of interest.

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References

1 Aicardi J, Lefebre J, Lerique-Koechlin A. A new sindrome: spasm in flexion, callosal agenesis, ocular abnormalities. *Electroencephalogr Clin Neurophysiol* 1965; **19**: 609–610.
2 Hoyt CS, Billson F, Ouvrier R, Wise G. Ocular features of Aicardi's syndrome. *Arch Ophthalmol* 1978; **96**: 291–295.
3 Font RL, Marines HM, Cartwright J, Bauserman SC. Aicardi syndrome: clinicopathological case report and electron microscopic observations. *Ophthalmology* 1991; **98**: 1727–1731.
4 McMahon RG, Bell RA, Moore GRW, Ludwin SK. Aicardi's syndrome: a clinicopathologic study. *Arch Ophthalmol* 1984; **102**: 250–253.
5 Bron AJ, Tripathi RC, Trapathi BJ. Development of the human eye. In: Bron AJ, Tripathi RC, Tripathi BJ (eds). *Wolff's anatomy of the eye and orbit*., 8th edn. Chapman and Hall: London, 1997, pp 621–664.

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