Research Article

Surgery and Clinical Practice

Efficacy of Cicatricort® in the Prevention of Keloids in Postoperative Plastic Surgery: A Retrospective Comparative Study (2023–2025)

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Received: 14 May 2025; **Accepted:** 19 Jun 2025; **Published:** 29 Jun 2025

Citation: Policarpo de Souza MA, Pontel MA. Efficacy of Cicatricort® in the Prevention of Keloids in Postoperative Plastic Surgery: A Retrospective Comparative Study (2023–2025). Surg Clin Prac. 2025; 2(1): 1-6.

ABSTRACT

***Introduction**: Keloid scars result from an exaggerated wound healing response characterized by excessive fibroblast proliferation and collagen deposition. Their formation is linked to persistent inflammation, dysregulated activation of growth factors such as TGF-β1, and imbalance between extracellular matrix synthesis and degradation. Due to the lack of effective preventive measures, this study evaluated the efficacy of Cicatricort®—a topical cream composed of acetylsalicylic acid, allantoin, vitamin E, and Sedum rosea root extract—in preventing keloid formation in patients undergoing plastic surgeries, including otoplasty, macrotia correction, blepharoplasty, rhytidoplasty, mammoplasty, abdominoplasty, and gynecomastia surgery.*

***Methods**: A retrospective analysis was conducted with 13,728 patients undergoing plastic surgeries. They were divided into two groups: a control group (n = 6,864) using corticosteroid-based scar creams between March 2023 and April 2024, and an experimental group (n = 6,864) using Cicatricort® for 90 days postoperatively from May 2024 to April 2025. Keloid incidence was assessed with a minimum follow-up of 24 months.*

***Results**: In the control group, 1.06% of patients developed keloids according to clinical records. In the Cicatricort® group, no keloid formation was observed. These findings suggest a strong preventive effect of the formulation, inhibiting fibrotic and chronic inflammatory cellular processes.*

***Conclusion**: Cicatricort® demonstrated significant efficacy in preventing keloid scars in plastic surgery patients by targeting key pathophysiological mechanisms of pathological scarring.*

Keywords

Keloid scar, Prevention, Surgery, Acetylsalicylic acid.

Introduction

Tissue healing is a dynamic and tightly regulated process involving sequential phases of inflammation, cellular proliferation, and extracellular matrix remodeling. Under physiological conditions, this results in the formation of a functional and inconspicuous scar. However, in certain individuals, the healing response is exaggerated, leading to hypertrophic or keloid scars. The latter are marked by uncontrolled scar tissue growth beyond the original wound margins, frequently associated with pain, itching, and significant aesthetic and functional impairment [1,2] (Figures 1 and 2).

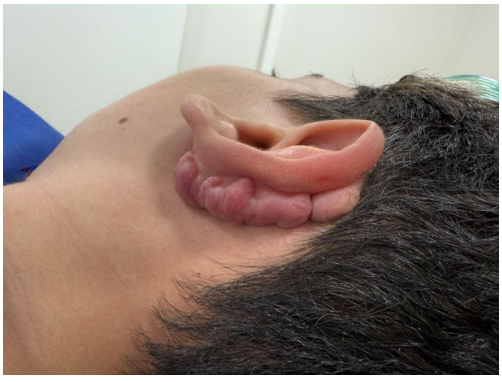

Figure 1: 11 months since the appearance of keloids after otoplasty.

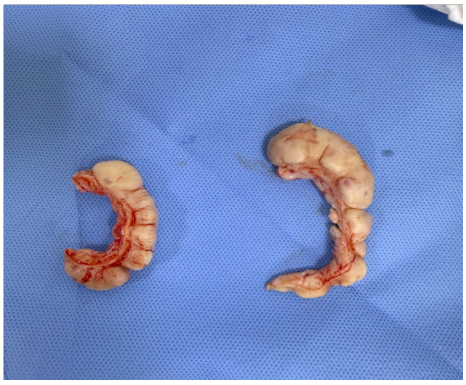

Figure 2: Surgical excision of a keloid scar.

