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Enhanced Analysis and Simplification of Scientific Findings with Emphasis on Katabasis and Anabasis on Safe and Effective Skin Cancer Treatment Using Curaderm

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Abstract

Skin cancer accounts for more cases annually than all other cancers combined. Traditional skin cancer treatments often fail to distinguish between cancerous and healthy skin cells, resulting in less effective outcomes and poor cosmetic results. In contrast, Curaderm cream, which contains antineoplastic BEC glycoalkaloids, selectively targets skin cancer cells while sparing healthy ones. This leads to effective healing with excellent cosmetic results. Here, we show that by using data from a Phase III clinical trial, the healing process works through two mechanisms: **1) Katabasis**—eliminating cancer cells by apoptosis. **2) Anabasis**—replacing them with healthy cells. Both processes occur at the same time, but their rates change during Curaderm therapy. These mechanisms explain the clinical observations that Curaderm-treated lesions first undergo transiently increased lesion sizes of up to 50%, followed by decreasing sizes until histological complete healing is achieved. At the end of treatment, the surface of the lesions becomes level with the surrounding healthy skin, is scarless, and is indistinguishable from untreated skin. These mechanisms ensure the complete removal and healing of basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) lesions without leaving scars.

Keywords

Skin Cancer, Basal Cell Carcinoma, Squamous Cell Carcinoma, Curaderm, BEC, Mohs, Anabasis, Katabasis, Glycoalkaloids, Scarring, Superior Cosmetic Outcomes, Scarless, NMSC

1. Introduction

Skin cancer is the most common type of cancer, occurring when skin cells grow uncontrollably to form tumours. Alarmingly, it accounts for more cases annually than all other cancers combined. Despite medical advancements, effective treatments remain a challenge.

Treatments of nonmelanoma skin cancer (NMSC) may involve:

- Surgical removal consisting of excision, Mohs surgery or curettage and electrodesiccation
- Cryotherapy
- Radiation
- Photodynamic therapy
- Imiquimod
- 5-Fluorouracil

Mohs micrographic surgery is considered the gold standard for treating nonmelanoma skin cancers (NMSCs). While it is effective, it has drawbacks, such as scarring and its invasive nature.

In 1987, it was reported that plant-derived glycoalkaloids solasodine glycosides, including solamargine, solasonine, mono- and di-glycosides of solasodine, known as BEC, induced remarkable anticancer effects with high therapeutic indices in cell culture, animals, and humans.

BEC in Curaderm are amphiphilic molecules that possess both hydrophilic and hydrophobic properties. The hydrophilic moiety, essentially the plant sugar rhamnose, binds to a mutant protein on malignant cancer cells. There are two types of binding of BEC on cancer cells, which are related to receptor affinity and numbers of receptors per cell. Normal non-cancer cells do not contain the mutant protein receptors.

After binding and internalisation of BEC by receptor-mediated endocytosis into cancer cells, the hydrophobic alkaloidal moiety, solasodine, triggers the cancer cell to commit suicide by the process of apoptosis.

A cream formulation containing high concentrations of BEC was shown to be effective and safe in the treatment of the premalignant lesions' actinic keratosis and malignant skin cancers, BCC and SCC.

Optimizing the bioavailability of BEC with skin cancer cells at the concentration of BEC that is in the range of anticancer efficacy in cell culture and animals, resulted in Curaderm, a very safe and effective treatment for skin cancers.

Curaderm, a topical treatment containing BEC glycoalkaloids, offers a positive alternative to treat skin cancer. Research shows that Curaderm eliminates cancer cells (evidenced by an initial increase in lesion size) and achieves complete healing, without scarring. Its results are comparable to or better than surgery [1] [2].

Studies reveal a gradual regression of basal cell carcinoma (BCC) with Curaderm [1] [3]-[5]:

- 40% reduction at 4 weeks;
- 78% at 8 weeks;

- 90% at 12 weeks, and;
- Over 95% at 14 weeks.

In contrast, placebo treatments showed minimal improvement and high recurrence rates [6]-[10].

This study goes further by isolating the specific effects of the active ingredient in Curaderm (BEC glycoalkaloids) [11]-[19] and demonstrates that the healing process works by balancing two key mechanisms:

1) Katabasis—cancer cell elimination by apoptosis.

2) Anabasis—replacement with healthy cells.

The comparison of Mohs surgery (which leaves scars) with Curaderm therapy (which results in no scarring) highlights the superior cosmetic outcomes of Curaderm.

Curaderm is registered at the health authorities in Australia, European Decentralised and Mutual Recognition Health Authorities, Russia, and Vanuatu and is being processed in USA.

