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Review Article

Recent Advances of Keloid Scar Management: Clinical Review

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Abstract

Keloids are benign dermal fibroproliferative disorders resulting from abnormal wound healing characterized by excessive deposition of collagen and progressive extension beyond the original wound borders. Despite numerous available therapeutic modalities, keloid management remains a considerable clinical challenge, as no single therapy has been universally accepted as the definitive gold standard for all scar subtypes. High recurrence rates persist across treatment modalities, largely due to heterogeneity in lesion characteristics, patient variables and therapeutic techniques. These challenges are further exacerbated by key limitations in the existing scientific literature, including a lack of high-quality Randomized Controlled Trials (RCTs), inadequate long-term follow-up durations, small sample sizes, heterogeneous outcome measures and frequent failure to clearly differentiate keloids from Hypertrophic Scars (HTS).

This narrative review synthesizes current evidence and assesses the effectiveness of a uniform multimodal protocol involving topical corticosteroids, surgical excision and intralesional corticosteroid injections in reducing keloid recurrence. Literature searches were conducted using PubMed, Google Scholar and ResearchGate, incorporating scoping reviews, RCTs, prospective clinical studies and quasi-experimental studies. The current literature review strongly supports multimodal or combination therapy as the most effective strategy for keloid management. Although Intralesional Corticosteroid Administration (ICA) remains a first-line intervention, its variable outcomes highlight the urgent need for standardized injection techniques, optimized dosing protocols and consistent reporting of objective results.

Keywords: Keloid; Topical Steroids; Corticosteroids; Hypertrophic Scar; Fibrosis; Wound Healing

Introduction to Keloid and Steroid Cream

Keloids and hypertrophic scars are among the most common therapeutic challenges that dermatologists face in their daily practice. Statistics have shown that more than 100 million patients worldwide experience scars after surgical interventions [1]. Keloids are known as fibroproliferative disorders of the skin or benign mesenchymal tumors, characterized by excessive tissue growth that often recurs even after excision and extends beyond the wound margin. They are common in African, Asian and Hispanic populations as they are considered high-risk populations with an increase of 5% to 16% [2]. The term "keloid" is derived from the Greek word 'khele' or 'chele', meaning 'crab claw', which describes their tendency for lateral expansion into surrounding normal tissue. Keloid scarring is not a recent affliction, with recorded history dating back to approximately 3000 BC in the Edwin Smith papyrus [3]. In 1806, the first clinical description of keloids was given by Alibert [4]. The common clinical manifestation of keloids is ill-defined papules with variant pigmentation and elevated, firm bosselated papules [5]. Reported symptoms and complaints can be pruritus, pain and psychological distress that can affect well-being and quality of life [6].

Pathophysiology of Keloids: From Fibroblast Activity to Mechanical Forces

The commonly known mechanism for the formation of keloids is the excessive collagen deposition and wound healing distortion due to chronic, localized inflammation in the reticular dermis. However, the exact underlying mechanism is complex and remains unknown. Etiologies such as wound tension, skin pigmentation, skin injury and genetic predisposition can alter the process of keloidogenesis. The common key elements that can define keloid pathogenesis are:

Fibroblast Activity and Abnormal Collagen Production: Keloids have a greater capacity to proliferate and produce high levels of collagen, proteoglycan and elastin. Keloids are characterized by over-proliferation and reduced apoptosis of the dermal fibroblasts [7]. Following injury, the inflammatory phase recruits cells such as neutrophils and macrophages to the wound site. The reticular layer of keloids and hypertrophic scars contains inflammatory cells, increased fibroblasts and newly formed blood vessels. One common pathology that contributes to the proliferation of extracellular matrix and upregulation of collagen synthesis is the overproduction of Transforming Growth Factor (TGF) and other cytokines. Proinflammatory factors such as interleukin (IL)-1, IL-1, IL-6 and tumor necrosis factor are upregulated in keloid tissues.

Keloids are characterized by extensive intradermal collagen deposition. Keloid tissue is composed of disorganized collagen, specifically Type I and Type III collagen, arranged haphazardly. Keloids contain thick bundles of closely packed type I, III, IV and V collagen. A unique feature of keloid scars is the presence of thick and uniformly stained collagen fibers known as keloidal or hyalinized collagen.

