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Review Article

Paradigms in periorbital scar management

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ABSTRACT

Periocular scarring following surgery or trauma is of great aesthetic and functional concern and is difficult to predict. In today's era, with increasing scientific knowledge and technological advances, both physicians and their patients are highly concerned with minimizing scar appearance as a rising number of patients feel disappointed with their scars and are frequently seeking help for functional and aesthetic improvement. Although various non-surgical and surgical treatment strategies are available it is still difficult to improve excessive scarring. Thus, the importance of thorough knowledge of eyelid anatomy and healing mechanisms along with appreciation of wound closure techniques like placing the sutures at natural cosmetic subunit junctions and along relaxed skin tension lines (RSTLs) in order to achieve scar camouflage and to ensure decreased tension on the wound cannot be more emphasised. Periorbital area should be tackled by the oculoplasty surgeons in view of their distinct anatomy and close proximity to the eye.

Scars are commonly treated with a combination of non-surgical techniques, including watchful waiting, scar massage, pressure therapy, silicone gel sheeting, topical or intralesional injections, cryotherapy, laser therapy or radiotherapy. Surgical approaches include pincushioning debulking, direct scar excision, broken line closure techniques, scar lengthening procedures (Z plasty, V-Y/Y-V advancement) and scar excision with lid reconstruction. Mastery of this content is essential for consistent operative success. For good cosmetic and functional outcomes, scar revision techniques should be thoughtfully tailored to the individual and scar subtype.

1. Introduction

Scar formation is a highly regulated wound healing process which occurs following skin or tissue injury.[1] It involves migration and proliferation of fibroblasts, collagen production and deposition and angiogenesis. Any derangements of this orderly process leads to scars that are exuberant. Periorbital and eyelid scars usually result due to mechanical trauma, chemical injury, burn or eyelid surgery. It can manifest as eyelid retraction, ectropion or entropion depending on the state of the anterior and posterior lamella of both upper and lower eyelid.[2] Cicatricial wound healing of the eyelids can result in significant eyelid scarring with subsequent progressive retraction leading to cicatricial

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ectropion (Figure 1 a & b).

2. Etiopathogenesis of Scars

Wound healing is a complex process involving hemostasis, inflammation, and tissue remodelling.

1. Hemostasis - Immediately following wounding, platelet degranulation and activation of the complement and clotting cascades occur which leads to the formation of a fibrin clot for hemostasis.[3]
2. Inflammation - Platelet degranulation is responsible for the release and activation of an array of potent cytokines, such as epidermal growth factor (EGF), insulin like growth factor (IGF-I), platelet-derived growth factor (PDGF) and transforming growth factor β (TGF-β), which serve as chemotactic agents for the recruitment of neutrophils, macrophages, epithelial cells, mast cells, endothelial cells and fibroblasts which leads to pathological scar formation via enhancing inflammation at the site of injury.
3. Proliferation – Within 48 to 72 hours after the initial event, the healing process transitions into the proliferation phase, which may last for up to 3 to 6 weeks.[4] Recruited fibroblasts synthesize a scaffold of reparative tissue, the so-called extracellular matrix (ECM). This granulation tissue is made of procollagen, elastin, proteoglycans and hyaluronic acid and forms a structural repair framework to bridge the wound and allow vascular ingrowth. There is further migration and proliferation of different cell types such as keratinocytes, myofibroblasts and mast cells. Myofibroblasts, which contain actin filaments, help to initiate wound contraction.

iv. Remodeling - the immature scar can transition into the final maturation phase, which may last several months. The abundant ECM is then degraded and the immature type III collagen of the early wound is modified into mature type I collagen.[4]

The transformation of a wound clot into granulation tissue thus requires a delicate balance between ECM protein deposition and degradation, and when this process is disrupted, abnormalities in scarring appear, resulting in either keloid or hypertrophic scar formation. Various risk factors for hypertrophic scars are described in Table 1.

Classification of Scars

Mustoe et al. have described a classification system for scars which is simple to understand and clinically useful.[9]

1. An ideal, mature scar is thin and flat, has a good color match with the surrounding skin, is oriented along the relaxed skin tension lines, and does not produce any distortion of the adjacent tissues.[10]
2. Immature scar - erythematous, itchy, painful area that gradually matures.
3. Contracted scar – occurs when a large dermal area has been damaged leading to permanent shortening of the skin, disfigurement or functional limitation.
4. Widened scar – occurs when scar is present in areas which are under high tension and when the tension is perpendicular to the wound edges.
5. Atrophic scar - depressed and flattened below the dermal area.
6. Linear hypertrophic scars - raised, erythematous scars that are confined to the borders of the surgical incision. They grow rapidly for 3–6 months after which reach a static phase. Finally, they regress on their own.
7. Widespread hypertrophic scars – are mostly seen after burn injuries. They are red, raised, and lies within the borders of the injury.
8. Minor keloid - focally raised and extends over the normal tissue.
9. Major keloid - erythematous and itchy scars which are large in size and raised > 0.5 cm and continues to grow for years.

