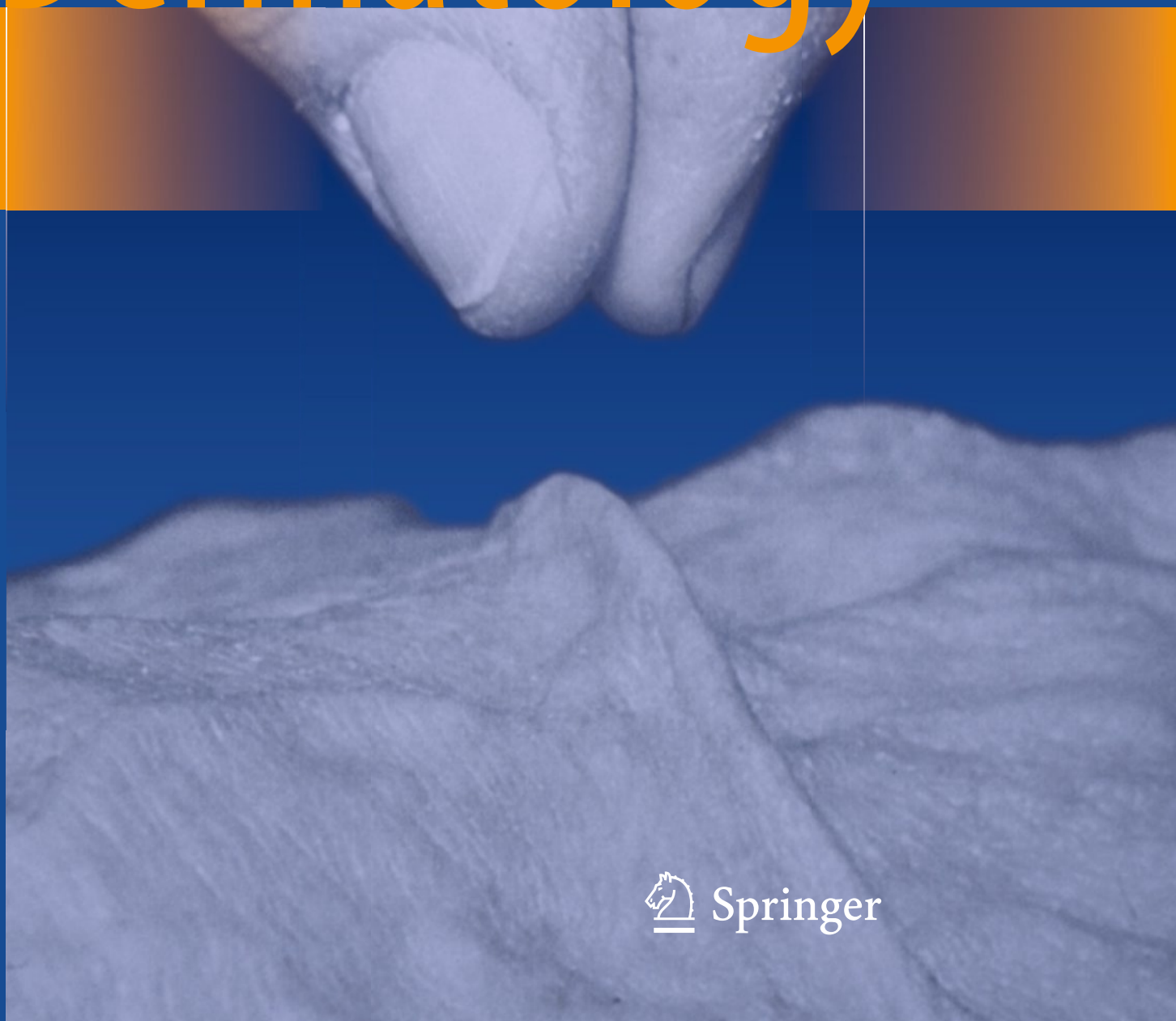
Robert A. Norman
Edward M. Young

Atlas of Geriatric Dermatology



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Preface

This atlas seeks to provide informative reading for dermatologists, geriatricians, internal medicine and family practice physicians, nurse practitioners, physician assistants, nurses, and medical assistants who provide care for the elderly. The work also offers ample material for medical, nursing, nurse practitioner, and physician assistant students. Dermatology residents and Fellows training in geriatric medicine will find the book to be a very helpful foundation in geriatric dermatology.

Glossary

Basic Dermatologic Terminology: Clinical

In examining patients who have skin problems, it is helpful to note the morphology of individual lesions, their pattern in relation to each other, and their distribution on the body. Since the earliest days of medicine physicians have been observing skin diseases and classifying them by these three criteria. Skin diseases are generally dynamic processes that evolve over their course. It is often helpful to identify **primary lesions**, which are the earliest abnormalities, and **secondary lesions**, to which they may evolve. Understanding this evolutionary process makes understanding the pathophysiology of the disease possible.

Primary lesions include a macule, papule, nodule, tumor, wheal, vesicle, and sometimes a pustule. A **macule** is a lesion in which the only abnormality is a change in color. Areas of color change larger than about 1 cm in diameter are referred to as **patches**. A macule may be **hypopigmented**, having a decrease in pigment, or may be **depigmented**, lacking pigment entirely (e.g., vitiligo). It may have an increase in the normal melanin pigment and be **hyperpigmented**. It may have an abnormal color, such as red or yellow. **Erythema** is the term used to describe reddening of the skin; it is generally associated with an increase in blood flow to an area. A yellowish discoloration in the skin may come from certain bile pigments, lipids, severe solar damage, and from yellow pigments in food and dye in some medications.

A raised or indurated lesion may be called a papule, nodule, or tumor. **Papules** are small, generally less than 1 cm in diameter. **Nodules** are somewhat larger. A very large nodule would be called a **tumor**, but this term also refers to any abnormal growth in any organ system. This term must therefore be used with special care, because most patients think that the term "tumor" is synonymous with cancer, which is not true.

A **wheal** (also called urticaria) is the medical term for a hive. It results from the leakage of fluid from blood vessels, which is then held diffusely in the tissue. This causes temporary raising of the tissue; this resolves within a matter of hours as the fluid finds its way back into the circulation through the lymphatics.

Small blisters are called **vesicles**, and larger ones are called **bullae**. These differ from wheals in that they are collections of free fluid within cavities rather than being held diffusely in the tissue. Therefore, they are not transient, as are wheals.

A **pustule** is a cavity filled with pus, which is made up of a mixture of fluid, cellular debris, and microorganisms. Sometimes pustules can be secondary lesions resulting from infection or from irritation of a papule or a vesicle.

Even if no primary lesions are present at examination, it is often possible to make a deduction on the basis of the pathophysiology of primary and secondary lesions.

Weeping and oozing lesions are seen secondary to the rupture of vesicles and bullae. Sometimes, the primary vesicles may have been too small to be seen except microscopically, as in some types of eczematous dermatitis.

Crusts represent dried fluid. Black crusts are usually from blood. Yellow crusts represent dried serum, as from bullous lesions. Brownish or honey-colored crusts, however, are secondarily infected with bacteria, as in impetigo.

A **plaque** is an elevated, plateau-like lesion, which develops from the coalescence of smaller lesions, such as wheals or papules.

A **scale** (also called a squame) is a dried-out bit of excess horny material. It may be secondary to inflammatory erythema, or it may be from excessive production. It is often helpful to describe scales further as thick, fine, or forming a collarette around the lesion.

These terms can be hybridized to describe lesions. Papulosquamous diseases are those that are raised and have scaling on their surfaces. A papulovesicular eruption has both papules and vesicles. Maculopapular eruptions have some areas of only erythema and other areas of erythematous papules.

Various terms are used to describe the shape of lesions. **Linear** refers to lesions in a line. **Annular** means ring-shaped. **Serpiginous** refers to those that wind in a snake-like pattern. **Geographic** refers to a map-like configuration. **Target** or **iris** lesions have a central point with a ring around it, and are usually seen in a condition called erythema multiforme. **Guttate** eruptions are those in which the lesions are small and in the shape of drops.

Other terms are used to identify other surface changes. **Verrucous** means wart-like. **Vegetation** refers to a large, moist, cauliflower-like growth. **Keratosis** is a term describing a circumscribed increase of the horny layer made up of keratin, the major protein in the epidermis. **Excoriations** are scratch marks. Chronic scratching or rubbing of the skin may cause thickening and the development of closely set, flat papules. This is called **lichenification**, which appears as exaggerated skin markings.

Maceration refers to continuous wetting of the skin, which produces thickening and whitening of the skin. It is more likely to occur on **intertriginous** skin. This refers to an area where adjacent skin surfaces rub against each other, such as the axilla and groin, trapping moisture. Excessively dry skin is said to be **xerotic** or **asteatotic**. **Eczematization** refers to a combination of weeping, oozing, vesiculation, erythema, crusting, and lichenification.

Various terms are used to describe abnormalities of structures within the skin. **Alopecia** refers to hair loss. **Hirsutism** or **hypertrichosis** are terms for increased hair. Inflammation of hair follicles is called **folliculitis**. When it is superficial, it appears as small pustules at the base of hairs. Large and deep infections of hair follicles are called **furuncles**. The merging of several adjacent furuncles is called a **carbuncle**. **Comedones** (singular, comedo) include blackheads and whiteheads; these are white, gray, or black plugs in the pilosebaceous openings, consisting of dried sebum, cellular debris, and bacteria. **Cysts** are noninflammatory collections of fluid or semisolid substances surrounded by a well-defined wall.

Telangiectases (singular, telangiectasia) are permanently dilated, small, superficial blood vessels. They usually blanch if they are pressed down, because the blood is within vessels. **Petechiae** are small hemorrhages from superficial blood vessels, which therefore do not blanch. Larger areas of bleeding into the skin are called purpura or ecchymoses.

Examination of the skin and the use of these terms allow most skin diseases to be put into the following categories:

Tumors
Pigmentation abnormalities
Papulosquamous diseases
Vesiculobullous diseases
Papular eruptions
Eczematous dermatitis
Hypersensitivity reactions
Cutaneous infections and infestations
Diseases of the skin appendages (hair, nails, glands, blood vessels)

Basic Dermatologic Terminology: Histologic

Hyperkeratosis is increased thickness of the stratum corneum.

Parakeratosis is the retention of nuclei in the stratum corneum. The stratum granulosum is usually thinned or absent (hypogranulosis) in the presence of parakeratosis.

Hypergranulosis is the increased thickness of the stratum granulosum. Hyperkeratosis is often associated with hypergranulosis.

Acanthosis is the increased thickness of the stratum spinosum.

Acantholysis is the loss of cohesion between epidermal squamous cells, with separation of squamous cells from each other (i.e., cells pull apart).

Intracellular edema is hydropic swelling with cytoplasmic pallor of the squamous cells of the stratum spinosum. When severe, this results in reticular degeneration (see below). This is often accompanied by intercellular edema.

Intercellular edema (**spongiosis**) is edema that develops between squamous cells of the stratum spinosum. The accumulation of colorless edematous fluid between the cells causes separation, with widening of the intercellular spaces and stretching of desmosomes. This imparts a spongy appearance to the epidermis. When severe, spongiotic vesicles form. This is often accompanied by intracellular edema.

Exocytosis is the migration of inflammatory cells from blood vessels in the superficial dermis into the epidermis.

Reticular degeneration is a consequence of severe intracellular edema that causes rupture of epidermal squamous cells, with the formation of multilocular vesicles from resisting cell membranes.

Spongiotic vesicle is the consequence of severe intercellular edema (spongiosis) that causes stretching and loss of the desmosomal attachments between squamous cells, with the formation of blisters in the epidermis.

Acknowledgements

I have great respect and gratitude for my patients, students, and family who have helped me to succeed with the vast challenge and diversity of Geriatric Dermatology. In particular, thanks to Joseph Salhab, OMS4, and my assistant Ibsen Morales for their help in the preparation of this book. Special thanks to Dr. Edward Young, Jr, MD for serving as my co-author. And many blessings to the memory and works of Victor Newcomer, MD, Albert M. Kligman, MD, and the other pioneers of Geriatric Dermatology that helped to set us on the right path. Thanks for caring for our elderly.

Contents

Part III Infections

Abbreviations

AA	Alopecia areata
AFX	Atypical fibroxanthoma
AK	Actinic keratosis
BCC	Basal cell carcinoma
BCE	Basal cell epithelioma (same as BCC)
BK mole	Dysplastic nevus syndrome
BP	Bullous pemphigoid
BXO	Balanitis xerotica obliterans (LS & A of penis)
CCN	Compound cellular nevus
CREST	Calcinosis, Raynaud's phenomenon, esophageal stricture, sclerodactyly, telangiectasia (scleroderma variant)
DF	Dermatofibroma
DFSP	Dermatofibrosarcoma protuberans
DH	Dermatitis herpetiformis
DLE	Discoid lupus erythematosus
DM	Dermatomyositis (also diabetes mellitus)
DSAP	Disseminated superficial actinic porokeratosis
EAC	Erythema annulare centrifugum
EBA(D)	Epidermolysis bullosa acquisita (dystrophica)
EIC	Epidermal inclusion cyst
EM	Erythema multiforme
EN	Erythema nodosum
ENL	Erythema nodosum leprosum
EPP	Erythropoietic protoporphyria
EPS	Elastosis perforans serpiginosa
GA	Granuloma annulare
GVH	Graft-versus-host disease
H-H	Hailey-Hailey (benign familial pemphigus)
HSP	Hertoch-Schonlein purpura
IDN	Intradermal nevus
ILVEN	Inflammatory linear verrucous epidermal nevus
IP	Incontinentia pigmenti
JCN	Junctional cellular nevus
JXG	Juvenile xanthogranuloma
KA	Keratoacanthoma
KS	Kaposi's sarcoma
lEE	Intra epidermal epithelioma
LI, L2, L3	Primary, secondary, tertiary lues
LP	Lichen planus
LS & A	Lichen sclerosus et atrophicus
LSC	Lichen simplex chronicus
MCTD	Mixed connective tissue disease

MED	Minimal erythema dose (phototherapy)
MF	Mycosis fungoides
MM	Malignant melanoma
MTX	Methotrexate
NF	Neurofibromatosis
NLD	Necrobiosis lipoidica diabeticorum
OSW	Osler-Weber-Rendu syndrome (also called HHT, hereditary hemorrhagic telangiectasia)
PAN	Peri(poly)arteritis nodosa
PAV	Poikiloderma atrophicans vasculare (may progress to MF)
PCT	Porphyria cutanea tarda
PG	Pyogenic granuloma or pyoderma gangrenosum
PLEVA	Pityriasis lichenoides et varioliformis acuta (Mucha-Habermann disease)
PMLE	Polymorphus light eruption
PRP	Pityriasis rubra pilaris
PUVA	Psoralen and ultraviolet A light photochemotherapy
PV	Pemphigus vulgaris
PXE	Pseudoxanthoma elastieum
RMSF	Rocky Mountain spotted fever
S & E nevus	Spindle and epithelioid (Spitz) nevus
SCC	Squamous cell carcinoma
SK	Seborrheic keratosis
SSSS	Staphylococcal scalded skin syndrome
TAD	Transient acantholytic dermatosis (Grover's)
TEN	Toxic epidermal necrolysis (a form of erythema multiforme)
TMEP	Telangiectasia macularis eruptiva perstans (a form of mastocytosis)
UP	Urticaria pigmentosa (mastocytosis)
VK	Verrucous keratosis
VV	Verruca vulgaris
XP	Xeroderma pigmentosum

Part I

Introduction to Geriatric Dermatology

Geriatric Dermatology: Overview

1

With the "graying of America," geriatric dermatology is a rapidly growing field. Many skin problems present differently in the elderly, require alterations in standard therapy, or both. In addition, banal-appearing skin rashes in the elderly may signal underlying systemic disease, especially cancer.

The purpose of this chapter is to alert dermatologists, internists, primary care physicians, and all other caregivers to some commonly encountered dermatologic complaints that may portend potentially life-threatening diseases in the elderly. A summary of the biology, histology, and physiology of aging skin provides useful background information for the chapters that follow.

Aging can be defined as a progressive loss of homeostasis that results in decreased organ function [1]. Intrinsic or chronologic aging refers to changes on sun-protected skin. Extrinsic aging, photoaging, and dermatoheliosis refer to changes on sun-exposed skin. These changes differ quantitatively and qualitatively from those of intrinsic aging.

Two major theories of aging exist [2]. The genetic program theory is an irreversible, orderly process, whereas the stochastic theory implicates "wear and tear" by random environmental assaults, particularly free oxygen radicals. These free radicals damage enzymes, DNA, and structural proteins. Both genetic and stochastic processes contribute to aging, depending on the replicative ability of the cell.

Skin changes of premature aging [3, 4] include atrophy, loss of fat, wrinkling, alopecia (hair loss), canities (graying hair), nail dystrophy, defective pigmentation, poikiloderma (triad of atrophy, hyperpigmentation and hypopigmentation, and telangiectasia), sclerosis (hardening), and ulceration. Many genetic syndromes have features of premature skin aging, most notably progeria (Hutchinson-Gilford syndrome), xeroderma pigmentosum, and cutis laxa.

Solar elastotic syndromes [5–7] describe changes seen in chronically photodamaged skin, They are not part of intrinsic aging per se, but are nevertheless commonly seen in elderly patients. The most common conditions include Favre-Racouchot syndrome (nodular elastosis with cysts and comedones), cutis rhomboidalis nuchae, actinic cheilitis and actinic keratoses, and poikiloderma of Civatte.

Aging research [5] has been confounded by four factors: (1) confusing chronologic aging and photoaging; (2) confusing development and senescence; (3) confusing aging with age-associated diseases (e.g. diabetes mellitus); and (4) confusing aging with age associated hormonal changes (e.g. menopause).

The epidemiology of skin disease in the elderly [8] can reveal several different patterns: some diseases increase steadily with age, others show a bimodal peak early and late in life, and some decline with age or show a single peak incidence in middle age.

Cellular and Molecular Changes in Aging Skin

Intrinsic or chronologic aging results in decreased mitogenic (replicative) potential or decreased life span of fibroblasts, melanocytes, and keratinocytes [2]. Decreased responsiveness to growth stimulators and increased responsiveness to growth inhibitors occur. When this responsiveness fails, malignancies develop. Photoaging accentuates these changes and also tilts the balance from cellular differentiation toward proliferation, with obvious implications for photocarcinogenesis.

Intrinsic aging adversely affects nucleic acids and cellular proteins, such as collagen, elastin, fibronectin (an adhesion molecule), intercellular ground substance, membrane composition, and catalase (an antioxidant enzyme that prevents free radical damage). Photoaging is characterized by severe solar elastosis, which results in massive dermal deposits of elastin-like material. This solar elastotic material probably originates from elastin rather than collagen, because it cross-reacts with antielastin antibodies and is susceptible to elastase, but is resistant to collagenase. It is only partially cross-linked by desmosine, however, so it lacks normal physiologic elasticity. These changes predispose the elderly skin to easy tearing, bruising, and wrinkling.

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Histologic Changes in Aging Skin

Clinically, aged skin exhibits roughness (dryness, xerosis), laxity, wrinkling, uneven pigmentation, and benign and malignant growths.

Histologically, flattening of the dermal-epidermal junction and effacement of the epidermal rete ridges and interdigitating dermal pegs are the most consistent findings in aged skin [9–15]. The stratum corneum and epidermal barriers remain well formed. Cellular heterogeneity (variation in cell size, shape, and staining characteristics) results in a diffuse epidermal dyscrasia (mild actinic keratosis) of photoaged skin. Melanocytes decline by 10–20 % each decade, resulting in poor tanning, decreased melanocytic nevi (moles), poor pigment transfer to keratinocytes, and guttate hypomelanosis on the arms and shins. Langerhans' cells are intraepidermal macrophages that decrease in density and in immune responsiveness to ultraviolet damage with age.

The dermal-epidermal junction shows reduplication of structural components, a loss of interdigitating basal cell "foot" processes, and decreased dermatoglyphics (fingerprints and other skin markings). This loss of dermal-epidermal adhesion predisposes to blistering from such diverse causes as cardiac edema, lichen planus, and autoimmune bullous pemphigoid.

The dermis becomes less dense, relatively acellular, and avascular. Loss of functional elastic tissue results in wrinkles, both temporary and permanent. Sun-protected aged skin shows fewer fibroblasts, macrophages, and mast cells. In contrast, photo aged skin has an increased number of inflammatory cells, especially partly degranulated mast cells. This "chronic heliodermatitis" [12] may stimulate fibroblasts to produce solar elastotic material.

The microcirculation and nerves undergo a gradual decline, predisposing to poor thermoregulation and decreased sensation for burning. Erythema ab igne ("redness from the fire") occurs from overuse of heating pads and space heaters and is manifest as a reticulate poikiloderma. The eccrine and apocrine sweat glands decline in number and activity, with decreased perspiration, body odor, and thermoregulation. Sebaceous glands paradoxically increase in size despite a decrease in function; this is related to decreased serum androgen levels. Sebaceous hyperplasia, rhinophyma (bulbous, greasy nose), and asteatotic (xerotic or nummular) eczema occur, the last exacerbated by overbathing and inadequate moisturizing. Hair decreases in number, caliber, and pigment density, resulting in male pattern androgenetic alopecia in men and diffuse hair thinning in women. Conversion of fine vellus to coarse terminal hairs occurs on the ears and nose of men and on the upper lip and chin of women, a common cosmetic annoyance. Nails undergo a slow irregular decline in growth, thinning of the nail plate, and longitudinal ridging, softening, splitting, and shaling. Subcutaneous fat atrophies on the cheeks and distal extremities but hypertrophies on the waist of men and thighs of women.

Aging Skin-Epidermal Changes

Melanocytes
- density doubles on sun-exposed skin.
- 10–20 % decline per decade.
- decreased tanning, decreased nevi, increased lentigines, decreased pigment transfer to keratinocytes, increased guttate hypomelanosis

Langerhans cells
- decreased density, decreased responsiveness

Aging Skin-Dermal-Epidermal Junction

Loss of basal cell cytoplasmic "foot" processes
Reduplication of lamina lucida and anchoring fibrils
Grenz zone or microscar in photoaged skin (NOT in sun-protected skin) consists of dense, horizontal band of collagen fibers and loss of elastin
Decreased dermatoglyphics, more pronounced on sun-exposed skin

Aging Skin: Dermal-Epidermal Junction Changes

Aging Skin-Dermal Changes

Decreased density, relatively acellular and avascular
Increased wrinkles-2 types
temporary-loss of elastic tissue from papillary dermis.
permanent-photoaged epidermis surrounded by solar elastosis

Aging Skin: Dermal Changes

Aging Skin-Dermal Changes

Decreased collagen-l % annual decline, altered fibers
Ground substance – gradual decline in sun-protected skin versus late increase in sun-exposed skin.
Clinical-easy tearing-stellate pseudoscars, traumatic purpura.

Aging Skin: Dermal Changes

Aging Skin-Dermal Changes

Dermal cells (fibroblasts, macrophages, mast cells)
Decrease in sun-protected skin.
Increased inflammatory cells in photoaged skin = chronic heliodermatitis. Possible stimulus to solar elastosis.
Vascular changes
Decreased capillary and venular thickness
Increased bruising, pallor, poor thermoregulation

Aging Skin: Dermal Changes

Aging Skin-Nerves

Nerves
Fewest changes in free nerve endings. ±decreased Pacinian corpuscles (pressure receptors)
decreased Merkel cells and Meissner corpuscles (light touch receptors)
decreased sensation for burn – erythema ab igne

Aging Skin: Nerves

Aging Skin-Glands

Eccrine sweat glands-decreased # and function, increased lipofuscin, decreased thermoregulation
Apocrine sweat glands-decreased secretion (decreased androgens), increased lipofuscin, decreased body odor
Sebaceous glands-decreased sebum production (decreased androgens), but increased gland size
Clinical: Sebaceous hyperplasia, rhinophyma, asteatotic (xerotic) eczema.

Aging Skin: Glands

Physiologic Changes in Aging Skin

Age related physiologic differences have been studied [1, 16, 17] in regard to the following: (1) proliferation and repair; (2) anaplasia; (3) percutaneous absorption and dermal clearance; (4) immunity; (5) enzyme activities; (6) mechanical properties; and (7) vitamin D synthesis [18, 19].

Proliferation and repair decrease with age, reflected by a slower epidermal renewal rate, decreased wound healing, increased wound dehiscence, and decreased re-epithelialization of blisters. Photoaged skin, however, undergoes an adaptive increase in DNA repair. Clinically, hyperproliferative disorders such as psoriasis and dandruff may be expected to improve with age, although many exceptions occur.

Anaplasia is disorderly growth that results in various benign, premalignant, and malignant growths in the elderly. Seborrheic keratoses and cherry (capillary) hemangiomas occur with age, usually independent of sun exposure. Actinic (solar) keratoses, basal cell carcinoma, squamous cell carcinoma, and malignant melanoma are provoked by sun exposure. Because of decreased numbers of melanocytes for pigment protection, decreased numbers of Langerhans' cells for immunosurveillance, and a lowered inflammatory response to eradicate neoplastic cells, photocarcinogenesis is a common problem in the elderly,

Age-related changes in percutaneous absorption and dermal clearance result in an increased incidence of contact dermatitis in the elderly. Decreased vasculature results in decreased clearance of chemicals from the dermis, with increased opportunity for the development of irritant (especially) and allergic contact dermatitis. The blunted inflammatory response of elderly skin can significantly delay diagnosis.

Immunity generally wanes with advancing age. A mild decrease in T-cell-mediated delayed hypersensitivity occurs in response to new allergens, although previously acquired immunity may persist. B cells remain constant with an increase in autoantibodies, clinically detected in bullous pemphigoid, pemphigus vulgaris, systemic lupus erythematosus, rheumatoid arthritis, and vasculitis. Decreased immediate (IgE) hypersensitivity to a radioallergosorbent test (RAST) has been reported. Clinically, these changes predispose to infections (especially fungal and viral), malignancy, and autoimmune disease.

Enzyme activity of the epidermis remains remarkably constant with age, although decreased catalase (antioxidant) activity allows free radicals to accumulate, and cause damage [20].

Mechanical properties of aged skin include decreased elasticity, increased laxity, decreased recovery from indentation pressure, decreased torsion extensibility, and decreased turgor. Elderly skin is susceptible to mechanical tearing from trauma and wound dressings, and to wrinkling from prolonged sleep [21].

Vitamin D synthesis declines with age. Along with overzealous sunscreen use, calcium -deficient diets (lactose intolerance), and estrogen deficiency, vitamin D deficiency can result in osteoporosis and hip fractures [22].

Aging Skin-Hair and Nails

Hair
decreased #, growth rate, shaft diameter
androgenetic alopecia
conversion of veil us to terminal hairs – cosmetic nuisance
Nails
slow, irregular decline in growth
decreased nail plate thickness, decreased lunula (matrix) size
Clinical: longitudinal ridges, soft, fragile, brittle nails.

Aging Skin: Hair and Nails

Physiologic Changes in Aging Skin

Proliferation and repair
Anaplasia
Percutaneous absorption and dermal clearance
Immune responsiveness
Mechanical properties
Vitamin D synthesis

Aging Skin: Physiologic Changes

Aging Skin-Proliferation and Repair

Decreased epidermal turnover (20 versus 30 days)
Decreased wound healing, increased dehiscence
Decreased re-epithelialization of blisters, increased infection,
Increased DNA repair in photoaged skin – adaptation
Clinical: hyperproliferative disorders, such as psoriasis and dandruff, may be expected to IMPROVE with age

Aging Skin-Anaplasia

Increased benign and malignant growths SK, AK, BCC, SCC
Increased photocarcinogenesis
Decreased immune surveillance by Langerhans cells
Decreased melanocytes for pigment protection
Decreased inflammatory cells to destroy neoplastic cells

Aging Skin – Mechanical Properties

Decreased elasticity, increased laxity
Decreased visco-elastic properties – altered ground substance and fibronectin-collagen adhesion
Decreased recovery from indentation pressure Decreased torsion (twisting) extensibility
Clinical: Increased susceptibility to tearing by trauma, wound dressings, skin sagging and wrinkling.

Aging Skin: Mechanical Properties

Aging Skin-Immune Responses

Decreased immune responsiveness
±Decreased T-cells, no change in B-cells Increased autoantibodies
±Decreased delayed hypersensitivity
Decreased immediate hypersensitivity
Clinical: Increased infections, especially viral & fungal
Increased malignancy
Increased autoimmune diseases-bullous pemphigoid, pemphigus, lupus

Aging Skin: Immune Responses

Aging Skin-Vitamin D Production

Decreased vitamin D production
Decreased dietary calcium (lactose intolerance) Decreased estrogen (menopause)
Increased sunscreen usage
Increased osteoporosis [23–25]

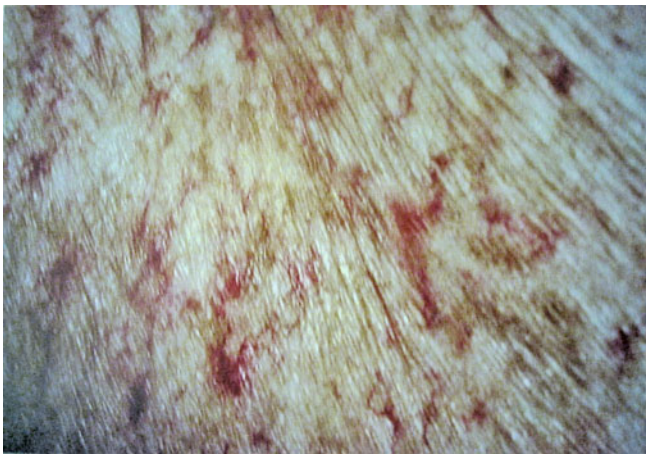


Fig. 1.1 Poikiloderma: hyperpigmentation, hypopigmentation, atrophy, and telangiectasia

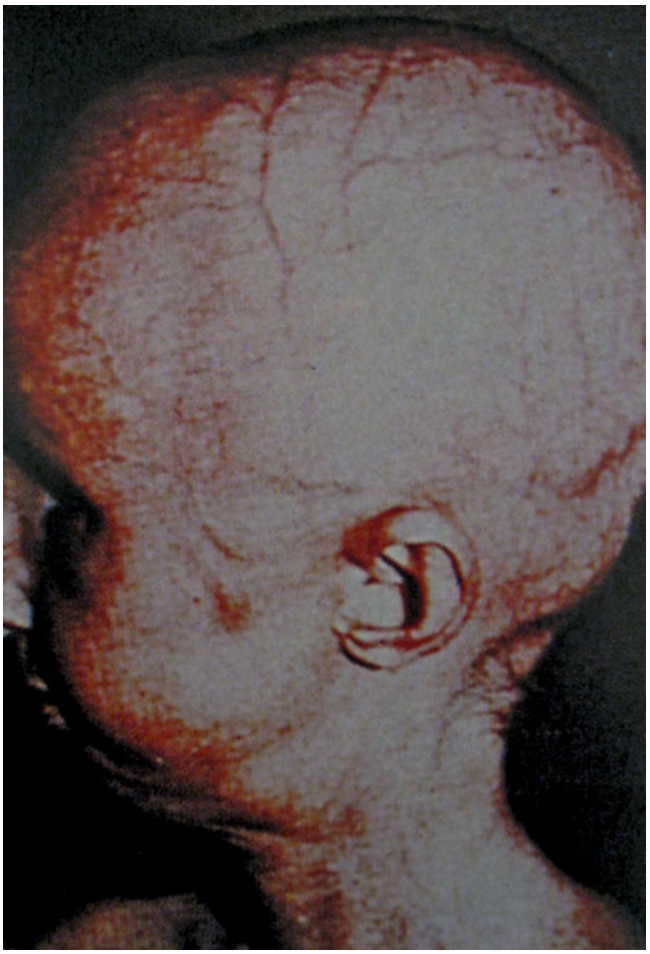


Fig. 1.2 Progeria (Hutchinson-Gilford syndrome). A profile of l3-year-old progeria patient showing craniofacial disproportion, micrognathia, almost total alopecia, and prominent venous pattern in the scalp. (Reproduced with permission from Gilchrest [1])

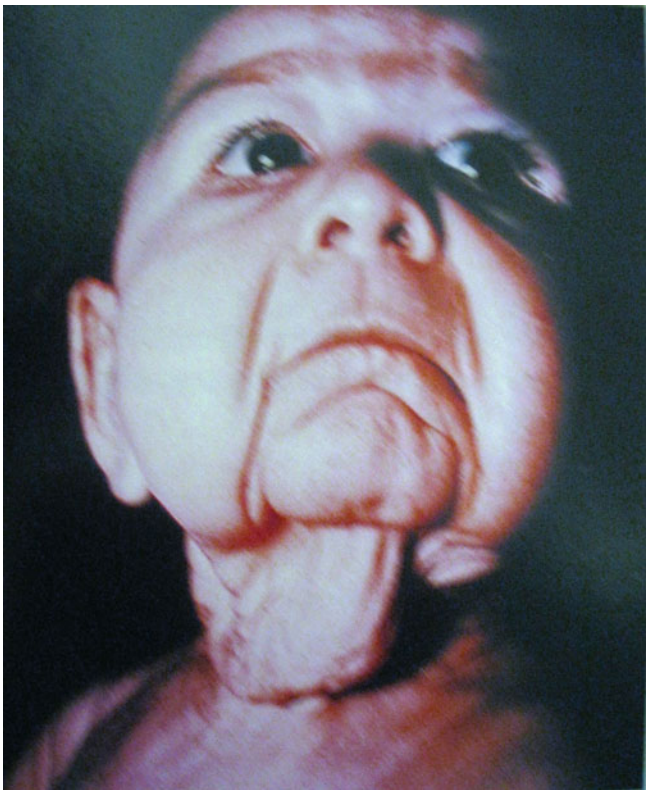


Fig. 1.3 Cutis laxa. This infant appears to be 80 years old

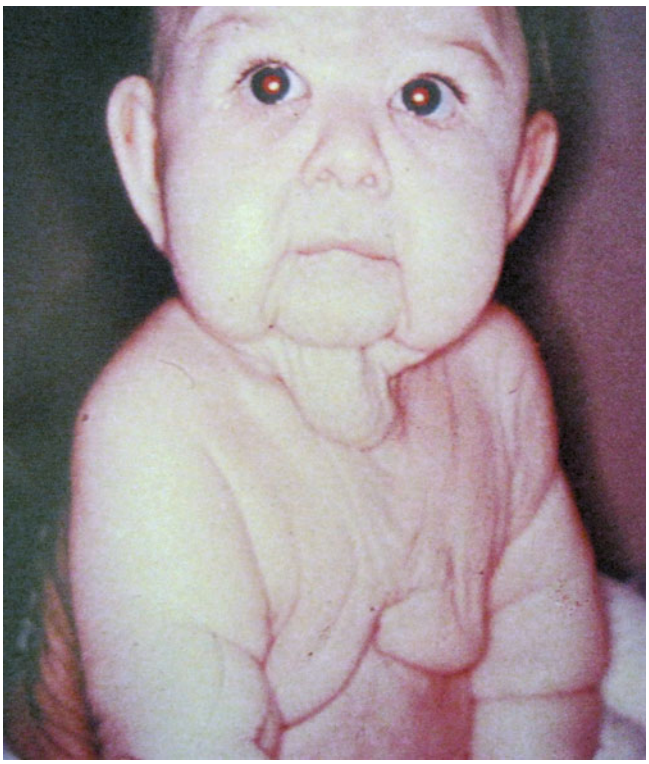


Fig. 1.4 Cutis laxa. This infant appears to be 80 years old

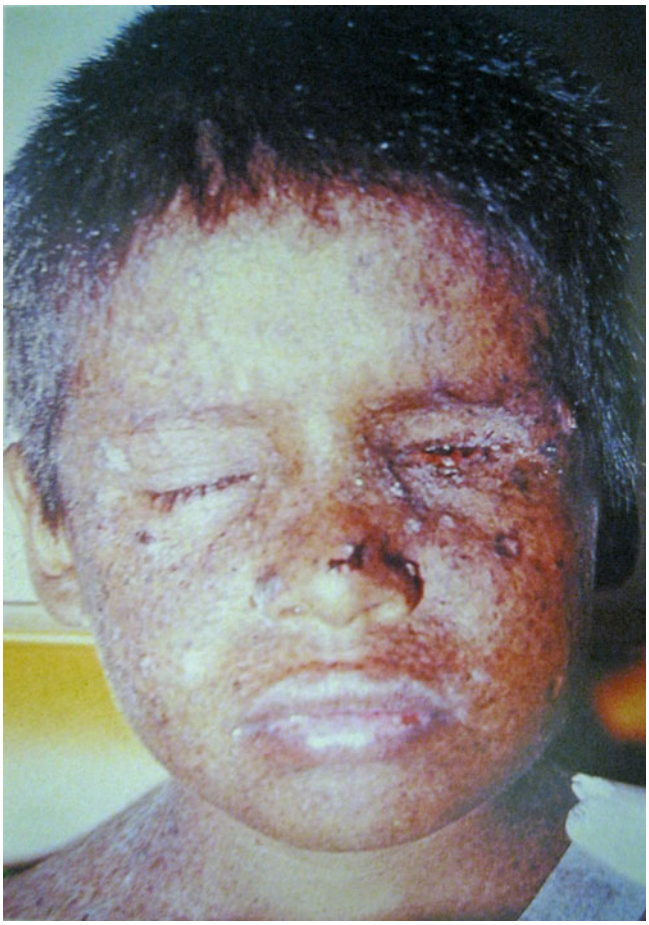


Fig. 1.5 Xeroderma pigmentosum. This child has already developed multiple skin cancers because of faulty DNA repair

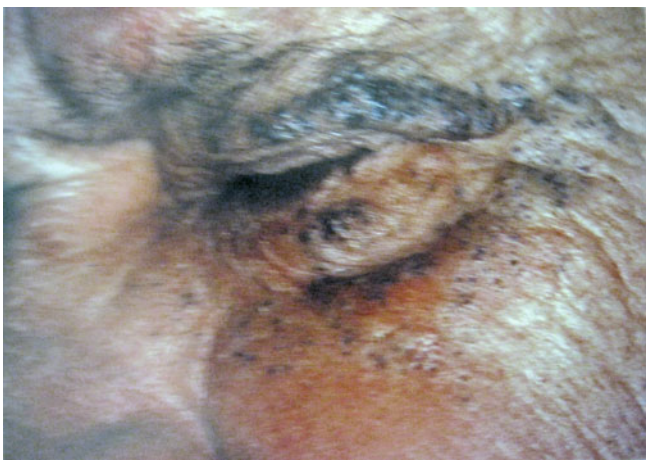


FIG. 1.6 Favré-Racouchot syndrome (nodular cysts and comedones)

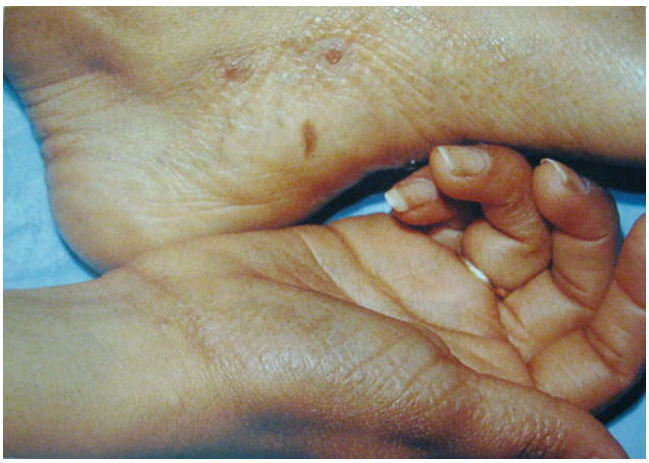


Fig. 1.7 Acrokeratoelastoidosis, also called collagenous and elastotic marginal plaques of the hands or digital papular calcific elastosis, shows flesh-colored cobblestone papules along sun-exposed edges of the hands and feet

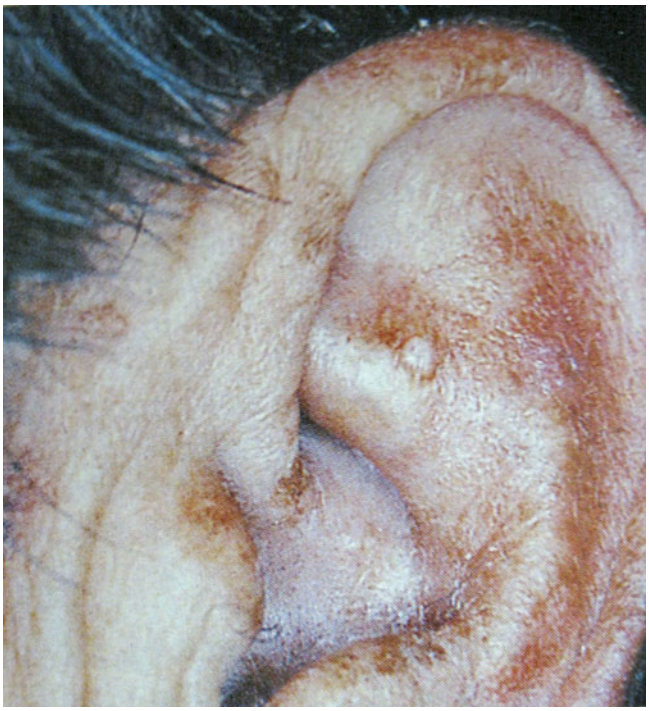


Fig. 1.8 Solar elastoma on ear (Reproduced with permission from Newcomer and Young [5])

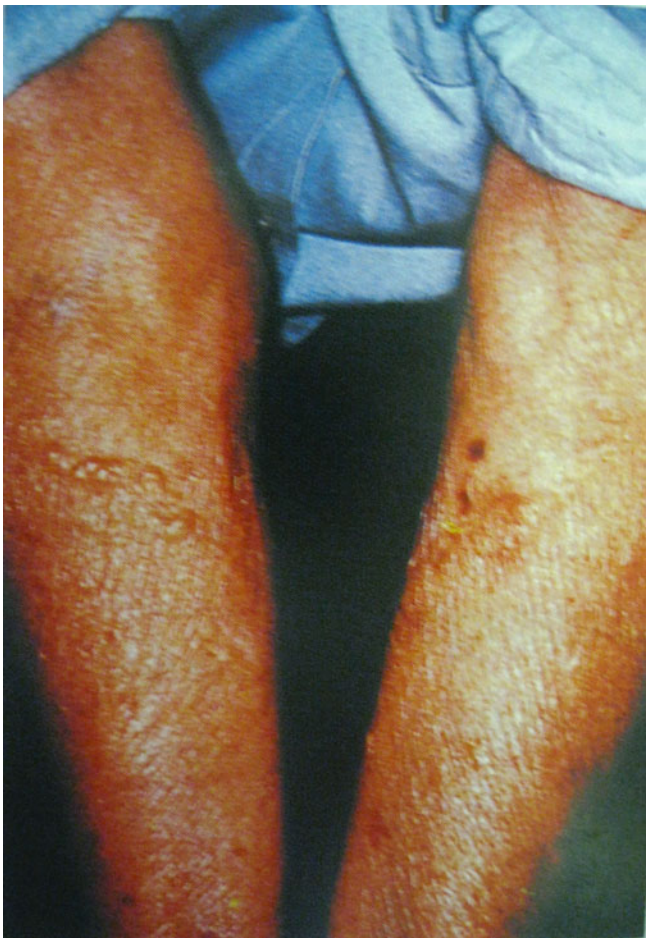


Fig. 1.9 Solar elastotic bands of the forearms (Courtesy of Dr. Sharon Raimer. Reproduced with permission from Newcomer and Young [5])

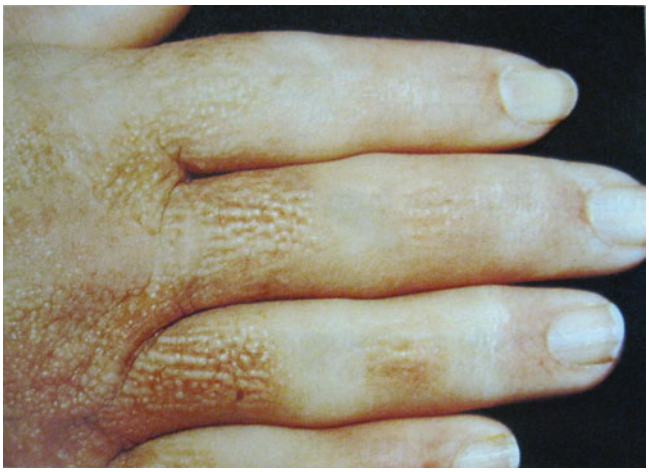


Fig. 1.10 Colloid milia consist of beaded translucent papules on the sun-exposed dorsal hands and fingers

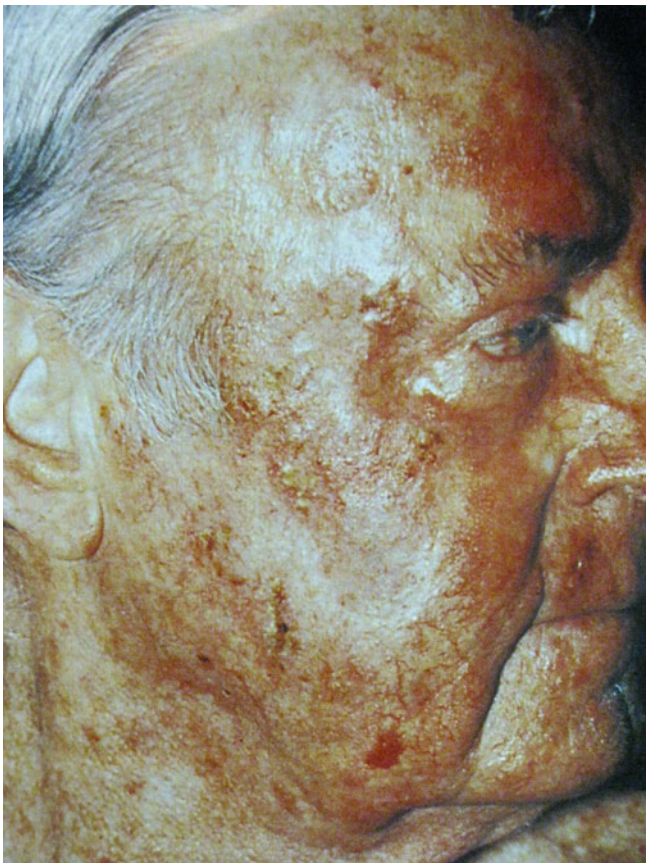


Fig. 1.11 Diffuse photodamage with multiple actinic keratoses and basal cell carcinomas

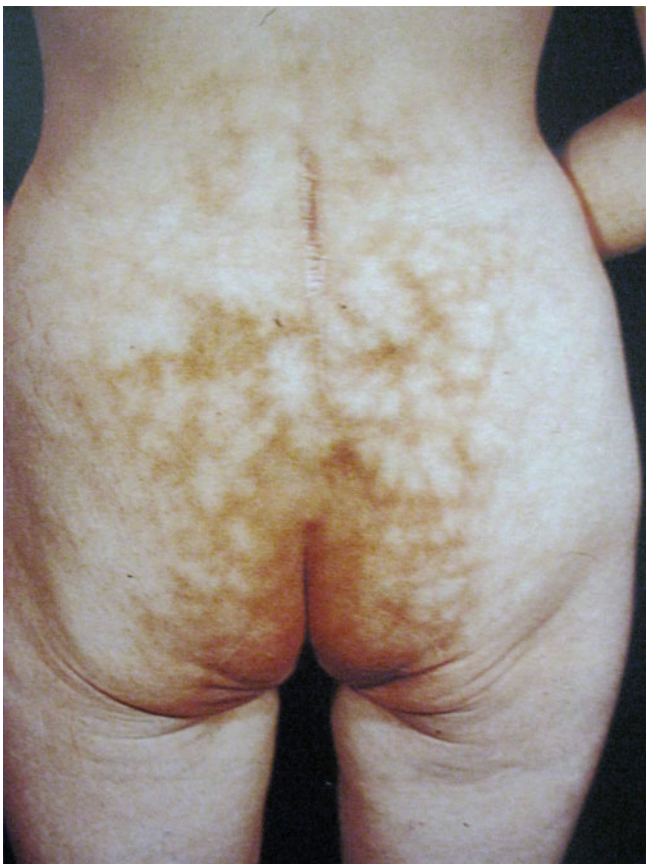


Fig. 1.12 Erythema ab igne: "redness from the fire"

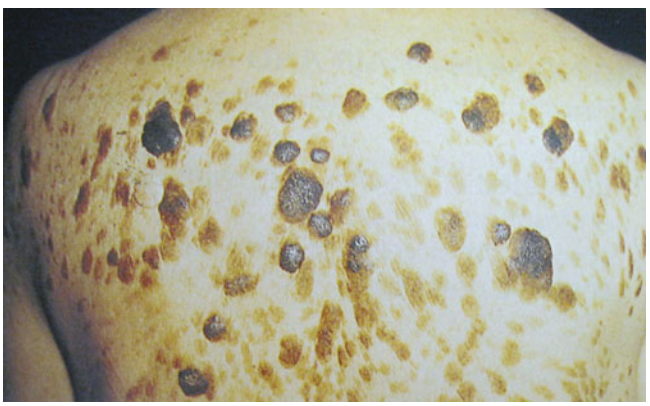


Fig. 1.13 Multiple seborrheic keratoses

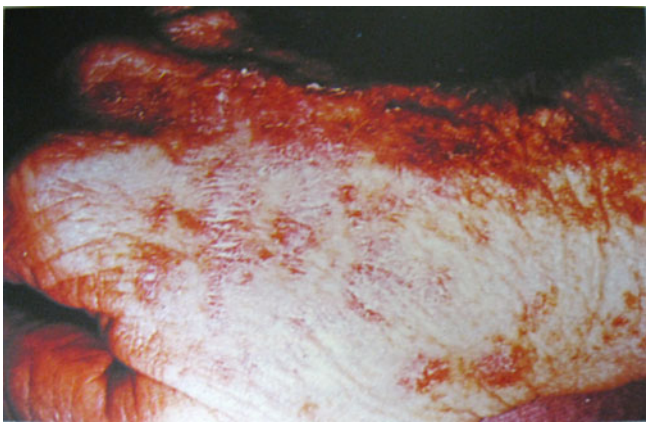


Fig. 1.14 Actinic keratoses on the hand

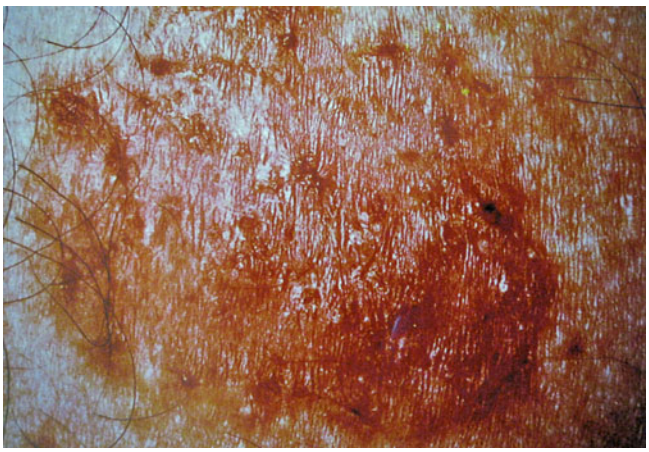


Fig. 1.15 Superficial basal cell carcinoma resembles eczema, tinea, psoriasis

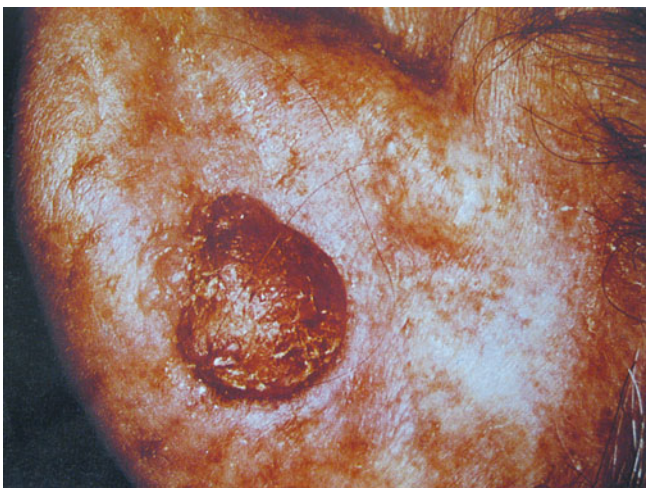


Fig. 1.16 Squamous cell carcinoma of posterior ear

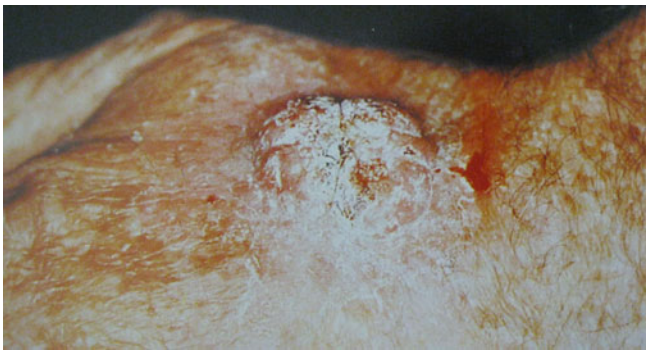


Fig. 1.17 Keratoacanthoma. The differential diagnosis includes squamous cell carcinoma

Fig. 1.18 Atrophic erythematous scaly patches

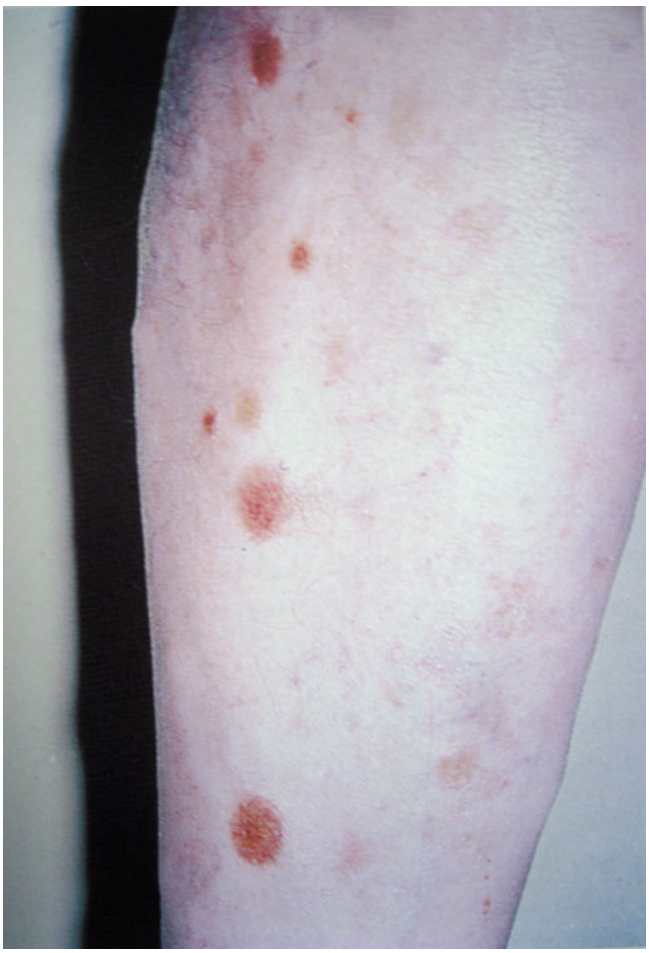


Fig. 1.19 Red scaling patches and plaques on the legs

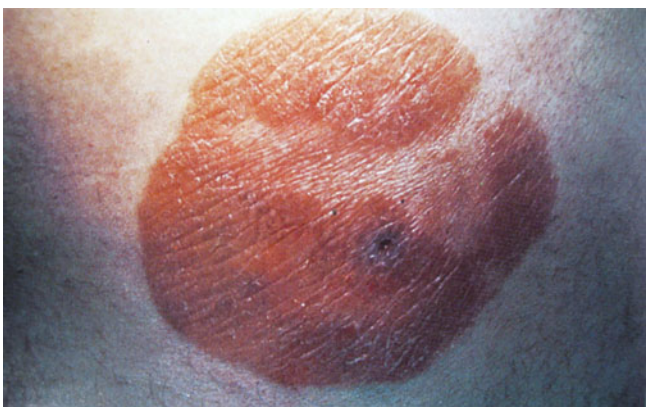
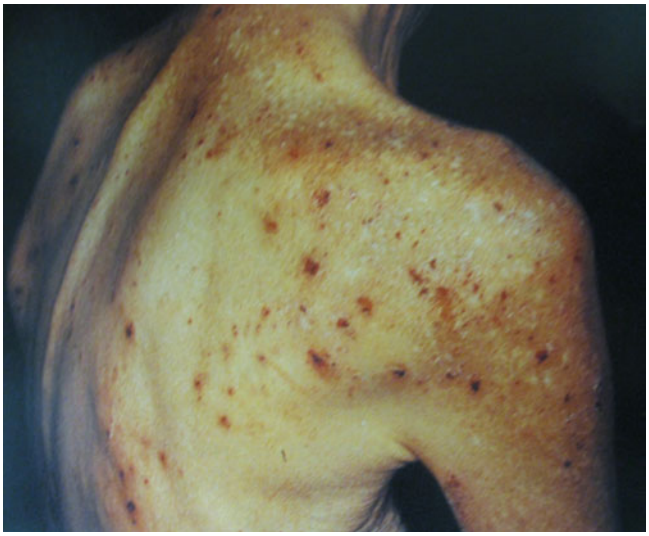
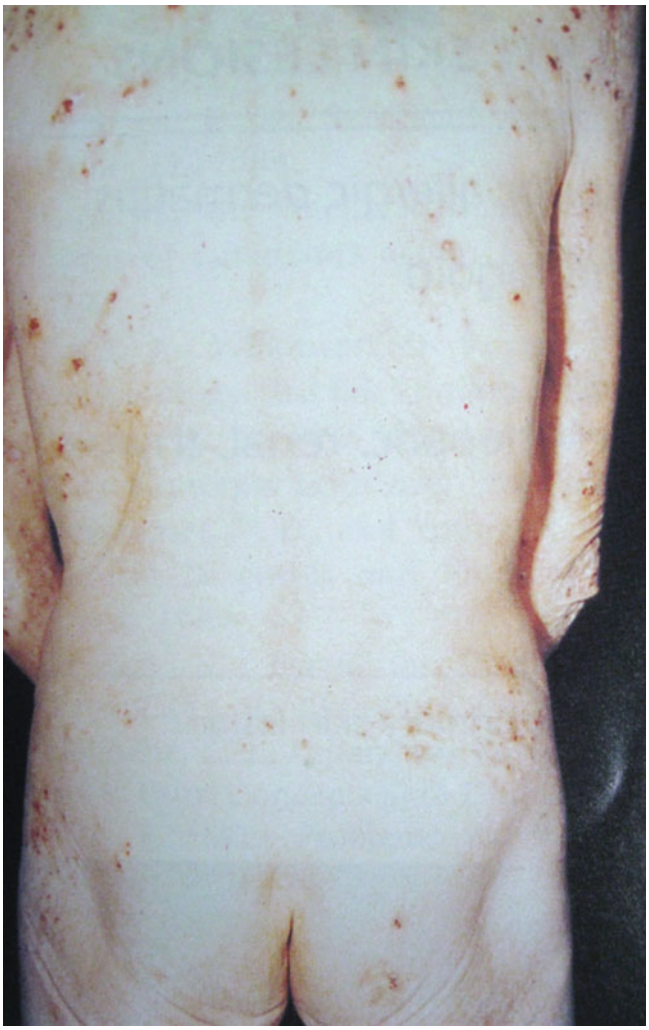


Fig. 1.20 Large psoriasiform plaque of mycosis fungoides (Courtesy of Dr. L. Swinyer)





Figs. 1.21 and 1.22 Excoriations, erosions, and rare papulovesicles on extensor surfaces

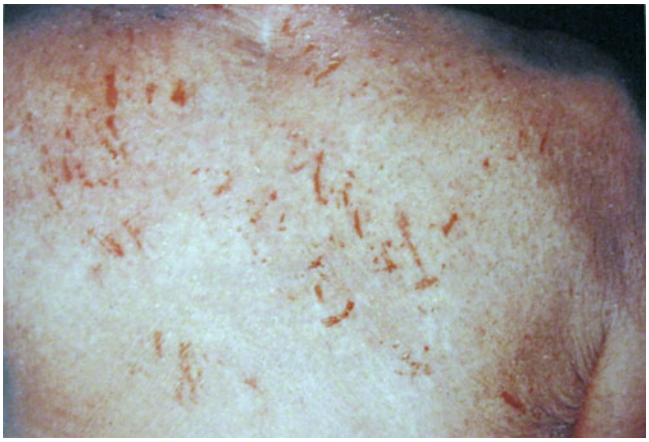


Fig. 1.23 Excoriations: patient had used a back scratcher

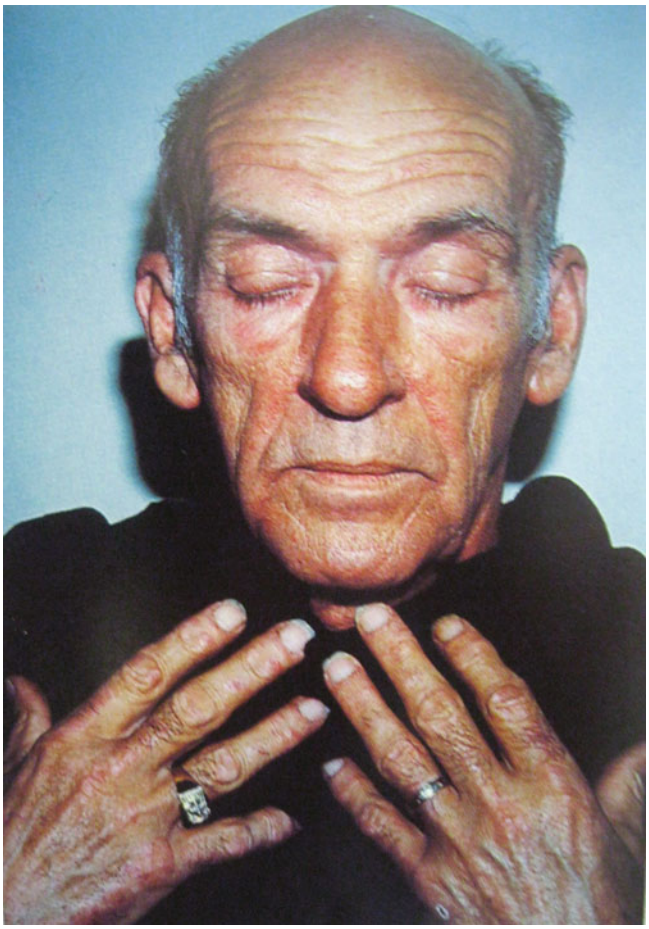


Fig. 1.24 A patient with generalized pruritus but no obvious rash

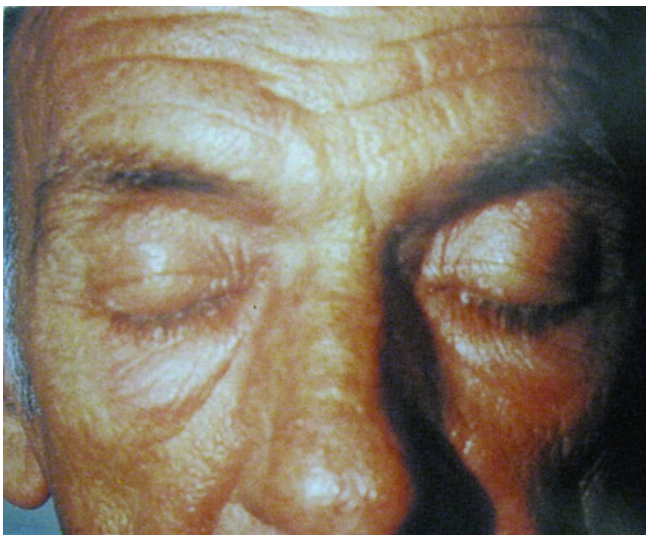


Fig. 1.25 Close examination reveals periorbital edema and heliotrope (lilac) eruption

