

Surgical and Non-Surgical Management of Hypertrophic Scars and Keloids in Plastic Surgery: A Contemporary Narrative Review

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ABSTRACT

Hypertrophic scars and keloids are fibroproliferative disorders of wound healing that remain among the most challenging conditions in plastic and reconstructive surgery. Although both entities share excessive extracellular matrix deposition, fibroblast activation, inflammation, and cosmetic burden, they differ in clinical behavior, recurrence risk, and therapeutic response. Hypertrophic scars usually remain confined to the original wound boundaries and may partially regress over time, whereas keloids extend beyond the initial injury and frequently recur after monotherapy. Contemporary management has shifted from isolated interventions toward multimodal, individualized treatment plans that integrate prevention, scar maturation control, tension reduction, silicone-based therapy, intralesional pharmacologic agents, laser-based modalities, microneedling, radiotherapy, and carefully selected surgical excision. This narrative review synthesizes recent evidence on surgical and non-surgical strategies for hypertrophic scars and keloids, emphasizing practical decision-making in plastic surgery. Particular attention is given to patient selection, anatomical location, scar phenotype, skin phototype, pediatric considerations, recurrence prevention, and combination therapy. The available evidence supports a stepwise approach in which early diagnosis, risk stratification, mechanical offloading, and conservative therapy are prioritized, while invasive modalities are reserved for symptomatic, functionally limiting, refractory, or cosmetically severe lesions. Despite advances in laser-assisted drug delivery, botulinum toxin, 5-fluorouracil, verapamil, bleomycin, and adjuvant radiotherapy, no single therapy is universally effective. A tailored, longitudinal, multimodal strategy remains the most rational framework for improving symptoms, function, appearance, and patient satisfaction.

KEYWORDS: hypertrophic scar, keloid, scar management, intralesional therapy, wound healing.

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INTRODUCTION

Hypertrophic scars and keloids are abnormal fibroproliferative responses that develop after cutaneous injury, surgery, burns, inflammation, acne, piercings, or trauma. In plastic surgery, their relevance extends beyond aesthetics because symptomatic scars may cause pain, pruritus, stiffness, contracture, restricted mobility, and major psychosocial distress. Contemporary algorithms emphasize that successful scar management begins before the scar is mature: risk assessment, atraumatic technique, tension minimization, infection prevention, and early postoperative surveillance are central to reducing pathologic scar formation [1].

Although hypertrophic scars and keloids are often grouped together, the distinction is clinically important. Hypertrophic

scars generally remain within the original wound margins, arise within weeks after injury, and may flatten with time. Keloids, in contrast, grow beyond the wound boundary, show persistent activity, and have a greater tendency to recur after excision alone. This difference explains why treatment selection should be based not only on scar thickness or erythema but also on growth pattern, location, symptoms, previous recurrence, patient age, skin phototype, and prior response to therapy [2].

Current evidence does not support a universal first-line treatment applicable to all scars. Instead, management should be individualized and frequently combined. Silicone therapy, pressure therapy, corticosteroids, 5-fluorouracil, bleomycin, verapamil, botulinum toxin, laser therapy, microneedling, radiotherapy, and surgical revision each have a role, but their

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efficacy depends on lesion phenotype and timing. This is particularly relevant in plastic surgery, where cosmetic outcome must be balanced against recurrence risk, adverse effects, pigmentation changes, wound complications, and patient expectations [3].

The modern therapeutic paradigm favors structured, multimodal care rather than isolated monotherapy. Recent systematic reviews and plastic surgery-focused discussions highlight that recurrence is most likely when keloids are excised without adjuvant therapy, while outcomes improve when surgery is combined with postoperative radiotherapy, intralesional therapy, pressure, silicone, or tension-reduction strategies. Therefore, the objective of this review is to provide a clinically practical synthesis of surgical and non-surgical management strategies for hypertrophic scars and keloids in plastic surgery, integrating recent evidence and figure-based concepts suitable for manuscript development [4,5].