3. Hypertrophic Scars Versus Keloids

A hypertrophic scar is an overgrowth of scar tissue that remains within the boundaries of a wound. It usually occurs within 4-8 weeks following injury. They have a rapid growth phase for up to 6 months, and then gradually regresses. They are less associated with skin type and are common with burn scars or deep laceration. Histologically, they contain primarily type III collagen which are well organized and oriented parallel to epidermis with abundant nodules containing myofibroblasts, large extracellular collagen filaments and plentiful acidic mucopolysaccharides.

Keloids are densely collagenous with thickened hyalinized collagen and non-encapsulated. They appear as firm, mildly tender, bosselated tumors with a shiny surface and sometimes telangiectasia. Keloids may develop up to several years after minor injuries and may even occur spontaneously in the absence of any known injury. The scar extends beyond the boundaries of the original injury and have delayed onset of development. The incidence of keloid in dark-skinned people is estimated to be 3 to 20 times compared to light-skinned people. Histologically, they contain type I and III collagens which are thickened, organized randomly in the dermis and containing pale-staining hypocellular collagen bundles with no nodules or excess myofibroblasts.

Hypertrophic scars have lower recurrence rates after simple excision as compared to keloids. Fortunately, periorbital scars are mostly hypertrophic.[11]

Scars may manifest as pruritus, tenderness, pain, and dysesthesias, all of which may result in sleep disturbances and disruption of daily activities. These symptoms are likely exacerbated by a variety of factors, including local friction, inflammation, stimulation of nerve endings in

Table 1: Risk factors for hypertrophic scar (HTS) formation [5–8]

1. Age	11-30 years age more predisposed
2. Race	Dark skinned population more prone (African > Caucasians), more pro-inflammatory cytokine CCL2 production
3. Mechanism of injury	Surgical wounds, clean wounds less associated with HTS than Burn wounds, tissue loss
4. Wound location	Skin subject to stretch is more prone
5. Wound infection	Bacterial colonisation a/w prolonged local inflammatory response, enhancing HTS formation
6. Suture material	Absorbable > non-absorbable (nylon suture, polypropylene suture preferred) Braided (vicryl) > Monofilament (monocryl)
7. Suturing technique	Wound approximation with minimum tension (Eg. use of zigzag suturing technique); Intradermal suturing with prolene are protective factors
8. Duration of suture removal	Delayed suture removal > 7 days predisposes to HTS formation
9. Coexisting systemic allergy	More mast cell degranulation supports scar formation
10. Smoking	Protective factor, decreases HTS formation by decreasing inflammatory phase of scar formation and decreasing collagen deposition
11. Genetics	Less role, more role with Keloid formation if family history present

and around the scars and increased local levels of β-endorphin. Certain scars may contribute to local hyper or hypohidrosis, exacerbating skin irritation and maceration in the setting of scar fragility. Cumulatively, these factors may interfere significantly with physical/ occupational rehabilitation efforts.

4. Evaluation

The lid should be evaluated for anterior lamellar shortening, posterior lamellar shortening and/or canthal tendon laxity. Proper evaluation and identification of the anatomic abnormality is necessary for good functional and aesthetical outcomes.

1. Scar appearance – scar size (measured by area, length, volume, height or width), scar colour, location and extent of scar, maturity of scar, whether linear or diffuse, raised or depressed, pigmentation of scar and any associated eyelid deformity due to scar.
2. Two - finger test - 2 fingers are placed on the skin below the eyelid margin and the skin is elevated superiorly (Figure 2). This provides extra anterior lamellar skin to the periorbital area. If after elevation of the skin, the retraction is resolved, then the cause of retraction is likely deficiency of the anterior lamella. If the retraction persists, the cause is likely in the posterior or middle lamella.
3. Lower lid distraction test - It involves grasping the lower eyelid and pulling it anteriorly away from the globe and a distraction of more than 6 mm is considered abnormal (Figure 3). It represents lid laxity and often canthal tendon weakness.
4. Snap back test - It also involves pulling the lid off the globe and assesses the speed with which the lid returns to its normal anatomic position. The lid should return to its position on the globe without blinking. If the patient needs to blink in order to bring the eyelid back into apposition with the globe, the test is abnormal.
5. Lateral traction test - A lateral traction is placed on the lid using a finger. If lateral tension on the lid corrects an observed entropion or ectropion the eyelid malposition may be caused by eyelid laxity and possibly disinsertion of the eyelid retractors.

5. Management

A meticulous preoperative evaluation is needed to decide the management plan and have good functional and aesthetical outcomes. As scar formation is an evolution, the timing of all surgical and nonsurgical interventions is critical. Goals of therapy for any scar should be established in conjunction with the individual patient and at a minimum, should focus on relieving symptoms, reducing comorbidities, decreasing scar volume, and maximizing functional and cosmetic outcomes. [12,13] Various treatment modalities are summarized in Table 2.

