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Tissue Necrosis Following Diode Laser-Assisted Transcanalicular Dacryocystorhinostomy

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Abstract

Advantages of transcanalicular laser-assisted dacryocystorhinostomy (TCDCR) over conventional external and endonasal dacryocystorhinostomy (DCR) have been purported to include decreased operating time, reduced morbidity, enhanced cosmesis, avoidance of general anesthesia, and a shorter recovery time. However, one case of skin necrosis has recently been reported to have occurred following diode laser-assisted TCDCR, and we now report three additional cases that were evaluated by the Ophthalmic Plastic Surgery services at the University of North Carolina and the University of California, San Francisco. Three patients developed full-thickness tissue necrosis over the medial canthus following TCDCR, and two of these patients experienced persistent tissue breakdown at the site following reconstructive repair.

In 1990, Massaro, et al. reported the first use of a laser in dacryocystorhinostomy (DCR).¹ Over the following decades, a multitude of new laser-assisted DCR techniques were reported, including the direct delivery of laser energy to the wall of the lacrimal sac via passage of a fiberoptic probe through a canaliculus.^{2,3,4} This technique has been termed transcanalicular laser-assisted DCR (TCDCR), and its purported advantages over conventional external and endonasal DCR include decreased operating time, reduced morbidity, enhanced cosmesis, avoidance of general anesthesia, and a shorter recovery time. While the success rate of TCDCR has consistently been demonstrated to be lower than external or endonasal DCR, the procedure has generally been considered safe.^{5,6,7,8} However, in 2010, Yenzi et al. published the first case of tissue necrosis following TCDCR.⁹ We herein report three additional cases of full-thickness tissue necrosis following TCDCR. One case was evaluated by the Ophthalmic Plastic Surgery service at the University of North Carolina (UNC) in 2010, and the other two were referred from a single

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outside practice to the Ophthalmic Plastic Surgery service at the University of California, San Francisco (UCSF) during a two month period in early 2011.

Case #1

A 63 year old Caucasian female was evaluated by the Ophthalmic Plastic Surgery clinic at the University of North Carolina in March, 2010 for a complaint of 6 months of constant epiphora in the right eye. She had no history of dacryocystitis, sinusitis, facial trauma, or eyelid, nasal, or sinus surgery. Evaluation confirmed a diagnosis of involuntal NLDO, and TCDCR was performed in the right eye in April, 2010. A 980 nm diode laser (ITI, LCC; Middleton, CT) was used in single pulse mode with 10 W/pulse and a pulse duration of 9.5 ms. Per the operative report, 1393 J of laser energy were delivered to create a full-thickness ostium of the lacrimal sac, lacrimal bone, and nasal mucosa. An anterior middle turbinectomy was performed, and a Merocel nasal pack (Medtronic, Inc.; Minneapolis, MN) and bicanalicular Crawford silicone tube were placed. No complications were noted. By 6 days post-operation, the patient had developed mild pain and tenderness over the right lacrimal sac, and medial canthal erythema was noted. The nasal pack was removed, the nasal exam was felt to be within normal limits for the post-operative period, and the patient was started on Vigamox ophthalmic solution (Alcon, Inc.; Fort Worth, TX) and Keflex (Eli Lilly & Co.; Indianapolis, IN). By day 10, the patient was noted to have "pale, devitalized tissue" at the medial canthus. Following debridement of the tissue, a small fistulous tract from the medial canthus skin to the lacrimal sac was observed. On post-operative day 13, the patient underwent surgical drainage of the medial canthal abscess with further debridement of the devitalized tissue and closure of the fistula. The bicanalicular stent was felt to be in proper position and was left in place. Tissue specimens obtained during the operation demonstrated necrotic tissue with inflammation. Following this procedure, the patient's wounds healed uneventfully, and the stent came out spontaneously at home during postoperative week 18. However, following this evaluation, the patient complained of persistent epiphora in the right eye.

