Sir,

Visual impairment in children

Certification of visual impairment is used to register persons as visually impaired or severely visually impaired according to set criteria. This data is reported nationally and allows comparisons between regions and ages, and is essential in planning health, social, and educational services.[1]

Given the size of the population of Bradford district, it would be expected that 1% of national visual impairment would be accounted for by the region. However, reported data suggests that Bradford district accounts for 13% of the national registered visual impairment and 16% of registered severe visual impairment in 0–4 year olds. This figure drops to 3.5% and 3% of visual impairment and severe visual impairment, respectively, in 5–17 year olds.

The age-specific rates of registered severe visual impairment and visual impairment in Bradford district are compared with the national average in Tables 1 and 2. These rates are significantly higher in Bradford district compared with the national average (Z-test, $P<0.001$).

There are a number of possible explanations for these differences. Bradford district has a higher prevalence than the national average of children born with some forms of disability that may be associated with visual impairment,[2] thought to be particularly prevalent in Black and minority ethnic (BME) groups. This may be due, in part, to a high prevalence of autosomal recessive and inherited disorders.[3] Areas with similar BME populations, such as Calderdale, Walsall, and Oldham, have none or very few cases of registered visual impairment. Therefore, this suggests that other causes may be contributing to the higher rate.

Another explanation may be differences in registering visual impairment between regions such as differing levels of access to an ophthalmologist. As Bradford district has a high prevalence of visual impairment, clinicians may be more confident in registering children at an early age than areas with lower prevalences.

Registration rates of visual impairment in children is higher in Bradford district than the rest of England. The cause of this is unclear and is likely to because of the combination of a genuinely higher prevalence of visual impairment locally and underreporting of visual impairment nationally.

Table 1 Rates of registered severe visual impairment per 1000 population in England and Bradford district

	Rate of severe visual impairment in 0–4 years per 1000 population (95% confidence intervals)	*Rate of severe visual impairment in 5–17 years per 1000 population (95% confidence intervals)*
England	0.22 (0.20, 0.23)	0.44 (0.43, 0.45)
Bradford and Airedale TPCT	3.30 (2.73, 3.87)	1.43 (1.17, 1.68)

Table 2 Rates of visual impairment per 1000 population in England and Bradford district

	Rate of visual impairment in 0–4 years per 1000 population (95% confidence intervals)	*Rate of visual impairment in 5–17 years per 1000 population (95% confidence intervals)*
England	0.19 (0.18, 0.20)	0.57 (0.55, 0.58)
Bradford and Airedale TPCT	2.28 (2.28, 2.76)	2.26 (1.94, 2.58)

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Sir,

Avulsed retinal vessels accompanying posterior vitreous detachment: a late complication of retinopathy of prematurity

Abnormal retinal vasculature and vitreous, in combination with aberrant vitreoretinal traction are factors in the development of late complications in adults with retinopathy of prematurity (ROP).[1] A case of intact arcades of retinal blood vessels separated from the retina (see Supplementary movie file), secondary to posterior vitreous detachment (PVD) in an adult with ROP is presented. We wish to highlight this rare late complication of ROP, which was described in adults by Tasman[2] in 1970 and in neonates by Kingham[3] in 1982.

Case report

A 59-year-old woman attended eye casualty 6 months after uncomplicated cataract surgery with a 1-day history of floaters in her right eye, She was a high myope with a history of ROP (twin birth, 10 weeks premature). In the past, she had suffered from recurrent vitreous haemorrhage in this eye. The left eye was phthiscal as a result of endophthalmitis after cataract surgery.

