Localised unilateral blepharochalasis

R M Manners, J R O Collin

In 1807 Beers referred to a condition which was later described as blepharochalasis (Gk = eyelid slackening) by Fuchs. The condition consists of recurrent attacks of oedema of the eyelids and in chronic conditions the skin becomes reddish, thin, and redundant. Periorbital sequelae include ptosis, pseudoepicanthic fold with underlying nasal fat pad atrophy, blepharophimosis, proptosis, lower lid malpositions, lacrimal gland prolapse, and cysts. The peak age of onset is in the teens and twenties with no sex predominance. The attacks of oedema occur with varying frequency during the active/early stage (monthly – annually) and last a few hours to a week or more. They continue for many years but gradually become less frequent until a quiescent/late stage is reached. Unilateral blepharochalasis has been reported to be quite rare. Histological changes in blepharochalasis include epidermal atrophy, a loss of elastin fibres, and dermal vasculitis with an increase in the size of the vessels and perivascular cuffing. We present a case of localised, unilateral blepharochalasis which was confined solely to the lateral half of the left upper and lower eyelids.

Case report

A 37-year-old man presented with a history of episodes of swelling of the lateral half of the left upper and lower eyelids which had started in his late teens and recurred initially every 2–3 months but more recently less frequently. The swelling was most prominent in the lateral half of the eyelids, lasted up to 24 hours, and was generally accompanied by a red discoloration of the lateral conjunctiva. The episodes were frequently associated with an upper respiratory tract infection. Medical history was normal with no history of atopy.

On examination during a quiet phase, the right eye showed no abnormality. The skin overlying the left lateral canthus had a reddish discoloration and was thin, telangiectatic, and redundant in the lateral aspect of the upper and lower eyelids (Fig 1). There was rounding of the lateral canthus with marked lateral canthal tendon laxity. The horizontal palpebral apertures measured 29 mm on the right and 25 mm on the left. There was no change in the area on Valsalva manoeuvre.

The lateral canthal region was explored under local anaesthesia via a lower lid blepharoplasty approach. No vascular lesion was found. The lateral canthus was reattached to the peristomeum of the lateral wall of the orbit using a 5/0 non-absorbable suture. Redundant skin was excised from the lateral half of the lower upper eyelids and the wound sutured with 6/0 non-absorbable sutures.

Examination of the excised skin showed findings consistent with blepharochalasis and no evidence of a vascular malformation. The result of 6 weeks postoperatively is shown in Figure 2. No attacks of swelling have occurred during the postoperative period.

Comment

Lateral canthal tendon laxity has been reported to arise in blepharochalasis owing to a dehiscence...
Superior oblique myokymia – a topical solution?

Kim Bibby, James S Deane, David Farnworth, John Cappin

Superior oblique myokymia (SOM) is a rare ocular motility disorder characterised by a monocular high frequency, low amplitude cyclotorsional tremor. It occurs intermittently, giving rise to sometimes obstructive symptoms of oscillopsia and diplopia.

Case report
A 50-year-old woman presented to the eye casualty department, Leicester Royal Infirmary with a 13-month history. She described oscillopsia and a feeling of tremor in her left eye. The symptoms occurred periodically and were particularly troublesome when she was reading. On examination, visual acuity was 6/6 unaided. Anterior segments, pupil reflexes, and funduscopy were unremarkable. Lid position was normal and she had a full range of ocular movements. Slit-lamp biomicroscopy disclosed a cyclotorsional tremor of fine amplitude with a vertical element. This was intermittent but could be induced by her looking down and to the right.

We prescribed betaxolol drops twice daily to the left eye for 1 month, and reviewed the woman after 2 months. In the 4 weeks she had been using the drops she had been asymptomatic, but her problem returned within 1 week of stopping treatment. On examination left SOM was apparent. She recommenced topical betaxolol and when reviewed at 3 and 6 months was totally asymptomatic. No SOM was observed on six separate occasions during these two outpatient appointments.

