CURRENT THERAPY IN DERMATOLOGY

1985 · 1986

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PREFACE

Knowledge is of two kinds. We know a subject ourselves, or we know where we can find information upon it.

Samuel Johnson, 1775

In the clinical practice of medicine we tend to become comfortable and knowledgeable about the management of routine dermatologic cases. However, since all patients are not routine and do not follow a prescribed pattern as a consequence of our therapy, we often need and seek additional information for specific problems. To be useful this information must be timely, practical, and authoritative. Standard textbooks of dermatology are helpful for understanding the basic information available about a specific disease, but customarily, they do not provide in-depth or up-to-date information about therapy. Specifically, textbooks rarely provide (1) a basic therapeutic approach to a problem which might help a clinician unfamiliar with a particular disease or (2) alternative forms of therapy to provide help for complicated problems.

The emphasis of this book is on the current therapy of specific cutaneous diseases. Both common and somewhat unusual diseases were selected for inclusion, but there was no attempt to cover all possible diseases. We believe that the coverage is appropriate for physicians who deal with skin disease, including family practitioners, internists, pediatricians, emergency physicians, and dermatologists.

Physicians recognized for their clinical expertise were asked to write the various chapters in the book, emphasizing their experience and personal approach in managing a patient with a given disease. We asked them to give specific drugs, their dosages, and their routes of administration in detail. Where there were several alternative modes of therapy we asked that these be given with a stated order of preference. Complications of therapy and the management of these complications were to be stressed. The differential diagnosis and the pathogenesis of the disorder were to be discussed only when they provided a rationale for management. Inevitably there was some overlap among subjects, but where divergent views or newer approaches to treatment were proposed, we allowed and actually encouraged repetition.

We would like to thank our colleagues for contributing their expertise in the preparation of this book. We especially want to thank Brian Decker for his guidance, and Sally Vasek and Gwen Warner for their dedicated help.

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PAPULOSQUAMOUS DISEASES

PSORIASIS

THOMAS F. ANDERSON, M.D.
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Psoriasis is a common, chronic heritable skin condition characterized by abnormal proliferation and differentiation of the epidermis in association with an exaggerated inflammation. Clinical involvement can vary from small guttate erythematous papules with little scale in a generalized distribution to well demarcated erythematous plaques with an adherent and flaking, silvery-white scale confined to the extensor surfaces. Macerated fissures in the intertriginous areas or severe scalp and nail involvement can also be seen. Psoriasis patients can also present with a generalized exfoliative erythroderma or pustular psoriasis.

Psoriasis involving the scalp, genitalia, or nail bed and matrix are all treated differently. Thus, treatment for psoriasis must be multifocal and individually designed. Other aspects necessary to consider in the design of appropriate therapy include the patient's physical or emotional state, sophistication, and expectations. The practical limitations of each therapy, including cost, constraints of the therapeutic setting, requirements for patient travel, time off work, compliance, and reliability must also be weighed.

In general, patients with psoriasis can be classified into three major groups based upon severity; mild, moderate, and severe. Mild disease can usually be managed with a combination of bland emollients, keratolytic therapy, or corticosteroids. Moderately severe disease usually requires the addition of tar or anthralin products and possibly ultraviolet light therapy. Severe disease may require all of the preceding in a hospital setting or systemic alternatives such as antimetabolites or psoralen and ultraviolet-A (PUVA) therapy. Experimental therapy with retinoids or other agents can also be considered.

TREATMENT

Bland Emollients

Perhaps the most inexpensive and widely used treatment for psoriasis is the frequent and liberal application of occlusive emollients such as Vaseline Petroleum Jelly, Eucerin, mineral oil, and Lubriderm. These oleaginous substances form a protective coating over the skin. Patients with active psoriasis often form new plaques in the exact sites of cutaneous injury. Emollients may work in part by decreasing this "Koebner phenomenon" by decreasing dry skin and excoriations. They also increase the hydration of the diseased psoriatic stratum corneum, thus facilitating the removal of the excess scale. Mild psoriatic plaques may stabilize the even regress (with decreased mitosis) with continued use of these moisturizing, occlusive emollients as the sole form of therapy.