2. Materials and Methods

2.1. Study Design

This study was a multicentre, double-blind, randomized clinical trial that compared the effects of Curaderm and Placebo on basal cell carcinoma (BCC). Both treatments were applied under the same conditions to eliminate bias [6].

2.2. Formulations Used

- **Curaderm** contains BEC glycoalkaloids (small molecules from Solanum plants) [12], salicylic acid, and urea. These ingredients work together to promote the shedding of skin cells and help destroy cancer cells.
- **Placebo** is an identical cream, but without BEC glycoalkaloids [6] [12] [20]-[22].

Both creams were applied to the lesions every 12 hours, covered with a dressing, for 8 weeks. Both treatments caused local irritation and erosion of the lesions, so there was no bias from the patients or investigators.

2.3. Assignment to Treatment Groups

Curaderm and vehicle cream (placebo) were randomly assigned to patients at a ratio 2:1, respectively. Ninety-four patients were randomized (n = 62, Curaderm, n = 32, placebo). The investigator and patients were blinded to treatment. Curaderm and placebo creams were visually indistinguishable.

2.4. Patient Inclusion Criteria

Patients 18 years and over, with 1) histologically confirmed BCC of any type and 2) a lesion of at least 0.5 cm in diameter were included in the study.

2.5. Patient Exclusion Criteria

Excluded from the study were patients 1) who were pregnant or lactating; 2) with

known sensitivity or allergy to Curaderm; 3) being immunosuppressed; 4) who had used 5-FU or topical tretinoin within the preceding 2 months; 5) with a history of recurrent BCC after surgery, cryotherapy, or radiotherapy.

2.6. Ethical Conduct of the Study

The study was conducted in full conformance with the principles of the Declaration of Helsinki, 1964 (as amended in Tokyo (1975), Venice (1983), Hong Kong SAR (1989) and South Africa (1996). The study was conducted according to International Conference on Harmonisation (ICH) Good Clinical Practice (GCP) guidelines (1996).

2.7. Data Analysis

The data for this study came from a multicentre, double-blind, randomized, vehicle-controlled, parallel-group clinical trial [2] [6]. To assess the effect of the antineoplastic BEC component in Curaderm, the placebo results were subtracted from the results of Curaderm. This allowed determination of the net effect of the active antineoplastic ingredient (BEC) in Curaderm. The lesion sizes were measured with a calibrated Micrometre.

3. Results and Discussion

In the Phase III clinical trial, there were wide variations with large standard deviations of the BCC pretreatment lesion sizes for the Curaderm and Placebo groups (Table 1).

Table 1. Diameter (mm) and summary statistics of BCC lesion sizes by treatment group before commencement of treatment.

Summary Statistic	Curaderm Group	Placebo Group
	Lesion Size (mm)	
Mean	12.1	13.2
SD	5.13	7.78
Minimum	5	7
Median	10	11
Maximum	30	50
No. Patients	62	32

The treatment response differences between Curaderm and Placebo are shown in Table 2. Probability (p) values were calculated using the Two-Sample t-Test, comparing the treatment groups to their pretreatment values.

Table 2 and Figure 1 show that Curaderm, containing the active BEC glycoalkaloids, initially caused a significant increase in lesion size during the first 6 weeks of treatment. After 8 weeks, the lesion sizes returned to baseline. Continuing treat-

ment led to a further reduction in lesion size, eventually making the treated area blend in with the surrounding skin, completing the healing process.

Table 2. Differences in lesion sizes for Curaderm and Placebo groups at various time points, compared to pretreatment sizes.

Weeks of Treatment	Mean Change in Lesion Size (mm)	Standard Deviation (SD)	p-value
2	4.4	5.54	< 0.001
4	6.6	11.16	< 0.001
6	5.9	9.56	0.011
8	0.5	10.67	0.197
26	−5.3	8.52	< 0.001
52	−3.7	3.54	< 0.001

