Review Article

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Scar management in pediatric patients: a current review

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ABSTRACT

Pediatric patients are particularly vulnerable to trauma and surgical interventions, both of which frequently result in cutaneous scarring. Although the complete prevention of injuries is not feasible, scar formation and long-term sequelae can be mitigated through evidence-based interventions. Current literature highlights that optimal scar management begins intraoperatively, with meticulous attention to reducing wound tension by aligning incisions along relaxed skin tension lines and achieving early closure under minimal stress. Postoperative strategies should be initiated within 2 to 3 weeks following wound closure and typically involve the use of silicone-based therapies (gels or sheeting) in combination with manual scar massage. Nevertheless, emerging evidence indicates that topical modalities alone may be insufficient in cases of erythematous or immature hypertrophic scars, where laser therapy has demonstrated superior outcomes. Pathological scar evolution—including atrophic scars, post-inflammatory hyperpigmentation, hypertrophic scars, and keloids—necessitates multimodal therapeutic approaches tailored to scar phenotype. For instance, atrophic scars may benefit from retinoid application and dermabrasion, whereas hyperpigmented lesions respond more favorably to retinoids, hydroquinone, and selective laser therapies. Hypertrophic scars and keloids require more intensive protocols, incorporating pressure therapy, intralesional corticosteroids, and laser-based interventions. In cases where pathological scarring persists beyond 12 months despite conservative and adjunctive therapies, surgical excision remains an appropriate intervention. Importantly, current evidence supports a stepwise, algorithmic approach to pediatric scar management, integrating preventive strategies, topical and device-based therapies, and surgical revision as clinically indicated.

Keywords: Pediatric patients, Pediatric scars, Scars, Scar treatment

INTRODUCTION

All wide-thickness surgical incisions can generally result in scar formation.¹ Full-thickness surgical incisions inherently carry a risk of scar formation; however, the ultimate outcome is strongly influenced by individual patient-specific factors and intrinsic healing capacity. Immature scars, defined as lesions less than one year old, often present with erythema and increased thickness. Histologically, these scars are characterized by a predominance of inflammatory cell infiltration and ongoing collagen remodelling. In contrast, mature scars, typically exceeding one year in age, are expected to

remodel into fine, linear structures with minimal chromatic disparity relative to the adjacent skin. 2

Pathological scar phenotypes—including atrophic scars, hyperpigmented lesions, hypertrophic scars, and keloids—represent the most challenging sequelae of wound healing. The variability in scar outcome is attributable to the complex interplay of genetic predisposition, wound biomechanics, and environmental factors, making consistent achievement of aesthetically favorable healing outcomes difficult. Nevertheless, adherence to evidence-based surgical and postoperative principles has been shown to mitigate unfavorable scar evolution.

In pediatric patients, scar management assumes heightened importance given the potential psychosocial implications. Notably, scars in children may expand proportionally as the child grows, increasing their visibility and psychosocial impact over time. Therefore, the optimization of scar aesthetics is a clinical priority, as these cutaneous alterations may significantly influence body image and self-perception during developmental years.³

METHODS

It is a descriptive-exploratory study type of bibliographic review. The literature search period is from 2015 to 2024 in electronic databases such as PubMed, Elsevier, and Web of Science. The keywords used in the MesH search were: pediatric patients; pediatric scars; scars; scar treatment.

Inclusion criteria

Search terms, level of evidence, summaries and keywords were included.

Exclusion criteria

Exclusion criteria were studies not related to the topic, outside the year limit, and not available.

They will be classified by year, type of study and level of evidence. For eligibility, a critical reading is carried out, level of evidence, documents available for analysis and according to the topic. A total of 35 sources were obtained for analysis and synthesis.

