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Estimated time to complete this educational activity: 1 hour

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TARGET AUDIENCE

This educational activity has been developed for dermatologists and other health care professionals involved in the diagnosis and management of skin disorders for which immune response modifier (IRM) therapy might be considered.

EDUCATIONAL NEEDS

Skin disorders related to photodamage pose a major health issue that has implications involving morbidity, mortality, and health resource utilization. Various types of destructive therapies offer the potential for high cure rates for lesions such as actinic keratoses. However, the therapies are not 100% effective, do not eliminate the risk of recurrence, and can cause scarring that patients often find unacceptable. Because photodamage to the skin arises over a long period of time, many patients may not be good candidates for destructive therapies.

Topical and systemic therapies appeal to many patients seeking less invasive alternatives to conventional approaches to treatment. However, some of these therapies have yielded mixed results. Recently, IRM therapy has evolved as another option for management of actinic keratosis, superficial basal cell skin cancer, and possibly other lesions associated with photodamage.

In this supplement to SKIN & ALLERGY NEWS, dermatology specialists share clinical case experiences that illustrate the application of IRM therapy in varied skin diseases.

LEARNING OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- identify and describe therapeutic options for skin diseases caused by photodamage.
- describe the results of a clinical application of IRM therapy for a patient with actinic keratosis.
- discuss one clinician's experience with an IRM as therapy for recalcitrant plantar warts.



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DERMATOLOGISTS' CASE FILES

Management of Malignant and Premalignant Skin Lesions

Introduction

Stephen K. Tying, MBA, MD, PhD

Cumulative exposure to sunlight and artificial forms of ultraviolet light have a major influence on skin aging and the risk of cutaneous malignancy. Patients often do not recognize the hazards of excessive sun exposure until years after the initial insult to the skin. Overt cutaneous manifestations of sun damage typically do not appear until 15 to 20 years after the original damage.

The cumulative effects of years of excessive sunlight exposure come in a variety of forms, ie, rhytides, lentiginos and changes in pigmentation, actinic keratoses, telangiectasias, and ecchymoses. Malignant lesions of the skin associated with photodamage include basal cell carcinoma, squamous cell carcinoma, lentigo maligna, and melanoma. Lesions may be accompanied by skin atrophy, inelasticity, and wrinkling. The skin also may become redundant, dry, coarse, and yellowed.^{1,2}

Premalignant and Malignant Skin Lesions

Actinic keratoses present as flat or slightly raised scaling lesions that range between red and brown in color. The condition is a common



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presenting complaint in dermatology and primary care practices. In one study of dermatology clinical practice, approximately 14% of patients presenting to dermatology clinics had evidence of actinic keratosis.³ The rate of transformation to squamous cell carcinoma is approximately 1% per year. However, estimated rates of transformation vary considerably, ranging from 0.0075% at 1 year for a patient with a single lesion to 6% to 10% at 10 years for a patient with multiple actinic keratoses.⁴

Basal cell carcinoma accounts for about 80% of all skin cancers.⁵ A lesion typically appears as a nonhealing plaque or papule, characterized by a translucent border and telangiectasia. Most basal cell skin cancers have a good prognosis, as the 5-year survival rate is about 95%.⁶

Squamous cell carcinoma accounts for most of the remainder of nonmelanoma skin cancers. In contrast to basal cell skin cancer, squamous cell carcinoma can metastasize. The incidence of this type of skin cancer has increased by as much as 200% in the past 20 years.^{7,8} The presence of multiple actinic keratoses increases the risk of progressive squamous

cell carcinoma by 10- to 50-fold.⁹

Treatment Options

A variety of surgical and nonsurgical therapies have demonstrated varying degrees of efficacy of the management of premalignant and malignant skin lesions. The treatment options fall into broad categories of surgical and abrasive/destructive methods and nonsurgical interventions.

Surgery and Abrasive/Destructive Techniques

Mohs micro-surgery has proven to be highly effective in the treatment of basal cell carcinoma, high-risk squamous cell carcinoma, and selected cases of malignant melanoma. When applied appropriately, Mohs surgery offers cure rates ranging between 95% and 99%.¹⁰

Cryosurgery has resulted in cure rates approaching 99% for actinic keratoses.¹¹ Favorable results also have been reported from selected patients with basal cell and squamous cell carcinoma. Recurrence rates of 1% to 10% have been reported for low-risk basal cell carcinoma and 10% for squamous cell carcinoma.¹² The technique has a more favorable scar profile than some other surgical and abrasive/destructive techniques, but use of cryosurgery precludes acquisition of a biopsy specimen to assess the adequacy of treatment.

