identified because of extensive involvement of the sac.[5] The location of the tumour in connective tissues of other organs and the positive immunostaining for CD-34 antigen suggest that this tumour originates from primordial endothelial cells.

Our patient presented in her sixth decade in contrast to the presentation in the other two cases in the second and third decades.[3] The 18 reported cases of orbital tumour were between the second and seventh decade (mean 45.7 years);[2] thus it seems that solitary fibrous tumour may be presented in a wide range of ages.

In the previous report of solitary fibrous tumour of the lacrimal sac,[4] the tumour presented as a solid medial canthal mass, whereas in our case, the patient presented with acquired nasolacrimal duct obstruction. Although this is only one case report, and lacrimal sac tumours are infrequent,[7] the findings emphasize the importance of a biopsy of the lacrimal sac during dacryocystorhinostomy in cases of abnormal appearing lacrimal sac in addition to systemic malignancy and certain inflammations[8].

References

1 Westra WH, Gerald WL, Rosai J. Solitary fibrous tumour. *Am J Surg Pathol* 1994; **18**: 992–998.
2 Kim HY, Lee SY, Kang SJ, Kim HJ. Solitary fibrous tumour of the orbit: a poorly recognized orbital lesion. *Acta Ophthalmol Scand* 1999; **77**: 704–708.
3 Scott IU, Tanenbaum M, Rubin D, Lores E. Solitary fibrous tumour of the lacrimal gland fossa. *Ophthalmology* 1996; **103**: 1613–1618.
4 Woo KI, Suh YL, Kim YD. Solitary fibrous tumour of the lacrimal sac. *Ophthalmic Plast Reconstr Surg* 1999; **15**: 450–453.
5 Suster S. Recent advances in the application of immunohistochemical markers for the diagnosis of soft tissue tumours. *Semin Diagn Pathol* 2000; **17**: 225–235.
6 Helm KF. Immunohistochemistry of skin tumours. In: Dabbs DJ (ed). *Diagnostic Immunochemistry*. Churchill-Livingstone: New York, 2002, p 318.
7 Stefanyszyn MA, Hidayat AA, Pe'er J, Flanagan JC. Lacrimal sac tumours. *Ophthalmic Plast Reconst Surg* 1994; **10**: 169–184.
8 Bernarnini FR, Molin M, Kersten RC *et al*. Routine histophthologic evaluation of the lacrimal sac during dacryocystorhinostomy. How useful is it? *Ophthalmology* 2001; **109**: 1214–1218.

S Rumelt[1], Y Kassif[1], I Cohen[2] and U Rehany[1]

[1]Department of Ophthalmology
Western Galilee-Nahariya Medical Center
PO Box 21, 22100 Nahariya, Israel

[2]Department of Pathology
Western Galilee-Nahariya Medical Center
PO Box 21, 22100 Nahariya, Israel

Correspondence: Shimon Rumelt
Tel: 972 4 9107635
Fax: 972 4 9107611

Sir,

Reduced visual acuity following standard ETDRS macular laser for clinically significant macular oedema
Eye (2003) **17,** 431–433. doi:10.1038/sj.eye.6700337

The early treatment diabetic retinopathy study (ETDRS)[1] showed that macular grid and focal laser therapy reduces the risk of moderate visual loss in diabetic eyes with clinically significant macular oedema (CSME). Although this treatment may not benefit every patient, side effects are fortunately uncommon. Reduction of visual acuity following treatment infrequently occurs because of subfoveal choroidal neovascularisation, subretinal fibrosis, macular haemorrhage and areas of capillary nonperfusion in continuity with the foveal avascular zone (FAZ).

A case is presented where loss of visual acuity occurred following focal laser treatment (in accordance with ETDRS guidelines) adjacent to an area of nonperfusion away from the fovea.

Case report

A 65-year-old man with type II diabetes mellitus reported a gradual decrease in right visual acuity over 6 months. He was known to have moderate nonproliferative diabetic retinopathy in both eyes and had presented 2 years earlier with CSME in both eyes. He had received macular laser treatment once to his right eye and once to his left. Fluorescein angiography was performed prior to further treatment and showed leakage temporal to the fovea, superotemporal nonperfusion and drop-out of perifoveal capillaries temporal to the macula (Figure 1). On examination, corrected visual acuity was 6/60 right and 6/36 left. There was a right posterior subcapsular lens opacity and moderate nonproliferative diabetic retinopathy in both eyes but no CSME. Uneventful phacoemulsification was performed. Two weeks postoperatively, right visual acuity was 6/9 corrected but CSME was identified temporal to the fovea. This was treated with uneventful focal laser. One month later the patient returned with a right visual acuity of 6/24. Clinical findings were

