

Guidelines of Care for the 10 most common dermatologic diseases:

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Disclaimer

Adherence to these guidelines will not ensure successful treatment in every situation. Further, these guidelines should not be deemed inclusive of all proper methods of care or exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding the propriety of any specific procedure must be made by the physician in light of all the circumstances presented by the individual patient. For the benefit of members of the American Academy of Dermatology who practice in countries outside the jurisdiction of the United States, the listed treatments may include agents that not currently approved by the U.S. Food and Drug Administration.

1. **Acne Vulgaris**
2. **Alopecia Areata**
3. **Atopic Dermatitis**
4. **Contact Dermatitis**
5. **Cutaneous Adverse Drug Reactions**
6. **Nail Disorders**
7. **Psoriasis**
8. **Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis, Onychomycosis, Piedra, Pityriasis, Tinea Capitis , Tinea Barbae, Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis.**
9. **Vitiligo**
10. **Warts: Human Papillomavirus**

1- Guidelines of Care for Acne Vulgaris*

Reference: 1990 by the American Academy of Dermatology, Inc.

I. Introduction

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II. Definition

Acne vulgaris is a follicular disorder that affects susceptible pilosebaceous follicles, primarily of the face, neck, and upper trunk, and is characterized by both noninflammatory and inflammatory lesions.

III. Rationale

A. Scope

Acne is a disease of high prevalence and affects many persons in their teen age years. Although peak prevalence may be at age 17, acne may begin as early as age 8 and is not uncommon in the 10- to 12-year-old age group in which it is often overlooked. Both sexes are affected equally, but males have, on average, greater degrees of severity. A substantial percentage of adults are affected either as a continuation of their teenage acne or its first appearance in the third or fourth decade. Most cases of acne subside and involute spontaneously and completely within a few years of their onset, but a small percentage remain active. The disease may cause considerable emotional distress because of its appearance, even when the disease has involuted if scarring has taken place. The psychosocial impact on teenagers may be devastating.

B. Issue

Acne vulgaris is a condition of unknown origin; however, multiple factors are known to contribute to its pathogenesis and its aggravation. Although there is no known predictable cure for the disease, numerous therapies are available that can substantially control its activity in most cases.

IV. Diagnostic criteria

A. Clinical

Patient history

To determine appropriate treatment a patient history should be taken. Information to be gathered may include the following:

Duration, to include progression to point of maximal severity

Location

Seasonal variation

Aggravation by stress

For women

Premenstrual flare-up

Menstrual history and pregnancy status

Increase of androgen-dependent hair

Thinning of scalp hair

Oral contraceptives and effect on acne

Hormone tests

Cosmetics and moisturizers: type and frequency

Current treatment(s): topical and systemic

Of acne

Of other diseases

Past treatment(s): topical and systemic

Of acne

Of other diseases

Family history of acne

Other skin disorders, especially but not limited to:

Atopy, personal or familial (because of occasional irritation to topical acne preparations)

Hidradenitis suppurativa

Drug allergies

General health, especially but not limited to:

Hepatic disease

Renal disease

Endocrine

Physical examination

Establishment of diagnosis should occur after review of the patient history and physical examination of the patient for the clinical criteria of acne.

Lesion type

Noninflammatory

Open comedones

Closed comedones

Inflammatory

Papules

Pustules

Nodules and/or cysts

Location

Face/neck

Back

Anterior chest

Extremities

Gradation

Mild, moderate, severe

For each predominant lesion type

Location

Complications

Scarring type

Atrophic

Localization

Severity

Discoloration

Hypertrophic

Localization

Severity

Discoloration

Keloids

Localization

Severity

Discoloration

Scarring grade

Definition

Location

Degree

Other associated findings include but are not limited to:

Postinflammatory macular lesions

Postinflammatory hyperpigmentation and hypopigmentation

Hirsutism for women

Alopecia for women

Asymmetry of distribution of acne

Excoriations

Diagnostic tests

There are no diagnostic tests for acne vulgaris. However, in some instances diagnostic tests are used to differentiate and identify acne-like eruptions or to detect the presence of systemic conditions that aggravate acne. Such tests include but are not limited to:

Bacteria culture (e.g., gram-negative folliculitis)

Hormonal assay (e.g., presence of androgen imbalance)

Biopsy when necessary to differentiate acne from other diseases

Inappropriate diagnostic tests*

Routine allergy testing

Hair analysis

*The tests listed are examples.

Exceptions

Not applicable

Evolving diagnostic test

Not applicable

Recommendations

Treatment

Topical treatment alone may be indicated for the following types of acne: mild to moderate comedonal lesions, superficial inflammatory (papular or pustular), and usually non-scarring. In addition, systemic treatment may be indicated for the following types of acne: moderate to severe (scarring or non-scarring) or that in patients with persistent hyperpigmentation. Systemic treatment may need to be used alone in patients who are intolerant to topical treatment or in whom such treatment has failed.

Nonsurgical

Topical therapy: most commonly used, but not limited to

Benzoyl peroxide

Benzoyl peroxide - erythromycin

Benzoyl peroxide - sulfur

Topical antibiotics

Tetracycline lotion

Clindamycin lotion, gel

Erythromycin lotion, swabs, gel

Meclocycline cream

Tretinoin

Salicylic acid

α -Hydroxy acid

Sulfur: including Vleminckx's solution

Resorcinol

Miscellaneous: astringents, soaps, cleansers

Systemic therapy: most commonly used, but not limited to:

Antibiotics, oral

Tetracycline

Erythromycin

Minocycline

Trimethoprim-sulfamethoxazole

Other

Isotretinoin, oral

Primary and only approved use is for severe, recalcitrant, cystic acne, refractory to conventional anti-acne measures, including systemic antibiotics. For women of childbearing potential, Appendix A guidelines apply.

Hormonal treatments may include the following:

Corticosteroids

Anti-inflammatory actions: high dose

Androgen suppressant action: low dose

Sex hormones (for women only)

Estrogen (oral contraceptive medication)

(c)Antiandrogens

Other treatments may include the following:

Dapsone

Diet (in selected cases)

Ultraviolet light

Superficial exfoliation

Carbon dioxide-acetone slush

Sulfur-resorcinol

Chemical

Surgical

Lesional therapy

Extraction of comedonal contents

Drainage of superficial pustules and cysts

Excision of sinus tracts and cysts

Intralesional corticosteroids

Cryotherapy

Dermabrasion (scars)

Filling materials (scars)

Surgical repairs (scars)

Other recommendations

Follow-up examinations should be a part of the optimal management of patient's acne, specifically to gauge degree (or lack) of improvement, tolerance to medications, need to augment or attenuate treatment depending on clinical response, and employment of lesional therapy. Intervals between visits will vary, dependent on, but not limited to, the severity of the problem, the intensity of treatment, and the need for frequent visits for lesional therapy.

In the early phase of treatment, more frequent follow-up visits are required than later when the condition has become less active. On average, the interval between visits will range from 1 to 12 weeks, with a median of 4 weeks.

Miscellaneous

Not applicable

Appendix A. Guidelines for prescribing isotretinoin (Accutane) in the treatment of female acne patients of childbearing potential*

PATIENT SELECTION

In the treatment of females of childbearing potential, Accutane should be used only for patients with severe, disfiguring, cystic acne.

Accutane must not be used by females who are pregnant or who may become pregnant while undergoing treatment.

Accutane should not be the therapy of first choice. It must be demonstrated that the patient is unresponsive to other standard therapies.

The patient must be reliable and capable of understanding the physician's instructions on the use of Accutane, the risks involved, and be willing to comply with these instructions.

The patient must be able to comply with effective contraceptive measures (which may include abstinence) for at least one month prior to, throughout, and for at least one month after treatment.

In the case of minors, the physical presence of the legal guardian is required when informed consent is obtained.

PHYSICIAN INSTRUCTIONS

The physician has the responsibility of explaining fully to the patient the expected outcome of fetal exposure to Accutane. This information must be understood by the patient and her legal guardian, if applicable. A written informed consent form must be signed by the patient and by the physician or third party witness, and in the case of a minor patient, by the patient's legal guardian. A copy of the signed form will be given to the patient and, in the case of a minor patient, to her legal guardian. The original consent form should be maintained in the patient's file.

If the patient is sexually active, she must be using a highly effective form of contraception. She must be capable of compliance. Gynecologic consultation is suggested for patients who have previously become pregnant while using seemingly adequate contraception. Accutane must not be prescribed unless it is documented in the patient's record that a highly effective form of contraception is used (which may include abstinence).

A blood pregnancy test must be administered and shown to be negative within two weeks prior to beginning Accutane therapy. Prescriptions for Accutane must not be dispensed until the pregnancy test is reported as negative. Therapy should begin in day two or three of the next menstrual cycle.

The physician should inform the patient that the Accutane package includes an insert containing information which she should read before she begins using the medication. The physician should encourage the patient to ask him or her questions concerning the risks associated with becoming pregnant while on the medication.

Prescriptions should be written for no more than two weeks beyond a scheduled follow-up visit. Visits to the physician should be at least monthly, and a repeat blood pregnancy test should be done monthly.

Patients must be advised that the maximum duration of a single treatment course is 20 weeks. Contraceptive use must continue for at least one month after therapy completion. A blood pregnancy test should be done one month after completion of therapy.

2- Guidelines of Care for Alopecia Areata

I. Introduction

The American Academy of Dermatology's Committee on Guidelines of Care is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists.

II. Definition

Alopecia areata is a disease that affects hair and sometimes nails, and is a disorder that may occur alone or in association with a variety of other medical conditions. The etiology of alopecia areata is unknown, but is characterized by hair cycle dysfunction and the presence of peribulbar and perifollicular mononuclear cell infiltrates. The disease may affect any hair-bearing area, but is most commonly seen involving scalp, eyebrow, eyelash, and beard hairs. Disease extent may be patchy or extensive. In extreme cases, total loss of scalp hair may result (alopecia totalis) or total loss of scalp and body hair may occur (alopecia universalis). However, these terms are relative as patients with alopecia totalis may also have patches of hair loss on other hair-bearing areas, and patients with alopecia universalis may still have scattered terminal or vellus hairs. Nail changes are observed in 10% or more of patients with alopecia areata. These may precede or coincide with the onset of hair loss and may or may not persist after the hair loss problem resolves.

III. Rationale

A. Scope

Alopecia areata is a common disorder with an estimated prevalence (number of cases existing at any one time) of 1 in 1000. However, the exact prevalence is unknown because many mild cases may never come to a physician's attention. There is no known race, sex, or occupational predilection for the development of alopecia areata. Alopecia areata can occur at any age and the course is highly variable. Relapses are common and patients may require follow-up for prolonged periods of time.

B. Issue

The cause of alopecia areata is unknown although most evidence supports the hypothesis that alopecia areata is an immunologically mediated disease. In alopecia areata, permanent destruction of hair follicles usually does not occur. Other diseases, such as vitiligo, or other autoimmune diseases, such as thyroiditis, may be seen in association with alopecia areata. Alopecia areata that develops in early childhood presents a management problem because it is not known whether other autoimmune diseases occur more commonly in such children compared with the normal population. It is not clear how carefully such children should be followed up as they age for the development of other autoimmune diseases.

IV. Diagnostic criteria

A. Clinical

1. Patient history

A thorough history is helpful because alopecia areata may occur with a variety of other diseases. The information obtained may also be helpful in choosing the most appropriate therapy. Pertinent history may include the following:

- a) Disease duration, age of onset, recurrences, and extent
- b) Symptoms (burning, itching, tingling before disease activity or concurrent with hair growth)
- c) Aggravation by stress
- d) Current and past treatment

(1) For alopecia areata

(2) For other medical conditions

e) Genetics

f) Medications

g) Personal or family history for alopecia areata may include the following:

(1) Autoimmune disease

(2) Endocrine disease

(3) Atopic diathesis

(4) Anxiety/depression/other psychiatric problems

(5) Eye changes

(a) Cataracts

(b) Retinal pigment abnormalities

2. Physical examination

An examination is done to determine whether the hair loss is compatible with the diagnosis of alopecia areata. The location and number of lesions, disease extent, and characteristics of the hair loss aid in the diagnosis. Common findings in the physical examination may include the following:

a) Hair

(1) Circumscribed oval or circular non-scaly patches of non-scarring hair loss

(2) Short, tapered hairs may be present within the affected areas (exclamation point hairs)

(3) Single or multiple patches of alopecia areata are most commonly seen on the scalp and face, but may also be found on other hair-bearing areas

(4) Disease extent may range from a single patch to loss of all scalp hair (alopecia totalis) or loss of all body hair (alopecia universalis).

(5) Scarring is not present.

(6) Clinically obvious scalp inflammation, although rare, may be seen.

(7) Variants from the common presentation exist and these may include the following:

(a) Ophiasis alopecia areata, in which hair loss occurs in a band partially or completely encircling the occipital and temporal scalp.

(b) Reticular alopecia areata, in which circumscribed patches of hair loss may be present in all stages of disease activity – active, stable, or regrowing.

(c) Diffuse alopecia areata, in which hair never grows very long as the patient chronically experiences recurrent episodes of hair loss and a short anagen growth phase.

(d) Alopecia areata in association with other hair disorders

b) Nails

Nail changes are observed in 10% or more of patients with alopecia areata. These may precede or coincide with the onset of hair loss and may or may not persist after the hair loss problem resolves. Findings may include the following:

- (1) Diffuse fine pitting of the nails
- (2) Thin and brittle fingernails and toenails
- (3) Longitudinal ridging
- (4) Koilonychia
- c) Other

3. Differential diagnosis of alopecia areata may include the following:

- a) Trichotillomania
- b) Tinea capitis
- c) Telogen effluvium and anagen effluvium
- d) Androgenetic alopecia
- e) Syphilis
- f) Systemic lupus erythematosus
- g) Traction alopecia
- h) Acquired immunodeficiency syndrome
- i) Early scarring alopecia
- j) Other

B. Diagnostic tests

Frequently, the diagnosis of alopecia areata is clinically evident. In some cases diagnostic tests may be indicated, such as the following:

1. Hair pull and microscopic examination of hair
2. Biopsy
3. KOH preparation and fungal culture
4. Immune, endocrine, and other laboratory studies, only as indicated
5. Serologic testing as indicated
6. Other

C. Inappropriate diagnostic tests

Biochemical hair analysis

D. Exceptions

Not applicable

E. Evolving diagnostic tests

Not applicable

V. Recommendations

A. Treatment

The success of treatment depends on the age of onset of the disease and the extent of hair loss. The prognosis tends to be worse when alopecia areata starts in early childhood or if the patient has alopecia totalis or universalis. The treatment of patchy alopecia areata is usually successful. However, the therapy for extensive alopecia areata may be prolonged and difficult. In selected cases observation and supportive therapy may be indicated. After treating a patient with extensive disease for 2 to 6 months, if no response is seen, another therapy may be tried. Therapies available for patchy and extensive alopecia areata include the following:

1. Nonsurgical

a) Scalp alopecia areata

(1) Anthralin

(2) Corticosteroids

(a) Topical

(b) Intralesional

(c) Intramuscular

(d) Oral

(3) PUVA therapy

(a) Systemic

(b) Topical

(4) Topical minoxidil

(5) Hair prosthesis

(6) Psychological care

(7) Evolving

(a) Contact allergens

(1) Topical dinitrochlorobenzene

(2) Squaric acid dibutylester (SADBE)

(3) Diphenylcyclopropanone

(b) Cyclosporine

(c) Isoprinosine

(8) Other

Eyebrow alopecia areata

Intralesional corticosteroids

2. Surgical

a) Partial hair replacement surgery, in selected cases

b) Cosmetic tattooing in selected cases

3. Other

B. Miscellaneous

Regular follow-up visits may be necessary. Therapy may be successful, but when discontinued, alopecia areata may relapse. In addition, although successfully treated patients may experience excellent hair regrowth, relapses may occur weeks, months, or years later.

3- Guidelines of Care For Atopic Dermatitis

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I. Introduction

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II. Definition

Atopic dermatitis is an acute, subacute, or chronic pruritic dermatosis with other cutaneous findings, such as xerosis, excoriations, and lichenification, often occurring in persons with asthma, allergic rhinoconjunctivitis, contact urticaria, or with a family history of the same. There is no biologic marker for the disease.

III. Rationale

A. Scope

Atopic dermatitis is a disease that may cause physical suffering, pronounced and total disability, and anguish for the patient and family. The reported incidence of atopic dermatitis indicates that a significant percentage of the general population is affected at some time in their lives.

B. Issue

The diagnosis of atopic dermatitis may be made with reasonable certainty, in most cases, from family history, patient history, and examination of the skin.

IV. Diagnostic criteria

A. Clinical

1. Major characteristics may include the following:

- a) Pruritus with or without excoriation
- b) Typical morphology and distribution
 - (1) Eczematous dermatitis
 - (2) Flexural lichenification or linearity in adults
 - (3) Facial and extensor involvement in infants and children
 - (4) Any of these patterns or combination of patterns can appear in both adults and children.
- c) Chronic or chronically relapsing dermatitis
- d) Personal or family history of atopy (asthma, allergic rhinoconjunctivitis, atopic dermatitis, contact urticaria)

2. Other characteristics may include the following:

- a) Xerosis
- b) Ichthyosis/palmar hyperlinearity/keratosis pilaris
- c) Early age of onset
- d) Cutaneous colonization and/or overt infections

(1) *Staphylococcus aureus*

(2) Herpes simplex and other viral infections

(3) Warts

(4) Molluscum

(5) Other

e) Nonspecific hand and/or foot dermatitis, increased susceptibility to irritant contact dermatitis

f) Nipple eczema

g) Cheilitis

h) Recurrent conjunctivitis

i) Infraorbital fold

j) Keratoconus

k) Anterior subcapsular cataracts

l) Orbital darkening

m) Facial pallor/facial erythema

n) Erythroderma

o) Pityriasis alba

p) Anterior neck folds

q) Perifollicular accentuation

r) White dermographism/delayed blanch

s) Impaired cell-mediated immunity

t) Other

3. Characteristic flare factors may include the following:

a) Sweating with enhanced pruritus

b) Intolerance to wool, any coarse fabric or non-absorptive occlusive garment, lipid solvents, and wet working conditions

c) Environmental or emotional factors

d) Food intolerance

e) Physical trauma

f) Other

B. Diagnostic tests

Uncomplicated atopic dermatitis with clear clinical markers does not usually require diagnostic tests. Definitive diagnosis or complicating factors, including flare factors, however, may require laboratory confirmation. Tests may include the following:

1. Complete blood cell count and differential, total count for eosinophilia
2. Serum IgE or antistaphylococcal IgE level
3. Smears for infectious agents (e.g., Tzanck test, KOH preparation) and immunofluorescence
4. Bacterial culture with antibiotic sensitivity, viral and fungal cultures
5. Skin biopsy
6. Patch tests
7. Scratch/prick/intradermal tests
8. Radioallergosorbent tests may be appropriate in special cases.
9. Immunologic testing
10. Food elimination
11. Psychological evaluation: Atopic dermatitis does appear to have a distinct tendency to flare in response to psychic stress such as anxiety, depression, anger, embarrassment, shame, and resentment. Patients subjected to experimental stress interviews manifest erythema and then pruritus in skin areas subject to dermatitis. If factors cannot be adequately evaluated and treated by the dermatologist, psychological evaluation and care, including testing, may be of value and should be considered.