From a histopathological and molecular perspective, keloids result from the hyperproliferation of dermal fibroblasts, with increased synthesis of type I and III collagen, overexpression of growth factors such as TGF-β1, and disorganized accumulation of extracellular matrix, all associated with persistent chronic inflammation and dysregulation of pro-fibrotic cytokines [3-5]. In addition, evidence indicates that genetic factors, younger age, darker skin phototype, and local tension at the surgical wound site are among the main risk determinants [6].

Despite advances in the treatment of pathological scars, preventing keloid formation remains a clinical challenge. Currently, there is no standardized and broadly effective topical pharmacological protocol for prophylaxis in predisposed patients. In this context, formulations with anti-inflammatory, antioxidant, and regenerative properties have gained prominence as promising therapeutic strategies.

Cicatricort® is a topical cream developed using a multifactorial approach, containing acetylsalicylic acid, allantoin, vitamin E, and Sedum rosea root extract. Each component has pharmacological properties targeted at key processes involved in pathological scarring, such as inhibition of TGF-β1, modulation of local inflammation, and reduction of tissue oxidative stress.

This study aims to evaluate the efficacy of Cicatricort® in preventing keloids in patients undergoing plastic surgeries, using a retrospective observational study design with a comparison to a control group.

Incidence of Keloid Scars in Plastic Surgery

The incidence of keloid scars following plastic surgery varies significantly depending on genetic factors, anatomical location of the incision, type of procedure, and complications such as infection or mechanical tension. Studies estimate that 5% to 15% of the general population develop some type of abnormal scar post-surgery, with keloids being less common than hypertrophic scars.

In genetically predisposed populations, such as individuals of African, Asian, and Hispanic descent, the incidence may exceed 40–45%, especially with a positive family history.

High-risk anatomical sites for keloid formation include:

- Earlobe
- Sternum (anterior thoracic region)
- Shoulders
- Back
- Incisions in high-tension or postoperatively infected areas

Additional Risk Factors for Keloid Formation

Several individual factors significantly increase the risk of keloid scar development, including:

- **Genetic predisposition:** A family history of keloids is strongly associated with higher susceptibility, indicating a hereditary influence [1].
- **Young age:** Keloid formation is more prevalent between ages 10 and 30, a period of heightened fibroblastic and immune activity [2].
- **Darker skin phototypes:** Patients with Fitzpatrick phototypes IV to VI (more pigmented skin) are at greater risk, especially those of African and Asian descent [3].
- **Local tension and wound infection:** Incisions under mechanical stress, repeated trauma, or surgical site infection hinder normal healing and favor fibrotic responses.
- **Altered immune reactivity:** Exaggerated or persistent inflammatory responses and elevated pro-fibrotic cytokines such as TGF-β1 and IL-6 contribute to abnormal scar tissue formation [4].

Early recognition of these risk factors is essential for identifying patients with high keloid formation potential and guiding prophylactic measures such as the use of topical anti-inflammatory and anti-fibrotic formulations.

Given these incidence rates and the lack of a standardized pharmacological prophylaxis, Cicatricort® emerges as a promising preventive solution, particularly in the following scenarios:

- Patients with risk factors (young age, darker or Asian skin, positive family history)
- Surgeries involving predisposed anatomical areas (ears, chest, scapular region)
- Elective procedures where prevention can be implemented from the early postoperative phase

The efficacy demonstrated in the study group—showing a 0% keloid formation rate among
6,864 patients—significantly exceeds the expected incidence, reinforcing the value of Cicatricort® as a candidate for inclusion in preventive care protocols.

Theoretical Review: Cellular and Physiological Aspects of Keloid Scar Formation

Tissue repair is a complex process, typically categorized into three sequential phases: inflammatory, proliferative, and remodeling. Under physiological conditions, there is a balance between extracellular matrix synthesis and degradation, resulting in a functional and minimally visible scar. However, in keloid scars,

this process becomes significantly dysregulated—particularly during the proliferative and remodeling phases—leading to the formation of exuberant scar tissue that extends beyond the original wound boundaries and may continue to grow indefinitely [1,2].

