epithelium and secondary inflammation. However, the second graft showed an unusual pannus associated with mucous plaques, corneal vascularisation, and an inflammatory infiltrate rich in eosinophils and macrophages. While some of these features can be interpreted as the consequence of prolonged epithelial ulceration, the possibility of an atopic reaction was considered and confirmed on further questioning and examination. The second graft therefore shows dual pathology of modified atopic keratitis in a patient with recurrent epithelial ulceration due to aniridia keratopathy.

Corneal abnormalities seen in aniridia have been attributed to deficiency of limbal stem cells and incursion of conjunctival epithelial cells onto the cornea. It has also been suggested that low levels of PAX-6 expression result in epithelial fragility related to cytokeratin (K3 and K-12) deficiency and in addition, an abnormal wound-healing response due to deficiency of matrix metalloproteinase-9.[4,5]

Mucous plaques are an uncommon complication of severe atopic and vernal keratoconjunctivitis, but can also be seen with herpes zoster ophthalmicus, keratoconjunctivitis sicca, and superior limbic keratitis.[6,7]

It is unusual for mucous plaques to develop unless very severe atopic disease is present. Our patient had marked corneal disease with large mucous plaques in the presence of minimal conjunctival involvement. We believe that a genetically determined abnormality of corneal epithelial healing in an aniridia patient with a very mild atopic conjunctivitis led to pronounced mucous plaque formation.

References

1 Duke-Elder S. Anomalies of the uveal tract-aniridia. In: Duke-Elder S (ed). *System of Ophthalmology*. CV Mosby: St. Louis, MO, 1963, pp 565–573.
2 Nelson L, Spaeth G, Nowinski T, Margo C, Jackson L. Aniridia. A review. *Survey of Ophthalmol* 1984; **28**: 621–642.
3 Muto R, Yamamori S, Ohashi H, Osawa M. Prediction by FISH analysis of the occurrence of Wilms' tumour in aniridia patients. *Am J Med Gen* 2002; **108**(4): 285–289.
4 Tseng S, Li D. Comparison of protein kinase C subtype expression between normal and aniridic human ocular surfaces: implications for stem cell dysfunction and aniridia. *Cornea* 1996; **15**: 168–178.
5 Ramaesh T, Martin J, Ramaesh K, Kaufman M, West J, Dhillon B. Corneal abnormalities in PAX+/−(small eye) mice mimic human aniridia-related keratopathy. *Inv Ophthalmol Vis Sci* 2003; **44**(5): 1871–1878.
6 Marsh RJ, Cooper M. Ophthalmic zoster: mucous plaque keratitis. *Br J Ophthalmol* 1987; **71**: 725–728.
7 Cameron J. Shield ulcers and plaques of the cornea in vernal keratoconjunctivitis. *Ophthalmology* 1995; **102**: 985–993.

S Saidkasimova, F Roberts and JL Jay

Tennent Institute of Ophthalmology
1054 Gt Western Rd
Glasgow G12 0YN, UK

Correspondence: S Saidkasimova
Tel: +44 141 211 1041
Fax: +44 141 211 2054
E-mail: shohistas@yahoo.co.uk

Eye (2005) **19,** 926–928. doi:10.1038/sj.eye.6701682; published online 17 September 2004

Sir,

Chronic myeloid leukaemia presenting as venous stasis retinopathy

I read with interest the comprehensive article by Sharma *et al*[1] reviewing the ophthalmologist's role in the ophthalmic manifestations of acute leukaemia. I would like to report a case of chronic myeloid leukaemia that presented to a DGH eye casualty.

Case report

A 29-year-old man presented with sudden onset diminution of vision in his left eye. He had glandular fever infection 10 years ago. Visual acuity was normal in right eye but restricted to 6/18, N10 in the left eye. Fundus examination revealed bilateral venous stasis retinopathy with scattered roth spots, the left eye being more severely affected than the right. The left eye also revealed macular haemorrhages (Figure 1).

On further history taking, he agreed to easy bruising, decreased appetite, and night sweats. General examination revealed gross hepatospleenomegaly in addition to multiple bruises of different ages on his body. Urgent blood test revealed marked leukocytosis with blood film consistent of Philadelphia-positive chronic myeloid leukaemia.