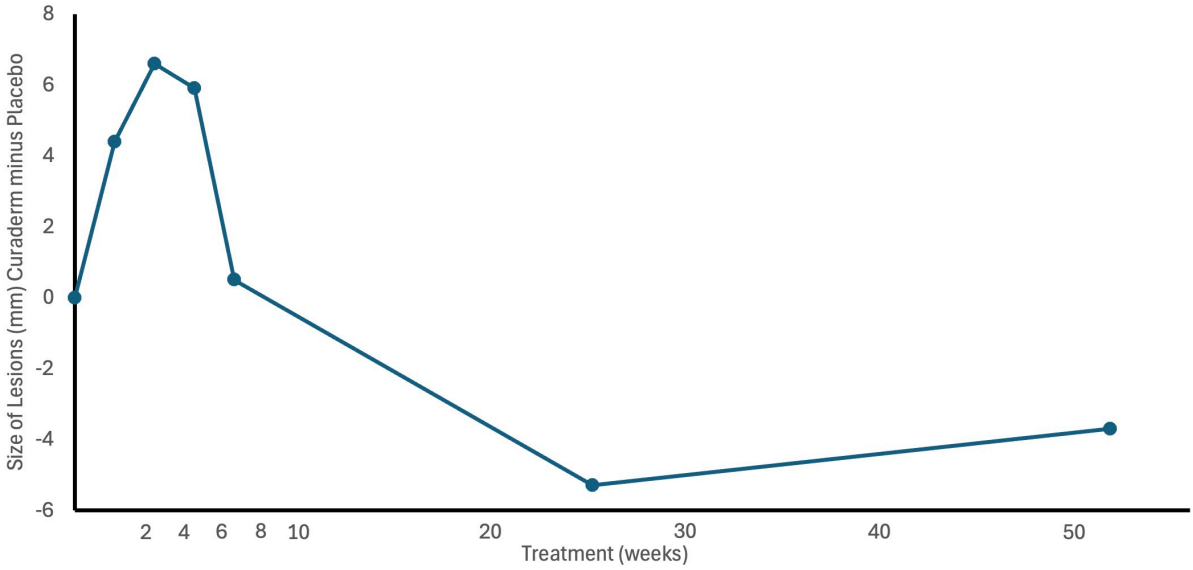


Figure 1. Progression of lesion healing of BEC glycoalkaloids under Curaderm therapy.

The statistically significant ($p < 0.001$ - 0.011) increase in lesion sizes lasted for up to 6 weeks of Curaderm treatment. By 8 weeks, the BCC lesions had shrunk back to sizes like their original pre-treatment state, with no significant statistical difference ($p = 0.197$). After 8 weeks of continuous treatment, the lesions continued to shrink and showed indentations, with their sizes becoming significantly ($p < 0.001$) smaller than the original pretreatment lesions. Eventually, the treated lesions were at the same surface level as the surrounding healthy skin.

Biopsies taken before, during, and after Curaderm treatment showed histopathological acanthosis, which is the thickening of the skin due to an increase in prickle cells in the stratum spinosum layer. This is a normal process in the development and maturation of tissue cells.

Physiologic hyperplasia was observed at lower rates up to 6 weeks of treat-

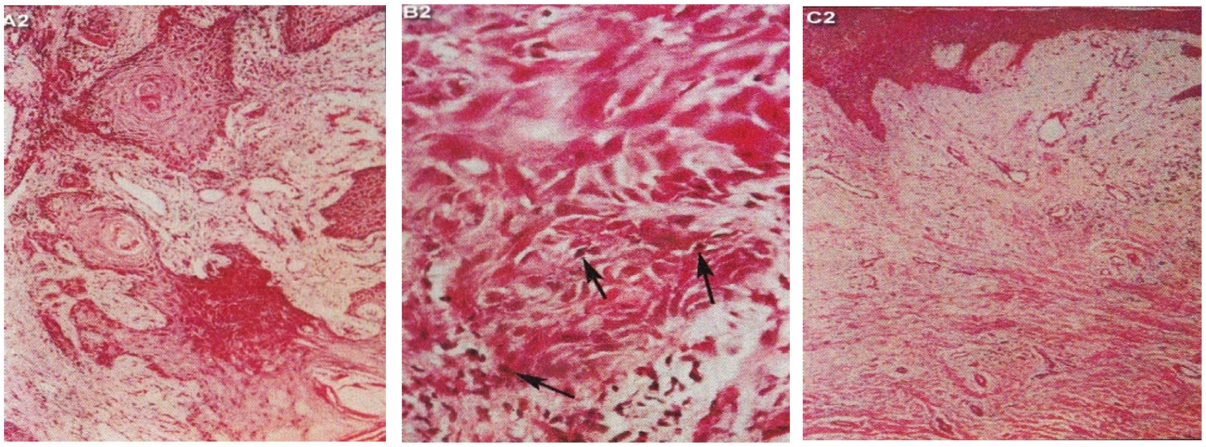
ment and increased after 8 weeks, continuing until clinical healing was observed.

These changes can be explained by two processes occurring during Curaderm therapy: **katabasis** (cancer cell death) and **anabasis** (replacement of cancer cells with normal cells).

Both processes happen at the same time, but their rates change. Katabasis was more prominent in the first 6 weeks of treatment, during which lesion sizes increased. By 8 weeks, lesion sizes stabilized, and both katabasis and anabasis were occurring at equal rates. After that, anabasis became the dominant process, leading to the complete healing of the lesions. At the end of treatment, the surface of the lesions was level with the surrounding healthy skin, indistinguishable from untreated skin.