At the cellular level, the main alteration observed is the hyperproliferation of dermal fibroblasts, which exhibit resistance to apoptosis and are responsible for the excessive production of type I and III collagen, fibronectin, and other matrix proteins. This hyperactivity is directly linked to the overexpression of TGF-β1 (transforming growth factor beta 1) receptors—a potent fibrogenic mediator involved in pro-scar signaling pathways [3,4]. Additionally, there is an imbalance between matrix metalloproteinases (MMPs), which degrade collagen, and their natural inhibitors (TIMPs), promoting the disorganized accumulation of extracellular matrix components [5].

Another key factor is the persistent presence of low-grade inflammation, driven by cytokines such as IL-1β, IL-6, and TNF-α, along with the maintenance of an M2 macrophage profile, which is known for its pro-fibrotic characteristics [6,7]. This prolonged inflammatory state, combined with immature angiogenesis, contributes to a pathological microenvironment and impedes the orderly resolution of the healing process [8].

Given this scenario, effective prophylaxis for keloid formation must include early inflammation control, modulation of fibroblast activity, and the maintenance of extracellular matrix homeostasis. In this context, topical formulations like Cicatricort®—which contains acetylsalicylic acid, allantoin, vitamin E, and Sedum rosea root extract—present meaningful therapeutic potential. Acetylsalicylic acid inhibits the prostaglandin inflammatory cascade and reduces TGF-β1 expression [9,13]; allantoin promotes epithelial regeneration and provides a soothing effect [14]; vitamin E functions as an antioxidant, protecting tissues from oxidative stress [15,16]; and Sedum rosea extract, known for its adaptogenic and anti-inflammatory properties, supports cellular homeostasis. Together, these components act synergistically to prevent the fibrogenic disturbances that result in keloid formation. (See Figure 3).

Materials and Methods

This is a retrospective, observational study based on clinical data from 13,728 patients who underwent plastic surgeries at our facility. The surgeries included otoplasty, surgical correction of macrotia, blepharoplasty, rhytidoplasty, mammoplasty, abdominoplasty, and gynecomastia. The study period was 24 months, and patients were divided into two groups:

- Control Group (n = 6,864): Patients who used conventional corticosteroid-based scar creams, such as desonide 0.5 mg/g, applied twice daily for 90 days.
- Cicatricort® Group (n = 6,864): Patients who used Cicatricort® cream twice daily for 90 days.

Inclusion criteria: patients aged between 7 and 55 years with no prior history of keloids in other body areas. Exclusion criteria included autoimmune diseases, prior corticosteroid use, or failure to follow up for at least 12 months. Keloid presence was clinically determined by a specialized medical team and recorded in standardized medical records. All data were anonymized to ensure confidentiality.