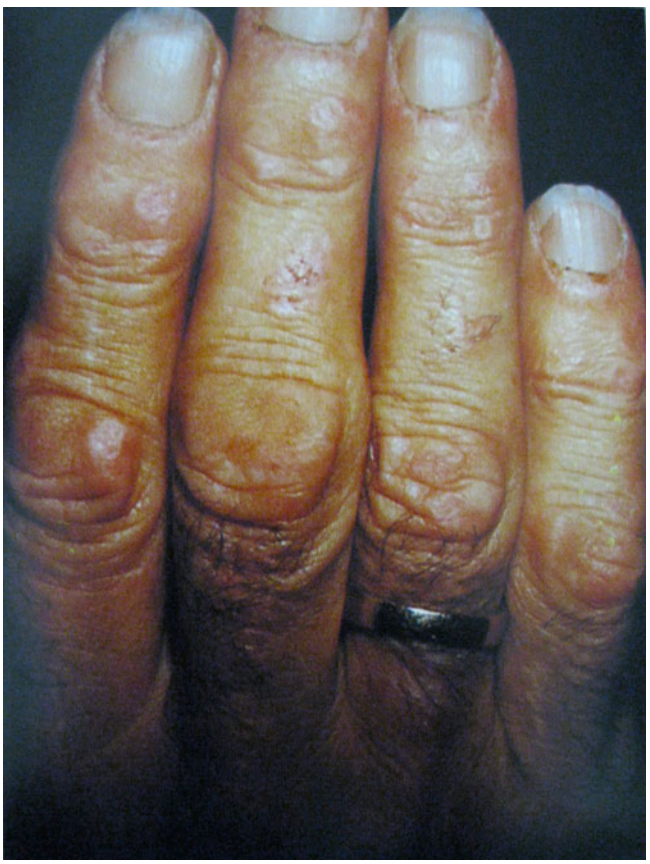


Fig. 1.26 Gottron's papules on the knuckles are suggestive of dermatomyositis

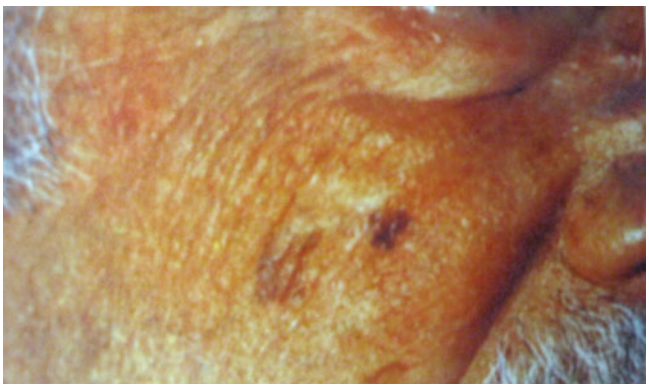


Fig. 1.27 Patient obtained cosmetic improvement with an over-the-counter bleaching cream, but delayed diagnosis

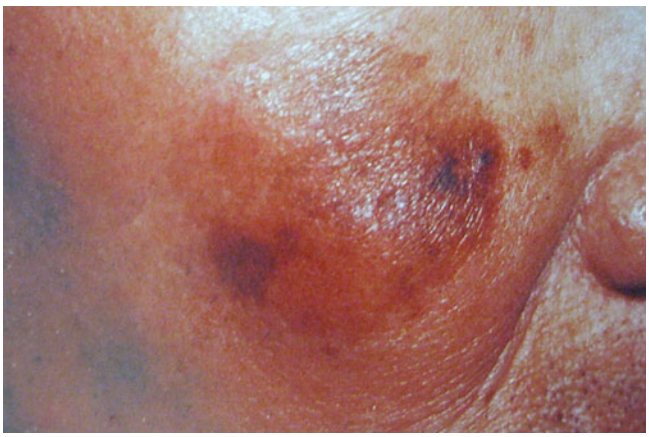


Fig. 1.28 Brown variegated patch on the cheek

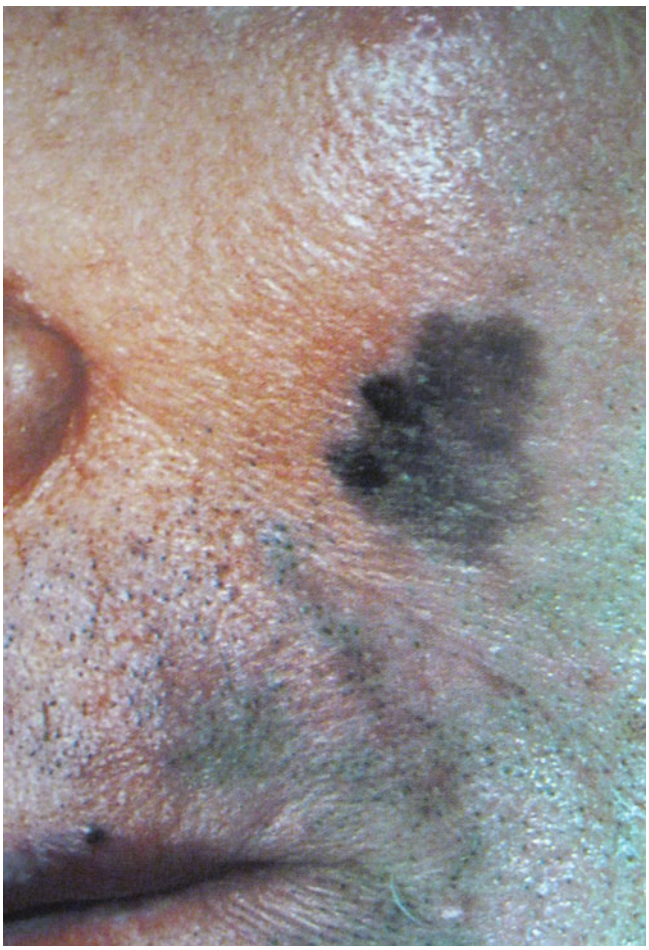


Fig. 1.29 Lentigo maligna melanoma is the final result of a long-neglected lentigo maligna (Courtesy of Dr. L. Swinyer)

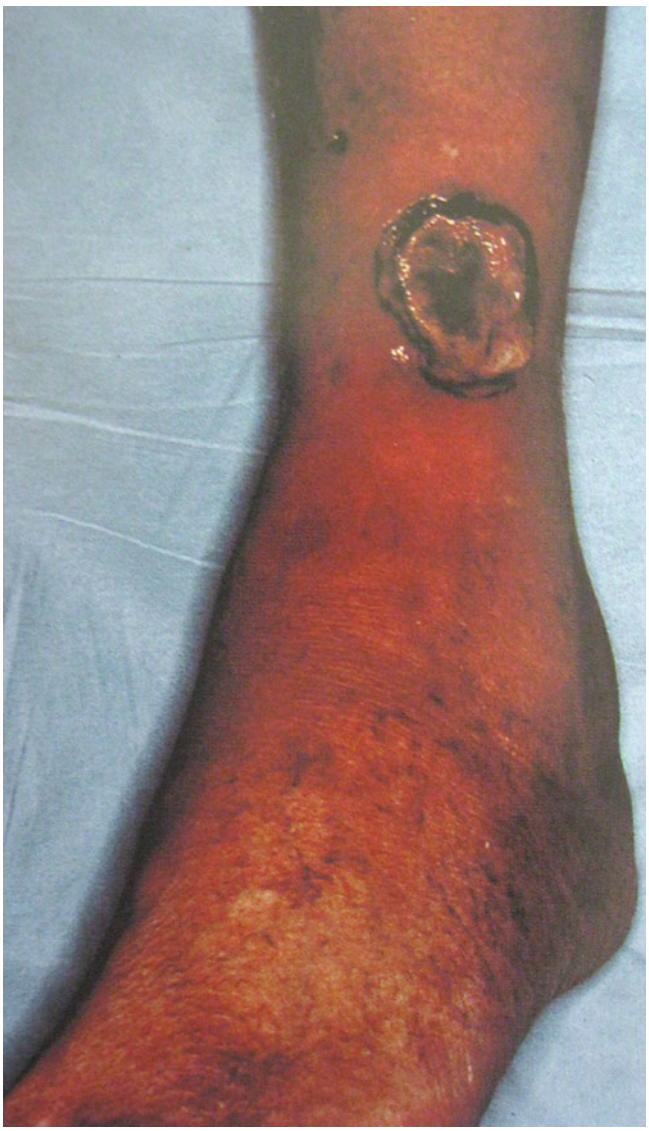
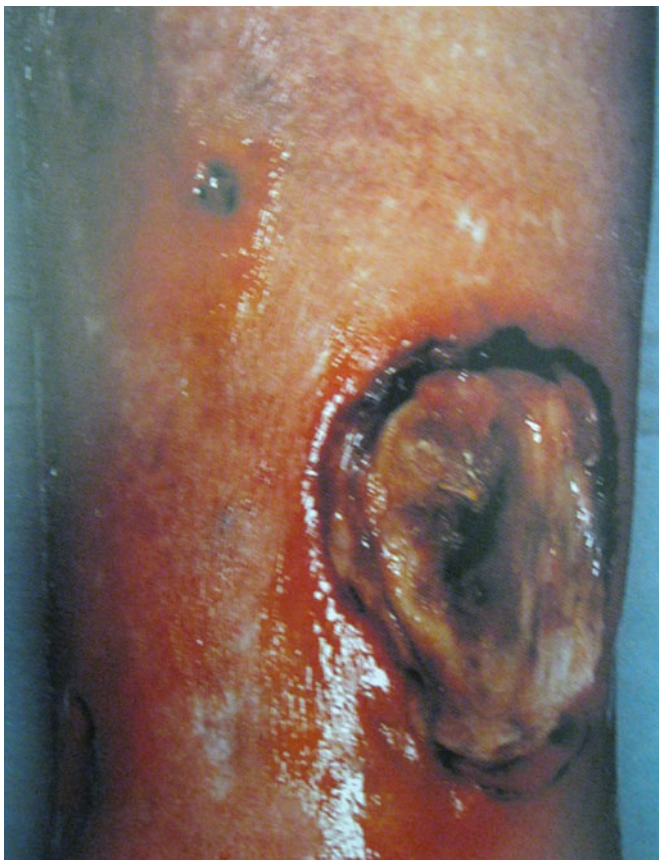
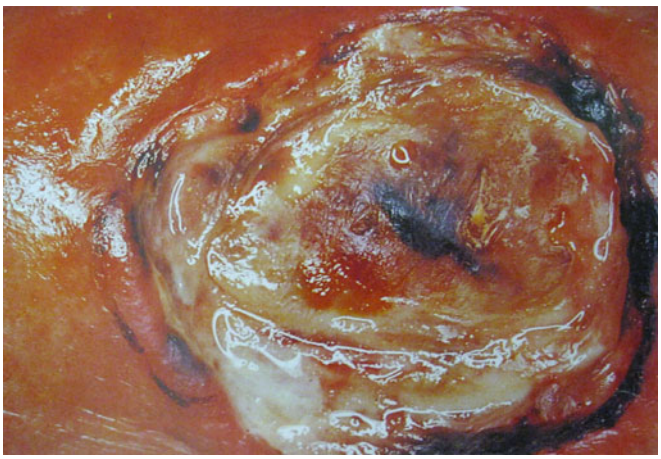


Fig. 1.30 A deep, dirty ulcer in a patient with normal arterial and venous circulation





Figs. 1.31 and 1.32 Close-up view showing jagged, undermined border

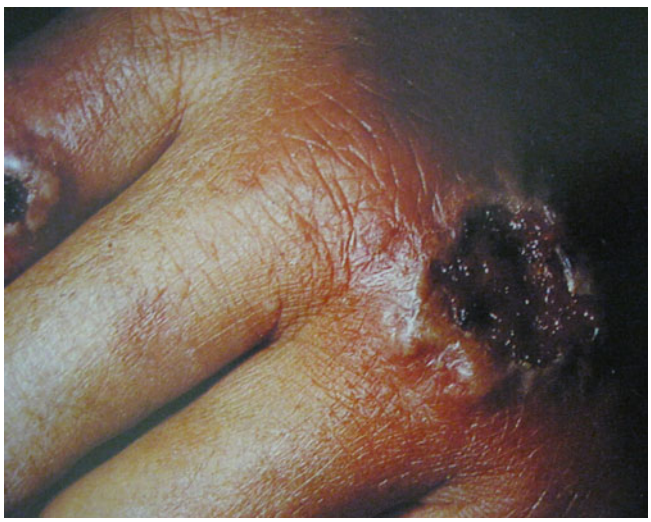


Fig. 1.33 Close-up of hand showing multiple ulcers that developed at sites of puncture wounds (pathergy)

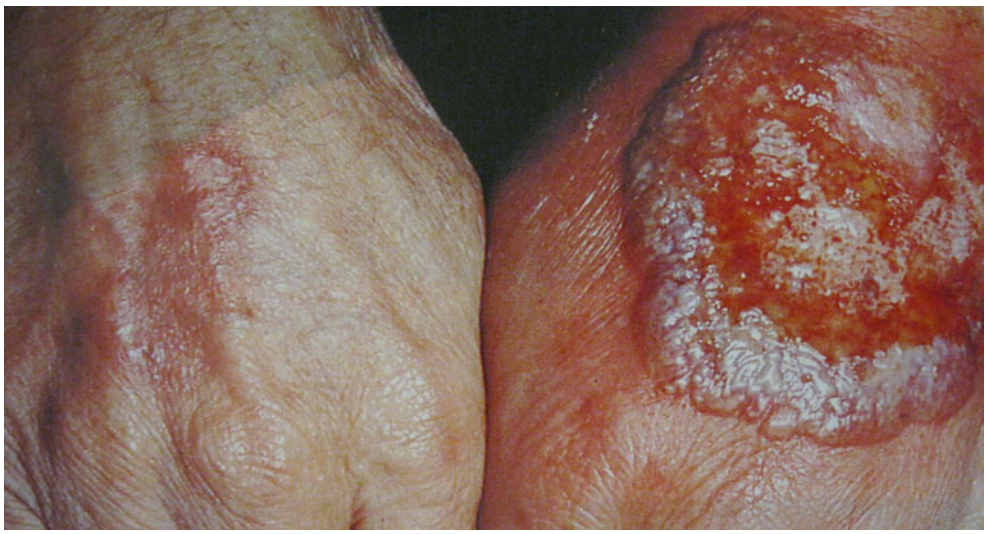


Fig. 1.34 Large, bullous pyoderma gangrenosum in a patient with myelogenous leukemia. Note the cribriform scarring on the opposite hand

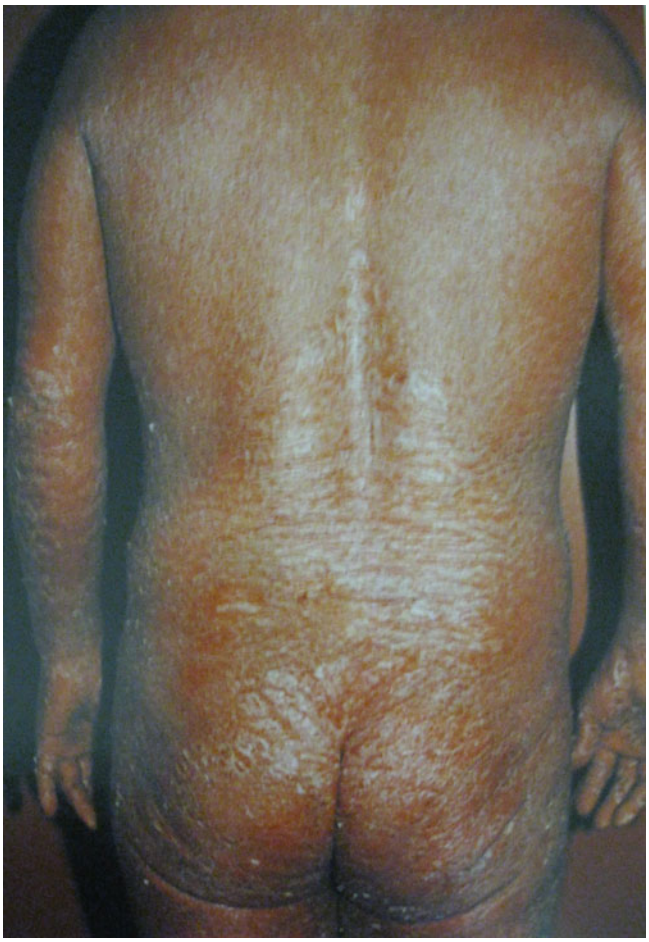


Fig. 1.35 "Red man" syndrome

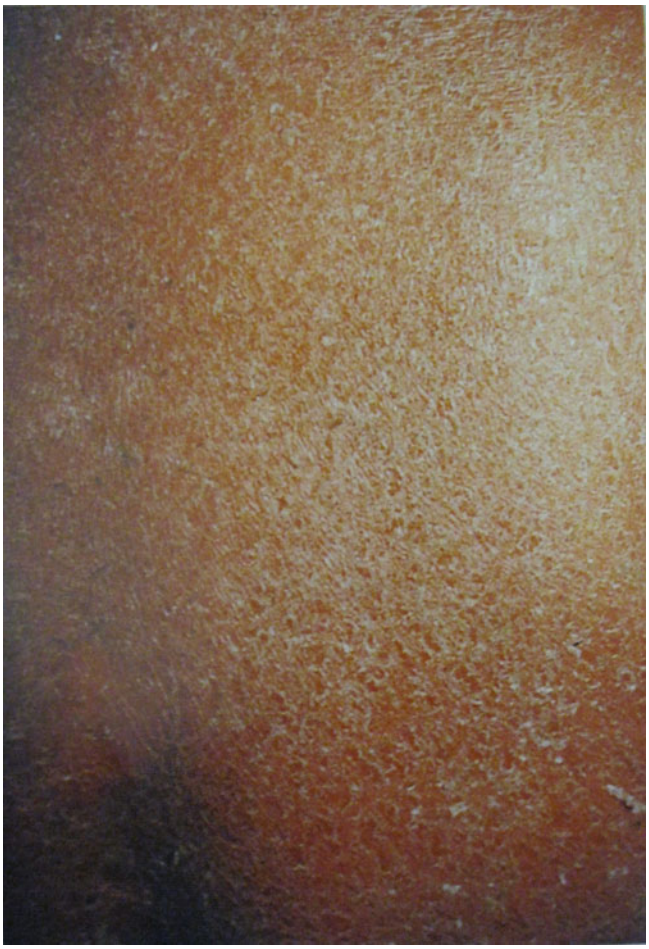


Fig. 1.36 Close up view of desquamative erythroderma

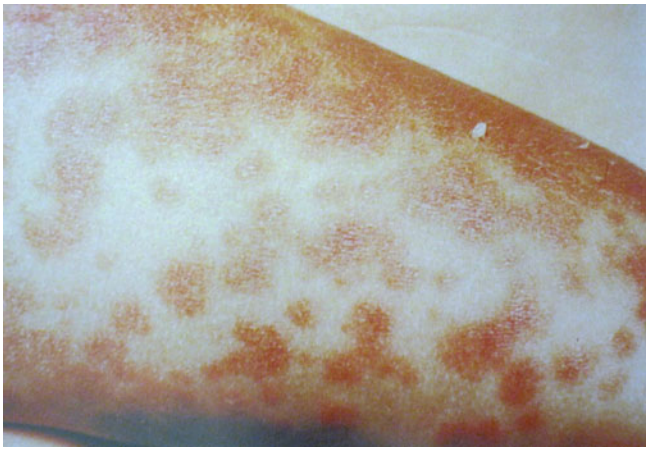


Fig. 1.37 Erythema multiforme lesions demonstrate non-blanching erythema and epidermal necrosis (Reproduced with permission from Newcomer and Young [5] Decreased responsiveness to growth)

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Part II

Inflammatory Conditions

Contact Dermatitis

2

Clinical Description

In the elderly, allergic and irritant contact dermatitis presents as variable, patchy erythema, often without vesicles (in contrast to younger patients) but with pruritus and/or burning.

Etiology and Pathophysiology

Contact dermatitis is a generic term applied to acute or chronic inflammatory reactions to substances that come in contact with the skin. Irritant contact dermatitis (ICD) is caused by a chemical irritant. Since ICD is a toxic phenomenon, it is confined to the area of exposure and is therefore sharply marginated and remains localized. Allergic contact dermatitis (ACD) occurs by an antigen (allergen) that elicits a type IV (cell-mediated or delayed) hypersensitivity reaction. ACD is an immunologic reaction that involves the surrounding skin and may spread beyond affected sites.

Topical preparations containing ingredients such as vitamin E, aloe vera, fragrances, parabens. quarternium 15, diphenhydramine (Benadryl spray, Caladryl lotion), neomycin (Neosporin). and PABA (para-amino benzoic acid) are common allergenic offenders [1, 2]. Overuse of soaps, cleansers, moisturizers, and cosmetics can produce irritation [3].

Histopathology

Histopathologic investigation reveals subacute dermatitis with spongiosis and lymphocytic (allergic) and/or neutrophilic (irritant) exocytosis.

Diagnosis

Patch testing can be of value when properly performed [4, 5]. The site of contact dermatitis suggests possible causes.

Differential Diagnosis (Table 2.1)

Because inflammation is attenuated in the elderly, other causes of generalized pruritus, including metabolic, infectious, and neoplastic conditions, must be considered. Unusual or persistent eczematous eruptions in the elderly warrant a careful search for neoplasms, infections, metabolic diseases, and drug ingestion. Sulfa-related medications are a common cause of contact dermatitis and phototoxic reactions.

Therapy

1. Discontinue use of offending topical agent(s) [15, 16]
2. Mild or mid-strength steroids are preferable to high-potency topical steroids in the elderly to avoid atrophy
3. Apply soothing, cool compresses, followed by bland emollients.

Prognosis

The prognosis is good once the offending agent(s) has been identified and removed.

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Table 2.1 Regional clues to contact dermatitis [6–16]

Face
Cosmetics
Perfume
Topical medication
Nail polish
Hair dye
Hair spray
Shaving lotion
Acne medication
Hatband
Man's beard
Cell phone
Ears
Hearing aid
Earphone
Ear drops
Earrings
Eyelids and periorbital area
Mascara
Eye drops
Nail polish (hand contact)
Pollens
Dust
Hair spray
Hair dye
Eyeglasses
Mouth, lips and periorbital area
Lipstick
Topical corticosteroids
Toothpaste
Mouthwash
Chewing gum
Fruit
Spices
Neck
Cosmetics
Perfume
Acne medication
Necklace
Scarf
Fur
Hands and forearms
Occupational substance
Ring
Plants
Gloves
Dust
Wristwatch
Topical medication
Anal region
Suppository
Antibacterial
Antiperspirant
Feet
Shoes, Footwear
Antifungal
Antiperspirant
Axillae
Deodorant
Antiperspirant
Genital area
Condom
Pessary
Clothing
Hygiene product
Topical medication
Things and lower legs
Clothing
Keys
Coins
Topical medication
Abdomen, chest
Rubber waistband
Pacemaker implant
ECG pads
Transdermal medication patches 3, 6-14

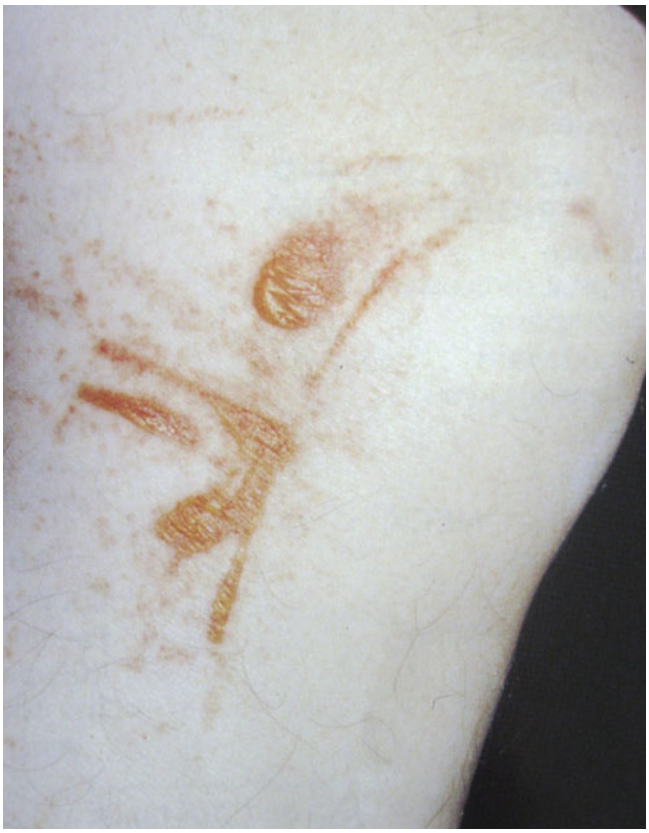

Fig. 2.1 Linear vesicles and erythema are characteristic of poison oak and poison ivy (Rhus) dermatitis

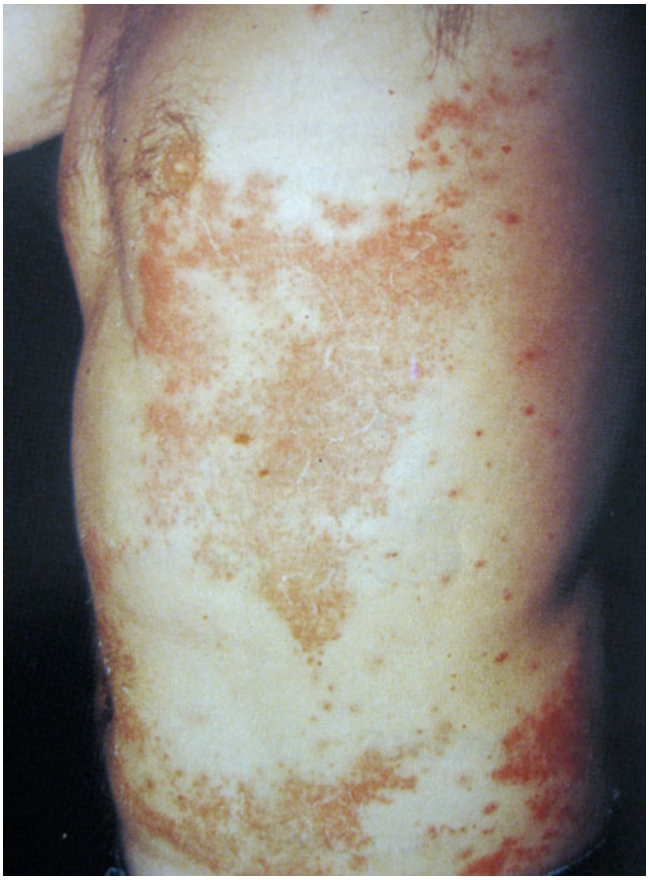

Fig. 2.2 Generalized poison oak or poison ivy dermatitis

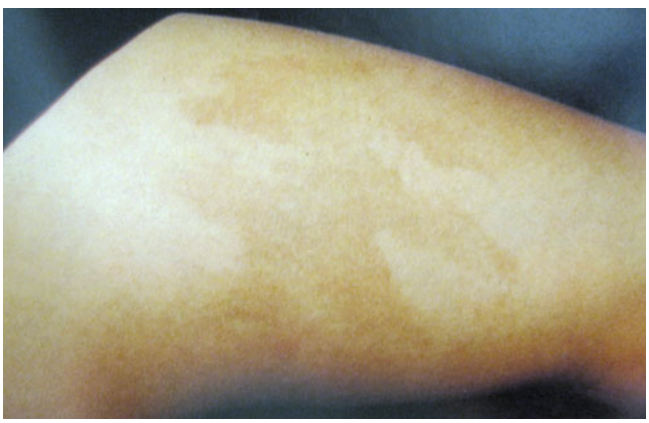

Fig. 2.3 Hyperpigmented streaks suggested berloque dermatitis (phytophotodermatitis) caused by citrus furocoumarins in perfume, cologne, and certain lemon and lime peel oils

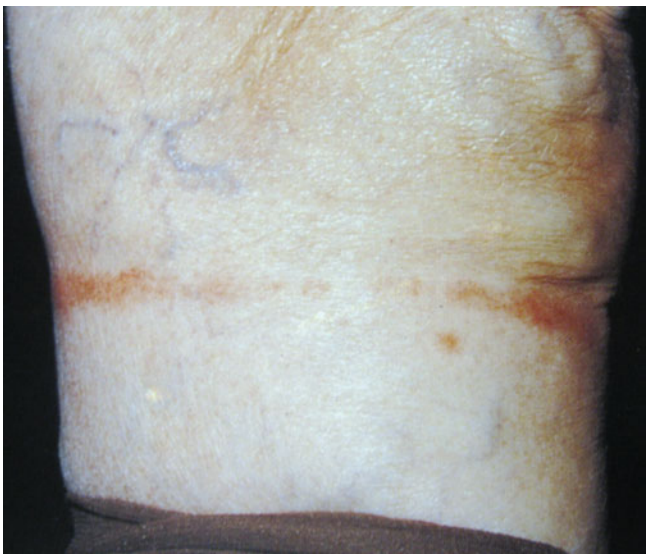

Fig. 2.4 Rubber dermatitis from elastic stockings

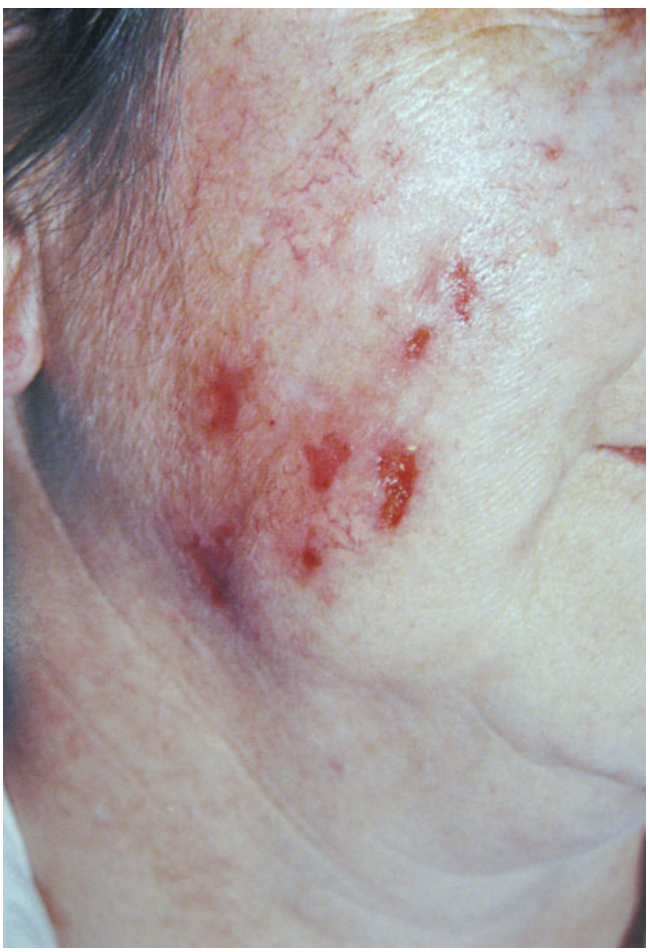

Fig. 2.5 Factitial (self-induced) dermatitis, also called dermatitis artefacta in a patient with delusion of parasitosis

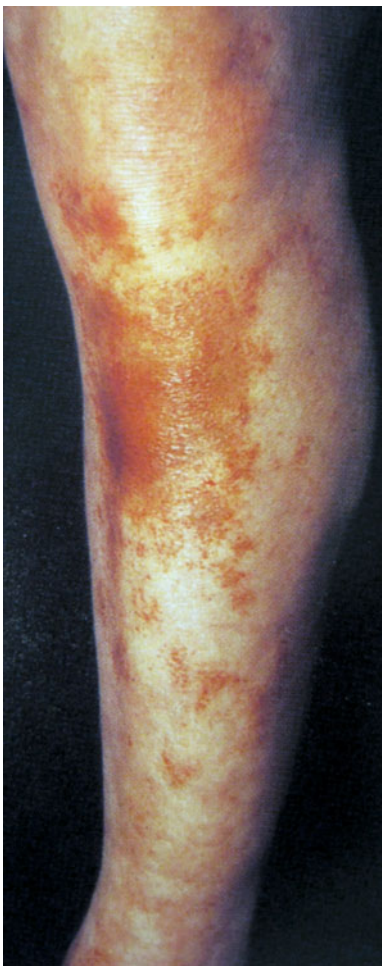

Fig. 2.6 Stasis dermatitis complicated by application of topical Neosporin ointment

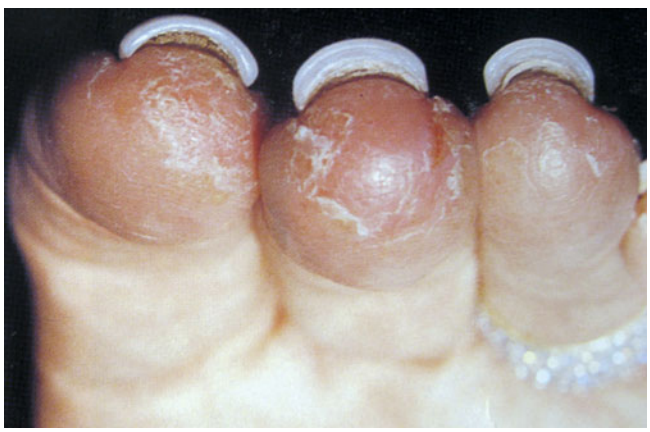

Fig. 2.7 Contact allergic dermatitis from artificial acrylic nails

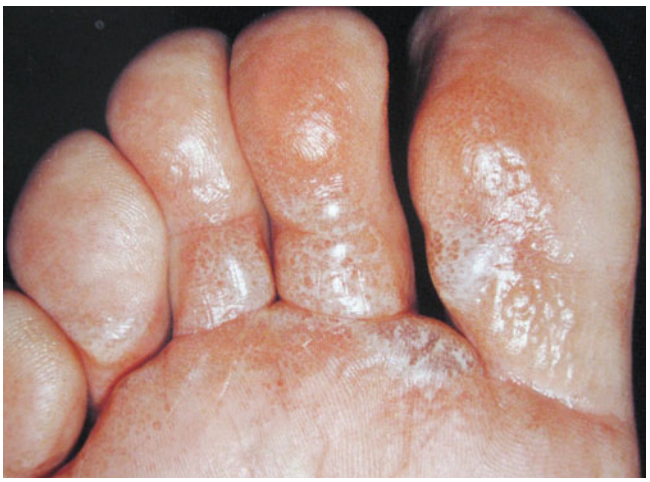

Fig. 2.8 Vesicular contact allergic dermatitis from shoe materials (leather, rubber, or glue)

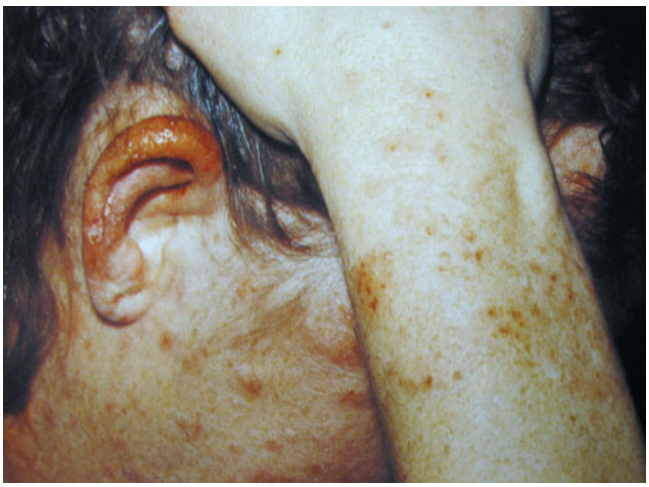

Fig. 2.11 Hair dye (paraphenylenediamine) dermatitis affects the ears, neck, and hands

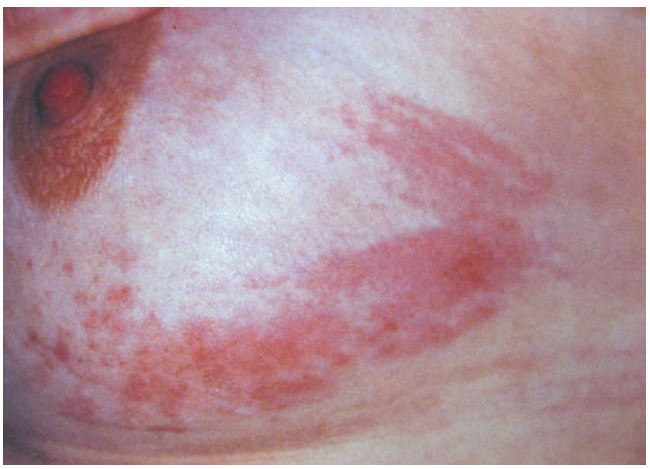

Fig. 2.9 Nickel dermatitis from an underwire bra

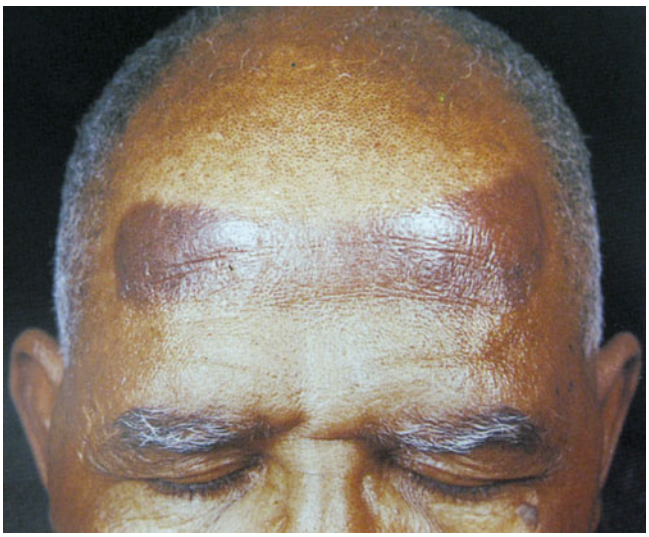

Fig. 2.12 Hatband dermatitis

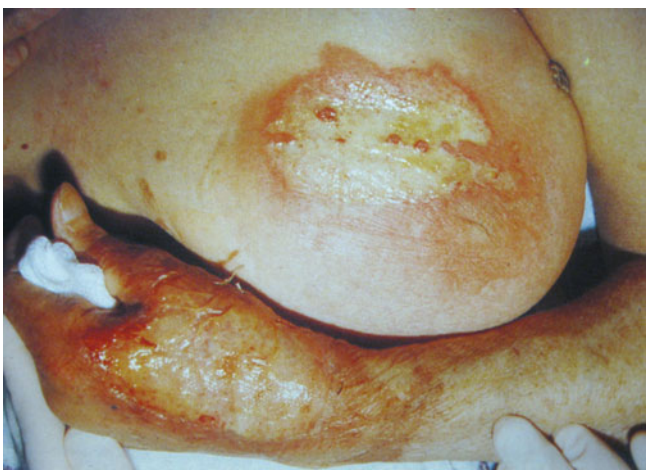

Fig. 2.10 Second-degree hot water burn from a pressure cooker

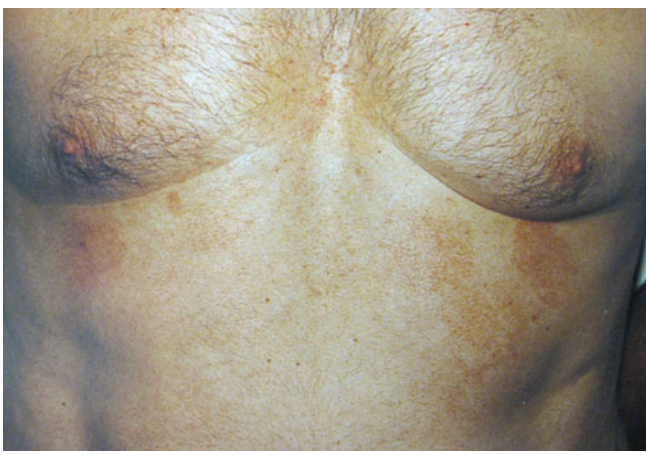

Fig. 2.13 Contact dermatitis from nitropaste (nitroglycerin transdermal patch)

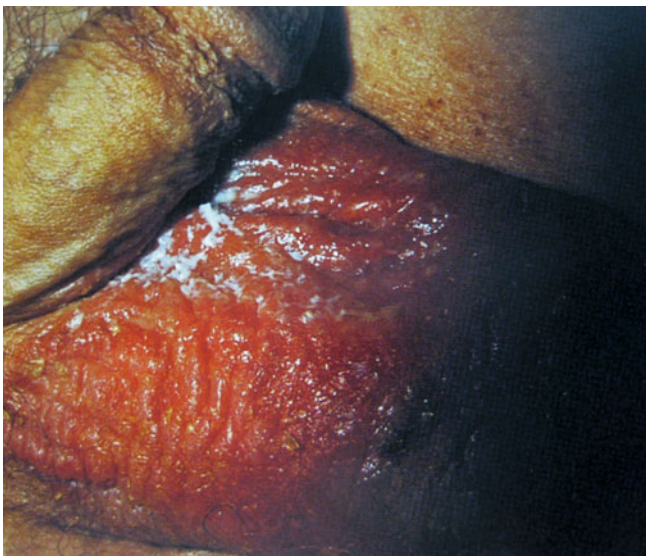

Fig. 2.14 Acute exudative contact dermatitis of the scrotum from Bounce fabric softener

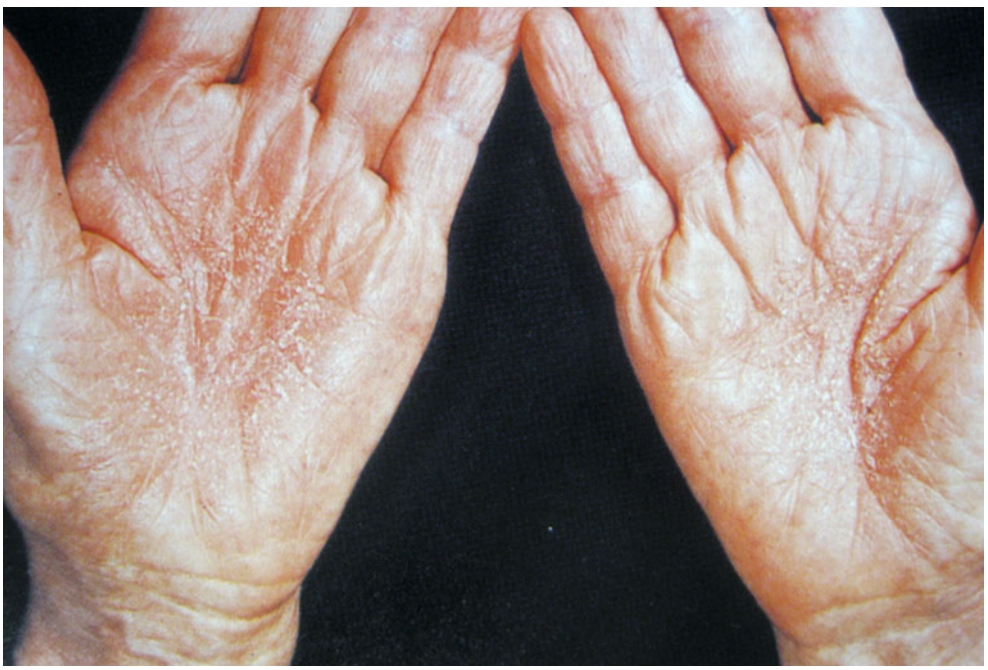

Fig. 2.15 Palm eczema may be aggravated by various topical allergens or irritants and by overwashing, certain foods (citrus, shellfish), emotional stress, infections (tinea pedis) or, occasionally, internal malignancies

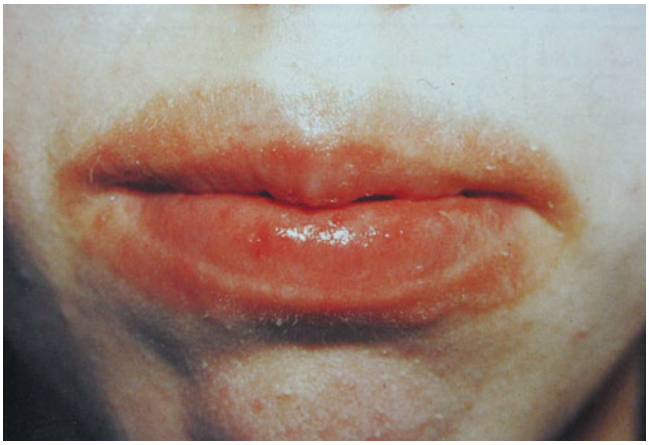

Fig. 2.16 Cheilitis venenata, contact dermatitis of the lips, caused by lip balm containing PABA, vitamin E, and aloe vera, three common allergens

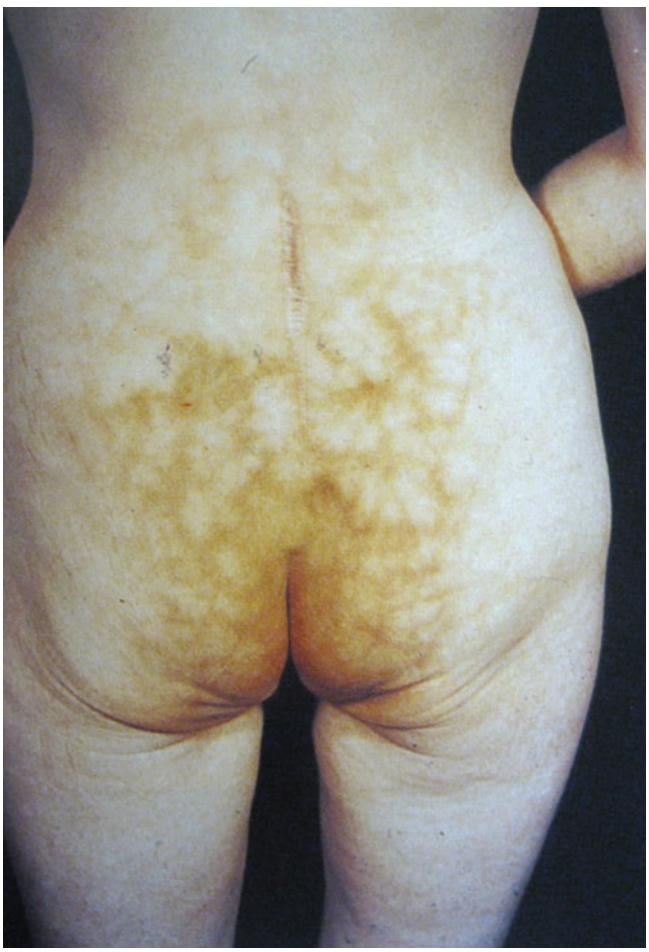

Fig. 2.17 Erythema ab igne ("*redness* from the fire"). This poikilodermatous (hyperpigmentation and hypopigmentation, telangiectasia, and atrophy) contact dermatitis is caused by excessive use of a heating pad

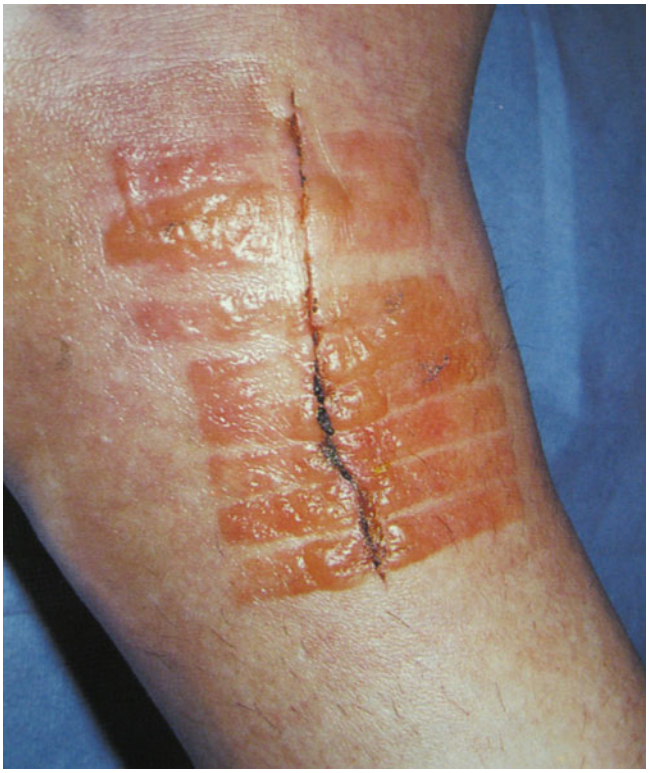

Fig. 2.18 Contact allergic dermatitis from Steri-Strip adhesive. (Reproduced with permission from Newcomer and Young [17])

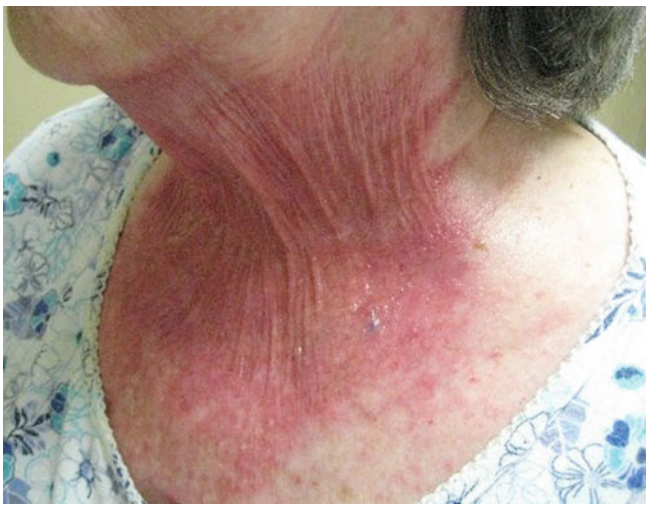

Fig. 2.19 Irritant dermatitis on the neck

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Androgenetic Alopecia

3

Synonyms

Synonyms include male and female pattern baldness.

Clinical Description

Androgenetic alopecia is a common disorder affecting both men and women. Androgenetic alopecia evolves differently in men and women. The incidence of male pattern baldness is generally considered to be greater than female androgenetic alopecia. The onset of adrogenetic alopecia is gradual and shows a transition from large, thick, pigmented terminal hairs to thinner, shorter hairs and finally to short, flaky, non-pigmented vellus hairs. More hairs are presented in the telogen (resting) phase due to shortening of the anagen phase as baldness progresses.

1. Male androgenetic alopecia – gradual thinning in temporal and frontal areas reshaping the anterior hairline.
2. Female androgenetic alopecia – diffuse thinning on the crown while generally maintaining the frontal and temporal hairline; [2] loss of frontal and temporal hairline warrants a search for an underlying androgen-secreting tumor.

Etiology and Pathophysiology

Androgenetic alopecia is a genetically determined condition inherited from either parent. There is strong evidence for a linkage to androgenetic alopecia on the 3q26 site on the X chromosome. The chromosome 20pII and the androgen-receptor gene also have an association to the cause of the condition. It may also be of polygenic inheritance due to evidence that follicles from balding areas of persons with pattern baldness are able to produce terminal hairs when implanted into immunodeficient mice. Androgen is necessary for the progression of alopecia, however it may not be the only factor contributing to it. Alopecia in mice has been reported in those that lack functional Vitamin D receptors due to inability for cyclic regeneration of hair follicles. Further research is needed to show a link to humans. The conversion to vellus-like follicles can begin at puberty and continues throughout adulthood.

Laboratory and Clinical Examinations

Serology ANA (to rule out SLE); rapid plasma reagin (RPR) test (to rule out secondary syphi-lis, CBC (Rule out iron-deficiency anemia), Chemistry--Serum iron, iron-binding capacity, TSH (Rule out thyroid disease)

In women with hair loss and evidence of increased androgens (menstrual irregularities, infertility, hirsutism, severe cystic acne, virilization), determine the

Testosterone: total and free, Dehydroepiandrosterone sulfate (DHEAS), and Prolactin

KoH Preparation (Rule out tinea capitis)

Scalp biopsy

Hair Pull test

Histopathology

A biopsy is usually unnecessary to make a diagnosis. If a biopsy is taken, it is usually sectioned transversely and may help in distinguishing non-scarring alopecias. In pattern alopecia, there is a decrease in the number, density, size, and caliber of the terminal hair follicles. An increased telogen-to-anagen ratio is often observed. The attached sebaceous glands may be preserved or experience hypertrophy.

R.A. Norman, E.M. Young, Jr, *Atlas of Geriatric Dermatology*,
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Differential Diagnosis

Alopecia Areata. It is a recurrent non-scarring type of hair loss that can affect any hair-bearing area *completely*. Onset can be sudden and hair loss can be asymmetrical, localized, or generalized. It is medically benign but can cause emotional and psychosocial distress in patients.

Telogen effluvium. This is a sudden onset of hair loss characterized by diffuse hair shedding. It is caused by severe illness, metabolic or hormonal stress, or medications, resulting from the transition of anagen phase to telogen phase of the hair cycle. Cases of telogen effluvium often occur in patients with underlying androgenetic alopecia. Treatable causes of telogen effluvium (eg, anemia, hypothyroidism) should be sought. Hair Pull test--Compared with the normal pull, in which 80–90 % of hair is in the anagen phase, telogen effluvium is characterized by a reduced percentage of anagen hairs that varies with the intensity of hair shedding.

Anagen effluvium. This onset of hair loss occurs after insult to the hair follicle that impairs its metaboloic activity. It is usually the result of chemotherapeutic agents. Tapered fractures in the hair shafts caused by damage to the matrix are characteristic of the condition. Pemphigus vulgaris has been reported to be a cause also.

Trichotillomania (**Complusive Hair Pulling**). Patches if broken hairs of irregular length are present, associated with psychiatric disturbances ranging from neurosis to depression to psychosis.

Tinea. Scales, pustules, and broken hairs, usually localized in patches, can be noted. Fungal hyphae and spores are discernible on potatssium hydroxide examination and culture.

Syphillis. Typically moth-eaten patchy alopecia, and other features of secondary syphilis (e.g., positive VDRL test result, papulosquamous lesions on trunk, palms, soles) are present.

Other problems to be considered include hypertension and smoking.

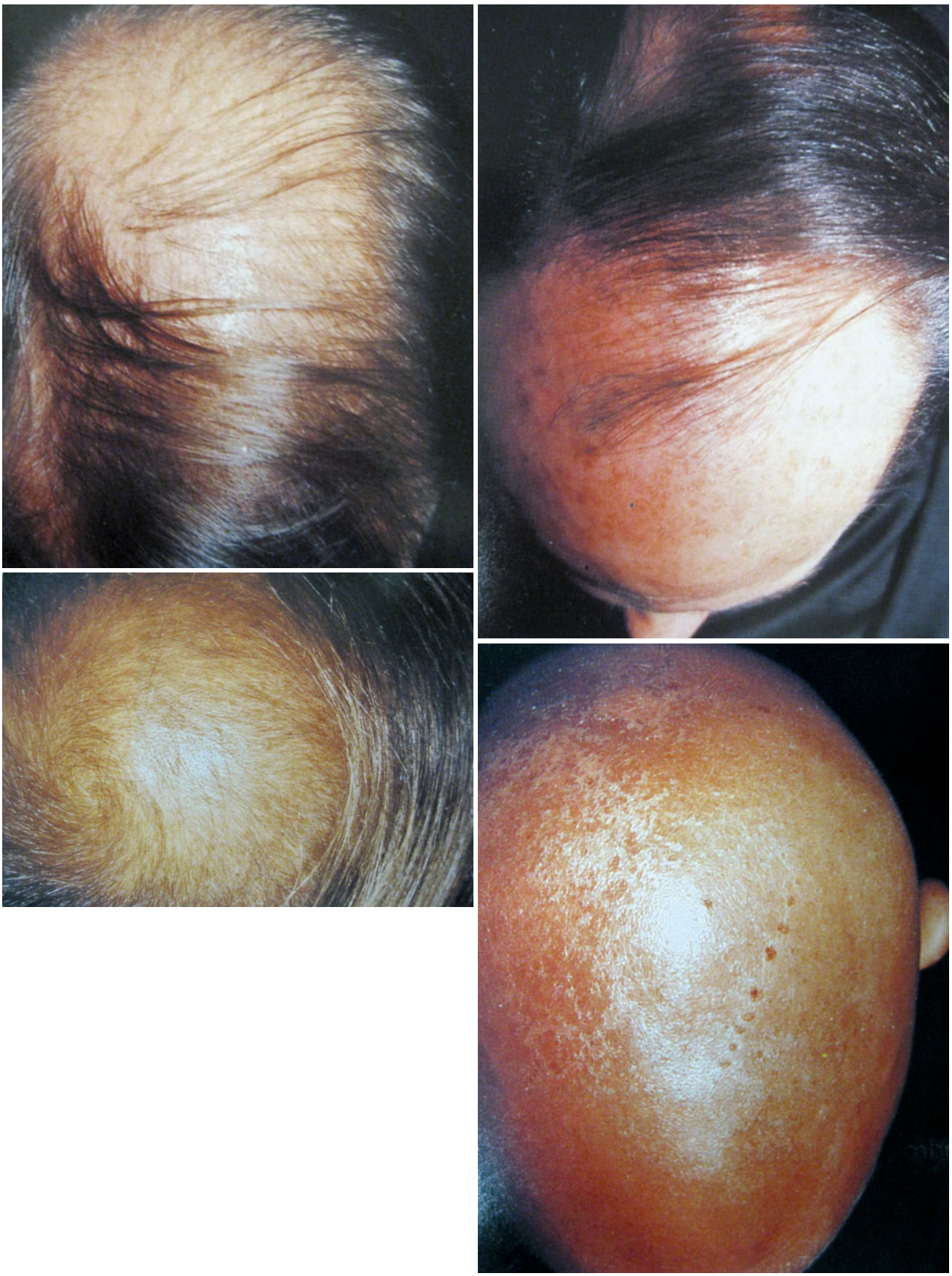
Therapy

The only two drugs that have been approved by the FDA for treatment of androgenetic alopecia are minoxidil and finasteride.

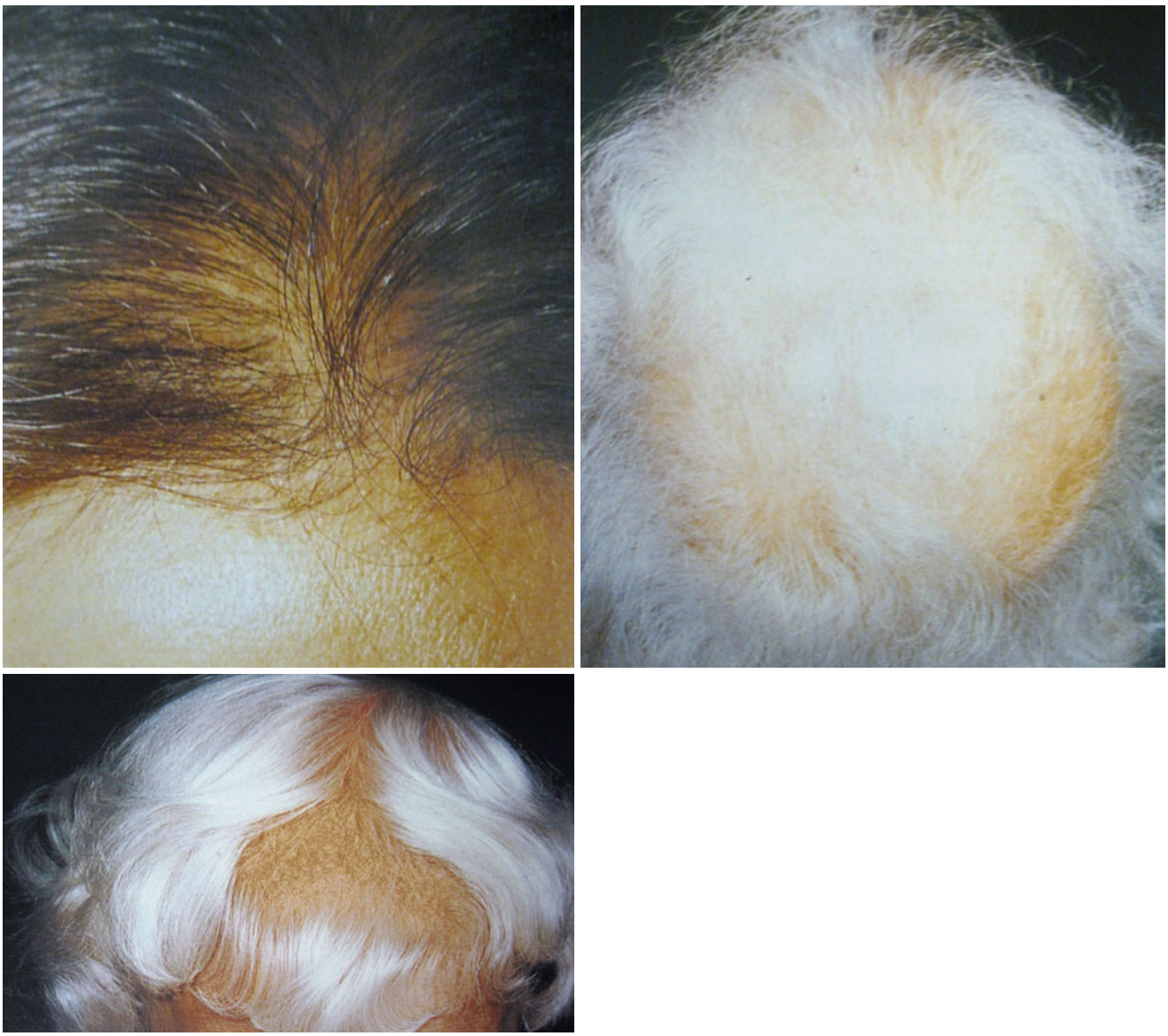
1. Minoxidil. The drug is marketed as a 2 or a 5 % topical solution, with the 5 % solution being somewhat more effective. It appears to lengthen the anagen phase and may increase blood supply to the follicle. Blood pressure, pulse, and weight should be monitored. Discontinuation produces a rapid reversion to pretreatment conditions. Side effects may include scalp irritation, headache, vision changes, and increased growth of facial hair.
2. Finasteride. This drug is given orally and is a 5-alpha reductase type 2 inhibitor. It can be used only in men because it can produce ambiguous genitalia in a developing male fetus. Finasteride has been shown to diminish the progression of androgenetic alopecia in males who are treated, and, in many patients, it has stimulated new regrowth.
3. Other drugs and therapy not approved by the FDA but may be potentially helpful include androgen suppressants or antagonists for women or low-level laser light therapy.
4. Other cosmetic treatments include hair transplantation (may cause infection), scalp flap, toupe, wig, mousse, conditioner, changing hair style, and coloring. Sometimes surgical treatment can be successful with satisfactory cosmetic results.

Prognosis

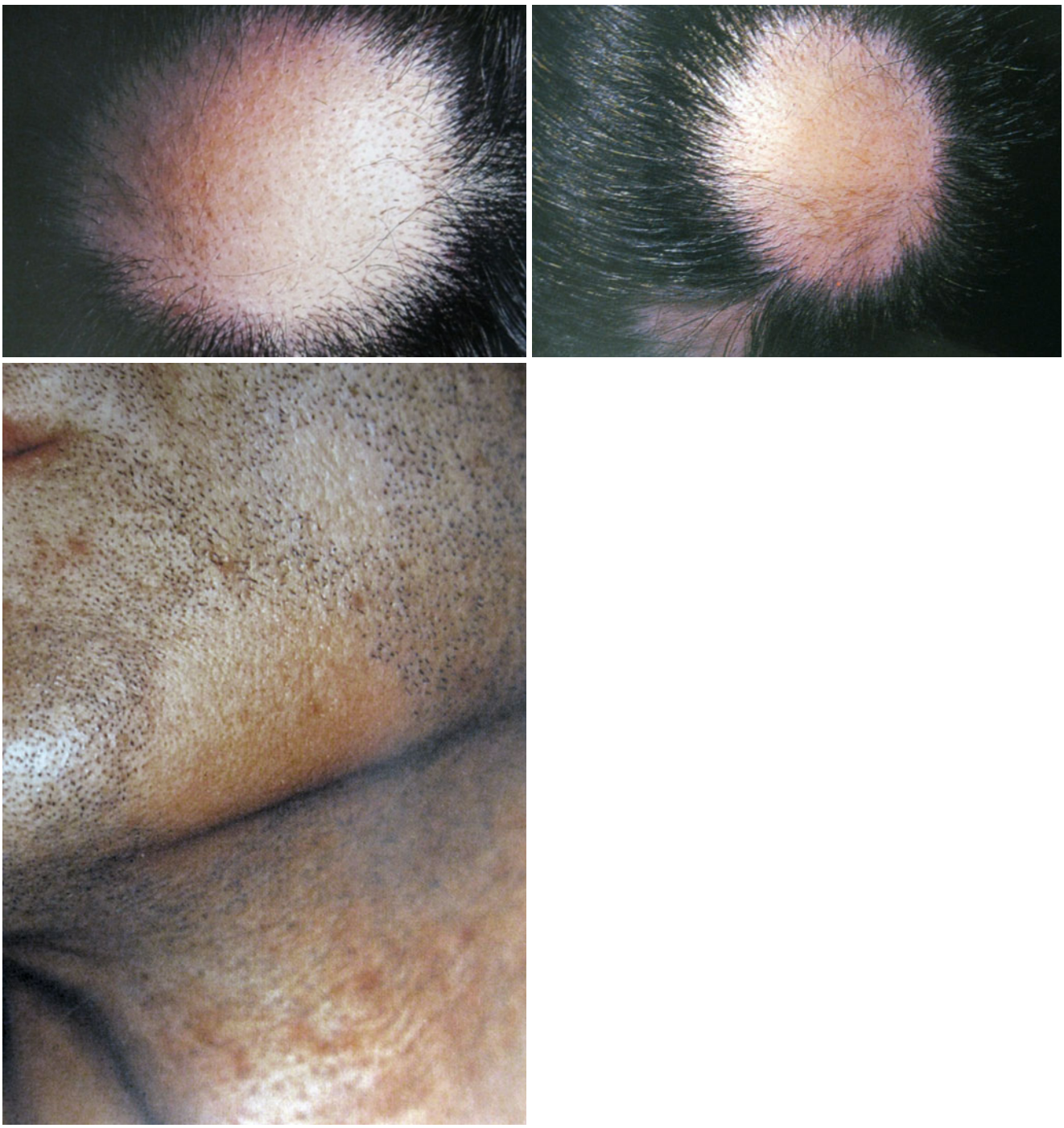
The prognosis is usually poor. Some patients progress to the point of complete baldness. Others may have patterned or unpatterned thinning, while retaining hair. Women who are treated may show a slow thinning of the crown, avoiding a complete hair loss in affected areas [1–6].



