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The authors have no financial or proprietary interest in this article

Eye (2006) **20,** 858–859. doi:10.1038/sj.eye.6702031; published online 12 August 2005

Sir,

Medically unexplained visual loss

We commend the authors[1] for a summary of the clinical characteristics of patients with 'medically unexplained visual loss (MUVL)'. We are pleased to note that all their patients had neuroimaging given the resource constraints. Our experience of over two and half years in managing patients with MUVL is similar except for a much lower rate of neuroimaging due to resource constraints—a common problem in hospitals up and down the country. We find the following 'checklist'

Patient sticker + Date

PLACE THIS CHART IN THE NOTES

PATIENT CLAIMING NPL / LP / HM:

☐ RAPD not present.
☐ OKN induced: **Drum (VA >3/60) or Mirror twisted infront of face (VA >LP)**
☐ Good forced choice preferential looking.
☐ Diplopia induced: **By 8prism base down over blind eye.**
☐ Fusion observed with base out prism.
☐ Stereoacuity present.
☐ Can't touch tips of fingers together with both / blind eye open: **This is a test of proprioception and not vision.**
☐ Bizarre writing**..**
☐ Deliberately avoids or crashes into objects.
☐ Visual recovery after a few days in hospital.
☐ Normal ERG / VEP: **remember abnormal VEP may be due to patient defocus. Focal defects can be missed. Dilated with refractive correction for test distance may help.**
☐ Dislikes strong light in the 'blind' eye.
☐ Fast visual location to an object dropped on the ground: **Make sure sound not a factor in ocular movement.**
☐ Inconsistencies with Worth lights and Bagolini glasses. **Describe ……**

PATIENT CLAIMING 6/9 – HM:

☐ Near vision @ 15' does not equal distance vision @ 6m. **6/60=N24 or J17 6/24=N10 or J9-11 6/12=N6 or J4-5.**
☐ Marked visual improvement with plano refraction: **+4 /-4 lens or rotating 2-6D +/- cylinders to cancel.**
☐ Suddenly stopping at a line on the Snellen chart: **most patients can usually see a few letters on the line below.**
☐ Can now read better with 'affected' eye with +4 fogging infront of the good eye.
☐ Same Snellen line read at 3m as at 6m.
☐ Improved acuity with +4 gradual reducing fogging down to their prescription.
☐ No RAPD: **this may be a small macular lesion.**
☐ Normal ERG / VEP: **remember abnormal VEP may be due to patient defocus. Focal defects can be missed. Dilated with refractive correction for test distance may help.**
☐ Ishihara inconsistencies: **Make sure patient not colour blind. Healthy eye behind a green lens will only see test plates #1 and #36. If any others seen VA > 3/60 in the 'affected' eye.**
☐ Inconsistencies with Worth lights and Bagolini glasses. **Describe ……**

PATIENT CLAIMING VISUAL FIELD DEFECT: **(any field may be artifact)**

☐ Visual fields not consistent between static / confrontation / kinetic. **(automated may look reliable)**
☐ Goldman spiral / star (**Most common) or** crossing / reversal of isoptres.
☐ Humphrey '4 leaf clover' field.
☐ Refixation with prism displacing the image into the blind field: **Eg if field < 20 degrees a 20 dioptre prism will displace the image into the blind field. Refixation should not occur if the field is truly blind.**
☐ Field loss vanishes with the knowledge of loosing driving license legality.
☐ 1m and 4m field of vision equal: **Field with 5mm pin @ 1m should be X4 the size with a hand @ 4m. Note that this only tests the central 15 degrees of vision.**
☐ Saccade outside 'seeing' field: **ask the patient first if they have any eye pain with eye movements, tell them you are checking their eye muscles (not vision). Use if they claim not to see in the periphery. Look directly at the object**

ADDITIONAL TESTS:

☐ Walking with arms stretched out: **Blind people do not do this.**
☐ "Look at your hand" – but patient looks elsewhere.

OTHER COMMENTS: …… ***NAME / SIGNATURE***

Figure 1 Checklist.

(Figure 1) very useful in documenting the clinical findings at each consultation. This 'checklist' not only guides the clinical examination based on presenting symptoms but also helps proper documentation—a vital part of defending the clinical decisions at a later date (especially if medico-legal issues arise). We place a lot of emphasis on reviewing the patient at regular intervals for at least 18 months and have a very low threshold for neuroimaging should the symptoms worsen during the review period.

Reference

1 Griffiths PG, Eddyshaw D. Medically unexplained visual loss in adult patients. *Eye* 2004; **18**: 917–922.

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Eye (2006) **20,** 859–860. doi:10.1038/sj.eye.6702033; published online 5 August 2005

Sir,

Optic disc and peripheral neovascularization in a young male

Case report

An 8-year-old Asian male presented to his optometrist for a routine review.

On examination, his visual acuity was 6/6 in both the eyes, anterior segments, intra-ocular pressures, and vitreous were normal. Bilateral disc and peripheral neovascularization was evident with peripheral retinal ischaemia and ghost vessels (Figure 1). The patient was born full term in the United Kingdom, and had mild childhood asthma and hayfever. Systemic assessment was normal and there was no family history. Fluorescein angiography confirmed the presence of bilateral disc and peripheral neovascularization with extensive areas of midperipheral capillary nonperfusion, without branch retinal vein or arterial occlusion. No delay in choroidal or retinal vessel filling was observed. Chest X-ray, Mantoux test, fasting blood glucose, ESR, CRP, serum ACE, sickle cell, ANA, anti-dsDNA antibodies, syphilis serology, anti-phospholipid antibodies, activated partial prothrombin time, prothrombin time, fibrinogen level, Protein C, and Protein S were all normal.

The patient was diagnosed with Eales' disease even though the clinical presentation was unusual for Eales' disease given the young age, and presence of both optic disc and peripheral retinal neovascularization. The patient was treated with bilateral panretinal photocoagulation under general anaesthesia. At 6 weeks postphotocoagulation the vision in the left eye dropped to hand movements following a pre-retinal haemorrhage. The patient underwent a left three-port pars plana vitrectomy with further laser panretinal photocoagulation. The neovascularization regressed, and was replaced with glial tissue (Figure 1). At 2 years after treatment, his condition remains stable and visual acuity 6/6 in both eyes.

Comment

Eales' disease is an idiopathic obliterative vasculopathy that is uncommon in the UK. The condition most commonly occurs in healthy adult males aged between 20 and 30 years, and the youngest case reported is in a 10-year-old.[1–3] To our knowledge, our 8-year-old patient is the youngest reported case of Eales' disease.

Eales' disease progresses through several stages periphlebitis, perivasculitis, neovascularization, vitreous haemorrhage, and tractional retinal detachment. Although the aetiology of Eales' disease is unknown, hypersensitivity to infectious agents, such as tuberculosis and autoimmunity have been proposed.[1] The treatment of Eales' disease requires the exclusion of systemic illness, and laser panretinal photocoagulation.[4] Vitreous haemorrhage may commonly occur and without treatment tractional retinal detachment result.[5–7] Prognosis is poor with delayed treatment, and in our patient prompt Argon laser photocoagulation and early vitrectomy following