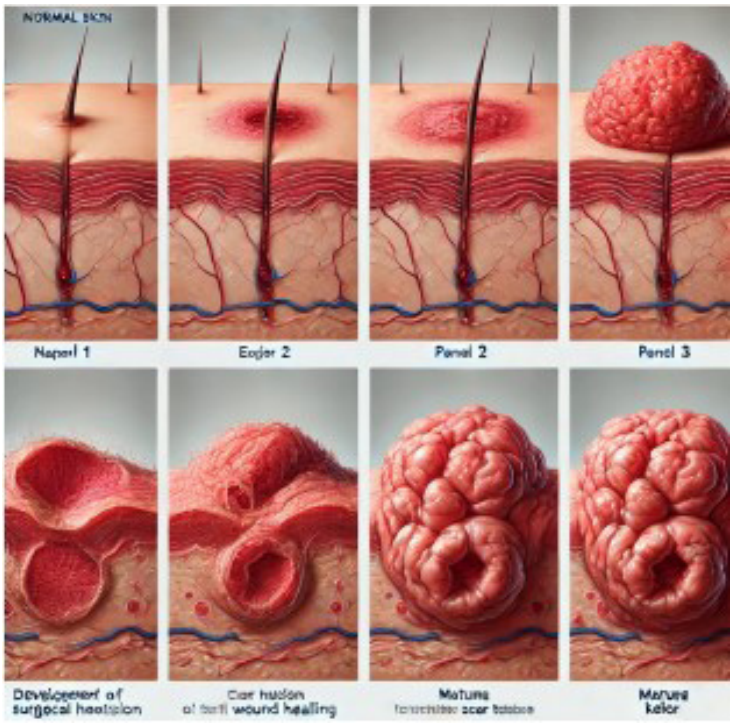

Figure 3: Cell formation of a keloid scar.

Results

Among the 6,864 patients in the control group, 1.06% developed keloids and required treatment between March 2023 and April 2024. The average time to keloid appearance was approximately 11.2 months (±2.4), ranging from 6 to 15 months.

In contrast, none of the 6,864 patients in the Cicatricort® group developed keloids, even those with identified risk factors such as darker skin tones and positive family history, during the period from May 2024 to April 2025.

Discussion

Several commercial products exist for the prevention of hypertrophic scars and keloids. A brief comparative analysis is provided below, highlighting Cicatricort® for its multifunctional formulation:

Commercial Products for Keloid Prevention

1. Contractubex® (Merz Pharmaceuticals)

- **Main ingredients:** Allium cepa (onion) extract, heparin, allantoin
- **Mechanism of action:** Antifibrotic, mild anti-inflammatory, and keratolytic effects
- **Clinical evidence:** Studies show modest efficacy in scar prevention and remodeling when used early [1].
- **Limitations:** Requires prolonged use (≥3 months); less effective on mature scars

2. Kelo-Cote® / Kelo-Cote Advanced Formula Gel (Alliance Pharma)

- **Main ingredients:** Silicone (polysiloxanes and silicon dioxide)
- **Mechanism of action:** Forms a protective film that hydrates and regulates local pressure and oxygenation
- **Clinical evidence:** Considered the gold standard in many treatment protocols; improves scar texture and pigmentation [2].
- **Limitations:** Lacks direct anti-inflammatory or antioxidant action; less effective on inflamed skin

3. Dermatix® (Meda Pharma)

- **Main ingredients:** Silicone gel and tocopherol (vitamin E)
- **Mechanism of action:** Silicone forms an occlusive barrier; vitamin E provides mild antioxidant action
- **Clinical evidence:** Reduces itching and provides gradual cosmetic improvement; more effective when used in the early stages [3].
- **Limitations:** Poor dermal penetration; inferior efficacy in older scars

4. Skinderma Pro® (Brazil)

- **Main ingredients:** Centella asiatica, onion extract, allantoin, vitamin E
- **Mechanism of action:** Healing and soothing agent with regenerative action
- **Limitations:** Limited clinical data; effects are more cosmetic than antifibrotic

5. Kelocote Spray® (for large areas or post-burn scars)

- Main ingredients: Silicone spray
- **Mechanism of action:** Same as the gel formulation, but with easier application for extensive areas
- **Indication:** Post-burn treatment, large surgical scars
- **Limitations:** Lacks direct anti-inflammatory action

6. Cicatricure® Gel (Genomma Lab)

- **Main ingredients:** Botanical extracts (onion extract, Centella asiatica, chamomile, aloe vera), allantoin, panthenol, beta-glucans
- **Mechanism of action:** Deep skin hydration, mild stimulation of epithelial regeneration, and soothing effect
- **Clinical evidence:** Promotes epithelialization and subjective improvement in scar appearance. No robust evidence for antifibrotic effects or modulation of TGF-β1 [1].
- **Limitations:** Classified as a cosmetic product with mainly moisturizing and anti-irritant properties; no proven direct effects on keloids or hypertrophic scars.