5.1. Scar prevention

1. Placing sutures at natural cosmetic subunit junctions is advantageous as these junctions improve scar camouflage.
2. Sutures should be placed in such a way that anatomical alignment of eyelid and periorbital structures is achieved during the primary repair.
3. Placing sutures along relaxed skin tension lines (RSTLs) as the scar may camouflage within the rhytids that form along RSTLs and also there will be decreased tension on the wound, which minimizes resulting scar formation (Figure 4). [14]
4. Achieving rapid epithelialization of wound as delayed epithelialization beyond 10–14 days is known to increase the incidence of hypertrophic scarring.

5.2. Non-Surgical Management

5.2.1. Massage therapy

Scar massage hastens wound healing, decreases itching, redness, and pain associated with an immature scar, and shortens the time required for scar maturation. Massage causes mechanical disruption of fibroblast fibers, increasing the pliability of the wound, leading to a softer scar. Massage is carried out using the pad of the thumb or fingers directly on the scar and rubbing in a circular matter. Massage can also be carried out with an emollient that keeps the epidermal layer hydrated, thereby reducing the vascularity and erythema. The pressure should be enough to move the scar and skin. Scar massage should be started as soon as possible and should be continued 2–3 times a day till 6–8 weeks after the trauma or surgery.[15]

In a review by Ault P et al.[16] which included published studies with a total 258 human participant with hypertrophic burn scars, showed that scar massage is effective in decreasing scar height, vascularity, pliability, pain, pruritus and depression in hypertrophic burns scarring. Ko et al.[17] have reported statistically significant reduction in scar thickness following massage.

5.2.2. Pressure therapy

Pressure therapy can be used for burn scars. In this pressure is applied over the burn area to cause compression of the scar which is believed to aggravate the hypoxic condition of the scar tissue. Thus, leading to degeneration of fibroblasts and collagen as well as a decrease in the collagen synthesis.[18] However, pressure therapy is not a practical approach for periocular scars. For periocular scars, silicone gel sheet is preferred as it provides occlusion and pressure along with hydration.

5.2.3. Silicone gel sheeting

This is a non-invasive approach for the prevention and treatment of keloid and hypertrophic scars. Silicone gel sheet is a soft, self-adhesive and semi-occlusive sheet which is thought to work by increasing the temperature, hydration, and perhaps reducing the oxygen tension of the occluded lesion and increasing collagenolysis and fibroblast degeneration causing it to soften and flatten. The silicone gel sheet also provides a hydrating environment for the epidermal layer and decreases the capillary activity, vascularity, and the metabolism of the scar tissue, thereby causing reduced collagen deposition.[19] Silicone gel sheeting is designed to be used on intact skin and should not be used on open wounds. To be effective, the silicon sheets must be worn over the scar for 12-24 hours/day for 2-3 months.[20] The use of silicone gel sheets is limited by daily patient's compliance. Silicon sheets may also be an adjuvant to surgical excision, intralesional corticosteroid and laser therapy.

5.2.4. Topical agents

5.2.4.1. Vitamin A. Topical and intralesional vitamin A and its retinoid derivative aid in wound healing and reduce pathologic scar tissue by affecting cell growth and differentiation, by modulating the extracellular matrix synthesis by fibroblasts.[21] Prutkin L et al. showed that Retinoic acid solution (0.05%) applied daily on scars demonstrated a reduction in size and decrease in itching.[22] Another clinical trial by Janssen de Limpens AM with 28 intractable scar cases treated with topical 0.05% Tretinoin showed a significant decrease in scar size in 79% cases.[23] Adverse effect included "retinoid dermatitis" (temporary cutaneous irritation characterized by erythema, peeling of skin, dryness, and itching), post inflammatory hyperpigmentation, telangiectasia photosensitivity and skin atrophy.[24,25]

5.2.4.2. Vitamin E. Vitamin E is a lipid-soluble antioxidant vitamin that has favorable effect on wound healing and scars. In a study by Palmieri B et al.[26] on 80 patients with hypertrophic scars and keloids, showed that incorporation of Vitamin E in silicone gel sheets led to a superior outcome (95%) than in the group using plain silicone gel sheets (75%) at the end of 2 months. Adverse effects include contact dermatitis, urticaria, eczematous dermatitis and reactions similar to erythema multiforme.[27,28]

5.2.4.3. Onion extract. Onion extract appears to improve the arrangement of collagen by its anti-inflammatory, collagen-inhibiting and bacteriostatic properties. Chanprapaph K et al.[29] in a study on 20 asian women with Pfannenstiel's cesarean section scars compared topical agents with Onion extract to plain petroleum emollients and showed that Onion extract leads to better scar management, whereas in another similarly designed study by Chung VQ et al.[30] on 24 patients with surgical wounds of atleast 4cm length, the investigators found no statistically significant difference between the two groups. However, no adverse effects have been reported, safety in periocular area use is questionable.