Case #2

An 83 year old Caucasian female attended our clinic in January 2011 for evaluation of a full-thickness skin defect in the left medial canthal region that developed following TCDCR with bicanalicular stenting at an outside practice. The patient had received radioactive iodine therapy (RAI) for metastatic thyroid carcinoma in 1986 and underwent Mohs micrographic excision with reconstruction for a basal cell carcinoma at the left medial canthal region in 2007. In early 2010, she developed epiphora and blurred vision in her left eye and was diagnosed with involuntal nasolacrimal duct obstruction (NLDO). Left-sided TCDCR was performed in October, 2010. An 810 nm diode laser (ITI, LLC; Middletown, CT) was used in single pulse mode with 12 W/pulse and a pulse duration of 9.5 ms. Per the operative report, 1300J of laser energy were delivered to create a full-thickness ostium of the lacrimal sac, lacrimal bone, and nasal mucosa. Cottonoids soaked in mitomycin C (0.3 mg/mL) were held in place at the ostium site for 3 minutes. A bicanalicular Crawford silicone tube was placed, and no complications were noted. As part of a standard post-operative regimen, the patient was treated with Tobradex ophthalmic suspension (Alcon, Inc.; Fort Worth, TX) and

Keflex (Eli Lilly & Co.; Indianapolis, IN). At 1 month, the patient had developed skin breakdown in the left medial canthal region which progressed to full thickness necrosis by two months post-TCDCR (see Figure 1A). Neosporin ointment (Pfizer, Inc.; New York, NY) was applied to the affected skin, and she was referred to the Ophthalmic Plastic Surgery service at UCSF. The silicone tubes were removed during the post-operative course by the outside ophthalmologist. At 3 months post-TCDCR, the patient underwent debridement of the defect and repair with a glabellar flap. An intra-operative incisional biopsy of the defect border demonstrated basal cell carcinoma; however, it was felt that the debridement of the surrounding tissue had likely been sufficient to remove residual basal cell carcinoma. Breakdown of the flap occurred at 8 weeks following her repair, and a decision was made to allow the wound to granulate without further intervention (see Figure 2A). At the most recent observation at 2 months status post repair, the wound had still not fully healed.

Case #3

A 63 year old Caucasian female attended our clinic in March 2011 for evaluation of a full-thickness tissue defect in the left medial canthal region that developed following TCDCR with bicanalicular stenting at the same outside practice. The patient had complained of 7 years of epiphora in her left eye and was diagnosed with involutinal NLDO. Left-sided TCDCR was performed in January, 2011. The same 810 nm diode laser (ITI, LLC; Middletown, CT) was used in single pulse mode with 12 W/pulse and a pulse duration of 9.5 ms. Per the operative report, 900 J of laser energy were delivered in an attempt to create an ostium; however, the surgeon noted difficulty in penetrating the tissue and abandoned laser treatment after noting blanching of the skin at the medial canthus. Cold saline compresses were applied to the area, and the procedure was completed via a non-laser-assisted endonasal approach with placement of a bicanalicular Crawford silicone tube. The appearance of a “first-degree burn” was noted over the medial canthal area at post-operative day 1, and skin breakdown was noted at post-operative day 7. Neosporin ointment (Pfizer, Inc.; New York, NY) was applied to the affected skin, and she was treated with Tobradex ophthalmic suspension (Alcon, Inc.; Fort Worth, TX) and Keflex (Eli Lilly & Co.; Indianapolis, IN). By 5 weeks, she had developed full-thickness tissue necrosis in the left medial canthal region (see Figure 1B). The silicone tubes were removed during the postoperative course by the outside ophthalmologist, and the patient was referred to the Ophthalmic Plastic Surgery service at UCSF. The patient underwent debridement of the defect and repair with a glabellar flap at 10 weeks post-TCDCR. During this procedure, multiple bony erosions were observed in the frontal process of the maxilla, which appeared consistent with laser damage. There was breakdown of the flap at 6 weeks following her repair, and the patient elected to undergo a second repair, this time with an advancement flap at 7 weeks post-TCDR (see Figure 2B). By 2 weeks following the second reconstruction, the patient again experienced flap breakdown, and it was decided to allow the wound to granulate (see Figure 3). At the most recent observation at 7 months following the initial repair, the wound had still not fully healed.