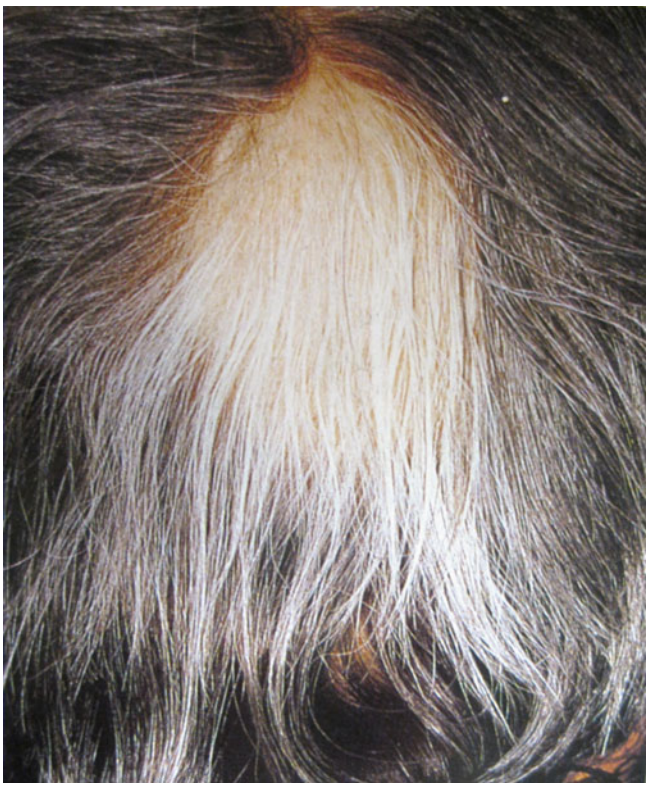
Figs. 3.1, 3.2, 3.3, and 3.4 Male pattern alopecia follows a predictable pattern (Hamilton stages): frontotemporal recession, followed by vertex balding. The occipital fringe is relatively resistant to hair loss. This is where donor follicles for hair transplantation are harvested

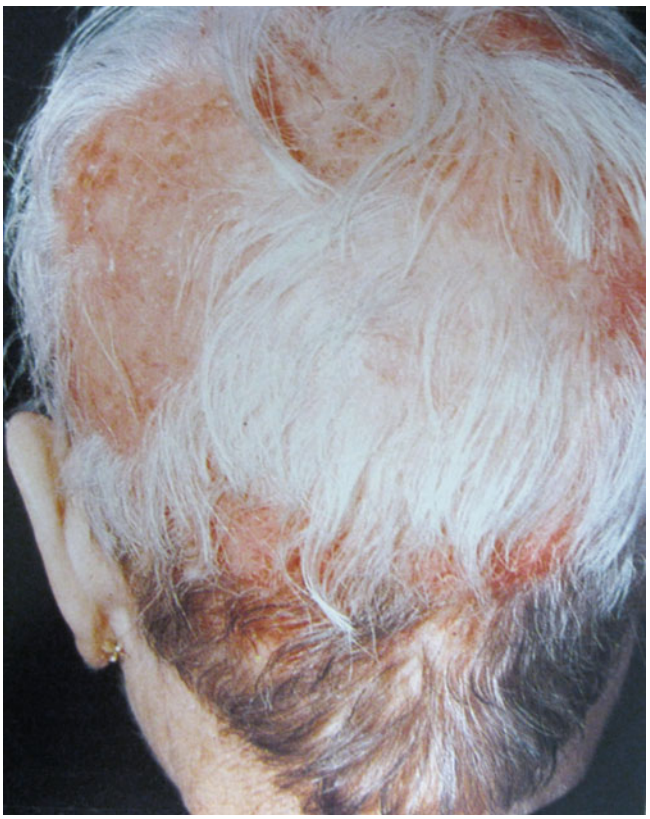


Figs. 3.5, 3.6, and 3.7 Female pattern alopecia demonstrates diffuse hair thinning with visible scalp showing through. Note sparing of the frontal hairline. The absence of the frontal hairline in women suggests hyperandrogenism from androgen-secreting tumor



Figs. 3.8, 3.9, and 3.10 Alopecia areata shows a localized bald area or areas with short, stubby, exclamation point hairs at the periphery





Figs. 3.11 and 3.12 Alopecia areata preferentially affects pigmented terminal hairs, with relative sparing of canities (gray or white hairs). This phenomenon can be localized, as shown here, or generalized. Patients who claim to have "turned gray" overnight actually have this variant of alopecia areata

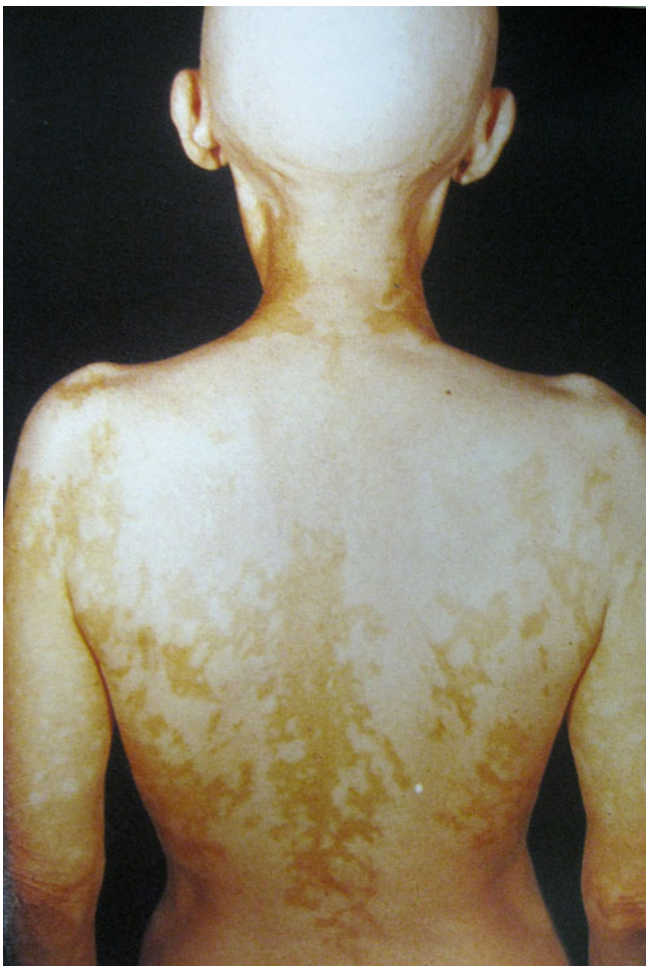
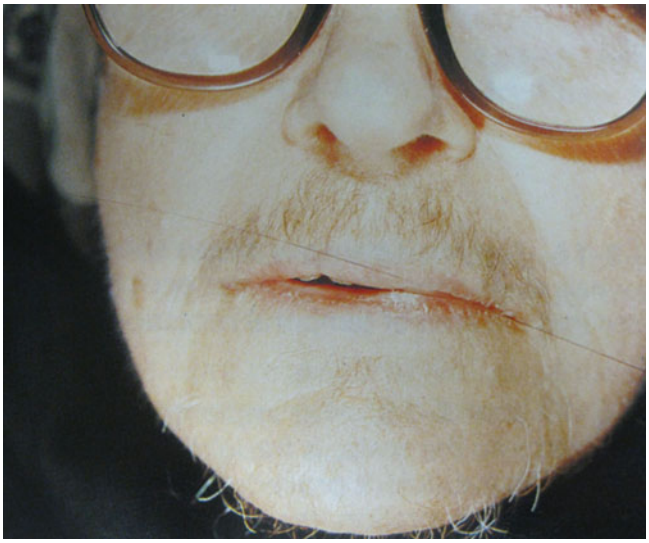
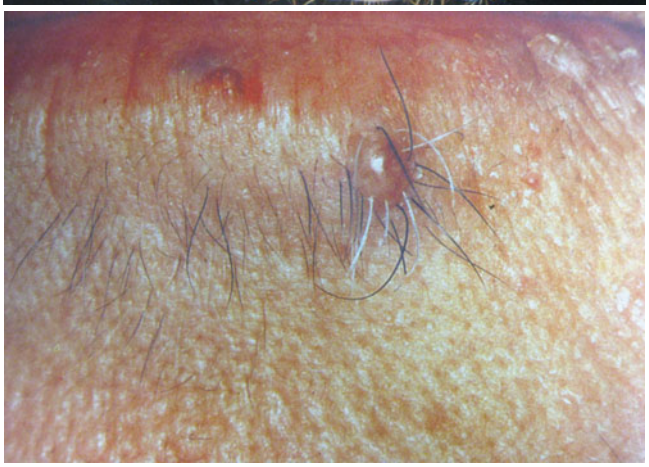


Fig. 3.13 Alopecia universalis is the total absence of body, scalp, and facial hair





Figs. 3.14 and 3.15 Hirsutism, usually a genetic variant, less commonly caused by androgen excess, is a vexing cosmetic problem in elderly women

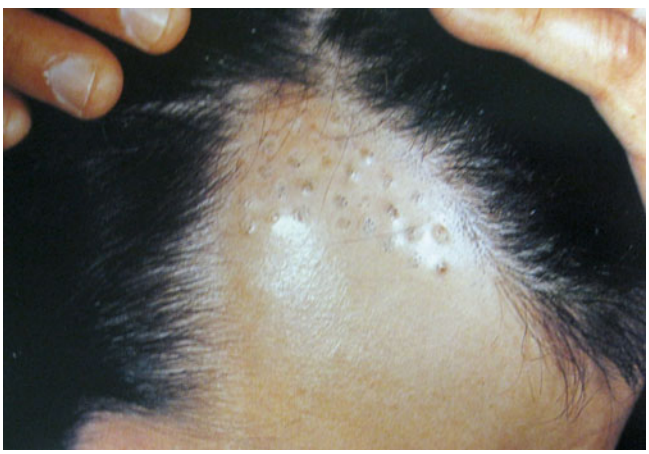
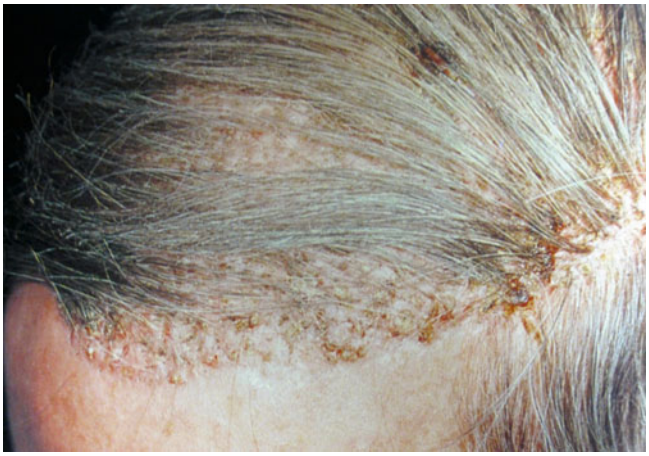
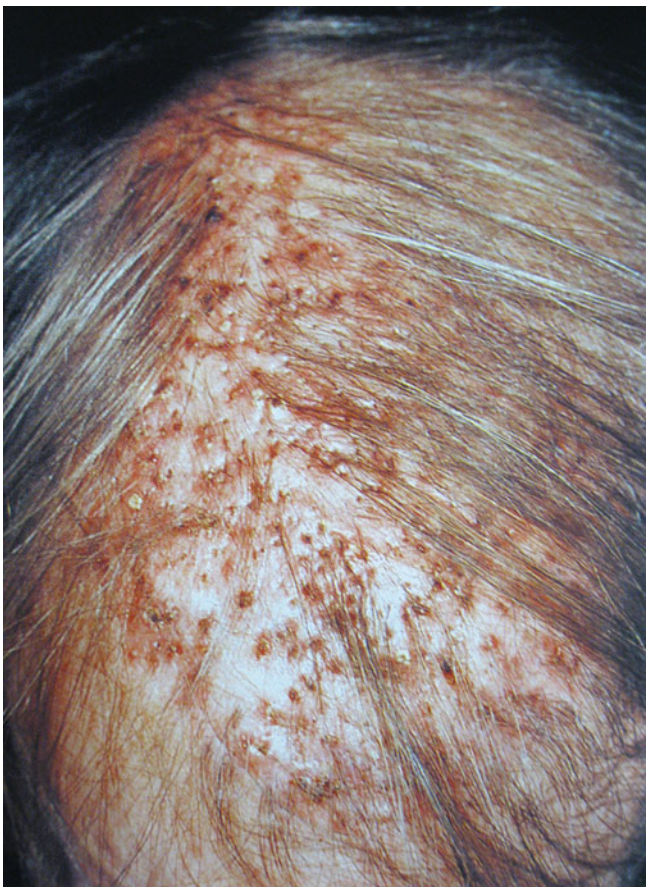


Fig. 3.16 Early hair transplantation shows insertion of donor hair plugs harvested from the occipital hairline and planted into the area of temporal recession





Figs. 3.17 and 3.18 Hair implantation, the insertion of foreign hairs into the scalp, is fraught with disastrous complications. This fraudulent procedure is in marked contrast to that of hair transplantation, the insertion of autologous plugs from the occipital scalp onto the balding scalp. Shown here are necrosis, keloid formation, and the rejection of implanted foreign hair

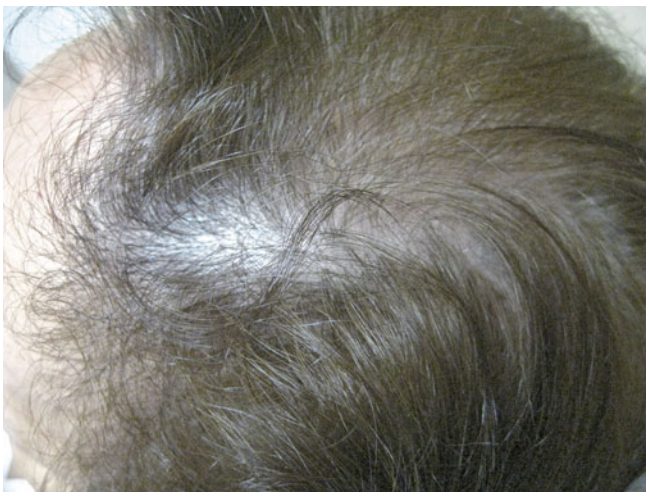


Fig. 3.19 Early pattern of hair loss

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Bullous Pemphigoid

4

Clinical Description

The dermatological manifestiations of bullous pemphigoid can be either subacute or acute, with widespread, tense bullae (blisters 1 cm or more in diameter). Pruritus is an accompany secondary effect. Urticarial lesions may also be present before blisters of the vesicular form. Generalized erythroderma can resemble psoriasis, atopic dermatitis and other conditions with exfoliative erythroderma. Pemphigoid nodularis has clinical features that resemble prurigo nodularis, with blisters arising on normal-appearing or nodular lesional skin. The incidence of bullous pemphigoid appears to be equal in men and women. The average onset is about 65 years of age. BP has been seen in children, but is rare.

Etiology and Pathophysiology

Bullous pemphigoid is a chronic autoimmune, subepidermal, blistering skin disease. It is characterized by the presence of immunoglobulin G (IgG) autoantibodies specific for the hemidesmosomal bullous pemphigoid antigens BP230 (BPAg1) and BP180 (BPAg2). The IgG autoantibodies bind to the skin basement membrane in patients with BP. This activates the complement and inflammatory mediators which in turn attracts inflammatory cells to the basement membrane. These cells release proteases, which degrade hemidesmosomal proteins and lead to blister formation. Serum levels of autoantibodies against BPAg2 are reportedly correlated with disease activity in some studies [1]. They are found to deplete cultured keratinocytes of the BPAg2 and weaken cell attachment in vitro, which further supports the pathogenic role of these autoantibodies [2].

Diagnosis

Diagnosis of bullous pemphigoid can be established by performing histophathologic analysis from the edge of a blister and direct immunoflourescence (DIF) of perilesional skin. DIF tests usually demonstrate IgG (70–90 % of patients) and complement C3 deposition (90–100 % of patients) in a linear band at the dermal-epidermal junction. If DIF is positive, an indirect immunoflourescence should be performed using the patient's serum. IDIF studies document the presence of IgG circulating autoantibodies in the patient's serum that target the skin basement membrane component. Other tests include Immunoblotting, Immunoprecipitation, ELSA, and Immunohistochemistry [3, 4].

Histopathology

Histopathologic investigation reveals subepidermal bullae with eosinophils. Eotaxin, an eosinophil-selective chemokine, is strongly expressed in the basal layer of the epidermis of lesional bullous pemphigoid skin. There is a correspondence with the accumulation of eosinophils in the skin basement membrane zone area.

Differential Diagnosis

Cicatricial Pemphigoid. This rare chronic autoimmune blistering disease affects the mucous membranes, including the conjunctiva and surrounding skin. Patients present with tense blisters and erosions on the head and neck or at sites of trauma [5].

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Dermatitis Herpetiformis (**DH**) is an autoimmune blistering disorder associated with a gluten-sensitive enteropathy (GSE) is characterized by grouped excoriations; erythematous, urticarial plaques; and papules with vesicles. The classic location for dermatitis herpetiformis lesions is on the extensor surfaces of the elbows, knees, buttocks, and back.

Drug-Induced Bullous Disorders. Bullous or blistering drug eruptions and drug-induced anaphylaxis and hypersensitivity syndromes are among the most serious types of adverse drug reactions. Withdrawal of the offending medication is the most important aspect of treatment of bullous drug reactions

Erythema Multiforme. Erythema multiforme may be present within a wide spectrum of severity. Erythema multiforme minor represents a localized eruption of the skin with minimal or no mucosal involvement.

Linear IgA Dermatosis. The classic primary lesions of linear IgA dermatosis are clear and/or hemorrhagic round or oval vesicles or bullae on normal, erythematous, or urticarial skin. Cutaneous manifestations may also include erythematous plaques, blanching macules and papules, or targetoid erythema multiforme–like lesions [6].

Therapy

The goal of therapy is to decrease blister formation, to promote healing of blisters and erosions, and to determine the minimal dose of medication necessary to control the disease process.

1. Corticosteroids, topical and/or systemic in doses sufficient to control bullae formation.
2. Patients treated with a systemic corticosteroid for longer than 1 month, should supplement with calcium and vitamin D should be instituted to prevent osteoporosis. Bisphosphonate, a specific inhibitor for osteoclast-mediated bone resorption (e.g., alendronate), should also be taken.
3. The most commonly used medications are anti-inflammatory agents (e.g., corticosteroids, tetracyclines, dapsone) and immunosuppressants (e.g., azathioprine, methotrexate, mycophenolate mofetil, cyclophosphamide). These agents inhibit the inflammatory process by inhibiting specific cytokine production and vascular permeability. They may also stabilize granulocyte membranes and prevent release of key enzymes [7].

Prognosis

Most patients affected with BP require therapy for months to years. Pemphigoid is difficult to control and long-term remission can be experienced. Patients also have an increased risk for hypertension, diabetes mellitus, and heart diseases. Bullous pemphigoid may be fatal, particularly in patients who are debilitated. The proximal causes of death are infection with sepsis and adverse events associated with treatment.

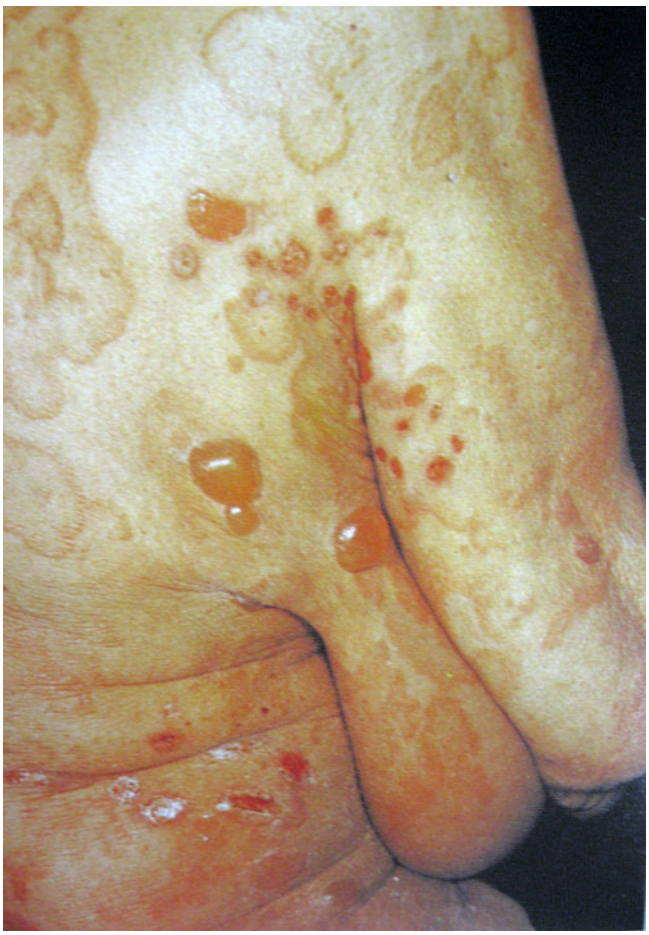
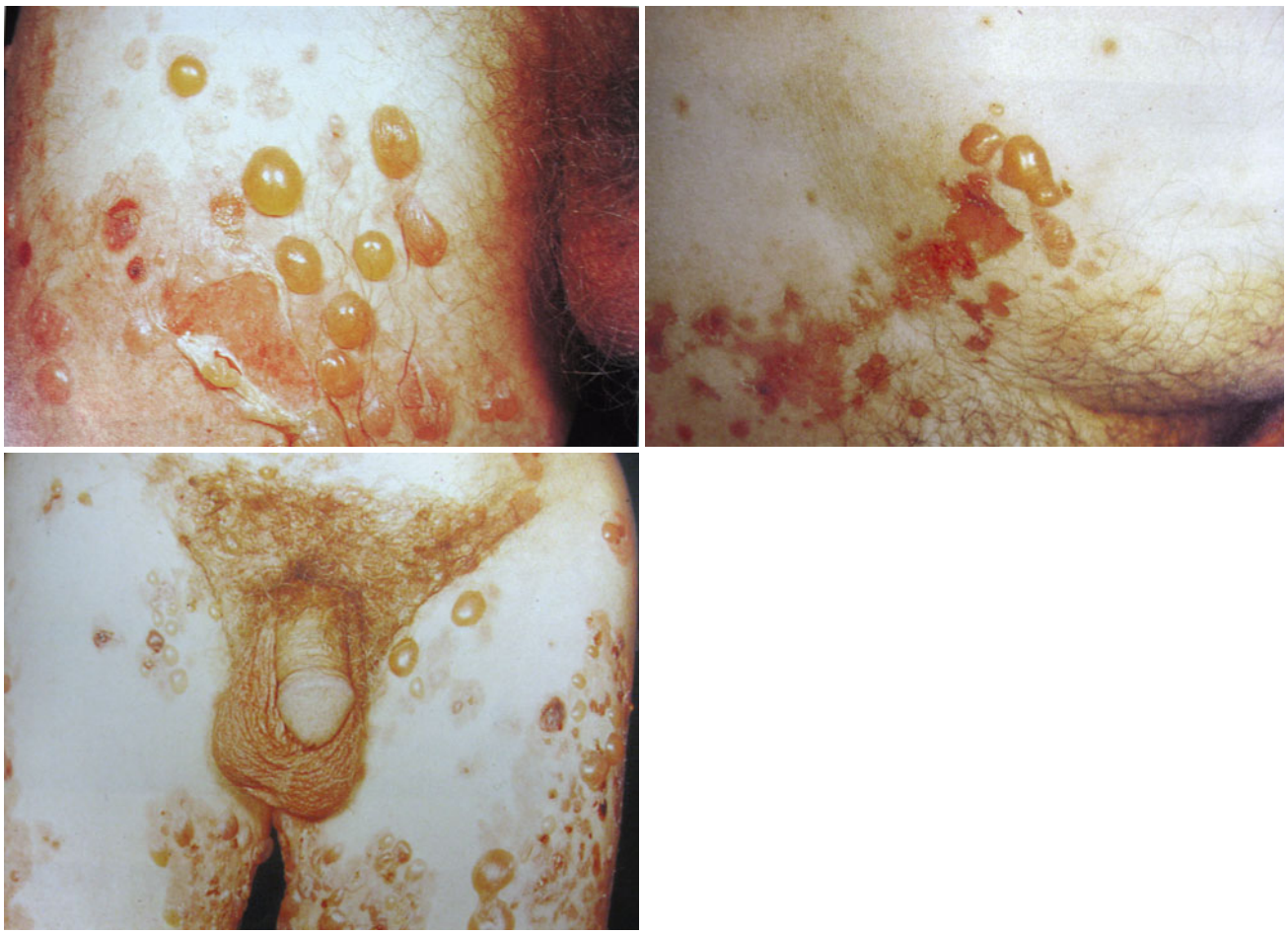


Fig. 4.1 Concomitant annular urticarial *lesions* and tense ounae suggest the diagnosis of bullous pemphigoid



Figs. 4.2, 4.3, and 4.4 Large, *tense* bullae and erosions in intertriginous regions suggest bullous pemphigoid

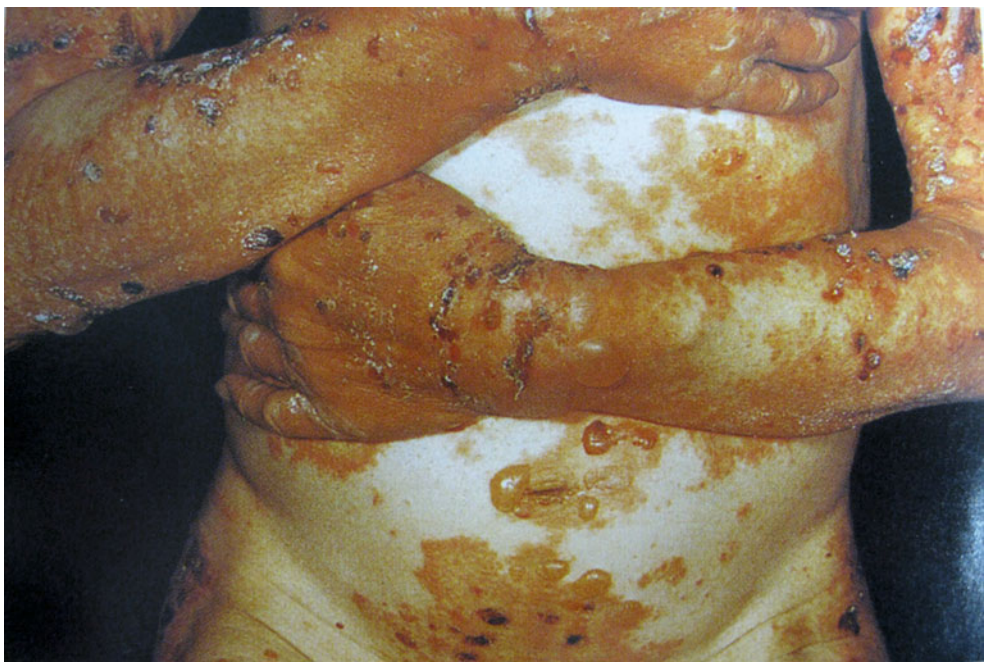


Fig. 4.5 Widespread truncal involvement with erythroderma of arms in bullous pemphigoid

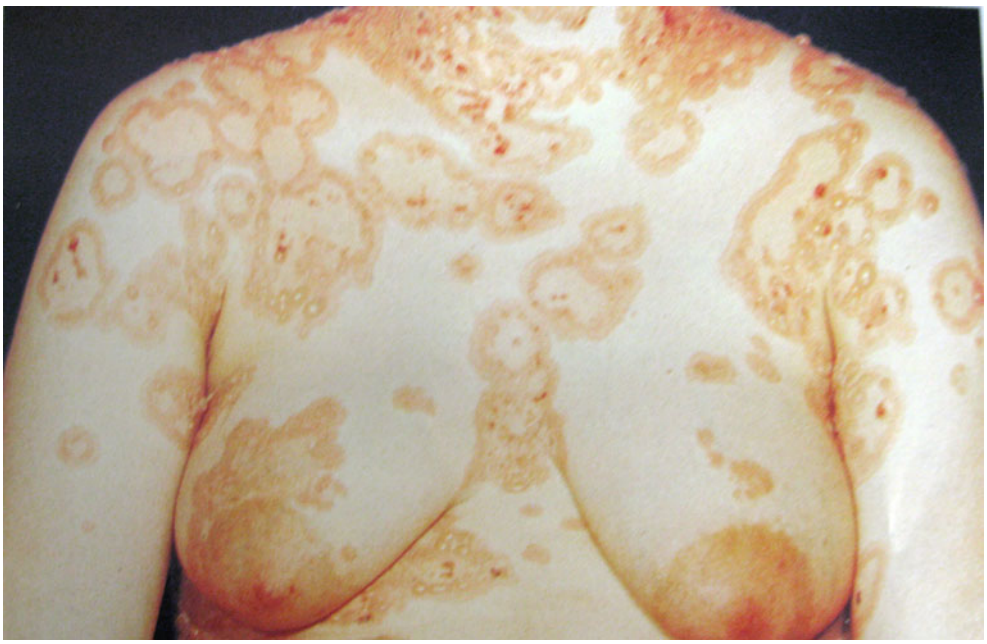
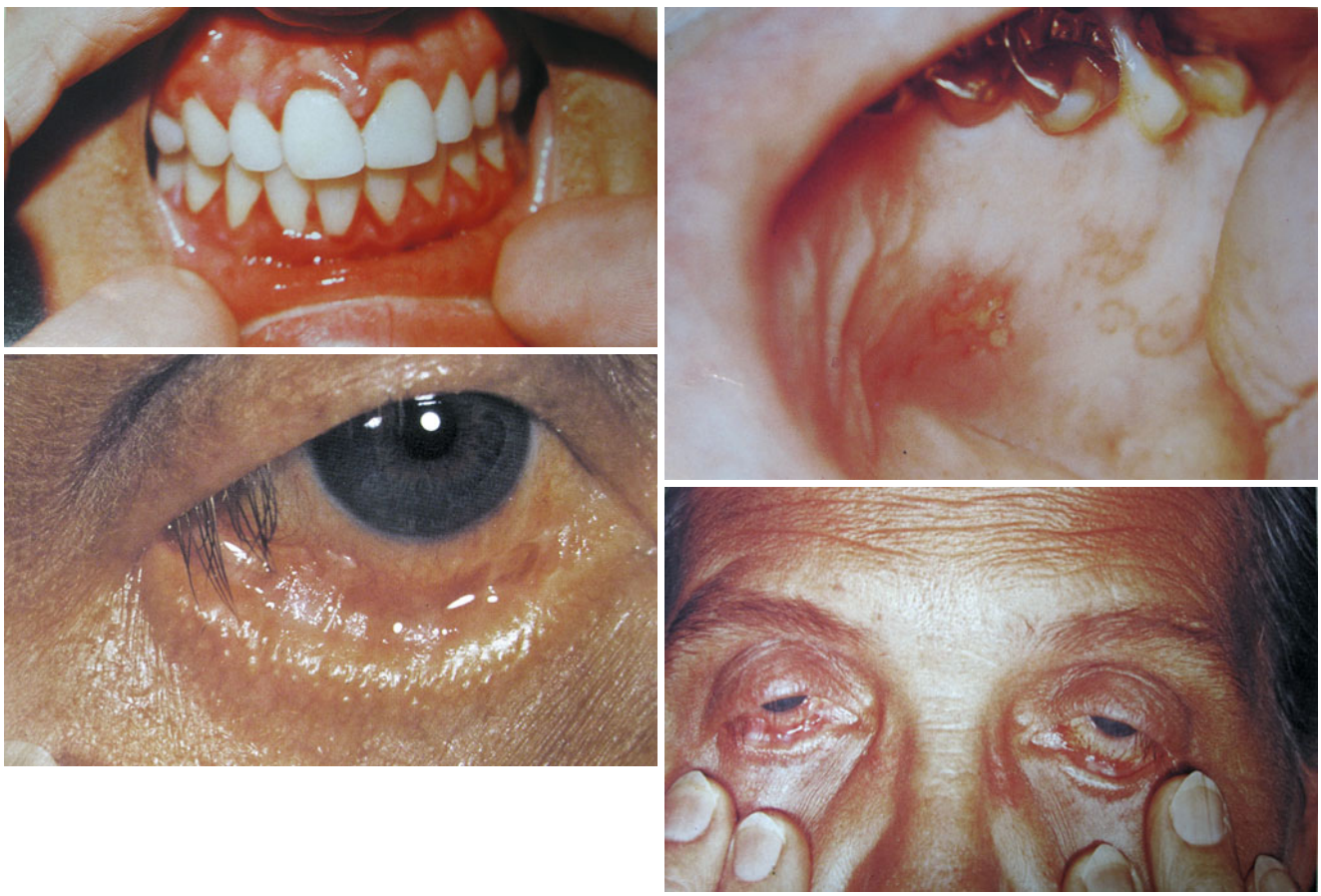


Fig. 4.6 Herpes gestationis is a bullous pemphigoid-like eruption of pregnancy that shares many clinical and histologic features of bullous pemphigoid. Direct immunofluorescence usually reveals linear (not tubular) deposits of C′ 3 along the dermal-epidermal junction with IgG in 40–50 % of the cases. Indirect immunofluorescence detects a herpes gestationis factor (complement-binding IgG anti basement membrane zone antibody) in 25–75 % of patients



Figs. 4.7, 4.8, 4.9, and 4.10 Cicatricial (Brunsting-Perry) pemphigoid features oral lesions (Figs. 4.7 and 4.8), notably desquamative gingivitis (which may also be seen in pemphigus vulgaris), ocular lesions (Figs. 4.9 and 4.10) including symblepharon (scarring conjunctivitis leading to blindness) and trichiasis (lnqrown eyelashes) and esophageal strictures, confirmed radiologically. These devastating complications belie the misnomer "benign mucous membrane pemphigoid"

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Dermatitis Herpetiformis

5

Synonym

This disorder is also known as Duhring's disease.

Clinical Description

Excoriations and tiny papulovesicles occur on the extensor surfaces of the elbows, knees, sacrum, and scalp [1–4]. You may find crusts and erosions on an erythematous base with postinflammatory hypo- and hyperpigmentation.

Etiology and Pathophysiology

Gluten-sensitive enteropathy and dermopathy result from the deposition of IgA in the skin and gut and from the activation of neutrophils.

Diagnosis

Direct immunofluorescence (DIF) of perilesional skin reveals granular deposits of IgA at the derrnoepidermal junction. Indirect immunofluorescence (IIF) of serum detects IgA-antiendomysial antibodies with a high sensitivity (79–90 %) and specificity (96 %) for dermatitis herpetiformis [3]. It can also signal the presence of villous atrophy of the patient's small intestine without resorting to intestinal biopsy studies [4].

Histopathology

Histopathologic investigation reveals reticulated subepidermal papillary abscesses with neutrophils.

Differential Diagnosis

The differential diagnosis includes scabies, generalized pruritus of any cause, allergic contact dermatitis, insect bites, papular atopic dermatitis, and neurodermatitis. With symptoms of pruritus, the patient may have previously been misdiagnosed and responded poorly to topical glucocorticoids [5, 6].

Therapy

1. Gluten-free diet
2. Dapsone, 25–100 mg daily (must check methemoglobin level. G6PD [glucose-6-phosphate dehydrogenase] enzyme level. and hematocrit)

Prognosis

The prognosis is fair, especially if a gluten-free diet is maintained. Occasionally, intestinal lymphoma develops.

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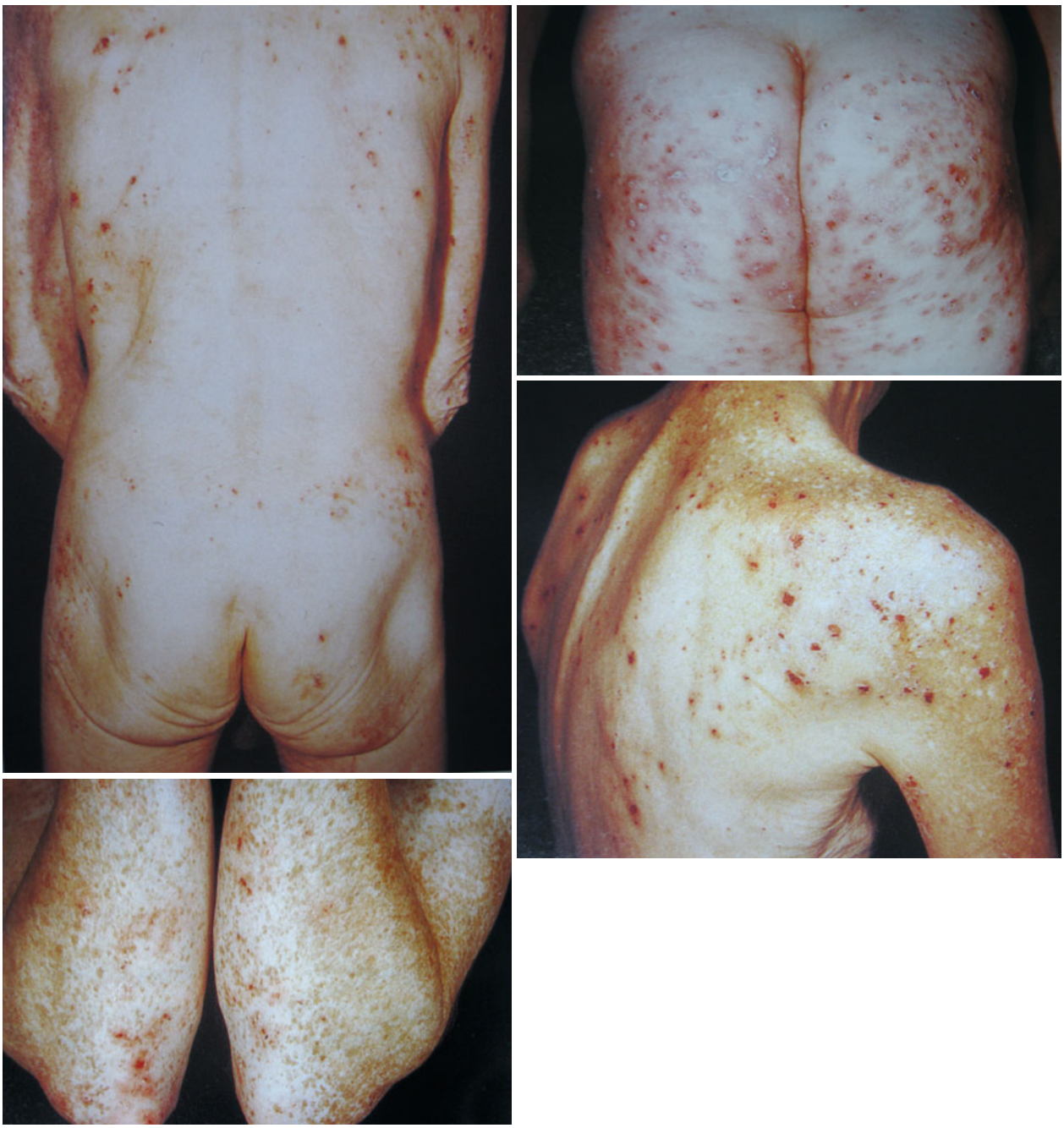
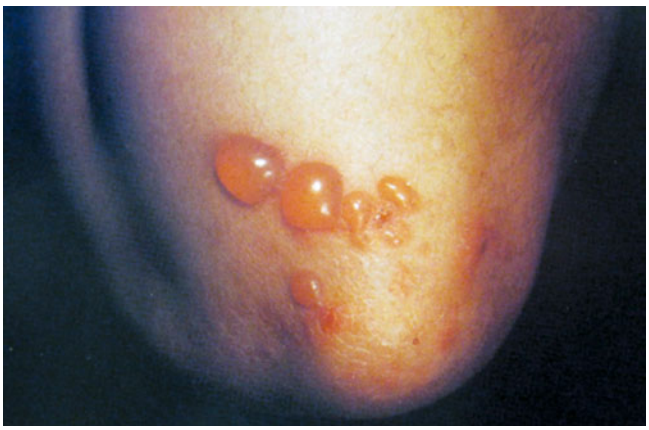
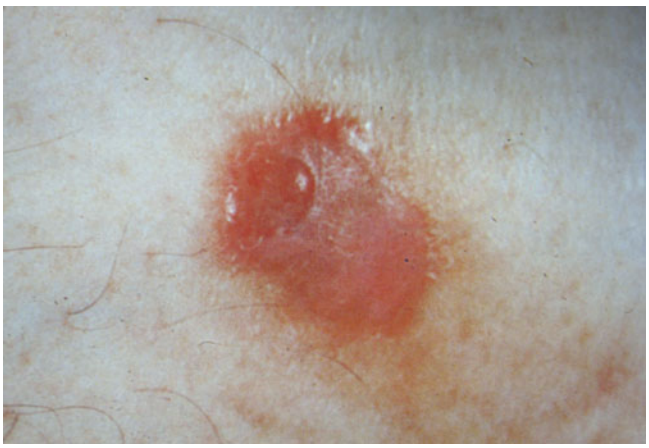
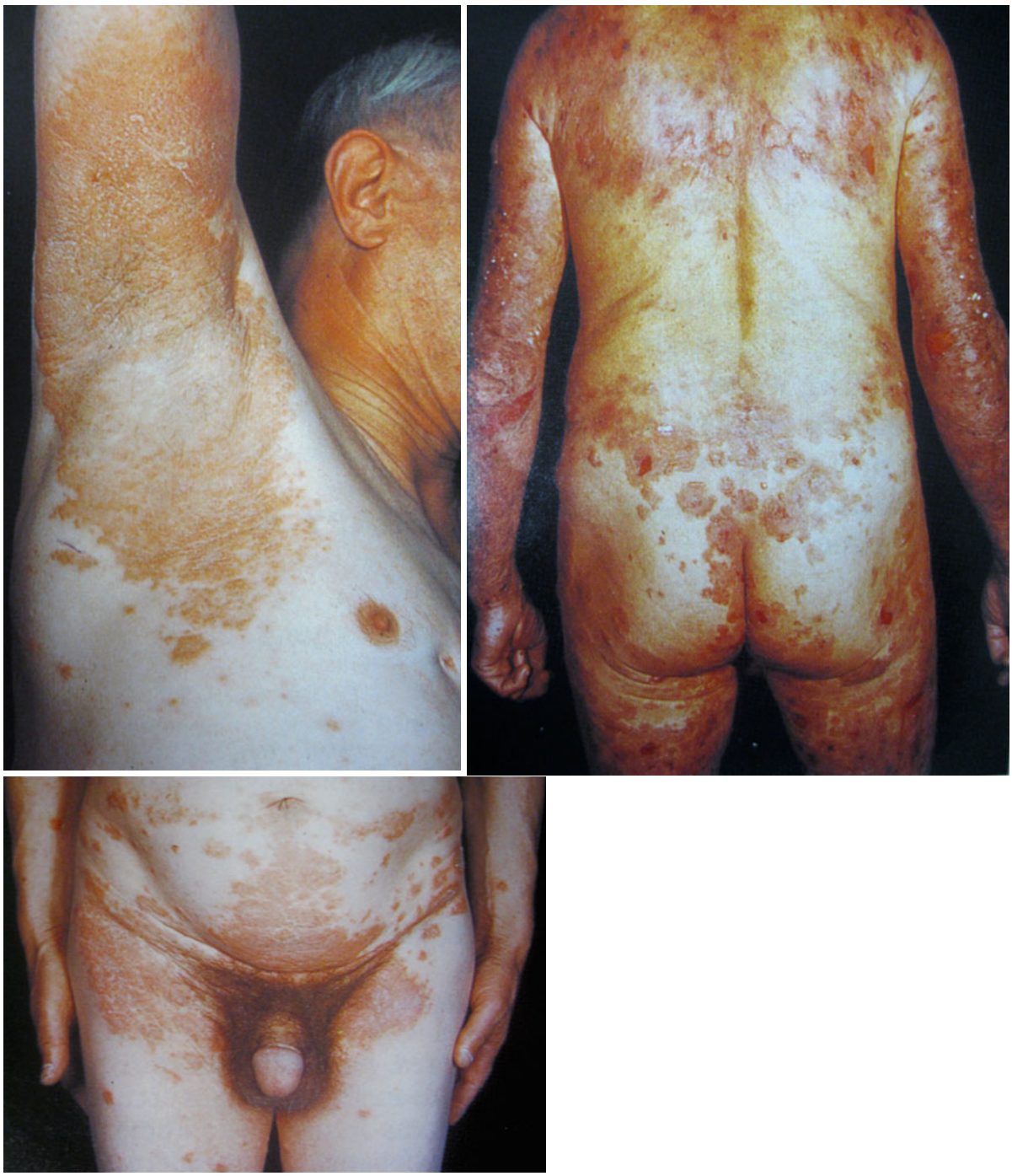


Figs. 5.1, 5.2, 5.3, and 5.4 Typical presentation of dermatitis herpetiformis shows widespread nonspecific excoriated papulovesicles, crusts, and erosions over the trunk (Fig. 5.1) especially the buttocks (Fig. 5.2), sacrum, scapulae (Fig. 5.3), elbows (Fig. 5.4), and knees





Figs. 5.5 and 5.6 Primary lesions consisting of intact vesicles, bullae, and papulovesicles are rarely found because of intense pruritus and scratching



Figs. 5.7, 5.8, and 5.9 Dermatitis herpetiformis simulating bullous pemphigoid with urticarial, sometimes hemorrhagic, plaques of the axillae (Fig. 5.7) and groin (Fig. 5.8). Note relative sparing of the central back (Fig. 5.9), a difficult area to scratch

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Erythema Multiforme

6

Classification

Erythema multiforme (EM) is subclassified into EM minor (Hebra's disease) and EM major [1–3]. EM major includes Stevens-Johnson syndrome and toxic epidermal necrolysis (TEN) [4–10]. Stevens-Johnson syndrome involves two or more mucosal surfaces (eyes, mouth. genitals). Toxic epidermal necrolysis (TEN, Lyell's disease) refers to the most severe, generalized form, an exfoliative erythroderma [6, 10].

Clinical Description

EM minor is characterized by multiple target or iris macules or vesicles with a dusky, gunmetal gray center and concentric red, pink, or violaceous rings, particularly on the hands and feet. Erosions occur on mucosal surfaces in Stevens-Johnson syndrome and TEN. The generalized exfoliative erythroderma of TEN resembles that of staphylococcal scalded skin syndrome. Polymorphic erythematous, urticarial, targetoid, vesicular, or erosive lesions may occur, but lesions tend to be monomorphic in anyone patient.

Etiology and Pathophysiology

Erythema multiforme is a cytotoxic reaction and/or vasculopathy with many causes, most commonly the following:

1. Drugs-Sulfa drugs (TEN) and penicillin are generally the causative agents, although almost any medication can produce EM, even topical agents [4].
2. Infections-herpes simplex (currently the most common cause of EM minor) and Mycoplasma pneumoniae (common cause of Stevens-Johnson syndrome).

Histopathology

Histopathologic investigation reveals epidermal necrosis, lichenoid lymphocytic infiltrate, usually without eosinophils (eosinophils may be seen in drug-induced cases), and subepidermal or dermal edema, which may progress to vesiculation.

Differential Diagnosis

The differential diagnosis includes urticaria, bullous pemphigoid, toxic shock syndrome, and staphylococcal scalded skin syndrome (Ritter's disease), which usually occurs in children following infections.

Therapy

The aim of therapy is to seek, treat, and eliminate the underlying cause:

1. A titrated suppressive dose of acyclovir 200 mg orally, two to four times daily, may prevent frequent, chronic, debilitating herpes simplex induced EM; 200 mg orally, five times daily for 5 days may shorten the duration of an acute attack, if given early.
2. Erythromycin is prescribed for infection by Mycoplasma pneumoniae.
3. Systemic steroids have been used to treat all forms of EM, but they must be given as soon as possible to be of any benefit. Their use is still controversial.
4. Meticulous burn care therapy can be life-saving in TEN.

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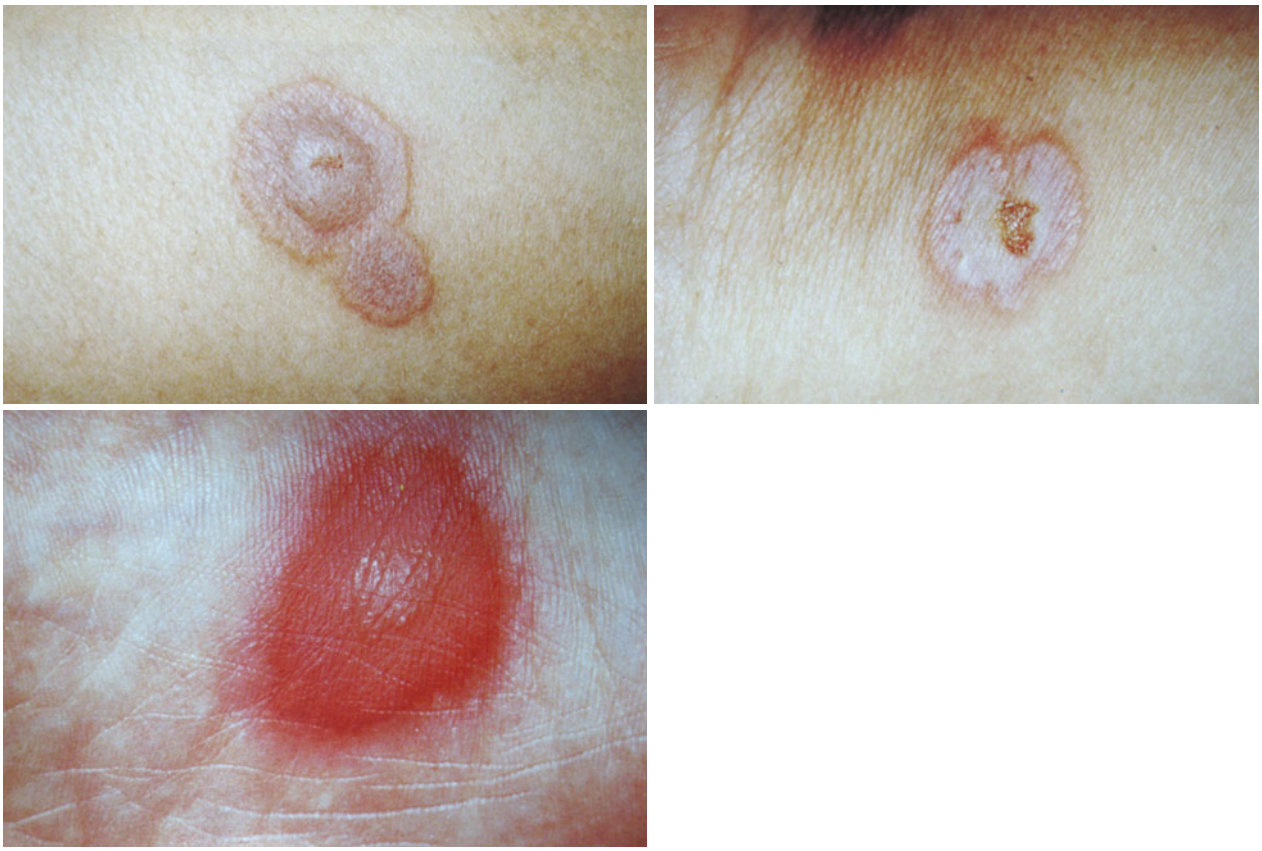
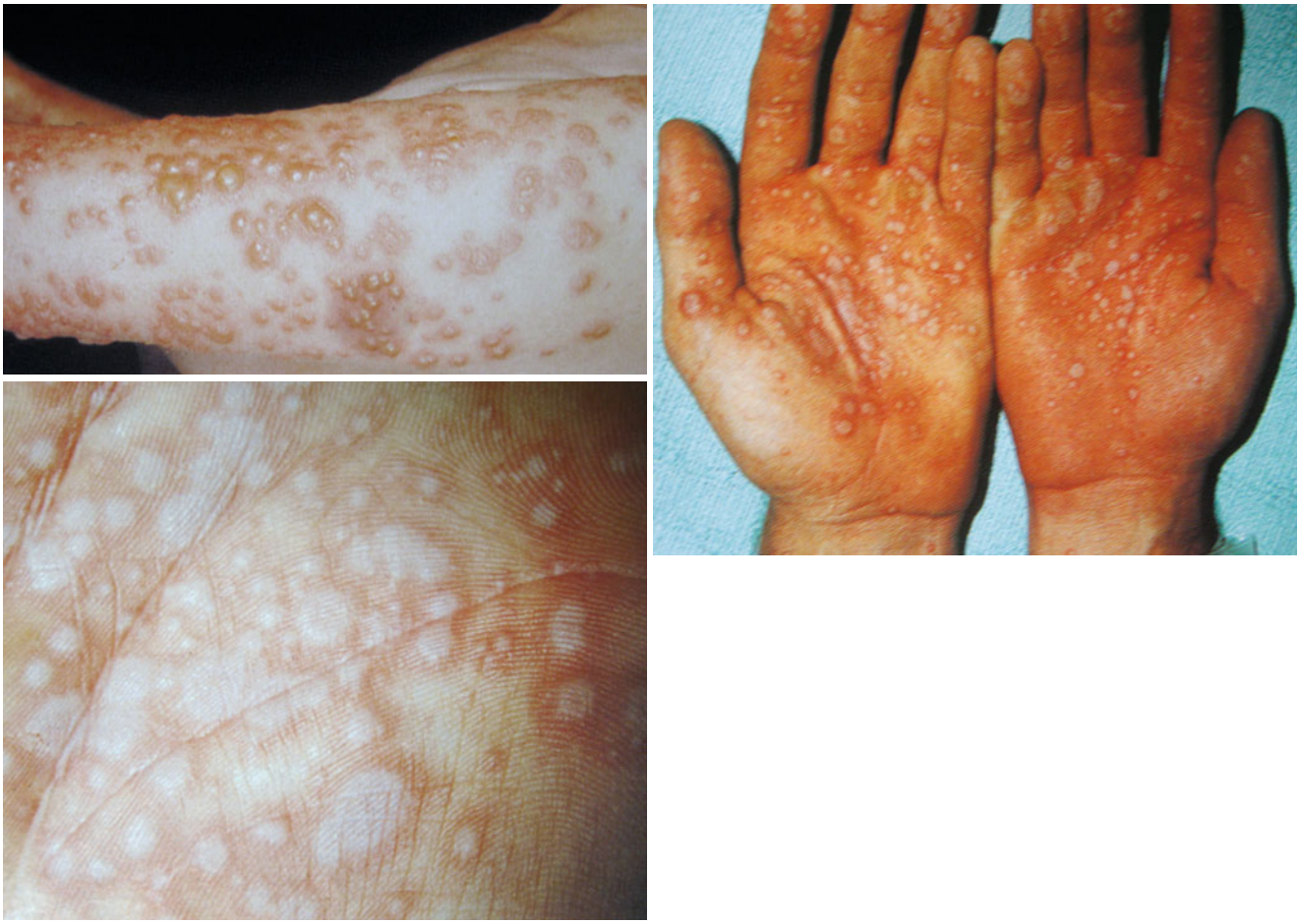
Prognosis

1. EM minor has an excellent prognosis for complete recovery, but recurrences are common.
2. Stevens-Johnson syndrome can lead to mucosal scarring (symblepharon and blindness, esophageal and urethral strictures) [11, 12].
3. TEN is potentially fatal even with treatment.



Figs. 6.1, 6.2, and 6.3 Target or iris ("herpes Bateman") lesions of erythema multiforme minor of von Hebra showing peripheral urticarial edema and central, gray-blue, necrotic epidermis



Figs. 6.4, 6.5, and 6.6 Vesiculobullous erythema multiforme of the arms and palms from herpes simplex virus

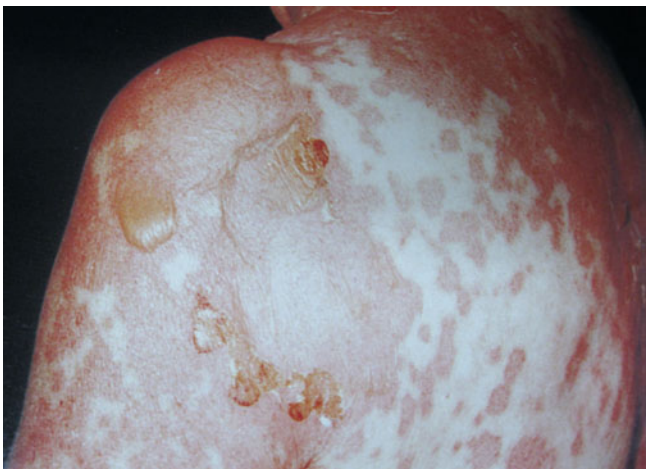


Fig. 6.7 Toxic epidermal necrolysis (Lyell's syndrome) is usually caused by drug allergy, especially to sulfonamides. Diffuse erythema rapidly progresses to flaccid, superficial bullae and erosions

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Grover's Disease

7

Synonyms

Grover's disease is also known as transient (but often stubbornly persistent) acantholytic dermatosis. "Acantholysis" is a histologic term meaning the separation of epidermal keratinocytes (spinous cells).

Clinical Description

This transient or often persistent eruption, lasting weeks to months, consists of scattered, discrete, 1- to 3-mm red, keratotic, pruritic papules and papulovesicles [1–3]. These usually occur on the central chest, back, and thighs in middle-aged and elderly men (more than women), especially after febrile illness or sweat-provoking activity [3].

Histopathology

Grover's disease has five histopathologic patterns, all characterized by acantholysis [2]:

1. Pemphigus vulgaris type-suprabasilar acantholysis
2. Hailey-Hailey type-full-thickness, "dilapidated brick wall" acantholysis
3. Darier's Type-acantholytic dyskeratosis with corps ronds and grains
4. Pemphigus foliaceus type-superficial acantholysis
5. Spongiotic type-acantholytic cells within spongiotic foci

Differential Diagnosis

The differential diagnosis includes the following:

1. Darier-White (Darier's) disease (keratosis follicularis). This is an autosomal dominant condition usually presenting at birth or childhood and sometimes late in life. Yellow-tan, crusted, keratotic, erythematous, follicular papules demonstrate suprabasilar acantholysis, lacunae, corps ronds (dyskeratotic keratinocytes), and grains (parakeratotic cells), which can also be seen in Grover's disease. Darier's disease preferentially affects the seborrheic areas. Other mucocutaneous findings include verrucous papules of the dorsal hands (acrokeratosis verruciformis of Hopf), oral mucosal cobblestone-like papules, and nail changes comprised of V -shaped notching, longitudinal ridging, and red and white lines.
2. Miliaria (sweat retention). Miliaria is not keratotic or follicular. Tiny, crystalline papulovesicles (miliaria crystallina), papules (miliaria rubra), or pustules (miliaria profunda) occur, depending on the depth of occlusion of the eccrine sweat glands.
3. Folliculitis. Pseudomonas aeruginosa hot tub folliculitis occurs in a bathing trunk distribution as a pruritic, follicular, pustular eruption that resolves spontaneously once the hot tub has been adequately chlorinated and/or avoided [4].

Therapy

1. Patient should avoid sweating and keep cool and dry.
2. Keratolytics-salicylic acid-sulfur preparations (sulfacetamide and precipitated sulfur) at night after a salicylic acid wash.
3. Mild topical corticosteroids
4. Low-dose etretinate or biologic therapy for the most severe, refractory cases.

Prognosis

Grover's disease may resolve in weeks or persist for months to years [5–7].

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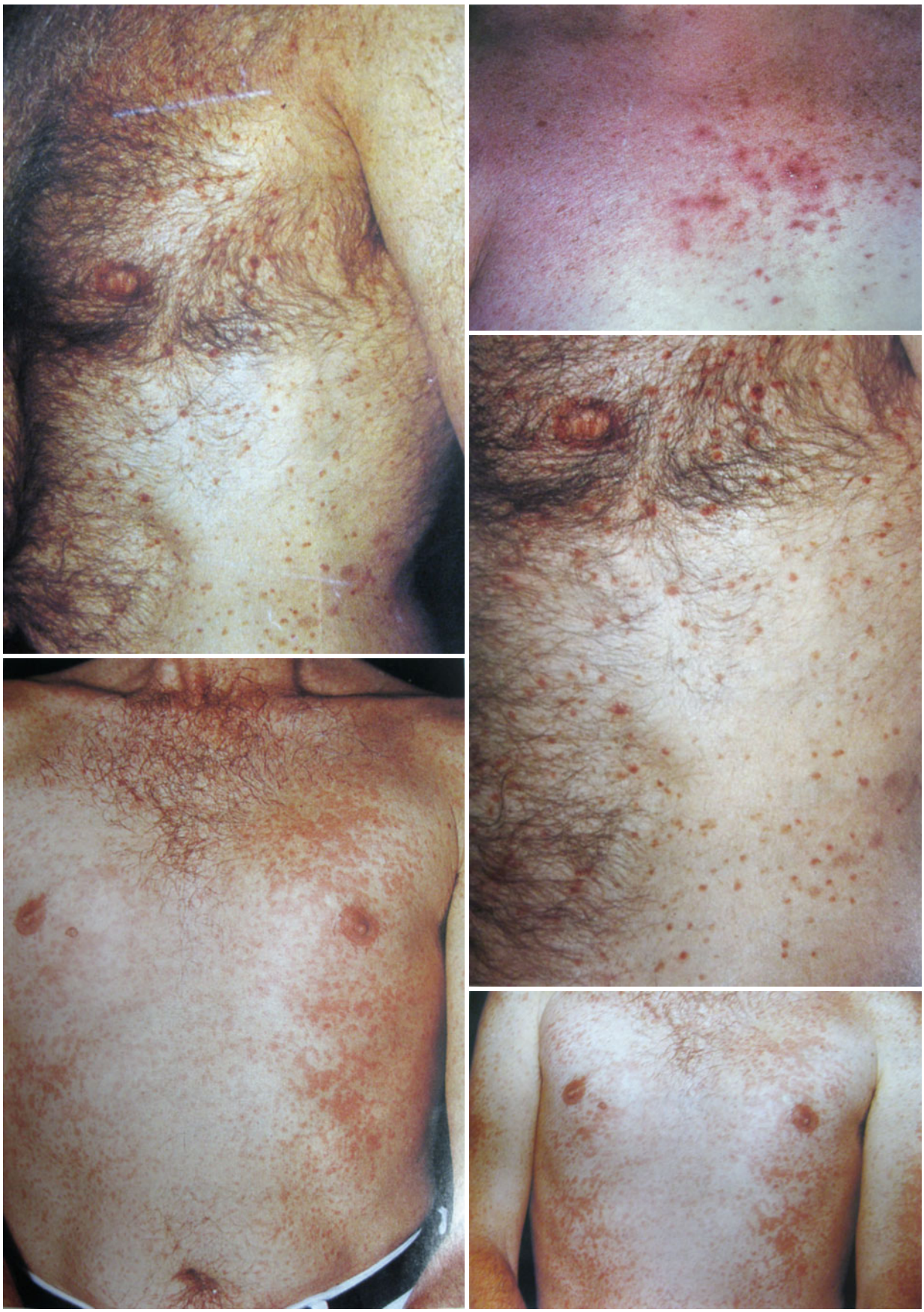
DOI 10.1007/978-1-4471-4579-0_7, © Springer-Verlag London 2014



Figs. 7.1, 7.2, 7.3, 7.4, and 7.5 Characteristic cfinical appearance of keratotic fo/licul-ar and nonfollicufar erythematous papulopustules on the central trunk

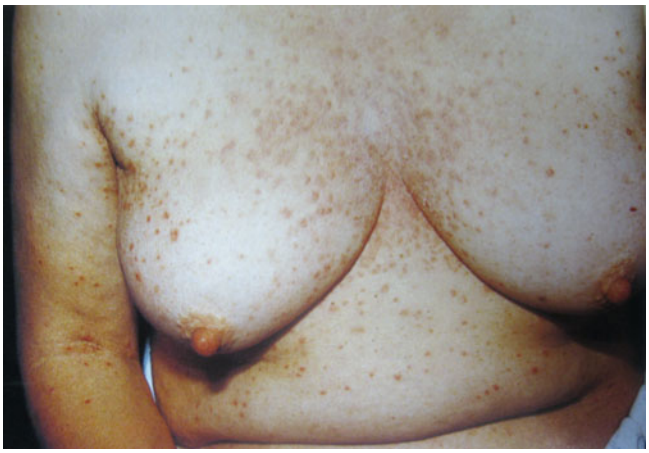


Fig. 7.6 Darier-White disease (keratosis follicularis) is an autosomal dominant condition presenting earlier in life (see text for details)

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Pemphigus

8

Clinical Types

1. *Pemphigus vulgaris*. Mucous membranes typically are affected first in pemphigus vulgaris. Patients have ill-defined, irregularly shaped, gingival, buccal, or palatine erosionslesions that may precede cutaneous lesions by weeks or months. Cutaneous lesions include flaccid blisters filled with clear fluid that arises on healthy skin or on an erythematous base.
2. *Pemphigus foliaceus*. The primary lesions are small, superficial blisters that are difficult to find because they are transient and transform into erosions. Typical pemphigus foliaceus has scaly, crusted erosions on an erythematosus base resembling those of sebhorrehic dermatitis.
3. *Pemphigus Herpetiformis*. Patients usually present with erythematous, vesicular, bullous, or papular lesions. Mucous membrane involvement is uncommon.
4. *Pemphigus IgA*. IgA pemphigus is characterized by tissue-bound and circulating IgA autoantibodies that target the desmosomal proteins of the epidermis. Lesions form within erythematous plaques or in skin without plaques. The initial, clear, fluid-filled blisters associated with IgA pemphigus fill with neutrophils and transform into pustules.
5. *Pemphigus Erythematosus*. Pemphigus erythematosus, also known as Senear-Usher syndrome, is an overlap syndrome with features of lupus erythematosus (LE) and pemphigus foliaceus. Lesions typically involve the scalp, the face, the upper part of the chest, and the back. Patients with classic pemphigus erythematosus present with small, flaccid bullae with scaling and crusting.
6. *Pemphigus vegetans*. Lesions in skin folds readily form vegetating granulations. In some patients, erosions tend to develop excessive granulation tissue and crusting, and these patients display more vegetating lesions.