On examination, the right eye was pseudophakic with visual acuity of 6/9. Fundoscopy showed the optic disc and retinal blood vessels dragged nasally (Figure 1a). In the peripheral fundus, there was chorioretinal atrophy and a falciform fold. A PVD accompanied by intact

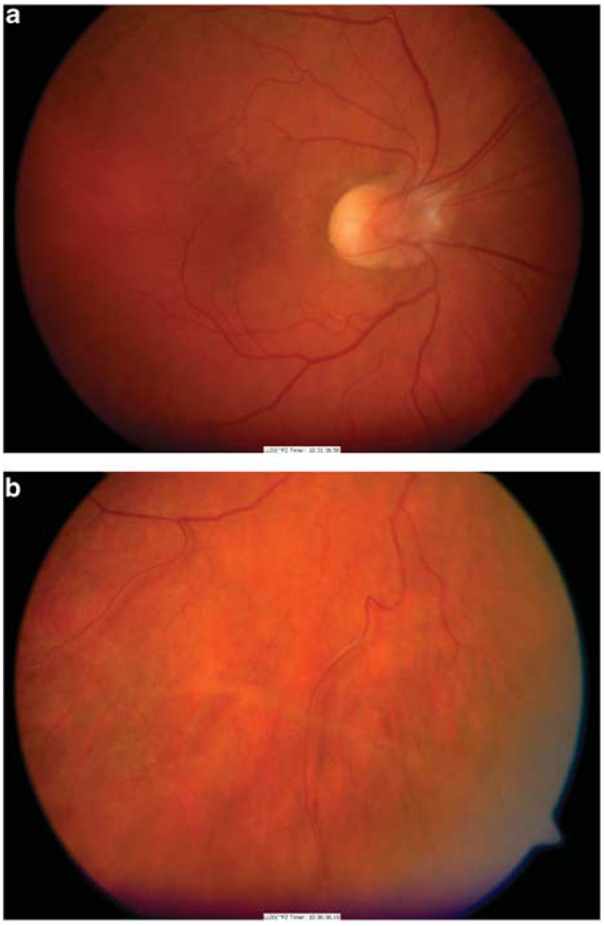

Figure 1 (a) A nasally dragged disc and retinal vessels. (b) Intact avulsed retinal vessels floating freely in the vitreous cavity.

avulsed retinal vessels floating freely in the vitreous cavity was present (Figure 1b). She had no retinal tear or detachment. OCT demonstrated areas of retinoschisis. Following review by the vitreoretinal team, a policy of observation was adopted. To date her visual acuity is 6/9, with no retinal detachment.

Comment

Various vitreoretinal complications have been described in the 'Boomer ROP' generation (born between 1940 and 1980).[4] Premature newborn survival improved during this time, but no defined treatment protocol was available for ROP giving rise to adult patients with various late-onset fundus findings. These include dragging of the retina, retinal detachment, retinal folds, lattice-like degeneration,[4] non-neovascular vitreous haemorrhage,[5] and myopia.[4]

Occasionally elevated blood vessels have been demonstrated secondary to vitreous traction.[6] We postulate that in this case separation of intact retinal vascular arcades from the retina occurred because of antero–posterior mechanical forces on already dragged retinal vessels under tension when PVD occurred. Kingham also advocated conservative management for such cases.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Mizuo-Nakamura phenomenon in Oguchi disease due to a homozygous nonsense mutation in the *SAG* gene

We report the clinical and electrophysiological findings in a case of Oguchi disease carrying a homozygous nonsense mutation in *SAG* (c.874C>T, p.Arg292X).

Figure 1 (a) Colour fundus photography of both eyes showing typical golden fundus reflex and pigment mottling in the far periphery. Discolouration is less profound in the macular area. (b) Fundus autofluorescence imaging of the right eye demonstrating no abnormality. (c) Axial cross-sectional image of the proband's right macula obtained using SD-OCT. In the parafovea, SD-OCT failed to detect the hyporeflective band (outer segments; OS) that is observed between the hyper-reflective layers associated with the inner/outer segment junction (IS/OS), and the RPE/Bruch's membrane complex (RPE/BM). (d) SD-OCT of the right eye of a 9-year-old control individual. Scans in c and d are to scale and acquired using the same SD-OCT protocol. Panel a with enlarged images of boxed regions (1, patient; 2, control) shows outer retina in detail. (e) Colour fundus photography of the posterior pole of the right eye using a non-mydriatic camera. After overnight (12-h) dark adaptation, a series of images were obtained over a 20-min interval. Disappearance of the golden reflex can be seen in the first image taken (top left). The golden colour gradually reappears after 10–15 flashes. Bottom right image is taken after 20 min and 32 flashes.