



Rethinking Tissue Preservation in Skin Cancer Treatment: Surgical Margins versus Molecular Selectivity

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Abstract

Mohs micrographic surgery is widely regarded as the surgical gold standard for the treatment of non-melanoma skin cancers (NMSC). It offers high cure rates, meticulous margin control, and well-established oncologic reliability. However, the increasing availability of molecularly targeted therapies invites critical re-examination of whether surgical excision must always define “complete cancer removal.”

Curaderm, a topical therapy containing the solasodine rhamnoside glycoalkaloid complex BEC, presents a fundamentally different approach. Its mechanism - selective induction of apoptosis in cancer cells through binding to mutant rhamnose-binding receptors – differs markedly from the tissue-removal paradigm of Mohs surgery. Comparison of these two modalities highlights a conceptual disconnect between traditional surgical clearance and modern molecular-cellular selectivity.

This communication examines that disconnect and argues that the standards for skin cancer clearance should evolve to include non-surgical, targeted cellular therapies capable of achieving comparable oncologic efficacy with superior tissue preservation.

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Introduction

Mohs micrographic surgery has long been considered the reference standard for treating many forms of non-melanoma skin cancer owing to its high cure rates and precise histologic margin control. Although its tissue-sparing intent distinguishes it from conventional wide local excision, Mohs surgery remains an excisional technique that necessarily removes a volume of normal tissue.

Advances in molecular and cellular oncology now raise an important question: can cancer clearance be

achieved without physical removal of healthy tissue? Curaderm therapy provides an instructive example of this emerging paradigm [1].

Results and Discussion

Mohs Surgery: Precision Excision, but Still Excision

The strengths of Mohs surgery are well documented. Its stepwise removal of thin tissue layers with immediate histopathological examination allows accurate mapping of tumor extensions and maximizes the likelihood of complete excision while sparing more tissue than standard excision strategies.

Nevertheless, even with its “tissue-sparing” intent, Mohs surgery inevitably removes surrounding normal tissue. Resulting defects frequently require flap reconstruction or skin grafting, particularly in cosmetically sensitive regions such as the ear, nose, eyelids, and lips. Scarring, contour distortion, and donor-site morbidity are therefore intrinsic to the procedure.

Figure 1 demonstrates this process:

- preoperative tumor,
- post-Mohs surgical defect,
- full-thickness skin graft reconstruction, and
- long-term postoperative appearance demonstrating graft-related textural and pigment variation.

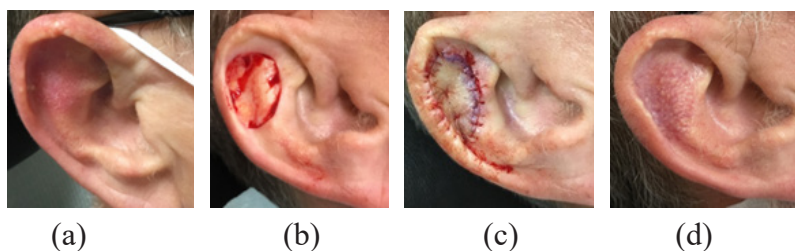


Figure 1: Mohs micrographic surgery requires tissue excision and reconstruction.

Representative images showing: (a) Preoperative basal cell carcinoma on the ear; (b) Surgical defect following Mohs excision; (c) Full-thickness skin graft reconstruction; and (d) Long-term postoperative appearance demonstrating graft-related contour and pigment variation. Images courtesy of Kathryn Potter, MD.

These outcomes are acceptable within surgical oncology but become increasingly conspicuous in an era emphasizing molecular precision and tissue preservation.

Curaderm: Molecular-Cellular Selectivity without Surgical Tissue Loss

Curaderm represents an alternative non-surgical paradigm. Its active component, the solasodine rhamnoside complex BEC, selectively targets cancer cells through binding to mutant rhamnose-binding receptors that are minimally expressed on normal keratinocytes. Following receptor-mediated endocytosis, downstream events include lysosomal destabilization, mitochondrial cytochrome-c release, Smac activation, and apoptosis. [2-9].