Figure 2 illustrates histological progress by analyses of biopsies of a squamous cell carcinoma (SCC) skin cancer before treatment with Curaderm (a), during treatment with Curaderm (b), and after completion of treatment (c).

Before treatment, SCC is very pronounced. At 8 weeks of Curaderm treatment, katabasis is low (arrows indicate limited cancer cells dying by apoptosis) amid predominant anabasis of noncancer squamous cells which are derived from basal cells. After completion of Curaderm treatment complete healing is observed with no scar tissue.



(a) (b) (c)

Figure 2. Histological diagnosis of a squamous cell carcinoma (SCC) before treatment (a), during treatment with Curaderm (b), after completion of treatment (c). Arrows indicate cancer cells dying by apoptosis during Curaderm treatment.

Figure 3 to Figure 5 clinically illustrate the process of Curaderm treatment. At the beginning of treatment (Figure 3(a), Figure 4(a), Figure 5(a)), lesion sizes increase, reflecting a high rate of katabasis (Figures 3(b)-(d); Figures 4(b)-(d); Figures 5(b)-(d)). Over time, anabasis predominates, and the lesions decrease in size (Figures 3(e)-(g); Figure 4(e), Figure 4(f); Figure 5(e), Figure 5(f)), ultimately resulting in complete healing (Figure 3(h), Figure 4(g), Figure 5(f)).

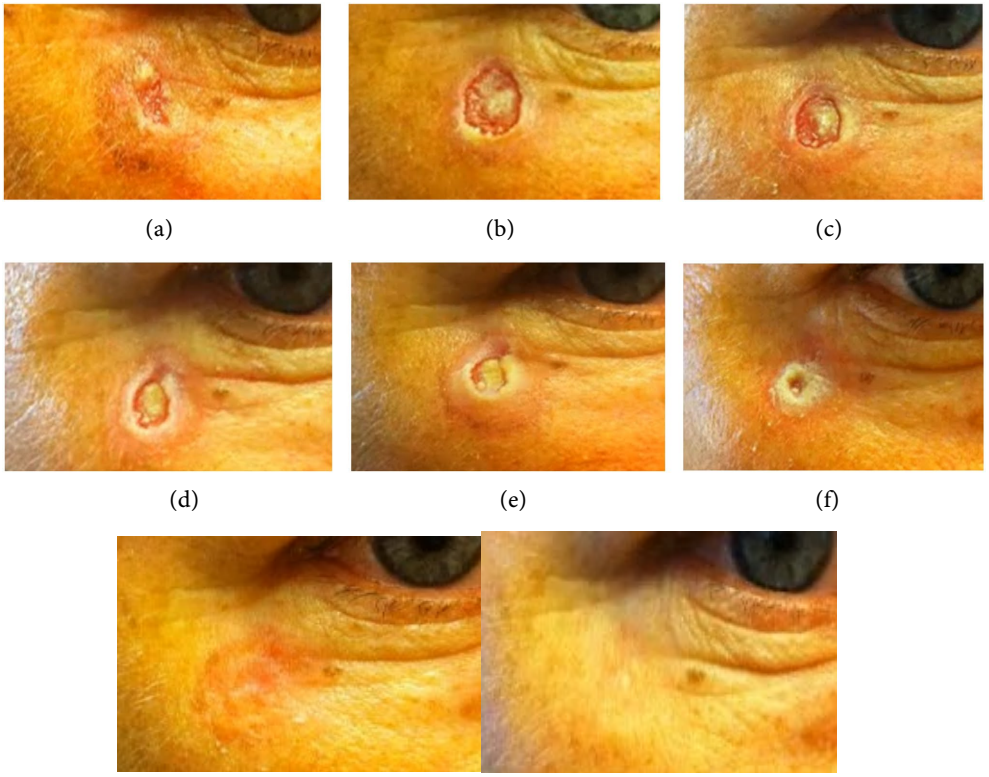


Figure 3. Treatment of a female patient during various time period stages: (a) before treatment; (b) after 3 weeks; (c) after 4 weeks; (d) after 5 weeks; (e) after 7 weeks; (f) after 9 weeks; (g) after 11 weeks; (h) after 12 weeks treatments with Curaderm.