REVIEW

Search strategy and article selection

A narrative literature search was performed using PubMed, PMC, and Google Scholar to identify recent articles published between 2022 and 2026 on hypertrophic scars, keloids, surgical scar revision, silicone therapy, laser therapy,

intralesional pharmacologic therapy, radiotherapy, microneedling, and emerging scar-modulating strategies. Priority was given to systematic reviews, meta-analyses, randomized controlled trials, clinical studies, and recent case reports with relevant clinical images. Articles were selected based on relevance to plastic surgery decision-making, therapeutic applicability, and availability of full-text clinical or mechanistic information.

Pathophysiological basis of hypertrophic scars and keloids

The development of hypertrophic scars begins with an exaggerated but anatomically confined wound-healing response. After cutaneous injury, normal repair progresses through hemostasis, inflammation, proliferation, and remodeling; however, persistent inflammation, excessive fibrin deposition, delayed re-epithelialization, abnormal angiogenesis, and prolonged fibroblast activation may shift this process toward hypertrophic scarring. This biological framework is particularly relevant in plastic surgery because many preventive strategies, including atraumatic technique, infection control, tension reduction, silicone therapy, and early scar surveillance, are directed at interrupting this transition before scar maturation becomes fixed [6].

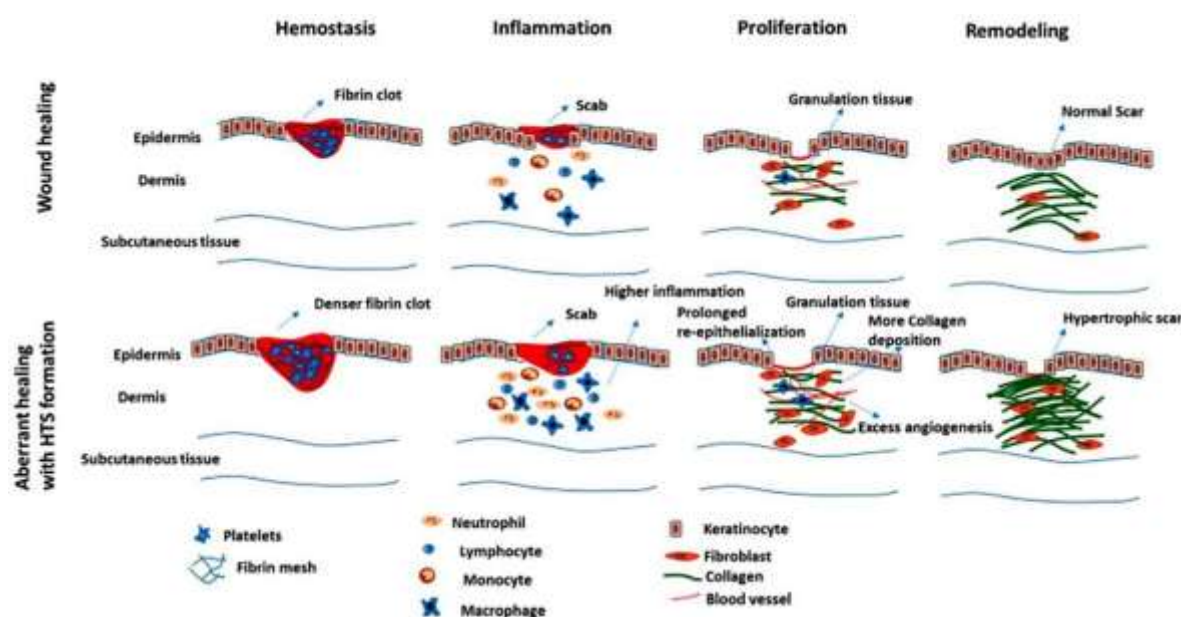


Figure 1. Phases of normal wound healing versus aberrant wound healing with the formation of hypertrophic scars.

Schematic comparison between physiological wound healing and dysregulated wound repair leading to hypertrophic scar formation. Increased fibrin clot deposition, persistent inflammatory-cell infiltration, prolonged re-epithelialization, excessive angiogenesis, and abnormal collagen deposition contribute to pathologic scar development. Adapted from Mony MP, Harmon KA, Hess R, et al. [6].