5.2.4.4. Imiquimod 5%. Imiquimod 5% cream is a topical immune response modifier which acts by stimulating interferon which increases collagen breakdown and also alters the expression of apoptosis-associated genes. Studies have reported contradicting results with some reporting positive effects on the recurrence rate of keloids[31–33] and others reporting no significant differences in recurrence rates.[34] Side effects include persistent inflammation, skin erosion and depigmentation.

5.2.5. Intralesional corticosteroids

Intralesional corticosteroids remain a mainstay in the treatment of hypertrophic scars and keloids with a response rate of 50% to 100% and a recurrence rate

of 9% to 50%. The efficacy of corticosteroids is likely secondary to their ability to suppress inflammation, inhibit nitric oxide synthase transcription with subsequent inhibition of collagen synthesis in fibroblast,[35] inhibition of scar fibroblast growth, fibroblast degeneration and down regulation of collagen gene expression in scars,[36] promote collagen degeneration and limit wound oxygenation and nutrition (Figure 5a and b).

Various steroid preparations that can be used for intralesional injection include hydrocortisone acetate, methylprednisolone, and dexamethasone. Triamcinolone acetonide (TAC) 10-40 mg/ml per course every 2 week is most commonly used.[37] Depending on the type of lesion two or three doses of TAC injection may suffice but some may require injection for 6 months or more with 2-4 weeks interval between injections.[38] Complications include atrophy, telangiectasia formation, delayed wound healing, widening of the scar and pigmentary alteration.[39]

5.2.6. Anti-metabolites

Antimetabolites interfere with the proliferative mechanisms of scar formation, mainly fibroblast proliferation and collagen production thus proving beneficial in scar prevention and reduction.

5.2.6.1. 5-Fluorouracil. 5-FU is a pyrimidine analogue with antimetabolite activity that is used in cancer chemotherapy.[40] Intralesional 5-FU minimizes scar formation by inhibiting cell proliferation through the disruption of DNA synthesis, inhibit collagen production and decrease fibrogenic marker, TGF α and TGF-b.[41] 5-FU produce better result in combination with steroid (Figure 6a and b), silicone gel sheeting or laser than monotherapy.[42,43] Khalid et al.[44] in 2016 in which he included 120 patients of keloid and hypertrophic scars divided in two groups. The group A patients received intralesional triamcinolone acetonide and group B patient received both 5-FU and TAC. 8 injections at weekly interval were given and patients were evaluated at the start of treatment and then at 4th and 8th week during treatment and then 4 weeks after end of treatment. Patients were assessed for mean reduction in scar height, efficacy and complications. Total 108 patients completed the study. The mean reduction in scar height in group B was markedly better than that of group A.Hatamipour et al. evaluated the outcomes of treatment of 50 patients with keloids at various sites with surgical excision followed by silicone gel sheeting in one group versus silicone gel sheeting and 5-FU injections in the second group. The second group achieved a 75% success rate as compared to 43% in the first, with an extremely low rate of recurrence (1%).[45] *Adv*erse side effect of intralesional 5-FU include pain, ulceration and burning sensation.[46]

5.2.6.2. Bleomycin. Bleomycin directly inhibits collagen synthesis through decreased stimulation of TGF β 1, inhibition of lysyl oxidase, an enzyme that helps in collagen synthesis and increase in fibroblast apoptosis.[47] Bleomycin has antitumor, antiviral and antibacterial activity and is a secondary metabolite of strain of streptomyces obtained from soil.[48] Intralesional injection of bleomycin results in significant improvement in scar height and pliability as well as reduction in erythema, pruritus and pain.[49,50] Dermal atrophy and hyperpigmentation may occasionally occur as side effects.[51]

5.2.7. Laser therapy

Laser therapy for scar has yielded varying success.[52] Laser such as carbon dioxide (CO2) and erbium: yttrium aluminium garnet YAG are ablative nonselective laser that target water molecule while those like 585 and 595 nm pulsed dye and neodymium (Nd):YAG lasers are non-ablative and chromophore usually oxyhemoglobin selective.[53] The 585-nm pulsed dye laser currently gives most encouraging results, as proven in a study by MP Goldman on 48 patients with hypertrophic scars treated with flashlamp-pumped pulsed dye laser, wherein 73% scars on the face for < 1 yr resolved in an average of 2.3 treatments.[54] The laser sessions are usually spread out over 2–6 sessions at weekly interval which may be sufficient for keloids or young hypertrophic scars.[55]

5.2.8. Interferon

Interferon (IFN) are cytokines with antiproliferative, antifibrotic and antiviral effects.[55] They decrease synthesis of collagen type 1 and 3. The antifibrotic effect is thought to be mediated through the antagonizing effect on transforming growth factor beta and histamine.[56] In a study by S Nanda on 28 patients with keloids were given Interferon α 2b injected intralesionally, 1.5 million IU (50 mg/ml, 0.5 to 2ml per session) once a week for a maximum of 12 weeks, reduces scar size by 50% in 70% patients and has proven superior to intralesional corticosteroid.[57]

