- 5 Matsuda Y, Kawamoto K, Kiya K, Kurisu K, Sugiyama K, Uozumi T. Antitumor effects of antiprogestrones on human meningioma cells *in vitro* and *in vivo*. J Neurosurg 1994; 80: 527–534.
- 6 Haak HR, de Keizer RJW, Hagenouw Taal JCW, v Seters AP, Vielvoye GJ, van Dulken H. Successful mifepristone treatment of recurrent inoperable meningioma. *Lancet* 1990; 336: 124–125.
- 7 Grunberg GM, Weiss MH, Spitz JM, Sadun A, Russell CA, Lucci L et al. Treatment of unresectable meningiomas with the antiprogesteron agent mifepristone. J Neurosurg 1991; 74: 861–866.

RJW de Keizer¹ and JWA Smit²

¹Department of Ophthalmology Leiden University Medical Center PO Box 9600, 23 00 RC Leiden The Netherlands

²Department of Endocrinology Leiden University Medical Center PO Box 9600, 23 00 RC Leiden The Netherlands

Correspondence: RJW de Keizer Tel: + 31 71 526 2374 Fax: + 31 71 524 8222 E-mail: R.J.W.de_Keizer@lumc.nl

Eye (2004) **18**, 954–958. doi:10.1038/sj.eye.6701370 Published online 19 March 2004

Sir,

Hemorheological changes in patients undergoing haemodialysis for chronic renal failure and retinal ischaemia

I read with interest the article by Pahor 'Retinal Light Sensitivity in Haemodialysis Patients'.¹ The author has shown that there was a significant reduction in retinal light sensitivity in 36% of patients undergoing haemodialysis (HD) for chronic renal failure (CRF) as indicated by the significant reduction in their field global indices: mean deviation, pattern standard deviation, and corrected pattern standard deviation in comparison with the control group. In the discussion, the author explained that this may be the result of the retinal circulatory disturbance secondary to hypertensive retinopathy and atherosclerotic changes in the carotid arteries that cause chronic ischaemic retinopathy due to increased red cell aggregation and its reduced deformability and increased blood viscosity.

First, I would like to add that the above haemorheological changes, in addition to increased

plasma fibrinogen, were reported in CRF patients undergoing HD and were found to increase the blood viscosity.^{2,3} These changes are presumed to precede and conduce to the development of astherosclerosis² in HD patients and, together with the microcirculatory disturbance known to develop in association with increased blood viscosity,^{4,5} could result in chronic retinal ischaemia. Erythrocyte abnormalities described in CRF patients seem to be particularly relevant in this context. In a review by Caimi,⁶ alteration in erythrocytes' membrane properties, due to oxidative stress, was suggested to lead to their increased aggregability and reduced deformability and to explain their reduced survival that, in addition to reduced erythropoietin synthesis by the impaired kidneys, causes anaemia. Although anaemia may counterbalance the hyperviscosity in these patients, it indeed increases the ischaemic brunt on the retina.

Second, the questions that follow from the interesting findings of this study are whether we will need to keep these patients under review to detect these early deficits in retinal function and whether it will be necessary to treat them upon detection of such signs. Noninvasive functional assessment by visual field testing or alternatively electrophysiological tests, as suggested in the study, would be appropriate and would serve the first purpose. Since hyperviscosity-induced changes in retinal and choroidal circulation were proved to reverse,⁷ one can only speculate that they could at least be partially reversed or slowed down with early treatment in HD patients. Maintaining good control of these patients' blood pressure, plasma cholesterol level and correction of anemia can certainly be paramount in that regard. Dipyridamole (Persantin) would be an additional therapeutic option that could improve the microcirculatory function through improving erythrocyte deformability.⁸ Notably, giving aspirin to these patients could potentially be hazardous because of their increased tendency for bleeding due to platelet dysfunction. However, considering that there is no evidence thus far that any treatment will have any beneficial effect on retinal function, the decision to investigate or to treat, I believe, will remain up to the clinician's discretion.