Keloids represent a more aggressive fibroproliferative phenotype than hypertrophic scars. Unlike hypertrophic scars, which remain within the original wound boundaries,

keloids extend into surrounding normal skin and frequently persist or recur despite treatment. Their pathogenesis involves genetic predisposition, chronic inflammation, fibroblast hyperactivity, excessive extracellular matrix deposition, immune dysregulation, and activation of profibrotic pathways such as TGF- β /Smad, JAK/STAT, MAPK, PI3K/AKT, and mechanotransduction-related signaling. Understanding these pathways supports the rationale for multimodal treatment rather than simple excision alone [7].

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Inflammatory-cell behavior is increasingly recognized as a therapeutic target in pathologic scarring. Macrophages contribute to wound repair through cytokine release, fibroblast regulation, angiogenesis, and extracellular matrix remodeling, but persistent macrophage activation may promote fibrosis and scar progression. Therapeutic strategies aimed at modulating macrophage polarization, reducing macrophage accumulation, inhibiting macrophage-derived profibrotic signaling, or interfering with exosome-mediated communication may become relevant adjuncts in future scar treatment protocols [8].

Contemporary therapeutic framework

Current management of hypertrophic scars and keloids is best understood as a multimodal framework rather than a single-treatment pathway. Therapeutic options can be grouped into surgical approaches, physical modalities, pharmacologic interventions, and biological or emerging therapies. In clinical practice, these categories often overlap: surgery may be combined with radiotherapy, silicone, pressure, or intralesional drugs; lasers may be used alone or for drug delivery; and pharmacologic therapy may be selected according to scar activity, thickness, symptoms, recurrence risk, and patient tolerance [9].

Clinical definition and therapeutic objectives

The first step in scar management is accurate clinical classification. Hypertrophic scars are raised, erythematous, and firm scars that remain confined to the wound margins, whereas keloids extend beyond the original wound and behave as benign but locally aggressive fibroproliferative lesions. In adults, treatment goals include reduction of height, erythema, induration, pain, pruritus, and contracture, while preserving function and achieving an acceptable aesthetic result. Because complete eradication is not always realistic, success should be defined by durable symptom control, flattening, improved pliability, reduced recurrence, and patient satisfaction rather than by disappearance alone [10]. Management principles differ in pediatric patients because growth, long-term cosmetic impact, procedural tolerance, pain control, and anesthesia exposure must be considered. Children may develop hypertrophic scars after burns, surgery, lacerations, or trauma, and early conservative therapy is particularly valuable because scar remodeling continues over time. Silicone therapy, pressure therapy, and cautious intralesional therapy may be appropriate, whereas surgical excision should be selected carefully and usually combined with adjuvant strategies when recurrence risk is high [11].

Patient selection and risk stratification

Skin phototype is a central variable in scar counseling and treatment planning. Patients with darker Fitzpatrick skin types have a higher predisposition to keloid formation and may experience more pronounced pigmentary alteration after inflammation, laser therapy, cryotherapy, or intralesional treatment. In these patients, management should incorporate

conservative energy settings, careful post-procedure photoprotection, avoidance of unnecessary trauma, and explicit counseling regarding recurrence and dyschromia. Quality-of-life outcomes should be assessed alongside objective scar measurements because the psychological impact may be substantial even when the lesion is clinically small [12].

Structured treatment algorithms are useful because hypertrophic scars and keloids often require sequential escalation rather than immediate aggressive intervention. A practical approach begins with assessment of lesion type, location, activity, symptoms, previous treatment, and recurrence history. Conservative measures such as silicone, pressure, massage, and tension reduction may be followed by technologies, injectable antifibrotic agents, pigment-directed therapy, or surgical intervention. This stepwise strategy is attractive in plastic surgery because it prioritizes gradual improvement while minimizing avoidable morbidity and recurrence [13].

Rationale for combination therapy

Combination therapy is central to contemporary scar management because different modalities target different components of pathologic scarring. Silicone and pressure reduce hydration imbalance and mechanical stimulation; corticosteroids suppress inflammation and fibroblast activity; 5-fluorouracil and bleomycin reduce fibroblast proliferation; lasers improve vascularity, pliability, and drug delivery; and surgery removes bulky disease but requires adjuvant recurrence prevention. The rationale for combining scar-modulating agents is strongest in refractory, recurrent, symptomatic, or anatomically high-risk scars [14].

