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Upper eyelid reconstruction due to scar contracture following major burn injury: A case series

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ABSTRACT

Background: Upper eyelid contracture is a regular occurrence after severe facial burns. Numerous problems, such as corneal ulcers and exposure keratitis, may result from it. It is difficult for plastic surgeons to operate in this situation to preserve worst-case scenarios and enhance palpebral function.

Methods: In this piece, we showcase two instances: (1) Eight months after suffering a chemical burn injury, a 26-year-old male was diagnosed with bilateral superior palpebral contracture with iris prolapse. Both eyes' visual acuity was 6/6 - 1/ƴ. A 75-year-old female patient was identified as having a corneal ulcer and a contracture on her left upper eyelid. Both eyes' visual acuity was 20/6 - 1/ƴ. In order to restore the top eyelid in both patients, we undertook a contracture release and a full-thickness skin graft.

Results: Evaluation five days after surgery revealed no evidence of graft lysis and that the graft had taken nicely. On the patient graft from the second, we discovered shifting, although new epithelization will take place. Both of the patients had good eyelid looks following restoration.

Conclusion: Plastic surgeons should be aware of the seriousness of significant burn injuries to the face. Patients must receive appropriate instruction on the significance of avoiding consequences from eyelid contractures. In this instance, creating the top eyelid via a skin graft and contracture release technique made sense. We acknowledge that one of the limitations of our study is the brief observational period of the patient's conditions.

Keywords: chemical burn injury, corneal ulcer, upper eyelid contracture.

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INTRODUCTION

Both as a result of wounds from elective surgery and wounds from burns, cuts, tattoos, acne, abscesses, and injections, the number of people with post-wound scars keeps growing. Because scars usually result in haptic sensibility to pain and contractures, they often lower one's quality of life. Scars can be broadly classified into hypertrophic scars and cicatrix scars. It can be challenging to distinguish between the clinical possibilities of those two kinds; incorrect identification might result in inadequate therapy and, occasionally, in erroneous surgical decisions.¹

The risk factors for keloids are believed to be connected to numerous factors. A history of keloids can raise the incidence of keloids. Some genes associated with an increased risk of keloids are HLA-B14, HLA-B21, HLA-BW16, HLA-BW35,

HLA-DR5, and HLA-DQW3. All races together, except albinos, will have keloids; additionally, the risk for this race is up to fifteen times higher. Keloids are more common throughout puberty and during pregnancy and become less common as a person ages. The cause is thought to be hormones. It's believed that mast cells play a role in the development of keloids.¹

CASE REPORT

We typically give away two cases in this article: (1) Eight months after sustaining a chemical burn injury, a 26-year-old male was found to have both iris prolapse and two-sided unparallel palpebral contracture. Perceptual acuity in both eyes is 6/6 - 1/ƴ. A 75-year-old woman was admitted to the hospital due to a corneal ulcer and contracture on her left upper eyelid. There was 20/6 - 1/ƴ visual acuity in both eyes.

We often conduct a full-thickness cutis and a contracture unleash for the two patients to rebuild the upper lid.

DISCUSSION

There are three stages in the healing process. A physical problem initiates the inflammatory stage, during which platelets, neutrophils, and macrophages release inflammatory mediators and cytokines that contribute to the recruitment of proliferative cells, fibroblasts, endothelial cells, and epithelial cells.² This stage lasts for two to three days after an injury occurs. Hemostasis is first achieved by vasoconstriction. Platelet-derived growth factor (PDGF) and transforming growth factor β (TGF- β) are released by the platelets during this phase. When neutrophils enter the wound, they fill the cavity. Neutrophils engulf dead tissue and shield the body



Figure 1. Pre-Operation for Case 1.



Figure 2. Pre-operation for case 2.



Figure 3. Intra-operation. (A) Case 1, (B) Case 2.

against infection. Moreover, monocytes will penetrate the area of the wound. Macrophages consume germs and residue from other cells and help provide growth factors that fibroblasts require to make an extracellular matrix and form new blood vessels to mend wounds. Consequently, the lack of monocytes or macrophages will hamper wound healing. Finally, mast cells and lymphocytes will reach the site of the wound, although it is yet unclear exactly what role they will play.³

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released by the platelets during this phase. When neutrophils enter the wound, they fill the cavity. Neutrophils engulf dead tissue and shield the body against infection. Moreover, monocytes will penetrate the area of the wound. Macrophages consume germs and detritus from other cells and help provide growth factors that fibroblasts require to make extracellular matrix and form new blood vessels to mend wounds. Consequently, the period of wound healing will be hampered by the lack of monocytes or macrophages. Finally, mast cells and lymphocytes will reach the site of the wound, albeit it is yet unclear exactly what role they will play.³

The longest stage of the wound healing process is called the Remodeling Phase, lasting from the third week to a year. Collagen remodeling and wound contraction are the hallmarks of this phase. Type I collagen is beginning to take the role of type III collagen. The collagen's rearrangement increases the wound's strength over time.³