Importantly, this cascade does not occur in healthy skin cells. Histological studies demonstrate preservation of normal tissue architecture accompanied by acanthosis and regenerative epithelial activity. Clinically, this process is characterized by katabasis (cancer cells elimination) and anabasis (repopulation with normal cells) without the need for excision, sutures, or grafts.

Figure 2 demonstrates this progression in a basal cell carcinoma treated with Curaderm. Early lesion enlargement reflects predominantly active katabasis, while subsequent contraction corresponds to anabasis and tissue restoration. Five-year follow-up confirms durable clearance and scarless healing.

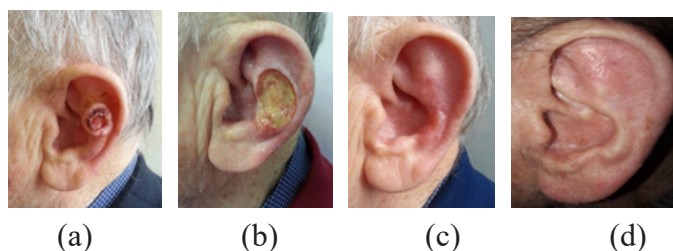


Figure 2: Curaderm therapy enables selective cancer-cell apoptosis and scarless healing.

(a) Baseline BCC. (b) Day 28 of treatment showing katabasis-related enlargement. (c) Day 60 where anabasis predominates and the lesion contracts. (d) Five-year follow-up demonstrating tissue preservation and absence of scarring.

The Analogy Between Mohs and Curaderm: Superficial but Misleading

Although Mohs surgery and Curaderm share the goal of complete cancer clearance, their similarity largely ends there. Mechanistically and clinically, they represent fundamentally different therapeutic strategies (Table 1).

Table 1: Comparison of Mohs Surgery and Curaderm Therapy

Feature	Mohs Surgery	Curaderm
Therapeutic Mechanism	Excision and Microscopic Margin Control	Molecular targeting of mutant receptors --> apoptosis
Impact on normal tissue	Removes tumour plus surrounding tissue	Spares normal cells
Reconstruction required	Common	None
Cosmetic outcomes	Variable, scars and graft distortion possible	Typically, scarless
Histological basis	Real-time margin assessment	Dynamic katabasis/anabasis
Cure rates	High	High

Equating the two therapies solely on the basis of “complete clearance” obscures these fundamental differences.

Clinical Implications

As dermatologic oncology moves toward personalized and precision-based care, therapies that maximise tissue preservation while maintaining oncologic efficacy are increasingly valued. This is particularly relevant in anatomical regions where even minimal tissue loss can result in disproportionate functional or cosmetic consequences.

Curaderm addresses several emerging and clinical priorities:

- Tissue Preservation, through selective cancer cell targeting
- Scarless Healing via natural regeneration
- Mechanistic Precision at the molecular-cellular level
- Long-Term Durability demonstrated by extended follow-up
- Accessibility through topical, non-surgical administration

These advantages do not diminish the importance of Mohs surgery, rather, they expand the therapeutic landscape, especially for early, localized basal and squamous cell carcinomas.

Redefining “Gold Standard” in the Molecular Era

Surgery is not being replaced but contextualized. As molecular-cellular therapies mature, the definition of "complete clearance" should extend beyond excision to include therapies capable of eliminating malignant cells while preserving normal tissue.

The disconnect between Mohs surgery and Curaderm reflects two distinct models of cancer treatment: one based on tissue removal, the other on cellular selectivity. Recognizing this distinction may better align clinical decision-making with contemporary oncological principles.

Conclusion

Mohs micrographic surgery and Curaderm both achieve cancer clearance, but through fundamentally different paradigms. Mohs relies on margin-based excision with attendant tissue loss and reconstruction. Curaderm harnesses molecular selectivity to induce apoptosis in cancer cells while preserving normal tissue and enabling scarless healing.

As precision oncology advances, tissue preservation, patient experience, and long-term cosmetic outcomes should be regarded as core components of treatment success. Integrating molecular-cellular therapies into appropriate clinical contexts may help realize the full potential of modern oncologic care.

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