C. Inappropriate diagnostic tests

Not applicable

D. Exceptions

Not applicable

E. Evolving diagnostic tests

1. Phenotyping and quantification of cellular aspects of immune response cells
2. Other

V. Recommendations

A. Treatment

There are no specific therapeutic modalities for complete control of atopic dermatitis. Management must be individualized to improve morbidity (itching and scratching) with the goal to allow most patients to function in a normal productive manner.

1. Nonsurgical

a) Systemic treatment may include the following:

(1) Antihistamines: single or multiple agents

(a) Hydroxyzine hydrochloride

(b) Doxepin hydrochloride

(c) Other

(2) Antiinfective agents

(3) Systemic corticosteroids in selected cases

(4) Others, which may include the following:

(a) Nonsteroidal anti-inflammatory drugs

(b) Antidepressants

(c) Phenothiazine

b) Intralesional corticosteroids in selected cases

c) Topical treatment may include the following:

(1) Corticosteroids, with the use of mid- to high-potency compounds for brief periods and low-potency compounds for maintenance therapy. When extensive areas of dermatitis are being treated ultra-high-potency corticosteroid compounds may be used for brief periods. Certain vehicles may cause irritation; thus alternative products or compounded preparations may be indicated.

(2) Tar preparations

(3) UV light

(a) UVA, with or without psoralen

(b) UVB

(c) Goeckerman and/or Ingram

(4) Emollients

(5) Pramoxine hydrochloride

(6) Therapeutic baths, compresses, and cleansers

(a) Tars

(b) Emollients

(c) Colloidals

(d) Skin cleansers

(7) Wet dressings

(8) Other

d) Hospitalization may be indicated or required for patients with severe flares or complications.

2. Surgical

Not applicable

3. Other and/or evolving

a) Immunotherapy

(1) Cyclosporine

(2) Methotrexate

(3) Azathioprine (Imuran)

(4) Interferon gamma

(5) Other

b) Oral ketotifen (a mast cell stabilizer)

c) Caffeine administered topically, 10% to 30% in petrolatum or water-in-oil base

d) Dietary

(1) Elimination diet

(2) Supplements

(a) Evening primrose oil

(b) Linoleic acid

(c) γ -Linoleic acids

(d) Fish oil

(e) Other

e) Hyposensitization

f) Others may include but are not limited to the following:

(1) Occupational and/or psychological counseling

(2) Bed rest

(3) Grenz ray, in carefully selected patients with localized disease

(4) Biofeedback

(5) Psychotherapy

B. Miscellaneous treatment considerations

Flare factors may include but are not limited to the following:

1. Infection

a) Bacterial

b) Viral

c) Fungal and yeast

2. Dry skin

a) Improper bathing

b) Use of strong soap, bubble bath, and detergent

3. Psychological stress

a) Psychotropic drugs may be used in selected cases.

b) Psychological counseling may be indicated.

4. Antigenic exposure

5. Contactants

a) Immunologic

b) Nonimmunologic

c) Irritants

(1) Wool or rough fabrics or materials

(2) Lipid solvents

(3) Wet working conditions

6. Sweating

a) Overheating

b) Saunas and steam

c) Excessive physical exertion

7. Physical trauma

8. Environmental factors

9. Food intolerance

4- Guidelines of Care for Contact Dermatitis

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II. Definition

Contact dermatitis is an altered state of skin reactivity induced by exposure to an external agent. Substances that produce this condition after single or multiple exposures may be irritant or allergic in nature and will often appear as an inflammatory process. Direct tissue damage results from contact with irritants. Tissue damage by allergic substances is mediated through immunologic mechanisms. The most common clinical expression of this induced inflammation is dermatitis (eczema).

III. Rationale

A. Scope

Contact dermatitis is a common reason for consulting a dermatologist and constitutes approximately 5.7 million physician visits per year. All age groups are affected, and there is a slight female predominance as reflected in patients seen for diagnostic patch testing. The complex nature of the chemical environment (natural and synthetic) in which we live brings the skin into contact with many potential exposures that may or may not pose a hazard, depending on individual susceptibility. There are more than 85,000 chemicals in the world environment today. Almost any substance can be an irritant, depending on the circumstances. Furthermore, more than 2800 substances have been identified as contact allergens. The potential for these substances to cause contact dermatitis varies greatly, and thus the severity of the dermatitis ranges from a mild, short-lived condition to a severe, persistent, job-threatening, and sometimes life-threatening disease.

B. Issue

Accurate diagnosis is the key to proper management of contact dermatitis. If the agent(s) causing the dermatitis can be found and successfully avoided, recovery can be anticipated; but if contact continues, the dermatitis may become chronic, disabling, and a serious threat to continued work and the activities of daily living. After prolonged and repeated episodes of dermatitis, a few patients may not fully recover, even with adequate medical care and following avoidance of its causes.

IV. Diagnostic criteria

A. Clinical

1. History may include the following:

a) General medical status

b) Onset

(1) Location

(2) Symptoms: Itching, burning, stinging

(3) Description: Redness, blisters, cracks, scales, welts, dryness, rash

(4) Other

c) Progression

(1) Relation between exposure and time interval of dermatitis

(2) Relation to home and recreation

(3) Relation to work

(4) Relation to activity: Sweating

(5) Relation to sun/air exposure, season, and time of day

(6) Other

d) Remissions

(1) Relation to weekends and/or vacations

(2) Response to treatment and rapidity of recurrence after discontinuing medication

(3) Relation to stress and/or anxiety

(4) Relation to water exposure

(5) Other

e) Work/job history

(1) Exact nature of work

(2) Duration of present activity

(3) Others similarly affected

(4) Changes in procedure or chemical exposure

(5) Protective measures: Type and effectiveness

(6) Related symptoms: Burning eyes, sneezing, wheezing

(7) Cleansing agents: Type and frequency of use

(8) Hand washing frequency and agents used

(9) Second job

(10) Review of Material Safety Data Sheets in relation to patient's job

(11) Other factors

f) Other exposure

(1) Hobbies and non-work activities

(a) Gardening, house plants, lawn care, and other outdoor activities (e.g., poison ivy, oak, and sumac, chrysanthemums, primula, Peruvian lily)

(b) Knitting, sewing, macrame

(c) Painting, ceramics, jewelry

(d) Cooking, baking

- (e) Woodworking, carpentry, gluing
- (f) Auto, motorcycle, truck repair
- (g) Photography and photographic developing
- (h) Sports
- (i) Other
- (2) Animals and substances on their skin or fur
 - (a) Dogs and cats
 - (b) Birds and caged animals
 - (c) Farm animals, horses
 - (d) Other
- (3) Cosmetics, fragrances, and personal care products
 - (a) Soaps and detergents
 - (b) Shampoos and other scalp/hair products
 - (c) Creams and lotions
 - (d) Perfumes, colognes, deodorants
 - (e) Nail polishes, artificial nails, and nail/cuticle products
 - (f) Consort or other interpersonal contact/products
 - (g) Other
- (4) Household activities and products
 - (a) Dishwashing products
 - (b) Laundry detergents
 - (c) Furniture waxes, polishes, and dusting agents
 - (d) Bathroom cleaning agents
 - (e) Floor care products
 - (f) Use of gloves (type and frequency)
 - (g) Other
- g) Family history
 - (1) Atopic background: Nature, prevalence, and severity including relation to eczema
 - (2) Ichthyosis, psoriasis, and hand eczema or other significant skin disorder
 - (3) Family members with contact dermatitis
 - (a) Relationship
 - (b) Age at onset

- (c) Type and severity of problems
- (d) Results of therapy and/or testing procedures
- (e) Other
- h) Medical history
 - (1) History of contact dermatitis: Nature, severity, and causative agent(s) if known
 - (2) Previous treatment
 - (a) At onset
 - (b) Self-treatment: over-the-counter treatments; relation to dermatitis
 - (c) By other physicians
 - (3) Medications: topical and systemic; past and present; relation to dermatitis as well as medication allergy
- i) Other

2. Physical examination may include the following:

- a) Location
 - (1) Symmetry
 - (2) Involved versus uninvolved skin
 - (a) Demarcation: Sharp or unclear
 - (b) Evidence of protection by clothing
 - (c) Distribution suggestive of photoexposure or air-borne pattern exposure
 - (d) Other
- b) Lesion type
 - (1) Acute
 - (a) Dermatitis (eczema)
 - (b) Vesicular/bullous
 - (c) Urticarial
 - (d) Excoriations
 - (e) Crusts
 - (2) Chronic
 - (a) Lichenification
 - (b) Pigmentary changes
 - (c) Atrophy
 - (d) Scarring
 - (e) Loss of hair

c) Other

B. Diagnostic tests

The patch test is the standardized diagnostic procedure of choice for allergic contact dermatitis. In general, practitioners should adhere to the following guidelines when performing patch tests.

1. Use test substances appropriately diluted. Standardized test kits containing a number of allergens are available.

2. The most widely used patch test material consists of strips of paper tape onto which are fixed 8 mm diameter aluminum disks. A small amount of allergen is placed within these disks, covering slightly more than one half the diameter of the disk.

3. Apply the patch to the upper or mid back, which must be free of dermatitis and devoid of hair. If shaving is necessary, it should be done only with an electric razor.

4. Leave the patch in place and keep dry for 2 days (36 to 48 hours) before removing unless symptoms of severe reaction occur.

5. Read tests:

a) The same day that patches are removed from the skin, allowing 20 to 30 minutes for erythema to resolve before interpreting results.

b) One additional reading 3, 4, or 7 days after tests initially applied

c) If two readings are impossible, a single reading 3 or 4 days (72 to 96 hours) after patches are initially applied.

6. Grade test reactions according to intensity:

a. 0 = no reaction

b. ? (+ or - reaction) = weak erythema only

c. 1+ = erythema with edema, covering at least 50% of the patch test site

d. 2+ = erythema and papules covering at least 50% of the patch test site. A few vesicles may be present.

e. 3+ = vesicles or bullae covering at least 50% of the patch test site

7. Interpret reactions cautiously:

a. 0 = no evidence of contact allergy

b. ? = doubtful existence of contact allergy

c. 1+ = possible contact allergy. 1+ is a common intensity of false-positive reactions.

d. 2+ and 3+ = probable contact allergy

e. If other stronger 2+ and 3+ reactions are present, the excited skin syndrome may be present.

8. Relate relevance of positive reactions to clinical dermatitis cautiously. Significance must be established by careful history and review of cutaneous exposures.

9. Additional tests of occasional value include the following:

a) Skin biopsy to differentiate from other diseases

b) Open application of a suspected allergenic product to the antecubital fossa twice daily for up to 1 week (the exaggerated use test or repeat open application test [ROAT]). This is applicable to "leave-on" products intended for use on the skin, not "wash-off" products

c) Prick or scratch test in the evaluation of contact urticaria. Emergency resuscitation equipment should be available. Contact urticaria may also be evaluated with an open test on sites adjacent to active dermatitis, as an alternative to prick or scratch testing.

d) Chemical analysis of environmental materials to determine whether they contain a substance to which the patient is patch test positive. The most commonly employed of these is the dimethylglyoxime test for nickel.

e) Potassium hydroxide preparation, fungal and bacterial cultures and appropriate laboratory examinations as needed

C. Inappropriate diagnostic tests

1. Occlusive patch test with irritant concentrations of material or material in which the irritant concentration is unknown

2. Paper immunosorbent test and radioallergosorbent (RAST) tests are not helpful in the diagnosis of contact dermatitis with the exception of contact urticaria when prick or scratch tests may be hazardous. In such case, the PRIST and RAST tests should be performed with caution.

3. Sublingual allergen application with vital sign monitoring as an indicator of adverse reaction

D. Exceptions

Not applicable

E. Evolving diagnostic tests

In vitro lymphocyte stimulation tests, migration inhibition factor, and other laboratory tests of lymphokine production remain investigational tools that at present are insufficiently standardized to allow clinical application.

V. Recommendations

A. Treatment

Topical treatment alone may be indicated for mild cases of contact dermatitis (i.e., limited site of involvement, acute contact dermatitis when the offending agent has been removed, or chronic contact dermatitis with limited symptoms). Systemic treatment may be indicated for control of itching and/or edema even in cases of limited extent. Systemic treatment may also be indicated for moderate to severe acute and/or chronic contact dermatitis.

1. Medical

a) Topical therapy: Most commonly used includes but is not limited to:

(1) Cool compresses with saline solution, water, milk, aluminum subacetate, or other agents

(2) Shake lotions, such as calamine

(3) Topical corticosteroid cream, ointment, lotion, gel, or spray

(4) Colloidal oatmeal baths

(5) Antibiotic creams or ointments, when secondary infection may be present

(6) Coal tar (in chronic eruptions)

(7) Emollients, lubricants, moisturizers (in chronic eruptions)

(8) Nonalkaline cleansers instead of soap

(9) Other

b) Systemic therapy: Most commonly used includes, but is not limited to the following:

(1) Antihistamines

(2) Corticosteroids, oral or intramuscular (Intravenous corticosteroids may be useful in severe acute cases.)

(3) Antibiotics, oral or parenteral (when secondary infection may be present)

(4) Other anti-inflammatory or immunologic agents

(5) Other

2. Surgical

Not applicable

3. Other

a) UVB radiation

b) Psoralen, topical or oral, and UVA radiation (PUVA)

c) Mechanical protection against allergens and irritants (e.g., gloves, protective clothing, protective barrier creams)

d) Removal or avoidance of causal allergens and/or irritants

B. Miscellaneous

Patient education about the nature of the dermatitis, triggering allergen or irritant factors, and suggestions for avoidance or substitution of these factors in the patient's environment may be helpful in management.

5- Guidelines of Care for Cutaneous Adverse Drug Reactions

Reference: 1996 by the American Academy of Dermatology, Inc.

I. Introduction

The American Academy of Dermatology's Guidelines/Outcomes Committee is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists. For the benefit of members of the American Academy of Dermatology who practice in countries outside the jurisdiction of the United States, the listed treatments may include agents that are not currently approved by the U.S. Food and Drug Administration.

II. Definition

An adverse cutaneous reaction caused by a drug is any undesirable change in the structure or function of the skin, its appendages, or mucous membranes. A drug is defined as a chemical substance or combination of substances that are ingested, injected, inhaled, inserted, instilled, or topically applied to the skin or mucous membranes. Adverse reactions may result from overdose, accumulation, pharmacologic side effect, drug-drug interactions, idiosyncrasy, microbiologic imbalance, exacerbation of existing latent or overt disease, Jarisch-Herxheimer reaction, hypersensitivity, autoimmune-like reaction, teratogenic effect, interaction of the drug and sunlight or other light sources (i.e., artificial tanning devices), or other unknown mechanism.

III. Rationale

A. Scope

Although adverse drug reactions are believed to be common, few data exist to document their incidence, seriousness, cost, and ultimate effect on health. Available information comes from four sources: (1) hospital studies; (2) epidemiologic reviews; (3) preapproval studies; and (4) voluntary reporting. In hospitalized patients an attempt to quantify cutaneous reactions disclosed a rate of 2.2 per 100 patients and 3 per 1000 courses of drug therapy

B. Issue

Many skin diseases mimic drug reactions. Consequently, one must have a high level of suspicion in evaluating possible drug reactions. Suspected cutaneous drug reactions are of numerous clinical types, result from various pathophysiologic mechanisms, demonstrate varying degrees of probable causation, and require various approaches to diagnosis and treatment.

Most cutaneous eruptions are not diagnostic of drug causation. Various cutaneous eruptions have different probabilities of being related to drugs. In addition, certain eruptions have particular drugs that are commonly associated with them. Therefore, an assessment of the eruption is important in determining the probability of drug association and set of drugs that may be responsible.

Only a few cutaneous eruptions are specific for drug causation. These include fixed drug eruption, argyria, and arsenical keratosis.

There are five issues to be considered in possible drug eruptions:

1. The assessment of the cutaneous eruption
2. The probability of a relation between the cutaneous eruption and the drug
3. If a drug eruption is probable, clinical and laboratory factors that might alert the clinician to the potential seriousness of the eruption

4. The management of the eruption
5. The prevention of future eruptions to include patient education

IV. Diagnostic criteria

A. Clinical

1. History may include
 - a. General medical history as indicated
 - b. Drug exposure (dosage, date started, duration, and interruptions in use)
 - c. Use of proprietary remedies (e.g., herbals)
 - d. Initiation of drug use and onset of reaction
 - e. Previous adverse drug reactions, cutaneous and otherwise, and type of adverse reaction
 - f. Re-exposure to a drug and exacerbation of eruption
 - g. Improvement after a decrease in dosage or stopping of drug
 - h. Disease states or injuries that may cause the eruption, or act as cofactors (e.g., infectious mononucleosis and ampicillin-related reactions, or HIV and trimethoprim-sulfamethoxazole (Bactrim) reactions)
 - i. Previous family or personal history of skin disease
 - j. Family history of hypersensitivity syndromes, anticonvulsant hypersensitivity syndrome
 - k. Environmental/occupational exposure to other substances that may be etiologic agent (e.g., sunlight, artificial tanning devices)
 - l. Clinical findings that may alert clinician that drug-induced cutaneous eruption may be serious (see Table I)
 - m. Other
2. Physical examination may include
 - a. General physical examination as indicated
 - b. Description of eruption
 - c. Distribution
 - d. Extent
 - e. Color
 - f. Secondary changes
 - g. Other relevant skin disease
 - h. Physical findings that may alert clinician that a drug-induced eruption may be serious (see Table I)
 - i. Other

Table I. Clinical and laboratory findings that should alert clinicians that a drug-induced cutaneous eruption may be serious

Clinical findings

Cutaneous

Confluent erythema

Facial edema or involvement of central part of face

Skin pain

Palpable purpura

Skin necrosis

Blisters or epidermal detachment

Positive Nikolsky's sign*

Mucous membrane erosions

Urticaria

Swelling of tongue

General

High fever (temperature >40°C)

Enlarged lymph nodes

Arthralgias or arthritis

Shortness of breath, wheezing, hypotension

Laboratory results

Eosinophil count >1000/mm³

Lymphocytosis with atypical lymphocytes

Abnormal results of liver function tests

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*The outer layer of the epidermis separates readily from the basal layer with lateral pressure.