Hypertrophic scars, also known as keloids, are believed to be partially caused by a protracted inflammatory phase. Fibroblast activity rises when immune cells proliferate in keloids, continuing to create the extracellular matrix. This is also assumed to be why the scar on the keloid extends past the edge or boundary of the wound. Regression may occur in hypertrophic scars due to decreased immune cell infiltration.³

According to a different viewpoint, TGF- β is crucial in developing this fibrotic tissue condition. Essential collagen and proteoglycan synthesis stimulants, TGF- β 1 and TGF- β 2, influence the extracellular matrix, promoting collagen production while preventing degradation. TGF- β 3, on the other hand, served the opposite purpose and was discovered to be more prevalent during the latter stage of wound healing. A proteoglycan called decorin can attach to TGF- β , neutralize it, and decrease the amount of extracellular matrix proteins. Fibrotic diseases may result from low decorin levels.³

It has been proposed recently that fibrosis can potentially be caused by apoptosis. During the initial stage, hypercellular and hypertrophic scars developed. Later on, during the remodelling phase, fibroblasts reduced



Figure 4. Control Post Operation for Case 1.



Figure 5. Control Post Operation for Case 2.

and eventually transformed into normal scars using apoptosis. This process started on the twelfth day following the injury. Studying hypertrophic scars from severe burns revealed that the apoptotic process took longer than expected—between 19 and 30 months after the damage.³

Facial burns may be present in up to 30% of thermal trauma patients admitted to intensive care units. Although the involvement of the eyelids and surrounding tissues is joint, heat damage seldom results in ocular loss. Hence, mechanisms like the blink reflex with eye closure, the Bell phenomenon of the eyeball, and defensive movements of the head and arms to stop the cause of a burn are causes. Restoring the lid lamellae, restoring facial symmetry, and protecting the cornea are the objectives of eyelid restoration. Inadequate reconstructive methods can lead to blinding keratopathy and corneal exposure. Amblyopia and visual deprivation are rare conditions, yet they do happen. The periocular skin burns more deeply than the skin exposed elsewhere because it is thin and devoid of subcutaneous fat. When it comes to eyeball injuries, eyelid burns (50–85%), and contractures (30–65%) are more common than conjunctival burns (3–11%), corneal

abrasions (7–22%), corneal burns (5%), and so on.⁴

It is thought that by receiving early and continuous therapy, secondary hazards such as orbital compartment syndrome, radiation keratopathy, corneal ulceration, and secondary pneumonia might be avoided. Subsequent readings lead to divergent interpretations regarding the function of split and complete-thickness skin grafts in eyelid reconstruction, the timing of operations for eyelid contraction, eschar excision and debridement, temporary suturing, and surgical tarsorrhaphy.⁵

Because every situation is different, the technique for reconstructing the eyelid depends on the type of defect and the available skin. The lid surgeon needs to be knowledgeable about reconstructive principles and methods.⁶

CONCLUSION

Plastic surgeons should be aware of the seriousness of significant burn injuries to the face. Patients must receive appropriate instruction on the significance of avoiding consequences from eyelid contractures. In this instance, creating the top eyelid via a skin graft and contracture release

technique made sense. We acknowledge that one of the limitations of our study is the brief observational period of the patient's conditions.

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CONFLICTS OF INTEREST

None.

ETHICAL APPROVAL

None.

CONSENT

A fully informed written consent has been obtained and documented in paper for the patient who is the subject of this case report.

BIBLIOGRAPHY

1. Sinto L. Scar Hipertrofik dan Keloid: Patofisiologi dan Penatalaksanaan. *Cermin Dunia Kedokteran*. 2018;45(1):29-32.
2. Tan J, Wu J. Current progress in understanding the molecular pathogenesis of burn scar contracture. *Burns & Trauma*. 2017;5. doi:10.1186/s41038-017-0080-1
3. Parry I, Richard R, Aden JK, et al. Goniometric Measurement of Burn Scar Contracture: A Paradigm Shift Challenging the Standard. *Journal of Burn Care & Research*. 2019;40(4):377-385. doi:10.1093/jbcr/irz038
4. Oosterwijk AM, Mouton LJ, Schouten H, Disseldorp LM, van der Schans CP, Nieuwenhuis MK. Prevalence of scar contractures after burn: A systematic review. *Burns*. 2017;43(1):41-49. doi:10.1016/j.burns.2016.08.002
5. Borrelli M, Geerling G, Spaniol K, Witt J. Eye Socket Regeneration and Reconstruction. *Current Eye Research*. 2020;45(3):253-264. doi:10.1080/02713683.2020.1712423
6. Siemionow M, Zor F. Burn Reconstruction: Future Perspectives—Facial Transplantation. In: *Handbook of Burns*. Springer International Publishing; 2020:1988-1999. doi:https://doi.org/10.1007/978-3-030-34511-2_28



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