Pharmacologic treatment should be selected according to scar biology and patient tolerance. Corticosteroids remain widely used because of their anti-inflammatory and antifibrotic effects, but adverse effects such as dermal atrophy, telangiectasia, hypopigmentation, pain, and recurrence after cessation limit their use as monotherapy. Antimetabolites, calcium-channel blockers, botulinum toxin, and bleomycin provide alternative or adjunctive pathways, although dosing schedules, injection intervals, and outcome measures remain heterogeneous across studies [15].

Traumatic scars deserve separate consideration because mechanism of injury, contamination, tissue loss, delayed closure, tension, and inflammation influence scar maturation. Randomized controlled trial evidence in traumatic hypertrophic scars and keloids supports the concept that early scar-directed intervention can improve symptoms and appearance, but heterogeneity remains substantial. In plastic surgery, traumatic scars should be reassessed over time, with treatment escalated when erythema, height, pruritus, pain, or contracture persist despite conservative care [16].

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Surgical management

Surgery is appropriate for selected lesions that are bulky, symptomatic, functionally limiting, deforming, recurrent, or refractory to conservative treatment. However, excision alone is generally inadequate for keloids because surgical trauma can reactivate the same profibrotic process that created the

lesion. For this reason, excision should be planned as part of a multimodal protocol that may include postoperative radiotherapy, intralesional corticosteroids or 5-fluorouracil, silicone, pressure therapy, meticulous closure, and long-term surveillance [17].

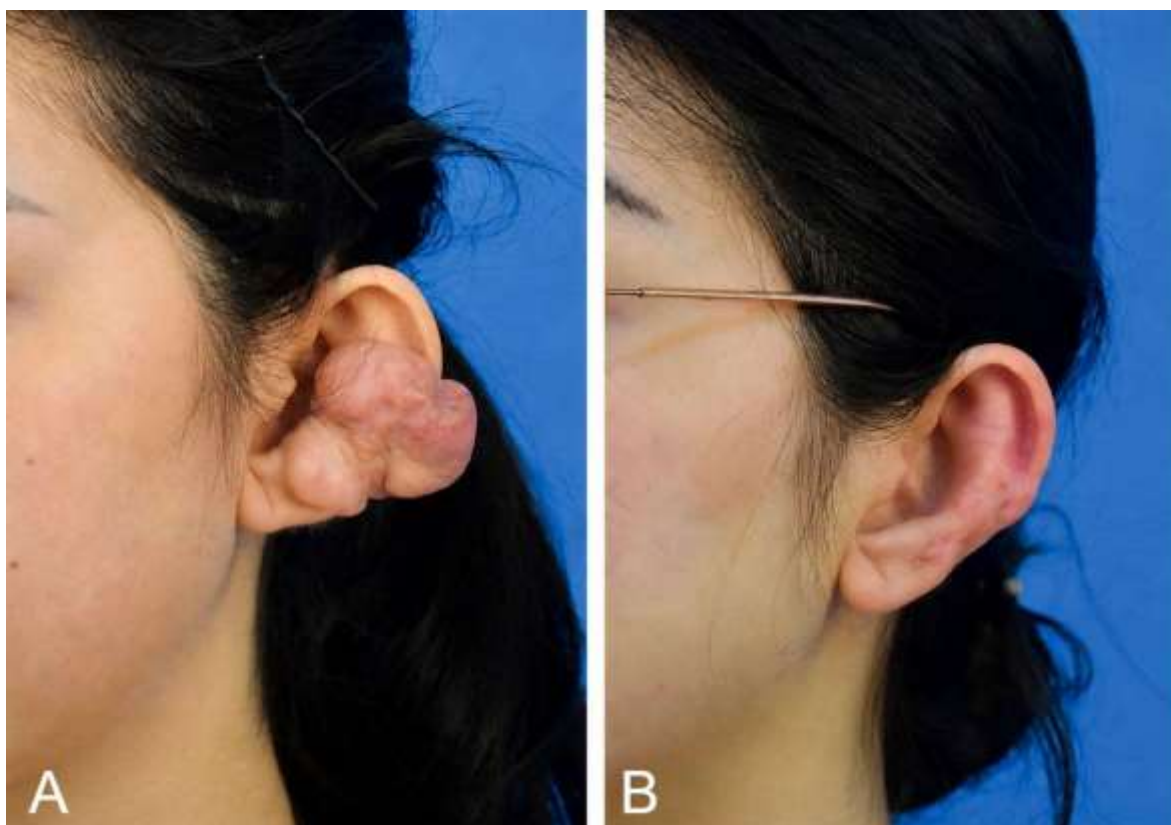


Figure 2. Ear keloid before and after comprehensive treatment.