B. Diagnostic tests and diagnostic aids

A diagnosis can often be made from the history and physical examination. The following tests may be useful:

1. Skin biopsy
2. Laboratory tests

a. Drug levels - of value when eruption is associated with overdose or other nonallergic type of reaction, or in comatose or non-communicative patient to establish presence of drug

b. Enzymes and metabolites

c. Complete blood cell and differential count

d. Other

3. Cessation of therapy

4. In selected cases re-exposure may be of value; consider the degree of drug reaction and risks

5. Prick or scratch test; emergency resuscitation equipment should be available

6. Patch test

7. Skin culture (bacterial, viral, fungal)

8. Microscopic tests

9. Textbooks of drug eruptions, literature searches, on-line databases, adverse drug reaction software

10. Other

C. Inappropriate diagnostic tests

Not applicable

D. Exceptions

Not applicable

E. Evolving diagnostic tests

In vitro lymphocyte toxicity assay, migration inhibition factor, and other laboratory tests of lymphokine production remain investigational tools that at present are insufficiently standardized to allow clinical application.

V. Recommendations

A. Treatment

1. Medical

a. The management plan is based on

1) Category of eruption (e.g., allergic, alteration in flora [ecological shift])

2) Severity of eruption

3) Natural history of eruption

4) Need for the drug and available alternatives

5) Other

b. The management plan may include

1) Appropriate consultation with the physician who prescribed the drug suspected of causing the eruption

2) Discontinuance of drug

a) The decision whether to continue to administer a drug that is known or assumed to be the cause of a reaction will be influenced by the

- (1) Severity and probable course of the reaction
- (2) Disease for which the drug was prescribed
- (3) Ease or difficulty with which the reaction can be managed
- (4) Availability of chemically unrelated drugs with similar pharmacologic properties

Attention is given to ensure that any substituted drugs are not pharmacologically and/or chemically related to the suspect drug.

b) The physician faced with the likelihood that the patient is suffering from a drug reaction has to consider overall patient management. In decisions regarding discontinuation of a drug, one must consider the risk/benefit ratio of each drug.

- 3) Alteration of dosage and/or administration
- 4) When indicated, hospitalization of patient who has a serious drug reaction
- 5) Other

c. Treatment of eruption

Commonly used agents include but are not limited to:

- 1) Topical and systemic corticosteroids
- 2) Antihistamines
- 3) Topical antipruritic agents
- 4) Baths, with or without additives
- 5) Emollients
- 6) Special treatments in severe reactions depend on the type of severe reaction
- 7) Other

2. Surgical

Occasionally needed in severe reactions (e.g., debridement of necrotic tissue in cutaneous necrosis)

B. Patient education

1. The physician should advise the patient regarding

- a. Drug(s) that may have caused the eruption
- b. Drug(s) to avoid, when practical
- c. Drug(s) that can be used

2. If there is some genetic implication for family members, such information should be conveyed.

3. In selected cases, the patient may obtain and wear commercially available jewelry that identifies the drug(s) to which the patient is allergic.

C. Miscellaneous

Adverse drug reactions may be voluntarily reported to the manufacturer or to the Food and Drug Administration Medical Products Reporting Program (MedWatch, 1-800-332-1088)

6- Guidelines of Care for Nail Disorders

Copyright 1996 by the American Academy of Dermatology, Inc.

I. Introduction

The American Academy of Dermatology's Guidelines/Outcomes Committee is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists. For the benefit of members of the American Academy of Dermatology who practice in countries outside the jurisdiction of the United States, the listed treatments may include agents that are not currently approved by the U.S. Food and Drug Administration.

Definition

Nail disorders include those abnormalities that affect any portion of the nail unit. The nail unit includes the plate, matrix, bed, proximal and lateral folds, hyponychium, and some definitions include the underlying distal phalanx. These structures may be affected by heredity, skin disorders, infections, systemic disease, the aging process, internal and external medications, physical and environmental agents, trauma, and tumors, both benign and malignant.

Rationale

Scope

Nail disorders comprise approximately 10% of all dermatologic conditions. When there is an abnormality of the nail unit, the patient may have pain or interference with function, or both. Nail disorders may affect walking, the picking up of fine objects, tactile sensation, and protective function. Functional effects may result in problems wearing shoes. In many societies the aesthetic aspect of the nail unit may affect occupation, employability, and interaction with other people.

There is no significant difference in distribution of nail disorders between sexes. However, ingrown nails appear to be more common in men, particularly young athletes who may have concomitant hyperhidrosis.

Nail disorders, although infrequent in children, increase in scope throughout life and affect a high percentage of the geriatric population. This is due in part to particular susceptibility of the nail to fungal infections, faulty biomechanics from arthritis, impaired circulation, greater susceptibility to neoplasms, and the use of systemic medications.

Under certain circumstances the space beneath the nail plate, a somewhat protected area, has been shown to harbor both fungal microorganisms and the scabies mite. With respect to fungi, such a reservoir could be a source of infection elsewhere in the person, particularly cutaneous spread to the feet in cases of onychomycosis. Because of scratching, the subungual presence of scabies may reinoculate previously treated skin and result in recurrence of the infestation.

The nail unit may show specific changes that are markers for a wide range of systemic disorders. These include collagen vascular, liver, renal, endocrine, cardiac, and neoplastic diseases. In addition, a number of symptom complexes exist (e.g., nail patella and yellow nail syndromes) in which the nail unit is an integral part of multisystem diseases. Consequently, evaluation of the nails is an important component of all physical examinations.

Issue

Nail disorders respond very slowly to therapy because of the inherent slow growth of the nail unit and because of poor absorption and impaired delivery of medications to the diseased portion of the nail unit. Although there are many medical treatments currently available for the control of nail disease, often surgical techniques may be concomitantly utilized to achieve a maximum benefit. Congenital anomalies may require surgical correction.

Diagnostic criteria

Clinical

History may include

General medical history, as appropriate

Onset, duration, progression of disorders

Presence or absence at birth

Location - upper and/or lower extremities; single and/or multiple digits

Occupational and/or environmental exposures

Precipitating and/or alleviating factors

Trauma

Other cutaneous and systemic disorders

Nail cosmetics and procedures

Past and present medications and drug allergies

Past and present treatments of nail (topical, systemic, surgical)

Emotional and stress factors

For patients undergoing nail surgery, include history of

Vascular compromise

Bleeding diathesis

Medications

Diabetes mellitus

Collagen vascular disorders

Arthritis

Past infections

Past surgical procedures

Other

Physical examination

Nail cosmetics may need to be removed for adequate examination. In some instances, all 20 nails may need to be examined.

General physical examination, as appropriate

Dominant hand and changes in the proximal and lateral nailfolds

Involvement of one or more fingernails, one or more toenails, and presence or absence of bony abnormalities

Thickness, consistency, color, surface changes, onycholysis (separation of nail plate from nail bed)

Nail changes according to which component of the nail unit is involved (plate, matrix, bed, hyponychium, folds, phalanx)

Other areas including skin, hair, and mucous membranes when indicated

Particular attention should be paid to hair abnormalities, mucous membrane and dental changes, and presence or absence of immunologic disorders.

The peripheral neurovascular status of the patient when indicated

Diagnostic tests

May include, but are not limited to:

Microscopic examination

Potassium hydroxide (KOH) preparation for dermatophytes and other microbiologic organisms

Tzanck smear for viral changes

Stains for bacteria (Gram stain)

Culture

Fungal – advisable to use both cycloheximide-containing medium as well as medium without cycloheximide because yeast and nondermatophyte molds may not grow in the presence of cycloheximide

Bacterial

Viral

Nail unit compression and transillumination

In vivo nailfold capillary microscopy

Nail clippings

Hyphae

Other

Biopsy for histologic evaluation

Indications for biopsy may include diagnosis of

Medical disorder

Infection, especially fungal

Neoplasm

Structural abnormalities

Pigmentary changes

Other

Biopsy guidelines

Take the smallest amount of tissue necessary to make a definitive diagnosis.

Perform biopsy on nail bed tissue whenever possible in preference to nail matrix tissue to avoid permanent dystrophy.

Utilize the procedure with the greatest benefit and potential to minimize permanent scarring or deformity.

Punch biopsies are adequate in most situations except when operating in the proximal nail matrix, where an ellipse or fusiform biopsy is preferable and where suture may be helpful. When biopsies of the nail bed and distal nail matrix are done, suturing is not mandatory.

An adequate biopsy may require extension to the periosteum.

Biopsy stains

Hematoxylin and eosin

Special stains: periodic acid-Schiff, silver stains

Direct immunofluorescence

Other

Chemistry and serologic evaluation is indicated for

Systemic disease (thyroid, renal, pulmonary, hematopoietic, endocarditis)

Collagen vascular disease (lupus erythematosus, scleroderma, dermatomyositis, rheumatoid arthritis)

HIV infection

Syphilis

Other

Adjunctive diagnostic studies that may be indicated in certain circumstances (e.g., to rule out tumors) include

Frequently used

X ray

Rarely used

Magnetic resonance imaging

Ultrasonography

Doppler studies

Other

Inappropriate diagnostic tests

Although the use of forensic and toxicologic evaluation for vitamins and trace metals may be useful, analysis of nails for nutritional evaluation is not of proven scientific value.

Exceptions

Not applicable

Evolving diagnostic tests

Not applicable

Recommendations

Treatment

A specific diagnosis should be established before commencing therapy whenever possible. Topical, intralesional, and/or systemic therapy is indicated for many nail disorders. Other modalities such as surgery, cryotherapy, radiation, phototherapy, and laser may be indicated. Patients should be advised that treatment of nail disorders is often a prolonged and gradual process. (See Table I for indications and treatment of some of the more common nail disorders.)

Medical

Topical therapy, commonly used but not limited to

Antifungals (See "Guidelines of Care for Superficial Mycoses: Onychomycosis," J AM ACAD DERMATOL 1996;34:116-21)

Antibiotics

Antivirals

Corticosteroids

5-Fluorouracil

Salicylic acid

Tar, anthralin

α-Hydroxy acids

Chemical avulsion (potassium iodide, salicylic acid, urea)

Other

Systemic therapy includes but is not limited to

Antifungals (See "Guidelines of Care for Superficial Mycoses: Onychomycosis")

Antibiotics

Antivirals

Corticosteroids

5-Fluorouracil

Photochemotherapy

Methotrexate for severe, incapacitating psoriasis

Biotin

Other

Intralesional therapy

Intralesional corticosteroids

Intralesional bleomycin

Other

Surgical

Surgical avulsion of the total nail is not routinely recommended except for the diagnosis and management of tumors and for the alleviation of severe pain.

Specific nail surgical procedures

All nail surgical procedures should be performed under aseptic conditions

Nail plate avulsion, partial or total

Nail matrix exploration

Biopsy of one or more portions of the nail unit

Partial or total matricectomy (chemical, laser, or cold steel)

Resection of the nail bed and/or nailfolds for ingrown nails, pincer nails, and other structural abnormalities

Crescent-shaped biopsy of the proximal nailfold for periodic acid-Schiff-positive globules and direct immunofluorescence

Perforation of the nail plate for relief of subungual hematoma

Cryosurgery

Laser surgery

Other

Suggested instrumentation

Variouly sized punches

Dual action nail nipper

English anvil nail splitter

Freer septum elevator

Dental spatula and other elevators

Skin hooks

Various scalpels including Beaver instruments

Appropriate needles and syringes for anesthesia (30-gauge needle preferable)

Tourniquet, when indicated

Cryosurgical unit or cotton-tipped applicator

Other

Anesthesia (See "Guidelines of Care for Local and Regional Anesthesia in Cutaneous Surgery, J AM ACAD DERMATOL 1995;33:504-9)

A digital block or distal ring block (perionychial block) may be performed. Application of a superficial freezing spray before the initial injection is suggested. Appropriate anesthetics include lidocaine, mepivacaine, and bupivacaine.

Postoperative care

Topical and/or systemic antibiotics when indicated

Bulky loose sterile dressing (2 x 2 inch or 4 x 4 inch) and tube gauze

Non-aspirin-containing pain medications for postoperative discomfort

Avoid use of aspirin and other nonsteroidal antiinflammatory drugs

Other

Phototherapy

X-rays

Laser

Other

Specific recommendations

Onycholysis

Establish cause, if possible, and treat or correct it

Strict irritant avoidance

No nail cosmetics (except under certain circumstances)

Topical antifungals and/or drying agents as indicated

Keep the nails short

Other

Psoriasis

Avoid trauma

Keep nails short

Avoid irritants

Consider topical, systemic, and/or intralesional corticosteroids

Phototherapy

Other

Lichen planus

Avoid trauma

Keep nails short

Avoid irritants

Consider topical, systemic, and/or intralesional corticosteroids

Other

Miscellaneous

Not applicable

Table I. Indications and treatments of nail disorders*

Indication	Anti-fungal	Anti-biotics	Anti-virals	Bleo-mycin	Cortico-steroids	5-Fluor-ouracil	Salicylic acid	Tar
Brittle nails								
Herpes simplex			T,S					
Lichen planus	S				T,S,I			
Onychomycosis	T,S							
<i>Pseudomonas</i>		T,S						
<i>Staphylococcus</i>		T,S						
<i>Streptococcus</i>		T,S						
Psoriasis	T,S				T,S,I		T	T
Symptomatic nail dystrophies (structural, idiopathic)								
Warts			T	I		T	T	

Indication	An-thralin	a - Hydroxy acids	Chem-ical avul-sion	Photo-chemo-therapy	Metho-trexate	Bio-tin	Sur-gical avul-sion	Cryo-surgery
Brittle nails		T				S		
Herpes simplex								
Lichen planus								
Onychomycosis			T				X	
<i>Pseudomonas</i>			T				X	
<i>Staphylococcus</i>			T				X	
<i>Streptococcus</i>			T				X	
Psoriasis	T		T	T,S	S†		X	
Symptomatic nail dystrophies (structural, idiopathic)								
Warts								X

I, Intralesional; S, systemic; T, topical; X, surgical.

*This table presents information on treatment of the more common nail disorders and is not inclusive of the spectrum of nail disorders.

†For severe, incapacitating psoriasis.

7- Guidelines of Care for Psoriasis

Reference: 1993 by the American Academy of Dermatology, Inc.

I. Introduction

The American Academy of Dermatology's Committee on Guidelines of Care is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists.

Definition

Psoriasis is a chronic skin disease that is classically characterized by thickened, red areas of skin covered with silvery scales. The extent of skin involvement can range from discrete, localized areas to generalized body involvement. The joints, nails, and mucous membranes may also be affected with the disease.

Rationale

Scope

Psoriasis affects 1% to 2% of the general population. It has no known cure and can affect all age groups. It may be symptomatic throughout life and may be progressive with age or wax and wane in its severity. Physical and psychological disability produced by the disease may range from minor to total. In addition to classical plaque psoriasis, a severe inflammatory form of the disease, erythrodermic psoriasis, or generalized pustular psoriasis may produce involvement of the total body surface and may have associated systemic or constitutional manifestations.

Issue

Morbidity

Patients with psoriasis have a disease that may be readily apparent to others because of the redness, scaling, and thickening of the skin. This disease can cause functional impairment, disfigurement of the skin, and emotional distress. The skin may itch, burn, sting, and easily bleed in the areas affected with psoriasis. Severe involvement of the hands, feet, and nails may make even routine activities, such as walking or dressing, difficult to perform, particularly in Reiter's disease, which is considered by many to be a severely disabling variant of psoriasis. Up to 30% of patients with psoriasis may have arthritic symptoms. Five to ten percent of those patients may experience functional disability from arthritis of various joints. Persons with erythrodermic (generalized redness of the body surface) psoriasis usually have difficulty in controlling body temperature. These and other factors directly related to psoriasis may cause a person to have difficulty in work performance, problems with social rejection, sexual dysfunction, and depression.

Mortality

Erythrodermic and generalized pustular psoriasis may be life-threatening because of systemic infections or cardiovascular or pulmonary complications. Severe social rejection and morbidity associated with the disease has led to suicide.

Diagnostic criteria

Clinical characteristics

A history and cutaneous examination should be performed. The history, clinical appearance, and location of lesions are the most reliable indicators of the disease.

Skin

The amount of scale, thickness of the lesion, and degree of redness may vary depending on the area of the body involved. The classic areas of involvement are scalp, face, elbows, knees, palms and soles, intertriginous folds, and genitalia. The lesion morphology may also vary and include papules and plaques of varying sizes as well as pustules. The surface area of skin affected may range from minimal to total body involvement.

Nails

Various degrees of nail deformity may be seen including pitting, onycholysis, and subungual hyperkeratosis.

Joints

Inflammatory changes can affect small or large joints, either singly or in combination, producing a wide range of arthritic changes and joint deformity.

Ears

A moderate degree of scaling within the ear canals is common. In severe cases this may produce an impaction of scale within the ear canal causing hearing impairment.

Mucous membranes

Psoriasis frequently involves the genitalia and may involve the lips, buccal mucosa, and other mucous membranes.

Miscellaneous

Approximately 30% of patients have a family history of the disease.

Severe cradle cap or diaper dermatitis may be forerunners of psoriasis.

Infections or stress may precipitate and exacerbate the disease.

Drugs, predominantly lithium, beta blockers, antimalarials, alcohol or non-steroidal antiinflammatory agents may aggravate the disease.

Diagnostic tests

Laboratory tests may include the following:

Biopsy

Histology is frequently used to differentiate psoriasis from other conditions (see "Differential Diagnosis"). The pathology is usually, but not always, confirmatory, particularly in guttate, palmoplantar, or erythrodermic psoriasis.

Other tests

Radiologic studies

Serologic studies

Culture for appropriate organisms

Microscopic (potassium hydroxide) examinations

HLA typing

Differential diagnosis

Many diseases mimic psoriasis. These may include but are not limited to dermatitis, fungal infections, discoid and subacute lupus erythematosus, lichen planus, mycosis fungoides, pityriasis rubra pilaris, Bowen's disease, para-psoriasis, and secondary syphilis.

Inappropriate diagnostic tests

Not applicable

Exceptions

Not applicable

Evolving diagnostic tests

Not applicable

Recommendations

The majority of patients with psoriasis may be able to control their disease with treatments either prescribed or delivered in a dermatologist's office. However, a significant number of patients may require specialized treatments and experienced nursing care that would necessitate the use of an ambulatory treatment center or admission to the hospital. Some of the treatment periods may be prolonged because of the severity and extent of the disease.