RESULTS

Procedures for proper scar management before scar formation

Meticulous planning of the surgical incision represents a fundamental principle in the prevention of adverse scar formation. Adherence to established surgical guidelines is essential to optimize scar cosmesis, with a preference for incision placement in anatomically concealed locations whenever feasible. When such placement is not possible, strategies should be employed to minimize scar visibility and long-term morbidity. Excessive mechanical tension at the wound edges during closure is a well-documented factor contributing to scar widening, hypertrophy, and overall poor aesthetic outcomes. To mitigate these effects, closure should be performed under the lowest possible tension, thereby promoting more favorable wound healing dynamics. ⁵

This objective can be achieved through alignment with the relaxed skin tension lines (RSTLs), which represent theoretical vectors of minimal tension and correspond to natural skin furrows generated by underlying muscular contraction. Incisions placed parallel to RSTLs demonstrate superior outcomes, as they distribute tensile

forces more evenly, reduce wound dehiscence, and consequently diminish the risk of hypertrophic scar formation.

Wound closure

Wound closure should minimize wound tension. Early primary wound closure is a critical step to induce wound epithelialization as quickly as possible because wound closure beyond 15 days increases the likelihood of wound hypertrophy.⁵ Large incisions that involve all skin layers should be closed in layers, beginning with the deep dermal layer and then the epidermal layer. There is strong evidence that deep dermal sutures decrease tension in the superficial layers and align the dermis, resulting in improved cosmetic results.⁶ Wounds closed in a single deep dermal layer had more prominent appearances at 3 months and more prominent scar color at 12 months compared with wounds closed in two layers.⁶ Fullthickness wounds closed in a single epidermal layer may result in parallel rows similar to those seen on a railroad track due to pressure necrosis of the skin beneath the suture.⁷ However, suturing with too little tension can lead to wound dehiscence.

Optimal wound closure must aim to minimize mechanical tension, as excessive stress across wound margins is a key determinant of hypertrophic scar formation. Early primary closure is considered a critical intervention to facilitate rapid re-epithelialization, with evidence indicating that delayed closure beyond 15 days significantly increases the risk of scar hypertrophy. On the other hand, patients reported less discomfort with absorbable sutures, which is beneficial in the treatment of pediatric patients. In the case of wide or full-thickness incisions involving multiple skin layers, a layered closure technique is strongly recommended.

This approach begins with approximation of the deep dermal layer, followed by closure of the superficial epidermal layer. Robust evidence supports that placement of deep dermal sutures reduces tension across the superficial layers, aligns dermal collagen fibers, and subsequently enhances cosmetic outcomes.

Clinical studies have demonstrated that wounds closed in a single deep dermal layer exhibited more pronounced scar elevation at 3 months and greater scar pigmentation at 12 months, compared with wounds treated using a dual-layer closure technique. Conversely, wounds managed with a single superficial epidermal suture layer are prone to the formation of parallel linear marks resembling "railroad tracks," largely attributable to localized pressure necrosis beneath the suture points.

On the other hand, insufficient suture tension carries the risk of wound dehiscence, underscoring the importance of achieving an optimal balance between minimizing closure tension and maintaining structural wound integrity.

Immature scars

Immature scars typically emerge following suture removal and represent a critical period for early intervention. Current evidence supports the initiation of adjunctive scar therapies within 2 to 3 weeks after scar formation to optimize aesthetic outcomes and reduce the risk of pathological scar development. Silicone gel and sheeting have shown effective results for the aesthetic improvement of scars and the prevention and treatment of pathological scars (Figure 1). Silicone should be applied at least 12 hours a day starting 2 weeks after surgery. Silicone gel can reduce scar volume and increase wound elasticity in 60–100% of cases.

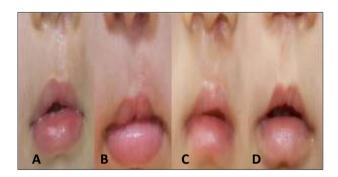


Figure 1 (A-D): Babies from the study group (silicone gel group) showing similar quality scars to the control group (silicone sheet group).¹¹

Among the available modalities, silicone-based therapies (gel or sheeting) combined with manual scar massage constitute the cornerstone of conservative management. Silicone has consistently demonstrated efficacy in both improving scar cosmesis and preventing the evolution of pathological scars, including hypertrophic scars and keloids. Clinical data indicate that daily application of silicone gel or sheeting for a minimum of 12 hours, commencing approximately 2 weeks postoperatively, results in significant clinical benefits. Specifically, silicone gel has been shown to reduce scar volume and enhance wound elasticity in 60–100% of treated cases, highlighting its role as a first-line intervention in pediatric scar management.