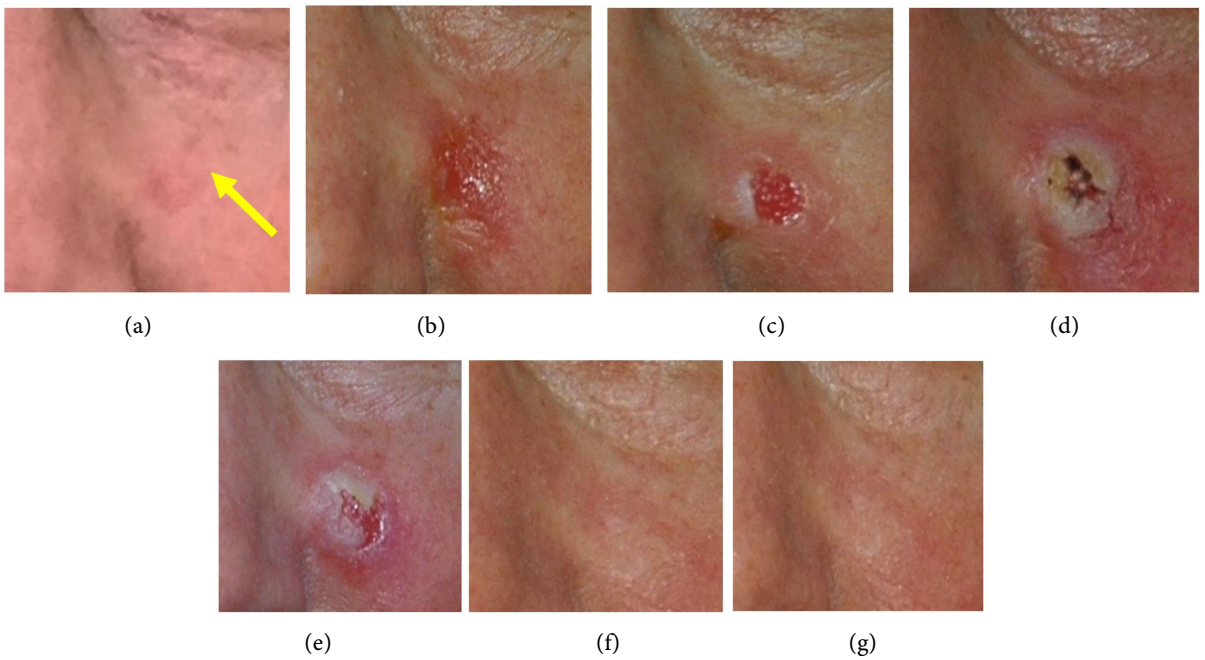
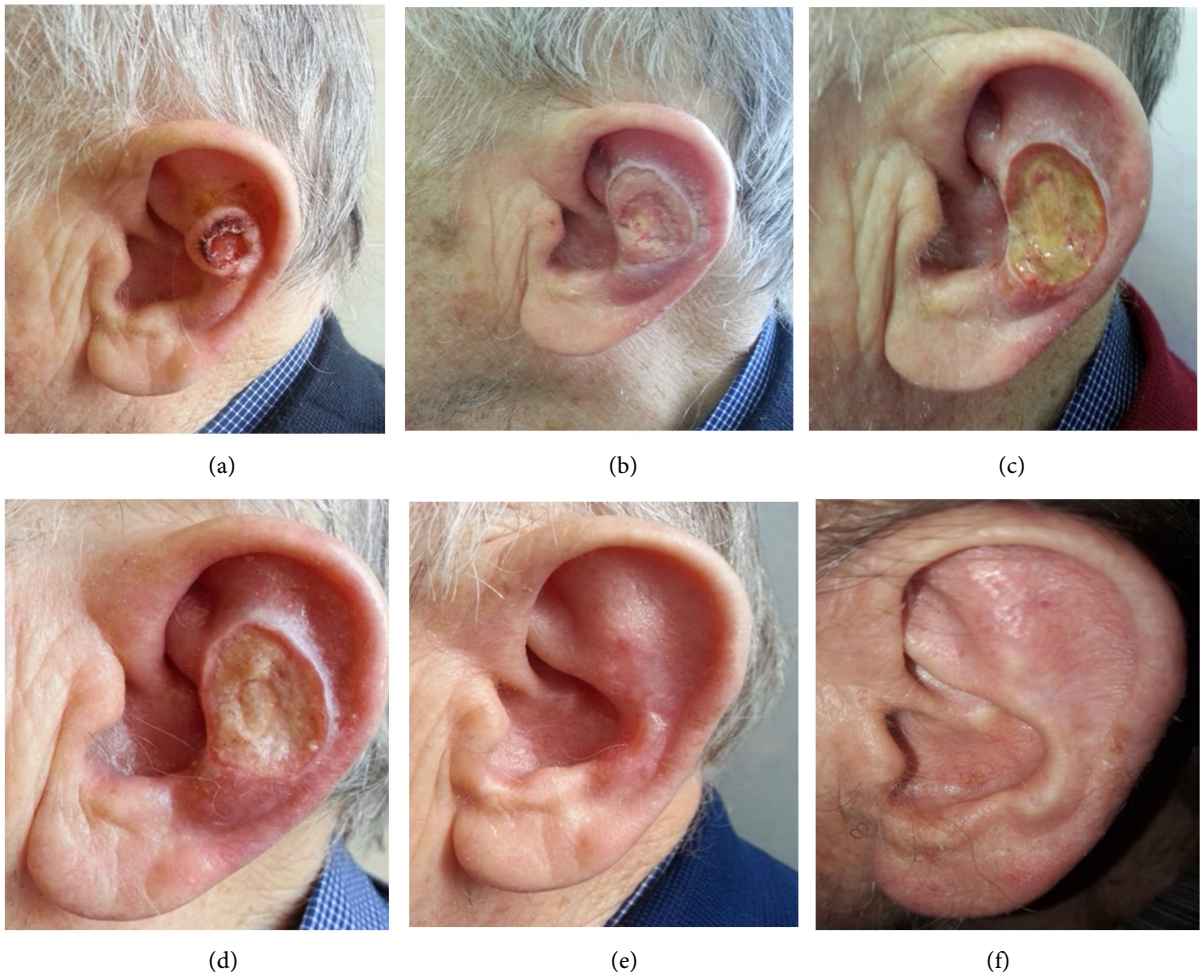