Continued on page 4

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Management of Patient With a Crusted Papule on the Nose

Robert T. Brodell, MD

The dermatologic toll of excessive exposure to sunlight continues to mount despite widespread recognition of the hazards and the availability of simple, inexpensive prophylaxis in the form of topical sunscreens, protective clothing, and limitations on sun exposure. More than 1 million new cases of skin cancer are diagnosed each year,¹ including 62,000 cases of melanoma, the fastest-growing category of cancer over the past 10 years.²

In addition to the threat of malignancy, photoaging robs the skin of its natural elasticity and tone, and a variety of neoplastic and hyperplastic skin lesions arise as a consequence of too much exposure to the sun. Actinic keratosis represents one of the most common dermatologic manifestations of photodamage. A precancerous lesion, actinic keratosis occurs in almost 20% of adults in the United States³ and accounts for 47 million visits to dermatologists' offices each year.⁴

Historically, cytotoxic therapies have formed the basis for treatment of actinic keratosis,⁵ providing cure rates as high as 99%.⁶ However, the number of effective nonsurgical treatment options has increased in recent years, and a growing proportion of patients affected by actinic and other sun-related skin lesions have begun to opt for these topical and systemic therapies. The anti-DNA thymidine analog 5-fluorouracil has the longest history in the topical treatment of actinic keratosis, but other agents that have shown promise include retinoids, cytokines, and topical preparations of nonsteroidal anti-inflammatory drugs.⁶

The immune response modifier



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(IRM) imiquimod has US Food and Drug Administration (FDA) approval for treatment of both actinic keratosis and superficial basal cell skin cancer. In two phase III vehicle-controlled clinical trials involving patients with actinic keratosis, imiquimod 5% topical cream reduced lesion counts by 87% compared to 14% in the control group, and the clearance rate in the two studies combined was 48% with imiquimod versus 7% with vehicle.⁷

Imiquimod also has FDA approval for treatment of superficial basal cell carcinoma. Two identical randomized clinical trials demonstrated clearance rates of about 75% after 12 weeks. In another study, imiquimod treatment led to a 12-week clearance rate of 90% and 1-year clearance rate of 84%.⁸

The IRM also has demonstrated efficacy for treatment of nodular basal cell carcinoma. In two phase II studies, daily application of imiquimod led to clearance in 71% of patients treated for 6 weeks and 76% of patients treated for 12 weeks.⁹ Another 12-week study showed complete clearance in 53% of patients and partial clearance in 37%.¹⁰

The following clinical case typifies the clinical experience of many physicians in treating basal cell carcinoma at the margins of biopsy specimens.

Clinical Case

A 60-year-old female patient presented with a 2-mm crusted, tan-colored papule on the left ala nasi. Physical examination of the lesion led to a differential diagnosis of hypertrophic-type actinic keratosis, ruling out basal cell carcinoma. A shave biopsy was performed, and the patient began

empiric treatment with polymyxin B/bacitracin ointment BID. Histopathology subsequently revealed an eroded nodular basal cell carcinoma that filled the dermis of the entire specimen and extended to all margins.

The patient returned 2 weeks later, and traditional treatment with Mohs surgery or excisional surgery was discussed (**Figure 1**).

Figure 1. Wound Following Shave Biopsy



Histopathology demonstrated that the basal cell carcinoma was not completely excised.

The discussion included the lack of metastatic risk with basal cell carcinoma, which allows for a consideration of medical treatment options. The patient was told that adjunctive treatment with imiquimod might improve the chance of avoiding additional surgery and might lessen scarring. In addition, exposure of the dermis as a result of the biopsy might allow better penetration of imiquimod and further improve the chances of effective treatment of the lesion. The patient elected to begin treatment with imiquimod BID.

Two weeks after initiation of imiquimod, slight inflammation of the lesion site was evident (**Figure 2** on page 3).

The patient continued imiquimod for 2 more weeks and then returned for another follow-up evaluation, which again revealed modest inflammation at the original site of the lesion (**Figure 3** on page 3).

In most cases, the wound would have been expected to heal by 6 weeks after biopsy. The

Continued on following page

A Middle-Aged Man With Psoriasis and Plantar Warts

Stephen K. Tyring, MBA, MD, PhD

Viral warts are a common presenting complaint in dermatology practice. Warts can cause substantial morbidity and adversely affect multiple aspects of a patient's quality of life. Immunocompromised patients unable to mount an adequate T-cell response to human papillomavirus (HPV) may develop multiple, extensive viral warts that are cosmetically unsightly, painful, and recalcitrant to conventional therapies.^{1,2}