Office treatment

Topical (Table I)

Emollients

Keratolytics

Corticosteroids

Tar

Anthralin

Evolving treatments

Vitamin D3 and derivatives

5-Fluorouracil

Nitrogen mustard

Cyclosporine

Nonsteroidal antiinflammatory agents

Antimicrobial agents

Other

Intralesional (Table I)

Corticosteroids

Evolving treatments

5-Fluorouracil

Cyclosporine

Other

Table I. Topical and intralesional therapy

	Effectiveness	Remission	Possible side effects*	Comments
Emollients	+	+	+A	----
Topical corticosteroids				
Mild potency	+	+	+AB	ab
Mid potency	++	+ / ++	++AB	abcd
Maximum potency	+++	++	+++AB	abcdf
Intralesional steroids	+++	+++	++C	bc
Tar	++	++	+ADE	def
Anthralin	++	++	++ADE	df
Keratolytics	+	+	+AE	cd
Other†				

Effectiveness: +, mild; ++, moderate; +++, high. Remission: +, <1 month; ++, 1-3 months; +++, >3 months. Possible side effects: +, mild; ++, moderate; +++, severe.

A, Inconvenience; B, topical corticosteroid side effects may be local and/or systemic and may include burning, irritation, itching, stinging, erythema, folliculitis, skin atrophy, telangiectasia, hypertrichosis, acneiform eruptions, hypopigmentation, perioral dermatitis, allergic contact dermatitis, maceration, secondary infection, striae, miliaria, HPA axis suppression, hyperglycemia, hyperglycuria, and manifestations of Cushing's syndrome; side effects tend to increase with increased potency; C, pain, discomfort, atrophy, telangiectasia, and hypopigmentation; D, staining; E, irritation.

Comments: a, Tachyphylaxis; b, increased risk of steroid side effects with increased potency, duration of treatment, and total dosage; c, possibility of systemic absorption may limit use in children; d, avoid eye contact and intertriginous use in children; e, increased photosensitivity; f, avoid use in body folds.

Consult Physicians' Desk Reference and drug information insert.

† Other evolving topical agents to be considered include (1) vitamin D3 and derivatives; (2) 5-fluorouracil; (3) nitrogen mustard; (4) cyclosporine; (5) nonsteroidal antiinflammatory drugs; (6) antimicrobial agents; (7) other.

Table II. Systemic, UVB/UVA phototherapy, and photochemotherapy (PUVA)

	Effectiveness and remission	Possible side effects	Patient monitoring	Clinical indications	Comments
Phototherapy UVB	+++	+A	++	All forms G	d,e,f,g
Photochemotherapy	++++	++B,F	++	All forms G	a,b,c,d,e,f

Methotrexate	++++	+++C,F	+I,II	All forms	a,b,c,e
Hydroxyurea	++	+++D,F	++II	All forms	a,b,c,e
Retinoids	+++	+++E,F	+II	All forms	a,b,c,e
Antibiotics	+++	+F	+II	All forms	b,c,e
Other*					

Effectiveness and remission: +, Partial clearing; ++, partial clearing and short remission (1 mo); +++, total clearing and short remission (1 mo); +++++, total clearing and long remission (months to years). Patient monitoring (patient and laboratory): +, Weekly to monthly; ++, monthly to every 6 mo; I, Roenigk HH Jr, Auerbach R, Maibach HI, et al. Methotrexate in psoriasis: revised guidelines. *J AM ACAD DERMATOL* 1988;19:145-56. II, *Physicians' Desk Reference* – thorough familiarization needed. Possible side effects: +, Mild; ++, moderate; +++, potentially serious, severe; A, Pruritus, sunburn leading to worsening of condition or Koebnerization, photoaging, increased incidence of skin cancer; B, Pruritus, photoaging, PUVA-induced freckles, increased risk of skin cancer cataracts; C, nausea, bone marrow depression, renal failure, ulcerative stomatitis, liver fibrosis, cirrhosis; D, bone marrow depression; E, dry nose, chapped lips, hair loss, hyperostosis, hypertriglyceridemia, hypercholesterolemia, hepatitis, cirrhosis, ocular and teratogenic changes; F, other – consult *Physicians' Desk Reference* and drug information insert. Clinical indications: G, Reduce frequency when active, inflammatory phase is over.

Comments: a, Should not be used in children except rare circumstances; b, contraindicated in pregnant patients; c, should be used with caution in female patients of childbearing potential; d, recommend periodic evaluation for skin cancer; e, may be combined with other treatment modalities; f, unsupervised UV light, including use of tanning booths is inappropriate. Likewise, home phototherapy should only be used with great caution under the direction of the patient's physician. PUVA (photochemotherapy) should not be attempted in the home unless under direct physician supervision; g, UVB alone more effective than UVA alone.

Other evolving agents to be considered include (1) cyclosporine, (2) ketoconazole, (3) vitamin D3, (4) Fumaric acid, (5) sulfasalazine, (6) other.

Phototherapy and photochemotherapy (Table II)

Ultraviolet B (UVB)

Psoralen + ultraviolet A (PUVA)

Evolving treatments

UVA/UVB combination

Other

Systemic (Table II)

Corticosteroids

In the vast majority of cases, this is not the treatment of choice because of a potential severe rebound of psoriasis and steroid side effects. Its use should be reserved for certain selected cases, disabling psoriasis, or psoriatic arthritis and is usually limited to short-term use.

Antibiotics

Useful in patients with evidence of cutaneous or systemic microbial infection, or colonization, especially in guttate psoriasis associated with streptococcal pharyngitis.

Methotrexate

Oral

Intramuscular

Hydroxyurea

Retinoids

Etretinate

Accutane

Acitretin

Evolving treatments

Cyclosporine

Vitamin D3

Sulfasalazine

Ketoconazole

Other retinoids

Fumaric acid

Fish oil supplements

Nonsteroidal antiinflammatory agents

Other

Combinations

Topical agents may be used sequentially or concomitantly with systemic agents, phototherapy, or photochemotherapy

Retinoids and phototherapy or photochemotherapy

Methotrexate and phototherapy or photochemotherapy

Other combinations

Surgical

Evolving treatments

CO2 laser surgery

Cryosurgery

Dermatome shaving

Adjunctive therapies

Antihistamines

Occlusive dressings and/or suits

Wet dressings

Antifungal creams

Localized hyperthermia

Specialized baths

Grenz ray therapy

Superficial radiation

Psychiatric counseling

Other

Patient education

This is an important part of any treatment plan and should include the effects that trauma, alcohol, infection, and stress may have on psoriasis. Expectations of the results of therapy and interactions that are possible between phototherapy and drugs and diseases are to be considered part of the patient education.

Other

Ambulatory treatment center

Ambulatory treatment centers provide specialized combination therapies such as Goeckerman and Ingram, in addition to standard office treatments for psoriasis (Section V.A.). Traditional treatment plans may vary depending on the individual center and they allow for extended periods of care under the supervision of a physician. The basic components of each treatment are listed.

Goeckerman

Four to 8 hours of care

Crude coal tar, other tars, and emollients

Occlusive dressings

Daily baths with oils, antiseptics or tar solutions

UV light treatments

Specialized dermatologic nursing care

Ingram

Anthralin in various concentrations and bases

Tar or emollient baths

UV light treatments

Specialized dressings

Specialized dermatologic nursing care

Additional services may include the following:

Specialized scalp treatments

Nail treatment

Patient education sessions

Psychological support groups

Other

Hospitalization

Criteria for admission may include the following:

Acute erythrodermic pustular, or rapidly developing psoriasis

Complications from previous therapy such as phototherapy, photochemotherapy, systemic or topical agents that would preclude their use in an outpatient setting

Coexistent illness such as difficult-to-control diabetes, hypertension, heart disease, or arthritis

Severe skin involvement with psoriasis covering more than 25% of the body surface

Severe psoriasis involving locations such as the scalp, face, hands, feet, or genitalia

Psoriasis that has not been controlled with previous outpatient therapy of at least 4 weeks

Psoriasis that is physically or emotionally disabling enough to limit the activities of daily living

8- Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis

Reference: 1996 by the American Academy of Dermatology, Inc.

I. Introduction

The American Academy of Dermatology's Guidelines/Outcomes Committee is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists. For the benefit of members of the American Academy of Dermatology who practice in countries outside the jurisdiction of the United States, the listed treatments may include agents that are not currently approved by the U.S. Food and Drug Administration.

II. Definition

"Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis" is one of six documents addressing superficial mycoses. Companion documents in this series include:

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis Versicolor

Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra

Mucocutaneous candidiasis is a mycotic infection of the skin and mucous membranes usually caused by the yeast, *Candida albicans*. However, other *Candida* species are occasionally responsible. Caution must be exercised when interpreting non *C. albicans* species. The standard terminology used in this document is cutaneous candidiasis; oral candidiasis (thrush); genital candidiasis; nail unit candidiasis; and chronic mucocutaneous candidiasis (CMCC) (Table I).

III. Rationale

A. Scope

Mucocutaneous candidiasis is a common disorder that affects all age groups, with no sex, race, or ethnic predilection. Mucocutaneous candidiasis is more common in persons who wear dentures, have diabetes mellitus, and in those immunocompromised by disease or by therapy. Most patients have disease limited to the cutaneous surfaces, especially areas of skin folds. However, immunocompromised persons may develop extensive cutaneous involvement. In some patients a serious, even life-threatening, systemic infection may develop. Systemic candidiasis is not addressed in this document. Mucous membrane involvement may be a marker for an immunocompromised state. The presence of oral candidiasis, especially in adults, may be an initial manifestation of diabetes mellitus, leukemia, lymphoma, malignancy, neutropenia, and HIV infection. Genital candidiasis may affect the vulva and vaginal area, as well as the perineal and crural folds, causing candidal intertrigo. Candidiasis may also affect the nail unit, particularly the nail plate and paronychia area.

B. Issue

Candida yeasts are part of the normal flora of the skin, mouth, intestinal tract, and vagina. Multiple intrinsic and extrinsic factors contribute to the development of clinical infection, called candidiasis. Candidiasis is endemic, and when host conditions are favorable, there may be person-to-person transmission. Although numerous systemic and topical therapies are available, unless predisposing factors are corrected, relapses and recurrences are common. Untreated, mucocutaneous candidiasis may become chronic and cause significant disability.

Some cases may resolve spontaneously, or on removal of predisposing factors, such as return to an immunocompetent state or discontinuation of antibiotic therapy. However, recurrences are common.

Risk factors for mucocutaneous candidiasis may include:

1. Cutaneous candidiasis

- a. Diabetes mellitus
- b. Tropical environment
- c. Obesity
- d. Use of systemic corticosteroids or antibiotic therapy
- e. Neutropenia
- f. HIV infection
- g. Other immunocompromised states
- h. Occlusion (e.g., diapers, casts, dressings)
- i. Diseases which disturb the integument (e.g., psoriasis, contact dermatitis)
- j. Other

2. Oral candidiasis

- a. Use of broad-spectrum antibiotics; systemic, topical, and inhalational corticosteroids; and cytotoxic drugs
- b. Radiation therapy
- c. HIV infection
- d. Other immunocompromised states
- e. Age (i.e., infants and elderly)
- f. Occlusion (e.g., dentures)
- g. Other

3. Genital candidiasis

- a. Women
 - 1) Use of broad-spectrum antibiotics and oral contraceptives
 - 2) Pregnancy
 - 3) Low vaginal pH
 - 4) Diabetes mellitus
 - 5) HIV infection
 - 6) Other immunocompromised states
 - 7) Poor hygiene
 - 8) Infected sexual partner

9) Other

b. Men

- 1) Uncircumcised penis
- 2) Maceration
- 3) Incontinence
- 4) Diabetes mellitus
- 5) HIV infection
- 6) Other immunocompromised states
- 7) Poor hygiene
- 8) Infected sexual partner
- 9) Other

c. Infants and children

- 1) Occlusion (e.g., diapers)
- 2) HIV infection
- 3) Other immunocompromised states
- 4) Other

4. Nail unit candidiasis

a. Prolonged exposure to water

- 1) Dish washers
- 2) Bartenders
- 3) Beauticians
- 4) Health care providers
- 5) Other

b. Occlusion

- 1) Artificial nails
- 2) Prolonged use of gloves
- 3) Other

c. Trauma to the nailfolds and cuticles

- 1) Nail biting
- 2) Manicure
- 3) Occupationally caused
- 4) Other

d. Other

5. Chronic mucocutaneous candidiasis (CMCC)

CMCC occurs as an autosomal recessive trait that appears in childhood and has several recognized clinical presentations. Despite the widespread or generalized skin and mucosal involvement with *Candida*, patients rarely have systemic or disseminated disease. Associated findings include alopecia, vitiligo, malabsorption disorders, and endocrine dysfunction. Infection with other microorganisms including *Staphylococcus*, *Streptococcus*, and *Haemophilus* species are quite common in these syndromes.

6. Other

IV. Diagnostic criteria

A. Clinical

1. History may include

a. General medical history, especially but not limited to:

- 1) History of weight loss or weight gain
- 2) Endocrine - diabetes mellitus
- 3) Risk factors for HIV disease
- 4) Presence of other risk factors (See III.B.)
- 5) Use of systemic medications

6) Other

b. Sexual practices

c. Recurrent infections

d. Duration of condition

e. Current treatment(s) topical and systemic of

- 1) Mucocutaneous candidiasis
- 2) Other disease

f. Past treatment(s) topical and systemic of

- 1) Mucocutaneous candidiasis
- 2) Other disease

g. Family history of diabetes mellitus and/or mucocutaneous candidiasis

h. Occupation

i. Dermatophytic infection, particularly tinea pedis, tinea cruris

j. Drug allergies

k. Other

2. Physical examination may include

a. General physical examination as indicated

b. Examination of the involved area, but special attention may be directed to

- 1) Skin folds
- 2) Diaper area
- 3) Corners of mouth and mucous membranes (e.g., perlèche)
- 4) Interdigital spaces
- 5) Scrotum, glans penis and foreskin, crural folds, gluteal area
- 6) Perianal area
- 7) Vagina, vulva
- 8) Axillae
- 9) Nail unit
- 10) Other

c. Extent of involvement

d. Clinical appearance (Table I)

e. Associated findings

- 1) Secondary bacterial infection
 - 2) Postinflammatory hyperpigmentation and hypopigmentation
 - 3) Onychodystrophy and onycholysis
 - 4) Excoriations
 - 5) Other
- f. Other

Table I. Location and presentation of mucocutaneous candidiasis infection

Terminology	Location	Presentation
Cutaneous	Intertriginous areas/skin folds	Inflammation, often with satellite and/or follicular pustules
Oral (thrush)	Oral mucosa including tongue, lips, gingiva, palate, buccal areas, pharynx	White patches; erosive and atrophic lesions may also occur; white confluent pseudomembrane resembling cottage cheese or milk curds may cover an erosive or denuded base
Genital	Vaginal mucous membranes Glans penis, prepuce may extend to scrotum	Pruritus, erythema, and often a creamy white discharge Erythema; occasionally vesicles, pustules
Nail unit	Proximal nailfold nail plate	Erythema; edema and a painful or purulent discharge; usually caused by <i>C. albicans</i> : onychodystrophy may result

Chronic mucocutaneous candidiasis	Skin, nails, mucous membranes	A heterogeneous group of disorders consisting of chronic <i>Candida</i> infections, usually by <i>C. albicans</i> , associated with variable aberrations in cell-mediated immunity and endocrinopathies; includes <i>C. granuloma</i>
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B. Diagnostic tests

After review of the patient history and physical examination, the diagnosis can often be established. Laboratory tests may confirm the diagnosis of mucocutaneous candidiasis. This verification is especially important when the use of systemic therapy is anticipated. Simple, inexpensive tests that can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include but are not limited to:

1. Potassium hydroxide preparation (KOH)

Material is obtained from the site of infection. If the lesion is a pustule, the purulent material can be used for the specimen. In cases of cutaneous involvement, the specimen can be obtained from the edge of a lesion. The material is placed on a glass slide and 10% to 20% KOH is added with or without dimethyl sulfoxide. A fungal stain, such as Chlorazol Black E, or Parker's blue black ink may be used to highlight the pseudohyphae. The presence of pseudohyphae and yeast forms confirm infection.

2. Other stains

Other stains may also be used to identify the yeast forms or pseudohyphae of *Candida*. These stains include, but are not limited to

- a. Gram stain
- b. Polychromatic multiple stain (PMS)
- c. Other

3. Fungal culture

Candida species are yeasts rather than molds and therefore, grow as yeast colonies on Sabouraud's glucose agar. The addition of cycloheximide to Sabouraud's agar may inhibit many species of *Candida* and other saprophytes. However, *C. Albicans* will grow on media containing cycloheximide. Examples of such media include Mycosel, Mycobiotic, and dermatophyte test medium. Although *C. albicans* will grow on dermatophyte test media, the agar will not exhibit a red color change as occurs with dermatophyte growth if read at the appropriate time interval, as indicated by the manufacturer's instructions.

4. Studies for differential diagnosis may include

- a. Bacterial culture to evaluate for secondary infection
- b. zank smear or viral cultures to evaluate for herpes virus infection
- c. Wood's light examination to evaluate for erythrasma, especially in intertriginous areas
- d. Skin biopsy to differentiate candidiasis from other dermatoses
- e. Other

5. Laboratory tests to evaluate risk factors, if applicable, may include:

- a. Endocrine tests

- 1) Blood glucose level
- 2) Thyroid function studies
- 3) Other
- b. Complete blood cell count
- c. HIV
- d. Other
- C. Inappropriate diagnostic tests
 1. Routine allergy testing
 2. Hair analysis
 3. Other
- D. Exceptions
- Not applicable
- E. Evolving diagnostic tests
- Not applicable
- V. Recommendations
 - A. Treatment
 1. Medical (Table II)
 - a. Alteration or improvement of cutaneous environment
 - 1) Avoid occlusion
 - 2) Promote dryness
 - 3) Promote good hygiene
 - 4) Other
 - b. Cutaneous, genital, and nail unit candidiasis
 - 1) Topical antifungal products include, but are not limited to
 - a) Imidazole/triazoles
 - (1) Clotrimazole
 - (2) Econazole
 - (3) Ketoconazole
 - (4) Miconazole
 - (5) Oxiconazole
 - (6) Sulconazole
 - (7) Terconazole (vaginal candidiasis only)

(8) Other

b) Ciclopirox olamine

c) Polyene antibiotics

(1) Nystatin

(2) Topical amphotericin B lotion

(3) Other

d) Topical corticosteroids may be used sparingly for short periods in conjunction with topical and/or systemic antifungals to reduce the inflammatory component.

e) Other topical agents

(1) Drying agents

(2) Powders

(3) Gentian violet

(4) Castellani's paint

(5) Potassium permanganate compresses

(6) Iodochlorhydroxyquin

(7) Other

f) Evolving

(1) Naftifine

(2) Terbinafine

(3) Other

2) Systemic therapy

Occasionally indicated, especially in widespread disease, recalcitrant disease, and immunocompromised patients

a) Fluconazole

b) Ketoconazole

c) Evolving oral antifungals

(1) Terbinafine (cutaneous candidiasis only)

(2) Itraconazole

(3) Other

d) Other

c. Oral or esophageal candidiasis

1) Topical antifungal products include, but are not limited to

a) Clotrimazole troches

b) Nystatin suspensions

c) Other

2) Systemic therapy

a) Fluconazole

b) Ketoconazole

c) Evolving oral antifungals

(1) Itraconazole

(2) Other

d) Other

d. Chronic mucocutaneous candidiasis

Although topical agents may be useful as adjunctive therapy, this disease is most commonly treated with systemic agents.