IFN therapy is costly treatment and has adverse effect which include painful injection requiring local anesthesia and dose dependent fever, chills, night sweats, fatigue, myalgia, headache, and flu like symptoms for 48-72 hours post injection.[58]

5.2.9. Cryotherapy

Cryotherapy is widely used for treatment of keloid and hypertrophic scars.[59] The mechanism of action is rapid repeated cooling and rewarming of tissue leading to cell death and tissue sloughing.[60] It has been used as monotherapy and in conjunction with other forms of treatment for bigger scars.[61] Cryotherapy has been reported to have efficacy of 50-85% on scar with moderate flattening and symptomatic relief.[62] Acute adverse effect of cryotherapy includes pain , necrosis, edema, and infection while the chronic effects include atrophy,

hyperpigmentation and hypopigmentation. [63,64]

5.2.10. Radiotherapy

Radiotherapy (RT) in the form of superficial X-rays, electron beam therapy and brachytherapy has been used successfully as an adjuvant therapy following surgical excision of keloids. [65] The mechanism of action is inhibition of neovascularization and proliferating fibroblasts, resulting in decreased collagen production. [66] Adverse effects includes pigmentary changes, telangiectasia, and skin atrophy, radiation retinopathy, cataract, glaucoma, dry eye, and destruction of lacrimal drainage system.

5.2.11. Botulinum toxin A (BTA)

BTA immobilizes local muscles thus reducing skin tension caused by muscle pull, and thus, decreases microtrauma and subsequent inflammation leading to aesthetic improvement of post-surgical scars. Gassner HG et al showed in a study on 31 patients with traumatic forehead lacerations, that botulinum toxin injections (15 U of BTA (Botox, Allergan, Irvine, CA, USA) per 2 cm intraoperative length) into the musculature adjacent to the wound within 24 hours after wound closure resulted in enhanced wound healing and less noticeable scars compared with placebo. [67]

5.2.12. Photodynamic therapy

Topical PDT generates reactive oxygen species, which in turn leads to cell apoptosis, membrane and mitochondrial damage and reduces type I collagen synthesis and fibroblast proliferation. In a study Ud-Din et al on 20 patients with keloids at various locations, showed that three treatments of PDT (37 J/cm2) at weekly intervals were effective in reducing pruritus and pain and in increasing pliability of symptomatic keloids. Also, when applied postoperatively after excision of keloids, no recurrence rates were seen at 9-month follow-up, with the exception of one patient. [68]

5.3. Surgical management

Various surgical interventions exist for scar revision; proper technique for improvement of cosmetically and functionally limiting scars of the periocular skin. Cosmetic surgical scar revision should be delayed for at least 6–12 months from the initial injury to allow for appropriate scar maturation.

5.3.1. Debulking of pincushioned flap

Pincushioning, also known as the trapdoor phenomenon, frequently arises as a complication in cutaneous flaps. Transposing and sliding flaps from the glabellar skin to the eyelid pose a notably elevated risk for this issue. This occurrence is likely attributable to intrinsic differences in skin thickness, challenges with appropriate flap sizing secondary to complex surface topography, limited capacity for primary undermining on the eyelid, and tendency for retention of edema. [69,70]

Original suture lines are sharply re-incised and the pincushioned flap is carefully lifted. The excess scar and fibrofatty tissue causing pincushioned flap is sharply debulked until the contour deformity is resolved. [71]

5.3.2. Direct scar excision

Scar is excised in a fusiform or elliptical manner and closed linearly after meticulous undermining. Undermining will help in decreasing the tension along suture line.

5.3.3. Broken line closure

Broken line closure technique also known as scar disruption, includes the W-plasty and the geometric broken line closure technique. In this technique, the scar is “irregularized” by breaking it up into short, and subsequently less perceptible segments. [71] The benefit of irregularising the scar is that long, straight scars reflect light homogenously and are therefore easily visible. An irregular line reflects insignificant light and is less visible leading to scar camouflage. [72]

W-plasty provides a regularly irregular scar, and geometric broken line closure provides an irregularly irregular scar.

5.3.4. Preventing medial or lateral canthal webbing

Eyelid reconstruction may result in medial or lateral webbing if the reconstruction creates vertical tension following wound repair. Excess anterior lamellar shortening may also cause webbing. A lateral canthal web occurs when the lateral aspect of the upper blepharoplasty incision is taken below the equator of the lateral canthus. It can also occur if excessive eyelid skin is removed laterally. During healing, scar contracture pulls the lax lower lateral eyelid skin superior and upper eyelid skin inferior, thus causing skin bridging across the lateral canthus. The key to avoiding webbing is to replace vertical tension with horizontal tension. [73]

5.3.5. Scar lengthening procedures

5.3.5.1. Plasty. In Z plasty, two, opposing, identically sized triangular skin flaps are created which when transposed, reorient the scar 90° while functionally lengthening the scar along the central limb of the Z-plasty. [74] This angle determines the degree of lengthening of the tissue, the larger the angle, the greater the length gain. An angle of 60° in Z-plasty gives a gain of 75% in tissue length and changes the scar direction by 90°. An angle of 30° lengthens the scar by 25%, an angle of 45° by 50%, angle of 75° by 100% and an angle of 90° by 125%. [75] An angle < 60° though easier to transpose, results in less scar lengthening and realignment of < 90°. Whereas, angle > 60° is avoided as it increases the force required for transposing the flaps, making the closure difficult. The scar lengthening component of the Z-plasty is useful for lid repositioning and resolution of medial

canthal webbing, while the reorientation associated with the procedure is helpful for placing the resultant irregularized scar into RSTLs and/or anatomic subunit junctions thus causing scar camouflage.

