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Eye (2009) **23**, 2121–2122; doi:10.1038/eye.2008.399; published online 16 January 2009

Sir,
Reply to Patel and Larkin

Thank you for your interest in our case and your insightful comments. Reaching a diagnosis of Eales' disease involves systematic exclusion of known causes of retinal vasculitis.¹ Although baseline investigations are important in all cases, individually tailored diagnostic testing based on patient symptoms and signs is recommended. Furthermore, in cases of diagnostic doubt, infection may be considered more likely if, after an initial improvement with therapy, the patient's disease rapidly becomes refractory to treatment.²

Our case did in fact undergo an extensive systemic work-up, which was not emphasised in the study. We also referred our patient to the physicians given the unusual nature of his presenting symptoms. A complete physical examination as well as medical, contact, and travel history did not reveal any abnormalities or risk factors. In addition to routine blood and urine testing, other investigations performed included fasting glucose and lipid profile, thyroid function tests, renal and liver function tests, serum homocysteine levels, coagulation screen, vitamin B12 and folate levels, serum ACE, CRP, ESR, full autoantibody screen, serum protein electrophoresis, and 72-h Mantoux testing. Radiological investigations performed comprised a chest X-ray, abdominal ultrasound, and carotid Doppler. All investigations completed did not reveal any systemic abnormality. We agree that prompt referral to the physicians and a full contact and travel history are important in ruling out other causes of retinal vasculitis, especially in cases with systemic features (eg, fever, weight loss, and altered bowel habit). In our case, the clinical effect of adjunctive treatment with pegaptanib was evidenced by the rapid regression of disc and retinal neovascularisation with no recurrence of vitreous haemorrhage for up to 9 months.

References

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Eye (2009) **23**, 2122; doi:10.1038/eye.2008.400; published online 16 January 2009

Sir,
Subthreshold diode micropulse panretinal photocoagulation for proliferative diabetic retinopathy

We read with interest the article 'Subthreshold diode micropulse panretinal photocoagulation for proliferative diabetic retinopathy' by Luttrull *et al.*¹ We would like to congratulate the authors for their work and cite a clarification.

The authors mention that the treatment parameters were designed to avoid the creation of clinically detectable photocoagulation lesions and that the effectiveness of subthreshold diode pan retinal photocoagulation (PRP) at this low irradiance level along with its efficacy in diabetic macular oedema is an evidence in favour of subthreshold laser in clinical practice. However, the mechanism of action of laser photocoagulation is thought to be different in these two conditions. The decrease in macular oedema is supposed to be mediated through the retinal pigment epithelium² for which even subthreshold energies may be sufficient. However, in proliferative diabetic retinopathy, destruction of the ischaemic retina thereby decreasing the angiogenic stimulus and improved oxygenation of the remaining retina are among the major hypotheses of the mechanism of action.³ Keeping in mind these factors, the likely mechanism of action of subthreshold PRP stated by the authors needs clarification.

Also, is it justified to treat all the patients with the same energy levels and to titrate it with the pain threshold that has a wide variation independent of the energy required for producing a visible lesion? Titrating the energy for a visible spot and then reducing the power or the time of the laser beam will be a better method for doing subthreshold PRP, as it will provide the required subthreshold energy for a given patient and amount of retinal oedema.

References

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