Phototherapeutic keratectomy re-treatment for recurrent corneal erosion syndrome

R Maini, M S Loughnan

Aims: Phototherapeutic keratectomy with an excimer laser is commonly used to treat recurrent corneal erosion syndrome. The aim of this study was to determine the success of a repeat phototherapeutic keratectomy for patients with persistent macroerosions following initial treatment with phototherapeutic keratectomy.

Methods: Eight patients who failed primary phototherapeutic keratectomy for recurrent corneal erosion syndrome were treated with repeat phototherapeutic keratectomy. All patients were treated with a superficial therapeutic ablation profile with a Visx, Nidek, or Summit 193 nm excimer laser. All patients were treated for both their primary treatment and re-treatment by the same surgeon. Retrospective analysis of the case records of all patients requiring re-treatment was supplemented with a telephone interview.

Results: Over a 5-year period (October 1995 to October 2000) 76 eyes were treated for recurrent erosion syndrome with phototherapeutic keratectomy. All patients had documented macroerosions and had failed previous treatment with a lubricant at night. Eight eyes (11%) continued to have macroerosions after this initial treatment; all opted for re-treatment with phototherapeutic keratectomy. Following re-treatment none reported symptoms consistent with a macroerosion. Six of eight patients (75%) are now symptom-free; 2/8 (25%) have an occasional foreign body sensation relieved by lubricants. Follow-up ranged from 9–60 months with a mean of 25.5 months.

Conclusion: Re-treatment with phototherapeutic keratectomy appears to be successful for patients with macroerosions complicating recurrent corneal erosion syndrome who have failed conservative management with ocular lubricants and a primary phototherapeutic keratectomy.

Recurrent corneal erosion syndrome (RCES) is a relatively common condition and may be classified as either dystrophic (dystrophic-RCES) where it occurs associated with an anterior corneal dystrophy, or non-dystrophic (ndRCES) where it occurs following superficial corneal trauma (post-traumatic RCES) or unrelated to any pre-existing corneal disease or past history of trauma (idiopathic RCES). Corneal dystrophies commonly associated with RCES include epithelial basement membrane (Cogan's microcystic or map dot fingerprint dystrophy), Reis-Buckler's, lattice, and granular dystrophies. Symptoms include tearing, pain, and photosensitivity and occur more commonly on waking. RCES symptoms may be classified as microerosions with milder symptoms of shorter duration (more common in dystrophy related RCES) or macroerosions, presenting with symptoms of longer duration—sometimes up to several hours, and more common in non-dystrophic RCES (ndRCES). Dystrophic RCES often leads to bilateral symptoms.

A variety of treatments are available for RCES, reflecting the lack of total efficacy of any single treatment. In the first instance conservative therapy with ocular lubricants is frequently used. More active intervention with a bandage contact lens, epithelial debridement, superficial keratectomy with a diamond burr, anterior stromal puncture, and Nd:YAG laser have been used with varying success. An associated meibomian gland dysfunction has been postulated with reduction in symptoms following treatment with oral doxycycline.

Recently, several authors have reported the use of excimer phototherapeutic keratectomy (PTK) for this condition.

Reported success rates for this treatment have varied from 69% to 86%. Recurrence rates are likely to be higher when PTK is used for dystrophic RCES.

There is little in the literature regarding re-treatment with PTK following primary treatment with PTK for RCES; one report demonstrated some success with re-treatment with shallow ablations (3 µm and 5 µm) following epithelial debridement with no detail regarding follow up. We present our results of re-treatment of ndRCES with PTK following primary PTK.

Methods

Over a 5-year period (October 1995 to October 2000) 76 eyes of 69 patients with either dystrophic or ndRCES underwent PTK. All the patients had documented macroerosions and had previously failed conservative management with ocular lubricants. All patients were treated by a single surgeon (MSL) using a superficial therapeutic ablation profile using a Visx, Summit, or Nidek EC5000 193 nm excimer laser. All treatments were performed following manual corneal epithelial debridement of the affected epithelium. This was defined by gentle debridement to remove only that area of the epithelium that was not firmly adhered to the underlying stroma. This usually involved most of the corneal epithelium including any area of microcysts. Ablation depth varied from 7.5 µm to 10 µm depending on the individual laser's parameters. All patients had topical chloramphenicol four times daily for 1 week and fluoromethalone acetate 1% four times daily for a week and twice daily for a week after ablation. In addition, all patients were asked to use a paraffin-based lubricant (Lacrilube) nightly for a month after treatment.

Eight eyes of eight patients had recurrent macroerosions following this initial treatment and all patients opted for re-treatment with PTK. All of these eight patients had ndRCES.

Retrospective analysis of case records of all patients requiring re-treatment was performed. This was supplemented by further follow up with a telephone interview. Patients were asked about the presence of any symptoms of discomfort, tearing, or photosensitivity and the use of any ocular medication.