Panel A shows a large, lobulated keloid involving the auricle before treatment, with distortion of the normal ear contour. Panel B shows the postoperative appearance after comprehensive treatment, demonstrating restoration of the auricular contour with no evident bulky keloid recurrence. Adapted from Wang et al. [18].

Plastic surgical principles are essential when operating on pathologic scars. Tension should be minimized through layered closure, fascial or deep dermal support, local tissue rearrangement, Z-plasty, W-plasty, geometric broken-line closure, or flap reconstruction when appropriate. In anterior chest keloids, where tension and recurrence are particularly problematic, perforator-based flap reconstruction may help redistribute mechanical forces and improve long-term contour. The operative goal is not simply removal of the scar but conversion of a hostile mechanical and inflammatory environment into a more stable wound-healing setting [19]. Adjuvant radiotherapy is one of the most important recurrence-reduction strategies after keloid excision, particularly for high-risk lesions of the chest, shoulder, ear,

and recurrent sites. The timing, dose, fractionation, and technique should be coordinated with radiation oncology, and patients must be counseled regarding risks, benefits, alternatives, and contraindications. Although recurrence may still occur, combined excision and radiotherapy generally outperforms excision alone and should be considered when the expected functional or cosmetic benefit justifies intervention [20].

Several invasive and interventional techniques have been incorporated into modern scar management, particularly for lesions that are bulky, recurrent, functionally restrictive, or cosmetically severe. These include surgical excision, tension-reduction closure, Z-plasty, local flap reconstruction, postoperative radiotherapy, microneedle-assisted delivery systems, and other procedure-based approaches. In plastic surgery, these techniques should be selected according to anatomical location, scar behavior, tissue availability, recurrence risk, and the patient's functional and aesthetic goals [21].



Figure 3. Massive cervicofacial keloid associated with chronic suppuration and mechanical skin traction.

Panels **A–C** show the preoperative appearance in anterior, left lateral, and right lateral views, demonstrating a large pendulous keloid involving the submental and anterior cervical region. Panels **D–F** show the postoperative appearance in anterior, left lateral, and right lateral views, with residual hypertrophic and keloidal scarring along the lower face, mandibular border, and neck. The original images were combined into a single composite figure and relabeled from **A to F**. Adapted from Banda et al. [22].

Silicone-based therapy and conservative care

Silicone gel and silicone sheeting remain foundational non-invasive therapies for prevention and treatment of hypertrophic scars. Their advantages include safety, accessibility, ease of use, and compatibility with postoperative care. Silicone may improve hydration of the

stratum corneum, reduce transepidermal water loss, modulate fibroblast activity, and improve scar color, thickness, pliability, pain, and pruritus. Adherence is crucial, and patients should be instructed that benefits require consistent use over weeks to months [23].

Postoperative scars are particularly suitable for silicone-based products because therapy can begin once epithelialization is complete and the incision is stable. In aesthetic and reconstructive surgery, silicone gel may be preferable for visible areas, mobile surfaces, or locations where sheets are difficult to secure, whereas sheets may be useful over flat or pressure-tolerant areas. Conservative therapy should be paired with sun protection, scar massage when appropriate, avoidance of unnecessary tension, and early review if erythema, pruritus, or hypertrophy progresses [24].



Figure 4. Clinical image of a linear hypertrophic scar following laparoscopic cholecystectomy.

The image shows a raised, erythematous, firm-appearing linear scar on the abdominal wall, with a measuring tape

placed below the lesion to document its approximate length and provide scale. Adapted from Drum Christie et al. [25].

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Laser and energy-based therapies

Laser therapy has become a major component of modern scar management, but the choice of device should reflect scar phenotype. Pulsed dye laser is often used for erythematous, vascular, immature scars, whereas fractional ablative lasers may improve thickness, texture, pliability, and contracture by creating microscopic treatment zones that stimulate remodeling. Evidence supports potential benefit, but protocols differ widely, and treatment should be individualized according to scar maturity, skin type, location, symptoms, and risk of pigmentary change [26].