Comment
Superior oblique myokymia was described as a distinct entity by Hoyt and Keane as a benign, periodic unioocular vertical and rotary microtremor. It is rarely associated with serious underlying pathology, but can be disturbing symptomatically. Susac and Smith report successful elimination of symptoms with the use of carbamazepine, and go on to advocate superior oblique myotony for intractable cases, or patients who cannot tolerate the drug. Propranolol is cited as a pharmacological alternative by Tyler and Ruiz.

Leigh et al describe success with the use of a topical β blocker in one patient. Betaxolol has weak membrane stabilising effects compared with other β blockers and is unlikely to work topically. However, it has a bioavailability of 89% and demonstrates significant reduction in finger tremor when administered parenterally. We hypothesise that enough betaxolol may be absorbed systemically to eliminate SOM. The drug is cardioselective and has fewer of the side effects associated with propranolol or carbamazepine. It may be of use as a first line treatment in those patients whose symptoms are intolerable.

NOTICES

Photonics West '95
The International Society for Optical Engineering (SPIE) will hold a conference entitled 'Photonics West '95' on 4–10 February 1995 at the San Jose Convention Center, San Jose, California, USA. This meeting consolidates three established California meetings, OE/LASE, Biomedical Optics, and the IS&T/SPIE Symposium on Electronic Imaging Science and Technology. Further details: SPIE, PO Box 10, Bellingham, WA 98227-0010, USA. (Tel: 206/676-3290; Fax: 206/647-1445.)

British College of Optometrists
The centenary conference of the British College of Optometrists will be held at Churchill College, Cambridge on 5–8 April 1995. Further details: BCO Conference Secretariat, Conference Contact, 42 Devonshire Road, Cambridge CB1 2BL. (Tel: 01223 323437; Fax: 01223 460396.)

1st International Conference on Ocular Aspects of Marfan’s Disease
The first international conference on ocular aspects of Marfan’s disease will be held at the University of Munster, Germany on 8 April 1995. Further details: H Gerding, H Busse, C Schroeter, Marfan Conference, Postfach 2322, 39013 Hamm, Germany. (Tel: 0049 2381-271746; Fax: 0049-2381-271743.)

United Kingdom Transplant Support Service Authority
There will be a ‘Cornell Transplant Meeting’ on 10 April 1995 to be held at the Postgraduate and Health Sciences Building, Central Manchester Trust. Further details: Julia Warr, UK Transplant Support Service Authority, Fox Den Road, Stoke Gifford, Bristol BS12 6RR. (Tel: 0117 9757555; Fax: 0117 9757577.)

Association for Research in Vision and Ophthalmology
The annual meeting of the Association for Research in Vision and Ophthalmology (ARVO) will be held on 14–19 May 1995 at the Fort Lauderdale/Broward County Convention Center, Fort Lauderdale, Florida, USA. Further details: Anne Meltzer, the ARVO Central Office, 9650 Rockville Pike, Bethesda, MD 20814-3998, USA. (Tel: (301) 571-1844; Fax: (301) 571-8311.)

4th International Symposium on Ocular Circulation and Neovascularisation

Vh International Symposium on Sjögren’s Syndrome
The Vh International Symposium on Sjögren’s syndrome will be held on 15–17 June 1995, in Noordwijkerhout, the Netherlands. Further details from: Conference Secretariat: A A Kruize, Department of Rheumatology F02.223, University Hospital Utrecht, p/o Box 85500, 3508 GA, Utrecht, the Netherlands. (Tel: +31 30 507357; Fax: +31 30 523541.)

International Society for Clinical Electrophysiology of Vision
The 33rd ISCEV symposium will be held in Athens, Greece, 16–20 June 1995. The congress is organised by the International Society for Clinical Electrophysiology of Vision. Further details: Secretariat, Erasmus Conference Centre, International Congress Organisers, 227 Kifissias Ave, 145 61 Kifissia, Greece. (Tel: (01) 6125022/3, 8054004; Fax: (01) 6125021.)

Corrections
We regret that there was an error in the paper by R M Manners and J R O Collin that appeared in the November issue of the journal (1994; 78: 881–2). Figures 1 and 2 were reversed but the captions were correct. Figure 2 showed a preoperative appearance of the patient and Figure 1 a postoperative picture.