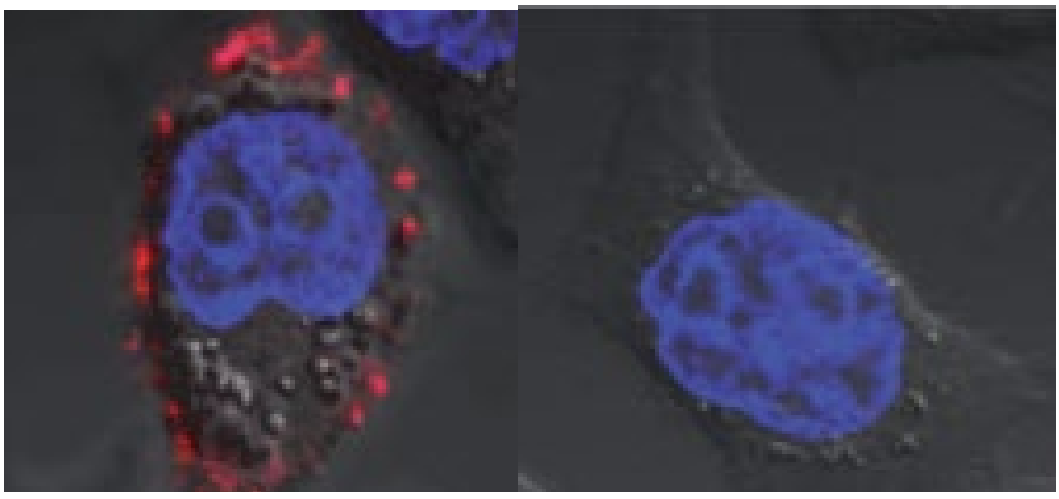
Figure 4. Treatment of a female patient during various time period stages: (a) before treatment, arrow indicates lesion site; (b) after 2 weeks; (c) after 3 weeks; (d) after 4 weeks; (e) after 5 weeks; (f) after 8 weeks; (g) 26 weeks after completion of treatments with Curaderm.



(a) (b) (c)

(d) (e) (f)

Figure 5. Treatment of a male patient during various time period stages: (a) before treatment; (b) after 17 days; (c) after 28 days; (d) after 60 days; (e) after 74 days; (f) after 5 years follow-up.



(a) Cancer Cell (b) Normal Cell

Figure 6. Identification of specific rhamnose binding protein (RBP) receptors on cancer cells by a fluorescence rhamnose probe (a). Normal non-cancer cells do not possess the RBP receptors (b). The red images indicate the binding of rhamnose conjugates (solamargine) to RBP receptors.

The antineoplastic action of BEC in Curaderm begins when BEC binds to mutant lectin carbohydrate-binding protein receptors on the surface of cancer cells [22]. The mutant BEC lectin receptors on cancer cells have been identified on skin cancer cells by fluorescence imaging analysis (**Figure 6(a)**), normal noncancer cells do not contain mutant BEC lectin receptors (**Figure 6(b)**) [4] [23]. This binding triggers receptor-mediated endocytosis, allowing BEC to enter the cancer cells. Once inside, BEC interacts with organelles like mitochondria and lysosomes, activating various apoptosis pathways that lead to cancer cell death [1] [4] [17].

Figure 7 shows microscopic morphological features caused by BEC leading to apoptosis, such as membrane blebbing, cell shrinkage, chromatin condensation, DNA fragmentation and apoptotic bodies [4]. In the body, the resulting cell debris from apoptosis (apoptotic bodies) is broken down by macrophage lysosomes. Unlike other treatments, Curaderm does not cause fibrous tissue formation or scarring, as seen in skin cancer lesions treated with Curaderm.