Laser-assisted drug delivery is an increasingly relevant technique in plastic surgery because fractional ablative lasers can create microchannels through dense scar tissue, facilitating penetration of topical or intralesional agents. This approach may improve delivery of corticosteroids or other scar-modulating drugs while potentially reducing injection burden. The concept is especially appealing for hypertrophic scars with broad surface area or keloids requiring combination therapy, although optimal drug choice,

concentration, number of passes, and treatment intervals remain areas of active investigation [27].

Fractional carbon dioxide laser monotherapy may improve selected keloids and hypertrophic scars by inducing controlled dermal remodeling, softening tissue, and improving scar texture. However, keloids are biologically active lesions, and laser monotherapy may be insufficient for thick, recurrent, or rapidly growing lesions. In darker skin types, conservative parameters and careful post-procedure care are important to reduce dyspigmentation. Fractional laser is best considered part of a broader strategy rather than a universal standalone treatment [28].

Early laser intervention after burn injury has gained interest because hypertrophic scar symptoms may become established during the active remodeling phase. Beginning laser scar revision within several months after injury may help address erythema, thickness, pruritus, pain, and stiffness before mature fibrosis becomes fixed. In plastic surgery and burn reconstruction, this supports proactive scar surveillance rather than delayed referral only after contracture or severe hypertrophy has developed [29].

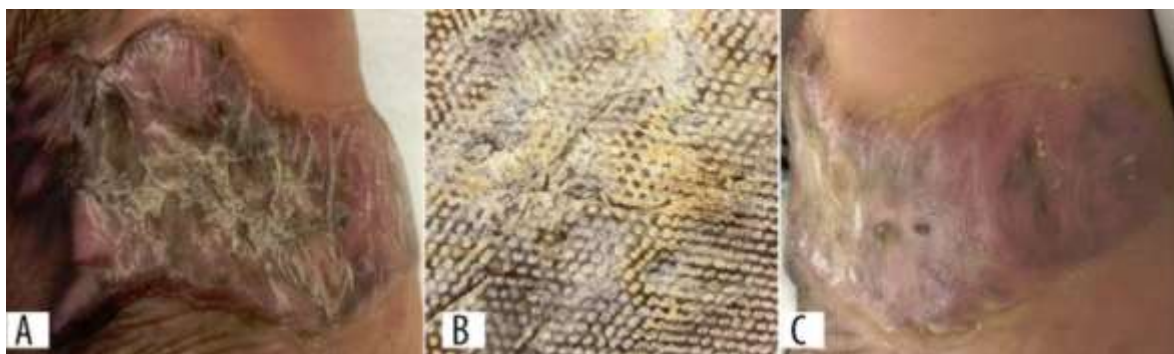


Figure 5. Clinical evolution of an extensive hypertrophic burn scar treated with multimodal laser therapy.

Panel A shows the hypertrophic burn scar before laser treatment, with thickened, erythematous, and irregular scar tissue. Panel B shows the scar after CO₂ laser treatment at 2 months, with visible fractional treatment marks and surface crusting. Panel C shows the scar after multiple cycles of combined 1540-nm and fractional CO₂ laser therapy at 3-month follow-up, demonstrating improvement in scar texture, thickness, and pigmentation. Adapted from Campolmi et al. [30].

Microneedling is a minimally invasive technique that may improve post-burn hypertrophic scars through collagen remodeling and increased dermal pliability. It is relatively accessible and may be combined with topical agents or other modalities. Compared with ablative lasers, microneedling may be attractive in resource-limited settings or in patients at higher risk for laser-associated dyspigmentation, although standardized protocols and long-term comparative data remain limited [31].

Comparative energy-based trials are useful because hypertrophic scars are heterogeneous and may respond

differently to ablative, vascular, or light-based devices. A prospective randomized comparison of ablative fractional carbon dioxide laser and narrowband intense pulsed light illustrates the need to match treatment to the dominant scar feature: erythema, thickness, pliability, dyschromia, or symptoms. In practice, combination or sequential device therapy may be reasonable when scars show mixed vascular and fibrotic characteristics [32].