The authors (Abiose et al) wish to make a correction to their blindness data presented in the paper that appeared in the January issue of the journal (1994; 78: 8–13). Since the manual analysis of visual field data set, which resulted in their presentation of data on those blind by virtue of visual field constriction, they have now entered the Friedmann field data onto microcomputer.

After data checking and a repeat analysis they have found 14 individuals who had been incorrectly classified as blind since they had seen one or more Friedman test points at 10° or more in the better eye.

Although these corrections do not change substantially the pattern and prevalence figure, they do alter to an important degree the visual field constriction data. In view of the rarity of data on this topic the authors feel that it is important to correct their report. They originally reported 42 individuals to be blind by visual field constriction (a prevalence of 0.6%). The correct figure for blindness by visual field constriction is now 28 individuals (0.4%). Three of those previously classified as blind by visual field constriction are now classified as visually impaired by acuity criteria, and a further three are now classified as unilaterally blind. Eight individuals are now classified as sighted. The table below gives pinhole acuity and cause of ocular pathology for each eye in the 14 individuals concerned.

A revised version of the abstract published with the paper, corrected to take account of these changes, is given below. The authors apologise for this correction. Further details of their visual field findings will be published shortly.

Revised abstract
During a field trial of ivermectin, 6831 people aged 5 years and above living in 34 mesoendemic onchocercal communities in Kaduna State, northern Nigeria, were examined for ocular disease. Visual function assessments included tests of visual acuity and visual fields. 185 individuals (2.7%) were bilaterally blind by acuity criteria with a further 28 blind by field constriction. The overall prevalence of blindness was 3.1%. A further 118 individuals were visually impaired by WHO criteria. Examination for the cause of blindness revealed that 43% of eyes in bilaterally blind patients were blind due to onchocerciasis. A further 11% were blind from optic atrophy of which in 24% is probably ocular in origin. Glaucoma was the next most common cause of blind eyes in the bilaterally blind (11%). Only 6% of eyes were blind from cataract as the primary cause. In the visually impaired population cataract was the most common primary cause of impaired/blind eyes (31%), followed by onchocerciasis (19%).

<table>
<thead>
<tr>
<th>Acuity left</th>
<th>Pathology</th>
<th>Acuity right</th>
<th>Pathology</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6/36</td>
<td>Onchocerciasis</td>
<td>PL</td>
<td>Chronic inflammatory disease</td>
</tr>
<tr>
<td>2</td>
<td>6/36</td>
<td>Onchocerciasis</td>
<td>6/36</td>
<td>Reclassify as visually impaired</td>
</tr>
<tr>
<td>3</td>
<td>6/36</td>
<td>Onchocerciasis</td>
<td>PL</td>
<td>Reclassify as visually impaired</td>
</tr>
<tr>
<td>4</td>
<td>NPL</td>
<td>Trachoma</td>
<td>6/9</td>
<td>Reclassify as unilaterally blind</td>
</tr>
<tr>
<td>5</td>
<td>6/18</td>
<td>Onchocerciasis</td>
<td>PL</td>
<td>Reclassify as unilaterally blind</td>
</tr>
<tr>
<td>6</td>
<td>6/9</td>
<td>Onchocerciasis</td>
<td>NPL</td>
<td>Reclassify as unilaterally blind</td>
</tr>
<tr>
<td>7</td>
<td>6/10</td>
<td>Glaucoma</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>8</td>
<td>6/18</td>
<td>Optic atrophy</td>
<td>6/18</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>9</td>
<td>6/36</td>
<td>Onchocerciasis</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>10</td>
<td>6/9</td>
<td>Retinitis pigmentosa</td>
<td>6/18</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>11</td>
<td>6/9</td>
<td>Onchocerciasis</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>12</td>
<td>6/9</td>
<td>Optic atrophy</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>13</td>
<td>6/9</td>
<td>Optic atrophy</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
<tr>
<td>14</td>
<td>6/9</td>
<td>Onchocerciasis</td>
<td>6/9</td>
<td>Reclassify as sighted</td>
</tr>
</tbody>
</table>