He was urgently referred to haematology where he received leukophoresis and cell and sperm storage. Oral hydroxyurea, allopurinol, and later imatinib-achieved cytoreduction and remission. His general and

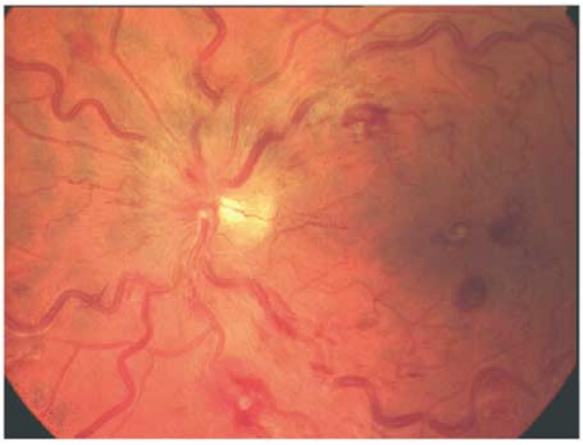

Figure 1 Fundus photograph of left eye showing venous stasis retinopathy, roth spots and macular haemorrhages.

ophthalmic condition improved with resolution of macular haemorrhages and roth spots.

I feel this case highlights the high index of suspicion of leukaemia necessary when a patient presents with roth spots or venous stasis retinopathy. As ophthalmologists, we play a pivotal role as prompt diagnosis and treatment can benefit patients with both acute and chronic leukaemia.

Acknowledgements

Financial interest: None

References

1 Sharma T, Grewal J, Gupta S, Murray PI. Ophthalmic manifestations of acute leukaemias: the ophthalmologist's role. *Eye* 2004; **18**: 663–672.

SM Salvi

Department of Ophthalmology
Royal Gwent Hospital, Cardiff Road
Newport NP20 2UB, UK

Correspondence: SM Salvi
Tel: +44 1633 238444
Fax: +44 1633 656294
E-mail: karansalvi@aol.com

Eye (2005) **19,** 928–929. doi:10.1038/sj.eye.6701683; published online 17 September 2004

Sir,

Pneumatic displacement of subretinal haemorrhage followed by transpupillary thermotherapy of choroidal neovascular membrane secondary to multifocal choroiditis

Multifocal choroiditis with panuveitis commonly occurs in middle-aged women. It is characterized by multiple punched out chorioretinal spots with vitritis.[1] This disorder is complicated by the development of choroidal neovascularization (CNV) or progressive subretinal fibrosis. Subfoveal CNV is an important cause of severe visual loss[2] in these patients. Methods of treating subfoveal CNV in multifocal choroiditis include observation (smaller than 100 μm),[2] oral corticosteroids,[3] thermal laser, surgical removal,[4] and photodynamic therapy (PDT).[5]

Transpupillary thermotherapy (TTT) is an established modality of treatment for occult CNV.[6] TTT for CNV in multifocal choroiditis has not been reported (Medline search).

We report a case of CNV secondary to multifocal choroiditis treated with pneumatic displacement of subretinal haemorrhage followed by TTT.

Case report

A 45-year-old male presented with an episode of diminished vision in the left eye of 15 days duration. A previous episode of decrease in vision in both eyes was followed by resolution of vision in the left eye only.

On examination, his best-corrected visual acuity was 1/60 in the right eye and 6/60; N36 at 20 cm in the left eye. Anterior segment examination was unremarkable. Fundus evaluation showed multiple chorioretinal atrophic scars in both eyes. One of the atrophic scars involved the fovea of the right eye. Subfoveal haemorrhage was noted in the fovea of the left eye (Figure 1a). Fundus fluorescein angiography (FFA) showed predominantly classic CNV with an indistinct temporal margin adjacent to a chorioretinal atrophy scar due to subretinal haemorrhage (Figure 1b). The subretinal haemorrhage was partially displaced with 0.5 ml of intravitreal air and prone positioning for 3 days (Figure 1c).

After 1 week, the patient's distant vision remained stable at 6/60 and near vision improved to N24 at 30 cm. FFA could now delineate the temporal margin of classic CNVM (Figure 1d). The options of PDT and TTT were discussed, and the patient chose to undergo TTT. TTT was delivered using an 810 nm diode laser (Iris Medical Oculight SLx, Iridex Corporation, Mountain View, CA, USA). The laser parameters were: power—130 mW; spot size—2 mm; duration—60 s. At 2 months post-treatment,