Mechanical Forces and Risk Factors: Wound skin tension and location of the scar are considered highly important contributors to pathological scar development and exacerbation. Keloid growth patterns align with the predominant direction of skin tension as well as the differentiation for HTS in diagnosis and treatment. Genetic components in specific ethnic groups, including African, Asian and Hispanic populations, are attributable to the formation of keloids. Darker skin tends to form keloids 15 times more likely than lighter skin pigmentation [8]. Other systemic factors, such as hormonal peaks during adolescence or pregnancy and conditions like hypertension, are known to worsen keloid formation.

Management

There has been no single therapy or gold standard for the treatment of keloids and it continues to be a challenge for experts in the dermatological field. In this section, further exploration of diverse keloid management.

Intralesional Injections (Corticosteroids and Antimetabolites)

Intralesional injections are a cornerstone of keloid treatment, particularly corticosteroids:

- *Corticosteroids (e.g., Triamcinolone Acetonide - TAC):* Corticosteroids or steroids are considered first-line therapy in the management of keloids and their prevention. For example, TAC is frequently used as an intralesional steroid because it exerts therapeutic effects by decreasing fibroblast proliferation, inflammation and reducing collagen synthesis, while also modifying extracellular matrix components [9]. In addition to the micro level of improvements, associated symptoms such as itching and pain are also reduced when applied to the keloid scar. The marked efficacy of intralesional steroids has led to the development of topical steroid applications. Steroid ointments or creams or steroid tapes/plasters are sometimes used as a first-line therapy, especially in the Neuroleptic Malignant Syndrome (NMS) protocol, which is optimized for Japanese patients [10]
- *Other Injections (e.g., 5-Fluorouracil - 5-FU):* 5-FU, a chemotherapeutic agent, that inhibits cell proliferation and enhances fibroblast apoptosis. 5-FU is used when keloids are not responsive to corticosteroid treatment or used in combination with corticosteroids to improve results further [11,12].

Surgical Excision

Surgical excision is a popular choice that is also recommended as a first-line therapy if disabling scar contracture is present and it involves physically removing the scar tissue [13]. However, excision of keloids without any adjuvant therapy should be strictly avoided due to a high rate of recurrence, which ranges widely from 45% to 100%.: To avoid recurrence, excision is almost always combined with adjuvant measures such as radiotherapy, intralesional corticosteroids, cryotherapy or 5-FU [14].

Silicone Sheets and Gels

Silicone-based products are sometimes called the gold standard of non-invasive therapies in keloid management and in preventing abnormal scarring, a primary conservative option for scar management. Their primary mechanism of action is occlusion and hydration of the stratum corneum, which leads to lower levels of collagen deposition and improved angiogenesis [15]. Silicone materials are available as sheets, gels or sprays. They are typically applied continuously for 12-24 hours per day for 3–6 months [16].

Laser Therapy

Laser therapy is commonly used as an adjunct treatment after silicone therapy and corticosteroids fail to provide sufficient improvement. The 585-nm Pulsed Dye Laser (PDL) improves scars by inducing capillary destruction, which generates hypoxemia and may alter local collagen production [17]. Ablative fractional lasers (e.g., CO_2) are on the rise in their use, with emerging evidence supporting their efficacy, sometimes over PDL for postsurgical scars [18].

Cryotherapy

Cryotherapy is a physical modality that can be used alone or in combination with other treatments and it functions by inducing cellular injury and necrosis of keloid tissue, typically administered using liquid nitrogen. It can be applied via contact, spray or intralesional injection [19]. Intralesional cryotherapy, which concentrates the cold within the lesion, is considered more effective than contact or spray methods. Cryotherapy combined with intralesional corticosteroid injection is a common traditional approach for keloids [20].

Keloids and Topical Steroid Creams

The use of topical steroid creams and ointments is usually used as an adjuvant therapy or in combination procedures in managing keloids. While Intralesional Corticosteroid Injection (ILCSI) remains the first-line treatment for keloids, topical steroid formulations are utilized to minimize the invasiveness and adverse effects associated with injections.

Types of Steroids and Rationale for Use

Corticosteroids have the main function of anti-inflammatory and immunosuppressive properties, inhibit fibroblast proliferation, reduce the excessive synthesis of collagen and Extracellular Matrix (ECM) components and suppress inflammation. However, the efficacy of traditional topical steroid formulations is often constrained by the stratum corneum barrier, resulting in low transdermal efficiency and short action time, preventing adequate drug concentration within the dense scar tissue [21]. The choice of topical corticosteroid is primarily based on its potency, which is categorized based on the method

Clobetasol Propionate (0.05% cream): It is classified as very high potency and is used in combination with silicone occlusion in comparison to intralesional triamcinolone [22].