Visual Comparison of Mechanisms of Action of Products for Keloid Prevention

Product	Anti-inflammatory	Antioxidant	Antifibrotic	Epithelial Stimulator	Physical Barrier (Silicone)
Cicatricort®	✅	✅	✅	✅	❌
Kelo-Cote®	❌	❌	❌	❌	✅
Contractubex®	✅	❌	✅	❌	❌
Dermatix®	❌	✅	❌	✅	✅
Skinderma Pro®	✅	✅	❌	✅	❌
Cicatricure® Gel	✅	✅	❌	✅	❌

Table 1: Updated Comparison Table: Keloid Prevention Products.

Distinct Advantages of Cicatricort®

- Acts on multiple cellular targets involved in keloid formation, including inflammation, oxidative stress, and fibrosis
- Combines four synergistic active ingredients—an uncommon feature in a single product
- Does not rely on the formation of a physical barrier (like silicone), which enhances absorption and facilitates use on small or mobile areas (e.g., ears)

The results demonstrate significant efficacy of Cicatricort® in preventing keloids following plastic surgery procedures—and, by extension, in any postoperative or traumatic wound.

In particular, Cicatricort® has shown remarkable effectiveness in preventing keloids in otoplasty patients. Each component of the formulation contributes through distinct therapeutic mechanisms.

Conclusion

Based on the cellular and molecular scientific evidence supporting the key components of Cicatricort®, and focusing on how each ingredient contributes to the prevention of keloid formation—with detailed and up-to-date references—the following mechanisms are highlighted:

1. Acetylsalicylic Acid (ASA)

o Cellular Mechanism:

- ASA irreversibly inhibits cyclooxygenase enzymes (COX-1 and COX-2), thereby reducing prostaglandin production—especially PGE2, a pro-inflammatory mediator involved in fibroblast activation and migration.
- It also inhibits the expression of TGF-β1, one of the main factors driving uncontrolled fibroblast proliferation and excessive collagen deposition in keloids [9,13].

o Relevant Effects:

- Decreased fibroblast proliferation and type I/III collagen synthesis
- Reduced inflammatory response at the wound site
- Inhibition of disorganized scar tissue formation

2. Allantoin

Cellular Mechanism:

- Allantoin promotes the proliferation of normal keratinocytes and fibroblasts, but does not stimulate the hyperactive fibroblasts characteristic of keloids.
- It functions as an epithelial modulator, encouraging orderly cell regeneration and alleviating inflammation without inducing pathological fibrosis [14].

o Relevant Effects:

- Promotes controlled epithelialization
- Soothes skin irritation and reduces mild inflammation and pruritus
- Does not activate fibrogenic pathways such as TGF-β or PDGF

3. Vitamin E (Tocopherol)

Cellular Mechanism:

- A lipophilic antioxidant that neutralizes free radicals, protecting cell membranes during oxidative stress in tissue repair.
- Inhibits the activation of NF-κB, a central pathway responsible for inflammatory cytokine expression and pathological fibroblast hyperproliferation and angiogenesis in keloids [15,16].

o Relevant Effects:

- Reduces oxidative damage and local inflammation
- Limits fibroblast migration and overactivation
- Improves elasticity and overall quality of scar tissue

4. Sedum rosea Root Extract (Rhodiola rosea)

o Cellular Mechanism

- An adaptogenic plant rich in rosavin, salidroside, and tyrosol, which modulate cellular responses to oxidative and inflammatory stress.
- Decreases expression of TNF-α, IL-6, and IL-1β—key cytokines involved in persistent inflammation during pathological wound healing.
- Enhances mitochondrial activity in normal keratinocytes and fibroblasts, helping to stabilize the tissue microenvironment [17,18].

o Relevant Effects

- Anti-inflammatory and antioxidant effects complementary to vitamin E
- Promotes cellular vitality and homeostasis
- Prevents the persistent pro-fibrotic environment that favors keloid formation

Final Statement

We conclude that Cicatricort® cream has proven to be highly effective in preventing keloids in patients undergoing plastic surgery. The evidence suggests it has the potential to become a therapeutic standard in the postoperative management of surgeries and traumatic wounds, particularly in areas prone to pathological scarring.

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