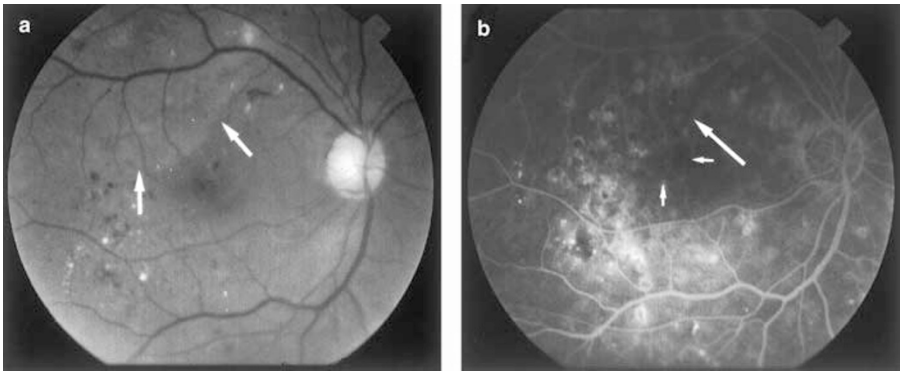

Figure 1 Photographs taken prior to temporal focal laser. (a) Red-free fundus photograph of right eye showing the area of superotemporal ischaemia (arrows). (b) Fluorescein angiogram showing area of superotemporal ischaemia (large arrow) and area of temporal ischaemia not continuous with FAZ (small arrows).

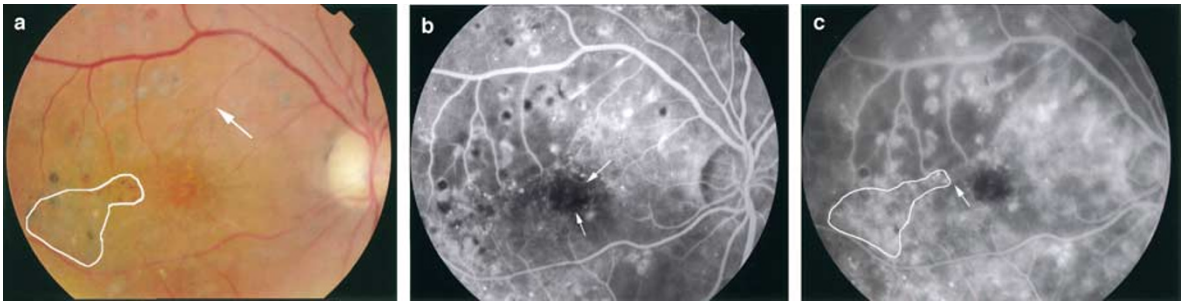

Figure 2 Photographs after temporal focal laser and visual deterioration. (a) Colour fundus photograph of the right macular region with the temporal area of retinal thickening (outlined) and the pre-existing area of superotemporal ischaemia (arrow). (b) Fluorescein angiogram of the right macular region showing an enlarged and irregular FAZ (arrows). (c) Fluorescein angiogram showing the treated area (outlined) and how this overlaps the temporal area of macular ischaemia (arrow).

unchanged and fluorescein angiography showed no foveal burn (Figure 2).

Comment

The ETDRS recommended macular laser treatment for eyes with CSME to reduce moderate visual loss and is supported by the Guidelines of The Royal College of Ophthalmologists on the management of diabetic retinopathy.[2] In particular, the ETDRS recommends, in areas of retinal thickening, focal laser treatment to areas of focal leakage and grid laser to areas of diffuse leakage and nonperfusion.

Rare complications can occur following routine treatment and include direct foveal burns, exacerbation of macular oedema with heavy extensive grid treatments, retinal haemorrhage owing to rupture of microaneurysms, development of choroidal neovascular membranes[3] and subretinal fibrosis.[4] Finally, laser treatment to the margin of an enlarged or irregular perifoveal capillary network should be specifically avoided[1] as it can further disrupt an already compromised FAZ and have an adverse effect on visual acuity.

This patient had evidence of nonperfusion to the superior macular prior to laser but the FAZ was intact. Fluorescein angiographic appearances were unchanged post-treatment. The aetiology of the visual loss in this case remains unclear but may be related to unrecognised disruption of macular capillaries supplying the perifoveal capillary network. These capillaries were at least 400 μm from the edge of the FAZ and adjacent to pre-existing ischaemia. It is possible that their function was compromised due to the adjacent ischaemia and that the focal laser treatment exacerbated this. Fluorescein angiography should be performed in all patients undergoing macular laser for diabetic macular oedema. Special consideration should be given to the treatment of areas of leakage adjacent to capillary network non-perfusion because of the risk of indirectly compromising the capillary network perfusion.