Discussion

Previous studies of TCDCR have shown it to be a relatively safe procedure. Drnovsek-Olup and Beltram performed 126 successive diode laser-assisted TCDCRs and reported ipsilateral lower eyelid swelling and bruising as their only complications¹⁰, and in 118 consecutive diode laser-assisted TCDCR cases reported by Hong et al., the only complication was a single instance of “thermal injury to the canaliculus” which was repaired with canalicular suturing.⁵ Furthermore, a prospective series of 25 cases of diode laser-assisted TCDCR by Plaza et al., reported “minimal” complications including bleeding and difficulty in dilation and probing of the canaliculi.¹¹ However, the occurrence of a small fistula connecting the canalicular system with the skin in one case, and the cauterization of the superior canaliculus in another, was reported from a series 29 diode laser-assisted TCDCR procedures by Eloy, et al in 2000.¹² In 2010 Yeniad et al. reported a case of localized tissue necrosis which developed within 2 weeks following diode laser-assisted TCDCR.⁹ Repair with an advancement flap failed, and culture specimens taken from the nasal cavity grew *Aspergillus*. The patient was treated with oral amphotericin B, and the wound eventually healed by granulation. Thermal damage from the diode laser was suggested as a cause for the observed tissue necrosis.

Our cases represent the third, fourth, and fifth reports of tissue necrosis following TCDCR. The case at UNC was the first encountered by the treating physician. Similarly, prior to the two consecutive complicated cases evaluated at UCSF, the referring ophthalmologist reported 144 uncomplicated cases of TCDCR. In this series, the same company manufactured all three diode laser units, and the single unit used in two of the three procedures was sent for evaluation following the first complication. The company found no evidence of malfunction and returned the laser. Following the second complication, the laser was again sent back to the company. Damage to the diode was reported, although the specific malfunction was unspecified.

In each case, the power setting of the diode laser (10–12 W/pulse) was below the level of 15 W/pulse which has been reported to cause heat lateralization and tissue charring and is comparable to the power settings used in other published series of diode laser-assisted TCDCR.^{10,11,12,13} However, total laser energy is not consistently reported in published series of TCDCR, and in the series by Drnovsek-Olup and Beltram, no significant complications related to thermal damage were seen with a total laser energy range of 195–685 J (average=245 J). While Yeniad et al. do not report the laser energy used in their case of tissue necrosis, the total energy amounts used in the present three cases (1393 J, 900 J, and 1300 J) are notably higher than those in Drnovsek-Olup and Beltram. During the initial reconstruction procedure of patient #3, punctate erosions were observed on the frontal process of the maxilla, which is considerably thicker than the adjacent and immediately posterior lacrimal bone. It is possible that, in this patient, the suture of the frontal process and the lacrimal bone extended more posteriorly in the lacrimal fossa, resulting in the application of increased laser energy in order to create a bony opening. The high quantities of laser energy could have caused local tissue damage sufficient to impair the normal wound-healing process and lead to the eventual necrosis of the affected tissue.

Case #2 also featured several complicating factors that could have contributed to local tissue breakdown. One such factor is the intra-operative use of topical mitomycin C. Mitomycin C is an antimetabolite that has been used in TCDCRs in an attempt to prevent closure at the lacrimal osteotomy site.^{5,8,11,14} A prospective case series by Henson, et al. was the first study to examine the use of adjunctive mitomycin C in diode laser-assisted TCDCR, and the success rate of 87.5% at 12 months was relatively higher than many other reports of TCDCR outcomes.^{5,8,12,13} Of the 40 reported cases, there were no complications attributed to the use of mitomycin C.

Another confounding factor is the history of RAI for the treatment of thyroid carcinoma. In this procedure, systemically administered radioactive iodine molecules (I131) are preferentially metabolized by thyroid follicular cells, leading to their destruction. The association between RAI and NLDO has been well established, and Morgenstern, et al. demonstrated the presence of sodium-iodide symporters in the epithelium of the lacrimal sac.^{15,16,17,18} It is possible that the local concentration of radioactivity contributed to damage of the surrounding tissue in this case.

Finally, the patient reported a history of basal cell carcinoma at the left medial canthus. She had previously undergone resection of the lesion and was presumed to be cancer-free at the time of her TCDCR. Following the development of local skin necrosis, a biopsy of the wound margin was taken in our clinic and showed no evidence of malignancy. Another biopsy of the wound margin taken intra-operatively was positive for superficial basal cell carcinoma, which raises the possibility that recurrent or residual malignancy (or the previous surgical manipulation of the canthal area) could have contributed to the eventual tissue breakdown. Given the extensive removal of tissue during the repair, it was felt that any residual carcinoma was likely to have been excised, and the patient elected to pursue a course of watchful waiting.

