Phototherapeutic keratectomy re-treatment for recurrent corneal erosion syndrome

R Maini, M S Loughnan

**SCIENTIFIC CORRESPONDENCE**

**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 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.

**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.

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**RESULTS**

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 which 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 then 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 after this initial treatment and all patients opted for re-treatment with PTK. All of these eight patients had ndRCES.

**RESULTS**

Eight of patients requiring re-treatment six (75%) were female. Age ranged from 38 to 79 years (mean 55 years). Date
of primary PTK, length of symptom free period, and date of re-treatment with follow up are documented in Table 1. Four of eight had a history of previous trauma (post-traumatic RCES) to the affected eye and the other four had no predisposing factors (idiopathic RCES). None of this group had bilateral disease. The period in months until failure of the primary PTK were 1, 1, 2, and 2.5 for the patients with a history of trauma and 1, 2, 5, and 8 for patients with no such history (Table 1). The ablation profile for each re-treatment is documented. Following re-treatment no patients reported symptoms consistent with a macroerosion. Six of eight (75%) patients were symptom free with no ocular medication and two of eight (25%) had occasional discomfort with a foreign body sensation; this was relieved by ocular lubricants.

**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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