Etiology and Pathophysiology

Pemphigus refers to a group of autoimmune or drug-induced blistering diseases of the skin and mucous membranes. These antibodies bind to keratinocyte desmosomes and to desmosome-free areas of the keratinocyte cell membrane which results in a loss of cell-to-cell adhesion, acantholysis. The antibody alone is capable of causing blistering without complement or inflammatory cells. Pemphigus is also reported to be linked to genetic factors. The elderly are more commonly affected. Occurrence is more common in patients with other autoimmune diseases [1–5].

Diagnosis

Direct Immunoflourescence (DIF) of preilesional skin reveals a "chicken wire" pattern of IgG deposition on intercellular cement substance. In the patient's serum, IDIF demonstrates the presence of circulating IgG autoantibodies that bind to epidermis. Circulating intercellular antibodies are detected using IDIF in 80–90 % of patients with pemphigus vulgaris.

Histopathology

Pemphigus is characterized histologically by intraepidermal blister and immunopathologically by the finding of in vivo bound and circulating immunoglobulin G (IgG) antibody directed against the cell surface of keratinocytes. Epidermal basal layer acantholysis is present in pemphigus vulgaris and vegetans. Subcorneal acantholysis is present in pemphigus foliaceus and erythematosus.

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Differential Diagnosis

Pemphigus can resemble several different imflammatory bullous diseases including bullous pemphigoid, dermatitis herpetiformis, erythema multiforme. Pemphigus vulgaris oral lesions may represent herpetic stomatitis or desquamative gingivitis. Pemphigus vegetans can resemble Hailey-Hailey disease, but differentiated by IF and histologic tests.

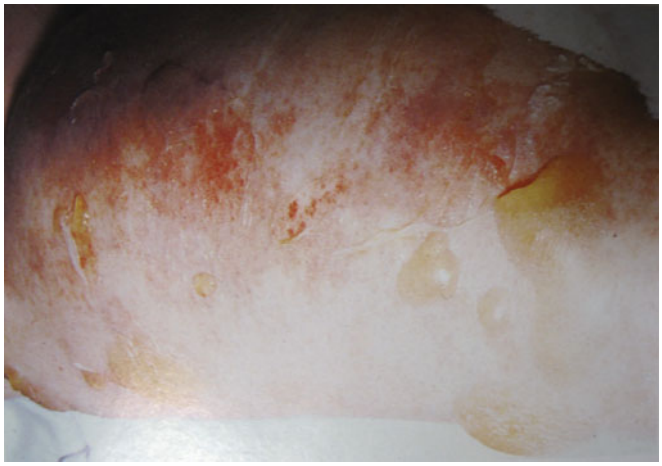
Therapy

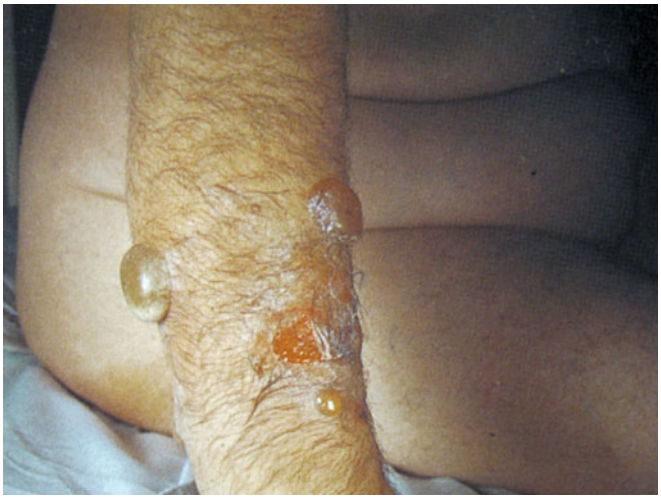
The aim of treatment is to decrease blister formation, promote healing of blisters and erosions, and determine the minimal dose of medication necessary to control the disease process.

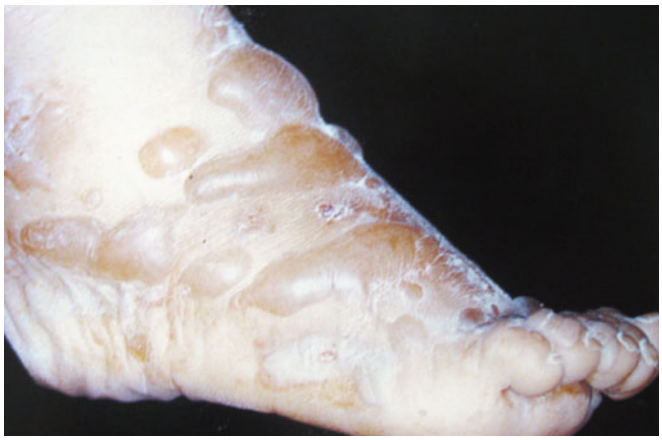
1. Corticosteroids have improved overall mortality. Prednisone, an anti-inflammatory agent, stabilizes lysosomal membranes and suppresses lymphocytes and antibody production.
2. Dapsone, Cyclophosphamide, Sulfasalazine, and Pentoxifylline have been reported as effective adjunctive treatments.
3. Mycophenolate, mofetil and azathioprine have been used as steroid-sparing agents
4. High-dose intravenous immunoglobulin has been reported to have a corticoid-sparing effect, but is quite expensive.
5. Many authorities now use rituximab as first- or second-line therapy. Rituximab (monoclonal antibody presumably targets B cells, the precursor of (auto) antibody-producing plasma cells. Given as intravenous therapy, it shows dramatic effect in some and at least partial remission in other patients.
6. Plasmapharesis reduces the level of circulating pemphigus antibodies.
7. Wet dressings, topical and intralesional glucocorticoid, antimicrobial therapy if documented bacterial infections, and correction of fluid and electrolyte imbalance. Topical care includes burn unit admission, whirlpool baths, topical antibiotics, and nonadherent dressings [6–8].

Prognosis

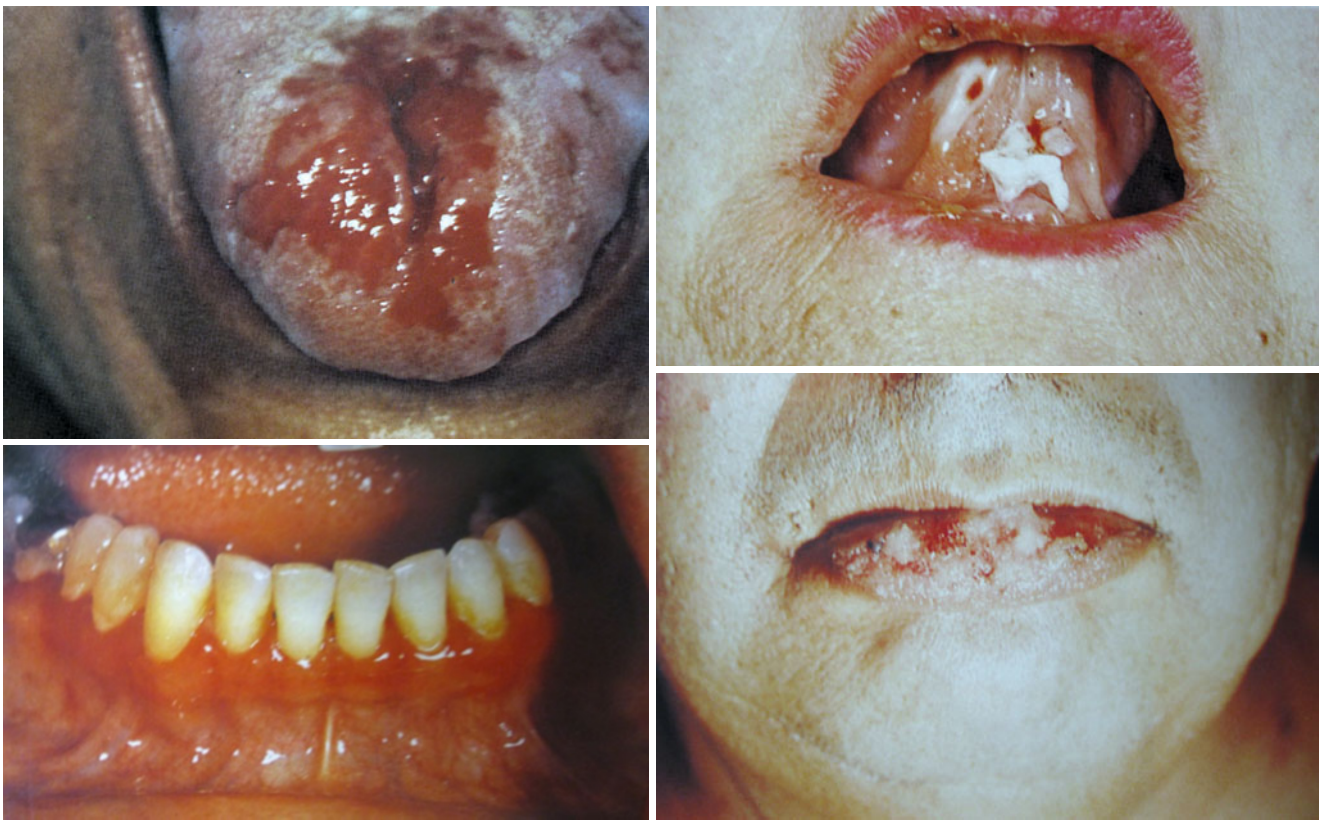
Therapy must be tailored for each patient, taking into account preexisting and coexisting conditions. The use of steroids has improved pemphigus related fatalities greatly, however untreated pemphigus is often fatal due to infections, fluid, and electrolyte disturbances. The outlook is worse in older patients and in patients with extensive disease.



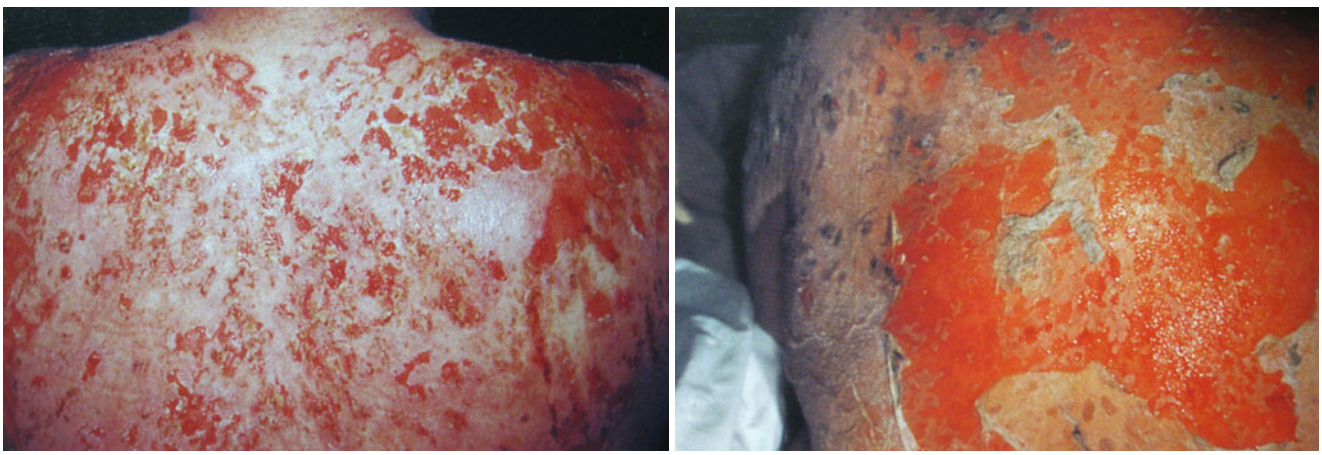




Figs. 8.1, 8.2, and 8.3 Flaccid bullae and erosions with a positive Nikolsky sign (production of blister by applying shearing force to clinically normal skin are the clinical diagnostic hallmarks of pemphigus vutqans



Figs. 8.4, 8.5, 8.6, and 8.7 Oral lesions, particularly ulcerations and erosions of the buccal mucosa, tongue, gingiva, and lips, are often the first sign of pemphigus, and occur in about 70 % of patients



Figs. 8.8 and 8.9 Extensive erosions must be treated as a severe burn with topical care and control of infection and of fluid and electrolyte balance

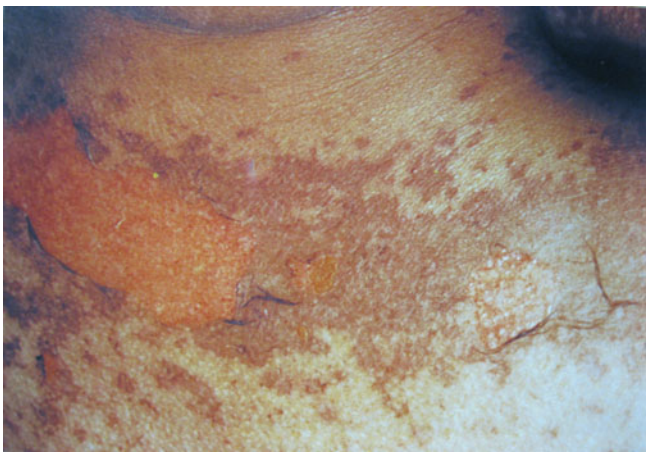
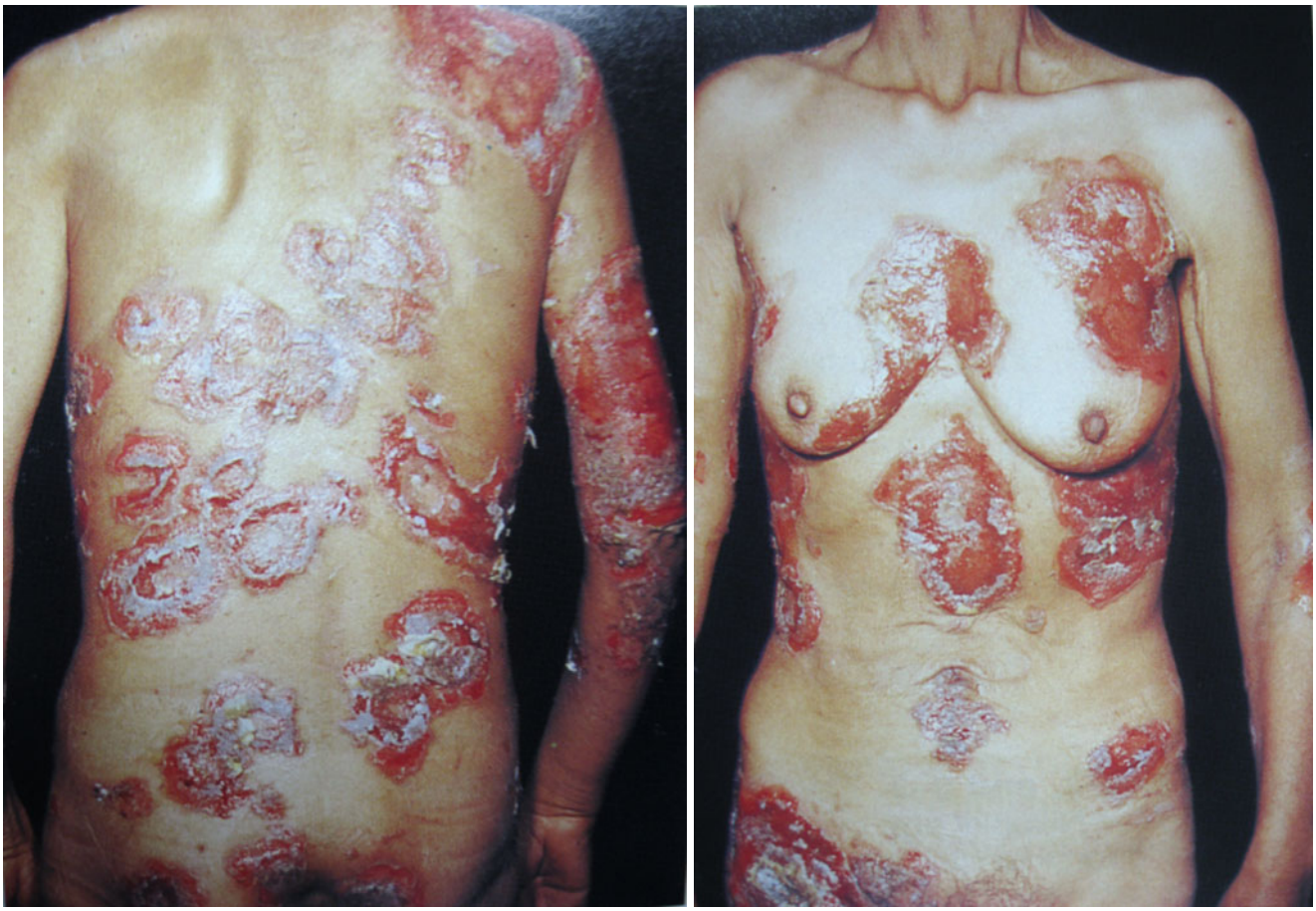


Fig. 8.10 Drug-induced pemphigus vulgaris and pemphigus foliaceus can occur with captopril, penicillamine, rifampin, penicillin, ampicillin, cephalexin, gold, propranolol, meprobamate, piroxicam, and phenobarbital



Figs. 8.11 and 8.12 Beefy, red erosions in a patient with severe pemphigus vulgaris

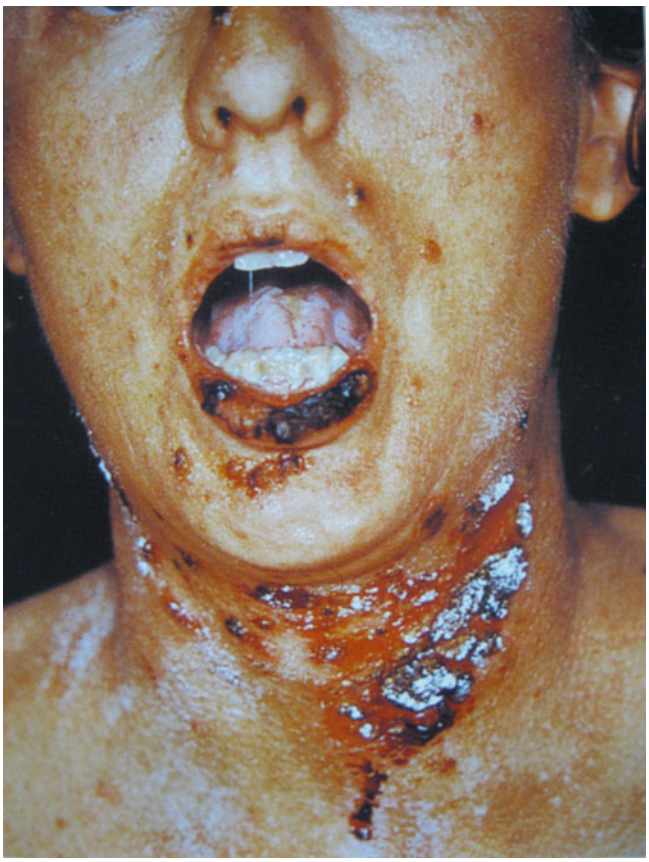
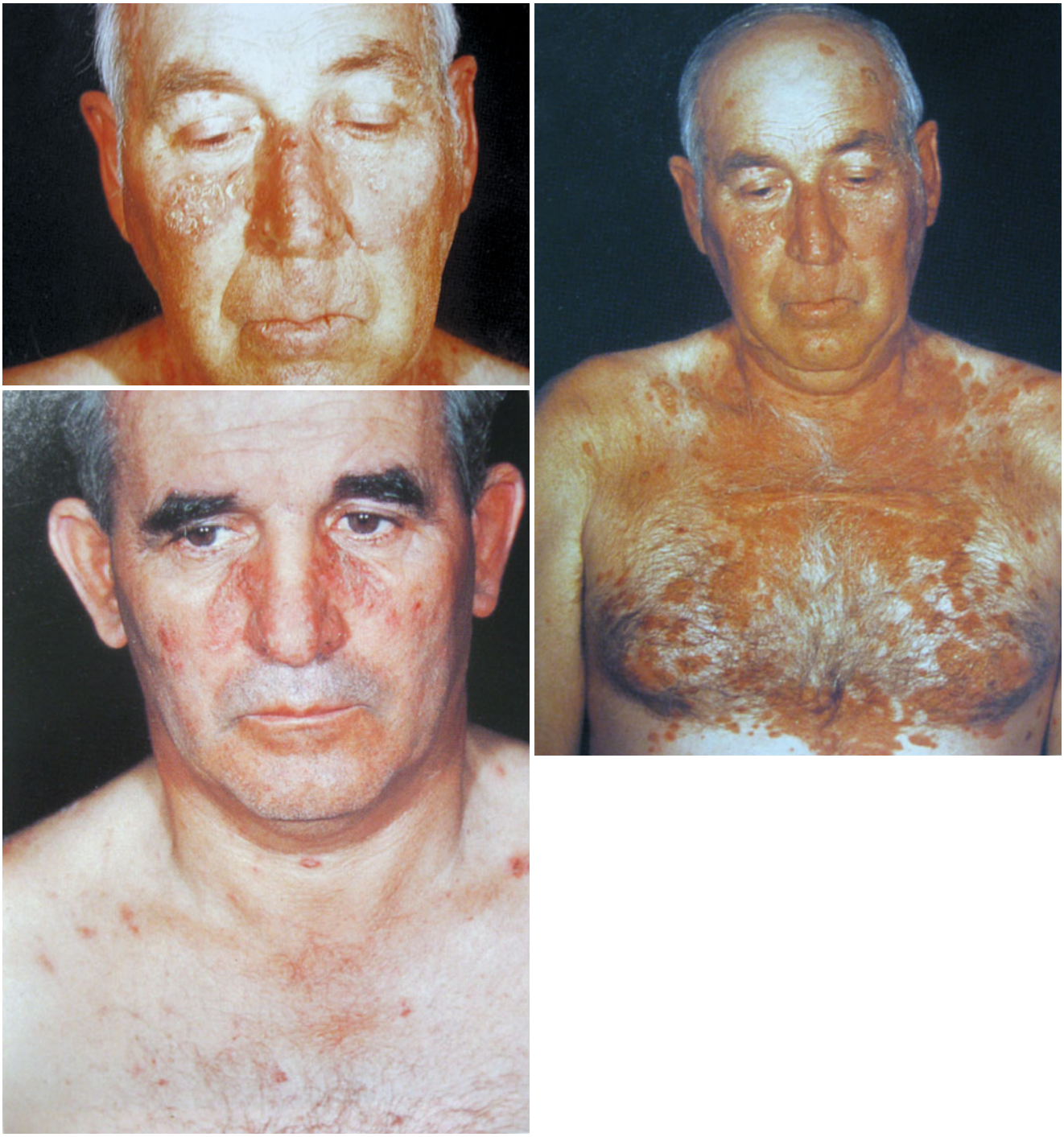
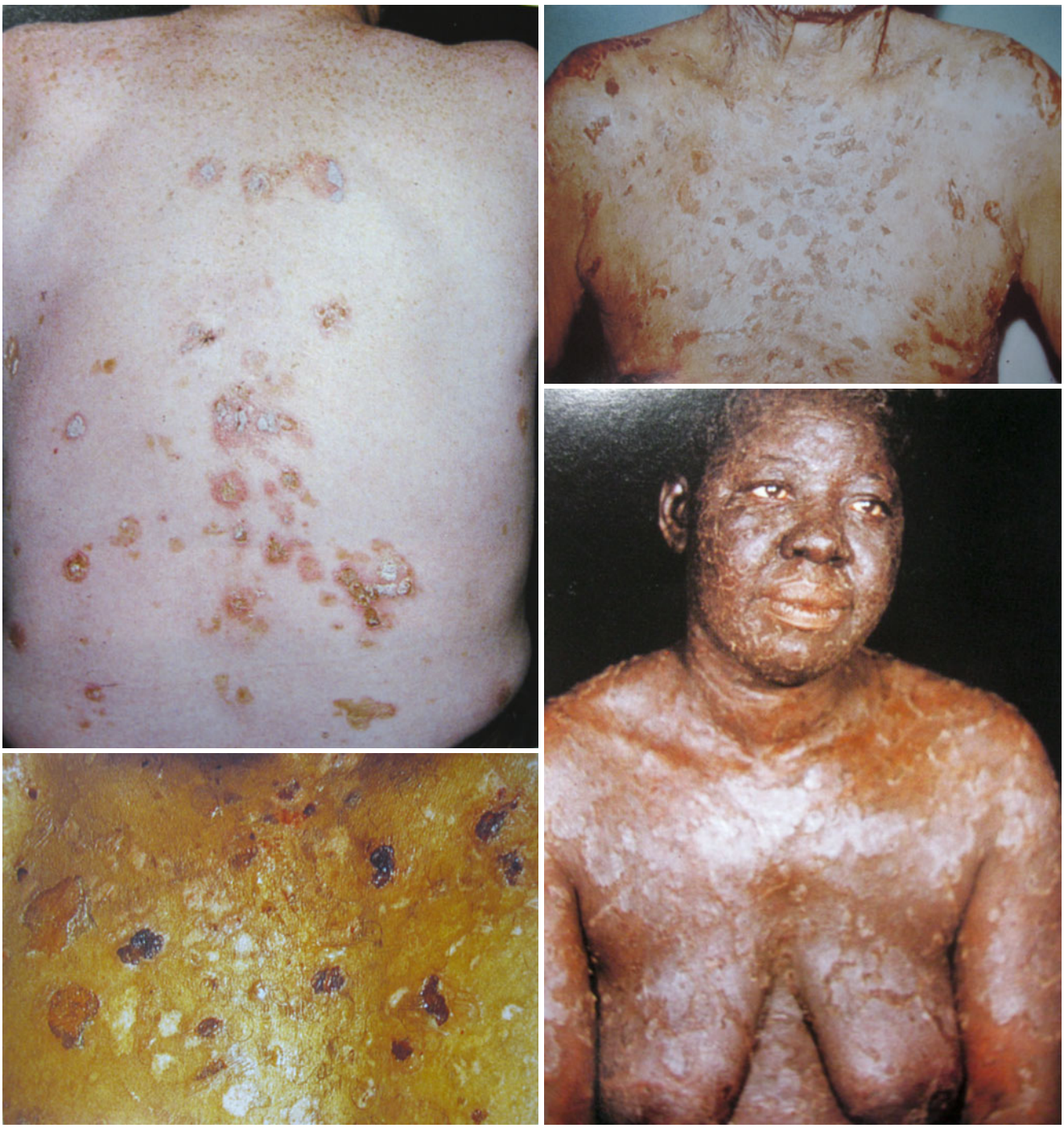


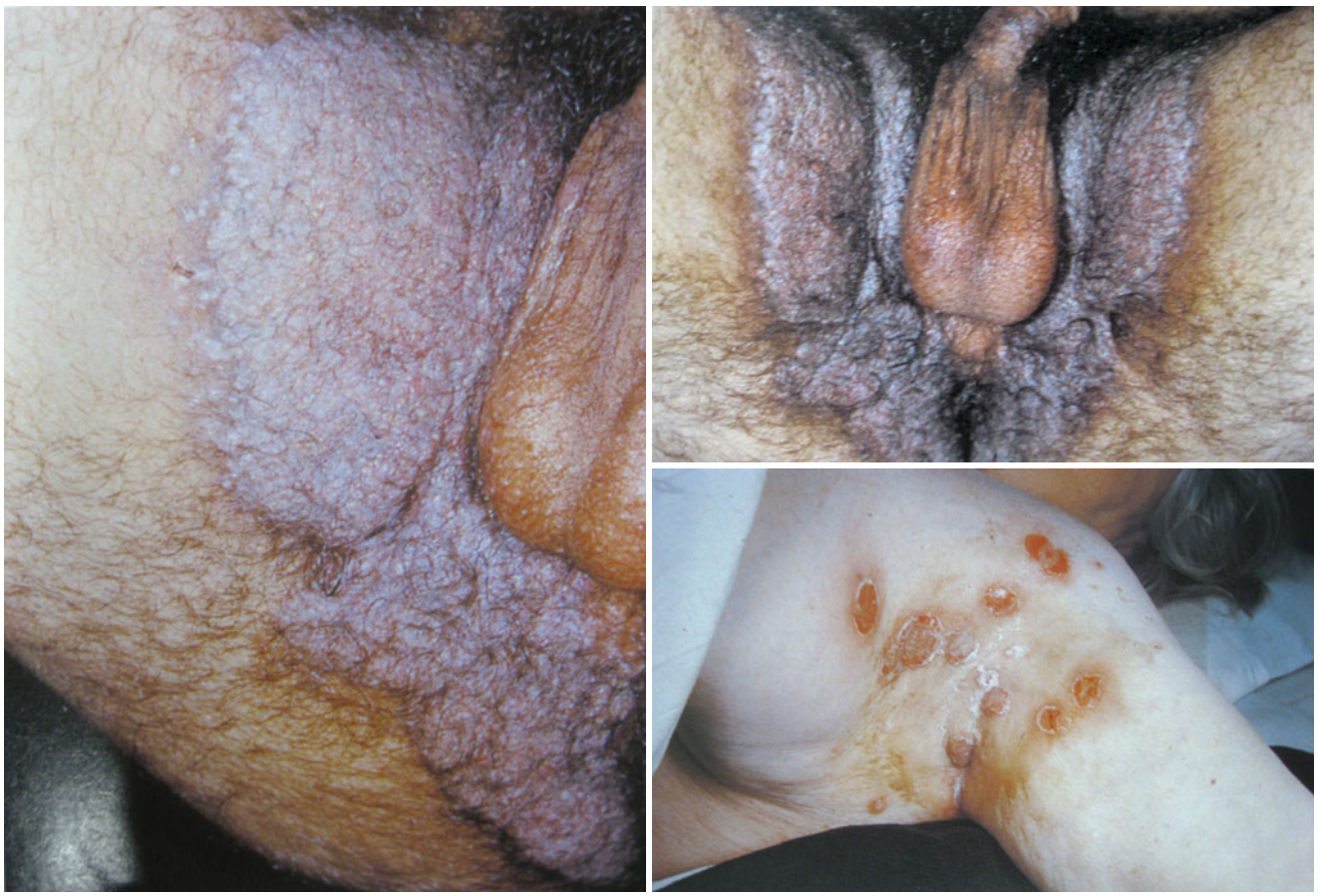
Fig. 8.13 Facial, oral, and neck lesions *in* a patient with pemphigus VUlgaris



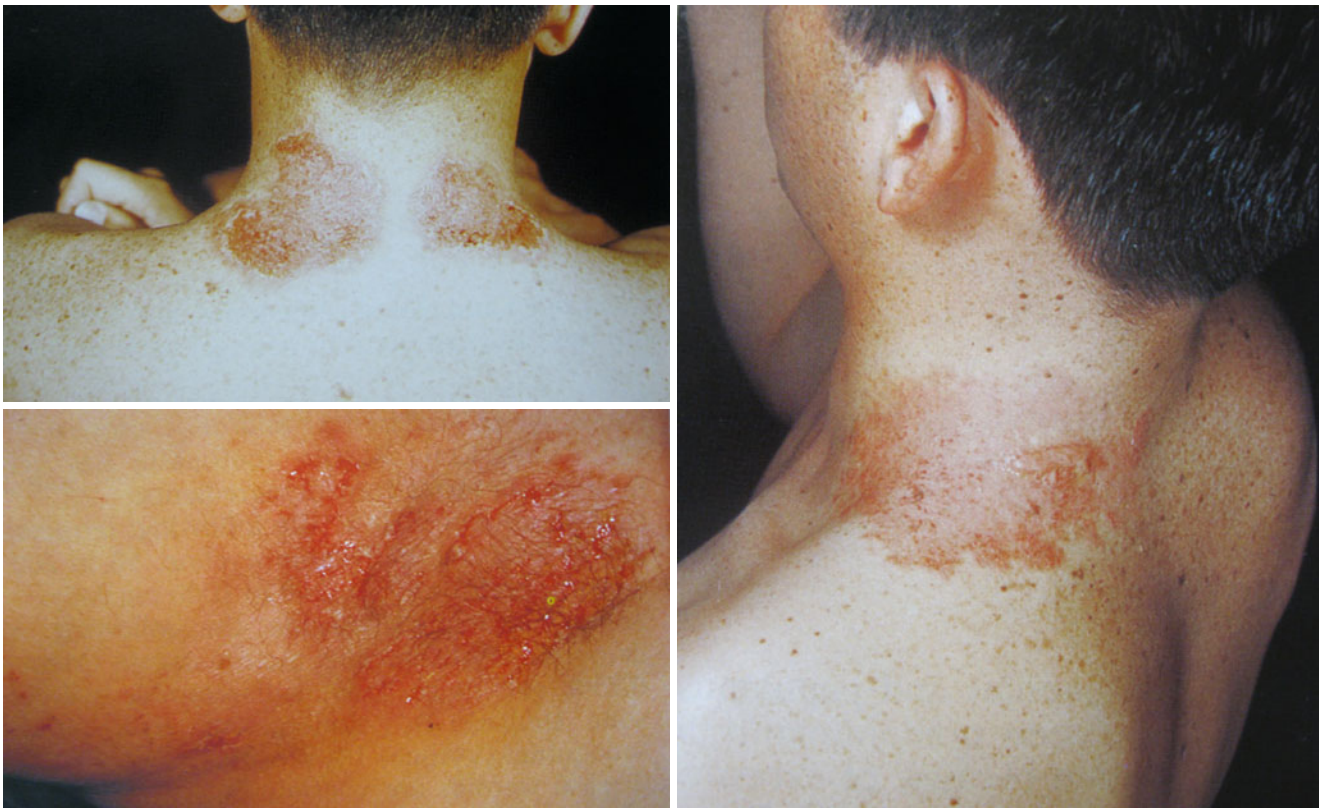
Figs. 8.14, 8.15, and 8.16 Pemphigus erythematosus (Senear-Usher syndrome) combines features of pemphigus vulgaris and lupus erythematosus: positive antinuclear antibody, facial butterfly rash, and positive direct immunofluorescence of IgG, M, and/or C3-a so-called lupus erythematosus band-along the dermal-epidermal junction, in addition to the intercellular staining



Figs. 8.17, 8.18, 8.19, and 8.20 Pemphigus foliaceus demonstrates superficial desquamation and erosions instead of fluid-filled blisters. It clinically resembles seborrheic dermatitis or, in generalized cases, exfoliative dermatitis



Figs. 8.21, 8.22, and 8.23 Pemphigus *vegetans* is manifested by verrucous, *vegetating*, moist eroded plaques in intertriginous areas, clinically resembling those of Hailey-Hailey disease (see Figs. 8.26, 8.27, and 8.28)



Figs. 8.24, 8.25, and 8.26 Hailey-Hailey disease (familial benign pemphigus) is an autosomal dominant condition with intertriginous vesicular plaques and erosions that rapidly become superinfected. Histologically, it shares features with both pemphigus vulgaris and Darier's disease (*see* Fig. 7.8). Direct and indirect immunofluorescence studies are *negative*, in contrast to pemphigus

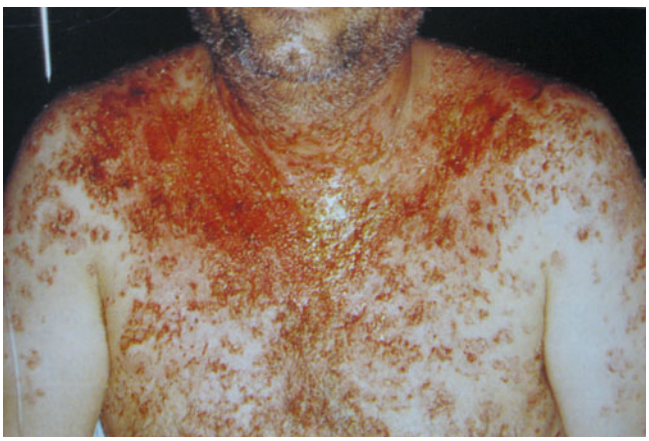
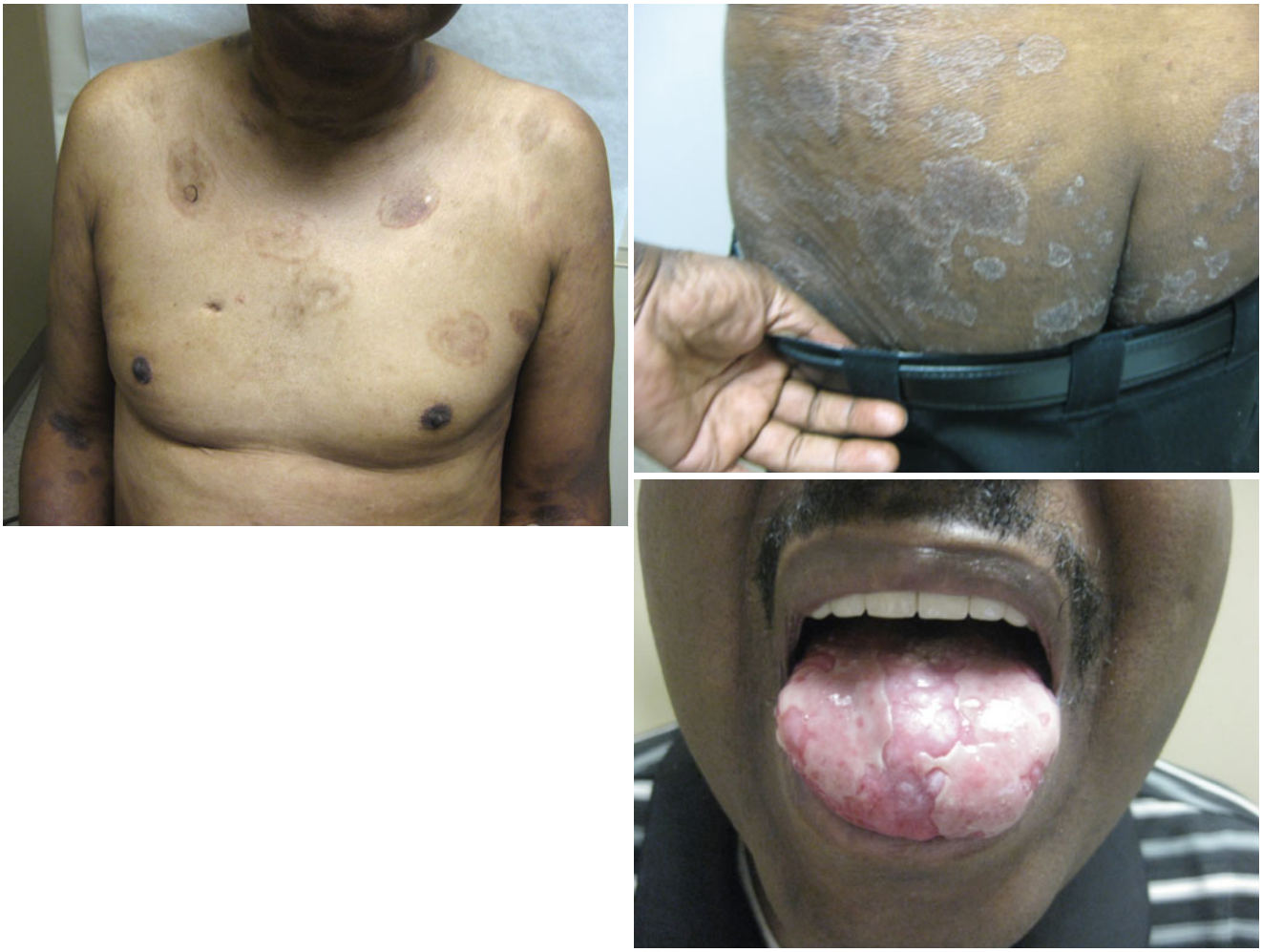


Fig. 8.27 Severe pemphigus vulgaris resembles a burn



Figs. 8.28, 8.29, 8.30 Pemphigus vulgaris affecting chest, buttocks and tongue

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Perioral Dermatitis

9

Synonyms

Perioral dermatitis is also known as perioral folliculitis or acne cosmetica.

Clinical Description

Perioral and perinasal red papules and papulopustules, sometimes pruritic, are seen predominantly in middle-aged and elderly women because of excessive use of such skin products as moisturizers and cleansing creams [1, 2].

Etiology and Pathophysiology

Perioral dermatitis may be a subset of rosacea.

Excessive use of moisturizers, cleansing creams, topical steroids, fluoride and/or tartar-control toothpaste [3], follicular infestation by Demodex folliculorum mite, and oral candidiasis have been implicated.

Histopathology

Histopathologic investigation reveals parakeratotic scaling, spongiotic dermatitis, and folliculitis with perivascular and perifollicular lymphohistiocytic and plasma cell infiltrate.

Differential Diagnosis

Typical rosacea involves the entire central face and occurs in elderly men and women. Acne vulgaris includes open and closed comedones, also involves the lateral face, and can occur in younger patients [4].

Therapy

1. Exacerbating cosmetics and/or toothpastes are eliminated.
2. Oral antibiotics include the following: tetracycline, 250–1,000 mg daily; minocycline, 50–200 mg daily; or erythromycin, 250–1,000 mg daily.
3. Topical antibiotics include the following: clindamycin; erythromycin; metronidazole 0.75 % gel, twice daily; sulfacetamide-hydrocortisone lotion.
4. Topical steroids are contraindicated except during the initial 2 weeks of therapy.
5. Oral candidiasis is treated, when present.

Prognosis

The prognosis is generally good.

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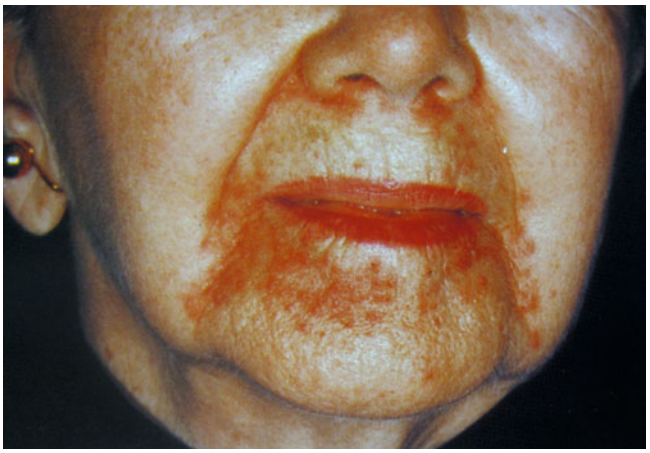
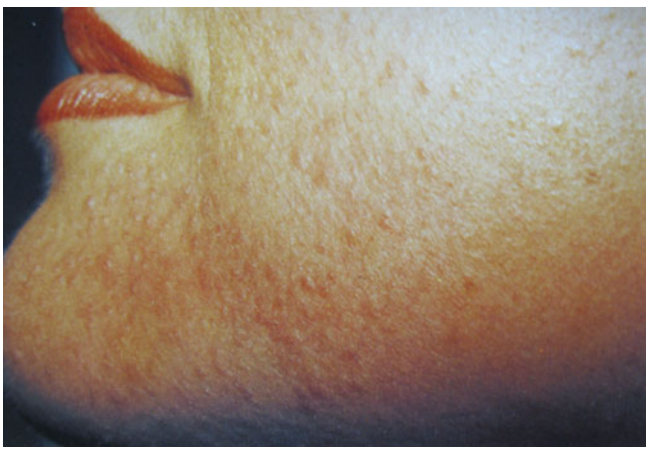
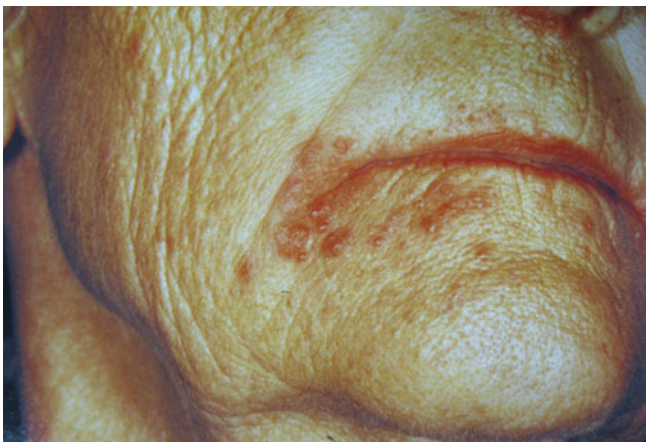
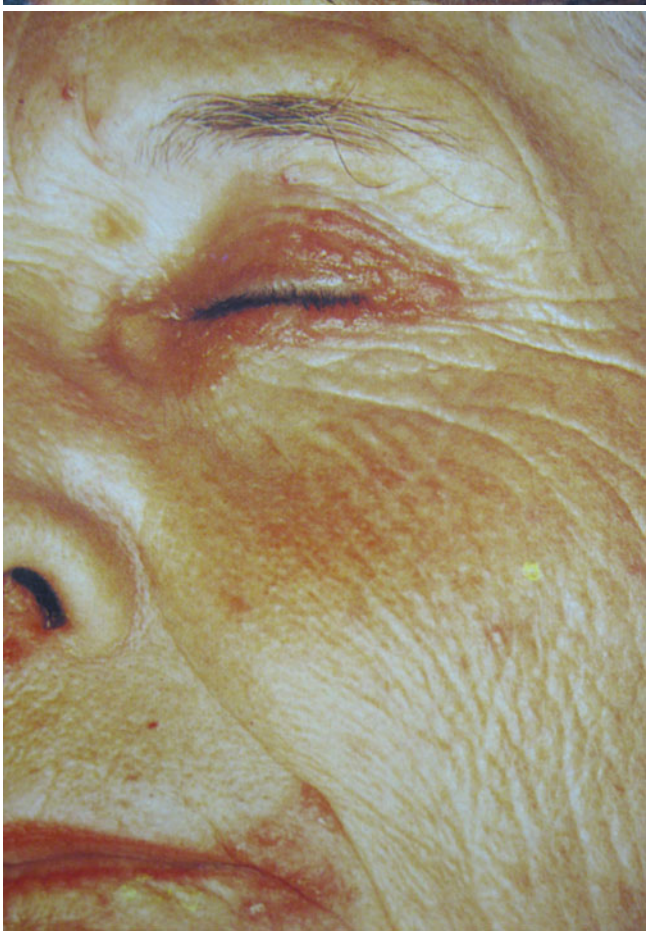




Figs. 9.1 and 9.2 Typical presentation of perioral follicular papulopustules, often caused by fluoride and tartar control toothpaste or by the overuse of cosmetics, especially moisturizers





Figs. 9.3 and 9.4 Perioral and periocular dermatitis from application of baby *oil*

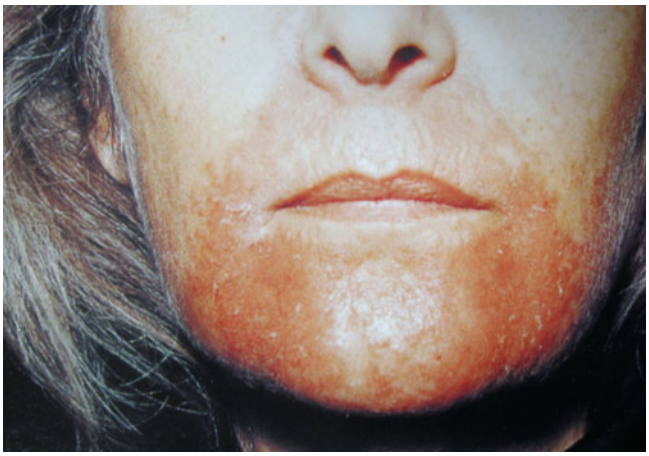


Fig. 9.5 *Perioral* dermatitis from prolonged application of potent fluorinated corticosteroids. This is also known as "steroid rosacea." The steroids initially improve the dermatitis but become progressively *less* effective. As the patient desperately applies more steroid to quell the eruption, the rash worsens

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Porphyria Cutanea Tarda

10

Clinical Description

The most common initial symptoms of porphyria cutanea tarda are cutaneous fragility and blistering of the hands, forearms, and, sometimes, the face. erosions may cause epidermal loss and infections. Healing is slow and painful, causing atrophic scars. Cutaneous findings are characterized by skin photosensitivity with increased skin fragility, facial hypertrichosis, blisters, scarring with milia formation, and skin hyperpigmentation on the hands and other sun-exposed areas. Urine sample will often show a grossly discolored with a tea- or wine-colored tint.

Etiology and Pathophysiology

Porphyria cutanea tarda (PCT) is the most common porphyryia occurring in adults. It encompasses a group of familial and acquired disorders in which activity of the heme synthetic enzyme uroporphyrinogen decarboxylase (UROD) is deficient. Many extrinsic factors play a role in the manifestation of PCT. Decreased production of UROD is created by oxidative stress on the liver. Iron, estrogen, ethanol intake, porphyrons, Hepatitis C, polychlorinated biphenyls, and polychlorinated cyclic hydrocarbons all have an effect on decreasing UROD production. Smoking may also have an effect associated with the earlier onset of symptoms in acquired PCT.

The patients who have the sporadic acquired form of PCT have normal UROD DNA sequences, but are exposed to large polyhalogenated cyclic hydrocarbons. The familial version most often manifests from autosomal dominant inheritance of a single mutation at the UROD locus. A rare recessive familial type of porphyria cutanea tarda in which both *UROD* alleles are mutated is termed hepatoerythropoietic porphyria. Excess iron enhances formation of toxic oxygen species, increasing oxidative stress and apparently facilitating porphyrinogenesis by catalyzing the formation of oxidation products that inhibit UROD [1–3].

Diagnosis

Screening for the gene mutation identified in hereditary hemochromatosis should be performed on all patients. Diagnosis involves the discovery of increased porphyrins in the blood, liver, stool, and urine. Direct Immunoflourescence (DIF) of the perilesional skin reveals variable perivascular deposits of IgG, IgM, and C3. Indirect Immunoflourescence (IIF) of the patient's serum usually yields negative results.

Histopathology

Skin biopsy specimens of blisters show subepidermal bullae with minimal dermal inflammatory infiltrate and dermal papillae protruding upward into the blister cavity (festooning). Thickening upper dermal capillary walls and dermoepidermal basement membrane zones are evident in routinely stained sections and accentuated with the periodic acid-Schiff stain. Elastosis, sclerosis of dermal collagen, and hyaline deposits may be seen in the dermis.

Differential Diagnosis

Other porphyrias, pseudoporphyrias, and photoaggravated bullous dermatoses can manifest with clinical features similar to those of porphyria cutanea tarda. They can also be induced by furosemide, naproxen, nalidixic acid, tetracyclines, dapsone, and pyridoxine [4]. Other diseases to be considered include Epidermolysis bullosa, epidermolysis bullosa acquisita, erythropoietic porphyria, hydroa vacciniforme, lupus erythematosus and bullous. Epidermolysis can be distinguished by electron microscopic localization of the basement membrane layer separation.

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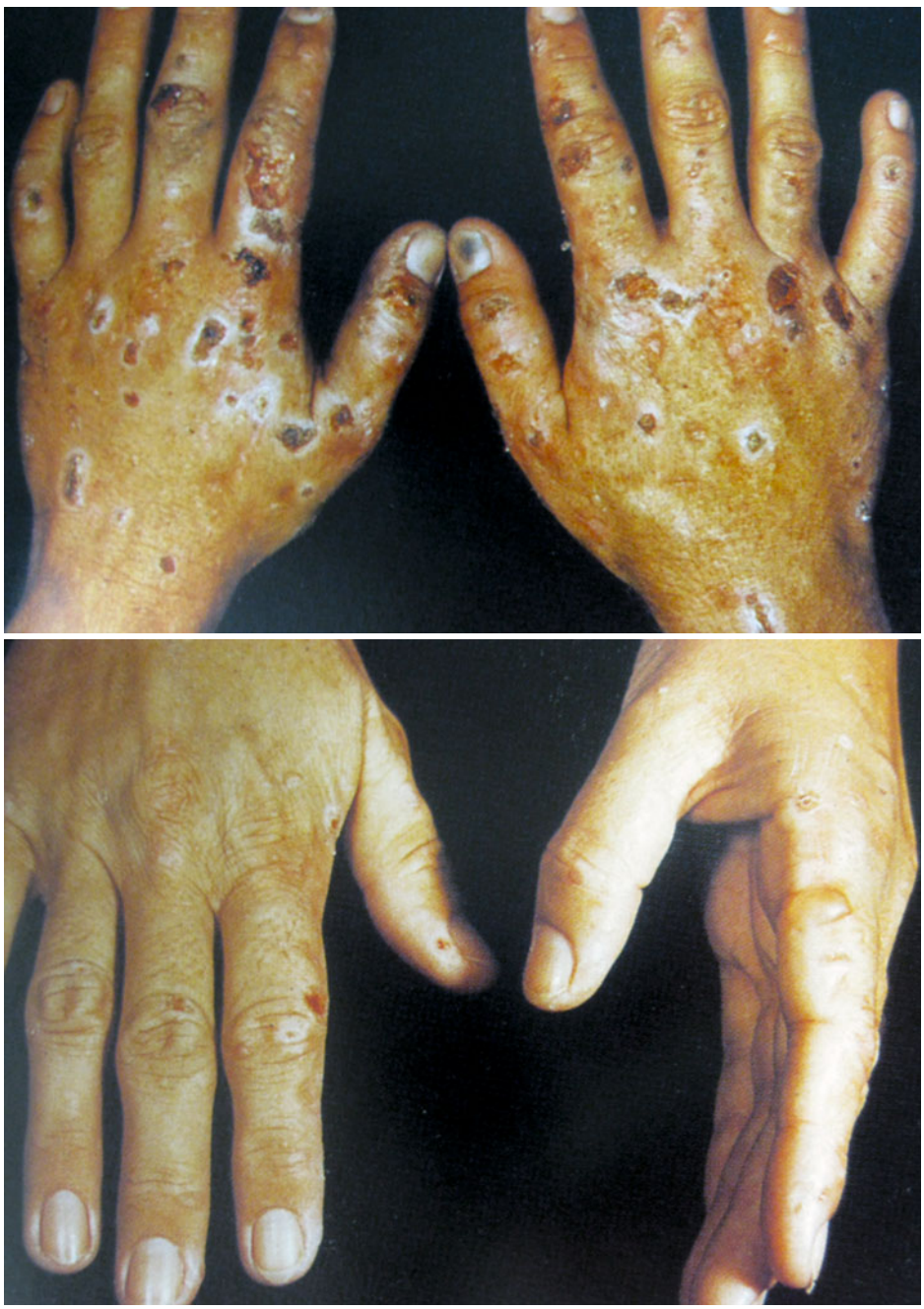
Therapy

1. Removal of extrinsic factor triggers. Iron and estrogen supplementation may reduce symptoms. Alcohol intake should be watched carefully to reduce the creation of increased free radical activity.
2. Patients should avoid sunlight exposure for maximum defense against photosensitivity.
3. Phlebotomy in severe cases may decrease the total iron load to 12–13 g/dl, which can lead to improvement. It may improve sclerodermalike symptoms in skin, but has not been proven to improve liver cell function.
4. Chelation with desferrioxamine is an alternative means of iron mobilization when venesections are not practical
5. Oral chloroquine phosphate (125–250 mg PO twice weekly) or hydroxychloroquine sulfate (100–200 mg PO two to three times per week), doses much lower than those used for antimalarial or photoprotective indications, can be effective. Eye exams are mandatory [5, 6].

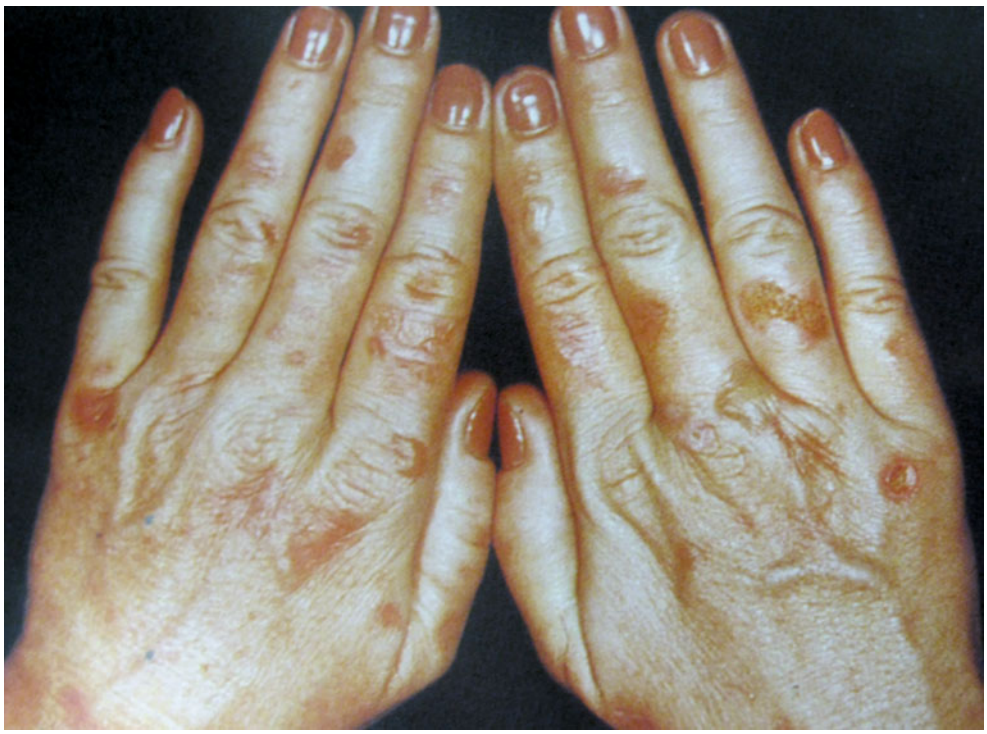
Prognosis

Levels of hemoglobin, serum ferritin, and plasma/serum or urinary porphyrins should be monitored during the course of treatment. Prognosis is fair to poor due to patients' abuse of alcohol restrictions.



Figs. 10.1, 10.2, and 10.3 Typical presentation of porphyria cutanea tarda (pa) showing vesicles, erosions, and crusts on dorsal hands. Patients commonly complain of excessive skin fragility rather than of sun sensitivitry.