The case reported by Yeniad, et al. was similarly complicated by a culture taken from the nasal cavity after the onset of necrosis that grew *Aspergillus*. However, it was unclear if the mold contributed to the development of tissue necrosis or if it was an opportunistic infection that established itself following breakdown of the skin barrier. The presence of confounders in these cases suggests that underlying factors may compromise the tissue and predispose patients to the development of skin necrosis following the application of laser energy, particularly in high amounts. However, an excessive amount of laser energy alone may be enough to cause necrosis, as evidenced by the absence of identified risk factors in cases #1 and #3.

Another striking feature of these cases is the failure of two of the three reconstructions. Despite two separate attempts at repair in case #3, there was recurrent flap breakdown, and the wound had not yet closed completely when last observed 7 months post-operatively. This implies a profound and lasting damage to local tissues, which is significant enough to impair the viability of healthy tissue transferred into the wound bed; notably, even tissue possessing a pedicle-based blood supply. The repair in case #2 was performed at 3 months post-TCDCR, and the first repair in case #3 was performed at 10 weeks post-TCDCR followed by a second attempt 7 weeks later. The exact timing of the repair in Yeniad, et al. is not

reported. The timing of the repair in both of these cases was influenced by the patients' expressed desires for improved cosmesis, and it is possible that further delays in the reconstruction attempts may have allowed the tissues to recover and increased the likelihood of success.

Lastly, skin necrosis may not be a complication unique to TCDCR. In 1998, Salour and Montazerin reported two cases of incision site skin necrosis following external DCR.¹⁹ However, one of these patients was found to have a post-operative blood glucose level of 355 mg/100 dl, and the other's surgeon reported the occurrence of "excessive cauterization" during the procedure. These factors were hypothesized to have contributed to poor wound healing, and, in contrast to the current cases, the one patient who pursued reconstructive surgery was successfully repaired with an advancement flap.

In conclusion, full thickness local tissue necrosis is a likely rare, but potential complication following diode laser-assisted TCDCR. Patients with certain underlying factors may be at greater risk; however, the presence of these factors does not appear necessary for the development of tissue necrosis. Additional research is necessary to determine the nature of the underlying tissue damage. Furthermore, once necrosis occurs, it may be refractory to subsequent reconstruction. These findings should be considered when comparing the risks and benefits of TCDCR versus other DCR procedures.