Keratolytic Therapy

In addition to occlusive emollients, plastic occlusive dressings (such as Saran Wrap, plastic gloves, or total body vinyl suits) have been shown to be useful in the treatment of hyperkeratotic psoriasis. Plastic occlusion not only softens and hydrates the horny layer but also increases the cutaneous penetration of topical drugs. One risk of plastic dressings and occlusive ointments is the overgrowth of cutaneous bacteria with resulting folliculitis or maceration. Therefore, occlusive dressings are generally used only intermittently

for 2- to 4-hour periods or, at most, overnight.

Chemical keratolytic agents are also useful in helping to remove excessive hyperkeratosis of the palms and soles or recalcitrant psoriatic plaques elsewhere on the body. Urea in a 20 to 40% concentration or salicylic acid in a 2 to 6% concentration are the most common agents used. Ammoniated mercury at a 5% concentration has been recommended for scalp psoriasis. Its use has been limited, however, by reports of renal tubular damage and allergic hypersensitivity with chronic use.

Propylene glycol solutions at a concentration by volume of 40 to 60% in water are particularly effective in removing thick scale when used under moist plastic occlusion. A commonly prescribed preparation, Keralyt, is a combination of 6% salicylic acid in a 60% propylene glycol gel. This can be applied to thickened areas such as the palms and soles, followed by the application of damp cotton gloves or stockings. Large plastic gloves, booties, or Saran Wrap sheets are then worn with tape at the wrists and ankles, forming a watertight but not constricting seal. After a 3- to 4-hour period during the day or overnight, this plastic dressing is removed and excess keratin atraumatically scraped away with a washcloth, a polyester fiber sponge (Buf-Puf) or an abrasive tool such as a pumice stone. Occlusive ointments must be applied immediately after this procedure to avoid desiccation of the remaining stratum corneum and the development of painful fissures. If a superficial erosion or painful fissure does occur, the area must be protected with an emollient or zinc oxide paste prior to any further treatment.

Urea at a 20 to 40% concentration in hydrophilic petrolatum dissolves hydrogen bonds in epidermal keratin and can be used as an alternative to Keralyt in this procedure.

Corticosteroids

Topical glucocorticosteroids are perhaps the most commonly prescribed treatment for psoriasis. These agents are vasoconstrictive, anti-inflammatory, and antimitotic; although expensive, they are generally non-staining and may only need to be applied once per day to once per week in individual cases. Ointment bases, although messy, are generally more effective than creams, especially when treating thick, hyperkeratotic psoriatic areas such as elbows and knees. Hairy areas are best treated with creams or lotions; solutions, gels, and sprays are the easiest preparations to apply to the scalp. Corticosteroid solutions are helpful in treating psoriasis of the ear canal and fingernails.

After the initial use of a high-potency corticosteroid to bring about fast resolution, the potency and frequency of application of these products are gradually reduced in order to prevent adverse cutaneous side effects such as striae, atrophy, telangiectasia, purpura, and acneiform eruptions. Glaucoma or cataracts may result from the chronic application of corticosteroids close to the eyes. Corticosteroids, when combined with plastic occlusive dressings can be complicated by hypertension, glucosuria in diabetic patients, glaucoma, and pustular psoriasis exacerbation after withdrawal. Continued use of topical corticosteroids is often associated with a loss of their effectiveness. A rebound phenomenon may follow precipitous discontinuation of corticosteroids. Considering the expense, side effects, and loss of effectiveness over time, topical corticosteroids are best used for short periods (less than 3 weeks) for mild psoriasis and never as the sole therapy for psoriasis.

We do not recommend the use of systemic corticosteroids in the treatment of psoriasis. However, intralesional corticosteroids can be a valuable treatment adjunct with a minimum of side effects. Small doses of highly insoluble forms of triamcinolone (such as Kenalog) can be injected into recalcitrant psoriatic plaques up to a total dose of 20 mg without any evidence of systemic effect. One milligram (0.1–0.4 cc) of triamcinolone acetonide injected from a concentration of 2.5 to 10.0 mg/cc into any one treatment site may result in a prolonged resolution of a psoriatic papule or plaque, lasting for weeks to months. The use of a 30-gauge needle or a mechanical or air-powered hypodermic gun may be helpful to reduce the amount of discomfort in treating isolated sites, e.g. scalp or fingernail, or recalcitrant plaque psoriasis. Side effects of intralesional corticosteroids include the development of sterile abscess or cellulitis, pain, atrophy, hyper- and hypopigmentation, and telangiectasia. Tachyphylaxis and rebound appear to be less evident with the intralesional dosage route. Many of these side effects are only temporary if care is taken to use the lowest possible dose and to avoid reinjection at the same site for 4 to 6 weeks.