Acoustic subcision represents another approach to scar remodeling by mechanically disrupting fibrotic bands and improving scar contour. Although early evidence is limited, the concept is relevant for hypertrophic scars with tethering, stiffness, or contour irregularity. In plastic surgery, subcision-based approaches should be used cautiously in active keloids but may be considered for selected hypertrophic scars after inflammation has stabilized and recurrence risk is acceptable [33].

Extracorporeal shock wave therapy has been investigated as a non-invasive modality for hypertrophic scars. Proposed mechanisms include modulation of inflammation,

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angiogenesis, fibroblast behavior, and extracellular matrix remodeling. Clinical use remains less established than silicone, corticosteroids, or lasers, but shock wave therapy may be an adjunct for symptomatic scars, especially when pain, stiffness, or pliability are major concerns [34].

Systematic review evidence on extracorporeal shock wave therapy suggests potential benefit but also highlights limitations in study design, scar type, treatment protocols, follow-up duration, and outcome measures. Therefore, shock wave therapy should not replace established treatments but may be considered as part of individualized care when conventional therapy is incomplete, contraindicated, or poorly tolerated [35].

Access to laser therapy is an important real-world issue. Even when evidence supports laser scar revision, patients may face barriers related to cost, insurance coverage, geographic availability, specialist referral, and timing after injury. For burn and trauma patients, delayed access can allow symptoms and contracture to progress. Plastic surgery services should therefore develop clear referral pathways and advocate for scar treatment as functional reconstruction, not merely cosmetic care [36].

Intralesional and injectable therapies

Botulinum toxin type A has emerged as a potential treatment for hypertrophic scars and keloids through mechanisms that may include reduction of local muscular tension, modulation of fibroblast activity, and decreased profibrotic signaling. Meta-analytic evidence from randomized controlled trials suggests that botulinum toxin may improve scar parameters in selected patients, although protocols vary in dose, dilution, injection depth, interval, and comparator treatment [37].

In pediatric post-burn hypertrophic scars and keloids, botulinum toxin type A may offer benefit while avoiding some adverse effects associated with repeated corticosteroid injections. However, children require careful pain control, dosing discipline, and longitudinal follow-up. Botulinum toxin should be considered an adjunct rather than a default

first-line therapy, particularly when conservative management remains appropriate or when scar activity is still evolving [38].

Intralesional botulinum toxin continues to be studied because it may be useful in scars located in high-tension areas or cosmetically sensitive regions. Its safety profile is generally favorable when administered correctly, but cost and availability may limit use. Future studies should clarify which lesions benefit most: early hypertrophic scars, recurrent keloids, surgical scars under dynamic tension, or patients who cannot tolerate corticosteroids [39].

Network meta-analysis of injectable therapies supports the broader principle that no single intralesional drug is ideal for every patient. Triamcinolone, 5-fluorouracil, verapamil, bleomycin, and botulinum toxin each have different efficacy and adverse-effect profiles. Treatment selection should consider scar thickness, pain tolerance, prior response, pigmentation risk, comorbidities, pregnancy status, lesion location, and the need for combination therapy [40].

Verapamil has been proposed as an alternative to triamcinolone because it may influence collagen synthesis and fibroblast activity while reducing steroid-related adverse effects. However, compared with triamcinolone, response may be slower or less pronounced in some studies. Verapamil may be useful when corticosteroid atrophy, telangiectasia, hypopigmentation, or patient intolerance is a concern, but it should be framed as one option within multimodal scar care [41].

The combination of triamcinolone acetonide and 5-fluorouracil is one of the most commonly discussed injectable regimens for keloids and hypertrophic scars. The rationale is complementary: triamcinolone reduces inflammation and collagen synthesis, while 5-fluorouracil inhibits fibroblast proliferation. Combination therapy may improve flattening and reduce steroid dose-related adverse effects, but injection pain, ulceration, hyperpigmentation, and recurrence must be monitored [42].



Figure 6. Clinical response of a pre-sternal keloid to intralesional therapy.