Figs. 10.1, 10.2, and 10.3 (continued)



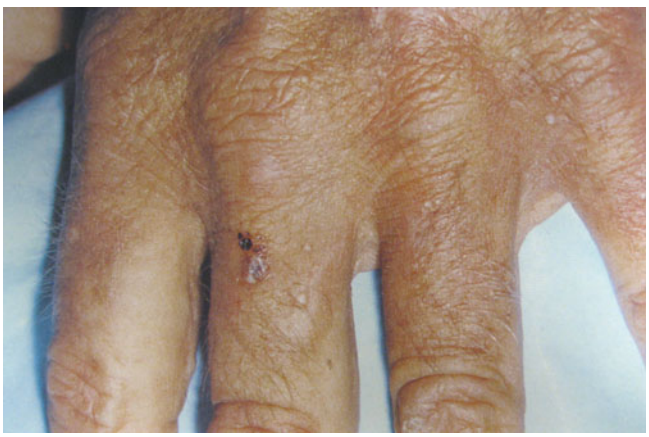


Fig. 10.4 Close examination of fingers reveals milia (tiny epidermal inclusion cysts) from incomplete healing of previous vesicles

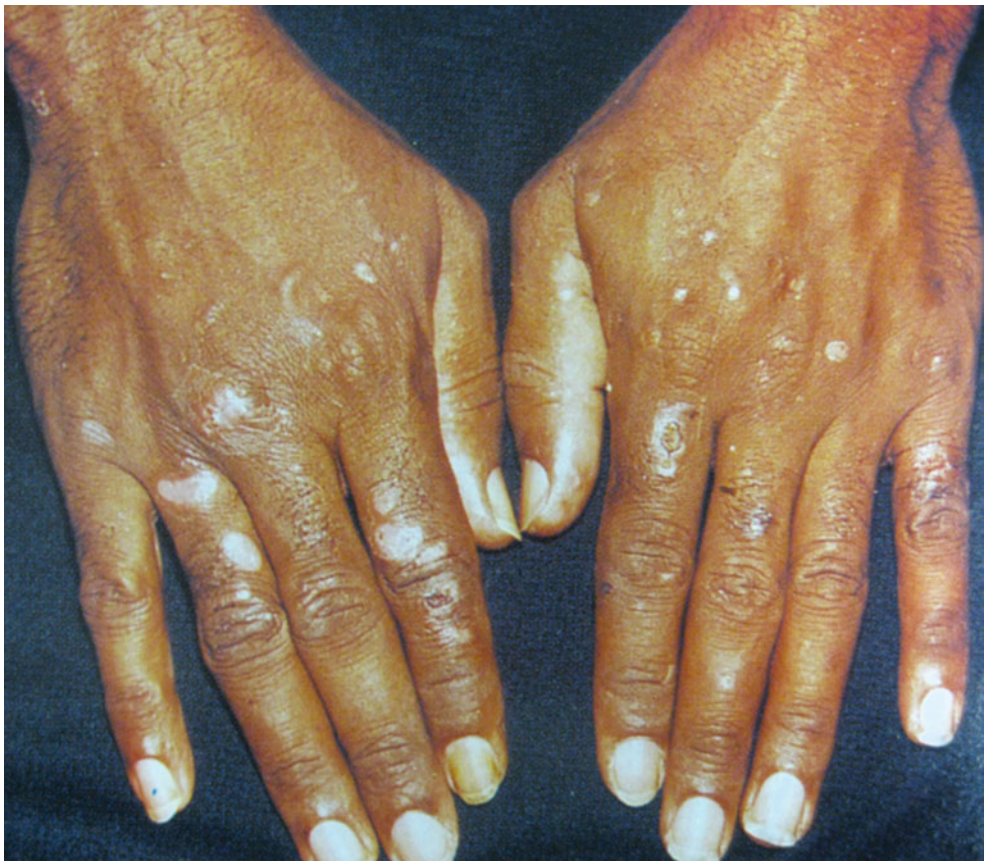


Fig. 10.5 Even dark-skinned individuals are susceptible to pa. The action spectrum (active wavelength) is in the *vis*ible range (*400-nm* wavelength, Sorer's band)

Fig. 10.6 Nail changes in PCT show distal subungual hemorrhage

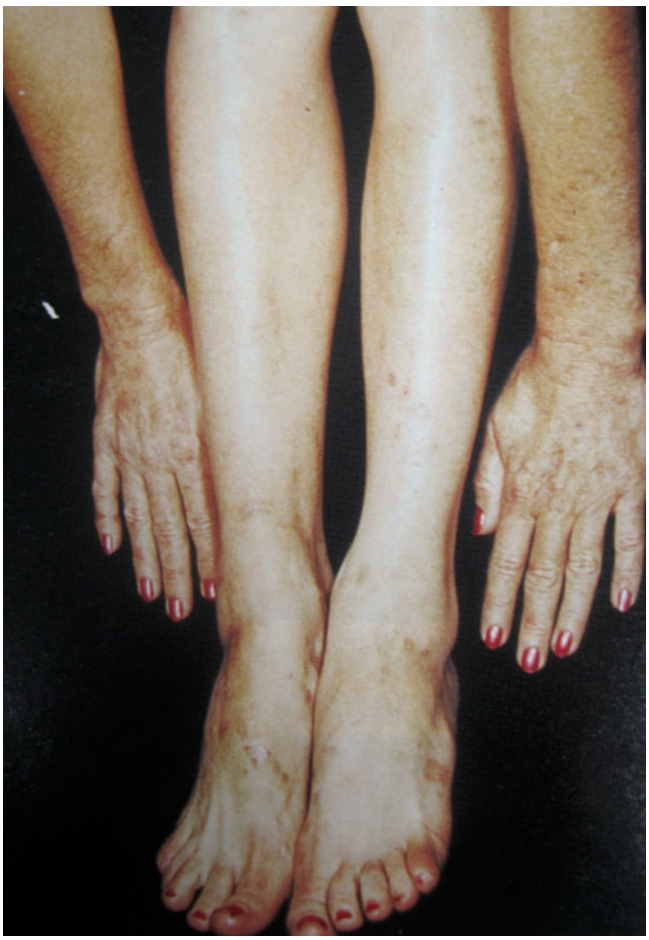


Fig. 10.7 Sun-exposed hands and feet are most susceptible. Gloves and closed-toe shoes are recommended for PCT patients

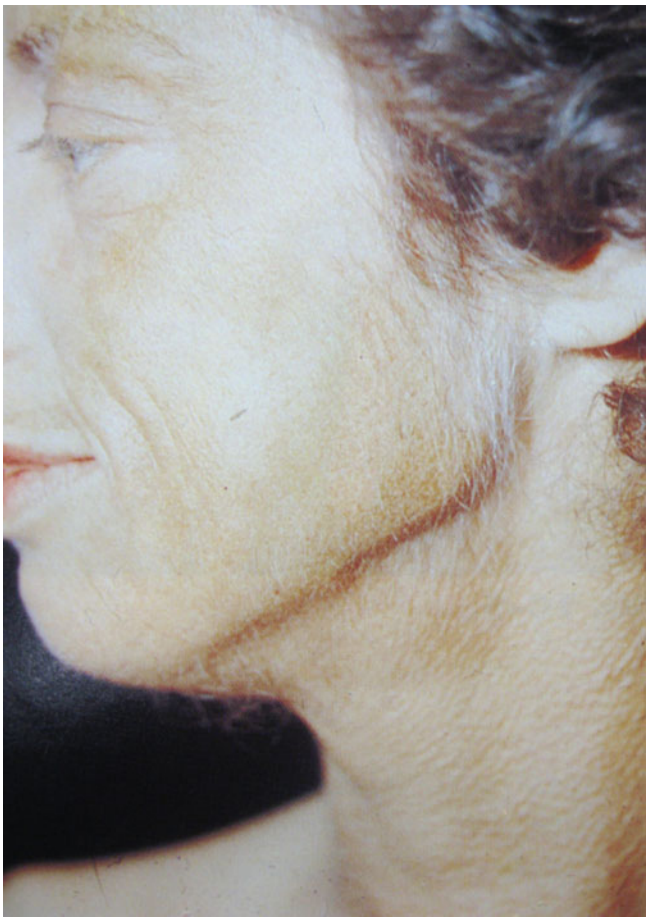


Fig 10.8 Facial hirsutism is common in porphyria patients

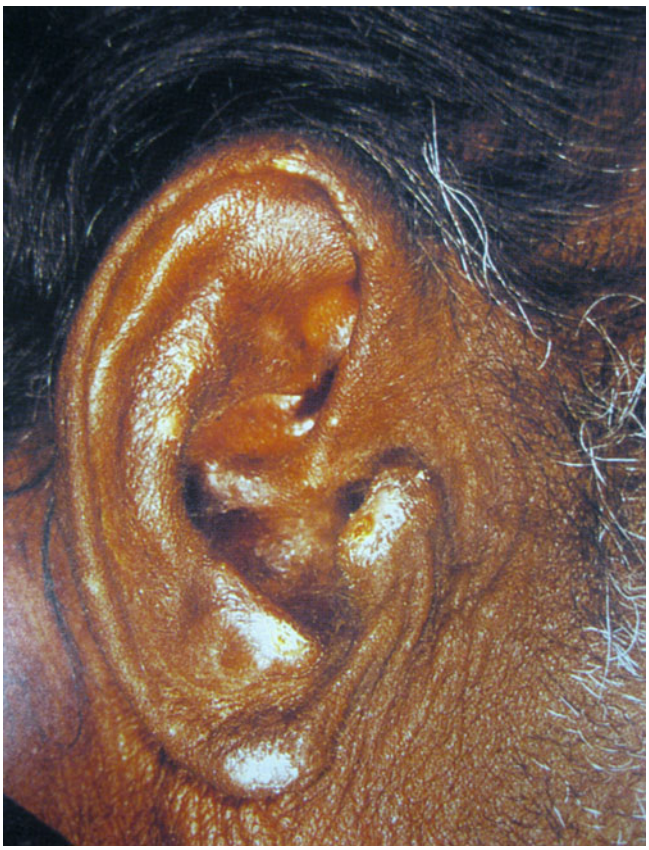


Fig. 10.10 These lesions on the ear resemble those of discoid lupus erythematosus. Direct immunofluorescence of the involved skin, however is usually negative in PO patients

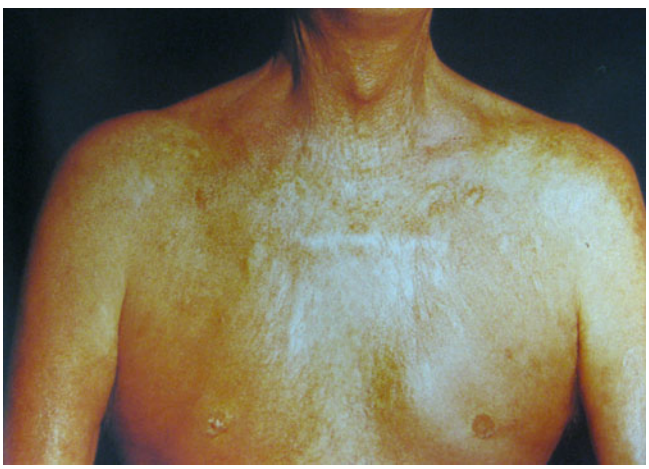


Fig. 10.9 Sclerodermoid variant of PCT shows hypopigrnented. indurated plaques and papules on the chest and ear

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Pruritus

11

Clinical Description

Pruritus is a common symptom that evokes scratching [1]. Pruritus is a problem commonly experienced by the elderly. Multiple factors may cause this problem. Excoriations (scratches) may be localized, generalized, or limited by mobility of the patient.

Etiology and Pathophysiology

Pruritus may be caused by various stimuli, inflammation, chemical mediators and mechanisms. Histamine is one of the most significant chemical mediators responsible for itching (others include bradykinin and prostaglandins). This chemical is stored in the mast cells in the skin and is released in response to various stimuli. They include:

1. Xerosis – Xerosis is the most common cause of pruritus in the elderly.
2. Contactants – Stimuli, such as wool fabric and touch may elicit itching.
3. Foods – citrus fruits (oranges, tomatoes, bell peppers), berries, and shellfish (shrimp, lobster, crab)
4. Medications – Asprin, Barbituates, Opioids, Morphine Sulfate, Penicillin, Chemotherapeutic agents, IV contrast dye and certain antifungal solutions
5. Infections and Infestations – Scabies, Pediculosis
6. Metabolic Causes – Diabetes mellitus, Cholestasis, Iron deficiency anemia, multiple sclerosis, renal and hepatic diseases, thyroid disease, Polycythemia Rubra Vera, HIV or AIDS, Neuropathy, delusions of parasitosis, Obsessive Compulsive Disorder, and Neurodermatitis.
7. Neoplasia – Hodgkin lymphoma, and other metastatic cancers
8. Dermatologic Causes – Atopic dermatitis, contact dermatitis, dematophytosis, lichen simplex chronicus, psoriasis, urticaria, pediculosis (lice), Scabies
9. Neurologic Causes – Nerve damage from aging and minor cerebrovascular accidents

Histopathology

Usually, histopathology investigation yields insufficient results unless an underlying skin disease is present. Lichen simplex chronicus can cause a hypertrophy or thickening or the epidermis and cutaneous nerves.

Therapy

1. The use of hydration, moisturizers, antihistamines, antipruritic lotions, topical and injectable steroids are generally best used in combination to help alleviate itching [2].
2. Topical therapy with lotions containing menthol, phenol, camphor and calamine. Pruritus due to dry skin can be improved with the application of emollient lotions. Topical doxepin, a potent histamine receptor antagonist, was extremely effective in controlling severe pruritus, especially in burn patients [3].
3. Oral therapy with antihistamines may be necessary. Non-sedating antihistamines (loratadine, fexofenadine, or cetirizine) may be prescribed. If these drugs prove ineffective, then a low dose of hydroxyzine may be ordered short term and is often prescribed for night time administration due to its sedating effects. Diphenhydramine is best avoided in the geriatric population as it may increase the risk for falls due to the anticholinergic properties of the medication [4].
4. UVB light and activated charcoal have proven useful for uremic pruritus and pruritus associated with hemodialysis [5].

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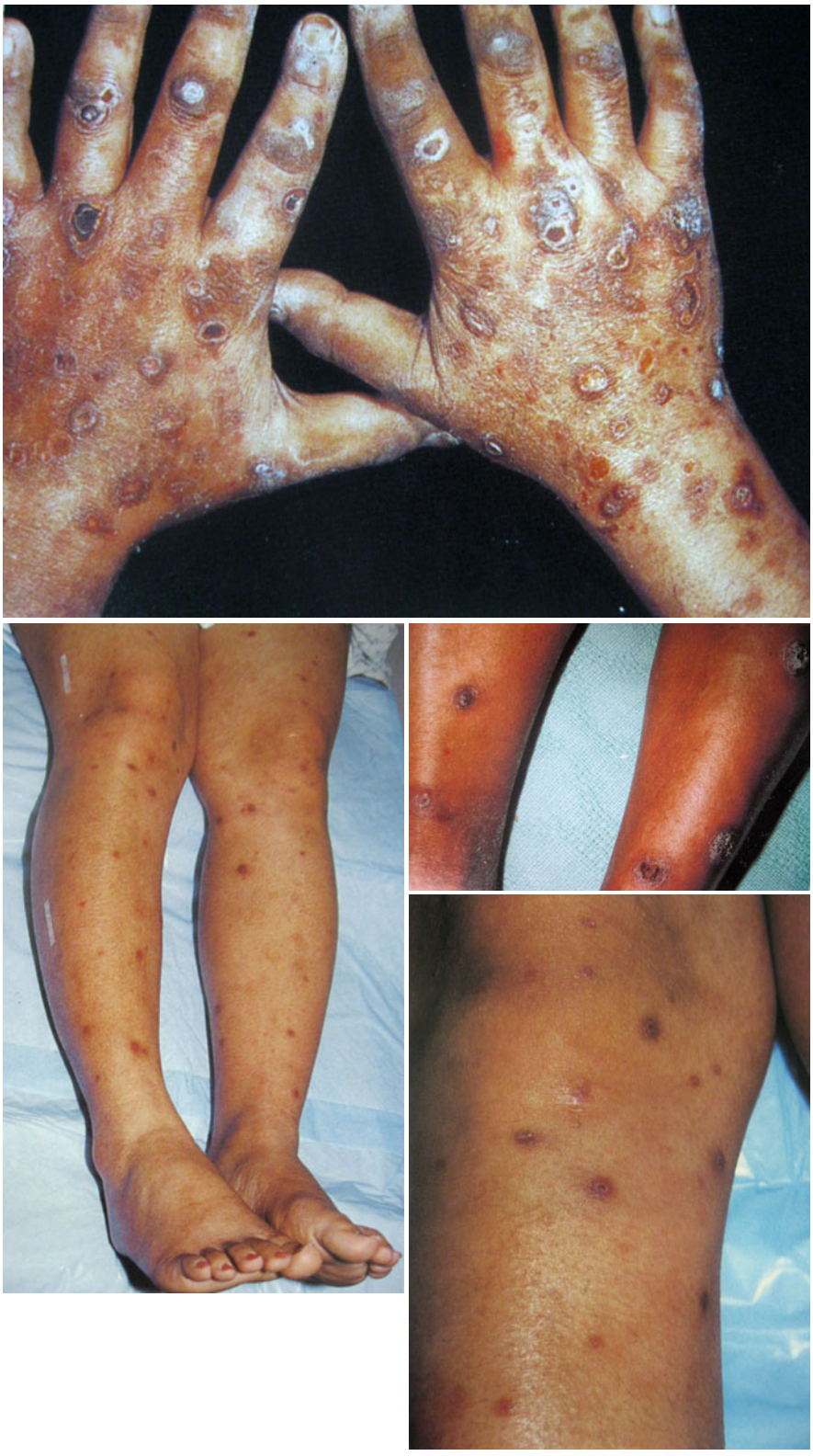
Prognosis

Prognosis usually depends on the underlying cause of pruritus. The careful history and physical examination should take into account the different types of itching and their duration, the quality of itching, and its distribution and timing. Healthcare providers should strive to make a full assessment with various laboratory tests to rule out internal malignancy. Superficial pruritus has a positive outlook with proper treatment and follow-up care [6–8].



Figs. 11.1, 11.2, 11.3, and 11.4 Prurigo nodularis (picker's nodule) is a localized, nodular form of lichen simplex chronicus (neurodermatitis) induced *by* chronic picking and scratching. Hyperpigmentation, erosions or ulcerations, and secondary bacterial infections can occur

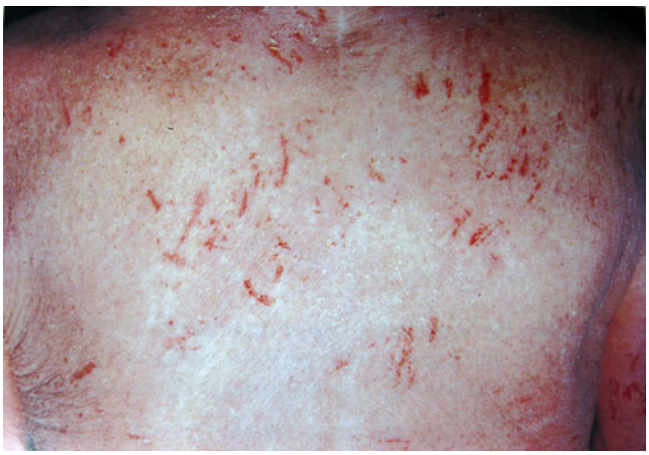


Fig. 11.5 Linear excoriations of the chest occurred in this patient with severe dry skin, or xerosis/see Chap. 19

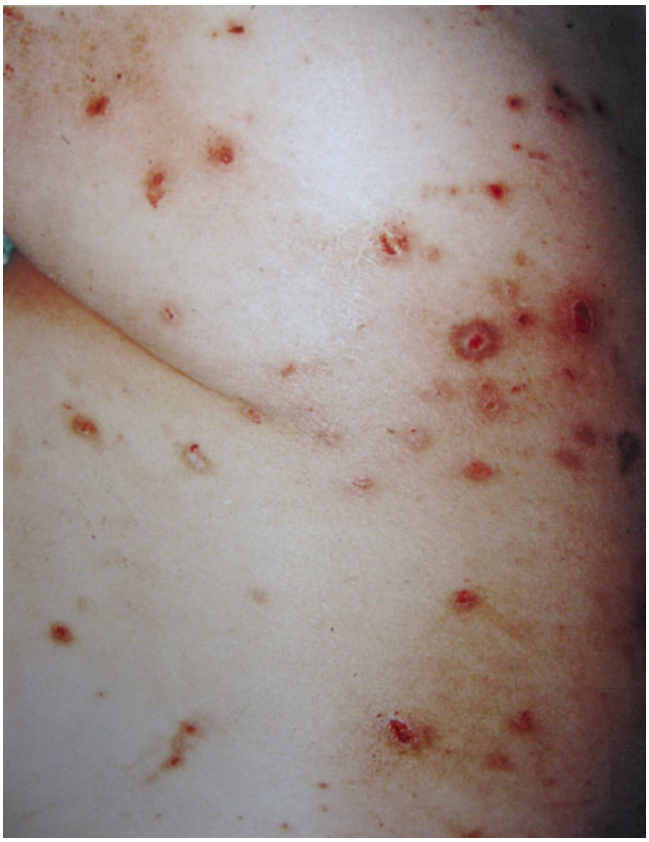


Fig. 11.6 Generalized crusted ulcerations and erosions developed in this patient with pruritus secondary to renal failure and hemodialysis. The peripheral hyperpigmentation may be permanent

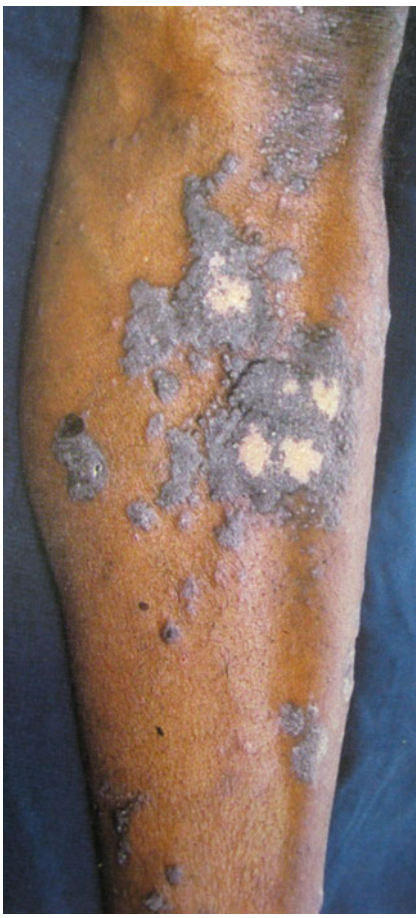
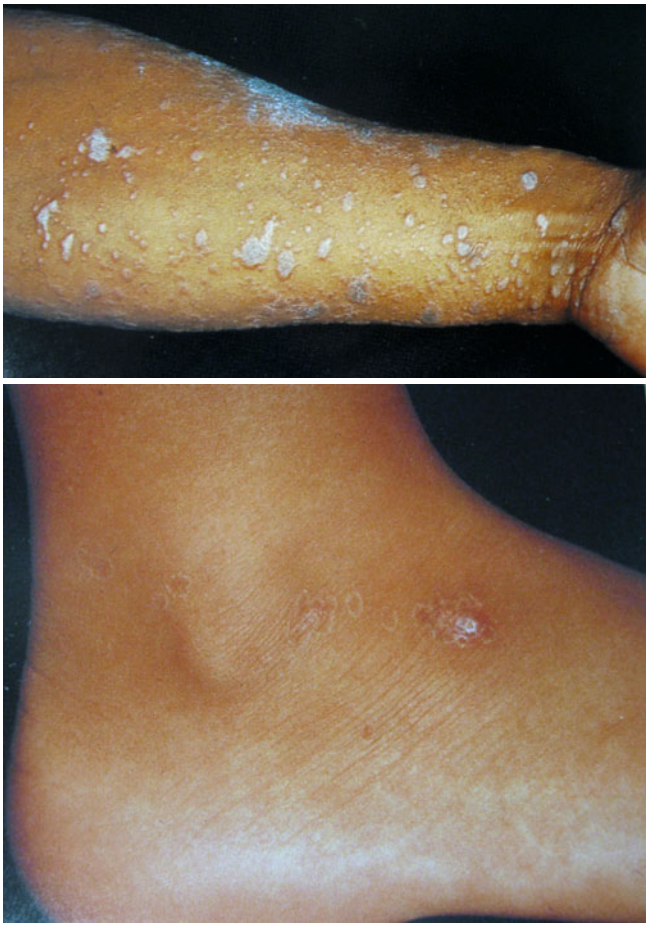


Fig. 11.7 Hypertrophic lichen planus clinically resembles lichen simplex chronicus. It is characterized by extremely pruritic purple papules and plaques. Note the ulcerations from deep scratching



Figs. 11.8 and 11.9 Typical lichen planus of the arm and foot. Pruritic, purple, polygonal papules and plaques display lacy, cross-hatched white lines (Wickham's striae)

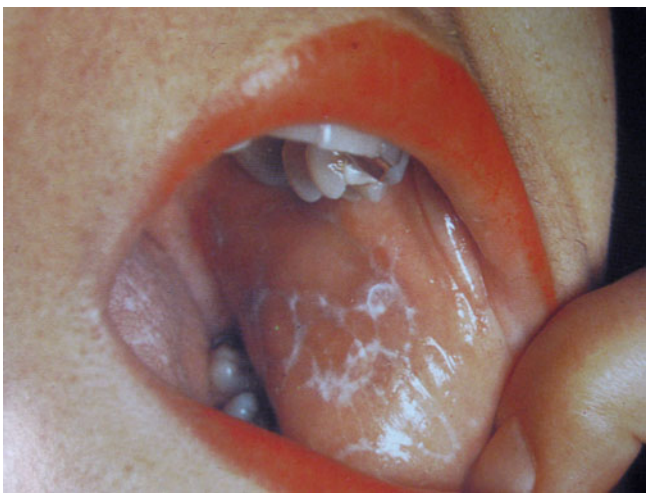


Fig. 11.10 Oral lichen planus. Note the reticulate racy white pattern on the buccal mucosa

Fig. 11.11 Lichen planopilaris. Lichen planus of the scalp causes scarring alopecia

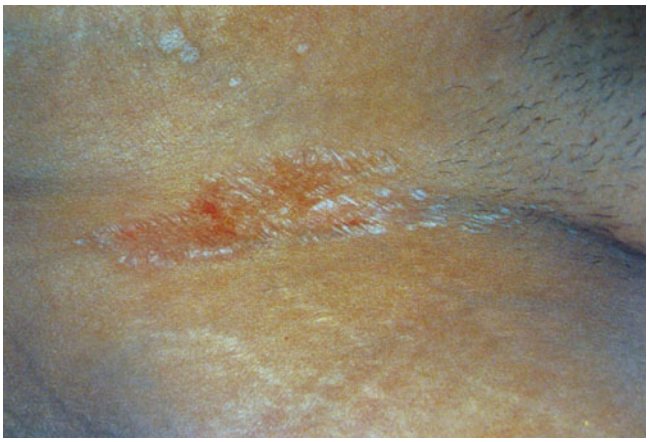


Fig. 11.12 Atrophic lichen planus of the groin clinically resembles lichen sclerosus et atrophicus and intertrigo

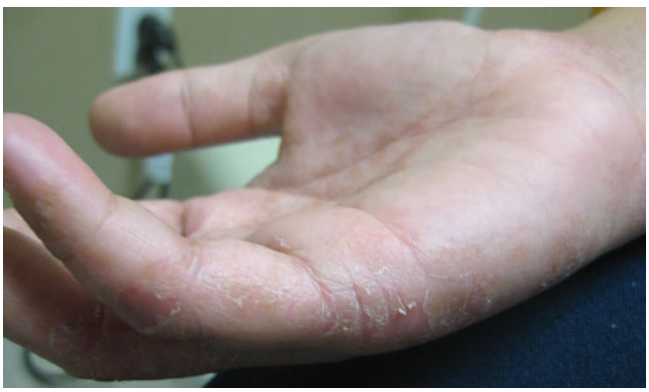


Fig. 11.13 Eczema with concurrent pruritus

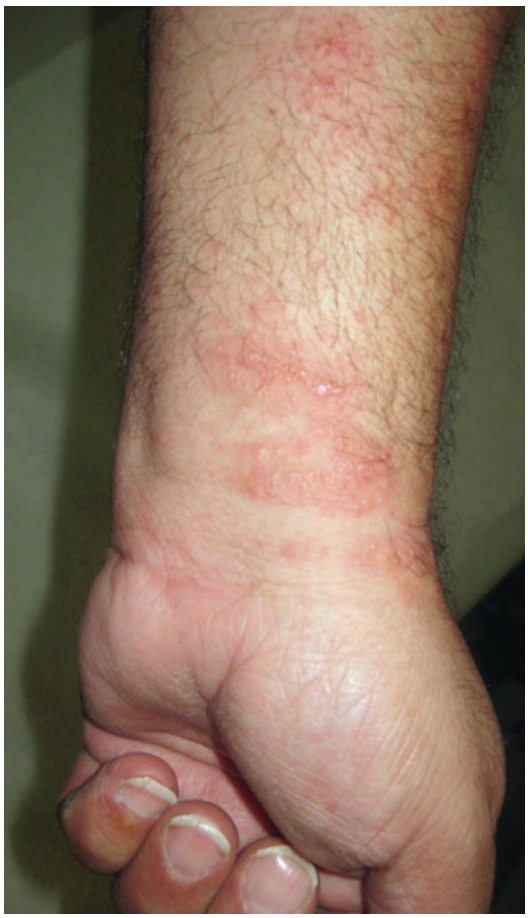


Fig. 11.14 Contact dermatitis in patient with localized pruritus

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Psoriasis

12

Clinical Description

Psoriasis is a common, noncontagious, multisystem, inflammatory dermatosis of the skin. The cause is unknown but studies suggest a genetic predisposition for the disorder. It commonly manifests itself on the skin of scalp, lumbosacral areas, intergluteal clefts, pubic regions, and joints such as elbows, knees, wrists, and ankles. Psoriasis affects 1–2 % of the world's population. It may begin at any age, but is more common in the second or third decade of life. Multiple types of psoriasis have been identified and distinguished by location and involvement:

1. Classic psoriasis: Exhibits scaly, red erythematous macules, papules, and plaques. Macules and papules can progress into well-demarcated, silver-white, adherent, micaceous plaques. Chronic stationary psoriasis is the most common type involving the scalp, extensor surfaces, genitals, and lumbosacral and retroauricular regions.
2. Plaque psoriasis: Characterized by raised, inflamed plaques covered with a silvery white scale. It is most common on the extensor aspects of the knees and elbows, the sacral region and the scalp. It can spread to the whole skin surface as seen in psoriatic erythroderma.
3. Guttate psoriasis: Presents as smaller, pink/red papules, 1–10 mm in diameter. It commonly manifests on the trunk. Lesions can be scaly.
4. Inverse or Flexural psoriasis: Occurs in regions of skinfolds and flexural surfaces such as the armpit, groin, and under the breast. It typically manifests in those past the age of 60 and is characterized by smooth, inflamed lesions without scaling due to high moisture environment on the body. There is a higher incidence of this type in obese patients.
5. Pustular/Erthrodermic psoriasis: Presents as sterile pustules on the palms and soles or diffusely (von Zumbusch's disease). In contrast to the other types, pustular psoriasis can be painful and cause soreness in affected areas. The generalized erythema poses the danger of hypothermia from heat loss. Dehydration, fevers, and chills are often accompanying complications because of the large area of skin affected.
6. Scalp psoriasis: Affects nearly 50 % of all patients and presents as erythematous plaques with silvery white scales on the scalp, scalp margin, and ears.
7. Nail psoriasis: Also affects up to 50 % of patients with an estimated lifetime incidence of 80–90 %. Affected nail plates thicken and crumble. Nail changes include pitting, onycholysis, yellow-tan discoloration, and subungual hyperkeratosis. Psoriatic Arthritis (PsA) affects nearly a third of those with skin symptoms. It produces stiffness, pain, and eventually severe deformity of the terminal interphalangeal joints.
8. Oral psoriasis: Manifests as whitish lesions on the oral mucosa. Severe cheilosis is also typically present. Geographic tongue is also considered to be a form of oral psoriasis.
9. Ocular signs also occur in approximately 10 % of patients with psoriatic skin disease [1–4].

Etiology and Pathophysiology

Psoriasis is a complex multifactorial disease. Causative factors include genetic and immune-mediated components. The exact cause is unknown but several theories point to various sources. Environmental factors include stress from cold, trauma, infections, alcohol and drugs such as iodides, lithium, antimalarials, and betablockers. Patients with psoriasis have a predisposition for the disease and the triggering event in many cases is an upper respiratory infection. A meta-analysis study by Riveira-Munoz et al. confirms that the deletion of LCE3C and LCE3B is a common genetic factor for susceptibility to psoriasis in the certain populations. Psoriatic lesions are associated with increased activity of T cells in the underlying skin. High levels of dermal and circulating TNF-α are usually present. Patients with HIV typically have a decrease in CD4 T cells, which leads to overactivity of CD8 T cells, worsening the condition. Guttate psoriasis often appears following streptococcal pharyngitis, cessation of steroid therapy, or use of antimalarial drugs. Median onset is 28 years and prevalence is African Americans is 1.3 % compared with 2.5 % in Caucasians [5, 6].

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Histopathology

Plaque psoriasis shows hyperparakeratosis, regular acanthosis, neutrophils in stratum corneum, intraepidermal neutrophils, and widely dilated dermal capillaries with perivascular lymphocytic infiltrate.

Therapy

1. Topical therapies commonly used include topical steroids, Vitamin A, tars, PUVA, light therapy, cortisone (Cordran) tape dressing. Intralesional steroids injections are sometimes used for thick plaques.
2. Oral therapies include Methotrexate, Cyclosporine, and oral steroids. These treatments provide temporary relief and short periods of remission but increase the risk of hepatoxicity, nephrotoxicity, and immunosuppression for the individual.
3. Many new medicines have been developed to target the immunologic and inflammatory cascades in psoriasis. Entanercept, adalimumab, and infliximab are anti-tumor necrosis factor therapies. These biologic treatments have proven to be quite effective [7, 8].

Prognosis

Cardiac disease, depression, and other related factors must be monitored carefully. Psoriasis is controllable but not curable, with intermittent relapses and remissions [9].

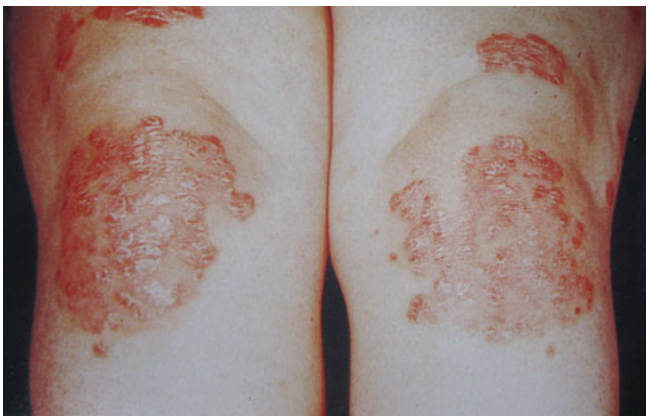


Fig. 12.1 Classic plaque psoriasis affects the extensor surfaces, especially the knees

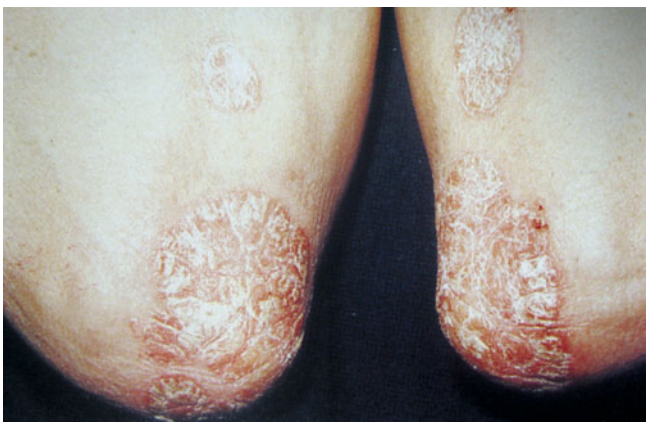


Fig. 12.2 Classic plaque psoriasis affects the extensor surfaces, especially the elbows

Fig. 12.3 Large plaque psoriasis involving the intertriginous areas under the breast, so-called "inverse psoriasis"

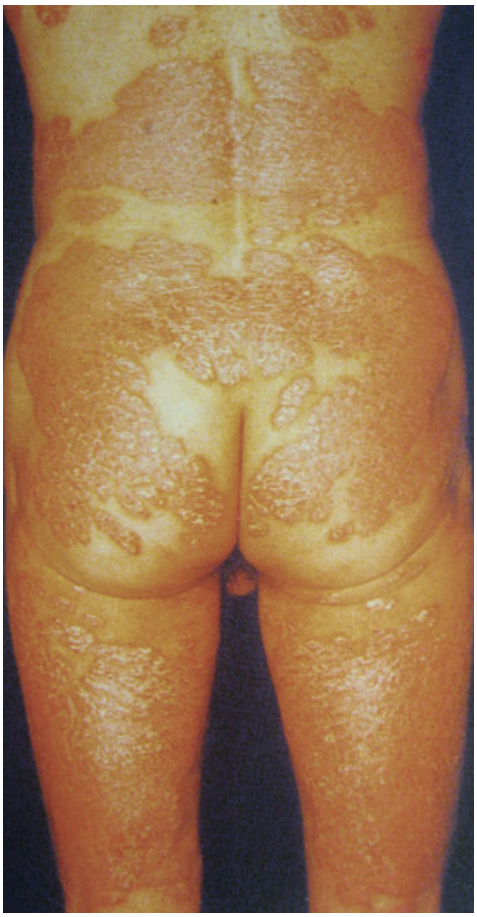


Fig. 12.4 Symmetric, geographic, generalized large plaque psoriasis of the trunk

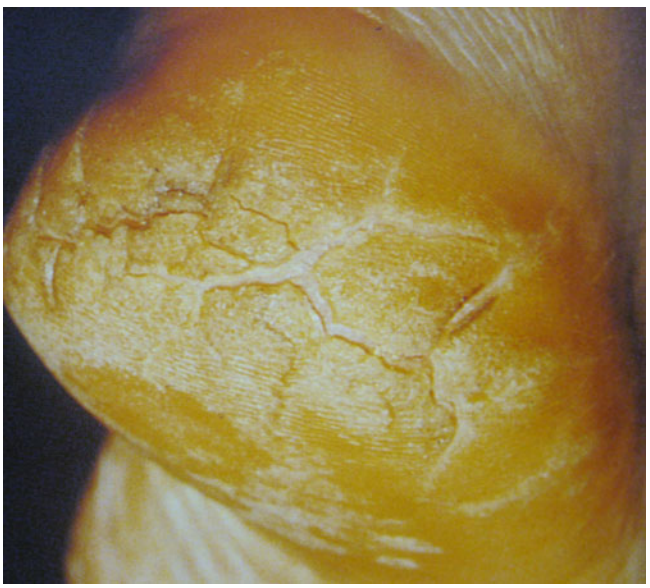


Fig. 12.5 Hyperkeratotic, fissured heels in plantar psoriasis

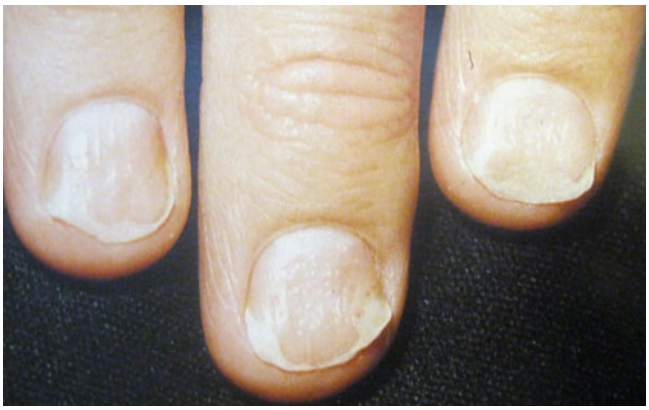


Fig. 12.6 Psoriatic nails show pitting, onycholysis (separation) and yellow-brown discoloration (oil spotting)

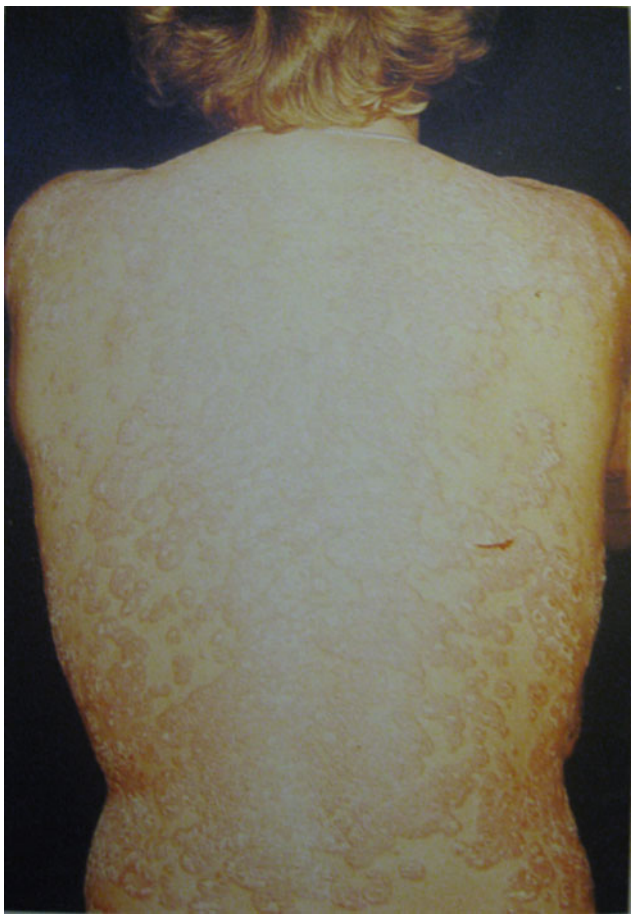


Fig. 12.7 The differential diagnosis of erythrodermic psoriasis includes generalized eczemas. mycosis fungoides, and pityriasis rubra pilaris

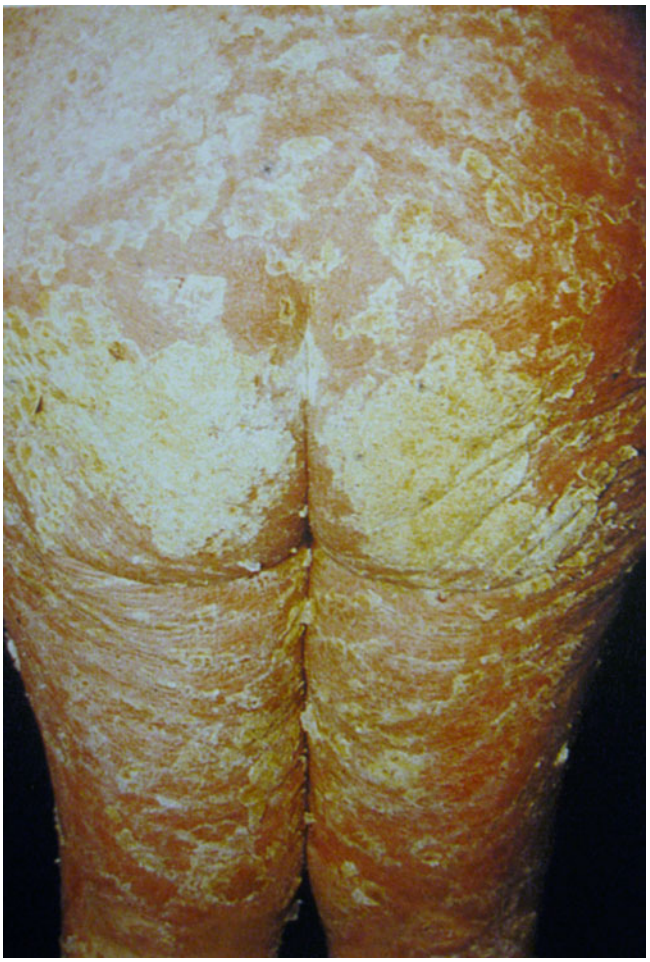


Fig. 12.8 Erythrodermic psoriasis with micaceous, silvery scale

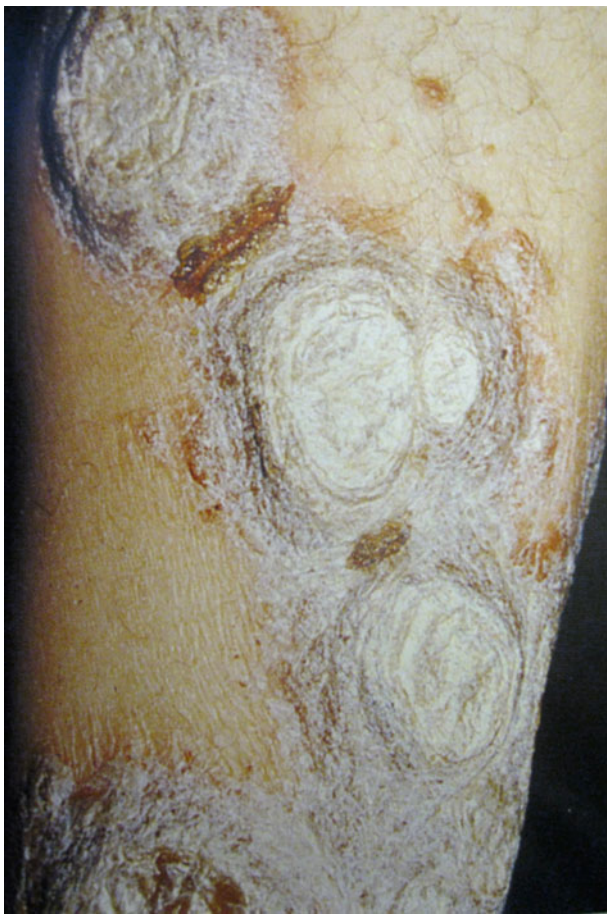


Fig. 12.9 Rupioid (oyster shell) hyperkeratotic plaques

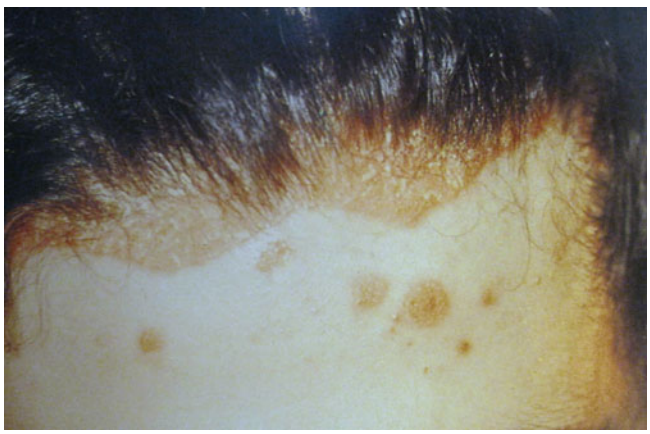


Fig. 12.10 Scalp psoriasis showing sebopsoriasis overlap syndrome

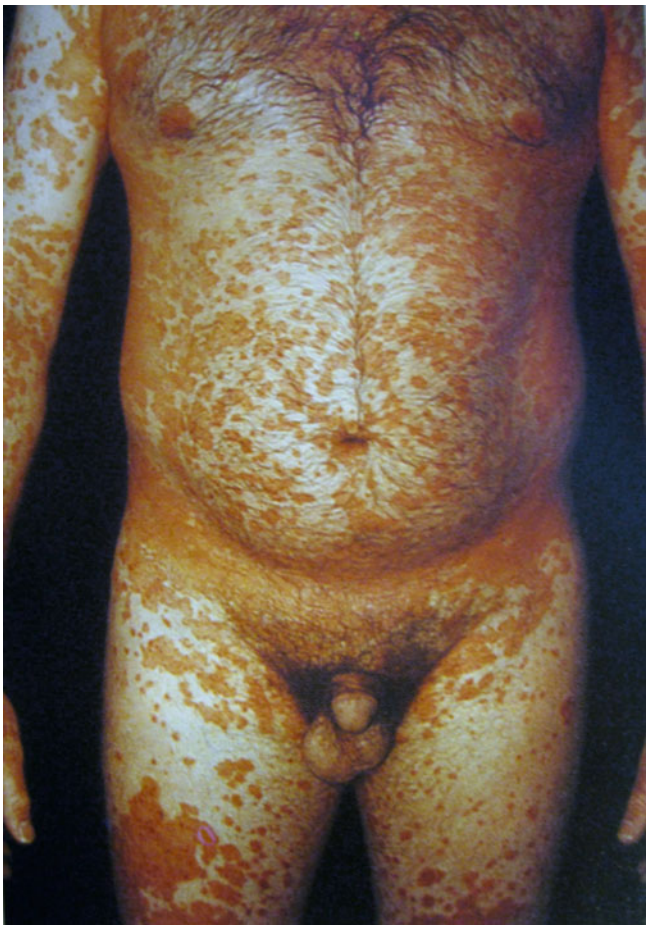


Fig. 12.11 Guttate (drop-shaped) psoriasis triggered by a streptococcal throat infection

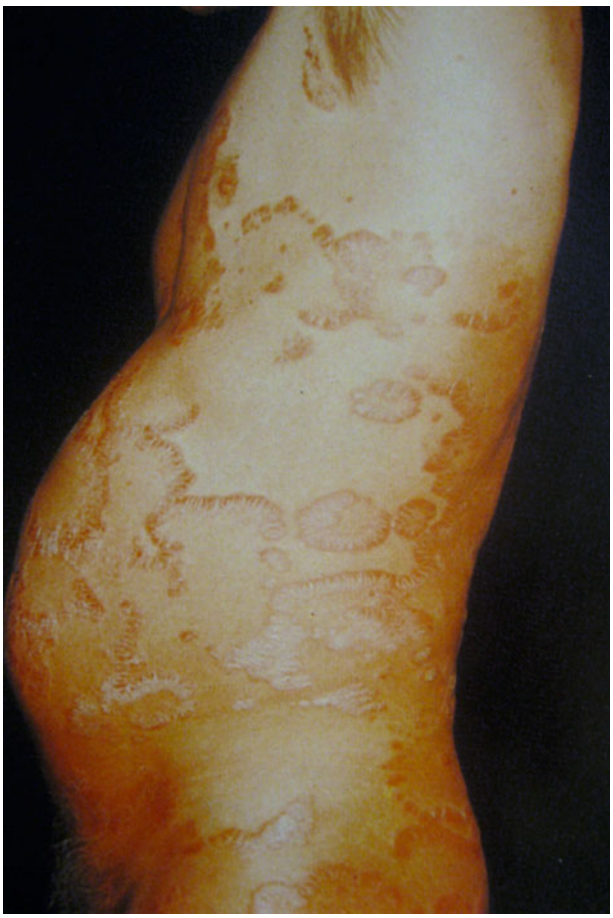
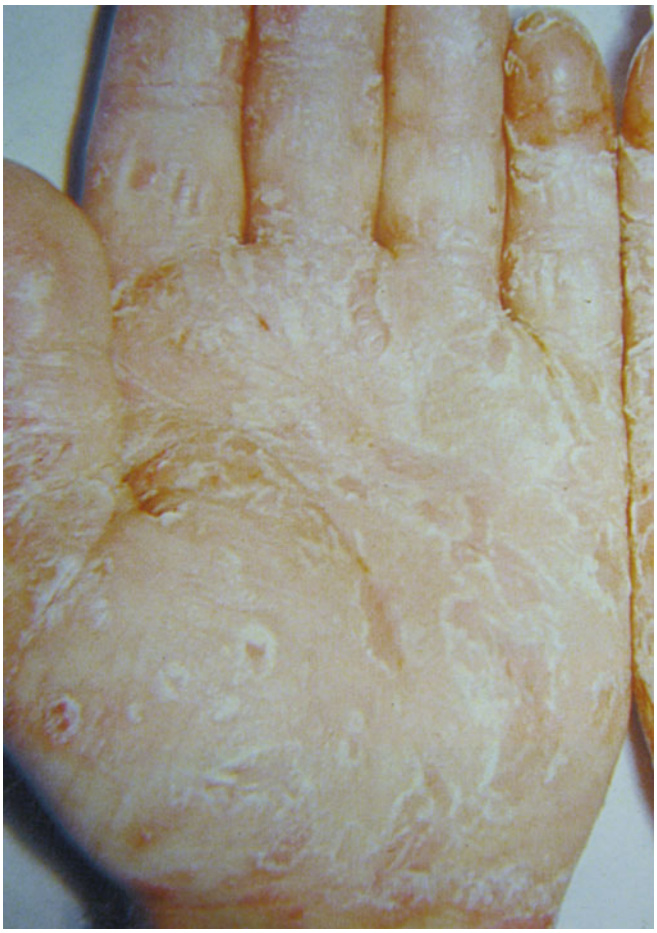
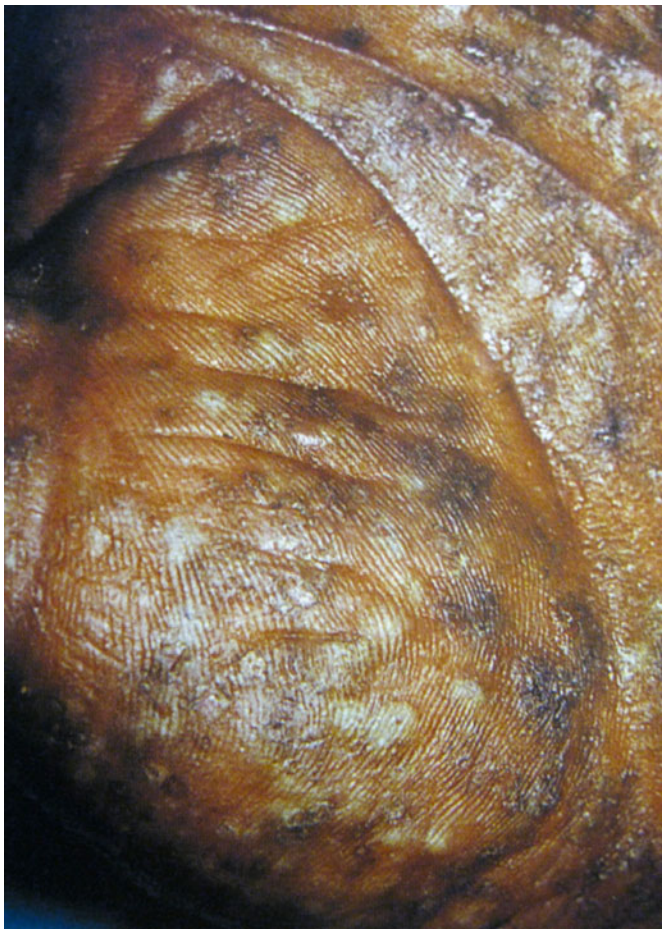


Fig. 12.12 Annular and arcuate geographic plaques showing central clearing and peripheral blanching (Woronoff's ring). The plaques resemble those of ringworm or mycosis funqoides





Figs. 12.13 and 12.14 Pustular psoriasis of the hands features sterile pustules that eventually become confluent and desquamative

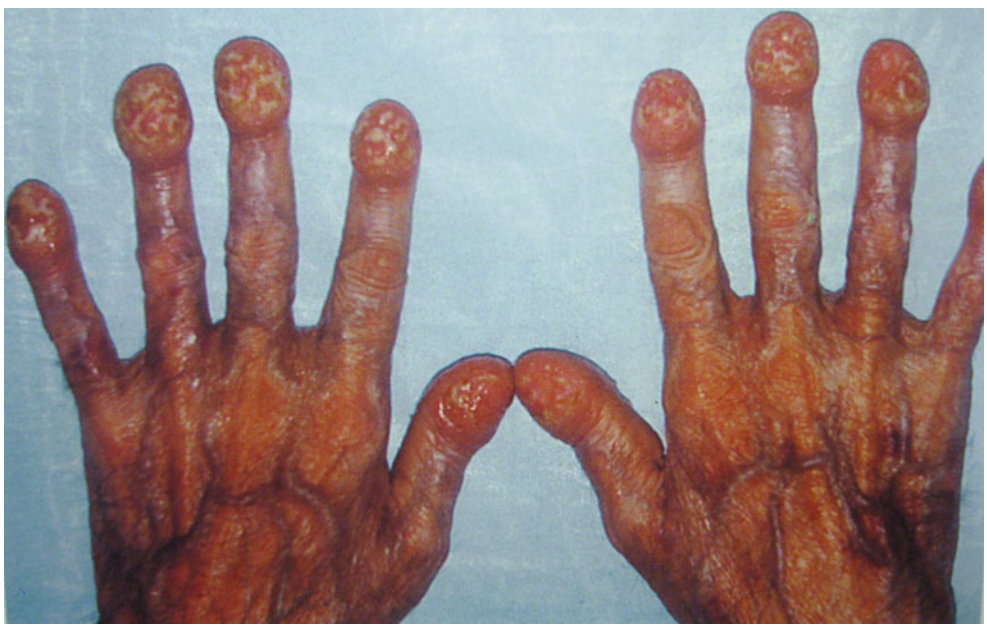
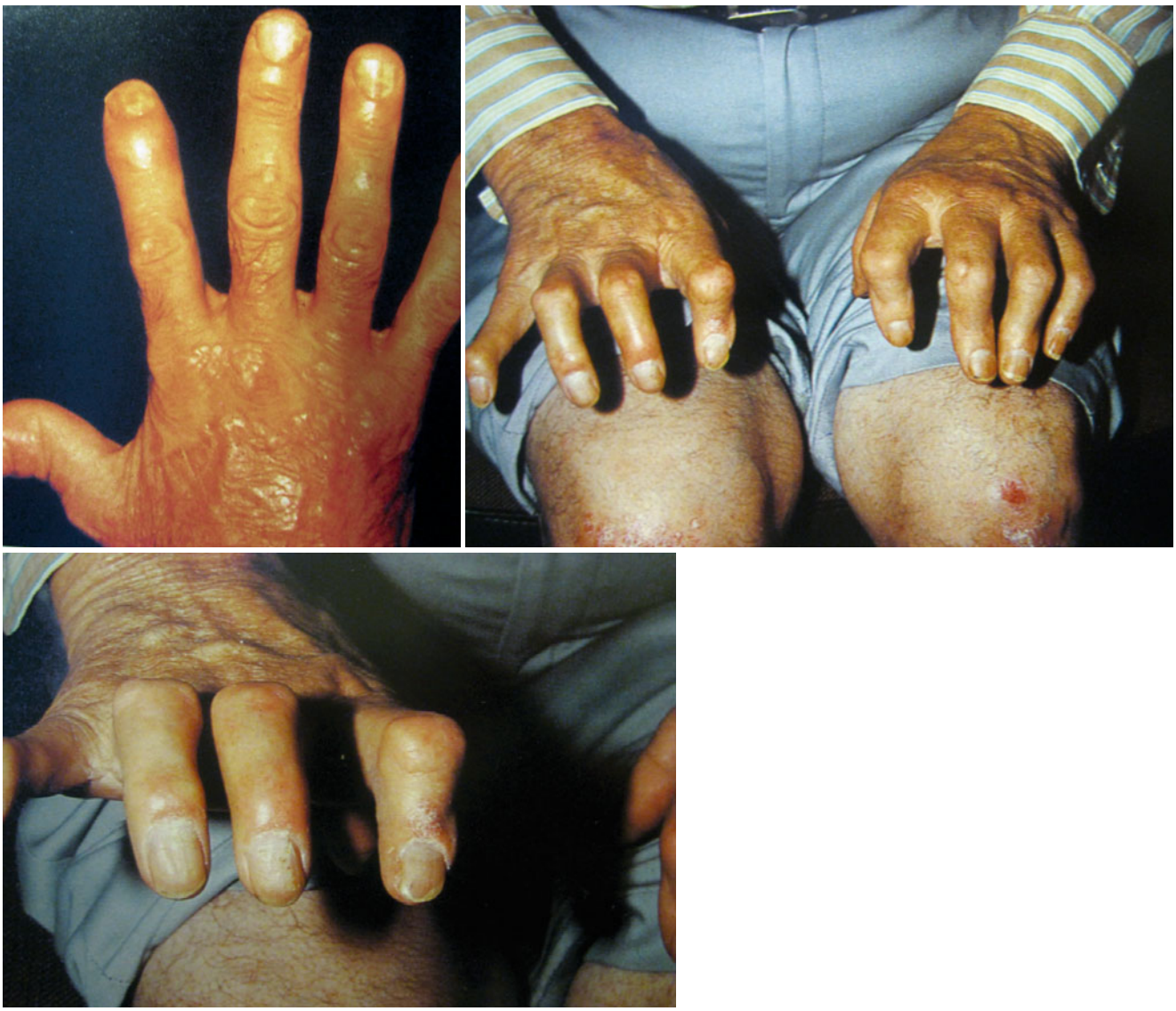
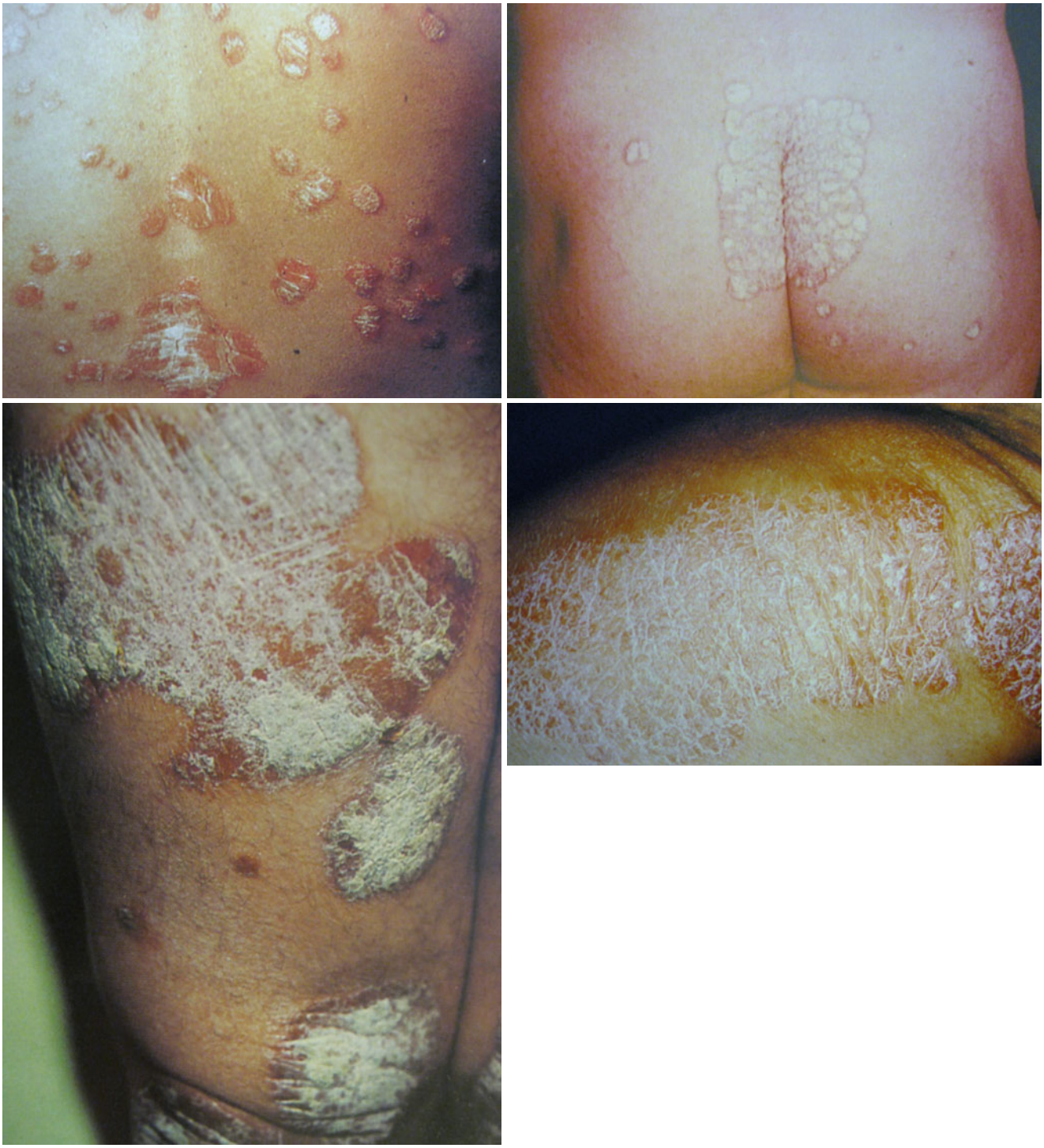


Fig. 12.15 *Severe* pustular psoriasis involving the fingertips and nail beds



Figs. 12.16, 12.17, and 12.18 Psoriatic arthritis can often be correlated with the severity of cutaneous and nail involvement



Figs. 12.19, 12.20, 12.21, and 12.22 Classic plaque psoriasis

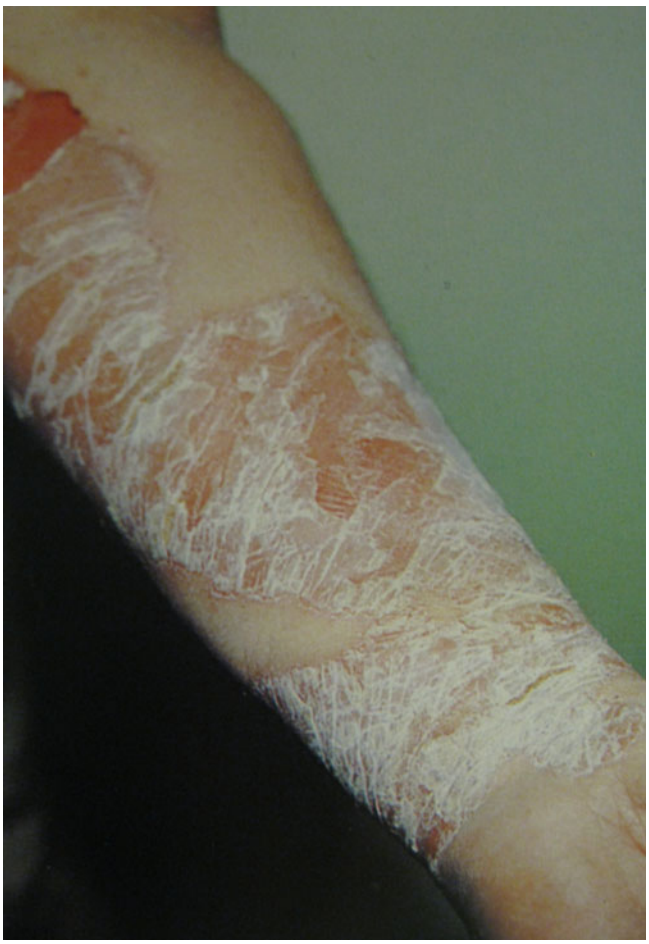
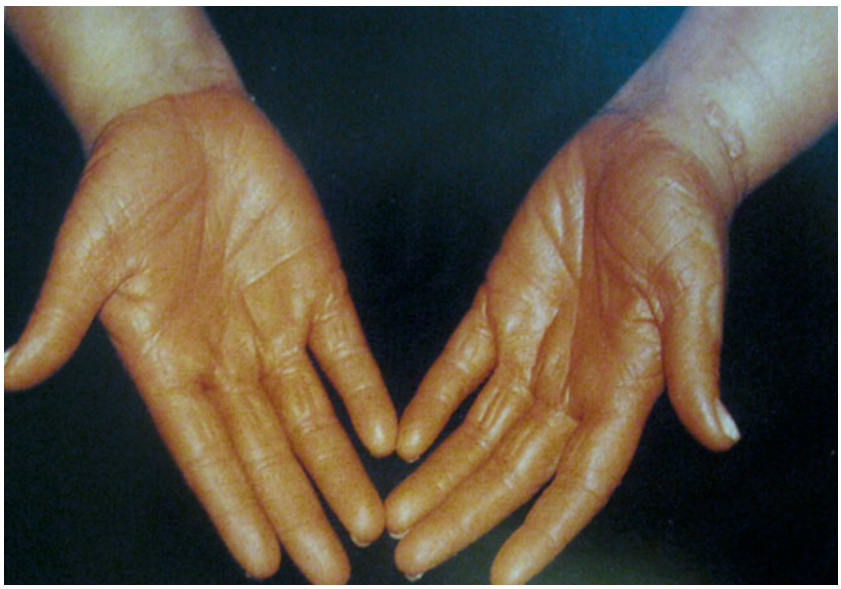
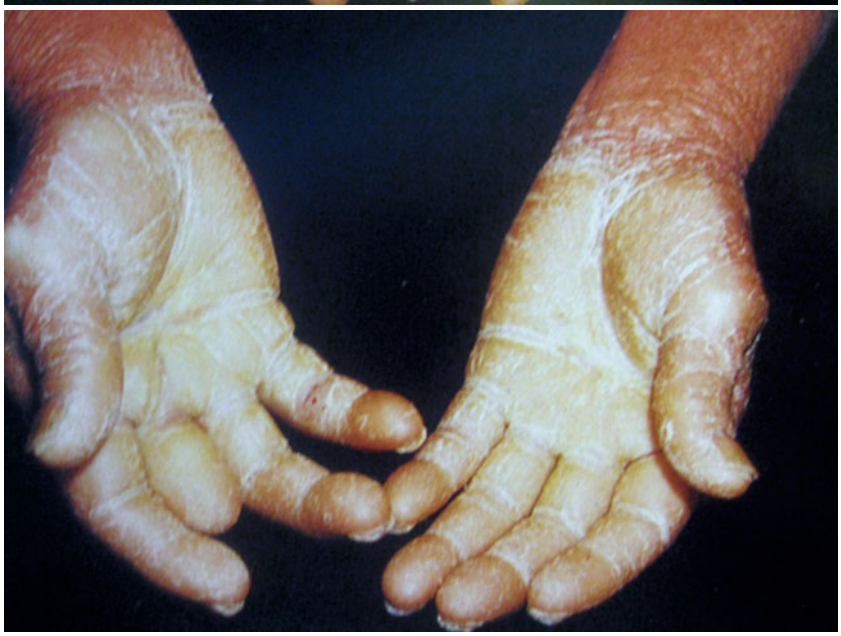
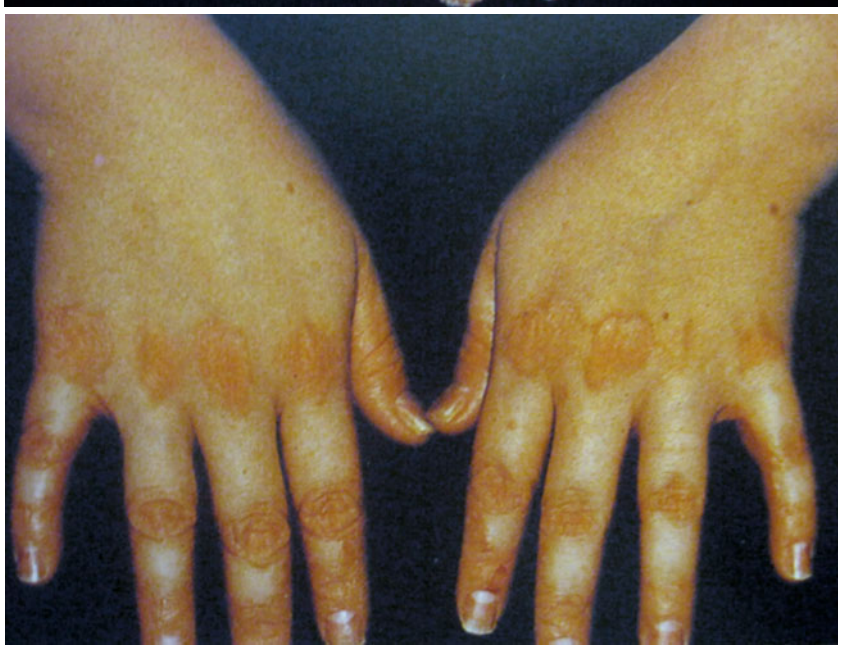


