(homocystinuria), and ADAMTS4 and ADAMTS10 (spherophakia). As per the principles of Occam's razor, we would suggest that the case Dr Athanasiadis *et al* report had a pre-existing zonular weakness and/or received higher-than-usual amounts of laser energy. It is important to emphasise that LPI should be performed by a skilled, experienced operator, using the lowest possible power to achieve a satisfactory iridotomy. We would advocate the use of sequential argon/YAG iridotomy in patients with thick, dark brown irides.[4]

Regarding the risk of cataract formation/progression after PI, similar principles to those outlined above apply. With excess power or inappropriately applied laser treatment, it is possible to induce lens opacities, but this can be avoided with careful and precise treatment. Studies suggesting that LPI accelerates the formation of age-related cataract are exclusively retrospective studies, or individual case reports. Some have used surrogate outcome measures, such as reduction in visual acuity, rather than lens opacity grading. We believe that our study,[2] which was carried out prospectively, in the largest number of treated cases so far studied, with a control group selected from the community, using a standardized objective assessment of lens opacity (LOCS III system), currently constitutes the most robust scientific assessment of the risk of lens opacity after laser iridotomy.

The choice of either laser iridotomy or lens extraction for management of angle-closure glaucoma should be informed by the ongoing MRC EAGLE trial (https://viis.abdn.ac.uk/HSRU/eagle/).

We are grateful to the journal for giving us the opportunity to reiterate these points.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Screening for childhood blindness and visual impairment in a secondary school in rural Malawi

Childhood blindness is one of five areas of disease control in Vision2020: The Right to Sight,[1] and it is estimated that there are 300 000 blind children in Africa.[2] However, data on the epidemiology of childhood blindness in sub-Saharan Africa is scant, as children are rarely included in blindness surveys. Historically, information has been obtained from schools for the blind and, more recently, the key informant method,[3] but this does not address children with milder forms of visual impairment. Screening for visual impairment in regular schools may also yield useful information, although children with debilitating visual impairment may be less likely to attend, and secondary school attendance is not universal in Africa.

We examined 1000 children (aged 11–19 years) attending secondary school in Malamulo in rural Malawi. Presenting visual acuity (VA, with spectacles if owned, but uncorrected otherwise) was assessed with Snellen Chart at 6 metres. If presenting VA was $<6/18$ ('visual impairment'), they were invited to attend Malamulo Hospital Eye Department for formal refraction and slit-lamp examination, following suitable permission. Spectacles were dispensed if necessary.

There were 39 students (3.9%) with presenting VA $<6/18$ in one ($N=20$) or both ($N=19$) eyes. Among them 20 (51.2%) were male. One student (0.1%) was blind (VA $<3/60$) bilaterally, due to high myopia (-16.0 dioptres), and one had unilateral blindness from amblyopia (due to strabismus). Causes of visual impairment are presented (Table 1). In all, 29 (14 in bilateral group and 15 in unilateral group) attended the

Table 1 Aetiology of visual impairment in a secondary school in rural Malawi

Aetiology	*Bilateral vision impairment N (%)*	*Unilateral vision impairment N (%)*
Myopia	12 (85.7)	1 (6.7)
Cataract	1 (7.1)	2 (13.3)
Corneal scar	1 (7.1)	4 (26.7)
Trauma	—	2 (13.3)
Keratitis	—	1 (6.7)
Amblyopia (due to strabismus)	—	2 (13.3)
Other refractive error	—	3 (20.0)
Total	14 (100.0)	15 (100.0)

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.

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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.

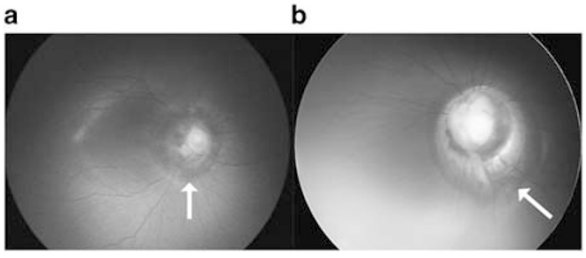

Figure 1 Fundus photographs of the right (a) and left (b) eyes of a premature infant with Aicardi's syndrome. Optic nerve colobomas were present in both eyes and a morning-glory disc anomaly was present in the left eye (b). Characteristic chorioretinal lacunae were present in a peripapillary location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

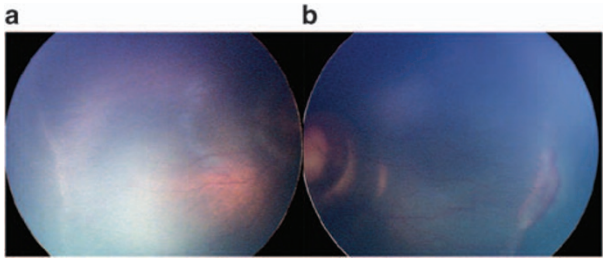

Figure 2 Colour fundus photographs of the temporal periphery of both eyes depicting zone II, stage I ROP in the right eye (a) and zone II, stage II ROP in the left eye (b).