References

- 1 Pahor D. Retinal light sensitivity in haemodialysis patients. *Eye* 2003; **17**: 177–182.
- 2 Martinez M, Vaya A, Alvarino J, Barbera JL, Ramos D, Lopez A *et al.* Hemorheological alterations in patients with chronic renal failure. *Effect of hemodialysis. Clin Hemorheol Microcirc* 1999; **21**: 1–6.
- 3 Wink J, Vaziri ND, Barker S, Hyatt J, Ritchie C. The effect of hemodialysis on whole blood, plasma and erythrocyte viscosity. *Int J Artif Organs* 1988; **11**: 340–342.

- 4 Knabben H, Wolf S, Remky A, Schulte K, Arend O, Reim M. Retinal hemodynamics in patients with hyperviscosity syndrome. *Klin. Monatsbl. Augenheilkd. (abstract)* 1995; **206**(3): 152–156.
- 5 Dobberstein H, Solbach U, Weinberger A, Wolf S. Correlation between retinal microcirculation and blood viscosity in patients with hyperviscosity syndrome. *Clin Hemorheol Microcirc* 1999; 20: 31–35.
- 6 Caimi G. Erythrocyte peroxide metabolism, plasma lipid pattern and hemorheological profile in chronic renal failure. *J Nephrol* 2002; **15**: 104–108.
- 7 Rezai KA, Patel SC, Eliott D, Becker MA. Rheumatoid hyperviscosity syndrome: reversibility of microvascular abnormalities after treatment. *Am J Ophthalmol* 2002; **134**: 130–132.
- 8 Saniabadi AR, Fisher TC, Lau CS, Bridges A, Taylor J, Belch J et al. Dipyridamole increases human red blood cell deformability. Eur J Clin Pharmacol 1992; 42: 651–654.

MA Elgohary

Opthalmology Department Essex County Hospital, Lexden Road Colchester CO3 3NB, UK

Correspondence: M A Elgohary Tel: +44 7939274217 Fax: +44 1206744742 E-mail: m.elgohary@doctors.org.uk

Eye (2004) **18**, 958–959. doi:10.1038/sj.eye.6701371 Published online 26 March 2004

Sir, Unusual presentation of dirofilariasis as a lacrimal mass

A 66-year-old Hong Kong Chinese female patient presented with a drooping of her right upper eyelid for 2 weeks. She was born in China and has never travelled abroad. Examination showed a partial ptosis of her right upper lid. It was caused by a palpable, firm, nontender mass with a size of 1 cm in diameter located at the superotemporal region of her right orbit. Ophthalmic examination was otherwise normal. Computed tomography scans of the orbit showed an enlarged lacrimal gland of the right eye associated with a nonenhancing hypodense lesion (Figure 1). No adjacent bony erosion was seen. Serological examinations including eosinophil count were normal. Chest X-ray was unremarkable. An excisional biopsy of the lacrimal gland was performed through lateral orbitotomy. The lacrimal gland was found to be well encapsulated and there was no adhesion to the surrounding structures.



Figure 1 Computed tomographic scan showing an enlarged right lacrimal gland associated with a noncontrast-enhancing hypodense lesion.



Figure 2 Haematoxylin and eosin stain. Solid arrow: multiple transverse sections of a single female *Dirofilaria* in the lacrimal gland. Empty arrow: lacrimal gland capsule.

Histopathology demonstrated a parasite surrounded by chronic inflammatory cells and significant eosinophils. The presence of thick cuticle, numerous external ridges, two internal longitudinal ridges with lateral chords, heavy musculature and double uterine tubes suggested that the parasite was a female *Dirofilaria* (Figure 2). The degeneration of the specimen did not allow further differentiation into subspecies. After the surgery, the eyelid contour returned to normal and there was no recurrence at the last follow-up visit of 1 year.

Dirofilariasis is a parasitic infection of domestic and wild animals, but it occasionally affects human as zoonotic disease. *D. repens* is the most frequently