5.3.5.2. V-Y and Y-V advancement. These techniques are indicated in scar lengthening in case of a small contracted scar, improving 'trapdoor' deformity, and elevation or depression of a free margin such as a scar causing ectropion.

The V-Y flap is helpful in "pushing" contracted free margins, while the Y-V flap can be utilized for "pulling" surrounding tissue. For V-Y advancement flap, a V-shaped incision is made through epidermis and dermis immediately below the distorted free margin or contracture.[71] After proper undermining, the trailing aspect of the V-shaped incision should be closed primarily with buried vertical mattress sutures, subsequently pushing the flap towards the desired free margin and leading to lengthening effect (Figure 7a).

The Y-V flap is useful for pulling a free margin into appropriate place after distortion by a pushing vector. A Y-shaped incision is made adjacent to the distorted free margin with the scalpel blade. After undermining, the V-shaped tissue is then advanced away from the free margin until appropriate position is achieved and then carefully sutured (Figure 7b).[71]

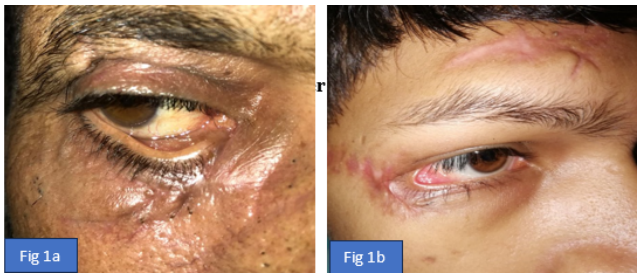

Figure 1: Shows right lower lid cicatricial ectropion.

Figure 2: Two - finger test shows that retraction is resolvedon elevation of lower lid suggesting anterior lamellar shortening

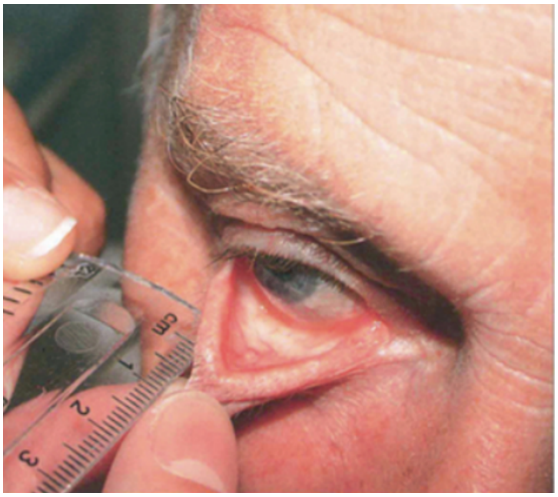

Figure 3: Lower lid distraction test

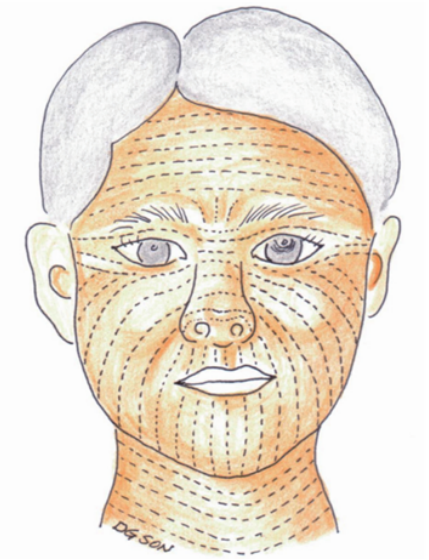

Figure 4: RSTL

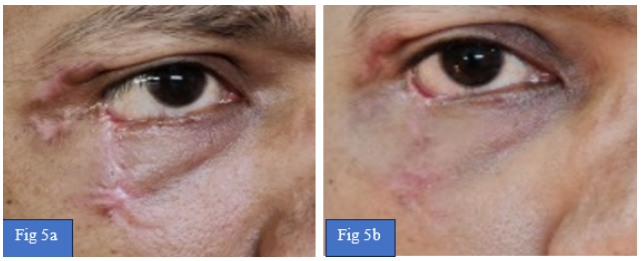

Figure 5: a and **b:** Shows scar at presentation and improved scar at 3 months follow up following four intralesional Triamcinolone acetonide (40mg/ml) injection at 2 weeks interval.