Mature scars

During the transition from immature to mature scar tissue, a dynamic remodelling process occurs in which inflammatory cells, epithelial cells, and the majority of fibroblasts undergo apoptosis, leaving behind organized bands of collagen fibres as the structural framework of the mature scar.¹⁰

Interventions aimed at reducing wound tension, as well as adjunctive therapies such as silicone gel application, massage therapy, and selective laser treatments, have demonstrated efficacy in promoting more favourable cosmetic outcomes during this critical remodelling phase.

Nevertheless, the maturation process is not always linear and may be complicated by the development of pathological scar phenotypes, including atrophic scars, hyperpigmentation, hypertrophic scars, and keloids. These lesions not only represent a biological dysregulation of wound healing but also exert profound effects on the functional and psychosocial well-being of pediatric patients. Consequently, early recognition and proactive management of aberrant scar formation are essential components of comprehensive pediatric care.

Atrophic scars can be broadly classified into three distinct subtypes—ice-pick, boxcar, and rolling scars—each of which has therapeutic implications that guide clinical decision-making. Topical retinoids have emerged as a cornerstone in the management of atrophic scars, demonstrating substantial efficacy both as monotherapy and in combination with adjunctive agents.¹¹

Clinical trials have shown that daily application of adapalene 0.3% gel resulted in significant improvements in skin texture and atrophic scar morphology, with reductions of approximately 50% at 12 weeks and exceeding 80% after 24 weeks of treatment. More recently, trifarotene, a novel FDA-approved topical retinoid, has demonstrated statistically significant improvements in scar remodelling with favorable safety and tolerability profiles. ¹²

Combination regimens have also yielded enhanced outcomes. Specifically, daily administration of adapalene 0.3% in conjunction with benzoyl peroxide 2.5% was associated with a marked reduction in scar burden and improved overall skin quality. The average patient age across these studies was approximately 16 years, underscoring the relevance of retinoid-based therapy in adolescent populations. Importantly, current evidence supports the safety and efficacy of topical retinoids and benzoyl peroxide in pediatric cohorts, although caution and individualized monitoring are recommended in younger children.

Microdermabrasion has been shown to be more suitable for pediatric patients; the procedure can be performed using local anesthetic and its complications are less severe. However, dermabrasion is more effective, particularly for deeper scars. Sessions can be performed every 2 to 8 weeks, with at least 4 to 6 treatments. Chemical peels, such as Jessner's solution and 70% glycolic acid peel, significantly improved results compared with microdermabrasion alone

DISCUSSION

Hyperpigmented

Hyperpigmented scars, frequently referred to as postinflammatory hyperpigmentation (PIH), result from an exaggerated melanogenic response following cutaneous inflammation. This process leads to localized overproduction and deposition of melanin within the epidermis and, in some cases, the dermis. Paediatric patients with darker skin phototype were disproportionately affected due to their higher baseline melanin content, which predisposes them to more pronounced and persistent PIH. If left untreated, PIH may persist for several months to years, potentially contributing to psychosocial distress, including reduced self-esteem and negative body image in children and adolescents.

Management of PIH requires a dual approach: addressing the underlying inflammatory dermatosis and implementing targeted interventions for pigment modulation. In paediatric populations, the most prevalent etiological factor is acne vulgaris, followed by other inflammatory dermatoses such as atopic dermatitis and impetigo. Preventive strategies remain central to reducing PIH burden, with daily use of gentle, non-irritating skin care regimens—including mild cleansers and emollient-based moisturizers—playing a protective role. 14

Furthermore, photoprotection constitutes a cornerstone of PIH management. Broad-spectrum sunscreens with a sun protection factor (SPF) of 30 or greater, combined with physical barriers such as protective clothing, reduce ultraviolet-induced melanogenesis and mitigate the exacerbation of hyperpigmented lesions. Early and consistent implementation of these strategies is essential to minimize disease chronicity and optimize both cosmetic and psychosocial outcomes in paediatric patients.15 Topical retinoids applied once daily are the first-line therapy for PIH caused by acne (Figure 2).