Historically, most therapies for common warts have aimed to destroy the infected tissue. Destructive techniques include surgery, laser treatment, adhesives, and cryotherapy. In addition, traditional prescription medical therapies are designed to destroy the affected tissue. These therapies include podophyllin, podophyllotoxin, various acid compounds, 5-fluorouracil (5-FU), bleomycin, retinoids, contact sensitizers, glutaraldehyde,

formaldehyde, and cantharidin.³

More recently, immunomodulators have become available for treatment of warts arising from HPV infection. Immunomodulatory agents include interferon, imiquimod (which currently has approval for treatment of anogenital warts), and cidofovir. Clinical trials of these agents have demonstrated wart clearance rates of 75% to 80% and low recurrence rates.³

A comprehensive review of commonly used local therapies for nongenital warts (including cryotherapy) revealed evidence of modest to moderate efficacy at best.⁴ Nonprescription topical compounds containing salicylic acid had the best evidence: six placebo-controlled trials demonstrating a cure rate of 75% compared to 48% for control groups.

Imiquimod has demonstrated efficacy as monotherapy for common warts⁵ and as an adjunct to ablative or occlusive therapy.⁶ The agent also has proved effective for

treatment of palmar and plantar warts in immunocompromised patients.⁷ The following case reflects the clinical use of imiquimod as therapy for general warts.

Clinical Case

A 45-year-old male patient presented with extensive psoriasis and expressed interest in treatment with the tumor necrosis factor- α (TNF- α) inhibitor etanercept. Physical examination revealed extensive plantar warts, which the patient said had been treated previously with liquid nitrogen, salicylic acid pads, and various combination therapies.

The patient asked for immediate treatment of the psoriasis and said he would return at a later date to consider treatment for the warts. The patient received etanercept 100 mg/week and returned 3 months later with virtually no residual evidence of the psoriasis. The plantar warts remained largely unchanged (**Figure 1**).

Figure 1. Treatment-Resistant Warts



The patient had extensive plantar warts that had not responded to multiple prior therapies.

Figure 2. Lesion Clearance



After 60 days of imiquimod therapy, the warts had disappeared.

The patient was offered a trial of imiquimod. However, the pro-

Continued on page 4

Continued from previous page

likely explanation for the lack of healing is that imiquimod caused local inflammation after binding to toll-like receptors on remnants of the basal cell carcinoma.

Follow-up at 3 months after the initial biopsy revealed complete healing of the lesion with little scarring and no evidence of recurrence (**Figure 4**).

Three months later (6 months after the initial biopsy), another follow-up evaluation also revealed no evidence of recurrence of the basal cell carcinoma. The patient was instructed to notify her dermatologist if there was any sign of recurrence at this site in the future.

Discussion

Although anecdotal, the results of this case suggest that using imiquimod on a cosmetically sensitive area after a biopsy has revealed basal cell carcinoma with marginal involvement can lead to an excellent cosmetic outcome and avoidance of addi-

Figure 2. Two Weeks Post-Therapy



After 2 weeks of treatment with imiquimod, the lesion shows considerable inflammation induced by the drug.

Figure 3. Four Weeks Post-Therapy



Healing is occurring with decreased inflammation despite continued treatment with imiquimod at week 4.

Figure 4. Three Months Post-Therapy



The lesion had completely healed with no evidence of persistent basal cell carcinoma at the 90-day follow-up visit.

tional surgery, which might produce additional scarring.

This case does not permit the definitive conclusion that imiquimod eradicated the residual cancer cells and prevented recurrence. Basal cell carcinoma present on biopsy margins might be destroyed by forces of the body's natural immune system. To resolve the uncertainty would require a prospective, randomized clinical trial comprising three treatment arms: placebo, Mohs surgery, and imiquimod. Because of the ethical difficulties of with-

holding treatment for a malignant lesion, such a trial cannot be conducted. However, Mohs surgery and imiquimod could be compared in a two-arm clinical trial.

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Introduction

Continued from page 1

Excision and curettage are used with success in selected patients with basal cell and squamous cell cancers. Microdermabrasion is useful for surface lesions and lesions in the upper third of the dermis. The technique also has been used prophylactically to prevent progression of actinic lesions to basal cell or squamous cell cancer. Potential complications of microdermabrasion include bleeding, erythema, eczematization, altered pigmentation, hypertrophic healing, and enlarged pores.¹³

Superficial and deep chemical peels have a role in the treatment of several types of malignant precursors, eg, actinic keratoses as well as solar lentiginosities.¹⁴⁻¹⁷ Laser resurfacing also has fairly broad applicability to sun-related skin damage. The treatment can be used to treat elastotic changes, comedones, fine wrinkling, and extensive actinic keratoses.¹⁸

Radiation is useful for treating basal cell carcinoma in older patients and others who have difficulty sitting through surgery. Good candidates for radiation therapy include patients with multiple lesions and those with unresectable tumors or metastases.¹⁰