1) Systemic therapy may include the following:

a) Ketoconazole

b) Fluconazole

c) Other

2) Evolving systemic therapy

a) Terbinafine

b) Itraconazole

c) Other

e. Other

Treatment of gastrointestinal tract colonization with agents such as oral nystatin may be helpful in preventing recurrences and as an adjunct to other treatment.

2. Surgical

Not applicable

3. Other

a. Treatment of sexual partner(s)

Because genital infections may be sexually transmitted, partners should be examined and treated appropriately.

b. Other

B. Miscellaneous

1. Follow-up

Follow-up examinations may be indicated, depending on extent, severity, and tolerance to medications, as well as the need to augment or alternate treatment based on clinical

response. Intervals between visits will vary, depending on, but not limited to the severity of the problem and the intensity of the treatment.

2. Monitoring of patients receiving systemic therapy

Periodic monitoring of hepatic, renal, and hematopoietic function may be indicated in patients treated with systemic antifungals.

3. Drug interactions

Oral antifungals have the potential for significant drug interactions and toxicities. The package insert and the *Physicians' Desk Reference (PDR)* should be consulted.

4. Contraindications and precautions for use of systemic antifungal therapy

a. Hypersensitivity to medication

b. Precautions (see package insert and the PDR)

c. Other

Table II. Medications for candidiasis

	Cutaneous	Oral	Genital	Nail Unit	CMCC
Clotrimazole	X	4	X	X	X
Econazole	X		X	X	X
Fluconazole	2	2	2	2,3	X
Itraconazole	3	3	3	2,3	3
Ketoconazole					
Topical	X		X	X	X
Oral	2	2	2	2	X
Miconazole	X		X	X	X
Oxiconazole	X		X	X	X
Sulconazole	X		X	X	X
Terconazole			X		
Naftifine	X		X	X	X
Terbinafine					
Topical	X		X	X	X
Oral	2,3				3

Ciclopirox olamine	X		X	X	X
Nystatin	X	X	X	X	X
Amphotericin B lotion	X		X	X	X
Topical corticosteroids	1		1	1	
Drying agents	X	X	X	X	X
Powders	X		X	X	X
Other	X	X	X	X	X

NOTES: Consult *Physicians' Desk Reference* or package insert. X, Indication; 1, topical corticosteroids may be used sparingly for short periods in conjunction with topical or systemic antifungals to reduce the inflammatory component; 2, oral antifungals may be indicated to severe disease, recalcitrant disease, and in immunocompromised patients; 3, evolving oral therapy; 4, troches.

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Reference: 1996 by the American Academy of Dermatology, Inc.

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Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis Versicolor Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra Onychomycosis is a general infection of the nail unit by fungal microorganisms, including dermatophytes, yeasts, and nondermatophyte molds. Tinea unguium refers to dermatophytic infection of the nail unit. Onychomycosis can occur in toenails as well as fingernails. The presentation includes onycholysis (separation of nail plate from nail bed), subungual debris or hyperkeratosis, discoloration of the nail plate, partial or complete destruction of the nail plate, and thickening of the nail plate. There are different clinical presentations of onychomycosis: distal subungual onychomycosis, proximal subungual onychomycosis, superficial white onychomycosis, and infection of the entire nail plate by *Candida albicans* in patients with chronic mucocutaneous candidiasis.

III. Rationale

A. Scope

Onychomycosis represents approximately 50% of all nail disorders. Onychomycosis is far more common on the toenail than it is on the fingernail. It may be associated with trauma to the nail and is commonly seen in association with tinea pedis. In fingernails, paronychia infections are common and are often caused by *C. albicans*. This may predispose patients to subsequent invasion of the remainder of the nail plate. Patients with diabetes mellitus have an increased susceptibility to candidal infections of the nails. Both sexes appear to be equally affected. Onychomycosis may occur at any age, but is unusual prior to puberty.

B. Issue

Onychomycosis is a common disorder that can result in significant morbidity. Clean, healthy nails are important in our society and dystrophic nails can be a social impediment and cause significant embarrassment, thereby affecting a patient's self-esteem. In addition, thickened nails can be painful, interfere with the function of the nail unit, affect the ability to use the hands and fingers, and cause pain on walking.

Onychomycosis occurs in association with trauma, systemic disease, and immunocompromised states including HIV infection. Toenail involvement may also be related to hyperhidrosis. There may be two subpopulations of persons who are infected with fungal microorganisms. In one group, recurrence occurs frequently regardless of therapy. These patients may have an impaired immune system that makes them predisposed to fungal infections and more difficult to treat. The other group has no apparent inherent defects in their

immune system. These patients do not usually get recurrences and can be treated more successfully. There may be an increased incidence of onychomycosis in the elderly population. Many elderly patients are unable to cut their thickened onychomycotic nails because of poor vision or arthralgia and rely on others to complete this task.

IV. Diagnostic criteria

A. Clinical

1. History may include

a. General medical condition, especially but not limited to:

- 1) Hepatic disease
- 2) Renal disease
- 3) Immunocompromised state
- 4) Endocrine disease – diabetes mellitus
- 5) Use of systemic medications
- 6) Other

b. Recurrent infections

c. Duration of condition

d. Current treatment(s) topical and systemic of

- 1) Onychomycosis
- 2) Other diseases

e. Past treatment(s) topical and systemic of

- 1) Onychomycosis
- 2) Other diseases

f. Family history of onychomycosis, tinea unguium, tinea pedis

g. Other skin disorders, especially but not limited to atopy, personal or familial

h. Other dermatophyte infections

i. Chronic urticaria

j. Other

2. Physical examination may include

a. General physical examination as indicated

b. Location

- 1) Fingernails
- 2) Toenails

c. Extent of involvement and the portion of the nail unit involved in the process (Examine all 20 nails.)

1) Distal subungual onychomycosis – the most common onychomycosis. The fungus invades the distal nail bed in the area of the hyponychium. Hyperkeratosis of the nail bed develops, which eventuates in onycholysis and finally, in thickening and discoloration of the nail plate.

2) Proximal subungual onychomycosis

The organisms invade under the cuticle and infect the proximal nail bed.

3) Superficial white onychomycosis

The fungal organisms directly invade the nail plate surface, which results in a crumbly nail plate surface.

4) Chronic mucocutaneous candidiasis

C. albicans can invade the entire nail plate. This is part of a syndrome and patients have other features in addition to nail disease. Candidal paronychia and onycholysis may also occur.

d. Associated findings

1) Inflammation

2) Tenderness

3) Thickening of the nail plate

4) Thickness of the nail bed

5) Presence of tinea pedis or other dermatophytic infections

6) Other

e. Other

B. Diagnostic tests

After review of the patient's history and physical examination, the diagnosis can often be established. Greater diagnostic accuracy occurs if the clinical diagnosis is verified by laboratory tests. This verification is especially important when the use of systemic therapy is anticipated. Simple, inexpensive tests that can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include, but are not limited to:

1. Potassium hydroxide preparation (KOH)

Material for the KOH examination is obtained from the infected part of the nail unit. In distal subungual onychomycosis, the hyperkeratotic, subungual debris at the most proximal area of the infected nail unit provides the ideal specimen. It is best obtained by débriding the thickened dystrophic nail as far back as possible. In the area of the nail bed, most proximal to the cuticle, debris can be removed with a small serrated curette. In circumstances in which it is difficult to get the subungual debris, the infected part of the nail plate can also be obtained for scale. In distal subungual onychomycosis, the undersurface of the nail plate can be scraped and used for KOH examination. However, the yield may not be as high. In superficial white onychomycosis, the infected nail plate surface can be scraped with a 15-gauge blade and this material used for the KOH preparation. In proximal subungual onychomycosis, scrape with a 15-gauge blade and remove healthy nail plate and culture-infected nail bed. The material is placed on a glass slide and 10% to 15% KOH is added. The addition of dimethyl sulfoxide to KOH solution is often helpful because of the thickness of the debris. A fungal stain, such as Chlorazol Black E, which is chitin specific, or Parker's blue black ink may enhance visualization of hyphae.

2. Fungal culture

The standard fungal culture medium is Sabouraud's glucose agar. The addition of an antibiotic, such as chloramphenicol, inhibits bacterial overgrowth that may inhibit the growth of pathogenic dermatophytes or nondermatophyte molds. In addition, cycloheximide can be added to the Sabouraud glucose medium. The addition of this antibiotic will inhibit the growth of many nondermatophyte saprophytes and permit the growth of dermatophytes. Cycloheximide is used in situations in which a dermatophyte is the suspected pathogen.

Accuracy may be increased by using two or more media. The second medium should be used to select out dermatophyte fungi. Examples include dermatophyte test medium, Mycosel, and Mycobiotic. Dermatophyte test media have a color indicator which changes the medium from yellow to red in the presence of a dermatophyte. However, the color reaction will obscure the features used to identify the colony morphology of many organisms. For those media in which a color change indicates the presence of dermatophytes, it is important to follow the manufacturer's instructions in terms of the number of days between culture inoculation and reading.

3. Nail clippings for histologic analysis

4. Nail biopsy may be considered to establish the diagnosis when other tests are negative.

5. Other

C. Inappropriate diagnostic tests

Not applicable

D. Exceptions

Not applicable

E. Evolving diagnostic tests

Not applicable

V. Recommendations

A. Treatment

It should be explained to the patient that topical therapy alone may not be successful in eradicating distal subungual onychomycosis. Systemic therapy for fingernails may require as long as 6 months and systemic therapy for toenails as long as 12 to 18 months. Patients may require more than one course of treatment because of recurrence or reinfection. Treatment decisions must be made on the individual patient and on the expectations and needs of the patient.

1. Medical

a. Topical therapy

1) Imidazoles

a) Clotrimazole

b) Econazole

c) Ketoconazole

d) Miconazole

e) Oxiconazole

f) Sulconazole

g) Other

2) Allylamines

a) Naftifine

b) Terbinafine

c) Other

3) Ciclopirox olamine

4) Polyene antibiotics

a) Amphotericin B

b) Natamycin

c) Nystatin

d) Other

5) Miscellaneous

a) Benzoic acid preparations (Whitfield's ointment)

b) Haloprogin

c) Tolnaftate

d) Other

6) Evolving topical therapy

a) Bifonazole/urea

b) Butenafine gel

c) Ciclopirox olamine lacquer

d) Tioconazole lacquer

e) Other

7) Other

b. Systemic therapy includes but is not limited to

1) Griseofulvin

2) Ketoconazole

3) Itraconazole

4) Terbinafine

5) Fluconazole

6) Other

c. Specific treatment

1) Distal subungual onychomycosis

a) Dermatophyte

If a dermatophyte is isolated, it is considered the pathogen. Systemic therapy includes griseofulvin or ketoconazole. Terbinafine, itraconazole, and fluconazole are evolving therapies that can also be used. Systemic therapy should rarely be given unless the diagnosis of onychomycosis has been confirmed by a KOH preparation, fungal culture, or nail biopsy. Both griseofulvin and ketoconazole require continuation of therapy until the nail unit becomes clinically normal.

b) *Candida albicans*

If *C. albicans* is cultured from the nail unit, it may be pathogenic. Other *Candida* species could be contaminants, and reculture may be indicated. In nail candidiasis, oral fluconazole, ketoconazole, and itraconazole (evolving) can be effective. Systemic griseofulvin is ineffective in *C. albicans* nail disease. Topical therapy may be effective adjuvant therapy, particularly if the proximal nailfold is infected.

c) Nondermatophyte molds

Nondermatophytes are seldom primary nail invaders. Exceptions include *Scopulariopsis brevicaulis*, *Scytalidium dimidiatum* (*Hendersonula toruloidea*), and *Scytalidium hyalinum*. At the present time, there is no effective oral agent for nondermatophytic fungal nail disease. Itraconazole can be effective in some instances of nail invasion with *Aspergillus* species.

2) Superficial white onychomycosis

This can be caused by *T. mentagrophytes* and several nondermatophyte molds including *Aspergillus terreus*, *Fusarium oxysporum*, and *Acremonium* species. Because this is an infection of the superficial nail plate surface, topical antifungals combined with surgical curettage or scraping of the infected portions of the nail plate may be effective.

2. Surgical

a. Surgical removal of the nail

1) Rarely used as a therapy

2) May be indicated depending on the degree of patient pain and discomfort.

b. Surgical curettage or scraping

May be effective in combination with topical antifungals in white superficial onychomycosis.

3. Other

Depending on the patient's age and under certain conditions, no therapy may be indicated for onychomycosis.

B. Miscellaneous

1. Follow-up

The nail should be trimmed back and the nail beds should be débrided.

a. Fingernail fungal infections - approximately once a month

b. Toenail infections - approximately every 6 to 8 weeks

2. Monitoring of patients receiving systemic therapy

Periodic monitoring of hepatic, renal, and hematopoietic function may be indicated in patients treated with systemic antifungals.

3. Drug interactions

Oral antifungals have the potential for significant drug interactions and toxicities. The package insert and the *Physicians' Desk Reference (PDR)* should be consulted.

4. Contraindications and precautions for use of systemic antifungal therapy

- a. Hypersensitivity to medication
- b. Precautions (See package insert and the PDR)
- c. Other

Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra

Reference: 1996 by the American Academy of Dermatology, Inc.

I. Introduction

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Definition

"Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra" is one of six documents addressing superficial mycoses. Companion documents in this series include:

Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis Versicolor

Piedra is the presence of superficial hair shaft nodules secondary to an infection of hair shafts with either *Piedraia hortae* (black piedra) or *Trichosporon beigelii* (white piedra).

Rationale

Scope

Piedra has a worldwide distribution. Black piedra occurs more frequently in tropical countries and is common in certain tropical areas of central South American and Southeast Asia. Its occurrence is uncommon in the United States. Black piedra affects mainly scalp hair and, less commonly, beard, mustache, and pubic hair. White piedra occurs in semitropical and temperate countries. All age groups and both sexes are affected. Familial spread may also occur. White piedra affects mainly beard, axillary, and perineal hairs, and less frequently, scalp hair. Eyelash and eyebrow involvement can occur.

Issue

If untreated, black piedra may persist for years. Removal of affected hair by clipping or shaving is curative, but may be unacceptable for many patients, particularly women. Once treated, recurrences are not common.

Genital white piedra may be due to a synergistic infection by *Coryneform* bacteria and *T. beigelii*, which colonize normal perigenital skin. Reinfection frequently occurs. Treatment of white piedra remains controversial. Eradication may be difficult despite laboratory evidence of sensitivity of the organism to antifungals. Disseminated *T. beigelii* infections can occur, especially in immunocompromised patients, and may lead to purpuric or necrotic cutaneous papules and nodules.

Diagnostic criteria

Clinical

History may include the following:

General medical condition, especially, but not limited to

Immunocompromised state

Other

Recurrent infections

Duration of condition

Current treatment(s), topical and systemic, of

Piedra

Other disease

Past treatment(s), topical and systemic, of

Piedra

Other disease

Other skin disorders

Drug allergies

Travel to regions where infection is endemic

Other

Physical examination may include

General physical examination as indicated

Location of involved hairs

Scalp

Pubic area

Mustache

Beard

Eyelashes, eyebrows

Axillae

Extent of involvement

Associated findings

Black piedra

Hard, black hair shaft nodules, firmly attached

Hair breakage, rarely

Normal surrounding skin

Other

White piedra

Soft, white, reddish green or light brown hair shaft nodules, loosely attached

Hair breakage

Normal surrounding skin

Other

Diagnostic tests

After review of the patient history and physical examination, the diagnosis can often be established. Greater diagnostic accuracy occurs if the clinical diagnosis is confirmed by laboratory tests. Simple inexpensive tests than can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include, but are not limited to:

Potassium hydroxide preparation (KOH)

Obtain material from hair shaft nodules. The material is placed on a glass slide and 10% to 15% KOH is added with or without dimethyl sulfoxide. A fungal stain, such as Chlorazol Black E, or Parker's blue black ink may be added to highlight the hyphae. A positive result shows:

Black piedra

Tightly packed pigmented hyphae, asci, and ascospores attached to hair shaft

White piedra

Loosely arranged hyphae, blastoconidia, arthroconidia attached to hair shaft

Fungal culture

Culture on Sabouraud's dextrose agar. *T. beigellii* is inhibited by cycloheximide, which is found in dermatophyte test medium (DTM), Mycosel, and Mycobiotic.

Other

Inappropriate diagnostic tests

Not applicable

Exceptions

Not applicable

Evolving diagnostic tests

Not applicable

Recommendations

Treatment

Medical

Treatment of choice for both black and white piedra is to remove the infected hairs.

Shave hair

Cut/clip hair

White piedra

Topical antifungal products include, but are not limited to:

Imidazoles

Ciclopirox olamine

2% Selenium sulfide

6% Precipitated sulfur in petrolatum

Chlorhexidine solution

Castellani's paint

Zinc pyrithione

Amphotericin B lotion

2% to 20% Glutaraldehyde

Other

Black piedra

Terbinafine (oral)

Other

Surgical

Not applicable

Other

Miscellaneous

Not applicable

Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis (tinea) Versicolor

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I. Introduction

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Definition

"Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis (Tinea) Versicolor" is one of six documents addressing superficial mycoses. Companion documents in this series include:

Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra

Pityriasis (tinea) versicolor is a superficial infection of the stratum corneum by the yeast *Malassezia furfur* (syn. *Pityrosporum orbiculare*). This yeast is part of the normal cutaneous flora. Pityriasis (tinea) versicolor is characterized by hyperpigmented and hypopigmented scaly patches, primarily on the trunk and proximal extremities.

Rationale

Scope

Pityriasis (tinea) versicolor is a common disorder that affects people of all age groups, but is most commonly seen in adults. Infants and children can also be affected, but often have an atypical presentation. This disease is typically worse in geographic areas with tropical ambient temperatures. Multiple factors are known to contribute to its pathogenesis.

Issue

Involvement of the cutaneous surface can occasionally be extensive, leading to emotional distress because of appearance. Symptoms vary from none to severe pruritus. Although numerous therapies are available, recurrences frequently occur after treatment, especially in tropical climates.