Panel a shows the pre-treatment lesion, characterized by a raised, hyperpigmented, firm-appearing keloid over the pre-

sternal region. Panel b shows the post-treatment appearance, with flattening of the lesion, reduced prominence, and

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residual hyperpigmented scarring. Adapted from Cheruvu et al. [43].

Recurrence after intralesional therapy remains a challenge, particularly in keloids with previous relapse or high-tension anatomical sites. Combining triamcinolone with other intralesional agents may reduce recurrence in some series, but reporting is inconsistent. For plastic surgeons, this reinforces the importance of documenting baseline scar dimensions, symptoms, photographs, treatment intervals, response, adverse effects, and follow-up duration [44].

Intralesional 5-fluorouracil is useful either alone or in combination, especially for lesions that are steroid-resistant or at risk of steroid-related atrophy. It may reduce scar height and symptoms but can cause injection pain, ulceration, crusting, or pigmentary alteration. Patient counseling and careful technique are essential, and treatment should be avoided or modified in patients with contraindications to antimetabolite exposure [45].

Randomized evidence comparing triamcinolone alone with triamcinolone plus 5-fluorouracil supports the clinical relevance of combination injection protocols. In practice, the balance between efficacy and tolerability determines adherence, because repeated painful injections can lead patients to discontinue therapy. Local anesthesia, spacing of sessions, dose adjustment, and adjunctive silicone or laser therapy may improve acceptability [46].

Bleomycin is another intralesional option for keloids and hypertrophic scars, particularly refractory lesions. It may reduce fibroblast proliferation and collagen production, but clinicians must consider pain, ulceration, necrosis, pigmentary change, and cumulative dose safety. Bleomycin is generally reserved for selected cases rather than routine first-line use, and informed consent should be explicit regarding benefits and risks [47].

Real-world evidence on bleomycin is valuable because clinical practice often includes lesions excluded from controlled trials, such as recurrent, large, symptomatic, or previously treated scars. Such data suggest that bleomycin can be effective in selected patients, but heterogeneity in protocols and follow-up limits definitive recommendations. In plastic surgery, bleomycin should be incorporated thoughtfully, particularly when other modalities have failed or are unsuitable [48].

Comparative data between bleomycin and triamcinolone highlight the need to individualize therapy rather than assuming that more aggressive pharmacologic treatment is always superior. Triamcinolone remains familiar, accessible, and inexpensive, whereas bleomycin may be useful for resistant lesions but carries distinct adverse-effect considerations. A rational injection strategy should start with patient-specific risk assessment and escalate only when expected benefit exceeds procedural burden [49].

LIMITATIONS

This review is limited by the heterogeneity of available studies, including variation in scar definitions, anatomical sites, treatment protocols, outcome measures, follow-up duration, and recurrence reporting. Many studies combine hypertrophic scars and keloids despite meaningful biological and clinical differences between the two entities. In addition, several emerging therapies are supported by small trials, retrospective cohorts, or systematic reviews of heterogeneous evidence rather than large, multicenter randomized studies.

Another limitation is that treatment outcomes are influenced by factors that are difficult to standardize, including surgical technique, wound tension, patient adherence, genetic predisposition, skin phototype, scar maturity, prior treatment, and local postoperative care. Therefore, the recommendations in this review should be interpreted as a practical synthesis for plastic surgery decision-making rather than a rigid protocol applicable to all patients.

CONCLUSIONS

Hypertrophic scars and keloids require individualized, multimodal management. The most effective strategy begins with prevention, risk stratification, atraumatic surgical technique, tension reduction, infection control, and early postoperative scar surveillance. Silicone therapy, pressure, intralesional agents, lasers, microneedling, shock wave therapy, and other non-surgical modalities can improve symptoms and scar quality, particularly when selected according to scar phenotype.

Surgery remains important for bulky, symptomatic, recurrent, functionally limiting, or cosmetically severe lesions, but excision should rarely be used alone for keloids. Adjuvant radiotherapy, intralesional therapy, silicone, pressure, and meticulous closure are essential to reduce recurrence. Future research should prioritize standardized scar classification, patient-reported outcomes, long-term recurrence data, skin phototype-specific protocols, and comparative trials of combination therapy. In current plastic surgery practice, the best results are achieved through staged, patient-centered care that combines biological control, mechanical optimization, and aesthetic reconstruction.

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