Table 2: Various nonsurgical and surgical interventions for periocular scar

Modality	Indications and use	Outcomes	Safety concerns
1. Massage therapy	Started as soon as possible Prevents scar	Effectively decreases scar height, vascularity, pliability, pain, pruritus	Safe Compliance issue
2. Silicone Gel sheeting	12-24 hrs/day for 2-3 months, on intact skin	Scar softening, flattening	Safe Compliance issue
3. Topical agents			
a) Vitamin A	0.05% preparations More used for keloid scars	Significant decrease in scar size	Retinoid dermatitis Post inflammatory hyperpigmentation Telangiectasia, photosensitivity, skin atrophy
b) Vitamin E	Incorporated in silicone gel sheets	~95% outcome shown a study in 2 months	Contact dermatitis, urticaria, eczematous dermatitis
c) Imiquimod 5%	5% cream Preventive > treatment	Studies have shown contradictory to no results as well	Skin erosion Depigmentation
4. Intralesional agents			
a) Corticosteroids	Mainstay treatment of HTS Triamcinolone acetonide (10-40 mg/ml) most commonly used, 2-3 doses, 2 weekly	Response rate of ~50-100% Recurrence rate of ~9-50%	Atrophy Telangiectasia Delayed wound healing Widening of scar Pigmentary changes
b) 5 FU	Combination therapy with steroid (9:1), silicone gel sheet, laser	Increases response rate by ~30%, decreases recurrence rate to as low as ~1%	Painful injection Ulceration Burning sensation
c) Bleomycin	1-2 IU given peri-ocular, 3-4 weekly	Added benefit in scar height, pliability, reduces erythema, pruritus, pain	Dermal atrophy and hyperpigmentation
d) Interferon α 2b	1.5 million IU intralesionally twice a day for 4 days	~50% reduction in scar size over 9 days Proven superior to steroid	Painful injection Flu like symptoms 48-72 hours post injection
e) Botulinum toxin A	15 U BTA per 2 cm length into scar adjacent musculature	Enhanced wound healing and less noticeable scar	Paresthesias Headache Ptosis if given in LPS muscle
5. Laser therapry	For pigmented scars 585-nm pulsed dye laser, 2-6 sessions, weekly	Per dose improvement in scar erythema and flattening ~57% Preferred in combination treatment	Post treatment purpura most common Pigmentary changes
6. Cryotherapy	Ideal treatment for Keloid scars with narrow base	~51-67% reduction in scar volume after single treatment, occasionally 100% reduction after second treatment	Pain, necrosis, edema, infection, atrophy, hypopigmentation
7. Radiotherapy	Adjuvant following surgical treatment of keloids usually	Prevents recurrence	Pigmentary changes, telangiectasia, skin atrophy, radiation retinopathy, cataract, glaucoma, dry eye
8. Photodynamic therapy	37 J/cm2, 3 doses, weekly	Decreased pruritus, pain, increased pliability of keloids, extremely low recurrence rates	Pain, erythema, edema, contact dermatitis, immunosuppression
9. Surgical management			
a) Debulking procedures	By debulking, direct scar excision, For smaller scars	Decreased size of scar, release of pincushioning effect around scars	Suture line tension
b) Scar disruption	W plasty and geometric broken line closure technique	Scar camouflage	Suture line tension
c) Scar lengthening procedures and lid reconstructions	Z-plasty, V-Y and Y-V advancement, lid reconstruction by free/advancement flaps	Improved cosmesis	Secondary surgical site creation

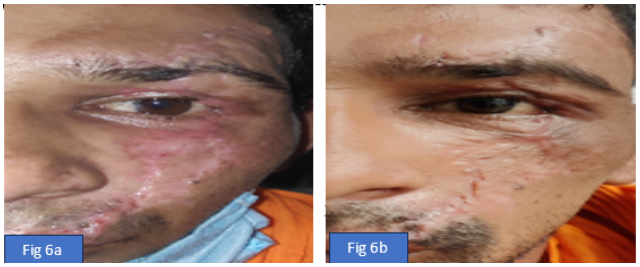

Figure 6: a and **b**: Shows scar at presentation and improved scar at 3 months follow up following four intralesional Triamcinolone acetonide (40mg/ml) + 5-Fluorouracil (50mg/ml) injections in ratio of 1:9 at 2 weeks interval.

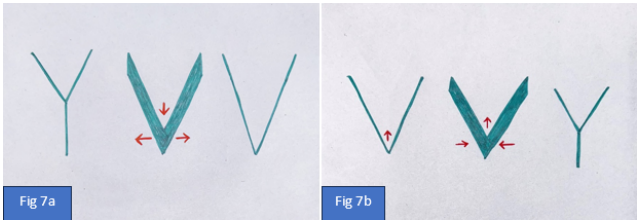

Figure 7: a and **b:** Shows technique of Y-V advancement and V-Y advancement.