Fig. 12.23 Auspitz's sign illustrates bleeding capillary punctae at sites of scale removal

Figs. 12.24, 12.25, and 12.26 Pityriasis rubra pilaris (PRP) is manifested by moccasin-type erythema and keratoderma of the palms and soles. Salmon pink-orange follicular and nonfollicular psoriasiform papules with a keratotic plug are best seen on the dorsal hands and fingers. The nonfollicular papules coalesce to form scaling plaques and, ultimately, scaling erythroderma







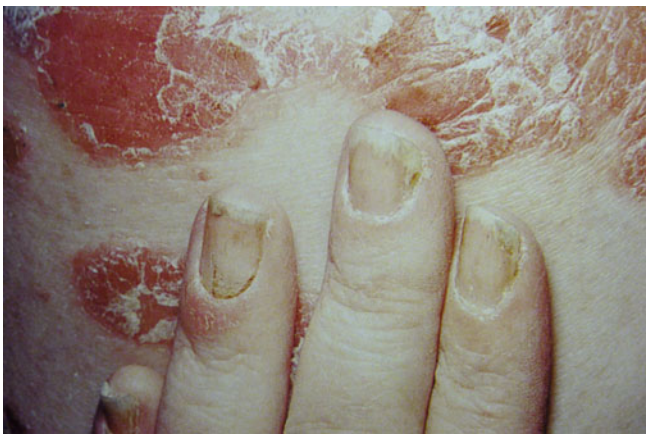


Fig. 12.27 Classic psoriatic skin and nail changes: pitting, onycholysis, subungual hyperkeratosis, and oil spotting

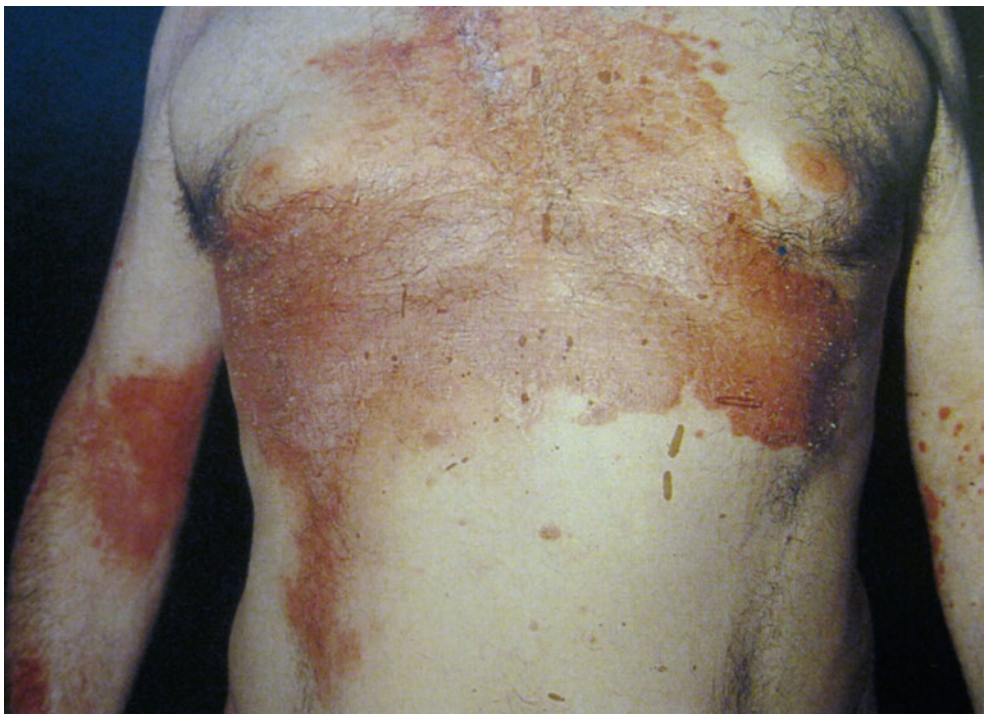


Fig. 12.28 Anthralin burn from overzealous topical application of anthralin and ultraviolet light

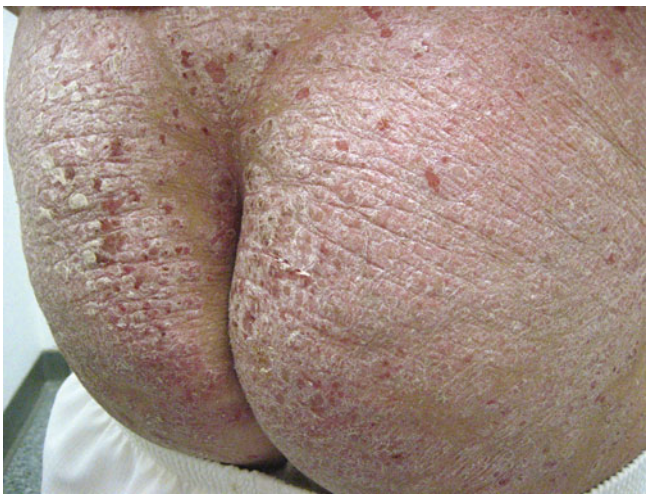
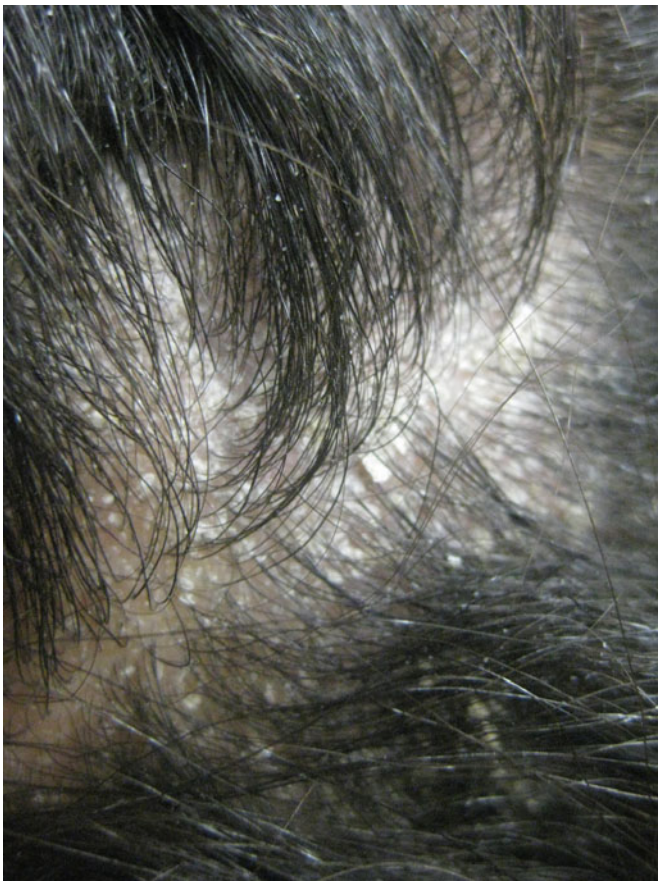
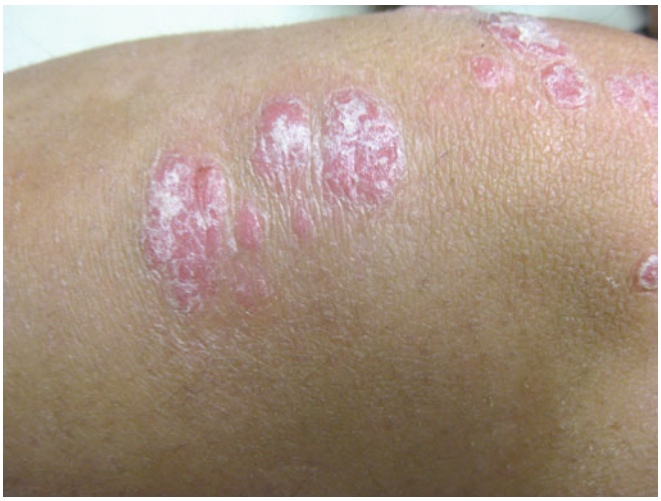
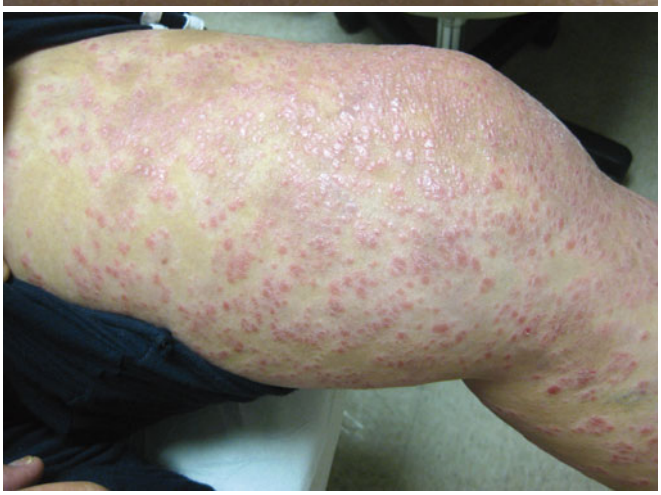


Fig. 12.29 Psoriasis on buttocks







Figs. 12.30, 12.31, and 12.32 Psoriasis on scalp and extremities

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Psychocutaneous Disorders

13

Entities

Psychocutaneous disorders involve the following:

1. Delusions of parasitosis (also called monosymptomatic hypochondriacal psychosis)
2. Neurotic (depressive) excoriations
3. Dermatitis artefacta (also called factitial dermatitis)

Clinical Description

Patients who present with these psychocutaneous disorders have an underlying psychiatric disorder.

Delusions of Parasitosis – Patients with these delusions function normally in other aspects of life, but comorbid anxiety, depression, and denial are common. The delusional patient is often defensive, angry, and hesitant to take any advice from the healthcare provider. The patient complains of infestations. Excoriations are often superinfected and without primary lesions [1].

Neurotic (depressive) Excoriations – Patient denies scratching although scarring and linear marking may be clearly evident. Erosions and superinfected ulcers are also present. There is a lack of scars and excoriations in areas that are hard to reach, such as the central back.

Dermatitis Artefacta – Patients vehemently deny self-trauma of skin lesions, sometimes to satisfy an unconscious need to be taken care of. Providers must be aware of the patient's psychosis and manipulative description as to the cause and duration of the lesions. The patient will also often appear unconcerned and bewildered when reporting the lesions. The lesions tend to be bizarre, angulated ulcers, cutaneous abscesses, sometimes accompanied by fever or malaise [2, 3].

Etiology and Pathophysiology

Patients with neurotic excoriations usually have depression or anxiety with obsessive-compulsive features. The Munchausen's syndrome variant has a sociopathic personality, while patients with dermatitis artefacta are most commonly diagnosed with the borderline personality disorder. Drug-induced "crank" psychosis from cocaine [4] and amphetamines may occur.

Histopathology

Biopsy is usually unnecessary and seldom helps. However, is recommended if necessary to rule out an underlying systemic disorder. Any material brought by the patient as "evidence" should be examined for scabies, lice, or other true infestations.

Therapy

The extensive damage from scratching and self-trauma to the skin requires intensive topical measures. Tar, bleach baths, emollients, and antibiotics, if infections are present, are helpful. Pramoxine cream or lotion, and other topical antipruritics can temporarily relieve any dysesthesia and pruritus. Antipsychotic drugs and psychiatric referral are recommended but the suggestion may not be taken well by this group of patients. Pimozide is commonly used but newer antipsychotics such as Risperidone and aripiprazole are equally effective and safer without the side-effects of weight gain and drowsiness. Depression symptoms can be treated with antidepressants to improve treatment responsiveness. Impulsive picking and scratching habits may be reduced or stopped with cognitive-behavioral methods or hypnosis and self-hypnosis [5–7].

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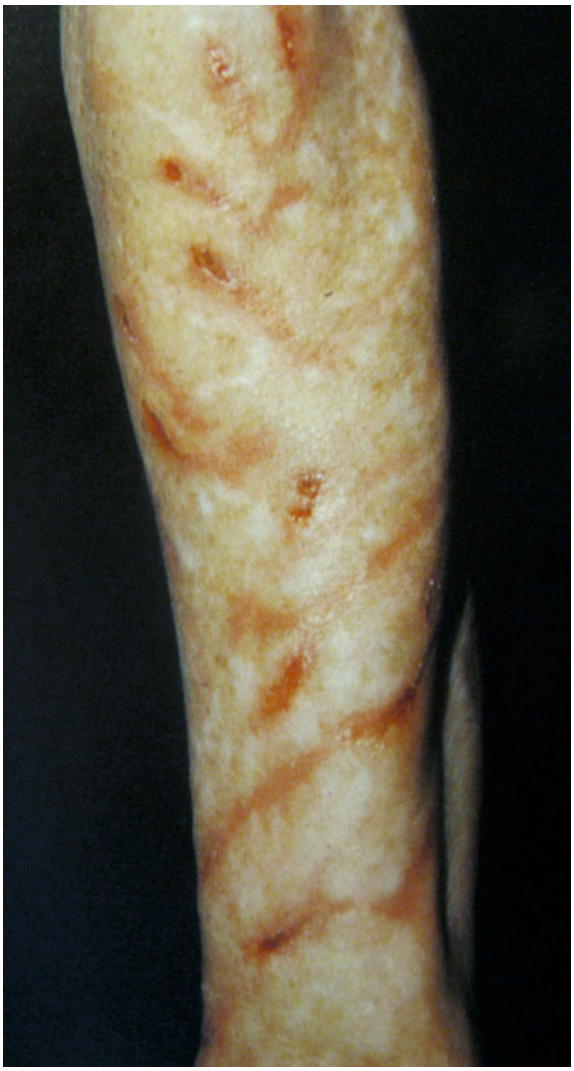
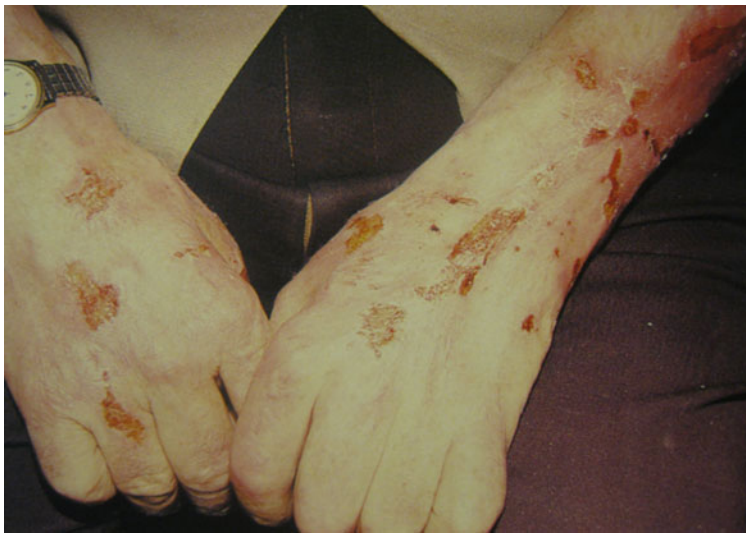
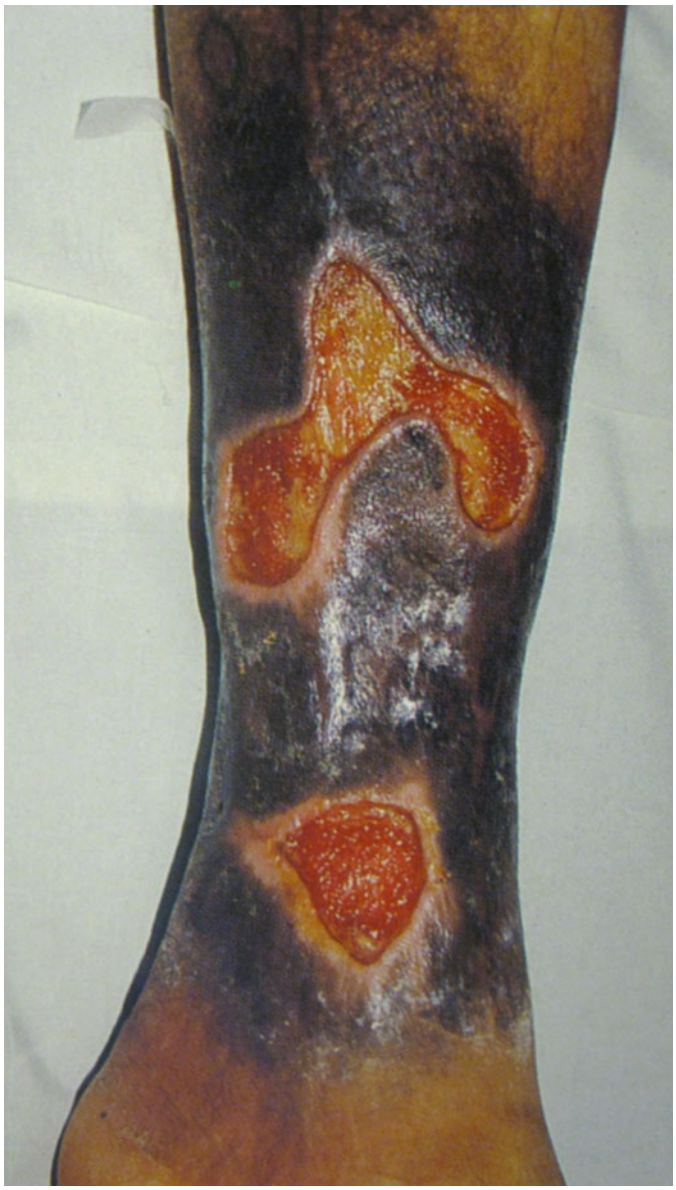
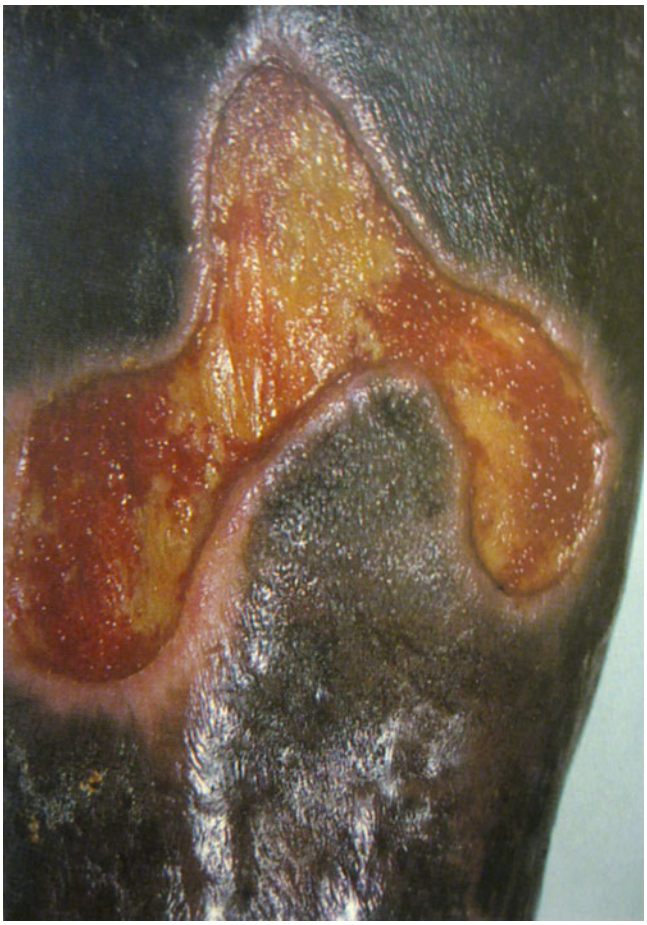




Figs. 13.1 and 13.2 Neurotic "depressive" excoriations are linear erosions and ulcerations on accessible areas of the body, especially the extremities, with notable sparing of the central back





Figs. 13.3 and 13.4 Factitial, self-induced ulcers exhibit bizarre shapes but often have a clean, healthy base, in contrast to ulcers of vascular, infectious, or neoplastic origin (Courtesy of Dr. H. K. Steinman)

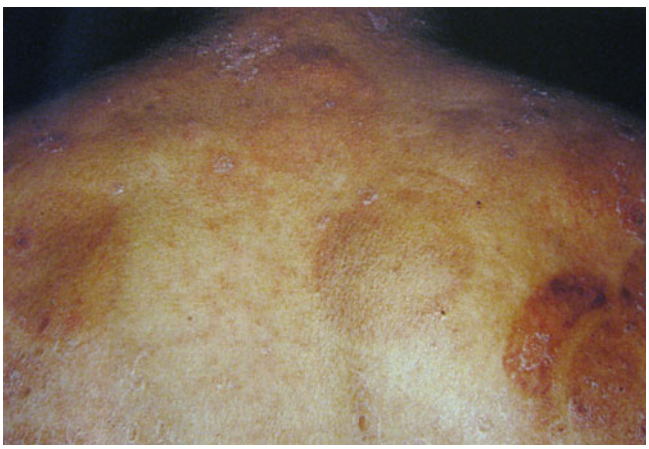


Fig. 13.5 These circular *ecchymoses* are a result of the common Asian practice of "cupping" to relieve infections and other ailments. Suction ecchymoses are produced when a *glass* cup containing a small candle is held against the skin until the candle burns out. Cultural and ethnic traditionsand practices must be considered to avoid misdiagnosing a patient *as* having a psychocutaneous *disorder* (Courtesy of Dr. H. K. Steinman)

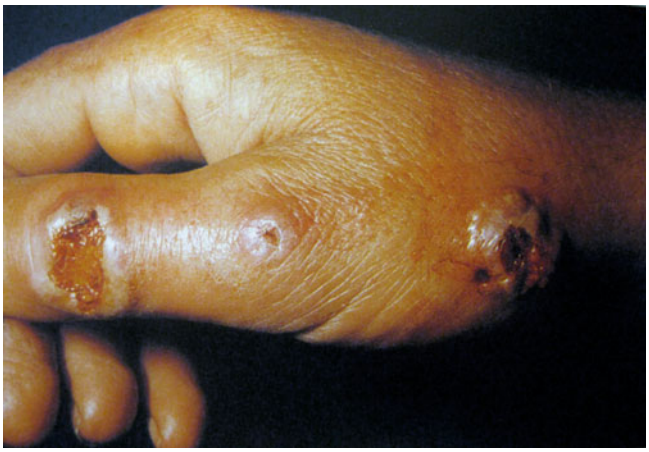
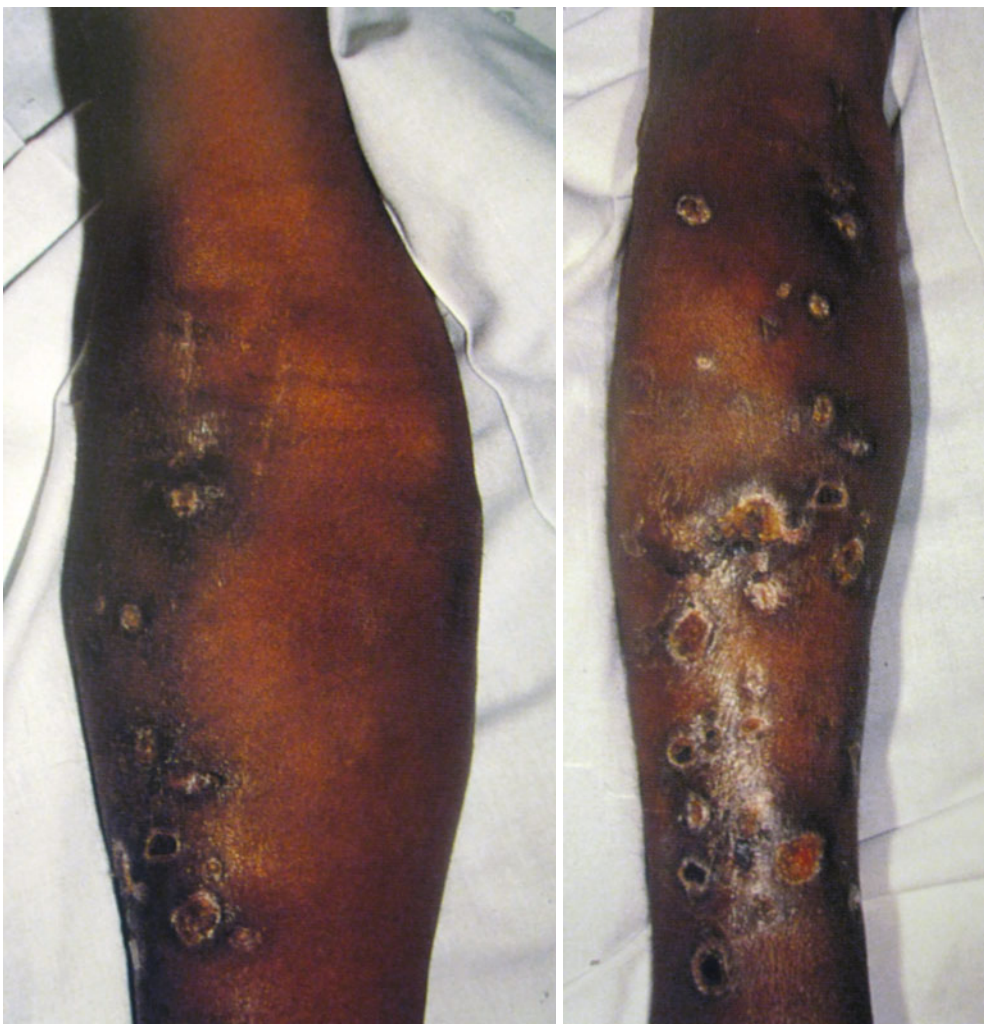
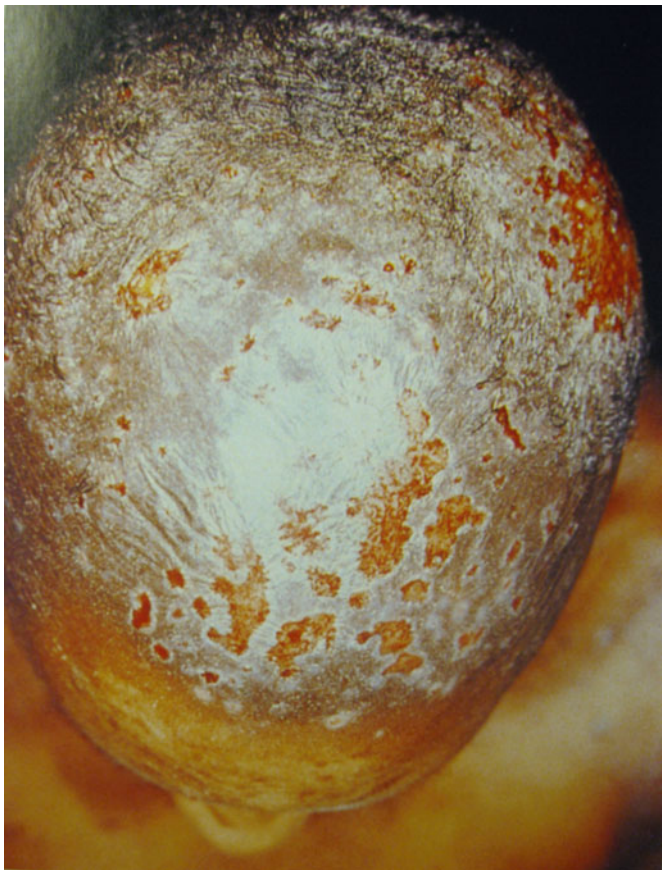
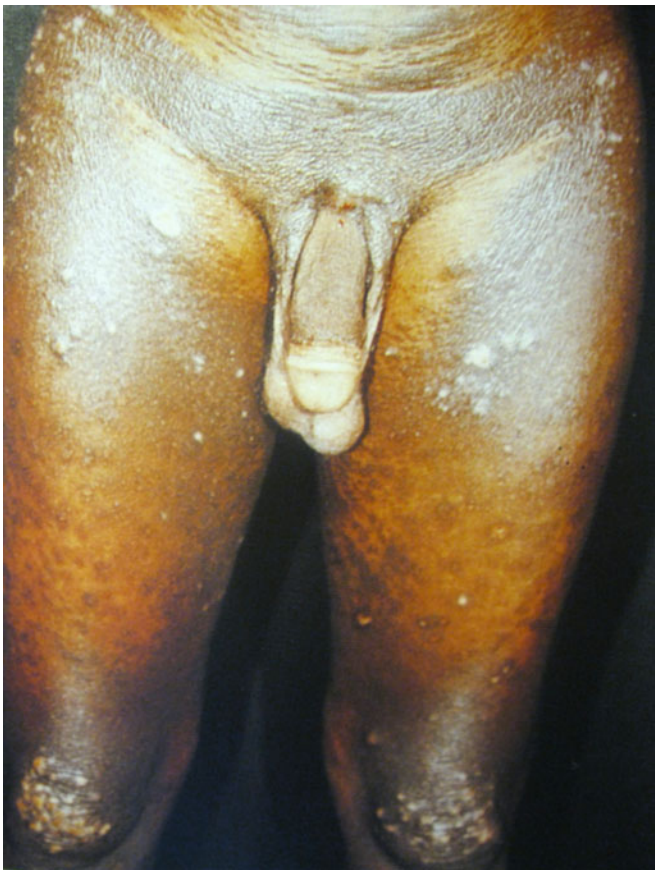


Fig. 13.6 Pyoderma gangrenosum involving the hands can be mistaken for factitial ulcerations, but the raised, fluctuant border with undermined edges and "dirty" base contrasts with the clean-based factitial ulcers seen in Figs. 13.3 and 13.4. Pyoderma gangrenosum can be seen in association with many internal diseases, especially inflammatory bowel disease, rheumatoid arthritis and, in this case, acute myelogenous leukemia



Figs. 13.7 and 13.8 Skin popping (intradermal injection by a narcotic addict). This form of self-induced trauma is also called factitial dermatitis and dermatitis artefacta





Figs. 13.9 and 13.10 "Neurodermatitis" shows lichenification (thickening of skin with accentuated markings) and erosions from chronic scratching

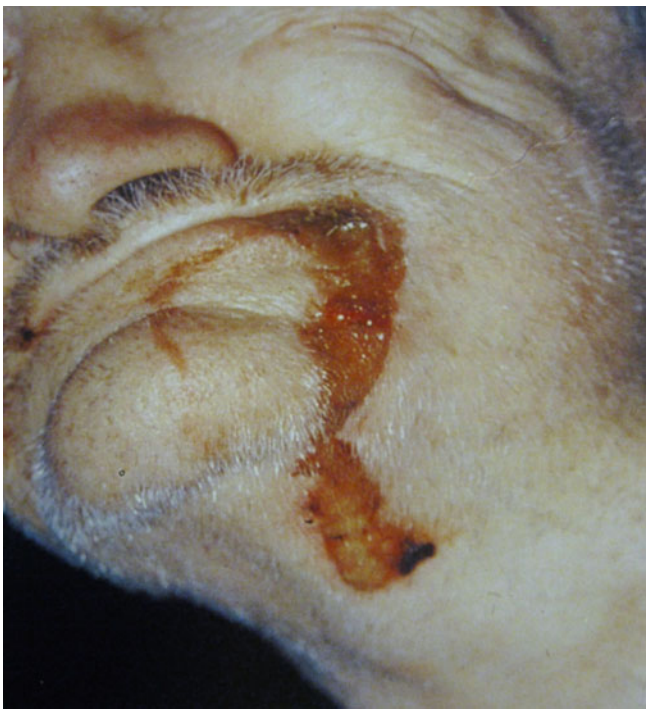


Fig. 13.11 "Slobber dermatitis" from chronic drooling mimics factitial dermatitis. Drugs, poorly fitting dentures, mandibular recession, and lax skin predispose to drooling and secondary infection with Candida albicans (perleche, angular stomatitis)

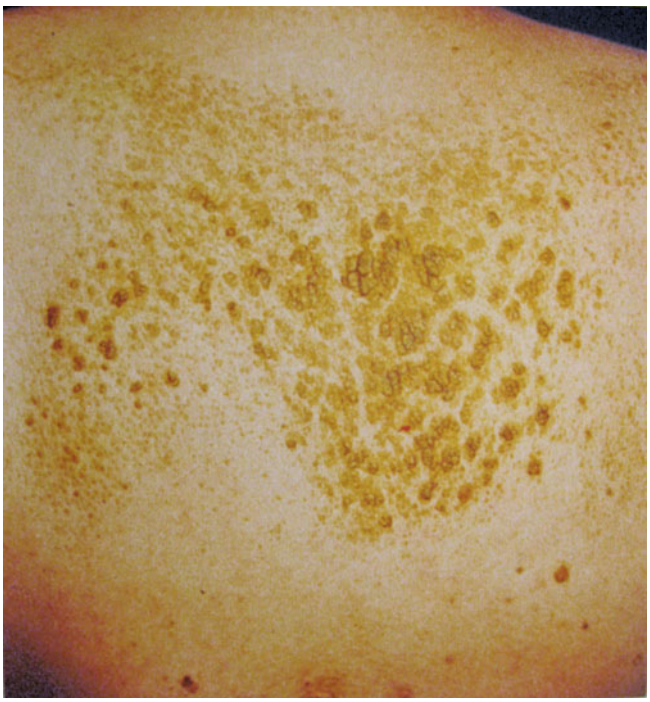


Fig. 13.12 Brown, crusty eruption on the midback resembles localized seborrheic keratoses, but these spots washed off with soap and water. This arthritic patient suffered from localized underbathing

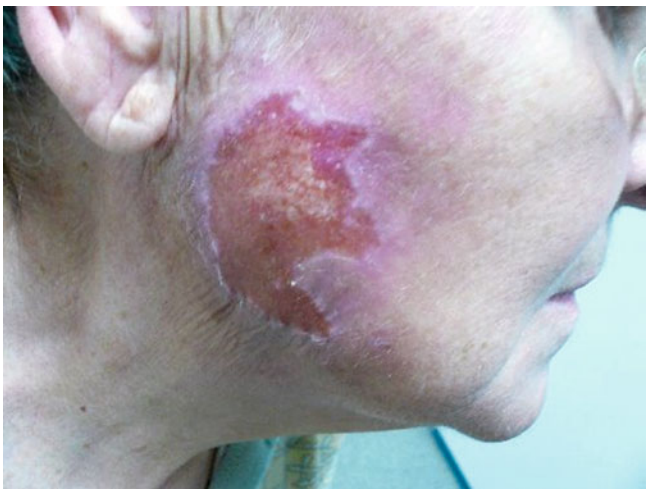


Fig. 13.13 Large excoriation on the right cheek

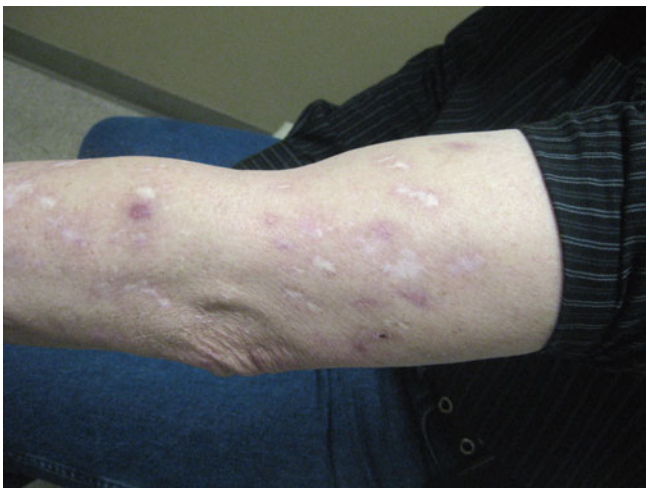


Fig. 13.14 Post-inflammatory hypopigmentation on arm of patient following self-excoriation

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Rosacea

14

Synonyms

Rosacea is also known as acne rosacea.

Clinical Description

The clinical presentation of rosacea typically includes red papules, papulopustules, and telangiectases affecting the central face. The National Rosacea Society has described a classification system based on four main subtypes: erythematotelangiectatic (vascular), papulopustular (inflammatory), phymatous, and ocular. Four variants (granulomatous, pyoderma faciale, perioral dermatitis, and steroid rosacea) have also been classified. Rosacea generally occurs when patients are at least in their 30s, but can develop in younger adults [1–3].

Erythematotelangiectatic (Vascular) Rosacea

The early vascular form is typically seen or described by patients as a recurrent blush. If it progresses, the blush may become longer lasting and telangiesctasias may form. Large spider angiomatas and edema may also develop in some individuals. A variant with a persistent woody induration has also been classified.

Inflammatory Rosacea

Inflammation in rosacea is similar to that of acne. Clinical aspects include small papules and pustules and can advance to deeper, more persistent granulomatous nodules.

Sebaceous Hyperplasia

Rosacea patients, especially males over 30, can experience an overgrowth of sebaceous glands. Rhinophyma, a hyperplasia of the nasal sebaceous glands, is linked to the rosacea. Nasal skin becomes slightly swollen and smoother, with pores and keratinous debris becoming more apparent.

Ocular Rosacea

Over half the patients diagnosed with rosacea have ocular manifestation confined to the eyelids and ocular surface with patients experiencing minor irritation, dryness, and blurry vision. Severe ocular rosacea can cause eye damage, lid margin telangiectasia, marginal corneal ulcers and inflammatory keratitis. Blepharitis and conjunctivitis are also common findings in these patients [4–7].

Perioral Dermatitis

Perioral dermatitis and periocular dermatitis are often present in patients with vascular rosacea. It is histologically similar to rosacea and responds to similar medications.

Pyoderma Faciale

Clinically described as eruptions of inflamed papules and yellow pustules in the central facial region. Pyoderma faciale is also histologically similar to rosacea and responds to similar medications.

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Steroid Rosacea

Caused by long-term use of corticosteroids. Patients initially show a sign of improvement; however, prolonged use can cause atrophy, vasodilation, and inflammatory papules.

Etiology and Pathophysiology

The exact etiology of rosacea is unknown, but the pathologic process has been described. Inflammation plays an important role in lesion formation. Inflammatory cells release proinflammatory cytokines and degradative enzymes that induce angiogenesis and damage dermal constituents. Facial flushing and vasodilation are more common in patients with rosacea. Alcohol, hot/spicy foods and drinks, fluorinated steroids and toothpaste, and *Demodex folliculorum,* a mite that lives in the lumen of the sebaceous follicles of the head, have been associated with rosacea. Cathelicidin and serine protease activity cause similar inflammation when injected into the skin. Rosacea has also been associated with stomach ulcers caused by the bacterium Helicobacter pylori [8–10].

Histopathology

Mild forms of rosacea are subtle and limited to vascular ectasia and mild edema. Moderate forms develop a perivascular and perifollicular lymphohistiocytic infiltrate and elastolysis. Severely rosacea shows noncaseating epithelioid granulomas and sinus tract formation. Follicular tuberculoid and sarcoidal granulomas are present in papular rosacea.

Differential Diagnosis

1. Ocular rosacea is often misdiagnosed as seborrheic dermatitis, as it commonly occurs in patients with rosacea.
2. A fixed blush on the lateral cheeks with fine follicular keratotic plugs characterizes keratosis pilaris.
3. Growth Factor Receptor Inhibitor "Acne" can resemble severe acne or rosacea and occurs during chemotherapy.
4. Lupus Erythematosus can be hard to differentiate from rosacea due to possible coexistence. The butterfly rash of rosacea can resemble lupus. Pustules and papules or blepharitis favors a diagnosis of rosacea. Fine scaling, pigmentary changes, follicular plugging, scarring, and tenderness favor lupus.

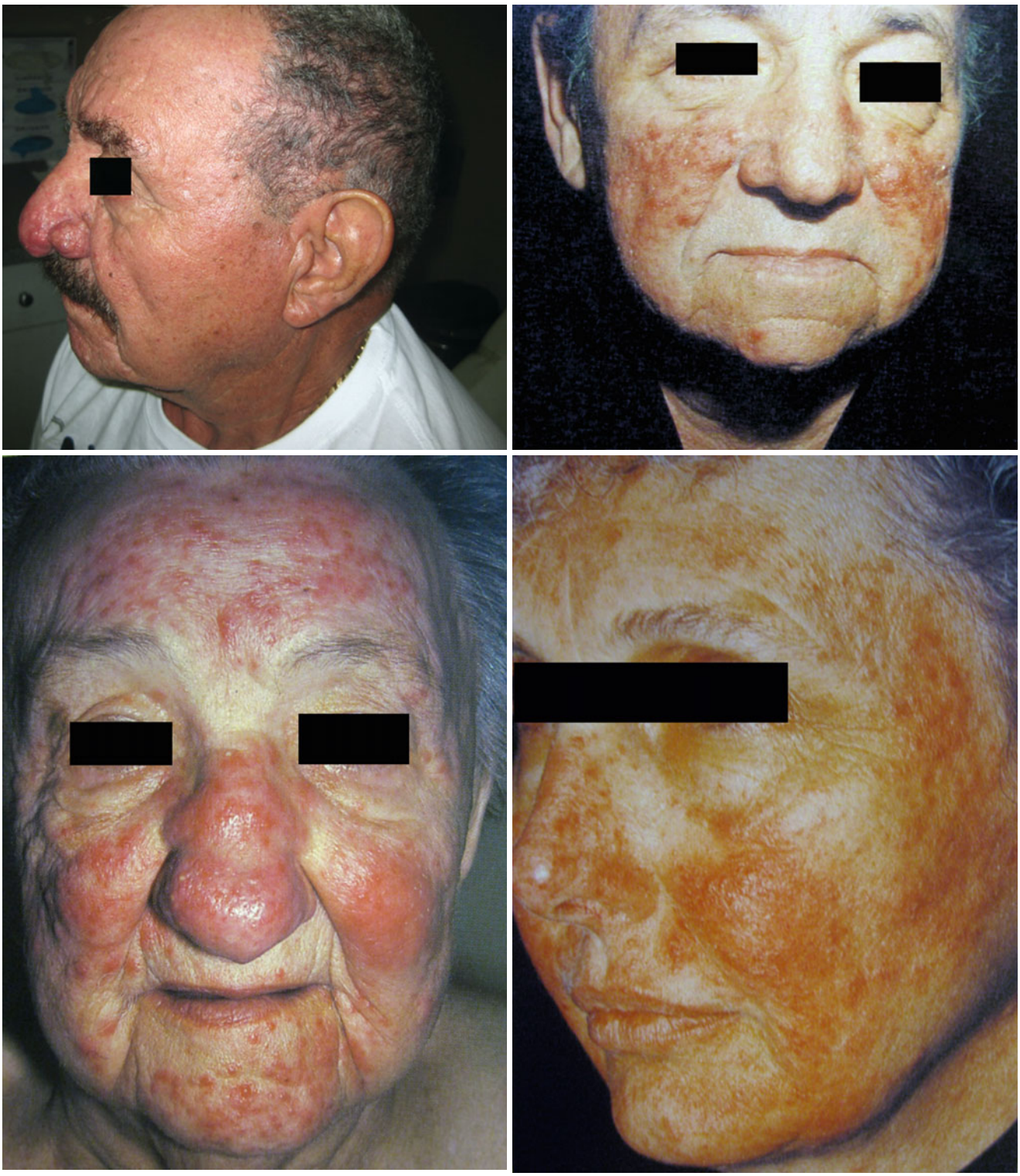
Therapy

Effective treatment of rosacea includes avoidance of triggers, topical and oral antibiotic therapy, both topical and oral retinoid therapy, topical vitamin C therapy, and cosmetic surgery. The subtype should be properly diagnosed before therapy. Therapeutic choices will depend on patient expectations, tolerance, previous therapies used, rosacea subtype, and severity.

1. Topical pharmacotherapeutic options include: azelaic acid, dapsone gel, clindamycin, clindamycin 1 %-benzoyl peroxide 5 % gel, erythromycin, metronidazole, or sodium sulfacetamide 10 % + sulfur 5 %.
2. For patients with moderate-to-severe papulopustular rosacea or those with ocular involvement, systemic therapy is often prescribed and options include doxycycline, erythromycin, metronidazole, minocycline, tetracycline, or in severe cases, low dose isotretinoin.
3. The telangiectatic component does not respond to either oral or topical therapy, and is best treated with laser and light-based therapies.
4. Surgical intervention may be required for the phymatous subtype [11–13].

Prognosis

The prognosis is generally good.



Figs. 14.1, 14.2, 14.3, and 14.4 Advanced rosacea shows central facial erythema, papulopustular eruption, and rhinophyma (enlarged bullous nose)

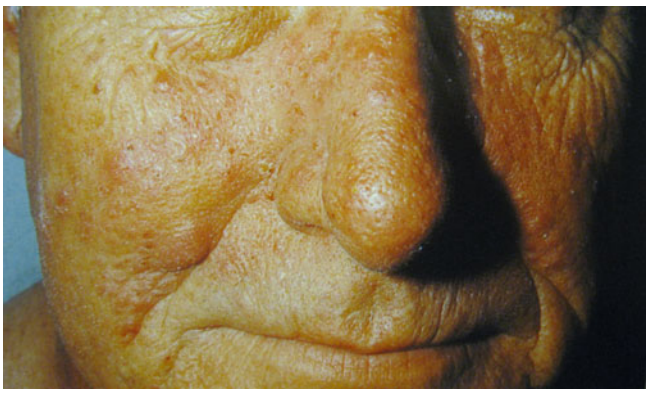


Fig. 14.5 Close-up of papulopustular rosacea, common in patients of Celtic ancestry

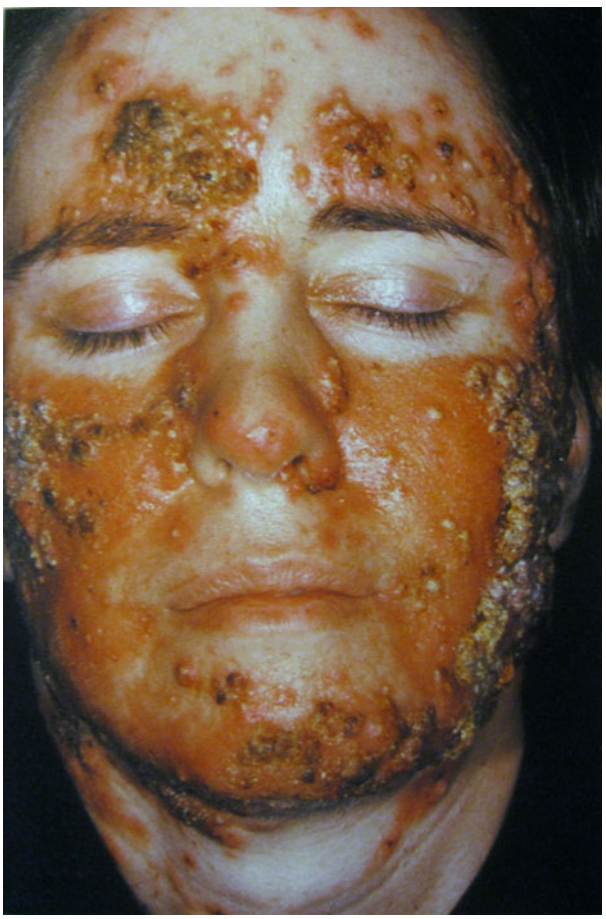


Fig. 14.6 Unusually severe necrotic, crusted rosacea responds well to treatment with isotretinoin. (Accutane)

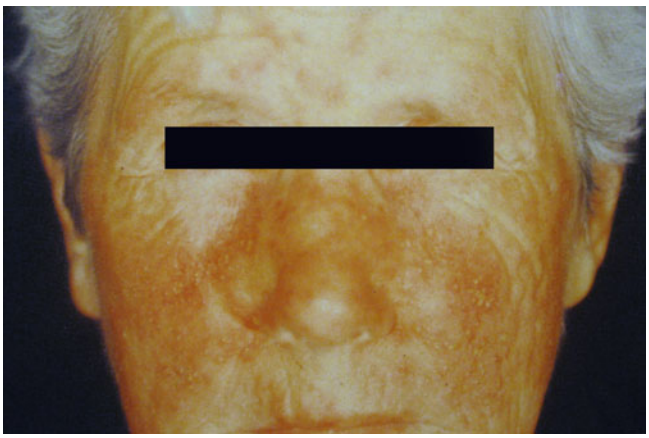


Fig. 14.7 The butterfly rash of rosacea can resemble those of systemic lupus erythematosus and polymorphous light eruption (see text)

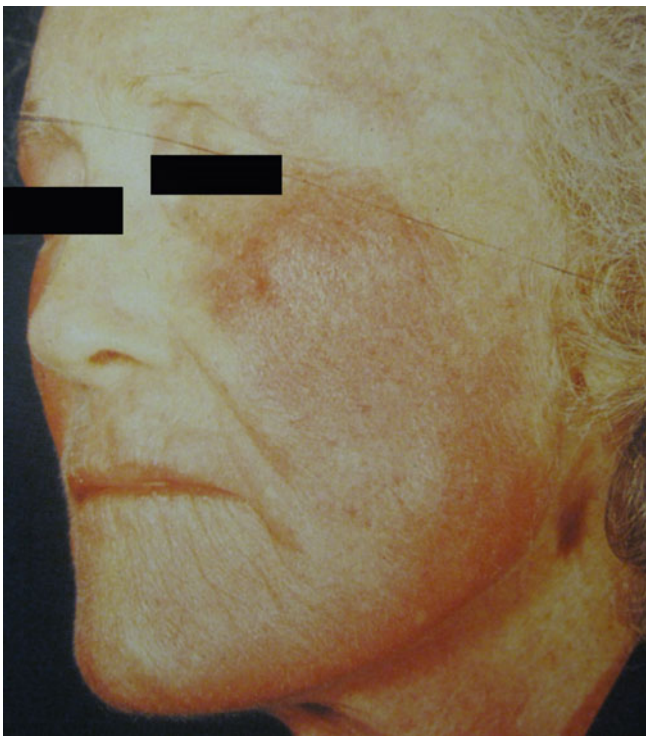


Fig. 14.8 Steroid rosacea is an iatrogenic complication of improperly prescribed and overused potent topical steroids. The telangiectasia and atrophy are often permanent

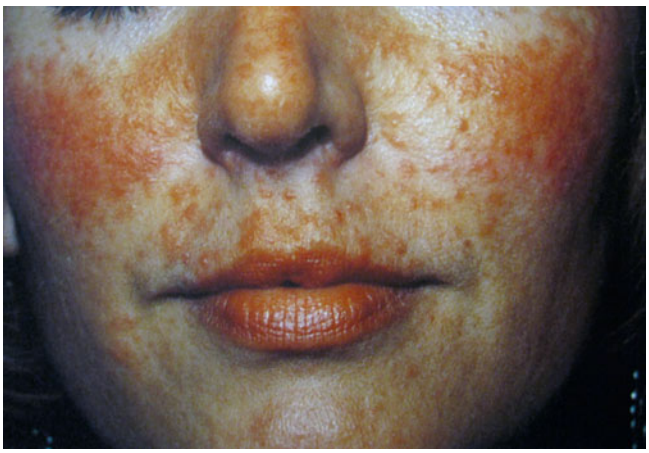
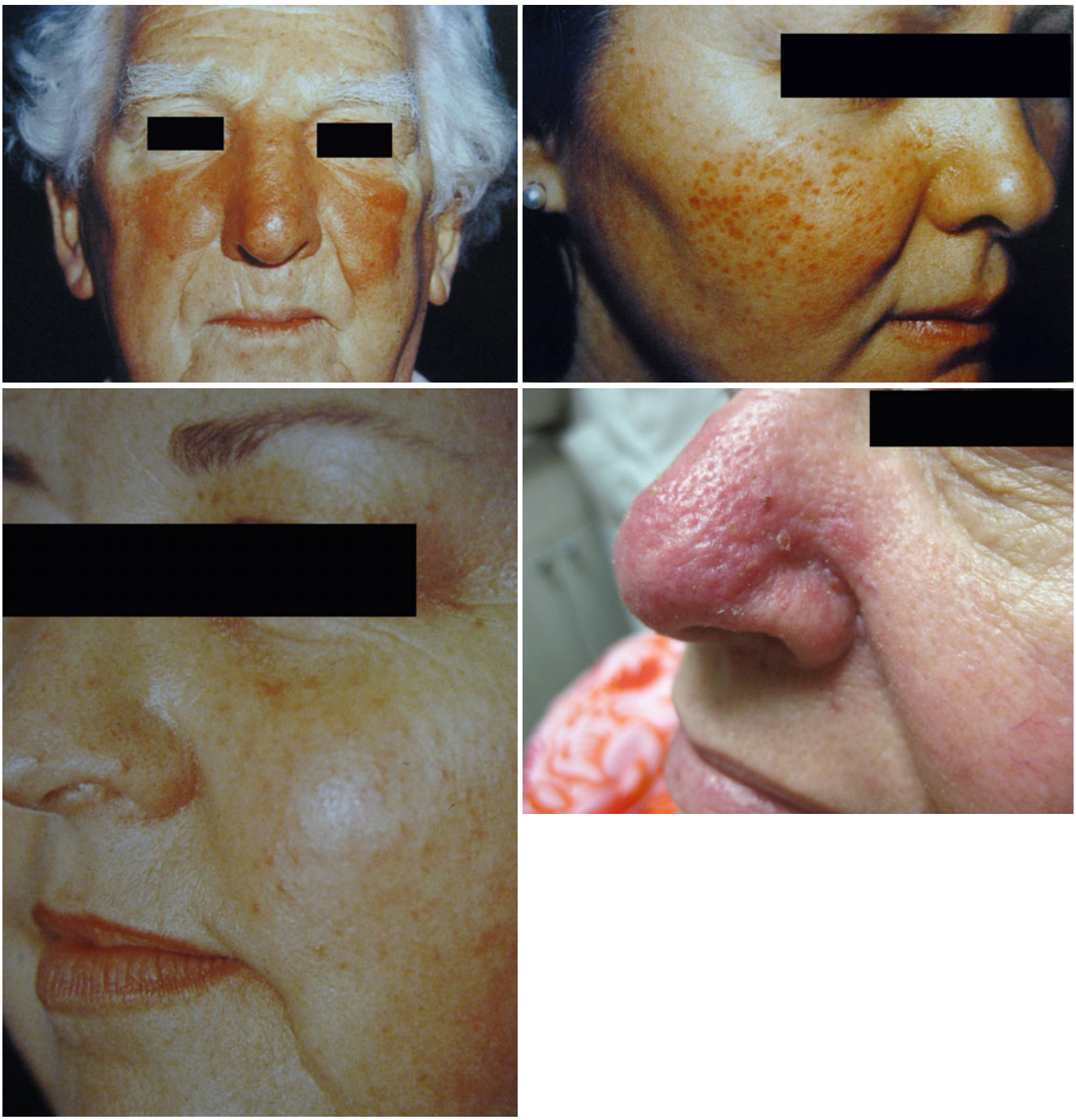


Fig. 14.9 Polymorphous light eruption (PMLE) is a photosensitivity disorder that clinically resembles rosacea and systemic lupus erythematosus (see text)



Figs. 14.10, 14.11, 14.12, and 14.13 Papular rosacea of the cheeks and nose

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Rhinophyma

15

Clinical Description

Rhinophyma is the most common manifestation of phymatous rosacea (see Chap. 14). Also called "whiskey nose" and "W.C. Fields syndrome", rhinophyma manifests as a large, bulbous, greasy nose with widely dilated, patulous sebaceous follicles. It occurs predominantly in men. It progresses from the nasal tip to the ala to the proximal nose. Rarely, rhinophyma affects the earlobes and chin also.

Etiology and Pathophysiology

Various causes have been postulated, including chronic flushing from many stimuli (e.g., excessive sun and heat, infestation with Demodex folliculorum mite), resulting in massive hyperplasia of the sebaceous follicles in the hyperplastic glandular form of rosacea. It is not necessarily caused by alcoholism; patients often request treatment to avoid this stigma.

Histopathology

Histopathologic investigation of rhinophyma reveals massive hyperplasia and hypertrophy of the sebaceous glands in a fibrotic connective tissue stroma.

Therapy

1. Surgery may be done in the form of hot wire loop (thermal cautery) "sculpting." CO_2 laser vaporization, or scalpel excision (decortication) [1, 2].
2. Isotretinoin (Accutane) may be prescribed for early cases, before fibrosis has occurred.

Prognosis

This condition is persistent if not treated, but rhinophyma is primarily of cosmetic significance [3–5].

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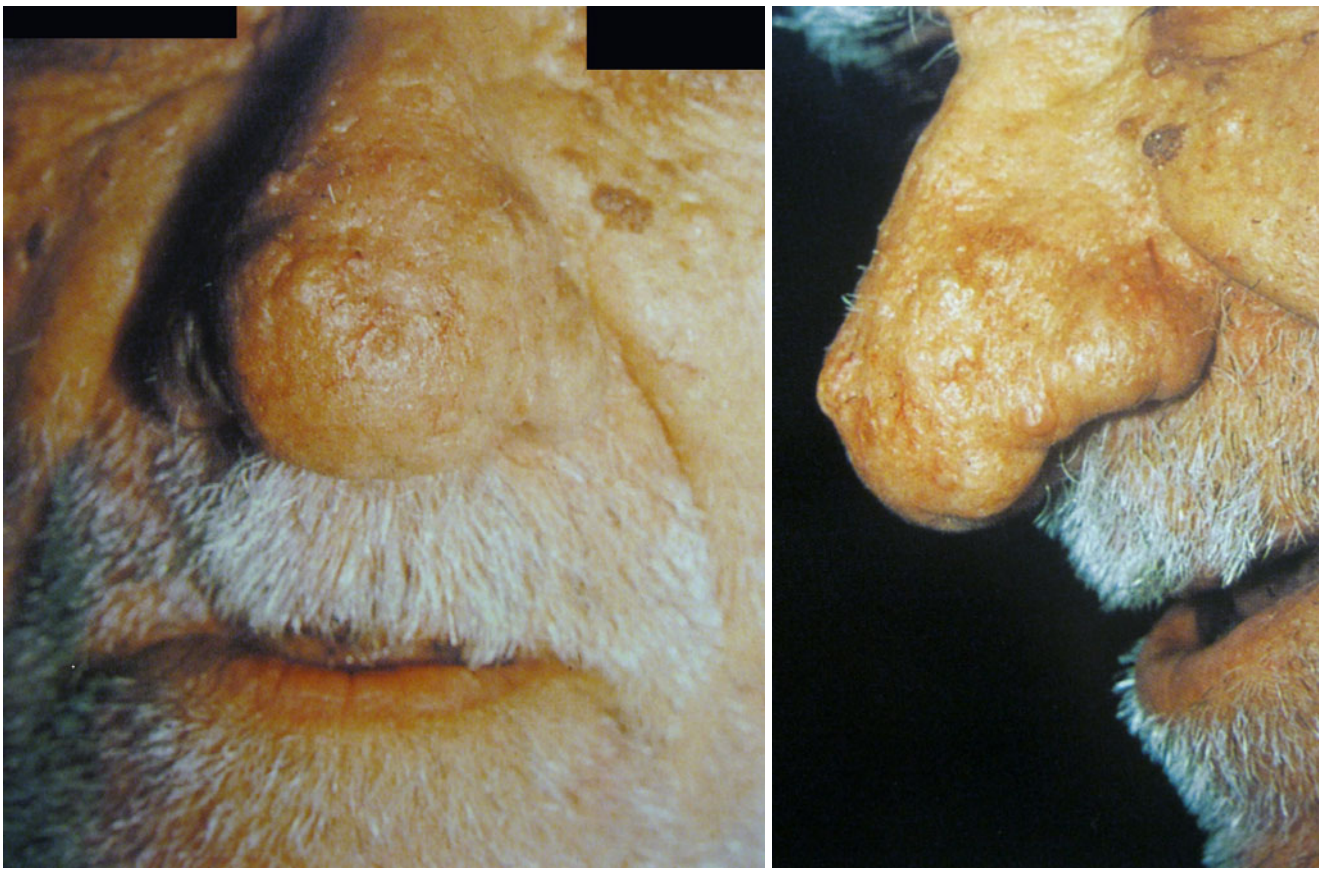

Figs. 15.1 and 15.2 Advanced rhinophyma showing a distorted, bulbous, lumpy nose studded with greasy, yellow papules. Histologic studies reveal massive sebaceous hyperplasia

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Seborrheic Dermatitis

16

Clinical Description

Seborrheic dermatitis is a common chronic inflammatory skin condition. It is characterized by the development of erythematous patches and scaling. Seborrheic dermatitis oleosa has red-yellow, greasy scale. Seborrheic dermatitis sicca has fine, white, dry scale. Both types most often appear on the face, scalp, upper chest, and back. It can also manifest in the gluteal cleft, umbilicus, groin, and genitals. Dandruff is a milder variant and treatment depends on severity of symptoms. The onset of these symptoms is gradual, but may be rapid in HIV-positive patients with a CD4 T-cell count of below 40 cells/mm [1–3].

Etiology and Pathophysiology

Seborrheic dermatitis affects approximately 11.6 % of the general population. It can manifest in persons of any race and is slightly worse in males. Patients with neuroleptic-induced Parkinsonism, familial amyloidosis, and trisomy 21 have shown an increased incidence of seborrheic dermatitis [4]. The cause of seborrheic dermatitis is not fully understood. It has been linked to skin colonization with yeasts of the genus *Malassezia.* It is hypothesized that the fungal metabolites react with the inflammatory free fatty acids released from sebaceous glands [5]. There is no genetic predisposition to the disease. Humidity and season, trauma, and emotional stress have been believed to worsen the condition.

Histopathology

Histopathologic findings of seborrheic dermatitis are broad. Characteristics include hyperkeratosis, acanthosis, focal spongiosis, and parakeratosis. Psoriasis is distinguished by thinned rete ridges and absence of spongiosis.