Acknowledgement

We would like to thank Richard Poynter, medical photographer at Moorfields.

References

1 Early Treatment Diabetic Retinopathy Study Research Group. Early photocoagulation for diabetic retinopathy ETDRS report number 9. *Ophthalmology* 1991; **98**(Suppl): 766–785.
2 Guidelines for Diabetic Retinopathy. London: The Royal College of Ophthalmologists, 1997.
3 Olk RJ, Lee CM. *Diabetic Retinopathy: Practical Management*. JP Lippincott: Philadelphia, 1993.
4 Guyer DR, D'Amico DJ, Smith CW. Subretinal fibrosis after laser photocoagulation for diabetic macular edema. *Am J Ophthalmol* 1992; **113**: 652–656.

EC Richardson, J Patel and PG Hykin

Moorfields Eye Hospital
City Road
London EC1V 2PD, UK

Correspondence: EC Richardson
Tel: +20 7566 2057
Fax: +20 7566 2972
E-mail: E0102richa@aol.com

Sir,

Ophthalmia nodosa secondary to tarantula hairs

Eye (2003) **17,** 433–434. doi:10.1038/sj.eye.6700335

Tarantulas are large spiders covered in a layer of velvety hairs, found in tropical and subtropical areas. Tarantulas belong to the Theraphosidae family, a subgroup of Mygalomorph[1] (Greek word mygale, field mouse!). They are regarded as the largest and hairiest spiders of all and therefore, to most enthusiasts, the most interesting species to keep. Over the last decade, they have become increasingly fashionable as pets in Britain, because tarantulas are easily available, interesting to watch, relatively slow moving, have a long life span (up to 20 years) and tolerate a certain amount of gentle handling.

All tarantulas are venomous and those that have less potent venom are particularly popular, including Chilean Rose (*Grammostola spatulatus),* Chilean Beautiful *(Grammostola Cala*), Mexican Red-Kneed (*Euathlus smithi*), etc.[1] Their defence relies mostly on painful bites with their erect fangs and barbed urticating hairs (hairs that can cause ocular, dermatological and respiratory irritation). These hairs are released when the tarantula panics by any gesture, seen as provocation. The tarantula raises its hind legs and vibrates rapidly across the dorsum of the abdomen,[2,3] where these hairs are located at a density of approximately 10 000/mm.[2] This stimulates a shower of urticating hairs spraying towards the predator and allows the spider to escape.

Furthermore, many tarantulas are notoriously unpredictable! This potentially harmful defensive behaviour is often not the highlight to the buyer.

Case report

A 14-year-old boy presented to our department with a 4-month history of intermittent ocular irritation. He had a few courses of topical antibiotics prescribed by his general practitioner, with no improvement. He had good general health with no suggestion of juvenile arthritis or autoimmune disorders. On direct questioning, we discovered his hobby was to look after his pet which he very proudly acquired 2 years ago—a tarantula—Chilean Rose. He recorded the tarantula had bitten him ('just like a wasp sting') and sprayed hairs to him on occasions during handling, which did not cause any concern.

On examination, his vision was 6/6 in both eyes. He had no facial erythema or lid swelling. There was mild follicular conjunctivitis, multiple tiny subepithelial corneal opacities, with mild anterior uveitis in the left eye, associated with a couple of large 'mutton fat' keratic precipitates. Careful slit-lamp biomicroscopy of the cornea anterior segment and gonioscopy did not show any tarantula hairs. Fundoscopy showed an area of inactive chorio-retinal lesion in the peripheral retina, with no evidence of vitritis.

He was started on topical steroid treatment and responded well. Treatment is continuing. He remained completely symptom-free. Family and patient have been made aware of the potential chronicity [2–4] of his eyes condition, but they have no intention of giving up the tarantula.

Comment

Ophthalmia nodosa was first described in 1904 as granulomatous nodules found on iris and conjunctiva in response to capillary hairs,[5] and now it is a term to describe an essentially ocular reaction to vegetation or insect hairs. [2,3,6] There are four types of urticating hairs, distinguishable by their pattern of barbs. Type III hairs are approximately 0.1–1.3 mm long, have shafts with a sharp-pointed head and numerous barbs. They travel like arrows and are the most apt to penetrate deeply into tissues. Tarantula hairs resemble sensory setae of caterpillars, are both type III,[2,6,7,11] and they are known to migrate relentlessly and cause multiple foci of inflammation in all levels of the eye.[2,6–8] Tarantula hairs