Photodynamic therapy combines a topical photosensitizer and

irradiation with a laser, light-emitting diodes, or some other light source. The treatment is used most often for patients with extensive solar keratoses, but some studies have demonstrated beneficial effects in superficial basal cell and squamous cell carcinoma.¹⁰

Topical and Systemic Therapies

The most widely used and evaluated topical therapy is 5-fluorouracil (5-FU). Topical 5-FU has demonstrated little efficacy in the treatment of squamous cell carcinoma in situ or superficial basal cell carcinoma. Electrochemotherapy has shown some potential in the treatment of malignant lesions but is neither accepted nor recommended for routine use.¹⁰

The biological response modifier imiquimod has demonstrated efficacy in a variety of premalignant and malignant lesions. In particular, five randomized controlled clinical trials have provided evidence of the safety and efficacy of imiquimod in the treatment of actinic keratosis.⁵ In reports of off-label use, the agent has shown promise for the treatment of lentigo maligna melanoma, melanoma in situ, cutaneous metastases of melanoma, and non-melanoma skin cancers.¹⁰

The vitamin A derivatives isotretinoin and tretinoin have shown potential for preventing

skin cancer.^{19,20} Other nondestructive therapies that have shown potential for treating skin lesions associated with excessive sun exposure include intralesional interferon- α -2a/2b²⁰ and certain topical nonsteroidal anti-inflammatory drugs such as diclofenac.²¹

Conclusion

Despite the well-recognized association between excessive exposure to sunlight and skin cancer, the incidence of malignant and premalignant lesions continues to increase. Multiple therapies have evolved for the treatment of malignant skin lesions and cancer precursors such as actinic keratoses. Dermatologists should become familiar with the various treatment options so as to offer the most appropriate therapy for an individual patient.

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Psoriasis and Plantar Warts

Continued from page 3

posed therapy introduced an element of uncertainty: Imiquimod's mechanism of action includes stimulation of TNF- α production. Whether coadministration of imiquimod and etanercept would adversely affect the therapeutic activity of either drug or of both drugs was unknown.

A decision was made to continue etanercept therapy and treat the warts by means of paring, freezing, and applying salicylic acid pads in the daytime, followed by imiquimod administered un-

der tape occlusion at night. The patient returned 1 month later, and the area affected by the warts had decreased markedly.

The patient elected to continue treatment with etanercept and imiquimod for another month. After a total of 60 days of imiquimod treatment, no warts remained visible (**Figure 2** on page 3).

At a 10-month follow-up visit, the patient's feet remained clear of warts. He also continued treatment with etanercept, and he remained free of psoriasis.

Discussion

This case is noteworthy for two

principal reasons. First, coadministration of etanercept and imiquimod resulted in no observed adverse effects, and both therapies proved highly effective in their treatment objectives. Second, the patient has remained free of plantar warts for almost a year after having dealt with the problem for much of his life. Though only a single case, the patient's experience suggests that imiquimod's antiviral effects are maintained long term.

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CME Post-Test and Evaluation

Instructions: For each question or incomplete statement, circle the most appropriate response. Four correct responses are required for credit.

1. Historically, what has been the most common approach to treatment of actinic keratoses?
 - a. Topical steroids
 - b. Destructive therapies
 - c. Chemotherapy
 - d. a & b
2. Which of the following best describes the current epidemiology of skin cancer?
 - a. Incidence has declined because of increased use of sunscreens and other protective measures.
 - b. Transformation from cancer precursor lesion to skin cancer has been slowed by effective new therapies.
 - c. >1 million new cases present annually.
 - d. Incidence of melanoma has increased more than that of any other cancer over the past 10 years.
 - e. c & d
3. What was the outcome of the clinical case involving recalcitrant plantar warts?
 - a. Treatment with an IRM led to complete clearance after almost a year of follow-up.
 - b. After initial surgical reduction of the lesions, adjunctive topical therapy led to complete clearance.
 - c. IRM therapy greatly minimized the extent of follow-up surgery.
 - d. IRM therapy blunted the activity of a TNF inhibitor, resulting in recurrence of psoriasis.
4. The case involving basal cell carcinoma:
 - a. Reinforced the role of surgery as the primary option for skin diseases related to excessive sun exposure.
 - b. Demonstrated that topical therapy is unlikely to have more than a modest adjunctive therapeutic role.
 - c. Suggested that IRM therapy might have cleared residual basal cell carcinoma at the lesion margins.
 - d. Provided compelling evidence that IRM therapy can eliminate the need for destructive interventions for photodamaged skin.
5. A form of IRM therapy currently is approved for treatment of:
 - a. Common warts
 - b. Squamous cell carcinoma of the skin
 - c. Lentigo maligna
 - d. Actinic keratosis
 - e. None of the above

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