Fluocinolone Acetonide (0.2% cream): A highly potent agent, which was used in a case series for keloids and was applied three times a day in 192 patients with variable conditions and including 12 cases of keloids. The results were variable, 2 cases showed clearance, 7 showed moderate response and 3 cases showed no response [23].

Methylprednisolone Aceponate (MPA) (0.1% cream): Agent potency is moderate and is used for the prevention of post-operative scars, such as following Cesarean section [24].

Betamethasone Valerate (0.025% cream or ointment): It is classified as medium potency and used as an ointment in the postoperative protocol, combining injections and topical application [21].

Other applications with variable potency: Triamcinolone Acetonide (TA) Lotion is used in an emulsion combined with an occlusive biosynthetic film (cynthaskin) [25]. Betamethasone Cream is used in combination with fractional ablative laser therapy (laser-assisted drug delivery) for resistant keloids [26].

Lower potency topicals are used with limited duration on sensitive areas such as facial scars to avoid any potential adverse effects. One protocol involving surgery and injections is using three different types of topical corticosteroids (Diflorasone diacetate, difluprednate and betamethasone valerate) continuously over six months and changing them every two months to minimize potential skin side effects (Table 1) [27].

Steroid	Potency	Concentration	Clinical Notes
Clobetasol propionate	Very high	0.05%	Effective under occlusion; alternative to TAC injection [27]
Fluocinolone acetonide	High	0.2%	Variable efficacy; early evidence [28]
Methylprednisolone aceponate (MPA)	Moderate	0.1%	Effective for early postoperative scars [29,33]
Betamethasone valerate	Medium	0.025%	Used postoperatively with injections [26]
Triamcinolone lotion	Medium	—	Used with occlusive films for symptom control [30]
Difluprednate/diflorasone	High	—	Used in rotation protocols post-surgery [32]

Table 1: Common topical steroids used in keloid management.

Topical Steroids: Efficacy, Successes and Improvements

Scar types and formulations are two main factors that predict the efficacy of topical steroid creams. The reported success of topical steroid creams has shown to relieve symptoms and improve scar characteristics with their consistent application. Topical corticosteroid therapy provides symptomatic relief from pruritus (itching) and pain. TA lotion combined with an occlusive film was used in a study and has reported that 6 out of 9 keloid patients who complained of itchiness experienced improvement after starting treatment [25]. In terms of scar height, one trial assessed the use of methylprednisolone cream for post-Cesarean scars after the three-month evaluation; the scar was significantly reduced compared to the control group [28]. In addition to the scar height, the overall appearance was improved after the use of topical TA lotion in 44% of keloid patients in at least three parameters out of five and they are colour, itchiness, elevation, texture and pain [25]. Furthermore, patient satisfaction was a positive factor for the potential use of topical steroids in keloids. One study compared methylprednisolone cream and silicone gel after a Pfannenstiel incision. Patients were satisfied with methylprednisolone group (48%) than in the silicone group (33%) or the control group (11%) and scored higher out of all groups (Table 2) [29]. Fig. 1 demonstrates the effectiveness of topical steroids on keloid scars.

Study	Steroid	Design	Key Outcomes
Yii & Frame (1996)	TA lotion + occlusion	Prospective	Improved pruritus, pain, texture
Meseci (2019)	MPA 0.1%	Clinical trial	Reduced scar height & vascularity
Nor et al. (2017)	Clobetasol + silicone	RCT	Comparable to IL-TAC
Ricciardi et al. (2025)	MPA vs imiquimod	RCT	MPA superior for prevention

Table 2: Outcomes of tropical steroid studies.