Diagnostic criteria

Clinical

History may include the following:

General medical condition, especially if use of oral antifungals is considered, may include the following:

Hepatic disease

Renal disease

Endocrine disease - diabetes mellitus

Use of systemic medications

Other

Duration, progression to point of maximal severity

Seasonal variation

Current treatment(s), topical and systemic, of

Pityriasis versicolor

Other diseases

Past treatment(s), topical and systemic, or

Pityriasis versicolor

Other diseases

Other skin disorders, especially, but not limited to, the following;

Atopy, personal or familial (because of occasional irritation to topical antifungal agents)

Seborrheic dermatitis

Drug allergies

Habitual use of heavy oils on skin

Other

Physical examination may include the following:

General physical examination as indicated

Location

Anterior aspect of the chest

Back

Extremities

Face, neck (more common in children)

Clinical appearance

Hyperpigmented lesions

Hypopigmented lesions

Erythematous lesions

Extent of involvement

Gradation

Mild

Moderate

Severe

Associated findings

Postinflammatory hyperpigmentation and hypopigmentation

Pruritus

Excoriations

Other

Other

Diagnostic tests

After review of the patient history and physical examination, the diagnosis can often be established. Greater diagnostic accuracy occurs if the clinical diagnosis is verified by laboratory tests. This verification is especially important when the use of systemic therapy is anticipated. Simple, inexpensive tests that can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include, but are not limited to, the following:

Potassium hydroxide preparation (KOH)

Scale from the affected area is placed on a glass slide, and 10% to 15% KOH is added with or without dimethyl sulfoxide (DMSO). If DMSO is included, gentle heating is generally not necessary. A fungal stain such as Chlorazol Black E, or Parker's blue-black ink may be added to highlight the hyphae and yeast cells. A confirmatory KOH preparation would reveal short, stubby hyphae and yeast cells. Patients may have a predominance of either.

Wood's light examination to demonstrate extent of involvement

Other stains

Other stains may be used to identify the hyphae and yeast cells. These stains include, but are not limited to, the following:

Paragon multiple stain

Other

Studies for differential diagnosis may include the following:

Fungal culture to exclude other mycoses *M. furfur* does not grow on routine agars without growth supplements and is, therefore, not routinely cultured.

Skin biopsy to differentiate pityriasis versicolor from other dermatoses

Other

Other

Inappropriate diagnostic tests

Routine allergy testing

Exceptions

Not applicable

Evolving diagnostic tests

Not applicable

Recommendations

Treatment

Topical treatment alone may be indicated for most patients. Systemic treatment may be indicated for persons with extensive involvement, with recurrent infections, and in whom topical agents as sole therapy have failed. Systemic therapy may be used with or without topical agents or may be used alone in patients intolerant to topical treatment.

Medical

Topical antifungal products include, but are not limited to, the following:

Imidazoles

Ciclopirox olamine

Miscellaneous

Selenium sulfide shampoos, lotions

Zinc pyrithione shampoos

Sulfur preparations

Salicylic acid preparations

Propylene glycol lotions

Benzoyl peroxide

Other

Other

Systemic therapy (see V.A. above)

Ketoconazole

Evolving

Fluconazole

Itraconazole

Other

Other

Surgical

Not applicable

Other

Miscellaneous

Follow-up

Follow-up examinations may be indicated, depending on extent, severity, and tolerance to medications, as well as the need to augment or alternate treatment on the basis of clinical response. Intervals between visits will vary, depending on, but not limited to, the severity of the problem and the intensity of the treatment.

Monitoring of patients receiving systemic therapy

Periodic monitoring of hepatic, renal, and hematopoietic function may be indicated in patients treated with systemic antifungals.

Drug interactions

Oral antifungals have the potential for significant drug interactions and toxicities. The package insert and the *Physician's Desk Reference (PDR)* should be consulted.

Contraindications and precautions for use of systemic antifungal therapy

Hypersensitivity to medication

Precautions (see package insert and the *PDR*)

Other

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae

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II. Definition

"Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae" is one of six documents addressing superficial mycoses. Companion documents in this series include:

Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis
 Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis Versicolor

Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra

Tinea capitis is a mycotic infection by dermatophyte fungi involving scalp hair follicles and adjacent skin. Tinea capitis is caused by select species in the genera *Microsporum* and *Trichophyton*. A variety of inflammatory and non-inflammatory lesions, often with associated alopecia, may be present. There are three recognized patterns: ectothrix, endothrix, and favus.

In ectothrix tinea capitis, hyphae fragment into arthroconidia (spores) outside the hair shaft. This eventually ends in cuticle destruction. There are inflammatory and non-inflammatory varieties of ectothrix tinea capitis. The non-inflammatory variety is often referred to as "gray patch," and the inflammatory variety, which may resemble bacterial furunculosis, is referred to as kerion.

In endothrix tinea capitis, arthroconidia (spores) occur within the hair shaft and cuticle destruction does not occur. There are also non-inflammatory and inflammatory varieties of endothrix tinea capitis. The non-inflammatory type is often referred to as "black dot" because of hairs breaking close to the scalp, leaving a "black dot" appearance on scalp skin.

Favus rarely occurs in the United States but is more common in other countries, particularly Eastern Europe and parts of Asia. This type is characterized by arthroconidia (spores), air spaces, and fragmented hyphae within the hair shaft.

Tinea barbae is similar to tinea capitis, but affects the hairs and follicles of beard and mustache areas. It is often caused by zoophilic dermatophytes.

III. Rationale

A. Scope

Tinea capitis has a worldwide distribution. The most common organisms in the United States are *Trichophyton tonsurans* and *Microsporum canis*. *T. tonsurans* generally causes an endothrix pattern of hair invasion and can result in inflammatory and non-inflammatory varieties. *M. canis* is more common in Europe and parts of South America. Currently, more than 90% of tinea capitis in urban areas of the United States is the endothrix variety produced by *T. tonsurans*. Worldwide, tinea capitis primarily affects preadolescent children. Adults are affected much less commonly. The infection may spread by person-to-person contact or by fomites. Some children and adults may be asymptomatic carriers and contribute to spread of infection. Tinea barbae is caused primarily by zoophilic dermatophytes. It is more common in rural areas.

B. Issue

Tinea capitis may be misdiagnosed as alopecia areata, trichotillomania, bacterial furunculosis, seborrheic dermatitis, psoriasis, telogen effluvium, and atopic or seborrheic dermatitis. In untreated cases, severe scarring with permanent hair loss may result. This has psychosocial ramifications and may affect the social adjustment and self-image of the child. Early diagnosis and appropriate therapy are therefore critical in preventing scarring. In general, systemic antifungal therapy is necessary in both tinea capitis and tinea barbae. Once treated, recurrences are uncommon. However, an asymptomatic carrier state may occur in some patients. This may contribute to the increased prevalence of some infections. Therefore in some instances, it is justified to treat asymptomatic adults and adolescent children who are exposed to the infected child.

IV. Diagnostic criteria

A. Clinical

1. History may include the following:

a. General medical condition, especially, but not limited to, the following:

- 1) Hepatic disease
- 2) Renal disease
- 3) Immunocompromised state
- 4) Use of systemic medications
- 5) Other

b. Tinea capitis in school or day care

c. Duration of condition

d. Current treatment(s), topical and systemic, of

- 1) Tinea capitis and tinea barbae
- 2) Other diseases

e. Past treatment(s), topical and systemic, or

- 1) Tinea capitis and tinea barbae
- 2) Other diseases

f. Other skin disorders, especially, but not limited to, atopy, personal or familial (because of occasional irritation to topical antifungal preparations)

g. Family history of tinea capitis and/or other dermatophytoses

h. Contact with infected animals

i. Contact with other persons with tinea capitis

j. History of shared combs, brushes, or hats

k. Recent travel

l. Other

2. Physical examination may include the following:

a. General physical examination as indicated

b. Location

1) Scalp

2) Beard area

3) Mustache area

c. Extent of involvement

d. Type

1) Inflammatory

a) Hair

Usually localized or diffuse loss, with remaining hairs loose and matted; extensive hair breakage

b) Scalp

Single or multiple erythematous plaques with follicular papules, nodules; crusting

2) Minimal inflammation or non-inflammatory

a) Hair

Patchy angular or round areas of loss; hairs become dull-gray; broken hairs and "black dots" (swollen hair shafts on scalp surface after hair has broken off)

b) Scalp

Rare papules or pustules, often referred to as kerion; can also present with severe crusting and resemble a pyoderma of the scalp

3) Favus

a) Hair

Minimal to extensive patchy loss; cup-shaped yellow crusts (scutula) surround hairs; matting

b) Scalp

Erythema, initially perifollicular; yellow crust

e. Associated findings

1) Hair loss

2) Secondary bacterial infection

3) Lymphadenopathy

4) Dermatophytid allergic reaction

5) Dermatophytosis on other body sites

6) Other

f. Other

B. Diagnostic tests

After review of the patient history and physical examination, the diagnosis can often be established. Greater diagnostic accuracy occurs if the clinical diagnosis is verified by laboratory tests. This verification is especially important when the use of systemic therapy is anticipated. Simple, inexpensive tests that can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include, but are not limited to, the following:

1. Potassium hydroxide preparation(KOH)

Scalp scrapings and epilated hairs and "black dots" are all materials that can be examined. The material is placed on a glass slide and 10% to 15% KOH is added with or without dimethyl sulfoxide (DMSO). A fungal stain, such as Chlorazol Black E, or Parker's blue black ink may be added to highlight the hyphae. Fungal elements are seen within the hair shaft (endothrix and favus) or on the outside of the cuticle (ectothrix).

2. Fungal culture

Several hairs from an infected area may be obtained for fungal culture. Because dermatophytes are the primary cause of tinea capitis and tinea barbae, media containing cycloheximide is helpful because it will selectively screen for dermatophyte growth. Appropriate agar choices include dermatophyte test medium, Mycosel, and Mycobiotic. Dermatophyte test media have a color indicator, which changes the medium from yellow to red in the presence of a dermatophyte. It is important to follow the manufacturer's instructions in terms of the number of days between culture inoculation and reading.

3. Wood's light examination

4. Studies for differential diagnosis may include

a. Bacterial culture to rule out secondary infection

b. Biopsy when necessary, e.g., if above diagnostic steps, in conjunction with clinical findings, fail to yield a diagnosis

c. Other

5. Other

C. Inappropriate diagnostic tests

1. Routine allergy testing

2. Other

D. Exceptions

Not applicable

E. Evolving diagnostic tests

Not applicable

V. Recommendations

A. Treatment

1. Medical

Oral antifungal agents are generally required for curing both tinea capitis and tinea barbae. Topical products may be used as adjunctive therapy in selected patients.

a. Systemic therapy includes, but is not limited to, the following:

- 1) Griseofulvin
- 2) Ketoconazole
- 3) Evolving systemic therapy
 - a) Fluconazole
 - b) Itraconazole
 - c) Terbinafine
 - d) Other
- 4) Miscellaneous adjunctive therapy
 - a) Corticosteroids (both intralesional and short-term oral corticosteroids may be beneficial in selected patients)
 - b) Systemic antibiotics may be indicated if a secondary bacterial infection is present.
- 5) Other
- b. Topical therapy (usually used as adjunct therapy)
 - 1) Removal of scale by gentle shampoos or compresses
 - 2) Medicated shampoos containing selenium sulfide, ketoconazole, or povidone in selected patients
 - 3) Topical antifungals may also be applied to the patient's scalp and may include the following:
 - a) Imidazoles
 - (1) Clotrimazole
 - (2) Ketoconazole
 - (3) Miconazole
 - (4) Oxiconazole
 - (5) Sulconazole
 - (6) Other
 - b) Allylamines
 - (1) Naftifine
 - (2) Terbinafine
 - (3) Other
 - c) Ciclopirox olamine
 - d) Miscellaneous
 - (1) Drying agents
 - (2) Powders
 - (3) Antibiotics (when applicable)

(4) Salicylic acid and other keratolytic agents

(5) Combination topical corticosteroid antifungal mixtures can be used but may need restrictions on duration of therapy. Caution must be exercised when using combination products that contain potent fluorinated corticosteroids.

e) Other

4) When *T. tonsurans* is cultured, family members may be treated with one of the following antifungal shampoos even if they are asymptomatic, because of the asymptomatic carrier state.

a) Ketoconazole shampoo

b) Selenium sulfide shampoo

c) Povidone-iodine

d) Other

2. Surgical

Surgical approaches to both tinea capitis and tinea barbae are rarely, if ever, indicated. Incision and drainage of kerion is not recommended.

3. Other

a. Clean environment and fomites.

b. Avoid sharing of brushes, combs, or hats.

c. Culturing family members and treatment with oral antifungal therapy may be indicated.

d. Search out infected pet and/or other animal carrier and recommend appropriate treatment

e. Shampoo scalp daily

f. Other

B. Miscellaneous

1. Keeping children out of school after starting therapy is controversial and may not be required for most infections.

2. Follow-up

Follow-up examinations may be indicated, depending on extent, severity, and tolerance to medications, as well as the need to augment or alternate treatment on the basis of the clinical response. Intervals between visits will vary, depending on, but not limited to, the severity of the problem and the intensity of the treatment.

3. Monitoring of patients receiving systemic therapy

Periodic monitoring of hepatic, renal, and hematopoietic function may be indicated in patients treated with systemic antifungals.

4. Drug interactions

Oral antifungals have the potential for significant drug interactions and toxicities. The package insert and the *Physicians' Desk Reference (PDR)* should be consulted.

5. Contraindications and precautions for use of systemic antifungal therapy

a. Hypersensitivity to medication

b. Precautions (see package insert and the *PDR*)

c. Other

Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis

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II. Definition

"Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Corporis, Tinea Cruris, Tinea Faciei, Tinea Manuum, and Tinea Pedis" is one of six documents addressing superficial mycoses. Companion documents in this series include:

Guidelines of Care for Superficial Mycotic Infections of the Skin: Mucocutaneous Candidiasis
Guidelines of Care for Superficial Mycotic Infections of the Skin: Tinea Capitis and Tinea Barbae

Guidelines of Care for Superficial Mycotic Infections of the Skin: Onychomycosis

Guidelines of Care for Superficial Mycotic Infections of the Skin: Pityriasis Versicolor

Guidelines of Care for Superficial Mycotic Infections of the Skin: Piedra

Tinea corporis, tinea cruris, tinea faciei, tinea manuum, and tinea pedis are fungal infections of the stratum corneum caused by certain species of the genera *Epidermophyton*, *Microsporum*, and *Trichophyton*.

The standard terminology used in this document is as follows:

Tinea corporis

Dermatophytosis of the glabrous skin of the trunk and extremities, characterized by both inflammatory and non-inflammatory lesions

Tinea cruris

Dermatophytosis of the proximal medial thighs and buttocks, characterized by inflammatory and non-inflammatory lesions often with invasion of hair follicles; cutaneous candidiasis in this region can mimic dermatophytic infection, but is usually associated with scrotal lesions.

Tinea faciei

Dermatophytosis of the non-beard areas of the face, characterized by inflammatory and non-inflammatory lesions

Tinea manuum

Dermatophytosis of the interdigital and palmar surfaces of one or both palms; the differential diagnosis includes infection caused by nondermatophyte fungi such as *Scytalidium hyalinum* and *Scytalidium dimidiatum* (*Hendersonula toruloidea*)

Tinea pedis

Dermatophytosis of the plantar surface and interdigital spaces of the foot, characterized by both inflammatory and non-inflammatory lesions; the differential diagnosis includes infection by nondermatophyte fungi such as *S. hyalinum* and *S. dimidiatum* (*H. toruloidea*)

III. Rationale

A. Scope

Ten percent to 20% of the population is estimated to be infected by a dermatophyte. Of these infections, tinea pedis is the most common, occurring in up to 70% of adults. The most common organisms in the United States are *T. rubrum*, *T. mentagrophytes*, and *T. tonsurans*. *T. verrucosum*, *M. canis*, *M. gypseum*, and *E. floccosum* also cause numerous infections. Dermatophytoses can be acquired from other people (anthropophilic), animals (zoophilic), and soil (geophilic). The most common source in the United States is infected people. *Trichophyton rubrum* is the most common dermatophyte both in the United States and the world and causes the majority of nonscalp skin infections. Peak prevalence of

dermatophytosis occurs after puberty. This is especially the case for tinea pedis, tinea manuum, and tinea cruris. Tinea corporis and tinea faciei may occur in the preadolescent period. Risk factors include contact with infected animals such as cats and cattle. Another risk factor for infection is the occurrence of anthropophilic tinea capitis, which may produce tinea corporis and tinea pedis in family members and persons in contact with the patient. Dermatophytes invade, infect, and persist in the stratum corneum and rarely penetrate below the surface of the epidermis or its appendages. The skin responds to the superficial infection by increased proliferation, which eventuates in scale and epidermal thickening.

B. Issue

When infection is caused by zoophilic organisms, the infection may spontaneously resolve after a period of pronounced inflammation. This is best demonstrated by *Trichophyton verrucosum* infections of the glabrous skin acquired by contact with infected cattle. The host response to the infection determines the physical changes on the skin. When dermatophytosis is caused by an anthropophilic dermatophyte, the lesion on the skin tends to be more chronic, persistent, and recalcitrant to therapy. This is especially true in tinea pedis caused by *T. rubrum*, which may persist for many years. After topical or systemic therapy, recurrence occurs in up to 70% of patients. Infections often require prolonged therapy with systemic and topical agents. Recurrence is caused partly by reinfection and the failure to eradicate the original infection. The persistence of infective fungal elements (spores) on the skin is probably a major factor in recurrence. Another issue with recurrence is incomplete eradication because patients may stop applying topical therapy when their symptoms are alleviated. With some infections the presence of a reservoir, such as the nail, may also explain frequent relapse and recurrence after a cessation of therapy. Dermatophytoses affect the quality of life. This is especially true for those who are HIV positive or otherwise immunocompromised because significant symptoms of pruritus or pain generally occur with infection. Subtle changes of dermatophytosis on the skin may allow an early diagnosis of HIV infection. Tinea pedis and tinea cruris are a major cause of symptomatic complaints including pruritus, pain, and in some instances disability. If untreated, tinea pedis may be complicated by onychomycosis. Dermatophytosis has been a problem in war time as combat troops have been disabled because of severe tinea pedis. Dermatophytosis can mimic many cutaneous diseases including psoriasis, parapsoriasis, eczema, and candidiasis. Because dermatophyte infections tend to be chronic, the annual cost for treatment in the United States exceeds \$400 million. It is important to diagnose infection as early and as accurately as possible.