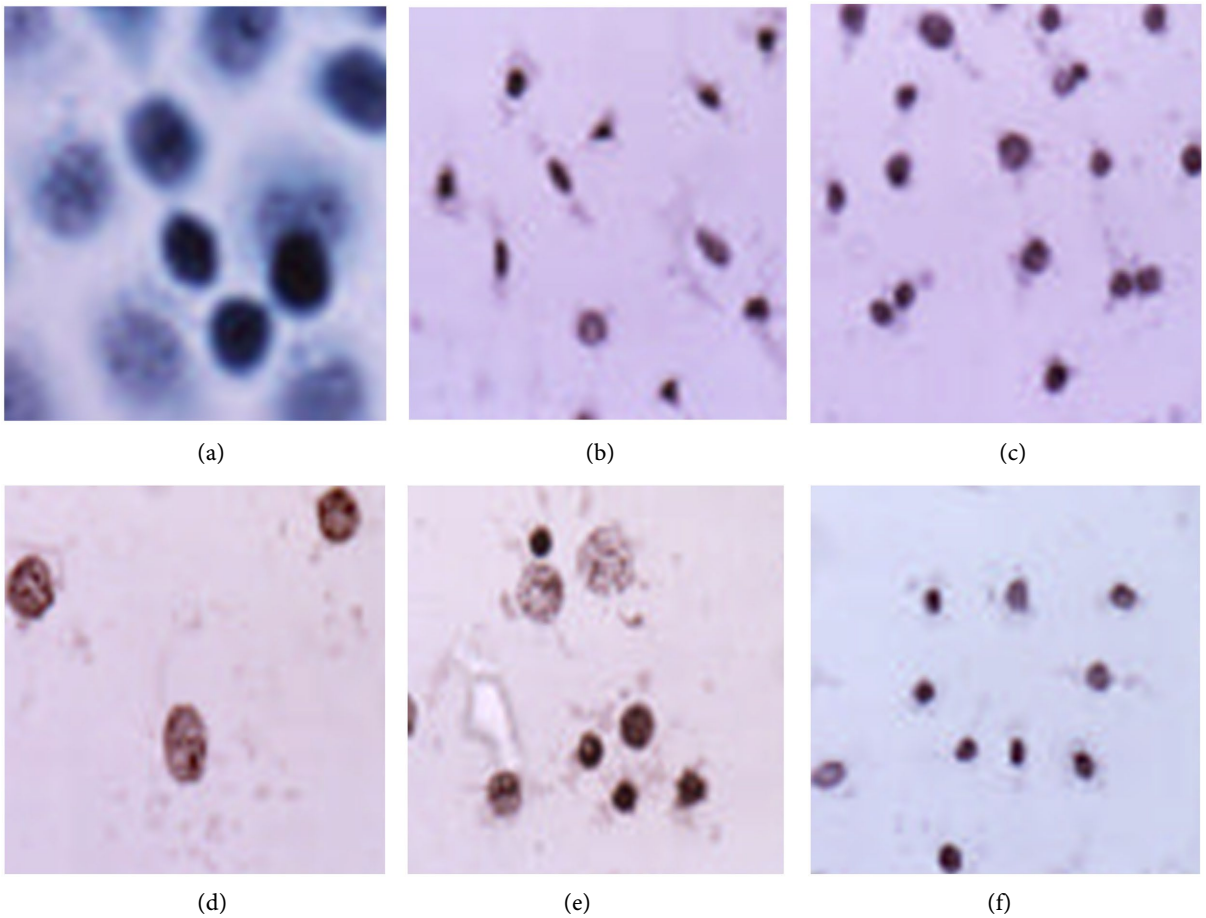
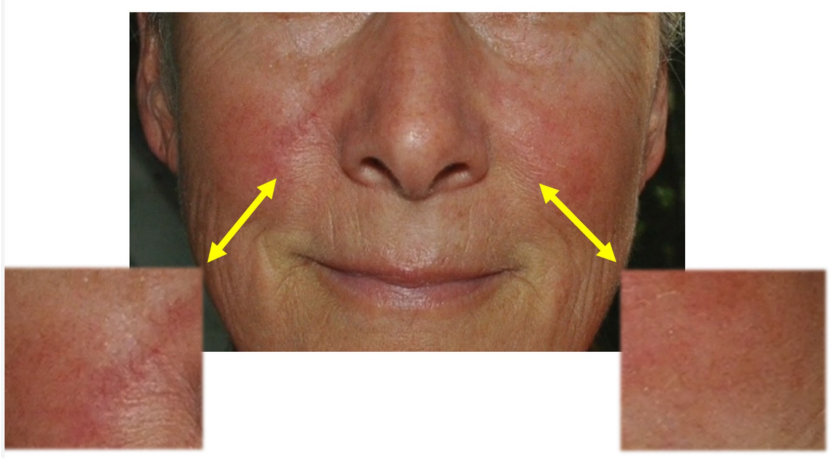