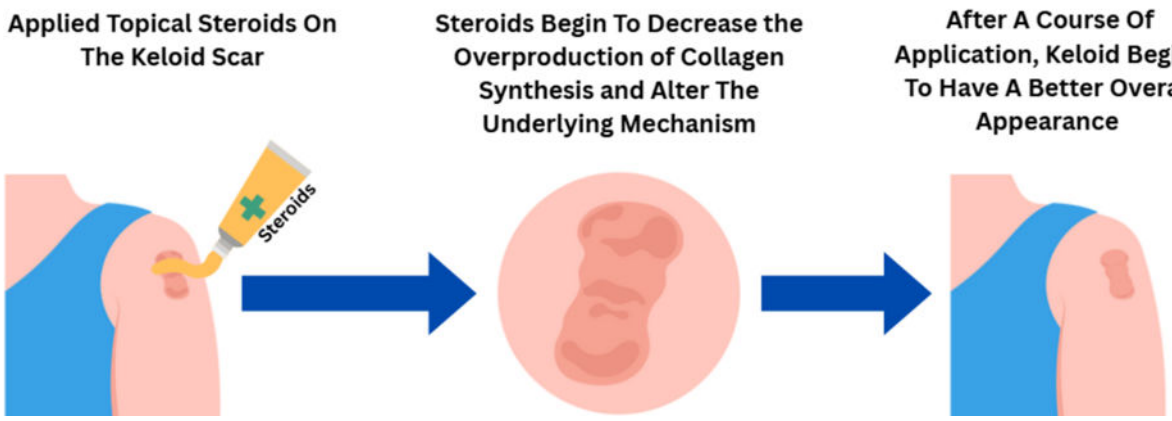

Figure 1: The efficacy of topical steroids in improving keloid scar.

The following figure shows the application of topical steroids on keloid scars by decreasing the overproduction of collagen and altering the underlying mechanism. The results improve the overall appearance of the scar.

Why Topical Steroids Aren't Always Effective (Limitations)

The common limitation of applying topical steroid monotherapy is due to various reasons, such as the maturity of the scar and dense keloids. One of the drawbacks of using topical steroids is the temporary effects after the treatment is discontinued. A prophylactic study of methylprednisolone cream for post-Cesarean scars, while positive effects were seen for vascularity and height at three months, the outcomes were similar to the control group at six months [28]. Poor absorption and penetration are common limitations that result in low transdermal efficiency, reduction of drug concentration and a short duration of action [28]. Lastly, a lack of large-scale randomized controlled studies can lead to practice reluctance [30].

Adverse Effects

The extended use of topical steroids is a critical consideration for scar management due to their adverse effects. Skin atrophy after long-term use of topical steroids is commonly associated with intralesional corticosteroid injections [31]. Telangiectasias are the formation of spider veins, which is a potential local side effect of prolonged topical corticosteroid use [21]. Hypopigmentation or general pigment changes are concerns associated with steroid use, particularly in patients with Fitzpatrick skin types IV-VI (skin of colour) [32]. Systemic complications, such as Cushing's syndrome, occur with high doses or prolonged application over large surface areas [33]. Other reported side effects include contact dermatitis, local irritation, itching, skin maceration and folliculitis [26].

Combination Therapies

Topical steroid creams and ointments are frequently used as part of multimodal treatment protocols to enhance efficacy and reduce recurrence rates. Fig. 2. Exemplifies the diverse combination therapies of topical steroids in keloid treatment.

1. Combination with Surgical Excision and Injections: The combination of five intralesional Triamcinolone Acetonide (TA) injections every 2 weeks after suture removal, with self-administered topical steroid ointment daily for 6 months, was used to reinforce the effect of injected steroids. This approach resulted in a low rate of keloid for 14.3% [27]. Similarly, the combination treatment of surgical excision and subsequent intralesional TA injection with a recurrence rate of less than 50% [34]
2. Combination with Occlusive Dressings (Silicone/Pressure Garments): To increase percutaneous absorption, incorporating topical steroids and occlusion. One trial compared Clobetasol Propionate 0.05% cream under silicone dressing occlusion versus intralesional TA for keloid treatment [22]. Silicone gel sheeting can serve as an adjunct to other therapies due to its low adverse effect profile and ability to modulate scar formation [35]. TA lotion combined with cynthaskin can form an adherent and transparent occlusive film used for exposed parts of the body with scars [25]. Another therapy used after surgery is using triple therapy by using TA cream and intrakeloidal injections with long pressure applications of silicone gel strip/sheets. The success of this regimen was recorded at a rate of 87.5% at 13 months, although it was noted to be time-consuming and tedious [36]

3. Combination with Laser-Assisted Drug Delivery (LADD): Combining LADD and topical steroids can facilitate topical drugs to have deeper penetration [37]. A study has shown that the use of topical TA with CO2 laser can decrease scar volume in comparison to intralesional TA injections [37]. Another study was conducted by combining topical betamethasone cream to treat keloids that are resistant to first-line therapies and with an improvement of 50% and reported a reduction of pruritus and pain [38]. The LADD approach is considered a safer and better aesthetic treatment option than standard injections (Table 3)