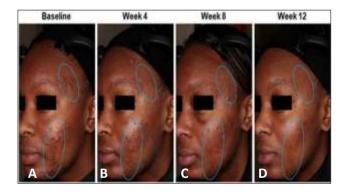


Figure 2 (A-D): Acne hyperpigmentation (indicated by circled areas) improvements in a 15-year-old who was treated with tazarotene 0.045% lotion once daily for 12 weeks.¹⁸

Hypertrophic

Hypertrophic scars are scars caused by excess inflammatory mediators and fibroblast proliferation during their formation (Figures 3A and B). Hypertrophic scars arise as a consequence of excessive inflammatory mediator release and fibroblast proliferation during the wound healing process. Although some hypertrophic scars may undergo partial regression within the first year

following their formation, their management continues to present significant clinical challenges. 16

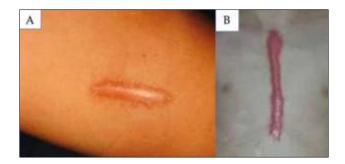


Figure 3: (A) Hypertrophic scar, and (B) chest hypertrophic scar.³⁰

In paediatric patients, the presence or history of hypertrophic scarring warrants particular attention, as these scars may exert both functional and psychosocial consequences. When wounds occur in areas subjected to high mechanical tension, or when early signs of hypertrophic changes are observed, pressure therapy is considered the first-line intervention. 18 Pressure therapy relies on the application of external mechanical forces through devices or garments such as compression dressings, snap buttons, or ear clips. The therapeutic mechanism is thought to involve localized ischemia, which subsequently enhances collagenase activity, modulates fibroblast function, and accelerates wound remodelling. This approach has been supported by evidence demonstrating improved outcomes in both scar thickness and elasticity, thereby reducing the clinical burden of hypertrophic scarring in paediatric populations. 19

When less invasive modalities such as silicone gel or sheeting and pressure therapy fail to achieve adequate improvement within two months, subdermal corticosteroid injections should be incorporated into the therapeutic regimen. Corticosteroids exert their effect by downregulating pro-inflammatory mediators, inhibiting keratinocyte and fibroblast proliferation, and inducing vasoconstriction, which collectively reduce nutrient delivery to scar tissue and suppress excessive collagen deposition.²⁰

Clinical evidence indicates that subdermal corticosteroid therapy can achieve regression rates of 50 to 100 percent in hypertrophic scars. The most widely used agent is triamcinolone acetonide (TAC), administered in doses ranging from 2.5 to 40 mg per site, typically mixed with a local anaesthetic to mitigate pain. Pain associated with intralesional corticosteroid administration can be a significant barrier to adherence in paediatric patients, underscoring the importance of appropriate anaesthetic techniques to improve tolerance and ensure treatment continuity.²¹

Alternative pharmacological therapies for the management of hypertrophic scars include 5-fluorouracil (5-FU) and

bleomycin. 5-FU is a pyrimidine analogue with potent antifibrotic activity, primarily achieved through the inhibition of fibroblast proliferation. While it may be administered as monotherapy, clinical evidence indicates that its efficacy is significantly enhanced when combined with intralesional corticosteroids such as triamcinolone acetonide (TAC). Studies have demonstrated that the combination of 5-FU and TAC produces superior outcomes compared with TAC alone, particularly in terms of reducing scar height, erythema, and improving patient-reported satisfaction.²²

For severe or extensive hypertrophic scars, monthly administration of approximately 0.9 ml of a 50 mg/ml 5-FU solution in combination with corticosteroids has been recommended. Bleomycin, a cytotoxic antibiotic, represents another therapeutic option. Its antifibrotic effects are mediated through the inhibition of collagen synthesis and the induction of fibroblast apoptosis, thereby contributing to scar regression. Both agents are generally reserved for refractory cases where first-line modalities have proven insufficient.²³ Monthly administration of 0.9 ml of a 50 mg/ml 5-FU solution combined with corticosteroids has been recommended for severe or large hypertrophic scars.24 Bleomycin is a cytotoxic antibiotic that inhibits collagen synthesis and induces apoptosis.²⁶

Keloids

Keloids are a form of pathological scarring resulting from the proliferation of fibrous tissue in the dermis. Unlike hypertrophic scars, keloids grow beyond the margins of the initial wound. Treatment for keloids is similar to that for hypertrophic scars, but should be more aggressive, as keloids are less likely to regress and can continue to grow for a longer period than hypertrophic scars.

