Anticipate Hyperpigmentation in Dark Skin Acne

**Therapies such as retinoids and benzoyl peroxide may trigger irritation and cause the skin to darken.**

**BY MICHELE G. SULLIVAN**

FROM THE AMERICAN ACADEMY OF DERMATOLOGY’S ACADEMY 2010 MEETING

CHICAGO – Patient education is just as important as clinical therapy when treating acne in skin of color. Even the best acne treatments can cause post-inflammatory hyperpigmentation in dark skin, Dr. Heather Woolery-Lloyd said.

“I always tell patients that these medications can be a little irritating and if you feel too dry or irritated, go to every other day or discontinue its use and try something else, because they can develop hyperpigmentation if the irritation continues,” said Dr. Woolery-Lloyd, director of ethnic skin care at the Baumann Cosmetic and Research Institute, Miami.

Black patients have about a 29% incidence of acne—a very common cause of hyperpigmentation. “The hyperpigmented acne macule is very common,” she said. “But, in black skin, comedonal lesions also show significant histologic signs of inflammation. So, if you are treating acne in skin of color, you’re also going to be treating hyperpigmentation.”

In their normal behavior with pants, long sleeves, and a hat, instead of sending patients to the sun,” said Dr. Craig Elmets. “In the result of overexposure to the ultraviolet light, but to repair the DNA damage that occurs as a result of overexposure to the sun,” said Dr. Craig Elmets. “In the stead of sending patients to the beach, cover up with long pants, long sleeves, and a hat, we can send them off to engage in their normal behavior with less worry about the long-term consequences.”

Some acne remain the first line of defense against cancer—in ultraviolet radiation, but to repair the DNA damage that occurs as a result of overexposure to the sun, said Dr. Craig Elmets. “In the stead of sending patients to the beach, cover up with long pants, long sleeves, and a hat, we can send them off to engage in their normal behavior with less worry about the long-term consequences.”

Sunscreens also have limited efficacy. “The label; in fact, studies show that if used improperly, patients have not reached the sunscreen factor. “The label; in fact, studies show that if used improperly, patients have not reached the sunscreen factor.”

A 2001 study allocated 30 patients with xeroderma pigmentosum who had metastatic or locally advanced basal cell carcinoma to dimericine or placebo, with the primary end point of new actinic keratoses. A number of agents being investigated for the chemoprevention of skin cancers have shown promising results in both animal and human studies.

Dimericine is a form of the bacterial enzyme T4 endonuclease. When encapsulated in a liposome and applied topically, the compound appears to boost the body’s DNA repair response by increasing base excision repair, Dr. Elmets said.

A 2001 study allocated 30 patients with xeroderma pigmentosum to dimericine or placebo for 1 year, in addition to sun-screen. Patients in the active group had a 68% reduction in basal cell carcinomas in comparison to the placebo group. Patients in the active group had a 68% reduction in basal cell carcinomas in comparison to the placebo group.

The compound is a systemic hedgehog pathway antagonist. “The hedgehog pathway is an important regulator of cell growth and differentiation during embryogenesis. But mutations are associated with basal cell carcinomas in both children and adults,” he said. Animal research has shown that inhibiting this pathway can reduce tumor growth.

In a study of 11 patients with metastatic or locally advanced basal cell carcinoma who took the drug at different doses, 18 achieved a response; 2 were complete and 16 were partial. Disease stabilized in 15 patients and progressed in 4 (N. Engl. J. Med. 2009;361:1164-72).

Ten-year trials, in progress or recruiting, focus on CDC-049’s safety and efficacy in a variety of cancers, including basal cell carcinoma, head and neck, and breast cancers. DMF0 (alpha-di-fluoromethylornithine, also known as efomorphine) irreversibly inhibits ornithine decarboxylase, an enzyme unregulated in many tumors. In a recent phase III trial of 219 patients with a history of nonmelanoma skin cancer, there was a 39% reduction in the total numbers of nonmelanoma skin cancers between the active and placebo group after 4.5 years of follow-up (more than 1,200 person-years). New basal cell carcinomas were 33% less common in the active group than in the placebo group (Cancer Prev. Res. 2010;3:25-34).

Finally, Dr. Elmets said, an ancient and familiar drink holds in intriguing possibilities. The primary component of green tea, EGCG, (epigallocatechin-3-gallate) is a potent antioxidant that appears to reduce histologic and clinical damage from exposure to ultraviolet light A and B. When EGCG is applied topically or given to animals to drink in their water, they show a dramatic reduction in new skin cancers. In humans, it reduces UVA and UVB erythema, he said.

Dr. Elmets disclosed that he has received research support from Pfizer Inc. and holds an intellectual property right on the use of EGCG as a skin cancer chemopreventive agent.

Topical, Oral Agents Show Promise as Skin Cancer Defense

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