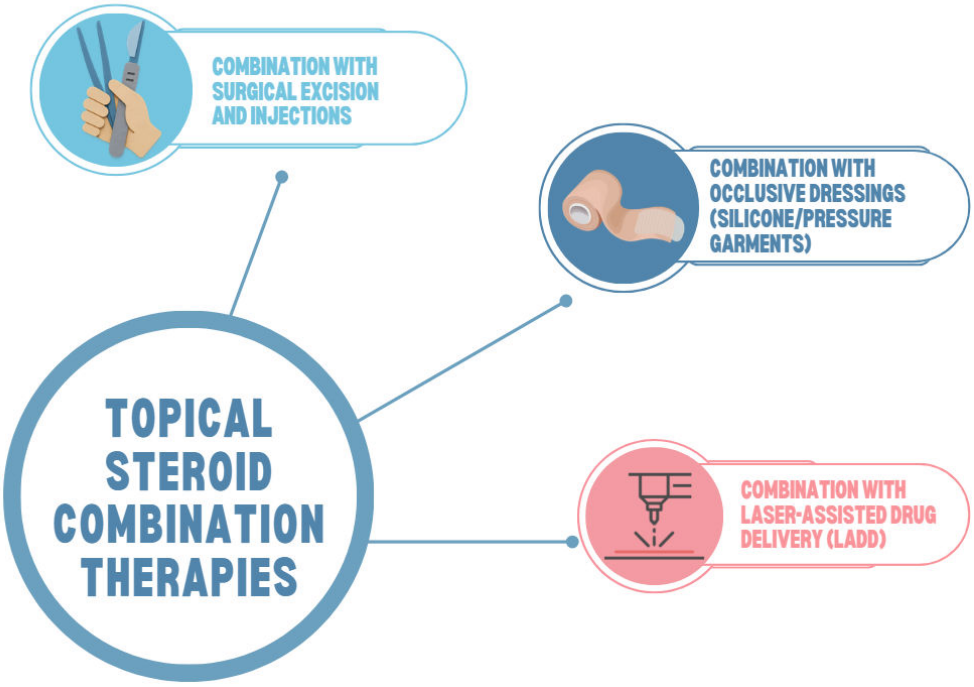

Figure 2: Topical steroid combination therapies in keloid management flow-chart.

Summary Table of Keloid Treatment Options

Treatment	Pros	Cons	Best Use
TAC (IL steroids)	First-line; reduces height & symptoms	Atrophy, pigment change	Most keloids; postop
5-FU	Good for steroid-resistant lesions	Pain, ulceration	With TAC
Excision	Immediate flattening	Very high recurrence alone	Only with adjuvants
Radiotherapy	Lowest recurrence	Cost; radiation risk	Large/recurrent
Silicone	Safe, non-invasive	Needs long-term use	Prevention; early scars
Pressure therapy	Effective for ears	Uncomfortable	Ear keloids
Laser (PDL/CO_2)	Improves color/texture	Cost; PIH	Symptomatic scars; LADD
Cryotherapy	Flattens thick nodules	Hypopigmentation	Thick lesions (light skin)
Topical steroids	Non-invasive; ↓ itch	Limited penetration	Early scars; adjunct
Imiquimod	Useful post-ear excision	Irritating	Small earlobe keloids
LADD	Deep drug penetration	Requires laser	Resistant lesions
Other agents	Option for refractory	Limited evidence	Specialist centers

Table 3: Summary of keloid treatment options.

Discussion

The Difference Between Keloid and Hypertrophic Scars

Both keloids and Hypertrophic Scars (HTS) have similarities, whether from their abnormal responses to wound healing to their common causes, such as cutaneous injury or irritation from trauma, tattoos, piercings, surgery, burns, insect bites, acne, abscess or vaccination. Crucially, superficial injuries that do not reach the reticular dermis do not cause keloidal or hypertrophic scarring. At the beginning of the injury, HTS and keloids are similar in scarring, but with healing, differentiation begins to occur, which is why misdiagnosis between HTC and keloids.