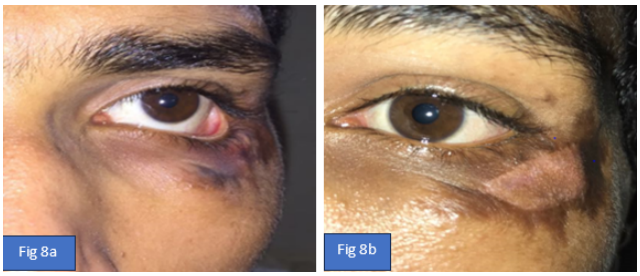

Figure 8: a: Shows scar at presentation and **b:** Shows improvement in lower lid contour at 4 weekspostoperative full thickness skin graft.

5.3.6. Scar excision and lid reconstruction

1. Anterior Lamellar Defects
   Eyelid defects involving the anterior lamella can be repaired by:
   (a) Direct closure with proper undermining - Small (< 25% of the eyelid) and superficial anterior lamellar defects are often closed directly with proper undermining.[76] Undermining helps to minimize tension on the suture line. This can be combined with anchoring sutures to support the deep tissues which will further take additional tension off the final closure.
   (b) Free skin graft – can be full or split thickness (Figure 8a and b).
      i. All skin grafts undergo primary and secondary contractures. Primary contracture refers to the immediate reduction in the size of a skin graft after harvesting, triggered by the passive recoil of elastin fibers within the dermis.
      ii. Given that full-thickness skin grafts (FTSGs) contain a larger portion of dermal tissue, primary contracture is more pronounced in FTSGs compared to split-thickness skin grafts (STSGs). On the other hand, secondary contracture, which occurs over time within the wound bed, is driven by myofibroblasts. This secondary contraction is more prominent in STSGs due to the absence of additional dermal layers to resist the pull of myofibroblasts, a feature present in FTSGs.
      iii. Consequently, in clinical practice, it's advisable to avoid placing STSGs in aesthetically sensitive regions like the eyelids as contracture-related deformities could arise.[77]
      iv. Full-thickness skin grafts often offer a suitable color match, contrasting with split-thickness skin grafts which may exhibit hypo or hyperpigmentation. Furthermore, the meshing process for split-thickness grafts notably impacts their aesthetic appearance. Split-thickness grafts are typically preferred for larger coverage areas. For eyelid FTSGs are preferred.
   (c) Donor sites include ipsilateral or contralateral eyelid skin, retroauricular skin, preauricular skin, supraclavicular area, upper inner arm.
   (d) Myocutaneous advancement flaps -
      i. Local random flaps
         A. V–Y advancement flap
         B. Rhomboid transitional flap
         C. Mustarde cheek rotational flap
         D. Tripier flap
         E. Fricke flap
      ii. Axial flaps
         A. Frontal Flap Based on the Frontal Branch of the Superficial Temporal Artery
         B. Forehead Flap Based on the Supratrochlear Artery (STA)
         C. Nasolabial Flap Based on the Angular Artery
2. Posterior Lamellar Defects
   Eyelid defects involving the posterior lamella can be repaired by:
   (a) Direct closure with or without lateral cantholysis
   (b) Free autogenous tarsal grafts –
      i. Tarsoconjunctival grafts from healthy eyelid

ii. Tarsomarginal grafts from healthy eyelid
iii. Oral mucosa graft (Lip mucoosa, Hard Palatal Mucoperiosteal (HPM) Graft, buccal graft)
iv. Auricular Cartilage Graft
v. Periosteum
vi. Fascia lata
vii. Sclera
viii. Nasal Chondromucosal Graft

(c) Tissue flaps
i. Local tarsoconjunctiva flap
ii. Hughes flap
iii. Reversed Hughes flap

3. Full Thickness Eyelid Defects
(a) Cutler beard flap
(b) Mustarde check rotation flap

Tenzel semicircular flap

6. Conclusion

Periocular scarring can have serious cosmetic and functional effects. Proper knowledge of eyelid anatomy and meticulous pre-operative evaluation is of utmost importance for good cosmetic outcomes. Although most scars are best treated with minimally invasive techniques or non-surgical methods prior to consideration of surgical revision. It is wise to wait for 6–12 months from initial injury for scar maturation if the scar is not causing any significant functional limitation. Early surgical intervention should be considered in case scar is leading to significant functional limitation. The eyelid reconstruction should be carried out with respect to the anterior and posterior lamellae, free margins and simultaneously respecting natural cosmetic subunit junctions and relaxed skin tension lines (RSTLs) to improve scar camouflage and to ensure a favourable outcome. Though there are several choices for the reconstruction and scar treatment but the selectin of procedure should be judicious to avoid unnecessary additional scar lines or cosmetic deformity in the face. Furthermore, the safety of the eye is of paramount importance and the procedure should be prioritised accordingly. The periorbital and eyelid procedures need to be done by an oculoplasty surgeon in view of their distinct anatomy and close proximity to the eye.

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8. Conflict of Interest

None.

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