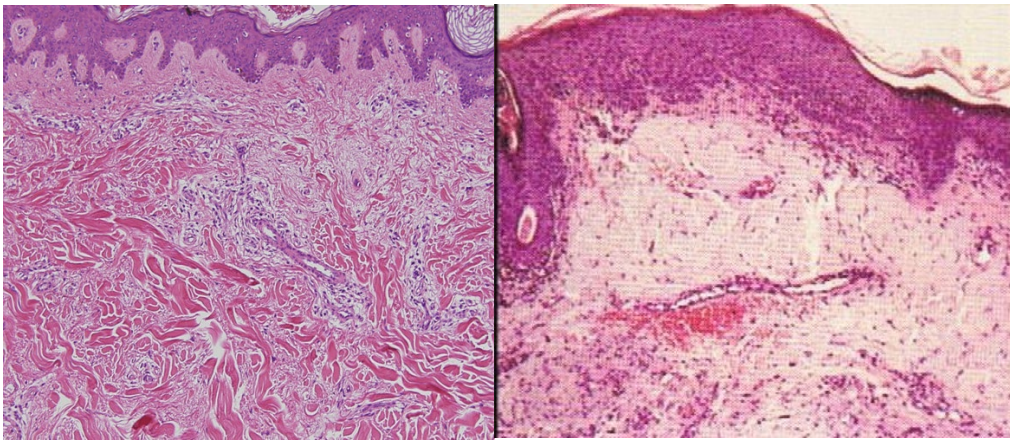
Figure 7. BEC in Curaderm rapidly and specifically kills cancer cells by the process of apoptosis resulting in scar-free healing: untreated cancer cells, the cells are all viable. (a) BEC causes the cytoplasm of the cancer cells to undergo dissolution, the nuclei contract and become dark staining (b) nuclei then enlarge; (c) the chromatin clumps; (d) and finally the nuclei disintegrate; (e) Only cellular debris (apoptotic bodies) are left after interaction of the cancer cells with BEC; (f) These events are characteristic of apoptosis, the process of programmed cell death. The cells were fixed and examined microscopically by the Papanicolaou method.

While Mohs surgery also effectively removes BCC with similar cure rates, it results in scarring. In contrast, Curaderm therapy does not cause scarring, as demonstrated in Figure 8 and Figure 9.



(a) (b)

Figure 8. Histologically diagnosed BCCs treated by Mohs surgery (a), and Curaderm therapy (b). This patient had two BCCs at similar positions, but facially opposite. Scar tissue is present after Mohs. No scar is present after Curaderm treatment, showing superior cosmetic outcomes.



(a) (b)

Figure 9. (a) illustrates histological diagnosis showing the severity of an established scar obtained by Mohs surgery after removal of a BCC, depicting horizontally oriented thicker and more densely packed hyalinised collagen bundles and loss of skin appendages. (b) shows a histological diagnosis of a biopsy after treatment of a BCC with Curaderm. Fine collagen and elastic fibres are present and are indistinguishable from an untreated skin biopsy.

The long-term safety and efficacy of Curaderm for the treatment of NMSCs [1]-[6] [11]-[19] [21] and actinic keratosis (AK) [24] [25] are well established. These achievements are due to the exceptional mode of action of the antineoplastic agent BEC present in Curaderm.

Adverse reactions experienced with Curaderm therapy range from burning sen-

sations, irritations, erosion, erythema, pruritus, swelling, postulation and ulceration. These transient effects are caused by the excipients in the formulation (as shown in placebo studies). These infrequent and minor side effects are usually caused by application of excessive amounts of Curaderm cream to the skin cancer lesions.

Histological success rates of over 95% are obtained if the Curaderm treatment procedure is followed, with no recurrences after 5 years. The duration of treatment period depends on the type, severity, size and location of the skin cancer. It is therefore highly recommended that the treatment with Curaderm is supervised by a health care professional to ensure complete success.

4. Conclusions

The interplay of katabasis and anabasis during Curaderm therapy underpins its effectiveness in treating NMSC. Unlike surgical methods, Curaderm facilitates natural skin regeneration without scarring, offering a patient-friendly alternative for NMSC management.

In the future, using artificial intelligence to predict treatment outcomes could further improve the effectiveness of this groundbreaking therapy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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