Intralesional corticosteroid therapy is widely regarded as the first-line treatment for keloids, with the most favorable outcomes achieved when corticosteroids are combined with adjunctive modalities such as compression therapy, laser ablation, or surgical excision. Radiation therapy, while sometimes employed in adult populations, is generally contraindicated in pediatric patients due to the potential for deleterious effects on developing tissues and the associated risk of secondary malignancy. In the pediatric population, intralesional TAC injections alone resulted in an 82.7% reduction in keloid size, and intralesional TAC used in combination with surgical excision and CO2 laser therapy resulted in complete keloid removal in 82.4% of patients.

In children and adolescents, intralesional triamcinolone acetonide (TAC) injections have demonstrated considerable efficacy, with studies reporting an average reduction in keloid size of approximately 82.7 percent. When TAC is administered in combination with surgical excision and carbon dioxide (CO₂) laser therapy, complete keloid resolution has been observed in up to 82.4 percent of cases. These findings underscore the importance of

multimodal treatment strategies tailored to the unique biological and psychosocial considerations of pediatric patients affected by keloid scarring.³¹ Injections should start at 4-week intervals for a few months, followed by injections every 6 to 10 weeks for several months, and finally titrated to every 12 weeks.³² As for intralesional bleomycin, intralesional doses of 1.5 IU/ml are recommended.

Small keloids that fail to improve within 8 to 12 weeks despite treatment with silicone gel or sheeting and intralesional corticosteroids, as well as larger keloids unresponsive to combined corticosteroid and 5-fluorouracil (5-FU) therapy, may be considered for laser-based interventions. Clinical data indicate that thick keloids exhibit limited responsiveness to 585-nm pulsed dye laser (PDL) therapy when used alone. However, improved outcomes have been observed when PDL is combined with intralesional corticosteroids or 5-FU injections, or when fractional CO₂ laser therapy is applied.

Although PDL may provide symptomatic relief by reducing pain and pruritus associated with keloids, its therapeutic mechanism primarily targets vascular components of scar erythema rather than scar bulk. Consequently, PDL is not recommended as monotherapy for the management of keloids but may serve as an effective adjunct within a multimodal treatment strategy.³³

Cryotherapy represents another therapeutic modality for the management of keloids, employing liquid nitrogen or argon gas to induce localized tissue necrosis through freezing. Intralesional cryotherapy, which targets the keloid from within, has demonstrated superior efficacy compared to conventional contact cryotherapy.³⁴ When used in combination with other modalities, particularly intralesional corticosteroids, contact cryotherapy may provide clinical benefit, though patients often require multiple sessions, with some reports indicating up to 20 treatments. Intralesional cryotherapy, by contrast, generally requires fewer sessions while achieving more substantial outcomes. A systematic review of eight studies reported mean scar volume reductions of 51 to 63 percent, accompanied by recurrence rates ranging from 0 to 24 percent in keloid patients treated with this approach. These findings suggest that intralesional cryotherapy may serve as a more efficient and effective adjunctive therapy within multimodal keloid management protocols.³⁵

CONCLUSION

Pediatric scars carry significant psychosocial implications, affecting not only the patients but also their families. Although evidence specific to pediatric scar management remains limited, fundamental principles of scar prevention and optimization can be universally applied across age groups. Preventive strategies should begin intraoperatively, with meticulous incision planning oriented parallel to relaxed skin tension lines and closure

techniques designed to minimize tissue trauma and wound tension.

In the postoperative phase, conservative modalities such as silicone gel or sheeting and scar massage should be prioritized for a duration of 2 to 6 months. For immature scars that remain erythematous, PDL therapy is recommended, whereas persistent thick immature scars may benefit from ablative CO₂ laser interventions. As scars progress to maturity, early identification of pathological scar phenotypes—atrophic scars, hyperpigmentation, hypertrophic scars, and keloids—is essential to initiate targeted therapies promptly and prevent long-term functional and psychosocial sequelae.

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