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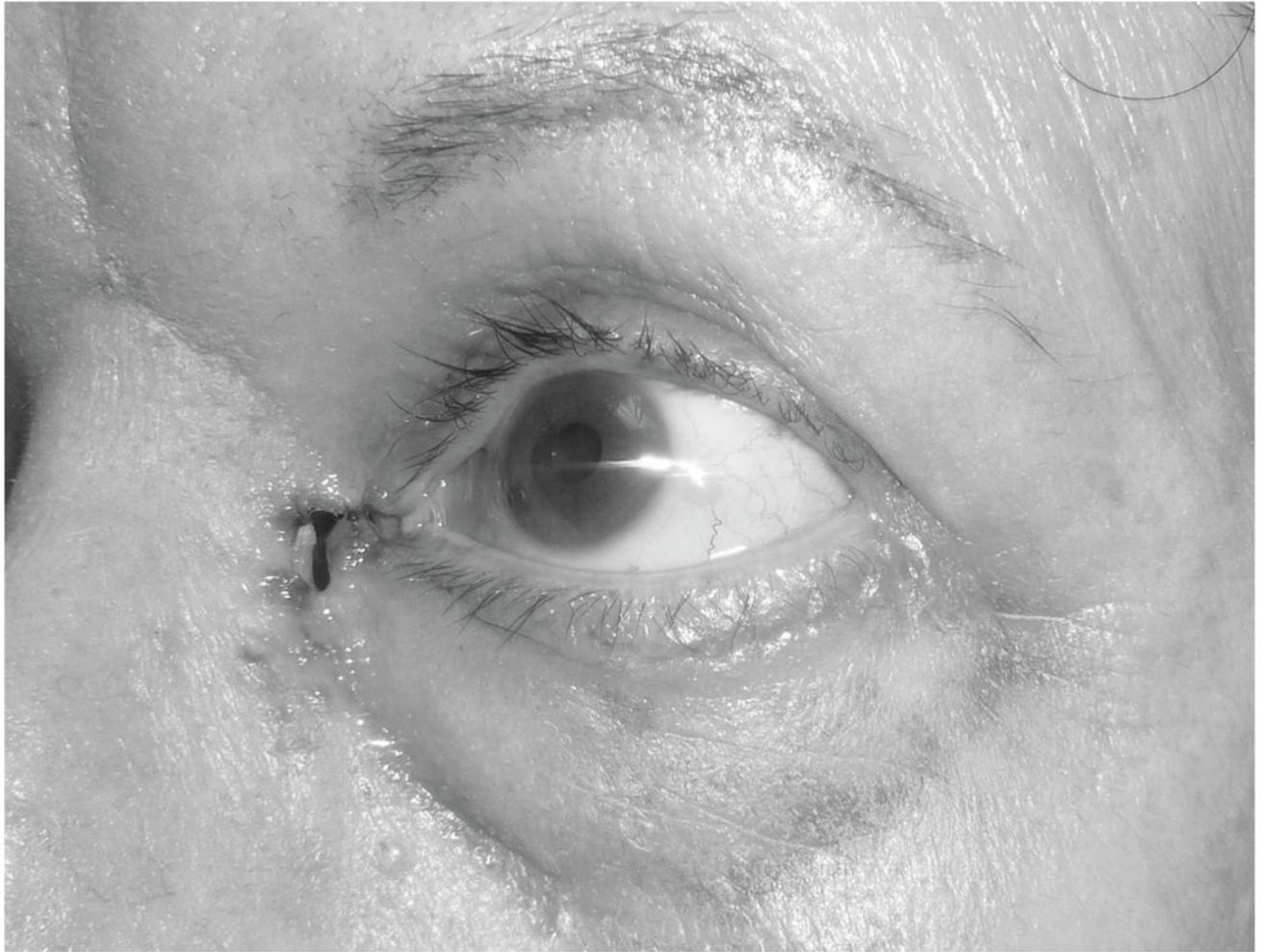


Figure 1.
(A) Patient #2; full-thickness skin necrosis over the left medial canthus. (B) Patient #3; full-thickness skin necrosis over the left medial canthus.

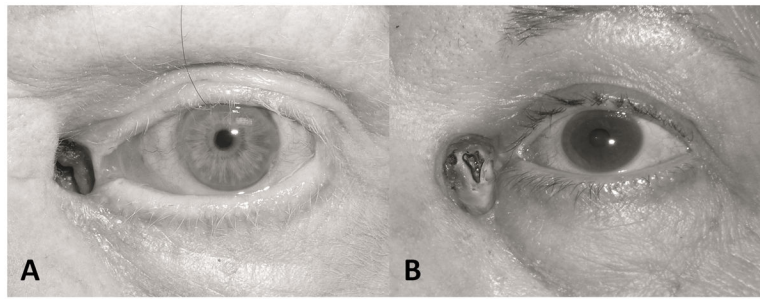


Figure 2.
(A) Patient #2; recurrence of skin necrosis following reconstruction. (B) Patient #3; recurrence of skin necrosis following the first reconstruction attempt.

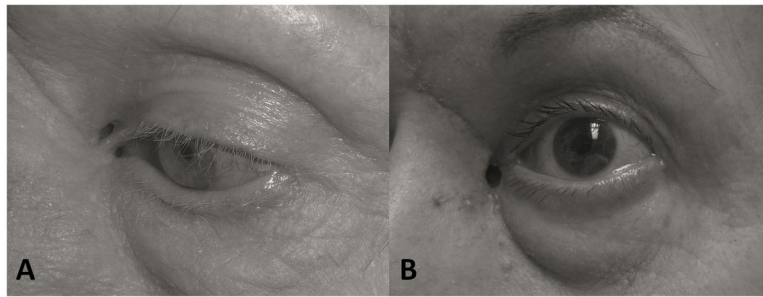


Figure 3.
Patient #2; recurrence of skin necrosis following the second reconstruction attempt.