3 Richard S, Campello C, Taillandier L, Parker F, Resche F. Haemangioblastoma of the central nervous system in von Hippel–Lindau disease. *J Intern Med* 1998; **243**: 547–553.

EJ Lee, L Membrey and S Vickers

Sussex Eye Hospital
Eastern Road
Brighton BN2 5BF, UK

Correspondence: S Vickers
Tel: +44 1273 606 126
Fax: +44 1273 693 674
E-mail: sarah.vickers@bsuh.nhs.uk

Eye (2005) **19,** 812–813. doi:10.1038/sj.eye.6701652; published online 10 September 2004

Sir,

Reply to Limbal stem cell deficiency: a clinical chamaelon

We read this case report with great interest. The authors described a case of a persistent corneal epithelial defect, stromal changes, and wound leak following a penetrating keratoplasty in an eye which had undergone a pterygium excision and conjunctival autograft.[1] The authors allude the epithelial defect noticed on the first postoperative day to be due to limbal stem cell deficiency. It may be plausible to provide an alternative explanation for the reported finding. The epithelial defect and corneal stromal changes were noticed to a site adjacent to the previous pterygia. Recent studies have shown evidence to suggest that the development of pterygia is linked to matrix metalloproteinases (MMPs) overexpressed by altered limbal epithelial basal cells.[2] MMPs are a family of more than 21 genetically distinct proteases, which are produced in small amounts under normal physiological conditions by fibroblasts and epithelial cells.[3] These MMP's being proteases dissolve and remodel extracellular matrix that includes fibronectins, collagen, and basement membrane.[3] During the development of pterygia, there is overexpression of MMPs that go on to dissolve Bowman's layer, which in turn triggers the fibrovascular pannus formation.[2]

The epithelial and stromal changes observed might have resulted from abnormal activity of MMPs from the previous site of the pterygium. Further, the figure shows the epithelial defect to be involving the donor corneal button as well on day 1. This was a very rapid change and manifestations of limbal stem cell deficiency are generally slow in onset.[4] The epithelial defect in this patient may be due to altered MMP expression resulting in dissolution of Bowman's layer leading to a corneal epithelial defect. The rapid healing of the epithelial defect following limbal stem cell graft may be contributed to the removal of source of the MMPs.

We would be most grateful for the view of the authors

References

1 Zaidi FH, Bloom PA, Corbett MC. Limbal stem cell deficiency: a clinical chameleon. *Eye* 2003; **17**(7): 837–839.
2 Dushku N, John MK, Schultz GS, Reid TW. Pterygia pathogenesis: corneal invasion by matrix metalloproteinase expressing altered limbal epithelial basal cells. *Arch Ophthalmol* 2001; **119**(5): 695–706.
3 Sivak JM, Fini ME. MMPs in the eye: emerging roles for matrix metalloproteinases in ocular physiology. *Prog Retin Eye Res* 2002; **21**(1): 1–14.
4 Dua HS, Azuara-Blanco A. Limbal stem cells of the corneal epithelium. *Surv Ophthalmol* 2000; **44**(5): 415–425.

SS Zaher and K Ramaesh

Tennent Institute of Ophthalmology
Gartnavel General Hospital
1053 Great Western Road
Glasgow, G12 0YN, UK

Correspondence: SS Zaher
Tel: +44 13 1441 5037
Fax: +44 141 211 2054
E-mail: sarah_zaher@hotmail.com

Eye (2005) **19,** 813. doi:10.1038/sj.eye.6701654; published online 3 September 2004

Sir,

Limbal stem cell deficiency: a clinical chameleon

Zaidi *et al*[1] infer that the corneal donor epithelial defect noted day 1 postkeratoplasty is secondary to limbal stem cell deficiency. How can this be so? A donor epithelial defect day 1 is surely due solely to loss of donor epithelium and has nothing to do with host limbal stem cell function. They have treated the donor epithelial defect with cyclosporin drops and intensive topical preservative-free steroids. This is inappropriate management for both stem cell dysfunction and persistent donor epithelial defect. They have then performed a limbal stem cell graft along with repeat keratoplasty, used the same inappropriate line of clinical management and observed a similar but less severe course of events. Therefore, they have neither demonstrated that limbal stem cell deficiency was the cause of the problem nor shown any convincing benefit from the stem cell graft.