IV. Diagnostic criteria

A. Clinical

1. History may include

a. General medical condition, especially, but not limited to

- 1) Hepatic disease
- 2) Renal disease
- 3) Immunocompromised state
- 4) Endocrine disease - diabetes mellitus
- 5) Use of systemic medications

6) Other

b. Previous occurrences

c. Duration of condition

d. Current treatment(s), topical and systemic of

- 1) Dermatophyte infections
- 2) Other disease
- e. Past treatment(s), topical and systemic of
 - 1) Dermatophyte infections
 - 2) Other disease
- f. Other skin disorders, especially atopy and contact sensitivity
- g. Occupational exposure
 - 1) Farm worker
 - 2) Zookeeper
 - 3) Laboratory worker
 - 4) Veterinarian
 - 5) Other
- h. Environmental and recreational exposure
 - 1) Gardening
 - 2) Contact sports
 - 3) Use of sports facilities
 - 4) Animals
 - 5) Other
- i. Drug allergies
- j. Chronic urticaria
- k. Other
2. Physical examination may include
 - a. General physical examination as indicated
 - b. Location
 - c. Extent of involvement
 - d. Type of lesions
 - 1) Non-inflammatory scaly
 - 2) Acute or subacute eczematous-like
 - 3) Chronic lichenified
 - 4) Nodular or granulomatous
 - 5) Bullous
 - 6) Pustular or resembling pyoderma

e. Associated findings

- 1) Infection involving the hair follicle and nail
- 2) Persistent hyperpigmentation and/or hypopigmentation
- 3) Secondary bacterial infection
- 4) Other

f. Other

B. Diagnostic tests

After review of the patient history and physical examination, the diagnosis can often be established. Greater diagnostic accuracy occurs if the clinical diagnosis is verified by laboratory tests. This verification is especially important when the use of systemic therapy is anticipated. Simple inexpensive tests that can be performed in the physician's office at the time of the patient visit may yield immediate results. Such tests include, but are not limited to the following:

1. Potassium hydroxide preparation (KOH)

Scale is obtained from the site of infection. The active border or the edge of a lesion is suitable for obtaining scale. In blistering lesions, the roof of the vesicle is an appropriate specimen. In pustular lesions, the purulent debris is appropriate. The material is placed on a glass slide and 10% to 15% KOH is added with or without dimethyl sulfoxide (DMSO). If DMSO is added to the KOH, heating is generally not necessary. A fungal stain such as Chlorazol Black E or Parker's blue black ink may be added to highlight the hyphae. A positive KOH will show numerous septate hyphae. A note whether the hyphae are nonpigmented or pigmented can be made because certain nondermatophyte infections (e.g., *Exophiala werneckii*) have pigmented hyphae.

2. Fungal culture

The standard fungal culture medium is Sabouraud's glucose agar. The addition of an antibiotic, such as chloramphenicol, inhibits bacterial overgrowth that may inhibit the growth of pathogenic dermatophytes or nondermatophyte molds. Media containing cycloheximide are useful when selectively screening for dermatophytes. Appropriate agar choices include dermatophyte test medium (DTM), Mycosel, and Mycobiotic. DTMs have a color indicator that changes the medium from yellow to red in the presence of a dermatophyte. However, the color reaction will obscure the features used to identify the colony morphology of many organisms. For those media in which a color change indicates the presence of dermatophytes, it is important to follow the manufacturer's instructions in terms of the number of days between culture inoculation and reading.

3. When a nondermatophyte mold is a possible pathogen, as may occur in certain cases of tinea pedis or tinea manuum, media that do not contain cycloheximide are useful. For example, *S. dimidiatum* and *S. hyalinum* can be causative pathogens in tinea pedis and tinea manuum. These organisms generally do not grow on media containing cycloheximide; therefore the use of noncycloheximide media can be helpful in these circumstances.

4. Studies for differential diagnosis may include the following:

- a. Bacterial culture to rule out secondary infection
- b. Wood's light examination to rule out erythrasma, especially in intertriginous disease and involvement of the scrotum
- c. Skin biopsy to differentiate a dermatophyte infection from other dermatoses
- d. Other

C. Inappropriate diagnostic tests

1. Routine allergy testing

2. Other

D. Exceptions

Not applicable

E. Evolving diagnostic tests

Not applicable

V. Recommendations

A. Treatment

1. Medical

a. Topical therapy

Topical treatment alone may be indicated for the following types of dermatophytoses: non-inflammatory tinea corporis, tinea cruris, tinea faciei, tinea manuum, and tinea pedis. Topical antifungal products include, but are not limited to, the following:

1) Imidazoles

a) Clotrimazole

b) Econazole

c) Ketoconazole

d) Miconazole

e) Oxiconazole

f) Sulconazole

g) Other

2) Allylamines

a) Naftifine

b) Terbinafine

c) Other

3) Ciclopirox olamine

4) Miscellaneous

a) Benzoic acid preparations (Whitfield's ointment)

b) Tolnaftate

c) Haloprogin

d) Drying agents

e) Powders

- f) Antibiotics (where applicable)
- g) Salicylic acid and other keratolytic agents
- h) Other

5) Combination topical corticosteroid antifungal mixtures can be used, but may need restrictions on duration of therapy and location of application. Caution must be exercised in the use of combination products, which contain potent fluorinated corticosteroids.

- 6) Other

b. Systemic therapy

Inflammatory dermatophytoses may require systemic antifungal therapy. Oral therapy may be required to treat hyperkeratotic areas as on the palms and soles, patients with disabling and/or extensive disease, patients intolerant to topical therapy or those for whom topical therapy has failed, patients with chronic infection, and patients immunosuppressed by disease or by therapy. Systemic therapy includes, but is not limited to, the following:

- 1) Griseofulvin
- 2) Ketoconazole
- 3) Evolving systemic therapy
 - a) Terbinafine
 - b) Itraconazole
 - c) Fluconazole
 - d) Other
- 4) Other

2. Surgical

Usually not indicated, except for drainage of superficial vesicles, bullae, and pustules

3. Other

- a. Elimination of risk factors such as avoidance of infected animals, soil, and people
- b. Preventive measures such as wearing protective footwear in public facilities
- c. If applicable, appropriate treatment of infected animals
- d. Other

B. Miscellaneous

1. Follow-up

Follow-up examinations may be indicated, depending on extent, severity, and tolerance to medications, as well as the need to augment or alternate treatment based on clinical response. Intervals between visits will vary, depending on, but not limited to, the severity of the problem and the intensity of the treatment.

2. Monitoring of patients receiving systemic therapy

Periodic monitoring of hepatic, renal, and hematopoietic function may be indicated in patients treated with systemic antifungals.

3. Drug interactions

Oral antifungals have the potential for significant drug interactions and toxicities. The package insert and the *Physicians' Desk Reference (PDR)* should be consulted.

4. Contraindications and precautions for use of systemic antifungal therapy

- a. Hypersensitivity to medication
- b. Precautions (see package insert and the *PDR*)
- c. Other

9- Guidelines of Care for Vitiligo

Reference: 1996 by the American Academy of Dermatology, Inc.

I. Introduction

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II. Definition

Vitiligo is a disfiguring medical disease of unknown origin that causes destruction of melanocytes in the skin, mucous membranes, eyes, inner ear, and occasionally in hair bulbs. The loss of melanocytes alters both structure and function of these organs and results in the absence of pigment.

III. Rationale

A. Scope

Vitiligo is a disease affecting about 1% of the world's population including 1 million to 2 million persons within the United States. It affects persons of all ethnic origins and both sexes. Although vitiligo can begin on any part of the integument, the first manifestations are loss of pigment (white spots) most commonly on the hands, feet, arms, face, and lips. Forty percent of patients have ocular pigmentary abnormalities and 5% will note some loss of visual acuity, poor night vision, or photophobia. The disease is usually progressive. Spontaneous repigmentation may occur; however, it is usually minor.

Involvement of exposed areas, such as the skin of the face or hands, and of the external genitalia often causes serious emotional stress. Studies document that at least two thirds of patients with vitiligo significantly underachieve their potential because of the psychosocial difficulties resulting from the disfigurement. Studies document that depigmented skin is biologically altered by the loss of melanocytes. Depigmented skin has a diminished immune/inflammatory response to a variety of noxious chemicals and other inflammatory stimuli.

B. Issue

The primary goal of therapy is to restore melanocytes to the skin. Such repigmented skin regains its normal immune/inflammatory functions. For those patients in whom vitiligo is so widespread that repigmentation is biologically not possible, an important goal of therapy is to improve the appearance of these patients so they can function normally in society. The best therapy for such patients may be to remove the small remaining blotches of normal pigmentation.

The physician who treats patients with vitiligo should have:

1. An understanding of melanocytes, their biology, and behavior, and
2. A thorough knowledge of the following:
 - a. Pharmacology of drugs used for treatment
 - b. Principles of phototherapy
 - c. Inherent risks and expected outcome of treatment

IV. Diagnostic criteria

A. Clinical

In most instances the diagnosis of vitiligo is based on the history and physical examination.

1. History may include

- a. General medical history, as appropriate
- b. Age at onset of depigmentation
- c. Course of the disease - stability, rate of progression
- d. Inflammation, irritation, or rash preceding the pigment loss
- e. Potential precipitating events including emotional stress, physical illness, sunburn, or other forms of cutaneous trauma occurring within 2 to 3 months before the onset of pigment loss
- f. History of photosensitivity
- g. Ocular or auditory dysfunction or inflammation
- h. Previous forms of therapy including dosage, effects, and/or toxicity
- i. Occupational hazards and hobbies that might cause chemically induced vitiligo
- j. Family history of vitiligo and early graying of the hair (i.e., significant loss of hair color before the age of 30 years)
- k. Personal or family history of associated conditions (e.g., thyroid disorders, premature graying, alopecia areata, diabetes mellitus, collagen vascular diseases, pernicious anemia, and/or Addison's disease)
- l. Emotional stress to the patient resulting from the pigment loss
- m. Other

2. Physical examination may include

- a. General physical examination as appropriate
- b. Acquired asymptomatic depigmented macules or patches usually without clinical signs of inflammation
- c. Hypopigmented lesions and/or trichrome macules or patches
- d. Occasional inflamed borders around depigmented lesions
- e. Initial lesions seen on the hands, forearms, feet, face, and lips
- f. The approximate surface area involved by depigmentation
- g. Patterns of vitiligo
 - 1) Generalized - symmetric macules or patches in a random distribution over much of the body
 - 2) Acral/acrofacial - depigmented macules or patches confined to the extremities and/or face
 - 3) Focal/localized - isolated macules or patches on one or two body sites
 - 4) Segmental - lesions restricted to one portion of the body such as one leg, one portion of the trunk or the face; lesions rarely if ever distributed in a dermatomal pattern
- h. Other important physical findings
 - 1) Acquired depigmented hairs within vitiliginous patches
 - 2) Poliosis of scalp hair, eyelashes, eyebrows, and/or beard hair

3) Pigmentary changes in the choroid and retinal pigment epithelium

4) Uveitis

i. For patients with fair skin, such as those with Fitzpatrick skin types I and II, detection of hypopigmented or depigmented patches of vitiligo may require the use of a Wood's lamp to delineate the areas of involvement. In patients with darker skin, a Wood's lamp examination can be helpful to assess the degree of hypopigmentation or depigmentation in individual lesions.

j. Baseline ophthalmologic examination if patients are to undergo photochemotherapy (see Drake et al. "Guidelines of Care for Phototherapy and Photochemotherapy")

k. Other

B. Diagnostic tests

1. In some instances diagnostic tests may be indicated to differentiate vitiligo from conditions that may mimic it clinically. These conditions may include but are not limited to:

- a. Cutaneous T-cell lymphoma
- b. Discoid or systemic lupus erythematosus
- c. Hansen's disease
- d. Late pinta
- e. Nevus anemius
- f. Nevus depigmentosus
- g. Piebaldism
- h. Pityriasis alba
- i. Postinflammatory depigmentation/hypopigmentation
- j. Sarcoidosis
- k. Scleroderma
- l. Tinea versicolor
- m. Other

2. Laboratory tests that may be helpful to detect the presence of associated systemic disorders may include but are not limited to tests for

- a. Adrenal insufficiency
- b. Diabetes mellitus
- c. Pernicious anemia
- d. Thyroid disease
- e. Other

3. One or more of the following tests may be useful:

a. Biopsy from the border stained with Fontana-Masson technique (for melanin) to differentiate vitiligo from some of the aforementioned conditions

b. If patients are to undergo photochemotherapy (see Drake et al. "Guidelines of Care for Phototherapy and Photochemotherapy"), consider baseline

- 1) Antinuclear antibody test
- 2) Liver and renal function tests

c. Other

C. Inappropriate diagnostic tests

- 1. Serum α -melanocyte-stimulating hormone levels
- 2. Serum corticotropin levels
- 3. Hair analyses
- 4. Trace metal analyses

D. Exceptions

Not applicable

E. Evolving diagnostic tests

Not applicable

V. Recommendations

A. Treatment

Treatment of vitiligo is important to restore the normal appearance, morphology, and function of the skin. Because melanocytes are indolent and slow to respond to current methods of therapy, therapy is often continued for 6 to 12 months to obtain an optimal response.

1. Medical

a. Psoralen photochemotherapy

Oral or topical psoralen photochemotherapy (or both) are the most efficacious treatments presently available in the United States (see Drake et al. "Guidelines of Care for Phototherapy and Photochemotherapy").

1) Mechanism

The reservoir for melanocytes that migrate into the depigmented skin comes from contiguous pigmented skin (melanocytes migrate about 2 to 3 mm into the depigmented skin) and from the hair follicle, the most important reservoir. Because of the absence of reservoirs, hair-bearing skin such as that on the forearms or legs in which the terminal (not vellus) hairs are totally depigmented are less likely to respond to medical treatments as is glabrous skin such as that on the palms, fingertips, and dorsum of the feet. The risks of PUVA must be weighed against small gains achievable when treating these latter types of skin.

2) Topical psoralen photochemotherapy

Home use of topical PUVA therapy is not appropriate. Topical psoralen photochemotherapy is often considered for patients with limited involvement (<20% of the body surface) or for children older than 5 years of age with localized patches of vitiligo.

a) In special circumstances, dermatologists with special expertise may use topical photochemotherapy for children younger than 5 years of age.

b) Oxsoralen lotion is usually diluted in ethanol, petrolatum, or hydrophilic petrolatum (Aquaphor) to a 0.01% to 0.1% concentration. Test doses of various concentrations may be performed before therapy is initiated.

c) The preparation is applied to the vitiliginous skin 15 to 30 minutes before UVA exposure. The initial UVA dose is usually 0.12 to 0.25 J/cm² and is increased to achieve mild erythema, usually in increments of 0.12 or 0.25 J/cm² weekly according to the patient's skin type.

d) Treatments are usually given one to three times per week, but not on two consecutive days.

e) A broad-spectrum sunscreen is applied to treated skin before leaving the physician's office, and patients are encouraged to wear appropriate clothing for sun protection

f) Outside activities must be avoided because of the high potential of the development of severe phototoxic reactions.

g) Photographs may assist in evaluating progress.

3) Oral psoralen photochemotherapy (PUVA)

Oral photochemotherapy is used for patients with more extensive vitiligo or for persons recalcitrant to topical therapy. Oral psoralens are not usually recommended for children younger than 12 years of age.

- a) Maximal repigmentation occurs with the new form of 8-methoxypsoralen (Oxsoralen-Ultra) given in a dose of 0.2 to 0.4 mg/kg. The dose must be individualized for each patient. The newer form should not be used interchangeably with the older form (Oxsoralen).
- b) The drug is ingested 1 to 1½ hours before UVA exposure.
- c) The usual initial UVA exposure is 1 to 2 J/cm².
- d) Subsequent treatment may be increased, usually in increments up to 1 J/cm², until moderate asymptomatic erythema is observed (a pinkness present on white skin).
- e) Treatments are given two to three times weekly, but never on two consecutive days.
- f) The dose of UVA is adjusted depending on the individual patient's response to treatment.
- g) For patients who do not tolerate 0.3 mg/kg, the dose of psoralen is decreased to 0.15 mg/kg.
- h) Patients must wear UVA-blocking glasses whenever using sunlight for illumination, from the time of exposure to psoralen until sunset that day, and are encouraged to wear UVA-blocking glasses when exposed to sunlight on the following day.
- i) After treatment, patients should avoid unnecessary exposure to sunlight and are encouraged to wear appropriate clothing for sun protection and to use a sunscreen on exposed areas.
- j) Genitalia are generally not treated except in special circumstances.
- k) Photographs may assist in evaluating progress.

4) Heliotherapy

Trisoralen and sunlight (heliotherapy) is a form of photochemotherapy.

- a) Trisoralen is prescribed initially at a dose of 0.3 mg/kg with initial sun exposure up to 15 minutes, ideally at the same time each day.
- b) The drug is ingested 2 to 4 hours before exposure to sunlight.
- c) Subsequent exposures are increased in increments of 5 minutes per treatment until the skin becomes moderately erythematous. The dose of Trisoralen and duration of sun exposure are monitored and adjusted on an individual basis depending on degree of erythematous response.
- d) Treatments are given two to three times weekly, but not on two consecutive days.
- e) Patients must wear UVA-blocking glasses whenever using sunlight for illumination, from the time of exposure to Trisoralen until sunset that day, and are encouraged to wear UVA-blocking glasses when exposed to sunlight the following day.
- f) After treatment, patients should avoid unnecessary exposure to sunlight for the remainder of the day and are encouraged to wear appropriate clothing for sun protection and to use a sunscreen on exposed areas.
- g) It is generally not recommended that patients with vitiligo of the genitalia be treated in this area except in special circumstances.
- h) Photographs may assist in evaluating progress.

5) UVA equipment

See Drake et al. "Guidelines of Care for Phototherapy and Photochemotherapy"

6) Side effects

See Drake et al. "Guidelines of Care for Phototherapy and Photochemotherapy"

b. Topical corticosteroid therapy

Topical corticosteroids are sometimes effective repigmenting agents. Optimal success of treatment with topical corticosteroids requires applications for 3 to 4 months or longer. Mid- or lower potency corticosteroids may be preferable to avoid the toxicity associated with long-term applications of high-potency corticosteroids. Lower potency topical corticosteroids should be considered in young children younger than 2 years of age who are not candidates for topical PUVA.

- 1) Corticosteroid cream is applied to depigmented skin once daily for 3 to 4 months.
- 2) The response is monitored with Wood's lamp examination at 6-week intervals.
- 3) Therapy is continued if repigmentation occurs, but stopped if there is no evidence of response after 3 months.
- 4) Photographs may assist in evaluating progress.
- 5) Possible side effects that require discontinuance of the medication include
- 6) Capillary telangiectases
- 7) Epidermal atrophy
- 8) Striae
- 9) Other

Depigmentation

1) Indications

Patients with vitiligo that is so extensive that repigmentation is highly unlikely

Patients who have more than 50% involvement of the skin and have demonstrated therapeutic resistance to efforts at repigmentation

Patients with disfiguring vitiligo on the face who are not desirous or willing to attempt repigmentation

Monobenzone is applied twice daily to areas with unwanted pigmentation until complete depigmentation is achieved. A trial on a small area is recommended before larger areas are treated.