Differential Diagnosis

Other diseases associated with seborrheic dermatitis include epilepsy, obesity, alcoholism, Leiner's disease, zinc deficiency, and tinea. Diagnosis is generally clinical. A biopsy may be needed in persons with an atypical presentation. Fungal culture will rule out tinea in younger patients. Seborrheic dermatitis may present similar symptoms as tinea capitis, erythrasma, psoriasis, atopic dermatitis, contact dermatitis, rosacea, vitamin B deficiency, zinc deficiency, or drug eruption.

Therapy

Therapy for seborrheic dermatitis is aimed at the inhibition of skin yeast colonization, reduction of pruritus and erythema, eliminating crusts and scales, and inflammation reduction. Topical therapies are the most common type of therapy because the condition is recurrent.

1. Antifungal agents are the mainstay of antiseborrheic therapy. *Azoles* work by inhibiting ergosterol, an important component of the fungal cell wall. Many also have anti-inflammatory properties. Ketoconazole is available over-the-counter in the forms of foams, gels, and creams or can be prescribed as a 200-mg/day regimen for 4 weeks. It may also be effective in combination with zinc and selenium. Itraconazole has an affinity for the skin, hair, and nails. The suggested regimen for oral itraconazole is 200 mg/day for 7 days. Bifonazole ointment has also been used effectively. Other agents include terbinafine (*allylamines*) and butenatfine (*benzylamines*) and Ciclopirox 1–1.5 % shampoo used two to three times per week until clearance and then every week to every other week for prophylaxis. Adverse effects from antifungals include contact dermatitis, burning, itching, or dryness.
2. Metronidazole is an effective gel formulation when applied twice daily for 8 weeks.

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3. Non-prescription antifungal agents include Selenium (shampoos) twice daily but may rarely cause hyperpigmentation. Pyrithione zinc is an active ingredient in many over-the-counter anti-dandruff shampoos and has both fungistatic and antimicrobial activities. It is available as 1 and 2 % shampoos and as a 1 % cream.
4. Treatment with topical corticosteroids, hydrocortisone and beclomethasone dipropionate may be beneficial for those with seborrheic dermatitis secondary to immunosuppression, such as those with an HIV infection.
5. Immunomodulators such as tacrolimus and pimecrolimus inhibit calcineurin and are anti-inflammatory, but should be only used for short periods.
6. Tar has fungistatic properties and the ability to reduce sebum production but has adverse risks of toxicity and carcinoma development.
7. UVA and UVB light have a direct inhibitive effect on *Malassezia spp*. but light therapy may have adverse effects of burning, itching, and possible increased malignancy after treatment [6–11].

Prognosis

Prognosis is generally good with topical antifungal therapy sustaining relapse-free periods. Corticosteroids, phototherapy, and other topical agents should be used as combination therapy with antimycotic agents to reduce unwanted adverse effects.

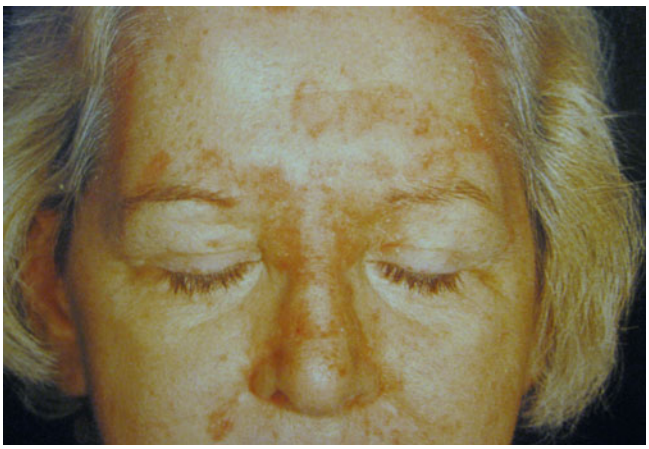


Fig. 16.1 Central facial seborrhea resembles the facial rash of lupus erythematosus

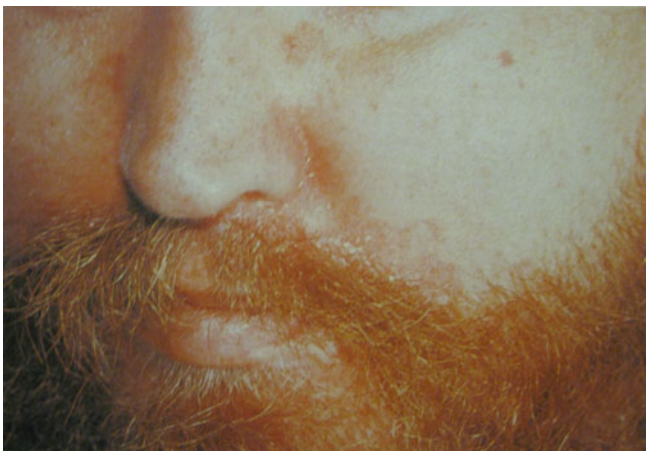


Fig. 16.3 Mustache and beard seborrhea is common

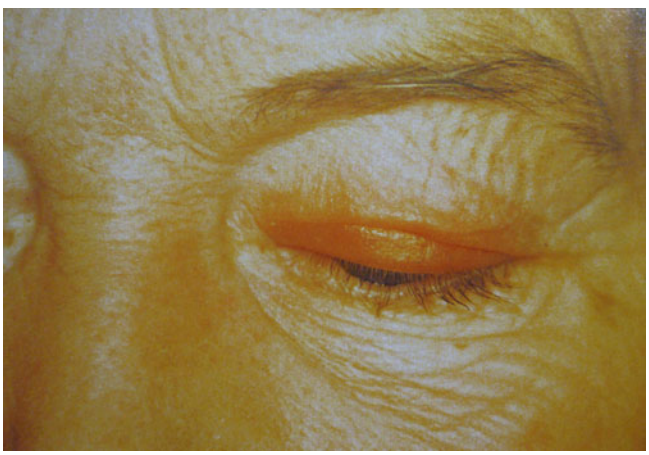


Fig. 16.2 Seborrheic dermatitis involving only one eyelid

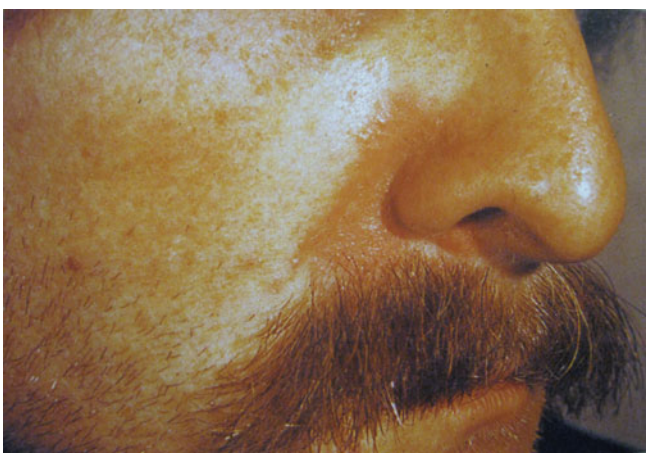
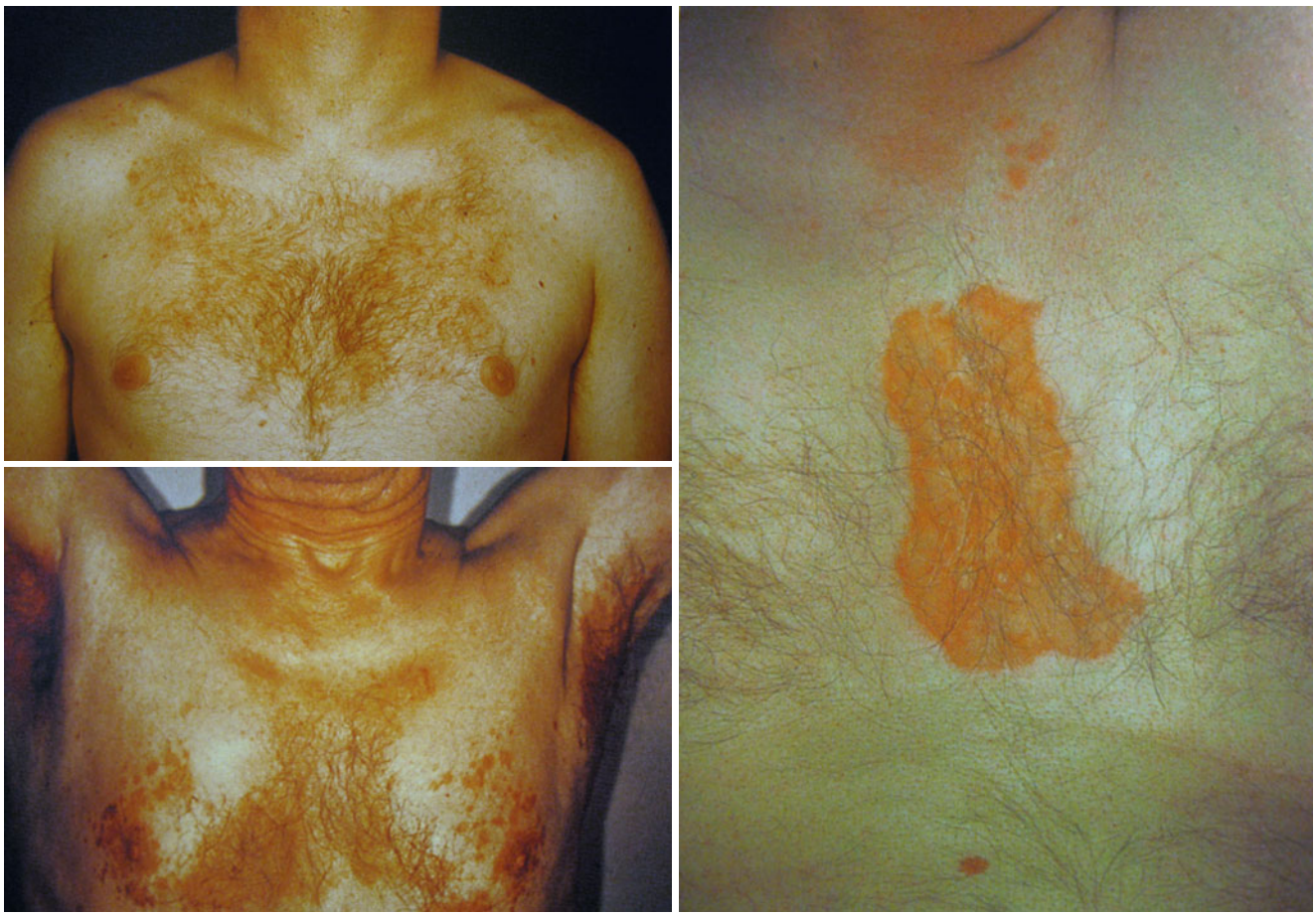


Fig. 16.4 Another example of mustache and beard seborrhea



Figs. 16.5, 16.6, and 16.7 Central chest seborrhea may be diffuse (Fig. 16.5) or localized (Fig. 16.6), with the latter resembling a patch of psoriasis or tinea (Fig. 16.7 courtesy of Westwood Pharmaceuticals, Buffalo, NY)

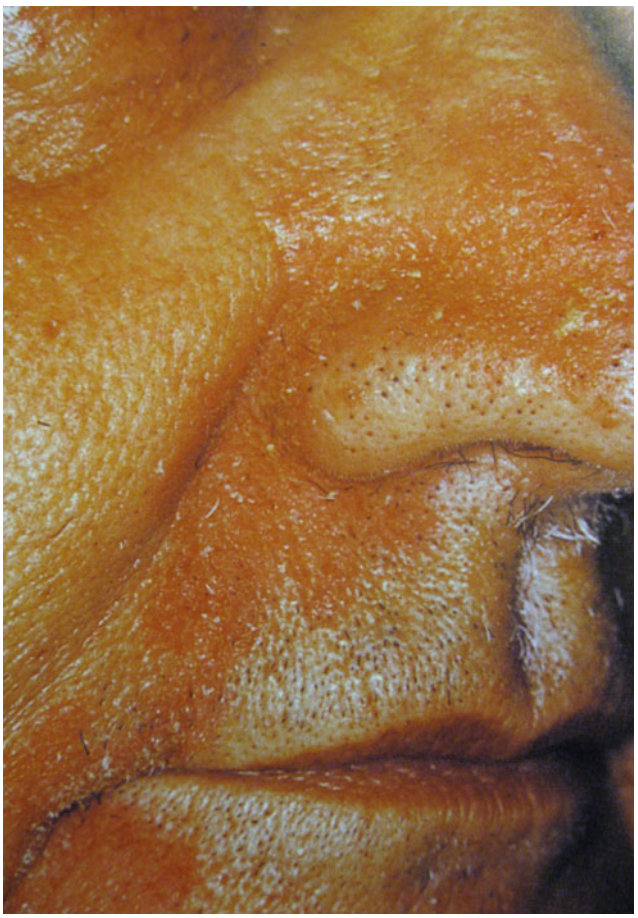


Fig. 16.8 Perinasal seborrhea

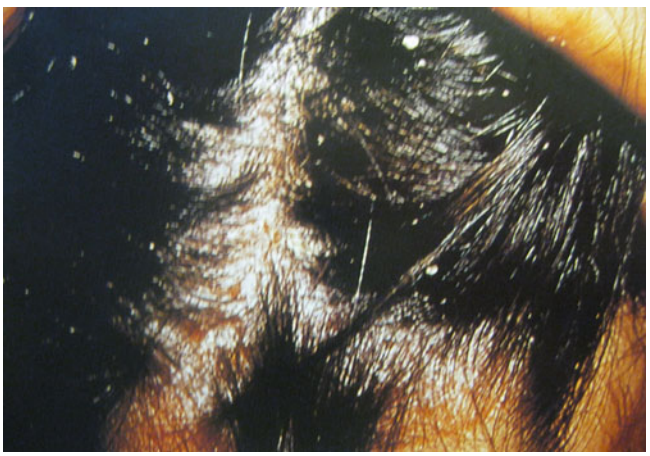


Fig. 16.9 Scalp seborrhea: greasy, yellowish scale with some resemblance to psoriasis (sebopsoriasis) (Courtesy of Westwood Pharmaceuticals, Buffalo, NY)

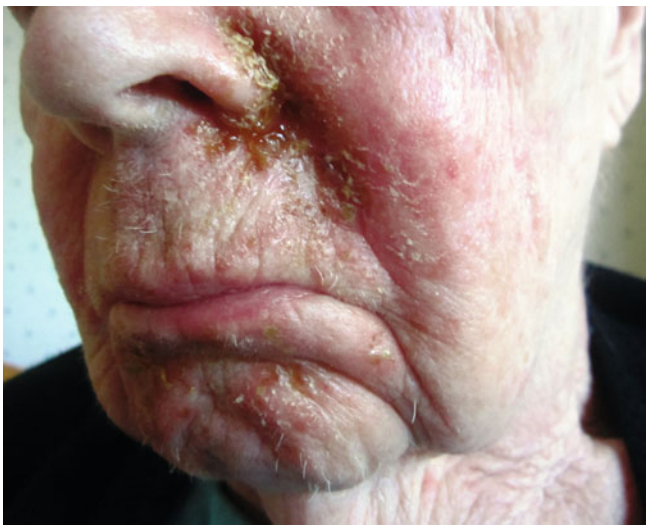


Fig. 16.10 Impetigo on face in patient with seborrhea

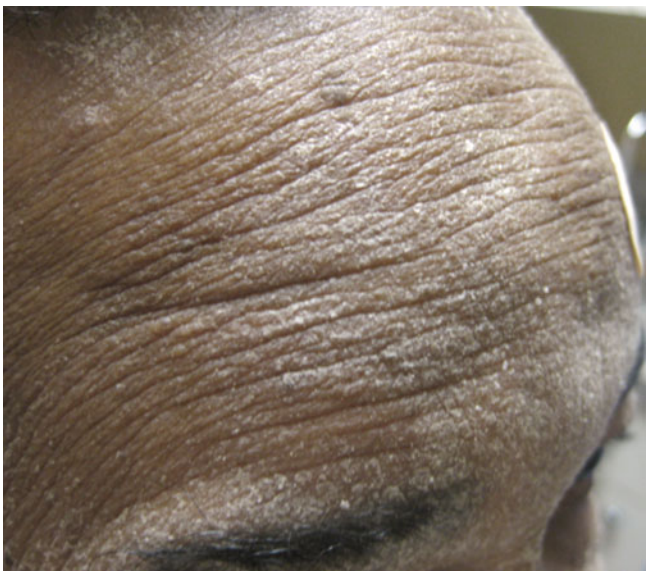


Fig. 16.11 Seborrhea of the forehead

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Solar Elastotic Syndromes

17

Definition

Solar elastotic syndromes include various clinically diverse conditions caused by photodamage. Histologically, they all show solar elastosis.

Etiology and Pathophysiology

Solar elastotic material is presumed to originate from elastin as a product of "sick" or sun- damaged fibroblasts (see Chapter I, sections on biology and aging skin). It reacts with antielastin antibodies, is susceptible to elastase, and resists digestion by collagenase. It lacks normal physiologic elasticity because of inadequate cross-linkage of elastic fibers by desmosine. Clinically, affected skin is highly susceptible to tearing [1, 2].

Clinical Conditions and Differential Diagnosis

1. Favre-Racouchot syndrome, also called nodular elastosis with cysts and comedones, is most prominent on the malar eminences of the cheeks. Comedonal acne occurs preferentially on the sebaceous areas.
2. Cutis rhomboidalis nuchae appear as deep, lozenge-shaped wrinkles on the posterior neck (often called farmer's or sailor's neck).
3. Elastomas of the ear, nose, chest appear as 1–4 mm yellow-white dermal papules that clinically resemble sebaceous hyperplasias.
4. Solar elastotic bands of the forearms clinically resemble scars from trauma (stellate pseudoscars).
5. Diffuse elastoses of the lips and cheeks are yellow papules and patches.
6. Actinic cheilitis histologically shows diffuse actinic keratosis of the lips, particularly the more sun-exposed lower lip. Progression to squamous cell carcinoma is possible. Because squamous cell carcinoma of the mucous membrane has a higher rate of metastasis than does squamous cell carcinoma on sun-exposed skin (30 % versus 1–2 %), early, complete excision of squamous cell carcinoma is mandatory.
7. Actinic granuloma is also called annular elastolytic giant cell granuloma, Miescher's granuloma of the face, and O'Brien's disease. The clinical differential diagnosis includes granuloma annulare (more common on the extremities) and necrobiosis lipoidica (diabeticorum). Some pathologists have suggested that actinic granuloma merely represents granuloma annulare or necrobiosis lipoidica on sun-damaged skin.
8. Acrokeratoelastoidosis marginalis, also called degenerative collagenous plaques of the hand and digital papular calcific elastosis, is common in truck drivers and others who drive for long hours with the thumb and index finger exposed to sunlight. It may also involve the sides of the feet.
9. Colloid milia occur as papules on the dorsal hand and fingers. Flat warts (verruca plana) are also occasionally exacerbated by sun exposure and exhibit the Koebner phenomenon (reproduction of additional lesions by trauma and/or autoinoculation by scratching).
10. Poikiloderma of Civatte is a poikiloderma (triad of hyperpigmentation and hypopigmentation, atrophy, and telangiectasia) of the lateral neck, especially in women with fair, thin skin. The differential diagnosis includes phytophotodermatitis from citrus oils (bergamot oil, psoralens) in perfume and cologne.
11. Ectropion (eversion and sagging of the lower eyelids) and ptosis (drooping of the upper eyelids) are ocular complications of severely sun-damaged skin that require ophthalmic plastic surgery. Untreated ectropion can result in purulent conjunctivitis and excessive tear retention; untreated ptosis can obscure vision with a window shade-like flap of lax skin.

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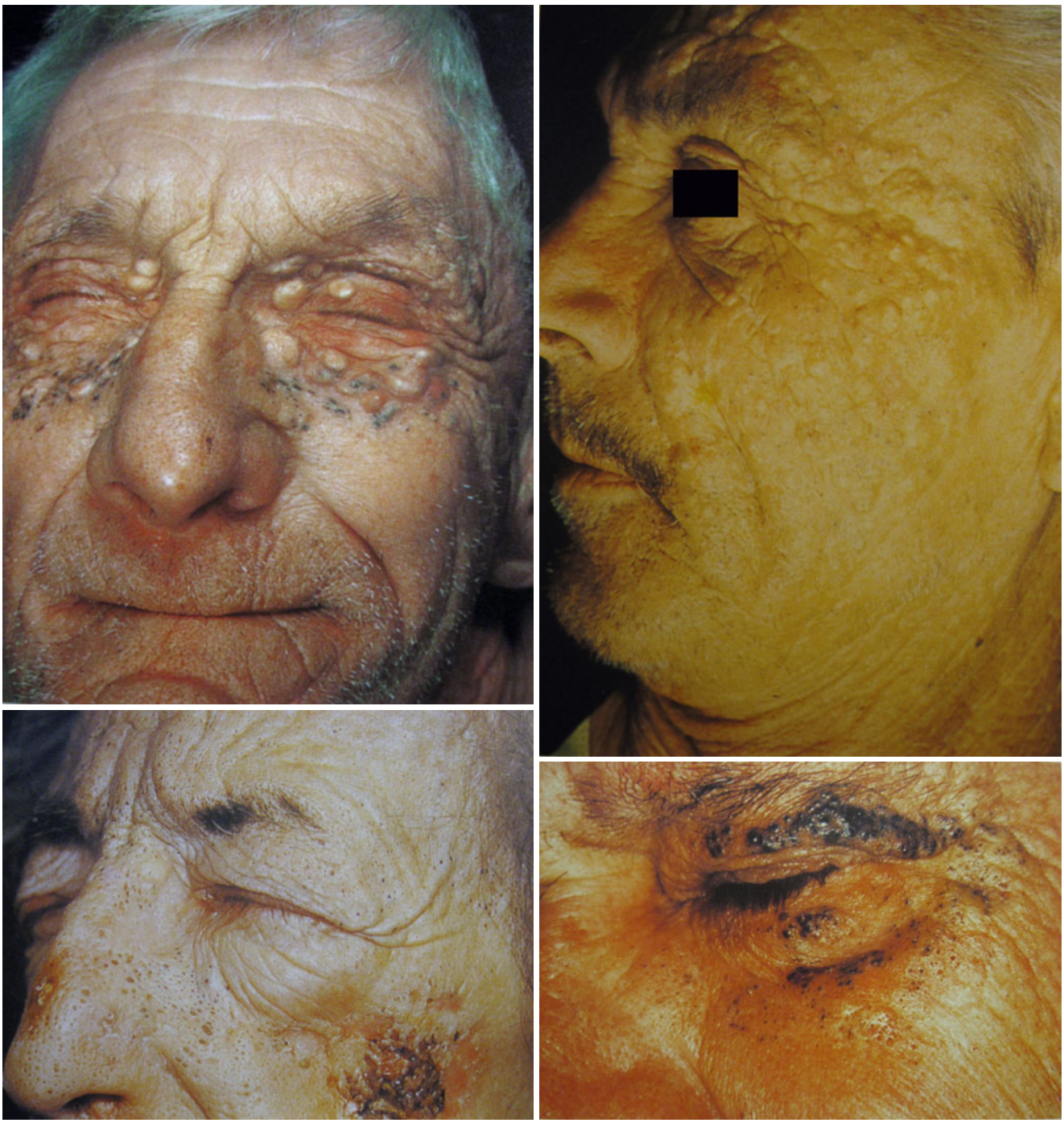
Therapy

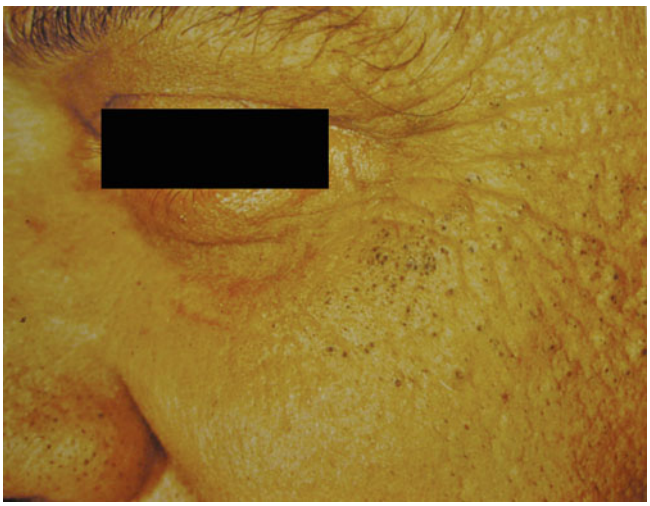
1. Further complications may be prevented by the appropriate use of sunscreens, sun avoidance, and wearing long-sleeved garments, wide-brimmed hats, and ultraviolet-blocking sunglasses [3].
2. Surgical repair of ectropion and ptosis may be carried out by an ophthalmic plastic surgeon.
3. The patient may use opaque sunscreens and/or cosmetics to camouflage poikiloderma of Civatte. Bleaching creams are of some value.
4. 5 - Fluorouracil topical chemotherapy, imiquimod, superficial surgical or chemical lip peel, or mucosal advancement for actinic cheilitis may be used.
5. Recalcitrant cysts and comedones in Favre-Racouchot syndrome may be incised and drained. Topical tretinoin can aid in chemoprevention [4, 5].
6. Cosmetically bothersome lesions can be removed by shave biopsy. Occasionally, cryosurgery can be used for solar elastomas, colloid milia, and acrokeratoelastoidosis. Lesions are usually multiple, making complete removal impractical.
7. Chemical peel with superficial trichloroacetic acid or deeper Baker's phenol may ameliorate diffuse elastosis.
8. Actinic granuloma can be surgically excised, if cosmetically practical.
9. No satisfactory treatment exists for cutis rhomboidalis nuchae or for large solar elastotic bands of the forearms.
10. Blue light, red light, and laser therapy have been used with various degrees of satisfaction for many patients with solar elastotic syndromes [6–8].

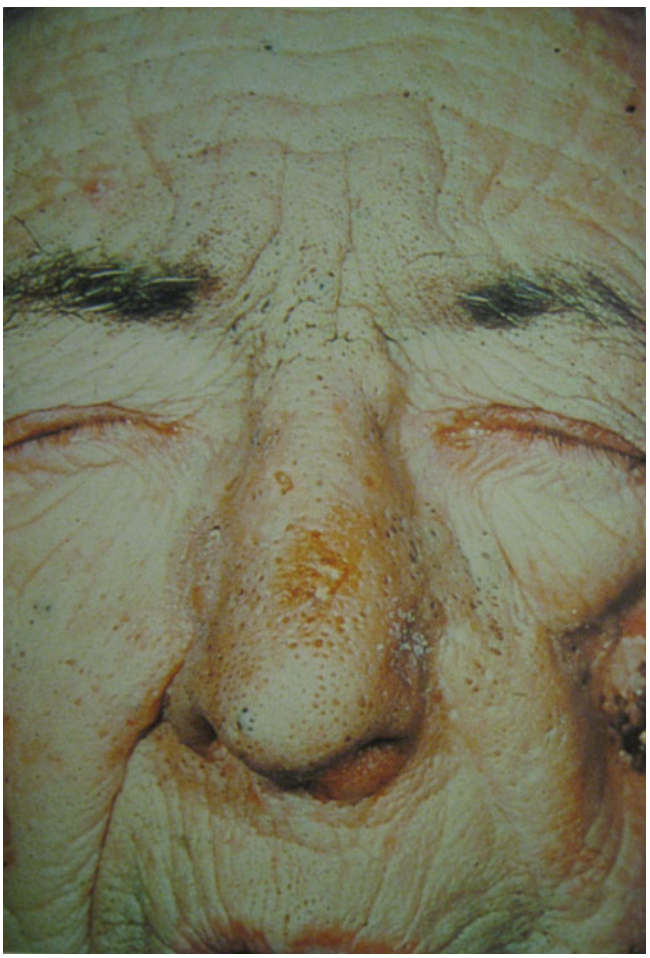
Prognosis

Progression to squamous cell carcinoma, with potential for metastasis, is the major risk of actinic cheilitis. The other conditions do not undergo malignant degeneration.

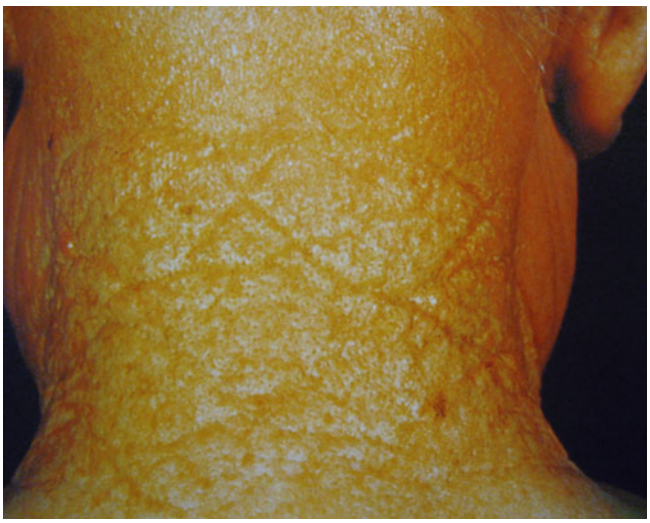


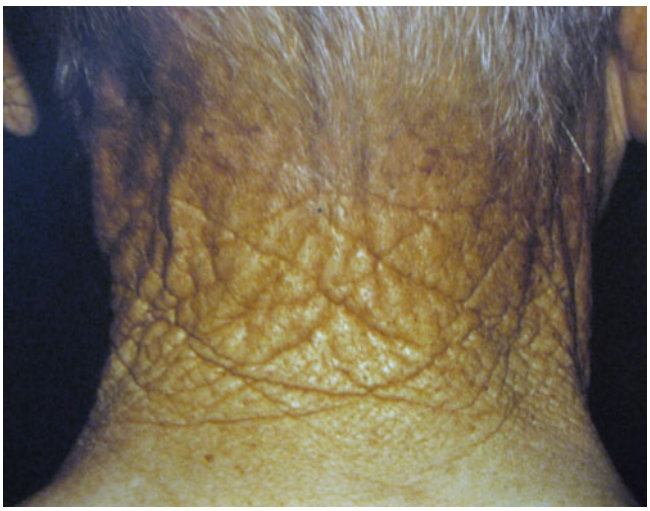
Figs. 17.1, 17.2, 17.3, and 17.4 Favre-Racouchot syndrome consists of open comedones (blackheads) and cysts on sun-exposed areas, especially the malar cheeks and periorbital skin



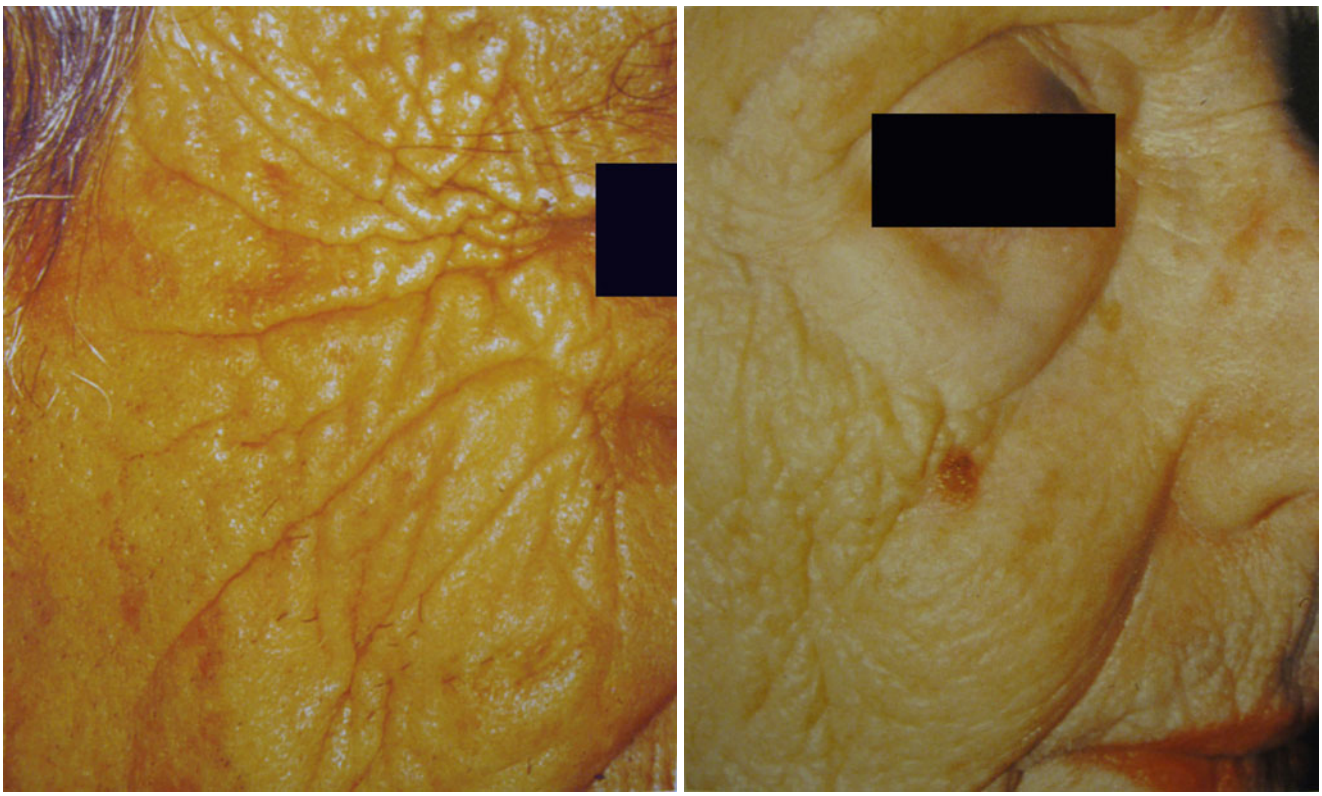


Figs. 17.5 and 17.6





Figs. 17.7 and 17.8 Cutis rhomboidalis nuchae. Note the rhomdoidal or lozenge-shaped furrows on the posterior neck. These are common in farmers, sailors, golfers, pool cleaners, and others who work outdoors



Figs. 17.9 and 17.10 Diffuse solar elastosis of the cheeks exhibits a sallow yellow color

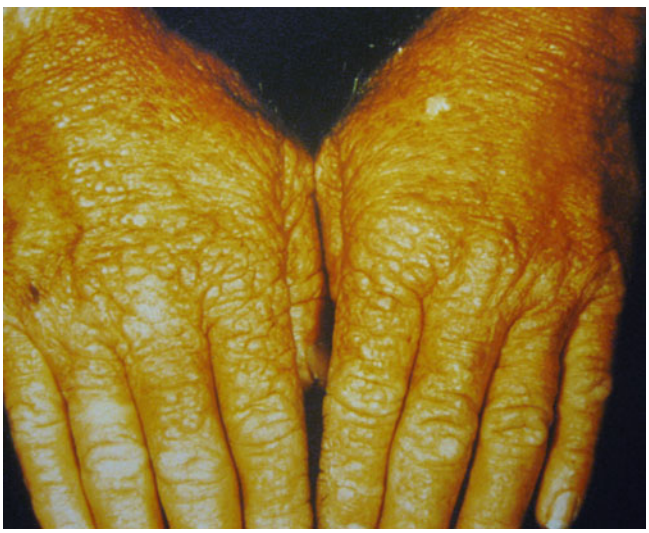
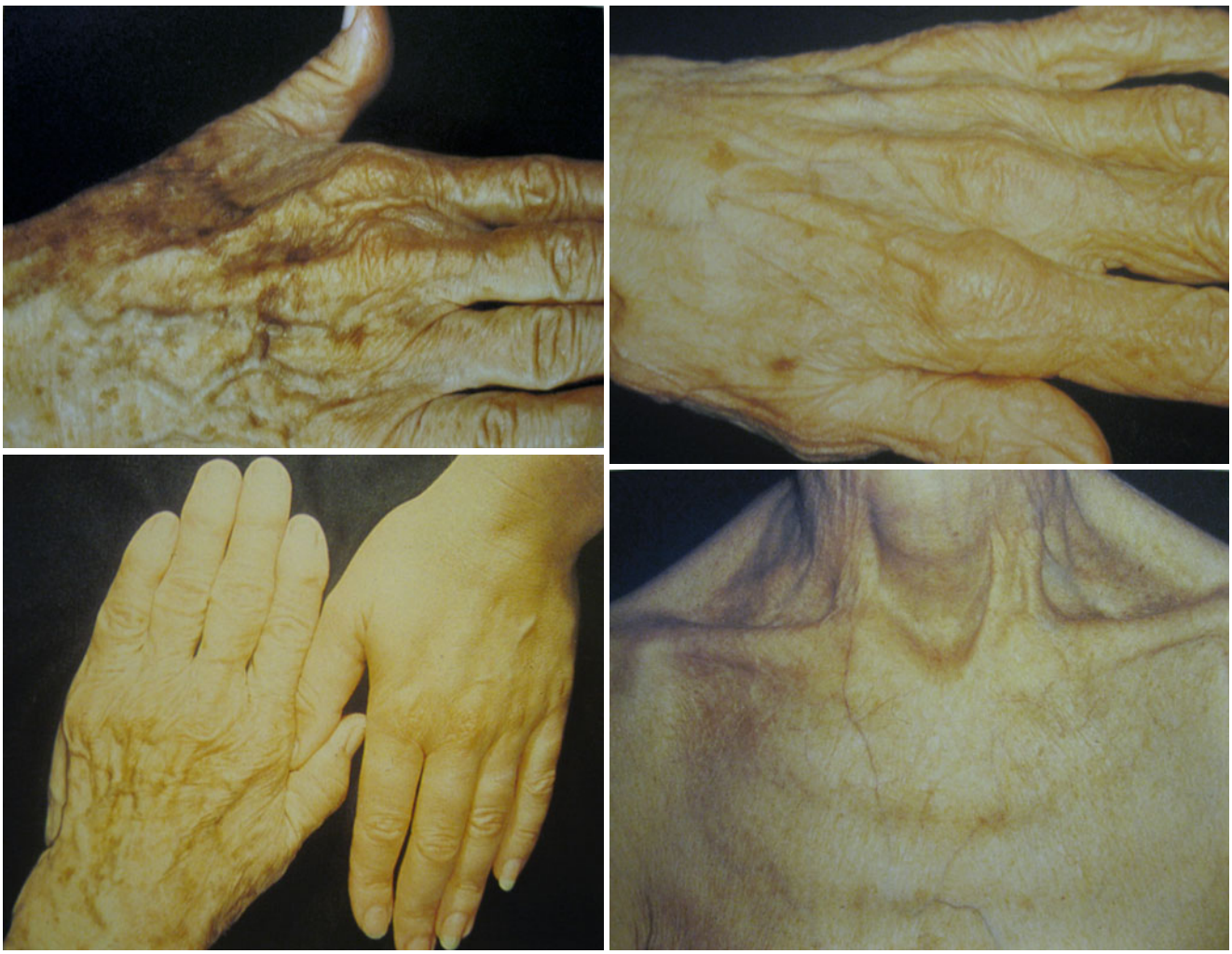
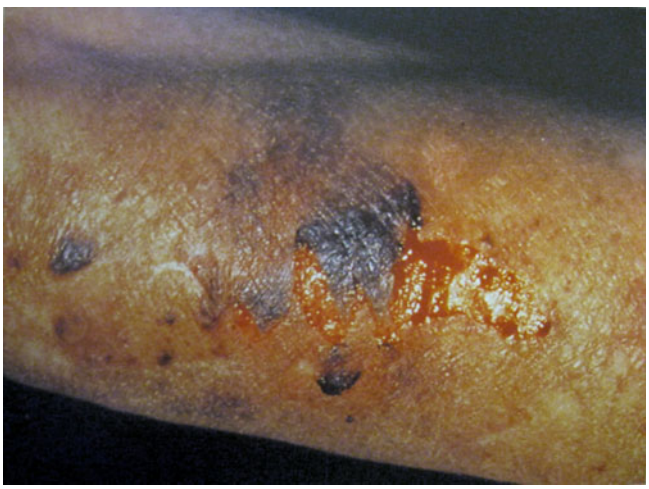


Fig. 17.11 Colloid milia. Discrete and confluent colloid (elastotic) papules can be noted on the dorsal hands and fingers

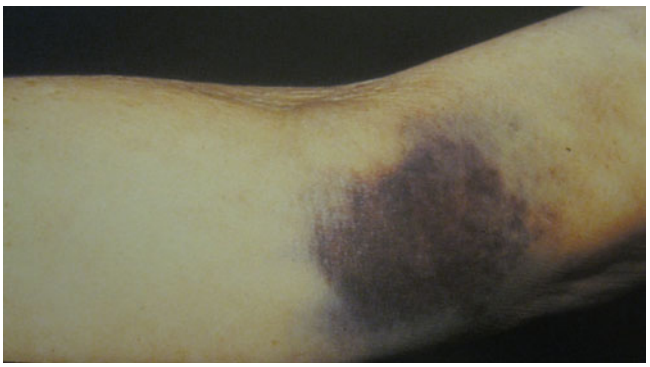


Figs. 17.12, 17.13, 17.14, and 17.15 Thin, atrophic, aged skin of the hands and chest

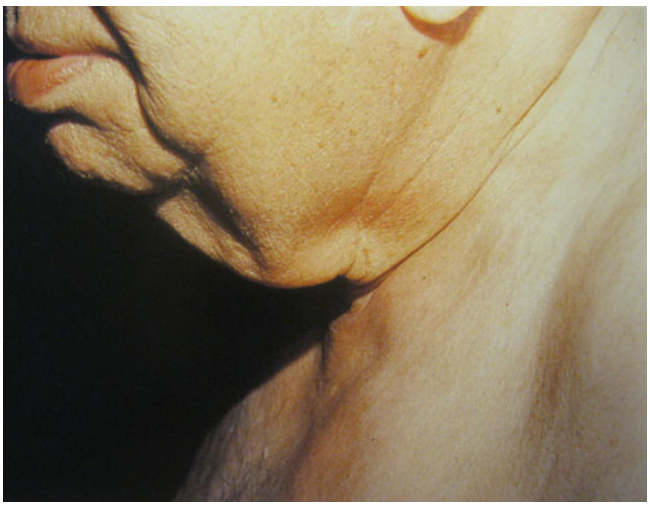
Fig. 17.16 Loss of elasticity is demonstrated by "tenting" of the skin after gentle pinching



Figs. 17.17 and 17.18 Actinic, solar, traumatic, or Bateman's purpura



Figs. 17.17 and 17.18 (continued)



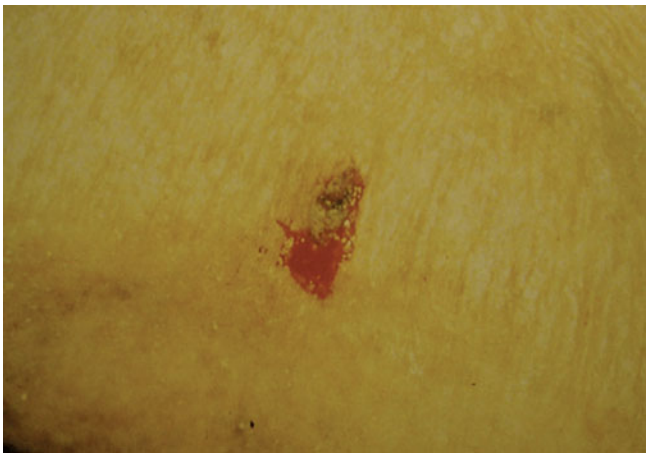
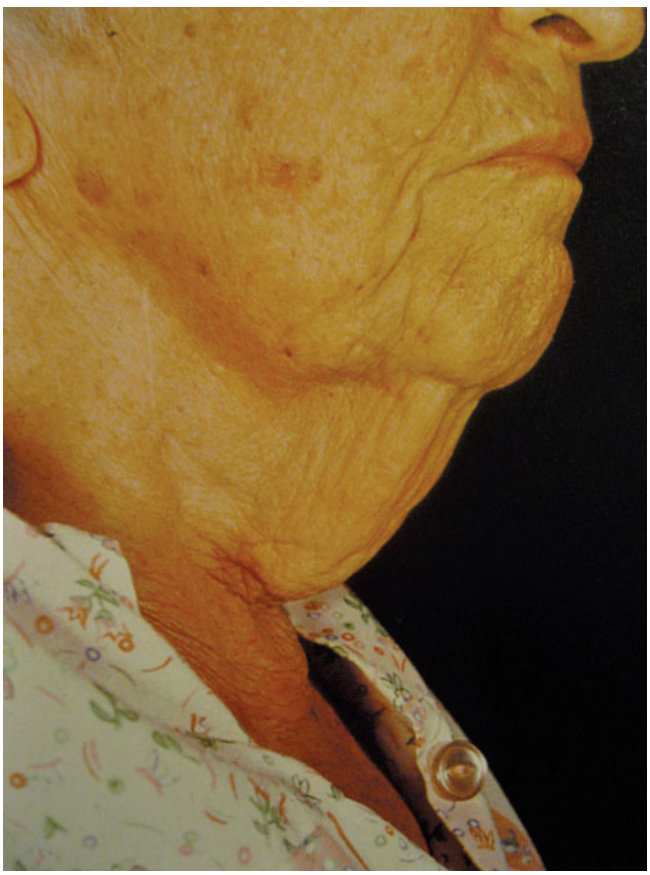


Fig. 17.19 Easily torn skin heals with white, star-shaped (stellate) pseudoscars



Figs. 17.20 and 17.21 Wattle ("turkey neck")

Fig. 17.22 Chronological aging versus photoaging (Courtesy of Johnson & Johnson, New Brunswick, NJ)

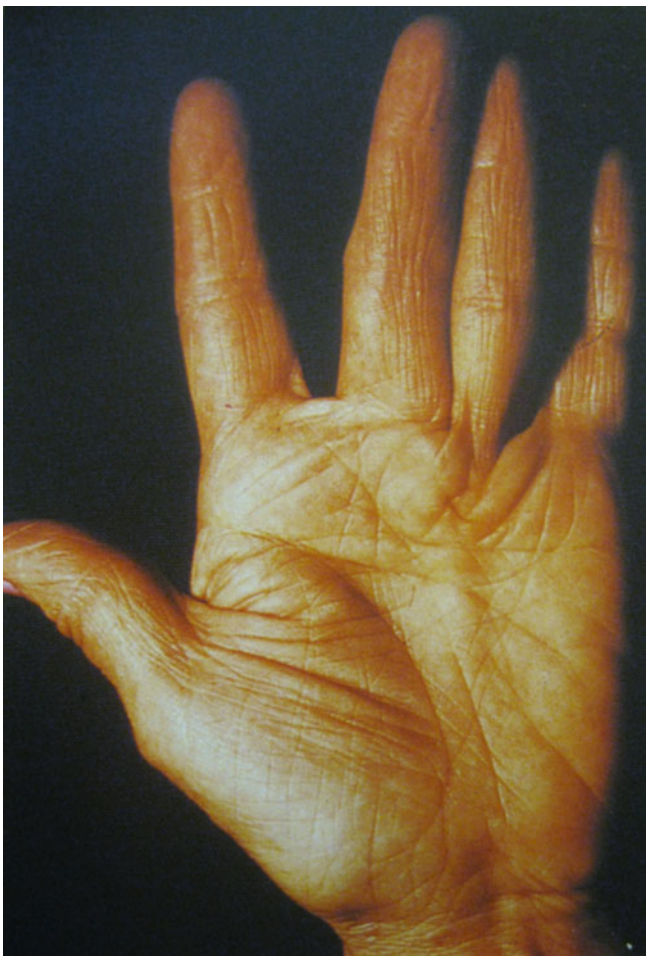


Fig. 17.23 Dupuytren's contracture is a fibrosis and shortening of the palmar tendons. The cause is unknown, although it occurs more commonly in alcoholics, diabetics, and the elderly

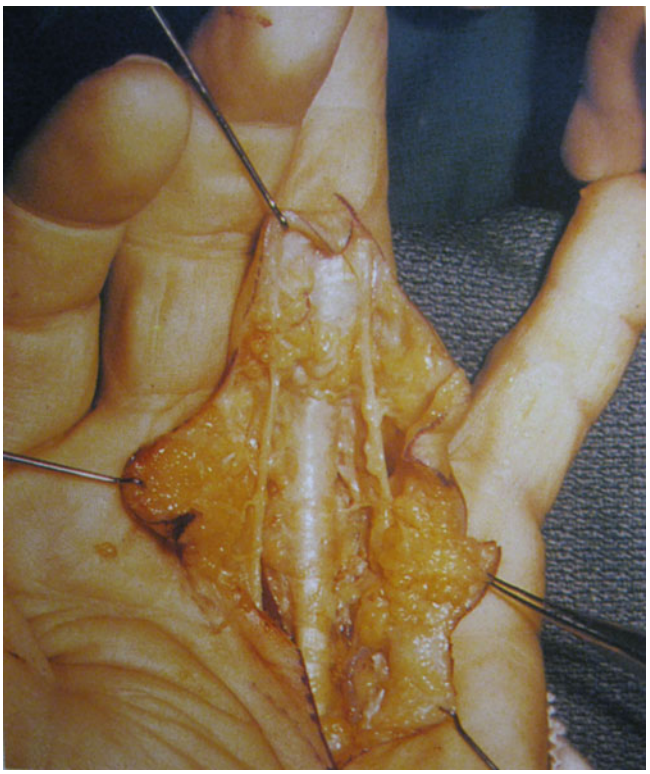


Fig. 17.24 Intraoperative repair of Dupuytren's contracture

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Urticaria

18

Clinical Description

Synonyms for urticaria include hives, welts, and wheals. Pink, edematous dermal plaques and papules often exhibit peripheral blanching and dermographism (wheal, flare, erythema—the triple response of Lewis). Individual lesions characteristically wax, wane, and migrate in less than 24 h, often in less than an hour.

Etiology and Pathophysiology

Allergic (lgE-mediated) or nonallergic (direct) mast cell degranulation results in histamine release, vasodilation, and dermal edema. Common causes include the following [1–4]:

1. Drugs-penicillin, aspirin, morphine, iodine
2. Infections-Streptococcus, hepatitis B, parasitic infestations
3. Foods-seafood, citrus, berries, chocolate
4. Stings-Hymenoptera (bee, wasp)
5. Physical factors-increased body core temperature (cholinergic urticaria), cold, physical pressure from tight clothing, jewelry, wristwatch, and other items.
6. Contactants, such as medications, chemicals, and foods, can cause contact urticaria.

Histopathology

Biopsy is usually not necessary or helpful except to rule out urticarial vasculitis. The minimal changes of dermal edema may not be apparent during processing, especially if epinephrine-containing anesthetic is used during biopsy.

Differential Diagnosis

The differential diagnosis includes the following:

1. Urticarial vasculitis-lesions persist for over 24 h as "fixed urticaria," may become hemorrhagic: biopsy reveals a leukocytoclastic vasculitis
2. Bullous pemphigoid-urticarial phase
3. Erythema multiforme
4. Angioedema-deep, subcutaneous urticaria that may affect the lips and throat; can be fatal

Therapy

1. The underlying cause, if found, must be identified, treated, and eliminated. A detailed history (drugs, previous diseases, foods, parasites, physical exertion, solar exposure) is of major importance. The history should differentiate between type of lesions--urticaria, angioedema, or urticaria + angioedema.
2. Antihistamines are more effective at preventing than relieving hives.
3. Subcutaneously administered epinephrine (Adrenalin) and other vasoconstrictors (Epi-Pen, ANA-Kit preloaded syringes) are prescribed to prevent anaphylactic shock. They are effective emergency treatments for hives and oral-laryngeal angioedema.
4. Systemic corticosteroids suppress urticaria but must be used cautiously until infectious causes have been eliminated.
5. Heavy exercising, overheating, sweating, and the use of caffeine, alcohol, coffee, tea, and other hot beverages should be avoided. Also, wearing loose-fitting clothing is helpful to reduce the occurrence of pressure urticaria [5].

Prognosis

Urticaria is often self-limited. In over 50 % of patients, no cause is detectable. Chronic urticaria (longer than 6 weeks) is especially difficult to treat, and often requires extensive evaluation, elimination diets, and environmental safeguards [6].

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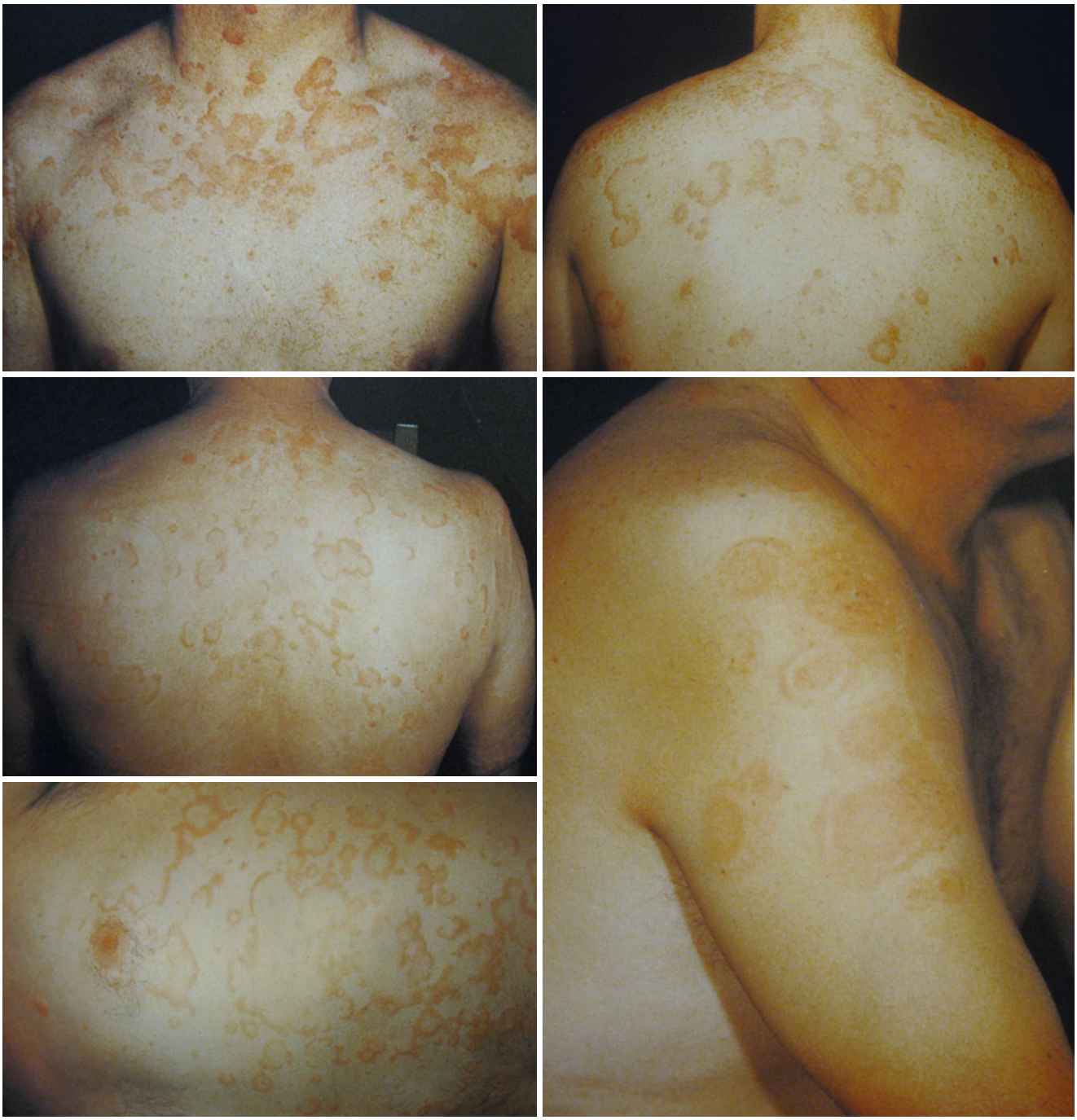
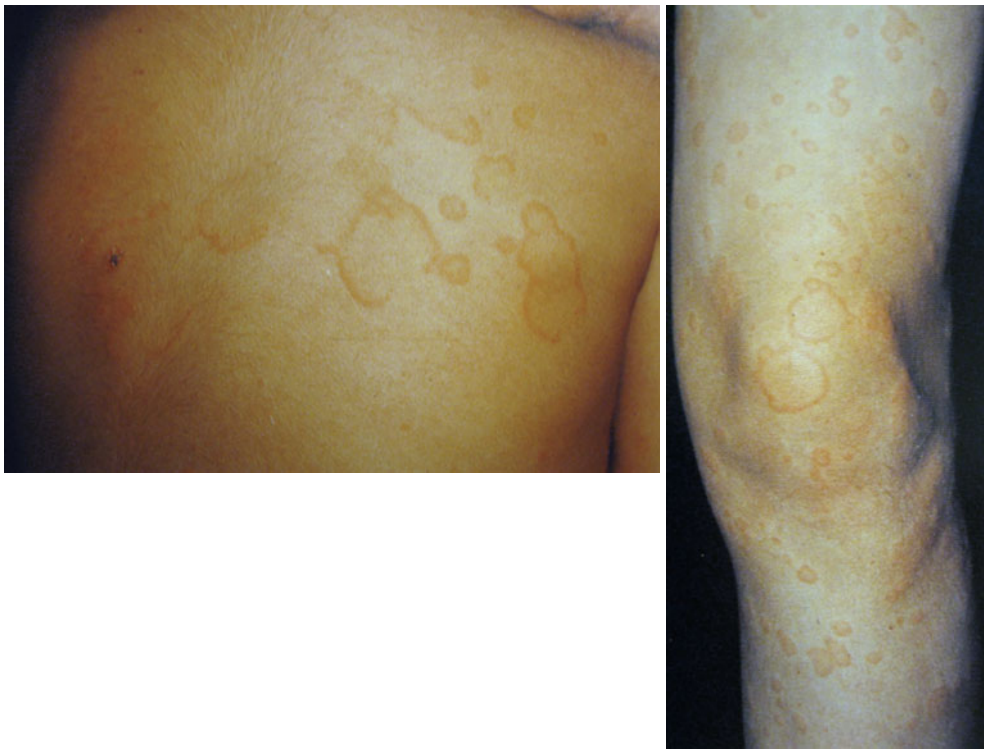
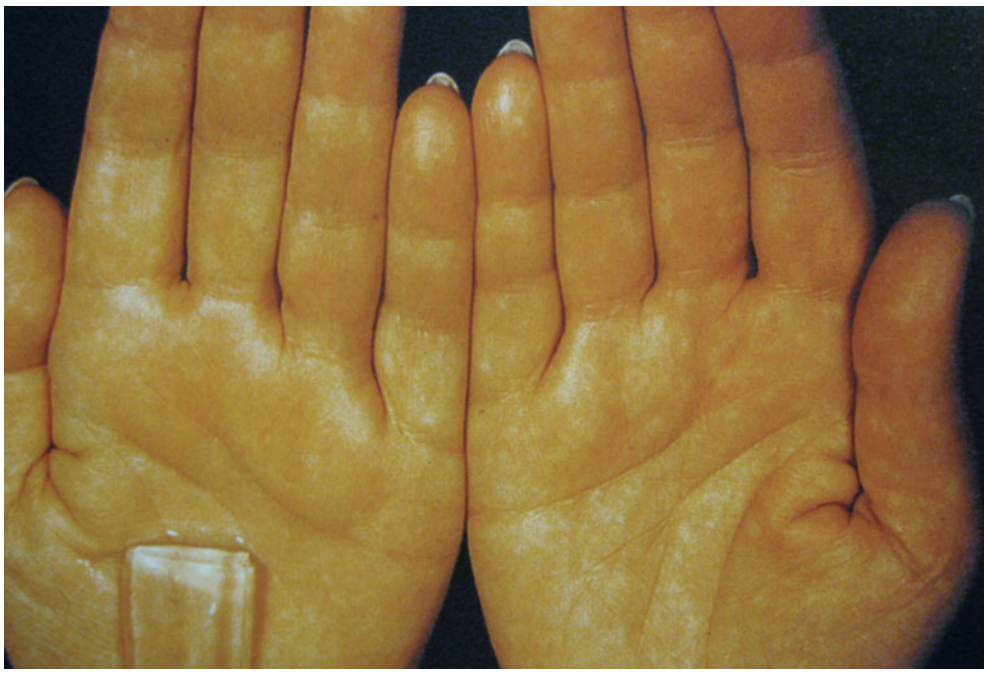
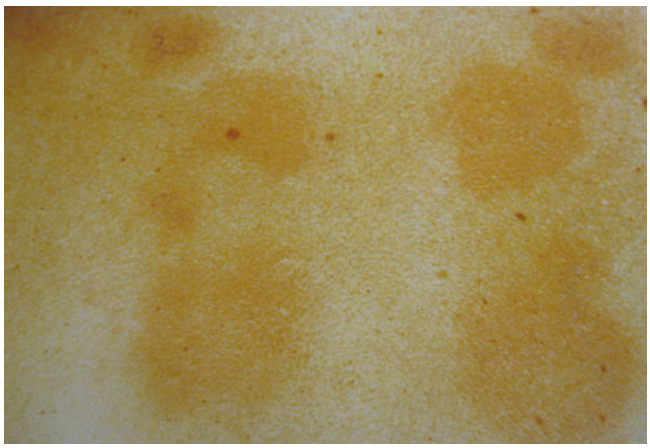


Figs. 18.1, 18.2, 18.3, 18.4, 18.5, 18.6, and 18.7 Typical urticaria: annular and arcuate edematous, erythematous dermal plaques without epidermal changes. Lesions are transient, lasting less than a day in any-one location. In contrast, urticarial lesions with hemorrhage lasting over 3 days suggest urticarial vasculitis, a true leukocytoclastic vasculitis

Figs. 18.1, 18.2, 18.3, 18.4, 18.5, 18.6, and 18.7 (continuned)







Figs. 18.8 and 18.9 Cold urticaria. Ice cubes placed on palms (Fig. 18.8) and back (Fig. 18.9) for 30 and 60 s induce urticarial wheals

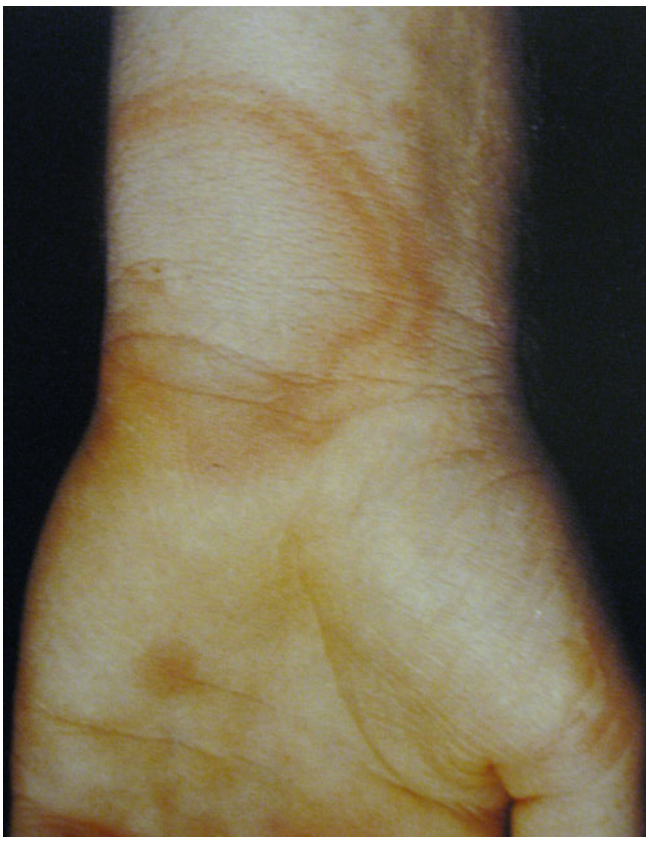


Fig. 18.10 Large annular, urticarial wheal

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Xerosis

19

Clinical Description

Synonyms for xerosis include asteatosis, dry skin, winter itch, and eczema craquelé. Manifestations of dry skin can progress from reddening and cracking to severe persistent pruritus and lichenification. The loss of hydration in the epidermis causes fissuring and cracking of the stratum corneum, making the skin look like antique porcelain (eczema craquelé). As the condition worsens, the skin scales and begin to flake, cracks may extend and deepen, and the skin feels uneven. Local or generalized pruritus may also be present. Chronic scratching and rubbing result in painful excoriations and infected with lesions that become thick and lichenified. Dry skin is the most common skin complaint in the elderly, affecting nearly 75 % of those 64 and older. In a recent study, the most common problem affecting nursing home patients was dry and pruritic skin. Dry skin is worse in the winter due to low humidity [1, 2].

Etiology and Pathophysiology

Xerosis is caused by dehydration through the hyperpermeable epidermis and stratum corneum. Increased age includes reduced sebaceous and sweat gland activity causing dryness. Xerosis has also been associated with zinc or essential fatty acid deficiency, renal disorders, hypothyroidism, neurologic disorders that decrease sweating, HIV, malignancies, obstructive biliary disease, and in those with radiation therapy [3, 4].