Results

Of eight patients requiring re-treatment six (75%) were female. Age ranged from 38 to 79 years (mean 55 years). Date
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Analysis of each re-treatment case

**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Trauma</th>
<th>Date PTK re-treatment</th>
<th>Ablation profile</th>
<th>Follow up (months)</th>
<th>Symptoms</th>
<th>Date PTK</th>
<th>Ablation profile</th>
<th>Ablation profile</th>
<th>Ablation profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>49</td>
<td>F</td>
<td>Yes</td>
<td>18/4/96 (S)</td>
<td>6 mm, 7.5 µm + *3</td>
<td>40</td>
<td>Symptom free</td>
<td>20/2/96 (V)</td>
<td>6.5 mm, 7.5 µm + 4 mm, 8 µm eccentric</td>
<td>32</td>
<td>Symptom free</td>
</tr>
<tr>
<td>2</td>
<td>51</td>
<td>F</td>
<td>Yes</td>
<td>18/6/98 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>34</td>
<td>Nil</td>
<td>18/2/98 (N)</td>
<td>9 mm, 8 µm + 3 mm, 8 µm</td>
<td>10</td>
<td>Nil</td>
</tr>
<tr>
<td>3</td>
<td>42</td>
<td>F</td>
<td>Yes</td>
<td>21/5/98 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>20</td>
<td>Symptom free</td>
<td>14/5/99 (N)</td>
<td>7.5 mm, 10 µm + 2 mm, 8 µm</td>
<td>10</td>
<td>Symptom free</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>M</td>
<td>No</td>
<td>3/6/98 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>4</td>
<td>Nil</td>
<td>4/8/98 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>12</td>
<td>Nil</td>
</tr>
<tr>
<td>5</td>
<td>79</td>
<td>F</td>
<td>No</td>
<td>19/10/98 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>10</td>
<td>Symptom free</td>
<td>11/5/00 (N)</td>
<td>7.5 mm, 8 µm + 3 mm, 10 µm</td>
<td>18</td>
<td>Foreign body sensation</td>
</tr>
<tr>
<td>6</td>
<td>38</td>
<td>M</td>
<td>Yes</td>
<td>5/8/99 (N)</td>
<td>9 mm, 8 µm + 2 mm, 8 µm</td>
<td>9</td>
<td>Symptom free</td>
<td>12/10/99 (N)</td>
<td>7.5 mm, 8 µm + 3 mm, 10 µm</td>
<td>18</td>
<td>Foreign body sensation</td>
</tr>
<tr>
<td>7</td>
<td>57</td>
<td>F</td>
<td>No</td>
<td>17/2/00 (N)</td>
<td>7.5 mm, 8 µm + 2 mm, 8 µm</td>
<td>3</td>
<td>Foreign body sensation</td>
<td>6/7/00 (N)</td>
<td>9 mm, 8 µm + 3 mm, 10 µm</td>
<td>10</td>
<td>Symptom free</td>
</tr>
<tr>
<td>8</td>
<td>57</td>
<td>F</td>
<td>No</td>
<td>13/10/99 (N)</td>
<td>7.5 mm, 8 µm + 2 mm, 8 µm</td>
<td>9</td>
<td>Foreign body sensation</td>
<td>13/10/99 (N)</td>
<td>7.5 mm, 8 µm + 2 mm, 8 µm</td>
<td>18</td>
<td>Foreign body sensation</td>
</tr>
</tbody>
</table>

**Note:** Where a "string of pearls" configuration was used the number of ablations ("pearls"), diameter, and depth of each ablation is given. Ablation diameter is given in mm and depth of ablation in µm.

**DISCUSSION**

Our results indicate that repeat PTK for ndRCES can be used successfully to alleviate symptoms of macroerosions despite initial treatment failure. The patients treated represent a subgroup of RCES patients who have recalcitrant disease, having failed conservative therapy and an initial PTK. Limitations of our study include the small number of patients requiring re-treatment and the use of a telephone survey to supplement case record analysis to define the extent of symptoms and usage of lubricants. Despite this we were encouraged to record complete symptomatic relief in the majority of patients (6/8) and a qualified success in the remaining two patients. Ohman et al. also found re-treatment to be of some success in a group of nine patients with unspecified follow-up.

No complications from either primary or repeat treatment were noted in our patients. This is likely to be due to the very shallow depth of each ablation. Using a PTK it is possible to treat affected areas that encroach on the visual axis with excellent visual rehabilitation. This is in contrast with other forms of interventional treatment—notably, anterior stromal puncture where postoperative scarring may limit treatment of the central cornea. One concern even with a PTK treatment profile is the possibility of inducing a refractive change. This appears to be less likely with a shallow ablation and modern PTK treatment profiles as opposed to those with older broad beam lasers. We did not specifically address the issue of refractive changes in our patients as several patients were treated during an attack and, as such, precise measurement of the patients' refraction preoperatively was not possible. Despite this none of our eight patients required a change in their pre-existing distance or near spectacles in the first several months postoperatively.

There is some evidence that upregulated matrix metalloproteinase activity alters the epithelial basement membrane in RCES with modulation of subepithelial fibronectin and laminin activity. Both are closely involved in the attachment of epithelial basal cells to the underlying basement membrane and poor adhesion secondary to these factors may give rise to the signs and symptoms of RCES. The mechanism of action of a PTK may be to strengthen the adhesion of the basal epithelial cells to the underlying tissue, this adhesion mediated via anchoring fibrils and hemidesmosomes. Animal studies have confirmed upregulation of both anchoring fibril constituents and hemidesmosomes following excimer ablation.

An in vivo confocal study has shown abnormal anterior stromal extracellular matrix in RCES. As the ablation is so shallow the primary PTK in the eight re-treated patients may have been insufficient to remove this tissue to allow adequate adhesion between the basal epithelial layer and underlying tissue. Re-treatment may have resulted in sufficient removal of tissue to allow effective adhesion to occur.

In conclusion, PTK appears to be an effective treatment for alleviating symptoms in the majority of patients with RCES. A repeat PTK appears to be effective in alleviating symptoms in those patients who fail a primary treatment.
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