Tar Products

A wide variety of products containing derivatives of crude or refined coal tar are available for

the treatment of psoriasis. Although the mechanism of action of tar products is unknown, these agents have been shown to suppress DNA synthesis when applied to mouse skin. Adverse reactions to tar therapy include folliculitis, photosensitivity, and allergic hypersensitivity, not to mention the noxious odor and staining properties of these agents. Animal and epidemiologic studies suggest that tar products may be carcinogens; however, a recent 25-year follow-up study of psoriatics treated with coal tar showed no increase in the incidence of cutaneous or internal malignancy.

Crude coal tar in a 1 to 5% concentration may be incorporated into zinc oxide paste, Aquaphor, or washable petrolatum to treat large, thick psoriatic plaques. Tar products should not be applied to intertriginous or hairy areas or under moist plastic occlusion, in order to avoid primary irritation and folliculitis. A less irritating alcohol extract of tar, liquor carbonis detergens (LCD), is commonly used in a 10% concentration in hydrophilic creams or lotions for generalized acute guttate psoriasis. Tar extracts formulated in creams or gels such as Fototar, Estar, or Psoragel, have recently been developed for outpatient use and for treatment of the scalp. These preparations are less messy and wash off easily. Although these agents decrease the incidence of staining and folliculitis, the gels tend to desiccate the skin, and they produce a burning sensation upon application. A wide variety of tar-based shampoos and bath and body oils is available without prescription.

Anthralin Products

Anthralin, which is a synthetic replacement for the naturally occurring antipsoriatic agent, chrysarobin, appears to improve psoriasis through a variety of actions. Like tar, it is antimitotic but it also may inhibit the production of a variety of mediators of inflammation. Unfortunately, it is a potent cutaneous irritant and is more likely than tar to stain the skin and clothing. It should be avoided in acute inflammatory psoriasis, flexural areas, and on the face. Accidental contact with the eyes may produce a severe chemical conjunctivitis.

Anthralin is generally compounded in a 0.1 to 2.0% concentration in a stiff zinc oxide paste, usually with the addition of salicylic acid in a 0.2 to 4.0% concentration as a stabilizing agent. Patients are treated with the lowest concentration initially, gradually raising the strength every few days until either the desired response or irritation occurs. A thick coating of paste must be applied carefully so as to cover only the psoriatic plaques. To prevent irritation and "koebnerization" of the surrounding skin, the plaques are often ringed with a protective coat of petrolatum or plain zinc oxide paste. The anthralin-covered plaques may then be dusted with talc and covered with tube gauze or stockingette bandages for 4- to 12-hour periods. Mineral oil, often with an added emulsifying agent such as 5% sodium lauryl sulfate, is then used to remove the paste prior to the next application. Anthralin has also been incorporated into ointments and liquid waxes at lower concentrations for extensive guttate psoriasis and scalp therapy. Staining and irritation can be lessened by using low concentrations of anthralin for long periods of time or high concentrations for short periods (under an hour).

Response to anthralin can be measured by the degree to which the psoriatic plaque acquires the anthralin stain and the amount of desquamation and thinning of the plaque which occurs with continued use. As the plaque thins, lesser concentrations of anthralin or shorter application periods must be utilized to prevent painful, edematous responses and potential koebnerization. Often the concomitant use of low-potency corticosteroids can blunt this irritant effect.

Ultraviolet Light Therapy

Sunlight and artificially produced mid-range "erythemogenic" ultraviolet irradiation (UVB) is an effective antipsoriatic therapy, presumably acting by directly suppressing DNA synthesis. Ultraviolet light damages epidermal DNA, producing nuclear photoproducts which must be repaired before further mitosis. This suppression of DNA synthesis is enhanced by anthralin and coal-tar products. Titrated exposures of increasing doses of ultraviolet light can be delivered to the skin through a template with a series of small openings. In this manner, the dose of ultraviolet light necessary to produce a minimum degree of erythema (minimal erythema dose) can be delineated. Starting at a dose just below that necessary to produce erythema, daily treatments are delivered gradually, raising the time of exposure by approximately 10 to 50 percent until minimum erythema is maintained approximately 6 to 24 hours after phototherapy. Twenty to 40 exposures are generally necessary in order to obtain the best results. The end point of treatment is the gradual desquamation and thinning of individual psoriatic

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