Possible side effects include

Dermatitis (usually responds to topical application of corticosteroids)

Pruritus

Xerosis

Conjunctival melanosis

Corneal pigmentation

Distal depigmentation - limited or widespread

Other

Considerations/precautions

Patients must be instructed to avoid direct skin-to-skin contact with others for 1 to 2 hours after application.

Depigmentation is permanent and irreversible, resulting in permanent photosensitivity. Sun protection measures should be employed. Although patients on occasion do regain some areas of color and repigmentation, this is not to be expected.

This form of therapy should be considered only under the most special circumstances for children younger than 12 years of age.

Most patients choosing depigmentation require time and assistance in making the decision to be permanently depigmented. It is helpful to involve spouses, parents, or other family members in making these decisions. Although the final cosmetic result will be excellent, repigmentation is usually preferable if success is possible.

Other

Adjunctive therapy

1) Sun protection measures

Appropriate clothing for sun protection should be worn and broad-spectrum sunscreens should be applied to all exposed skin (both pigmented and depigmented) to prevent photodamage.

2) Cosmetic camouflage

Cosmetic camouflage with stains or makeup is acceptable to some patients, particularly in persons with limited involvement in exposed areas.

Supportive care

Patients often require supportive care that can be offered by the dermatologist. For some patients referral to a psychiatrist or psychologist may be appropriate.

4) Other

Other

Surgical

Autologous skin grafts

Small (2 to 4 mm), round, full-thickness grafts are harvested with a trephine from a pigmented site and transplanted into an area of vitiligo. Undesirable effects include scarring, a cobblestone-like texture, incomplete, or spotty repigmentation, koebnerization at the donor site, and, less often, infection.

b. Autologous epidermal grafts

The donor epidermis is separated from the underlying dermis by suction alone or suction and heat. The roof of the induced blister contains viable melanocytes. The recipient bed is created in a similar manner or by freezing with liquid nitrogen. The recipient blister roof is discarded and the donor blister roof is placed on the recipient defect. By the use of transplanted epidermis alone, the risk of scarring decreases significantly. Undesirable effects include incomplete or spotty repigmentation, donor site koebnerization, and infection.

c. Autologous epidermal grafts and PUVA

Autologous epidermal grafts are transplanted as described above. After healing is complete, PUVA therapy is introduced. The PUVA induces melanocyte migration away from the grafts and into the surrounding vitiliginous skin.

d. Micropigmentation (tattooing)

Micropigmentation involves the tattooing of vitiliginous skin in an attempt to match the surrounding normally pigmented skin. Iron oxide pigment injected into the dermis is most often utilized. An exact match of pigment is difficult to obtain.

e. Other

Evolving

Dermabrasion and topical 5-fluorouracil

Vitiliginous skin is superficially dermabraded and 5% 5-fluorouracil is applied twice daily for 7 to 10 days. Complete, but darker, repigmentation may result. Undesirable effects include long recovery period, infection, scarring, and aggravation of vitiligo.

b. Autologous, in vitro cultured, melanocytes grafts

Melanocytes are harvested from a small fragment of pigmented skin from the patient. The melanocytes are introduced into tissue culture and their number expanded. The cultured cells are transplanted onto vitiliginous skin.

c. Khellin and ultraviolet A (UVA)

Khellin is a furanochromone. Combined with the UVA it is reported to be as effective as PUVA therapy in the treatment of vitiligo without the phototoxicity associated with the psoralens. An oral dose of 50 to 100 mg is given 2½ hours before UVA exposure. UVA dosage depends on patient's skin type and ranges from 5 to 15 J/cm². Recent studies reported elevation in liver enzymes in patients receiving topical or oral khellin.

d. L-Phenylalanine and UVA

Reports vary about the response of vitiligo to L-phenylalanine and UVA.

L-Phenylalanine, 50 mg/kg, is given orally 30 minutes to 1 hour before UVA exposure.

The dosage of UVA radiation varies from 2 to 12 J/cm², depending on the patient's skin type. Contraindications for this therapy include phenylketonuria, impaired liver and kidney function, malignant skin disease, pregnancy, breast-feeding, history of arsenic exposure, prior radiotherapy, and autoimmune disorders.

Other

Other

Many reported cures for vitiligo are extant. Data regarding efficacy, toxicity, safety in pregnancy, chemistry, pharmacology, and other factors are not available.

Miscellaneous

Patient education

Education of patients regarding the disease, its usual course, and the risks and expected outcome of treatment are essential components of good dermatologic treatment. The National Vitiligo Association, P.O. Box 6337, Tyler, TX 75711 (telephone: (903)534-2925) provides patient education materials.

10- Guidelines of Care for Warts: Human Papillomavirus

Reference: 1995 by the American Academy of Dermatology, Inc.

I. Introduction

The American Academy of Dermatology's Committee on Guidelines of Care is developing guidelines of care for our profession. The development of guidelines will promote the continued delivery of quality care and assist those outside our profession in understanding the complexities and scope of care provided by dermatologists. For the benefit of members of the American Academy of Dermatology who practice in countries outside the jurisdiction of the United States, the listed treatments may include agents that are not currently approved by the U.S. Food and Drug Administration.

II. Definition

Warts are benign tumors that commonly involve the skin and other epithelial tissues. The etiologic agents for these infections are a class of double-stranded DNA viruses called papillomaviruses. Warts are generally classified by their clinical features and morphology (e.g., common, flat, filiform) or by location (e.g., genital, plantar, respiratory papillomatosis).

III. Rationale

A. Scope

Human papillomaviruses (HPV) infect individuals of all ages. Clinical lesions are most common in children and young adults, with an estimated incidence of 10%. Although the prevalence of HPV in the adult population is not known, various diagnostic techniques, including serology and DNA hybridization, suggest that exposure to the virus and subclinical and latent infection may be very common. Infection occurs as a result of person-to-person spread, including that of sexual transmission, vertical transmission, and from exposure to virus in the environment. In fact, HPV infection is now the most common sexually transmitted disease. In the past 20 years, according to figures from the Centers for Disease Control and Prevention, there has been a sixfold increase in the number of visits to physicians' offices for treatment of anogenital warts. Genital warts in children may or may not be associated with sexual abuse, particularly in those 3 years old and younger, because vertical transmission is a distinct possibility. There has also been an increase of HPV infection among immunocompromised patients (e.g., transplant patients and in patients who test positive for HIV. Patients with kidney transplants have a 40% prevalence rate of HPV.).

B. Issue

Although some warts spontaneously regress, warts in some adults and in those who are immunocompromised can persist for years. They can be a source of physical discomfort and psychologic trauma, as well as contagion. Some HPV types, such as 16, 18, and 31, have been associated with certain types of carcinoma involving the reproductive tract, the skin, mouth, pharynx, and larynx, which makes HPV infection an even greater concern to patients and physicians. Their oncogenic potential is an

important issue. The role of HPV in directly inducing malignant tumors is unclear. Cofactors possibly play a role in this process; however, there is active investigation in this area.

There is clear evidence that HPV can exist in a subclinical state - viral protein and infectious particles are present - but significant change in the skin surface is not appreciated with the naked eye. Magnification along with the application of 5% acetic acid may be useful in visualizing this state. Therefore treating only clinically abnormal-appearing skin does not necessarily treat the field of viral particles around each lesion. Latent infection also exists in which viral DNA is present in tissue but where complete virus particles are not assembled. Recognition of this state is only possible using certain diagnostic techniques (e.g., DNA hybridization or polymerase chain reactions (see Section IV.B.)). The degree to which contagion is possible in latent and subclinical infection is not known. It would appear that the immune system plays a significant role in the ultimate expression of HPV. It is possible that many individuals may never be cured of this virus but may "express" at various times the spectrum of HPV from clinically obvious lesions to latent infection. Whether it is possible to eradicate the virus completely is unresolved. The issue does not primarily lie with the otherwise healthy individual with warts on the finger, but rather with the patient who has HPV of the genital area and/or mucous membranes, as well as with the immunocompromised patient.

IV. Diagnostic criteria

A. Clinical

1. History may include the following:

a) Duration: Include progression to point of maximal severity (e.g., one asymptomatic periungual wart for 20 years with no change in size vs 10 painful warts on the sole developing over 2 months).

b) Location: Mucous membranes adjacent to cutaneous lesions may also be involved (e.g., consider involvement of rectal mucosa in perianal warts, urethral and bladder involvement in penile warts, and vaginal or cervical involvement in vulvar warts).

c) Current and past treatment: Include successful and unsuccessful treatments and possible complications (e.g., severe pain, scarring, hypopigmentation and hyperpigmentation, extension of warts)

d) Family history of warts

1) Natural history of warts in family and others in family presently with warts

2) Epidermodysplasia verruciformis - a rare familial disorder with HPV that may lead to squamous cell carcinoma

e) History of contagion: Sex partners with warts or cervical dysplasia

f) Other skin disorders

Abnormalities in cell-mediated immune responsiveness of the skin may predispose persons to HPV as well as other cutaneous viral and fungal infections.

g) General health

1) Immunosuppressed state (e.g., AIDS, lymphoproliferative disorder, transplant, cancer treatment, lupus, etc.)

2) Diabetes: Poor circulation may affect treatment plan as well as being associated with greater risk of infection.

3) Cold intolerance: May need to use liquid nitrogen therapy with caution, especially on digits

4) Other

h) Drugs (e.g., prednisone, chemotherapeutic agents, smoking, oral contraceptives, topical estrogen creams may predispose persons to infection)

i) Pregnancy status

1) Warts may worsen in pregnancy and regress afterwards.

2) Certain precautions must be observed. (See Section V.A.1.f. and V.A.2.h.)

j) Sunlight exposure: In transplant patients and patients with epidermodysplasia verruciformis, sunlight exposure may serve as a cocarcinogen.

k) Other

2. Physical examination

An appropriate physical examination of the skin should be performed to determine the type, location and extent of lesions. Diagnosis is usually made on clinical grounds but may require other diagnostic tests (see Section IV.B. and IV.E.)

a) Classification

Lesions are commonly classified by their clinical location and morphology.

1) Cutaneous

(a) Common warts (*verrucae vulgaris*)

(b) Flat warts

(c) Plantar and palmar warts

(d) Mosaic

(e) Other

2) Anogenital

Can involve the vulva, vagina, penis, scrotum, urethra, and rectum

(a) Condylomata acuminata: Cauliflower-like growths

(b) Subclinical lesions: Flat or micropapillary papules

(c) Bowenoid papulosis: May appear as subclinical flat warts on the genitalia but histologic features are those of an intraepidermal squamous cell carcinoma

3) Extracutaneous

(a) Conjunctival warts

(b) Oral condylomas and focal epithelial hyperplasias

(c) Laryngeal papillomatosis (can extend into the bronchopulmonary epithelia)

(d) Other

b) Extent

Extensive warts may suggest an immunocompromised state, epidermodysplasia verruciformis, or other significant disease states and may affect treatment plan.

c) Other

1) Appropriate mucous membranes should be examined by the physician or referred to a specialist:

(a) Oral

(b) Laryngeal

(c) Urethral

(d) Vaginal and cervical

(1) Especially if vulvar lesions are present or if patient's sexual partner has HPV

(2) If cervical dysplasia is present on Papanicolaou smear

(3) In some instances of vulvovaginal pruritus, increased discharge, or pain

(e) Anorectal

In the case of anogenital warts examination of the patient's sexual partner(s) may be indicated. Concern for sexual abuse may be raised in children with anogenital warts. However, anogenital warts in children are not always a sign of sexual abuse, particularly in children 3 years old and younger. Vertical transmission is a distinct possibility.

2) Evidence of scarring from previous procedures may influence whether surgical or nonsurgical treatment approach is taken.

3) Other disease findings, including but not limited to the following:

(a) Skin changes of AIDS

(b) Wartlike lesions distributed over sun-exposed areas in immunocompromised patients (especially those with transplants) may need to be differentiated from squamous cell carcinomas

(c) Other

4) Diagnostic tests

The diagnosis is usually made on clinical grounds. However, there are instances in which it will not be obvious (e.g., verrucous lesion in patients with AIDS or other immunocompromised patients, long-standing warts unresponsive to multiple treatments, condylomas, bowenoid

papulosis, or when HPV is suspected on a mucosal surface). In such cases the following diagnostic tests may be useful.

1. Common

a) Biopsy with hematoxylin-eosin staining: Not a sensitive test for HPV but can rule out other disorders. However, koilocytosis is pathognomonic for HPV infection. Latent infection can be very difficult to diagnose on hematoxylin-eosin-stained sections.

b) Acetic acid 5% with magnification can be performed in most instances of anogenital HPV infections, but false-positive and false-negative results are not uncommon.

c) Cytology of cervix: Koilocytosis can often be diagnosed on a Papanicolaou smear. Cytologic examination may also be coupled with colposcopy to increase sensitivity in selected cases.

d) Anoscopy: Indicated when perianal warts are present

e) Urethroscopy and/or cystoscopy: Does not seem to be helpful at the present time, unless microscopic hematuria is present.

f) Culture or other diagnostic tests may be appropriate for detection of other sexually transmitted diseases (e.g., gonorrhea, syphilis, chlamydia, hepatitis B, and herpes simplex)

g) Other

2. Less common

a) Immunohistochemical detection of papillomavirus structural protein

1) Confirms presence of fully assembled and presumable contagious HPV virions

2) May confirm clinical or subclinical infections (high specificity but low sensitivity)

b) Electron microscopy: Can detect viral particles but the process is laborious and insensitive.

c) DNA hybridization techniques

1) Detects clinical, subclinical, and latent infections (sensitive and specific)

2) Useful in determining the type of HPV: Certain types appear to have greater oncogenic potential (e.g., 16, 18, 31)

d) Polymerase chain reaction: Ultra-sensitive (false-positive results have been reported)

C. Inappropriate diagnostic tests

Not applicable

D. Exceptions

Not applicable

E. Evolving diagnostic tests

1. Serologic techniques

2. HPV has recently been cultured using special techniques. This method of detection of HPV is only a research tool at the moment.

3. Other

V. Recommendations

A. Treatment

1. Major goals of treatment

- a) To increase the clinical disease-free interval
- b) To decrease the bulk of clinically diseased tissue in an effort to "assist" the immune system in dealing more effectively with this virus
- c) To decrease transmission of HPV to adjacent or distant body sites or to other persons by decreasing clinically infected HPV tissue.
- d) To indicate the desirability of routine, lifelong follow-up for early detection of dysplasia, especially in patients with anorectal warts
- e) To avoid aggressive, potentially scarring procedures for asymptomatic subclinical disease with the expectation of curing the patient
- f) In the case of genital warts, the patient's sexual contacts may be referred for evaluation. The use of certain types of condoms may be beneficial in reducing contagion but cannot offer any protection against wart transmission from other uncovered perineal areas where HPV may be present.

g) Other

2. General indications for treatment

- a) Desire for treatment
- b) Painful, bleeding, disfiguring, or disabling lesions
- c) Itching, burning, or dyspareunia associated with genital warts
- d) Large numbers or large warts
- e) Cervical dysplasia associated with HPV
- f) Prevention of spread
- g) Warts in immunocompromised patients: Some lesions may develop into squamous cell carcinoma
- h) Pregnancy with desire for a vaginal delivery and large number of warts of the genitalia and/or cervix. Cesarean section may sometimes be considered to decrease the possibility of laryngeal warts developing in infants.

i) Other

3. Treatment modalities may vary according to a number of factors:

- a) Age of patient
- b) Duration of warts
- c) Location of warts
- d) Extent of warts
- e) Type of warts (mosaic, flat warts)
- f) Patient's immune status
- g) Other disorders patient may have: Diabetes, cold intolerance

- h) Pain tolerance
- i) Inconvenience of some treatments requiring multiple trips
- j) Risk of scarring
- k) Experience of the physician with certain modalities (e.g., laser)
- l) Other

4. Medical treatment

The listed treatments may be used singly, in combination with each other, or with a surgical modality.

a) Common

1) Caustics/acids: Salicylic acid, lactic acid, monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, nitric acid, and others

2) Cantharidin

3) Podophyllin resin: Especially in anogenital HPV. A purified form of this resin containing podofilox as 0.5% solution is available.

4) Tretinoin

5) Bleomycin: Intralesional

6) 5-Fluorouracil: Topical

7) Other

b) Less common

1) Oral etretinate or vitamin A

2) X-ray: Not in laryngeal papillomatosis or epidermodysplasia verruciformis

3) Heat and tape occlusion

4) Other

c) Evolving treatments

1) Interferon alfa: Intralesional or intramuscular

2) Other

d) Inappropriate treatments

At the present time no type of wart vaccine is recommended. Because the placebo response is high, unsupported systemic treatments should be questioned.

5. Surgical treatment

The listed treatments may be used singly, in combination with each other, or in combination with other nonsurgical modalities.

a) Common

(1) Cryosurgery

(2) Carbon dioxide slush (dry ice and acetone)

(3) Electrosurgery & curettage

(4) Blunt dissection

(5) Carbon dioxide laser may be used for the treatment of extensive, recurrent, or recalcitrant warts. It may be used in conjunction with other modalities including electrosurgical debulking, interferon, and/or postoperative 5-fluorouracil.

b) Less common

(1) Excision

(2) Other

6. Other treatments

a) No treatment (spontaneous resolution) to allow development of immunity to the virus

b) Hypnosis and other forms of suggestion

7. Evolving treatments

a) Photodynamic therapy

b) Pulsed dye, Q-switched, and copper vapor lasers, which are directed at the vascular component of the wart, may be useful.

c) Induction of delayed-type hypersensitivity (e.g., squaric acid dibutylester, topical *Rhus*, intralesional tuberculin, dinitrochlorobenzene)

d) Colchicine, cimetidine

e) Other

B. Patient education

Patients should be aware of the following:

1. The wart virus persists after therapy and some degree of infectivity may remain even in the absence of clinical lesions. Patients with a history of anogenital warts need appropriate clinical follow-up because of the potential oncogenicity of some HPV types.

2. Some warts can regress spontaneously; therefore no treatment may be an option.

3. The behavior of individual lesions is not totally predictable and lesions may not respond optimally to treatment.

4. The presence of local as well as systemic immunity may be necessary to eradicate the clinical manifestations of HPV.

C. Miscellaneous

The use of many of these nonsurgical approaches may be contraindicated during pregnancy or in females likely to become pregnant during the treatment period, or in children. Treatment intervals may vary considerably from patient applied daily treatments to multiple office visits. Patients should be aware that long-term follow-up especially in genital warts, may be necessary.