Differentiation

The primary clinical distinction between keloids and HTS is growth pattern. Keloid scars tend to extend from their original boundaries in their direction of healing, with a distortion of the lesion and a pseudo-tumoral nature by invading the surrounding healthy skin. Whereas HTS remain within the boundaries of the original wound and yet regress over months and years spontaneously after the initial injury [39].

Another difference is the pathology between HTN and keloids; keloids are characterized by the presence of thick, haphazardly arranged collagen fibers called keloidal or hyalinized collagen bundles, which are fewer in HTS. HTS collagen bundles are typically smaller, flatter, less demarcated and arranged in a wavy or parallel pattern. However, many lesions exhibit intermediate characteristics, making clinical differentiation challenging. Some experts propose that keloids and HTS are fundamentally the same fibroproliferative skin disorder and differ only in the intensity, frequency and duration of the chronic inflammation of the reticular dermis. HTS are typically limited in their wound margins, respond normally to growth hormones and may contain mainly Type III collagen arranged parallel to the epidermal surface (Table 4) [40].

Feature	Keloids	Hypertrophic Scars
Growth	Extend beyond wound borders	Remain within wound margins
Regression	Rarely regress	Often regress over time
Collagen	Thick, hyalinized, disorganized bundles	Parallel, wavy collagen bundles
Inflammatory state	Chronic and persistent	Controlled and self-limited
Recurrence after treatment	Very high (up to 100% post excision)	Lower recurrence

Table 4: Clinical and histopathological differences between keloids and hypertrophic scars.

Future Directions of Steroid Applications in Keloids

The future of keloid therapeutic management is progressively evolving from empirical treatments toward targeted molecular therapies and advanced biological agents [30]. A deeper understanding of the molecular pathways responsible for pathological scarring has identified Transforming Growth Factor-beta (TGF-β) as a primary target, with future treatments potentially utilising recombinant TGF-β3, interleukin-10 and imatinib mesylate to inhibit fibrogenic signalling [6]. Innovative research is exploring the use of RNA interference (RNAi) and microRNA-based therapies, such as VEGF siRNA, to suppress angiogenesis and silence specific transcription factors like Runx2 that drive excessive collagen deposition [41]. Furthermore, mechanistic checkpoints observed in tumor growth, for example, fibroblast activation protein alpha (FAP-α) and CTHRC1, are being investigated as novel targets to halt the aggressive, invasive nature of keloid tissue [5]. Technological advancements in drug delivery systems are also central to upcoming management strategies, shifting toward non-invasive transdermal methods to replace painful injections. These include dissolving microneedle patches, nanocarrier systems like transfersomes and Laser-Assisted Drug Delivery (LADD), which creates microscopic channels to ensure a uniform distribution of medications throughout the dense scar matrix [21]. Additionally, cell-based therapies involving Mesenchymal Stem Cells (MSCs), Adipose-Derived Stem Cells (ADSCs) and autologous fat grafting are undergoing large-scale trials to determine their efficacy in modulating chronic inflammation and improving tissue pliability [41].

Conclusion

The treatment of pathological keloid scars remains fundamentally challenged due to the lack of standardized therapy and the limited understanding of the primary underlying mechanism. This lack of understanding necessitates further investigation of long-term, large-scale RCTs and the development of treatment protocols to define true outcomes and recurrence rates. Additionally, a major current limitation is that ICAs are invasive and painful, severely impacting patient compliance, alongside the risk of local adverse effects like skin atrophy and dyspigmentation. Future directions are focused heavily on creating new formulations, combination treatments and drug delivery systems to overcome these drawbacks, notably including the use of Laser-Assisted Drug Delivery (LADD), where fractional lasers create micro-channels to enhance the deep penetration of topical steroids, as well as developing advanced transdermal systems like microneedle arrays and nanocarriers for controlled and sustained delivery. Finally, leveraging advances in molecular understanding, research is progressing toward targeted molecular therapies that intervene in fibrotic pathways (e.g., TGF-β) and testing emerging options by pushing the field toward more effective, personalized medicine.

Conflict of Interest

Authors declare no conflict of interest.

Authors' Contributions

All authors have contributed equally to this work and have reviewed and approved the final manuscript for publication.

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Consent for Publication

Informed consent for publication was obtained from the patient involved in this case report, as documented in the manuscript.

Informed Consent Statement

Informed consent was obtained from the participant involved in this study.

Ethical Statement

Not applicable

Data Availability Statement

Not applicable

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