Histopathology

Biopsies are rarely performed since dry skin can easily be detected clinically.

Differential Diagnosis

1. Ichthyosis vulgaris – an autosomal dominant disorder associated with atopic dermatitis, hyperlinear palms, and keratosis pilaris.
2. Nummular dermatitis – red, annular, scaly, dry patches on arms and legs.
3. Pityriasis alba – presents as white, fine scaling or non-scaling patches on the face and trunk of younger patients.

Treatment

1. Artificial humidification in homes through vaporizers and humidifiers.
2. Changing bathing habits: Bathing less frequently, using warm instead of hot water, and showering instead of soaking in a tub can help reduce natural oil loss.
3. Patients should drink plenty of water daily and wear protective clothing in cold, dry weather.
4. Milder superfatted soaps and cleansing creams and help reduce excessive skin oil loss. Products with alcohol and fragrance should be avoided, as they also lead to dryness.
5. Moisturizers such as ceramide-based creams and lotions can be very helpful for protecting and restoring the epidermal water barrier. Petrolatum, lanolin, cocoa butter, olive oil, and heavy mineral oils can be greasy and sticky, but work well when combined with humectants such as glycerin, propylene glycol, pyrrolidone, carboxylic acid, sodium lactate, and urea [1, 5].

Prognosis

Prognosis for patients with xerosis is generally good. However, underlying conditions such as systemic and genetic

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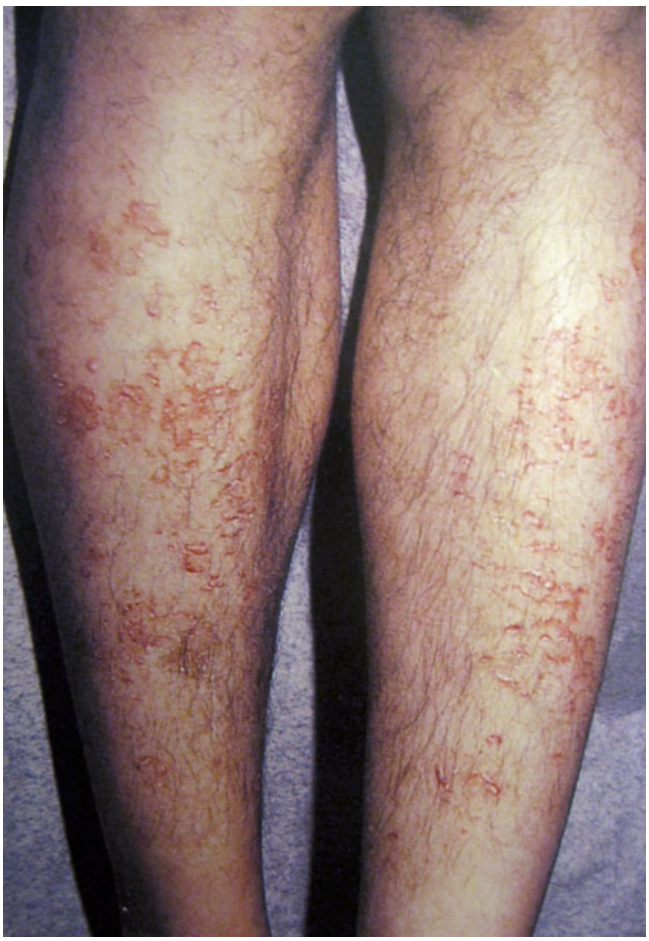
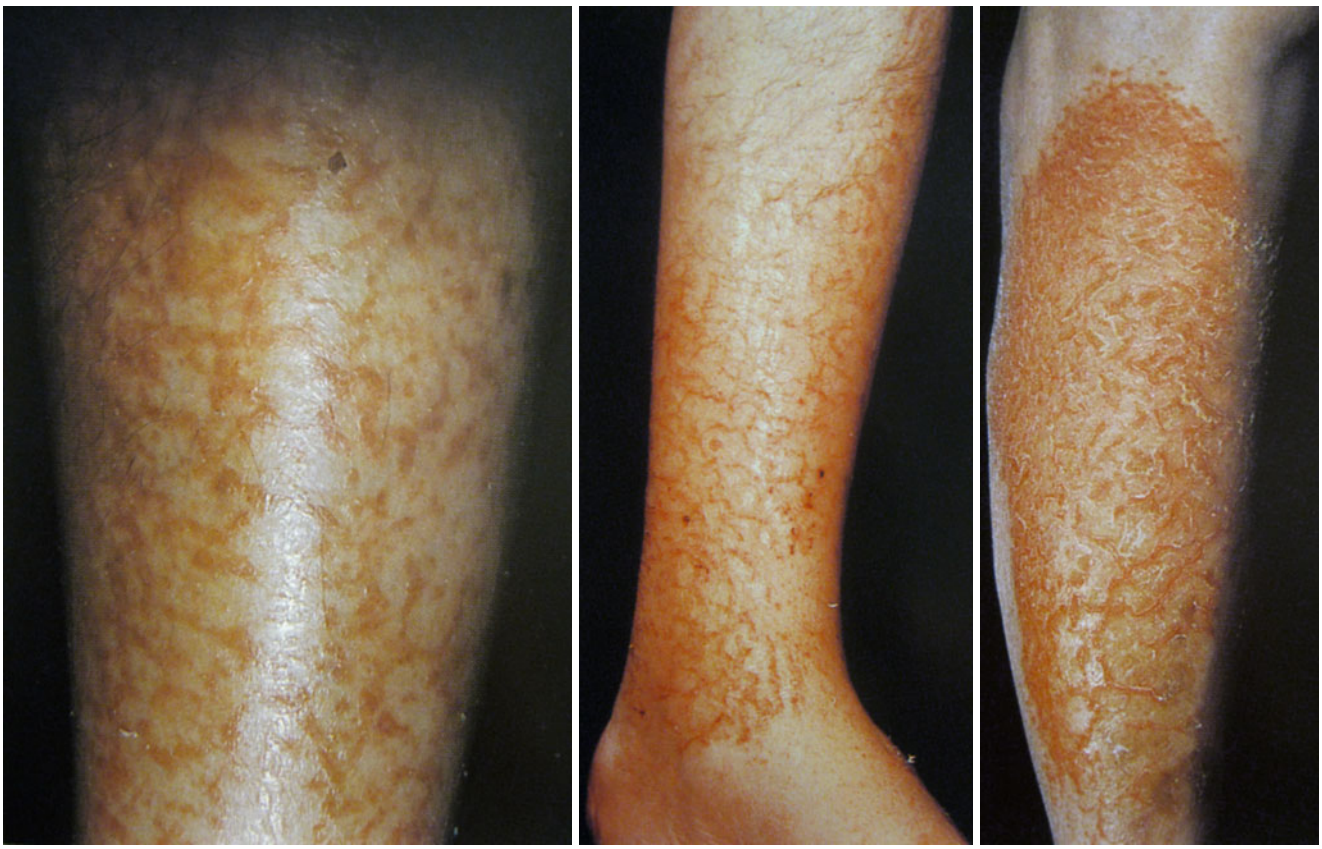


Fig. 19.1 Moderate xerosis shows eczematous, circular patches (nummular dermatitis) and reticulated (net-like) erythema of eczema craquele



Figs. 19.2, 19.3, and 19.4 Severe eczema craquele may become superinfected, resulting in cellulitis

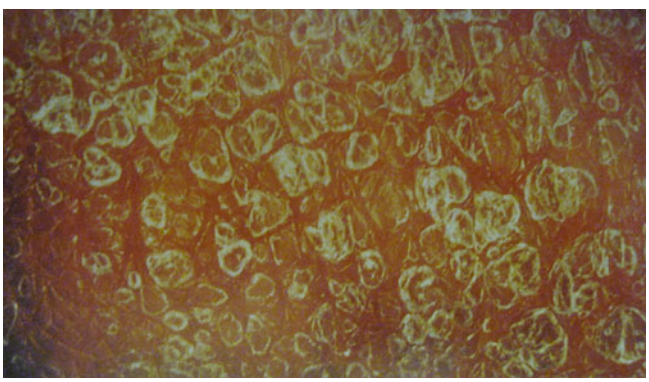


Fig. 19.5 Close-up views of asteatotic skin reveal a tessellated or tile-like crazing or mosaic pattern of scale and erythema (Reproduced with permission from Newcomer and Young [6])

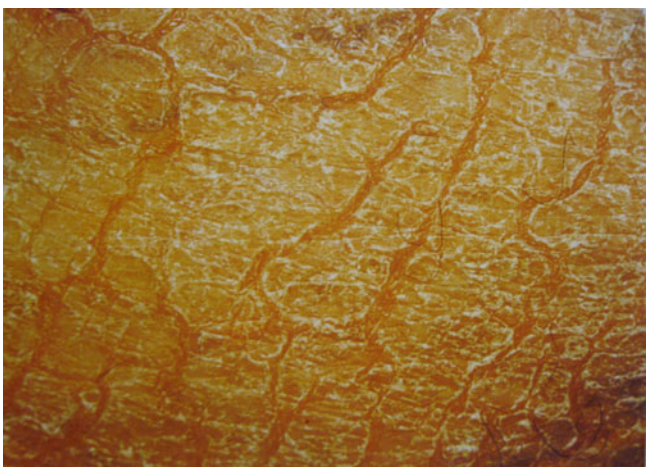


Fig. 19.6 Close-up views of asteatotic skin reveal a tessellated or tile-like crazing or mosaic pattern of scale and erythema

complications may cause chronic xerosis that can only be controlled with treatment. New formulas of moisturizers and soaps have improved treatment outcomes.

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Part III

Infections

Superficial Fungal Infections

20

Clinical Conditions

Several clinical conditions are associated with fungal infections:

1. Dermatophytosis, commonly called tinea or ringworm, usually presents as red or brown scaling, annular patches and plaques on the scalp, face, trunk, extremities, or interdigital web spaces. Hair loss may be prominent [1].
2. Candidiasis, also called moniliasis or yeast infection, usually presents as beefy red moist patches and plaques, often studded with small yellow pustules, on moist skin, nails, and mucous membranes, especially the tongue. Thrush refers to a thick white coating of candida that can be scraped off the tongue or buccal mucosa.
3. Tinea (pityriasis) versicolor presents as white, tan, or pink lightly scaling, discrete, and confluent macules, papules, patches, and plaques over the upper trunk and neck. It is less common in elderly patients because of decreased perspiration and oil production (compared to teenagers and young adults)
4. Dermatophytid (id) reactions are secondary eruptions, usually on the fingers and hands, that follow allergic sensitization to fungi. They may follow spontaneous inflammation of a primary lesion (usually on the lower part of the body) or as an irritation from overtreatment. Usually, a series of vesicles appears on the palm or along the side of the finger. They may itch or become infected and painful.

Etiology

These clinical conditions have various causes:

Dermatophytosis. Three genera of dermatophytes invade keratinized areas of the body, such as the skin, hair, and nails-Trichophyton, Microsporum, and Epidermophyton

Candidiasis. *Candida albicans* flourishes as an opportunistic invader in the settings of diabetes, antibiotic or estrogen administration, and immunosuppression (from cancer and AIDS). Candida invades keratinized as well as non-keratinized surfaces, including mucosa [2–5].

Tinea Versicolor. *Pityrosporum orbiculare* and *P. ovale* (*Malessezia furfur*) yeasts thrive in an environment of oil and excessive perspiration.

Dermatophytid Reactions. Although fungi (usually *Trichophyton mentagrophytes*) can be demonstrated at the primary site, they are usually not found in the id. However, the id does exhibit a positive reaction to a skin test with a standard Trichophyton vaccine. The id rash disappears after fungi have been cleared from the primary site [6–9].

Diagnosis

The following diagnostic procedures are helpful.

1. Scrapings of infected scales, nail debris, pustules, and/or affected plucked hairs are treated with KOH (potassium hydroxide) to dissolve keratin. Microscopic examination reveals branching hyphae (dermatophyte), budding yeasts, and/or pseudohyphae (Candida albicans, Pityrosporum ovale).
2. Cultures on Sabouraud agar reveal characteristic colony morphology in 1–2 weeks. DTM (dermatophyte test medium) contains an indicator dye that turns red when dermatophytes are present.
3. Histologic investigation, using special stains for fungal organisms (PAS, periodic acid-Schiff) is occasionally helpful if cultures are negative.
4. Wood's lamp (UVA [black light]; demonstrates fluorescing dermatophytes (*Microsporum audouini*, *M. Ierruglneum*, *M. canis*, *M. distortum*) and tinea versicolor.

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Differential Diagnosis

The differential diagnosis includes the following:

1. Tinea--eczema, psoriasis, seborrheic dermatitis, pityriasis rosea, secondary syphilis, and all the papulosquamous diseases. A fungal culture and KOH examination should be done on all scaling eruptions. Ringworm is an annular eruption whose differential diagnosis includes the annular erythemas, such as erythema annulare centrifugum and erythema chronicum migrans associated with Lyme disease
2. Candida--bacterial intertrigo and folliculitis, inverse psoriasis
3. Tinea versicolor--papulosquamous conditions as above, plus vitiligo, guttate hypomelanosis, and pityriasis alba (for white areas).
4. Dermatophytid reaction--contact dermatitis, dyshidrotic eczema (pompholyx), herpetic whitlow (herpes simplex virus). The hands and feet should be examined as a dermatologic unit for fungus.

Treatment

1. Topical antifungals constitute first-line therapy for localized, uncomplicated, fungal infections of the skin [1]. Three categories of topical antifungals include the imidazoles (clotrimazole, econazole, ketoconazole, miconazole, oxiconazole, sulconazole), an allylamine (naftitine), and ciclopirox olamine. If a topical antifungal from one category does not work, it is prudent to try antifungals from the other categories before resorting to systemic therapy. Antifungal powders are also useful.
2. Systemic therapy, using griseofulvin (for dermatophytes only), ketoconazole (for both dermatophytes and candidiasis), or fluconazole is necessary for extensive generalized tinea corporis, for deep infections involving the hair root (Majocchi's granuloma), candidiasis, and for impenetrable structures such as nails and thick-skinned palms and soles. Side effects, mainly hepatotoxicity and hematologic depression, or interactions with blood thinner such as warfarin, limit their use in the elderly.
3. Local measures include keeping the skin cool and dry and reducing hyperkeratosis with keratolytic agents, such as 12 % ammonium lactate lotion, tretinoin cream or gel, or salicyclic acid. Laser ablation can improve onychomycosis (fungal load in nails).
4. Dermatophytids resolve spontaneously once the primary fungal infection has been treated. Topical or internal steroids may be necessary to control severely pruritic or painful ids. Caution must be used to prevent exacerbating the underlying fungal infection [10, 11].

Prognosis

Most superficial fungal infections can be controlled by topical therapy. Tinea pedis can progress to gram-negative toe web infection; the gram-positive bacteria are killed by penicillin-like products of the dermatophytes. Cellulitis of the foot and leg can ensue. Candidiasis can occasionally progress to sepsis, an often fatal complication,

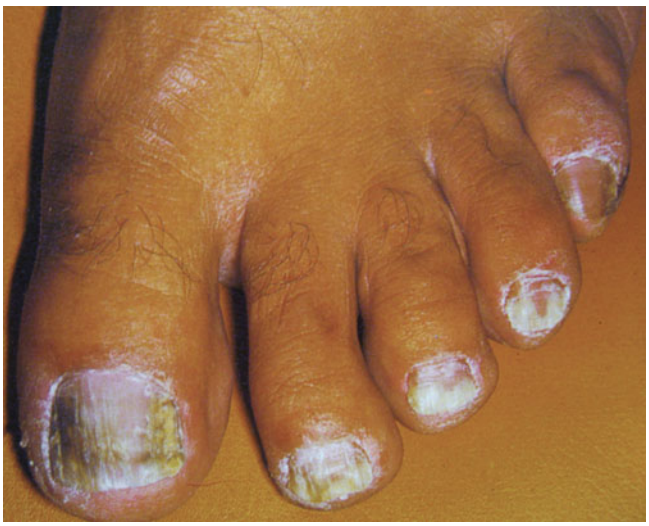


Fig. 20.1 Onychomycosis of the toenails. *Trichophyton rubrum* is the most common cause. It causes a dry, scaling, noninflammatory tinea pedis

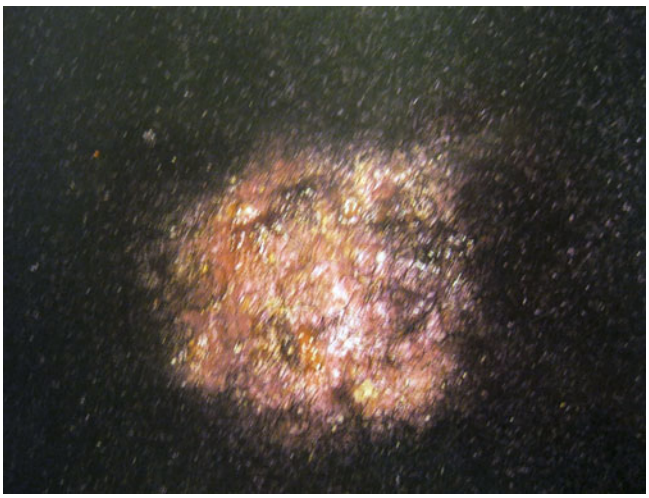


Fig. 20.2 Tinea capitis shows an area of alopecia with broken hairs. *Trichophyton tonsurans* is the most common cause

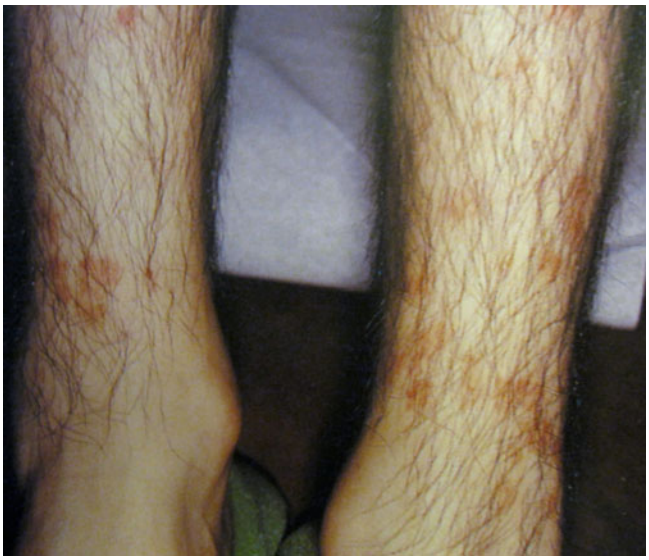
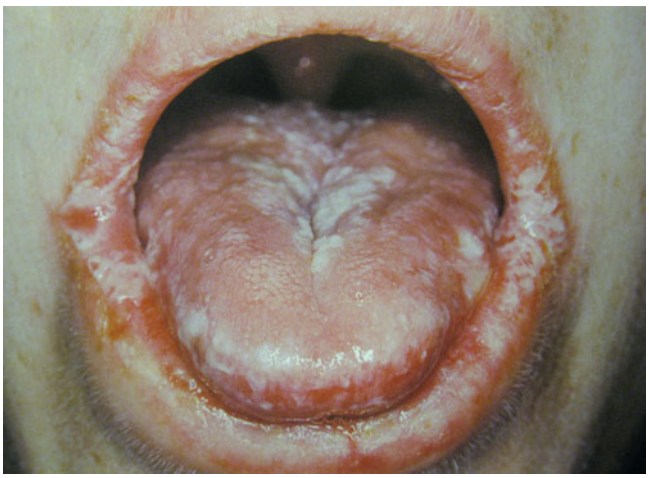
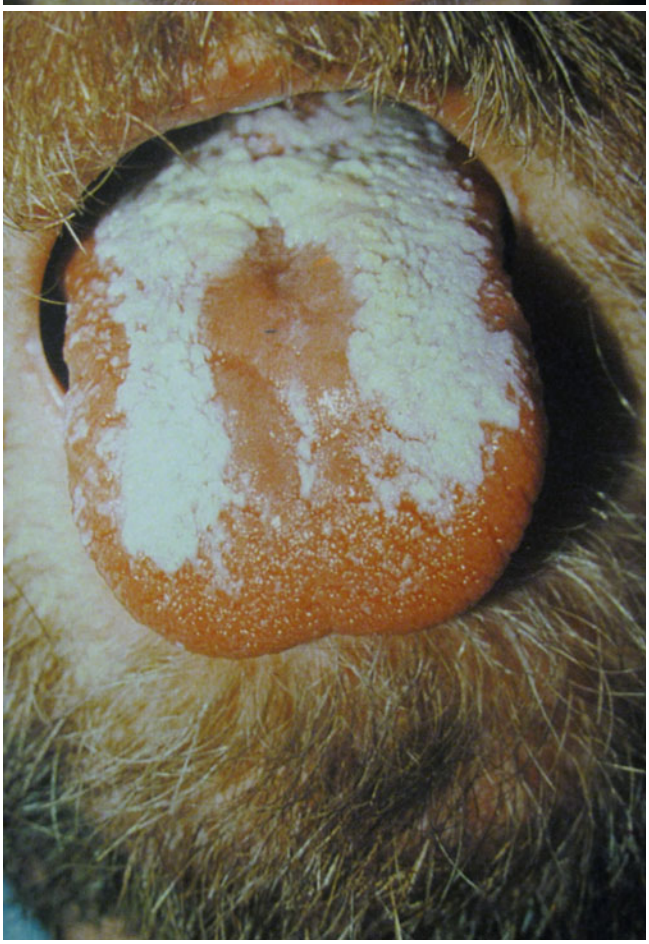
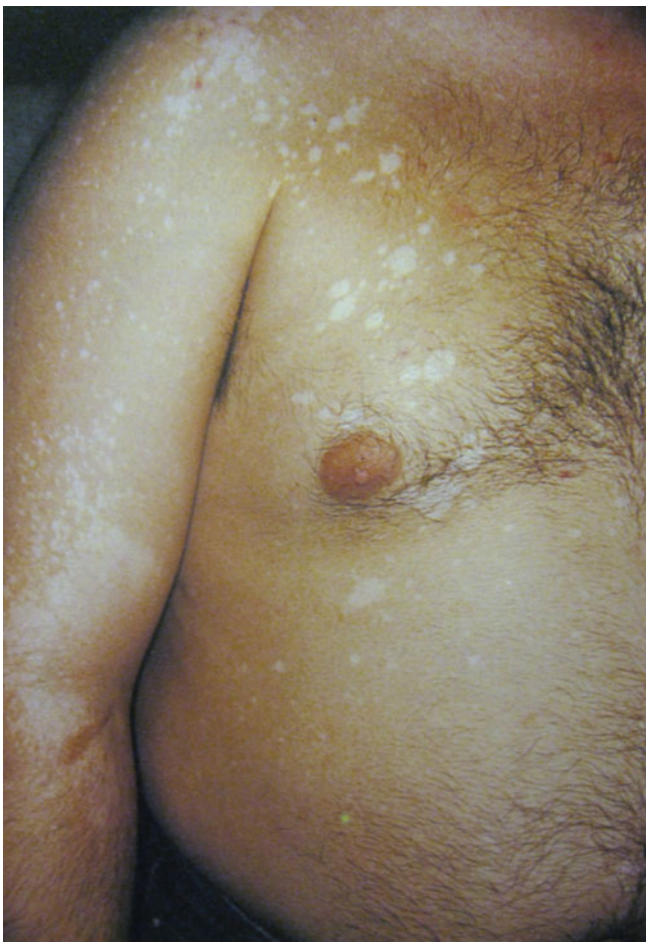


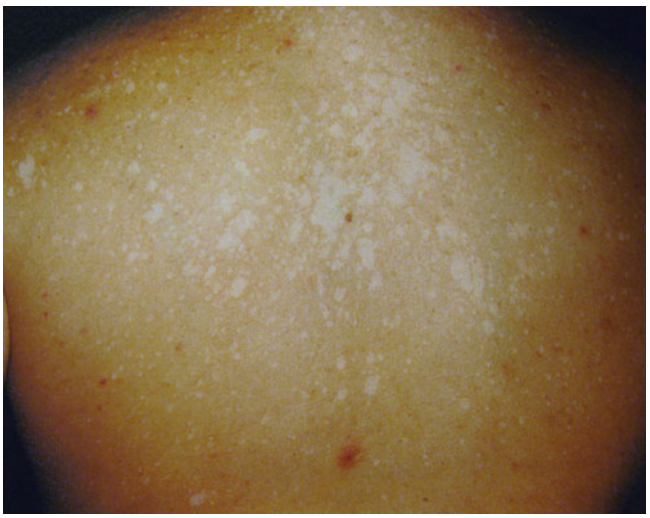
Fig. 20.3 Tinea corporis of the legs caused by *Microsporum canis* contracted from patient's infected cat. Wood's lamp examination shows *yellow-green* fluorescence





Figs. 20.4 and 20.5 Candidiasis. The white cottagecheese-like-exudate can be scraped off the tongue, exposing a tender, beefy, red, raw mucosal surface





Figs. 20.6 and 20.7 Tinea versicolor. Hypopigmented, slightly scaling patches on the trunk are less common in elderly than in younger patients

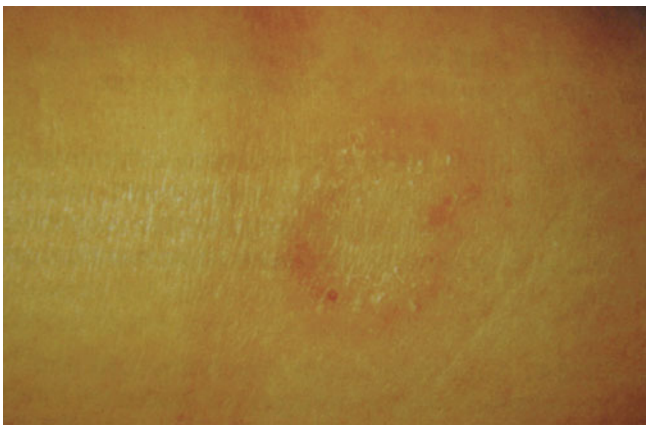
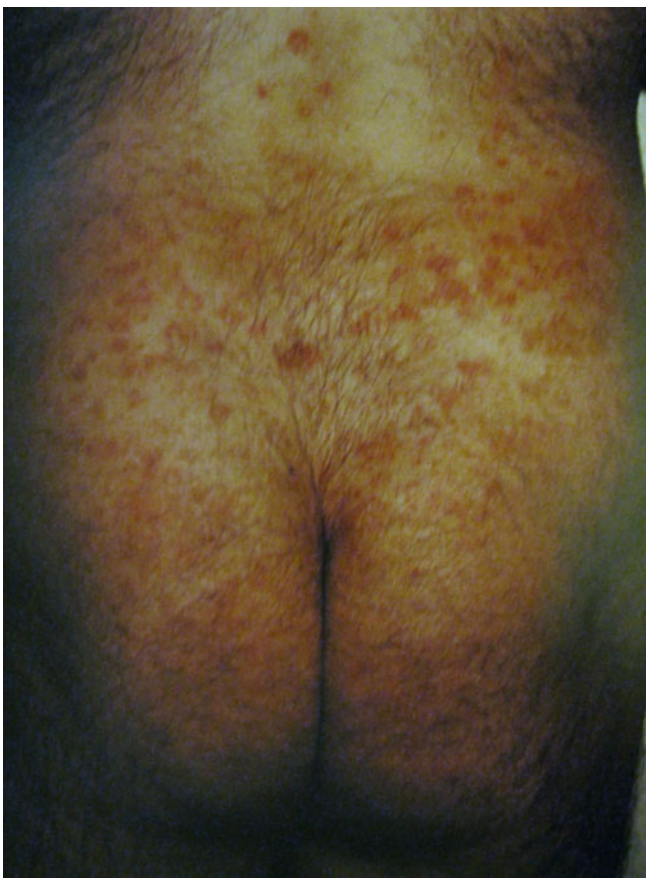
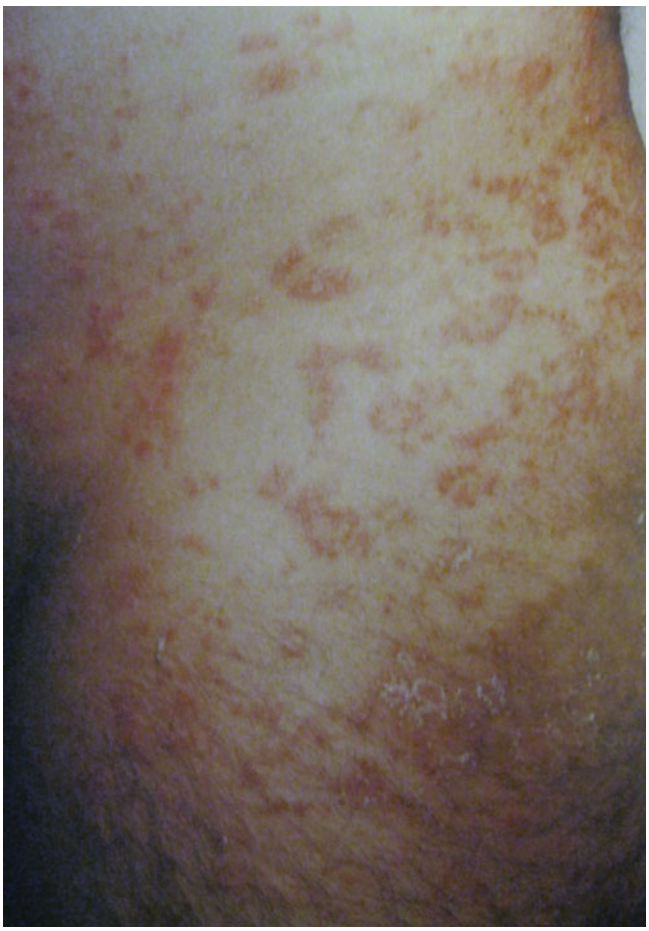


Fig. 20.8 Ringworm. This annular, vesicular, inflammatory eruption is caused by *Trichophyton mentaqrophytes*



Figs. 20.9 and 20.10 Ringworm. This annular, scaling eruption is caused by *Trichophyton rubrum*



Figs. 20.9 and 20.10 (continued)

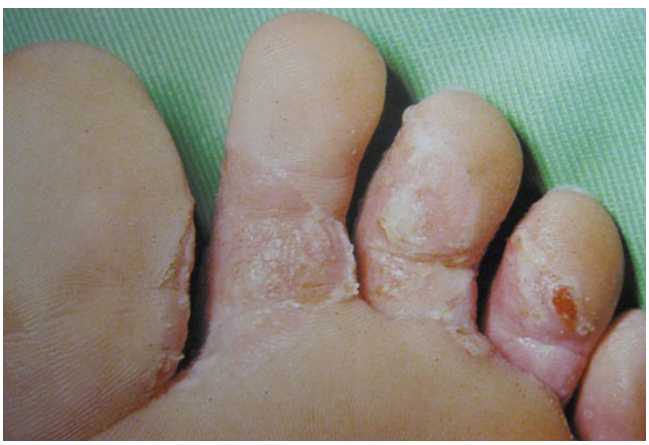


Fig. 20.11 Tinea pedis is an intensely inflammatory infection caused by *Trichophyton mentagrophytes*. In chronically wet or macerated areas, gram-negative toe web infection must be suspected and treated with topical antifungals and antibiotics

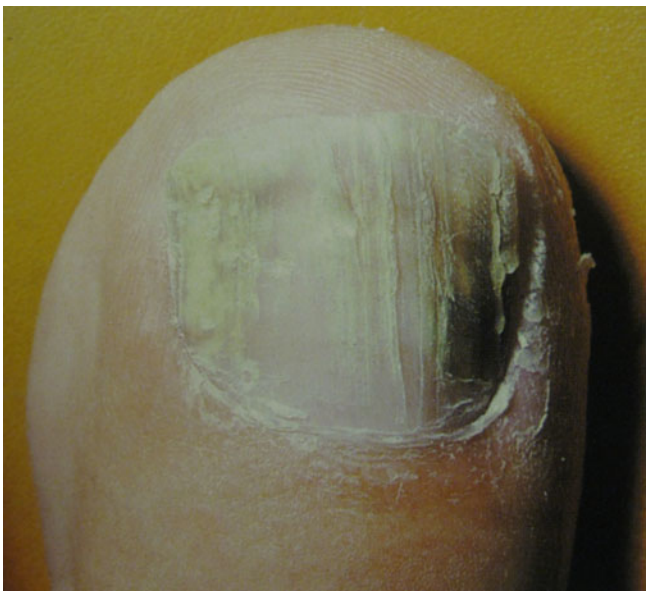


Fig. 20.12 Superficial white onychomycosis and blue-green *Pseudomonas aeruginosa* (bacterial) infection of the great toenail

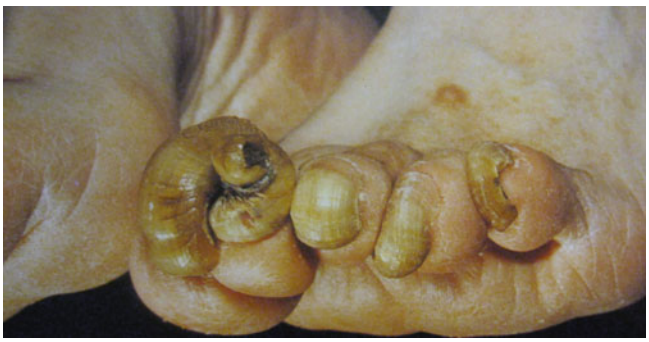


Fig. 20.13 Onychogryphosis refers to overgrown, curved nails that are not necessarily infected with fungus. Arthritic elderly patients are often unable to trim their toenails

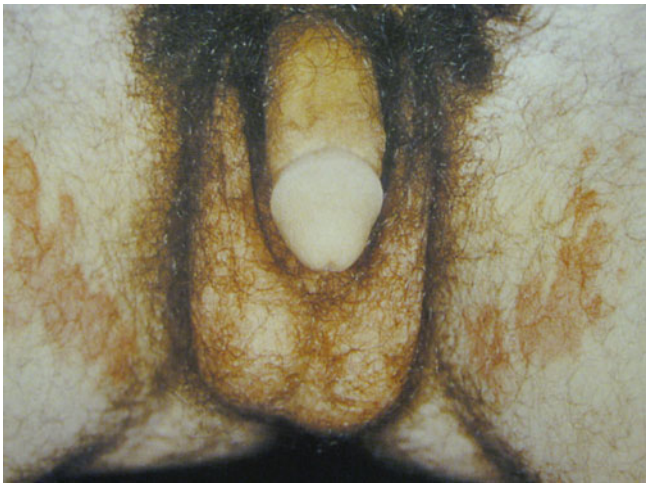


Fig. 20.14 This unique drug eruption resembles that of Epidermophyton floccosum-*type* tinea cruris, but no fungal elements were detected by scrapings or culture. This eruption represents the initial manifestation of a penicillin allergy that localized to the area of a previous fungal infection of the groin

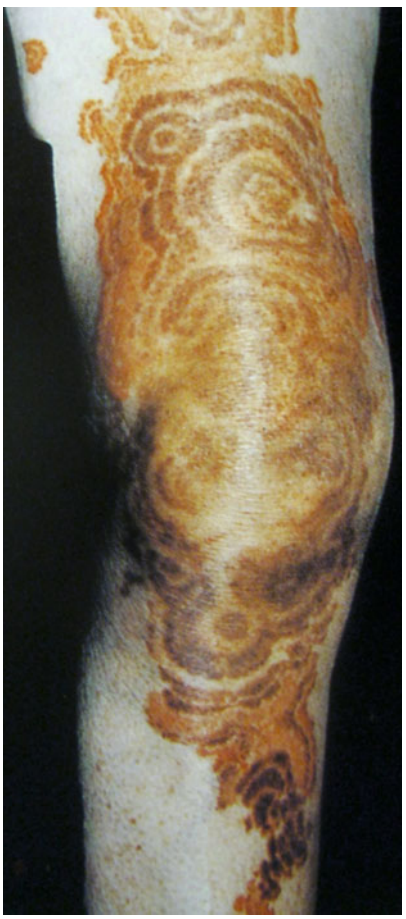


Fig. 20.15 This bizarre drug eruption simulates ringworm

Fig. 20.16 Erythema chronicum migrans is an annular erythema associated with Lyme disease. It resembles ringworm

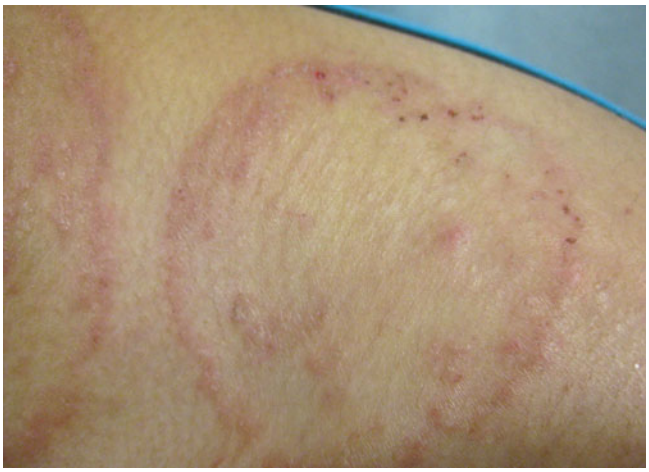


Fig. 20.17 Extensive tinea corporis

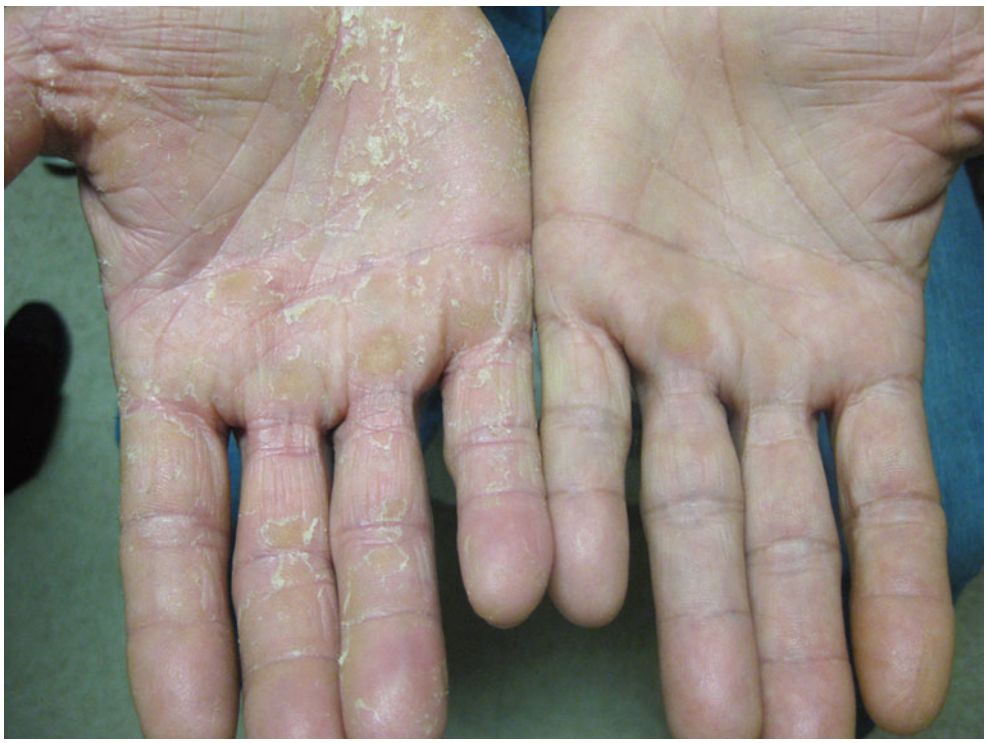


Fig. 20.18 One-hand, two-foot disease

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Herpes Simplex

21

Synonyms

Synonyms for herpes simplex include cold sores, fever blisters (herpes simplex labialis), and genital herpes (herpes simplex genitalis).

Clinical Description

Herpes simplex is less common in elderly than in younger patients [1, 2]. Recurrent episodes consist of localized, painful, grouped, tense vesicles on a red, erythematous base. Vesicles resolve in 7–10 days. A prodrome of tingling or burning lasts 1–3 days before vesiculation occurs.

Etiology and Pathophysiology

Skin inoculation with herpes simplex I or II virus results in retrograde transmission of virus down the nerve, with a reservoir of virus in the ganglion and antegrade transmission of virus back to the skin during attacks. Aggravating or precipitating factors include the following:

1. Stress
2. Sunburn (especially herpes labialis)
3. Menstrual periods
4. Illness
5. Immunosuppression from drugs or cancer, particularly defects in cellular immunity caused by lymphoma, leukemia, or AIDS

Patients with severe, progressive herpes should be examined for these underlying factors.

Histopathology

Histopathologic investigation reveals intra epidermal reticulated vesicles with characteristic multinucleated giant cells. These may be seen by examining a Tzanck preparation (the undersides of the blister roof and blister base are scraped and visualized with modified Wright-Giemsa [Tzanck] stain).

Differential Diagnosis

The differential diagnosis includes herpes zoster, which shows dermatomal distribution of lesions but is rarely, if ever, recurrent [3].

Therapy

No treatment is necessary in most cases, but the following may be used, if required:

1. Acyclovir 200 mg orally, five times daily for 5 days for severe episodes, (capsule [200, 800 rng] and elixir [200 mg/5 ml] forms available) [4–11]
2. 2. Acyclovir, 800 mg, orally twice daily for 5 days for severe recurrent episodes of male gertital herpes [7]
3. Acyclovir, 200 mg orally, once, twice, three or four times daily for chronic long-term (I-year) suppression of frequent (twice monthly or more) episodes [7]
4. L-Lysine, several other agents, have a 30 % placebo effect [12–15]

Prognosis

The natural history progresses to less severe, less frequent episodes.

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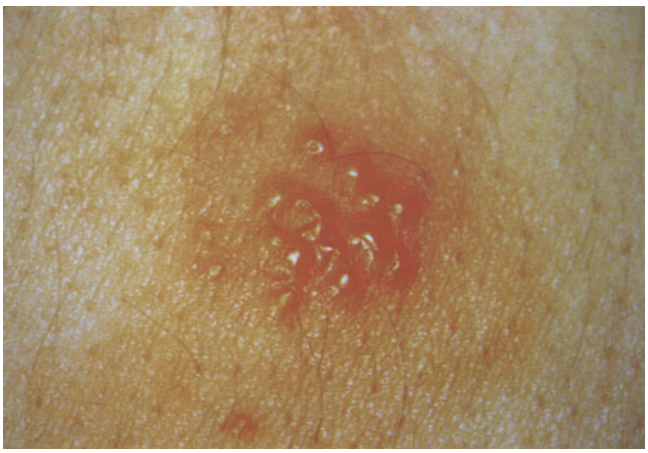
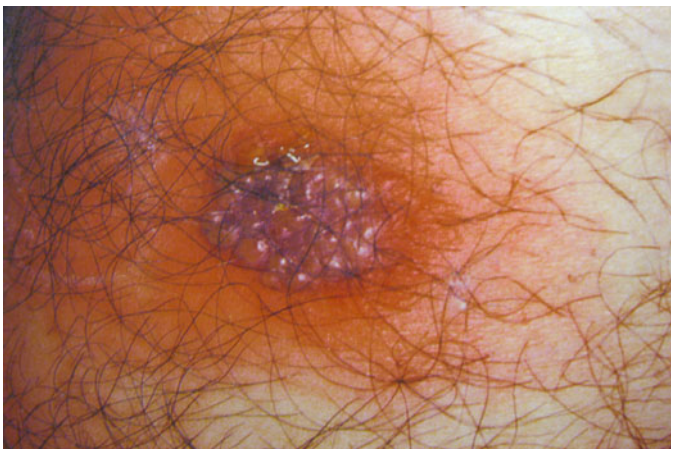
DOI 10.1007/978-1-4471-4579-0_21, © Springer-Verlag London 2014





Figs. 21.1 and 21.2 Classic presentation of herpes sim*plex* is grouped *vesicles* on an erythematous base

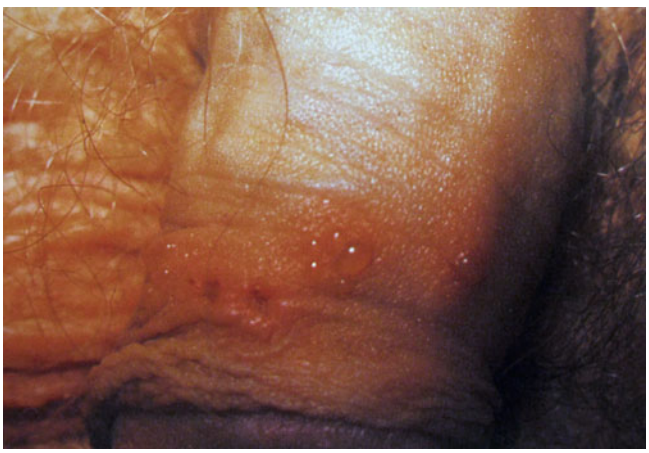


Fig. 21.3 Herpes simplex genitalis

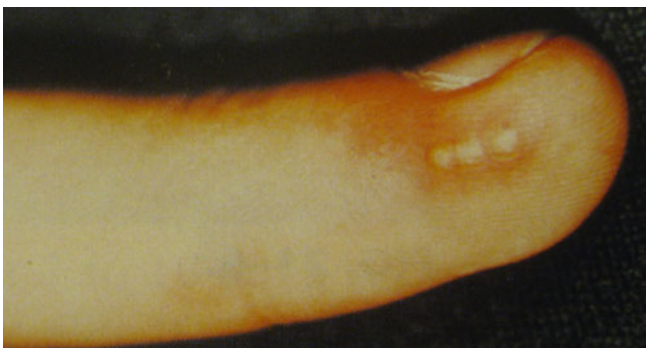


Fig. 21.4 Herpetic whitlow is an occupational hazard of dentists and dental hygienists who fail to wear *gloves*

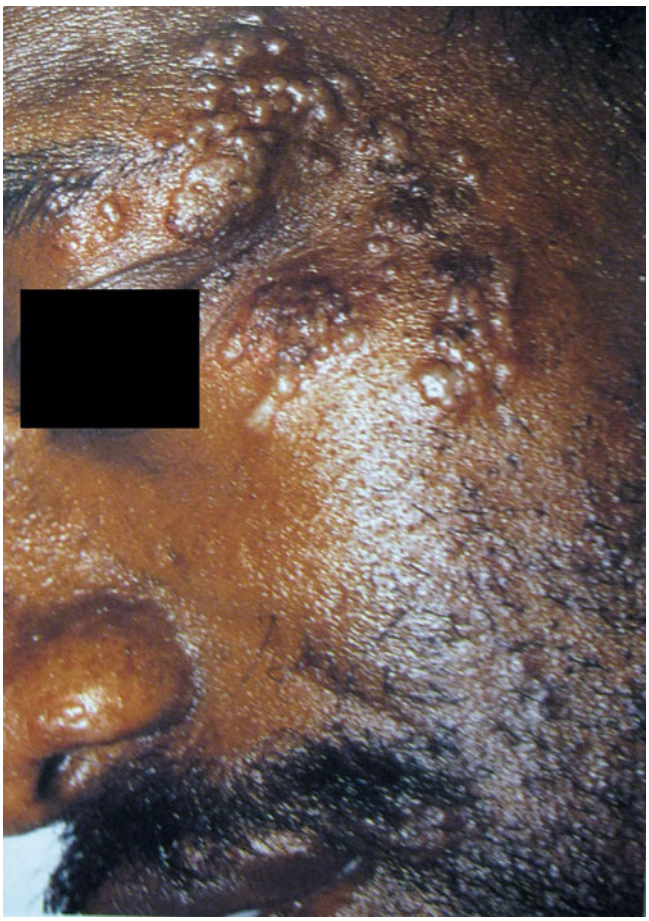
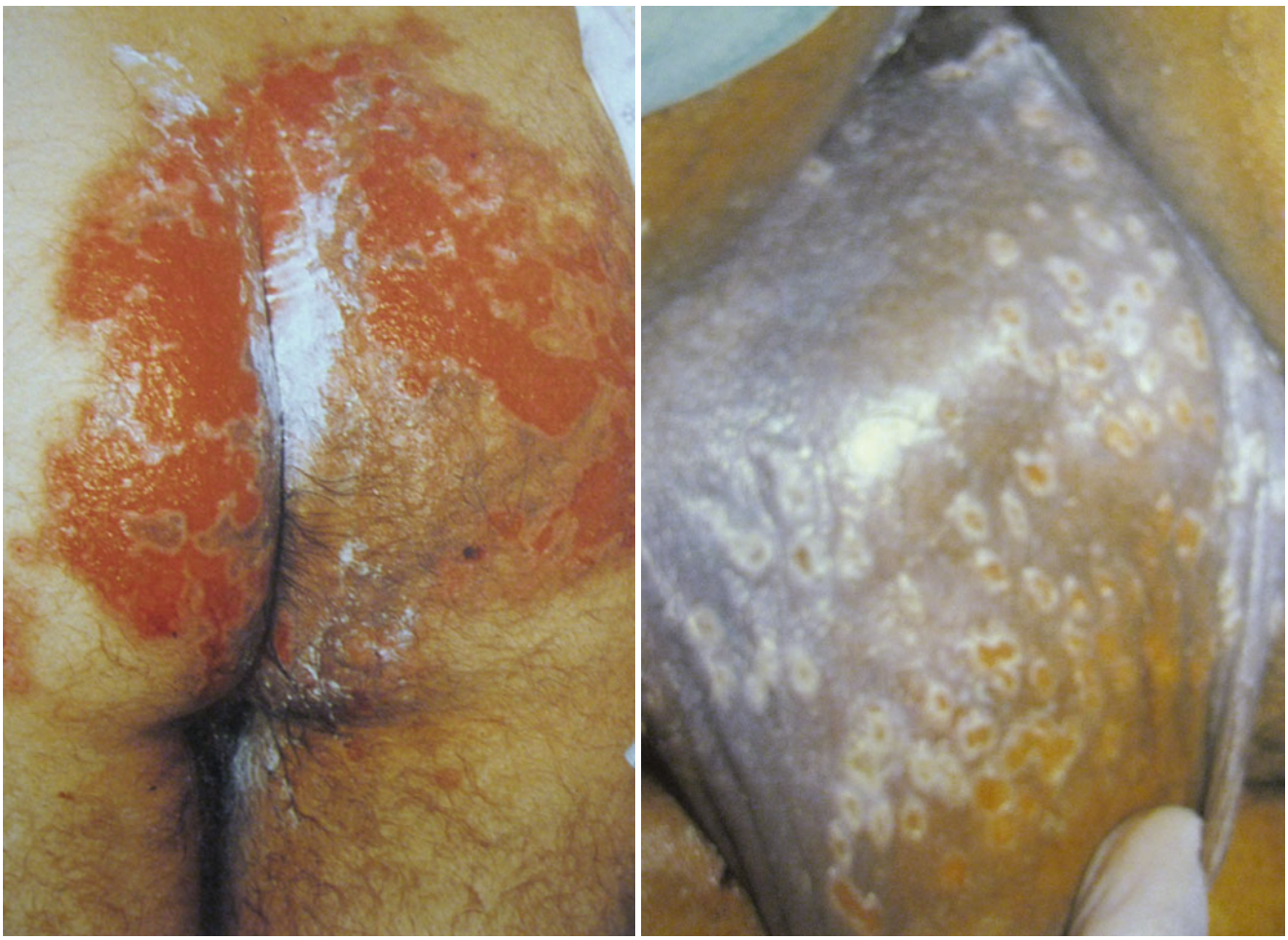


Fig. 21.5 Herpes simplex ophthalmicus, involving the first branch of the trigeminal nerve, can result in dendritic keratitis of the cornea and temporal lobe encephalitis



Figs. 21.6 and 21.7 Severe erosive sacral, gluteal, and scrotal herpes simplex in immunocompromised patients with lymphoma and AIDS

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Herpes Zoster

22

Synonyms

Synonyms for herpes zoster include shingles and zoster.

Clinical Description

Classical lesions consist of a dermatomal distribution of painful grouped vesicles on a red base [1–3].

Special forms include the following:

1. Ramsay-Hunt Syndrome-affects cranial nerves Vll and VIII (facial and auditory) and can lead to facial paralysis, hearing loss, and vertigo
2. Zoster sine herpete-pain without vesicles
3. Hutchinson's sign-vesicles on nose tip signify involvement of nasociliary branch of trigeminal nerve V, ocular zoster must be suspected, and ophthalmologic referral given
4. Other motor nerve syndromes-can result in ocular muscle palsies
5. Postherpetic neuralgia (PHN)-pain beyond 1 month after onset of the rash.

PHN is the most frequent complication of herpes zoster. Its frequency is determined largely by age, and perhaps by immunosuppression. It does not necessarily correlate with the severity of the acute zoster [4, 5].

Etiology and Pathophysiology

Herpes zoster is a localized recurrence of varicella zoster virus infection. In immunocompromised adults, the eruption may generalize. Underlying cancer should be sought, especially cellular immune defects, such as lymphoma, leukemia, and AIDS.

Histopathology and Diagnosis

The histopathology of zoster is the same as for herpes simplex, but viral culture can help differentiate, if necessary. Growth occurs in 3 days for herpes simplex, and in 7 days for varicella-zoster. "Recurrent" zoster is often herpes simplex, although recurrent herpes zoster has been reported.

Prevention

Herpes zoster vaccine (Zostavax, Merck & Co., Inc.) was licensed and recommended in 2006 for prevention of herpes zoster among adults aged 60 years and older. In March 2011, the Food and Drug Administration (FDA) approved the use of Zostavax in adults aged 50 through 59 years. The FDA approved the expanded indication for Zostavax based on a study of approximately 22,000 adults aged 50 through 59 years in the United States and four other countries [6, 7].

Therapy

For acute herpes zoster, the following treatment may be used:

1. High-dose acyclovir 800 mg orally five times daily, for 7–10 days, reduces pain, hastens lesion healing, and may reduce the incidence of postherpetic neuralgia. Available in capsule (200, 800 mg) and elixir (200 mg/5 ml) forms, acyclovir should be taken with sufficient water to minimize the risk of crystallization in the kidney. Valacyclovir is also quite effective, 1 g three times daily for 10 days.
2. The use of early systemic corticosteroids to prevent PHN is controversial. In patients over 50 years of age, who

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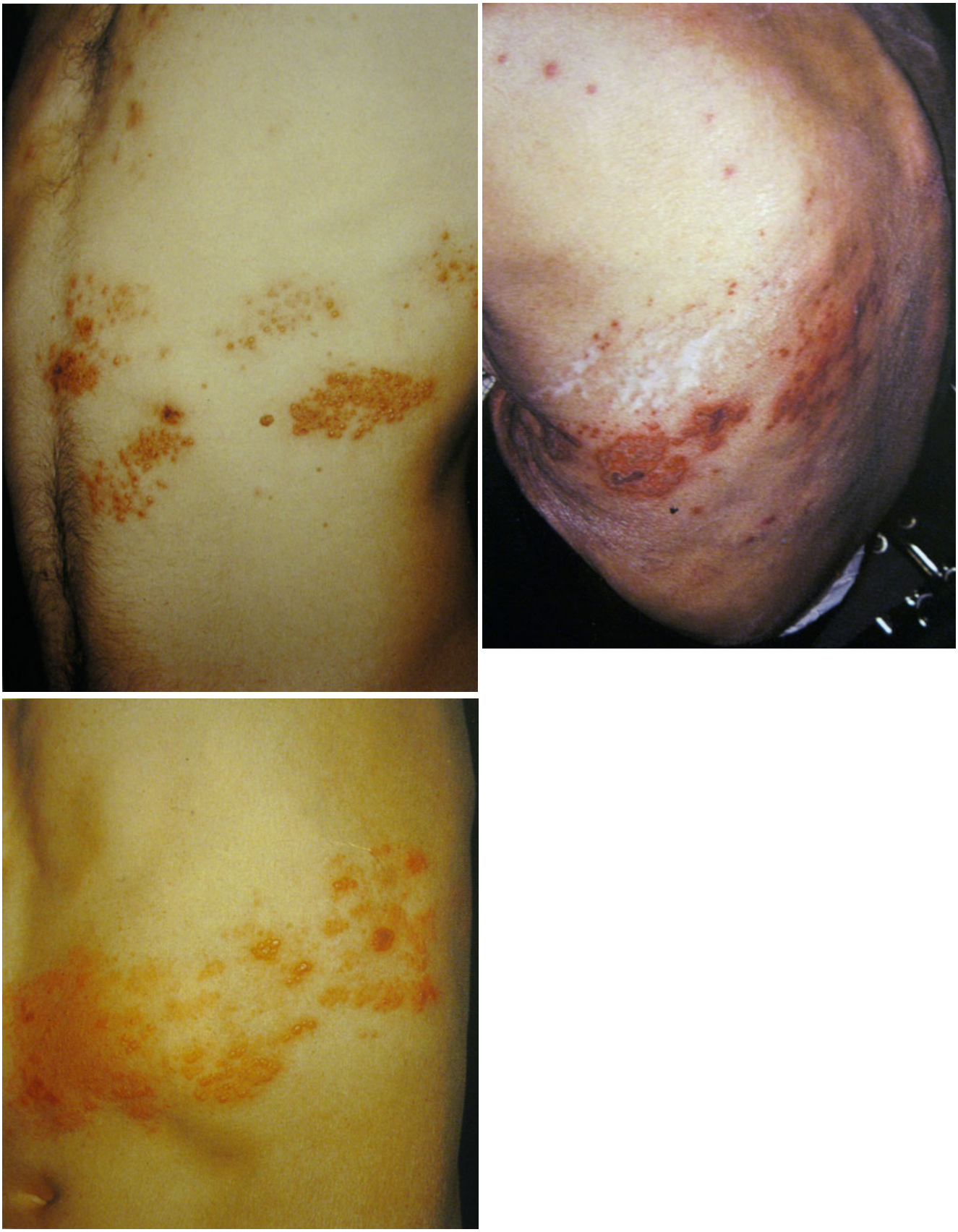
have had zoster for less than 6 days, prednisone taper (60 mg, orally for I week, then 40 mg orally for I week. and 20 mg orally for I week), or triamcinolone acetonide 40 mg 1 M, have been used.

Postherpetic neuralgia, once established, is difficult to treat, but the following may be tried.

1. Analgesics, anticonvulsants (carbamazepine), and antidepressants (amitryptyline) have been used, with varying effectiveness. These drugs must be used with great care in elderly patients because of central nervous system side effects.
2. lntralesional steroids, intralesional anesthetics (lidocaine), and a transcutaneous electrical nerve stimulation (TENS) unit may relieve pain locally.
3. Topical capsaicin cream qid depletes substance P, a chemical mediator of pain.

Prognosis

Postherpetic neuralgia can be a severe, painful problem, occasionally driving patients to suicide.



Figs. 22.1, 22.2, and 22.3 Classic herpes zoster showing a dermatomal distribution on the trunk

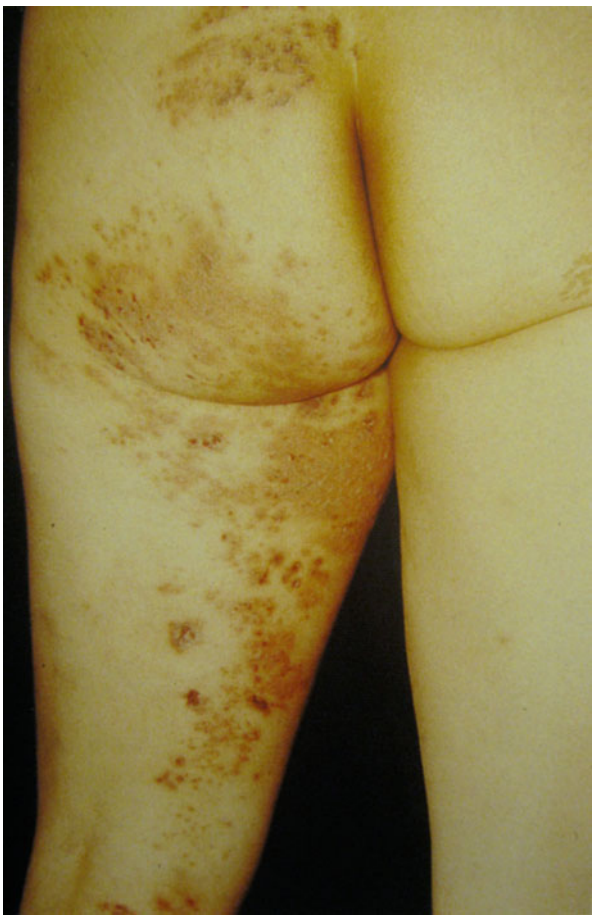
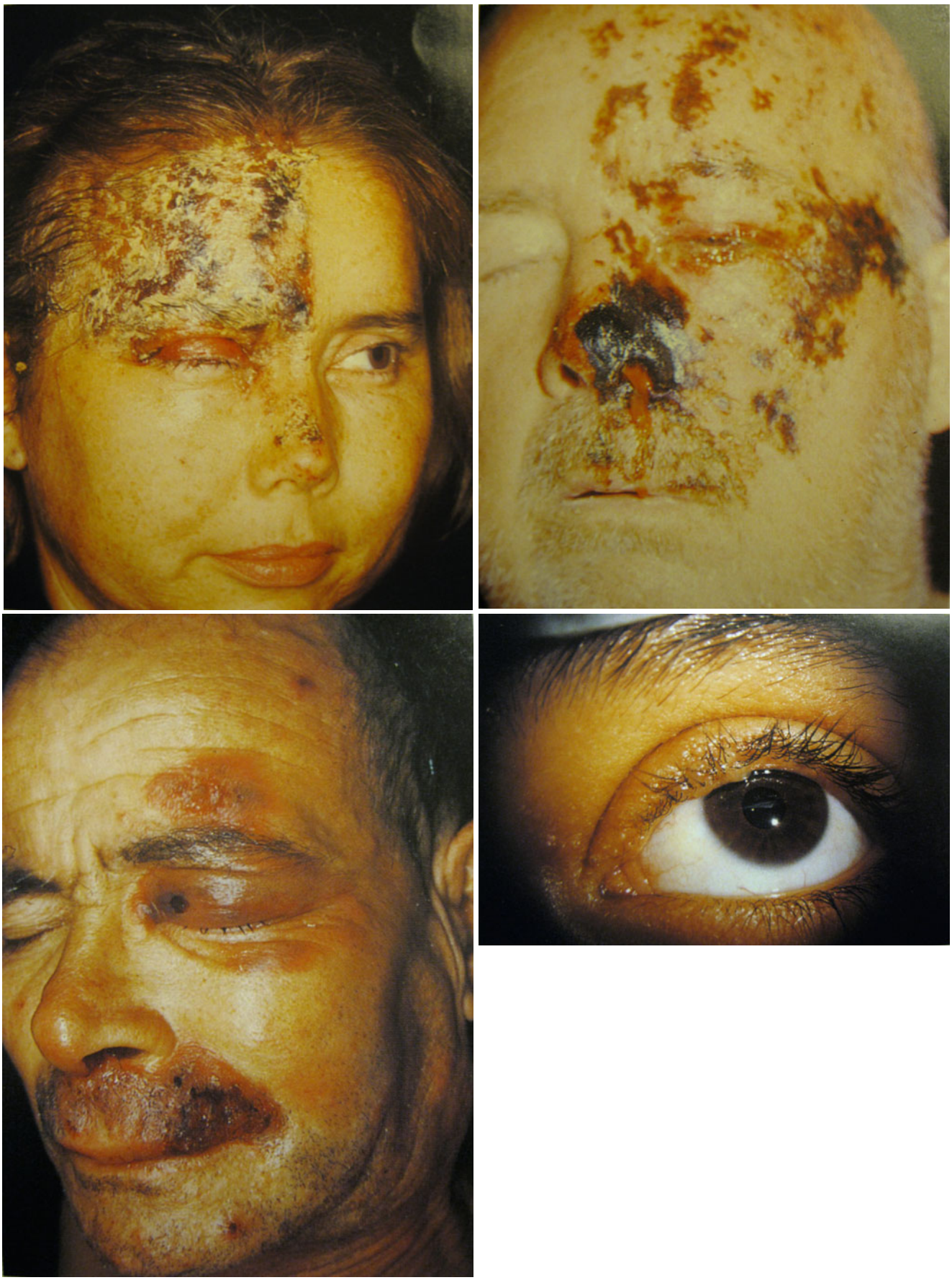


Fig. 22.4 Lumbosacral herpes zoster causes sciatica



Figs. 22.5, 22.6, 22.7, and 22.8 Herpes zoster ophthalmicus from involvement of the first branch of trigeminal nerve V. Note Hutchinson's sign of nasal tip involvement. An ophthalmologic examination is essential

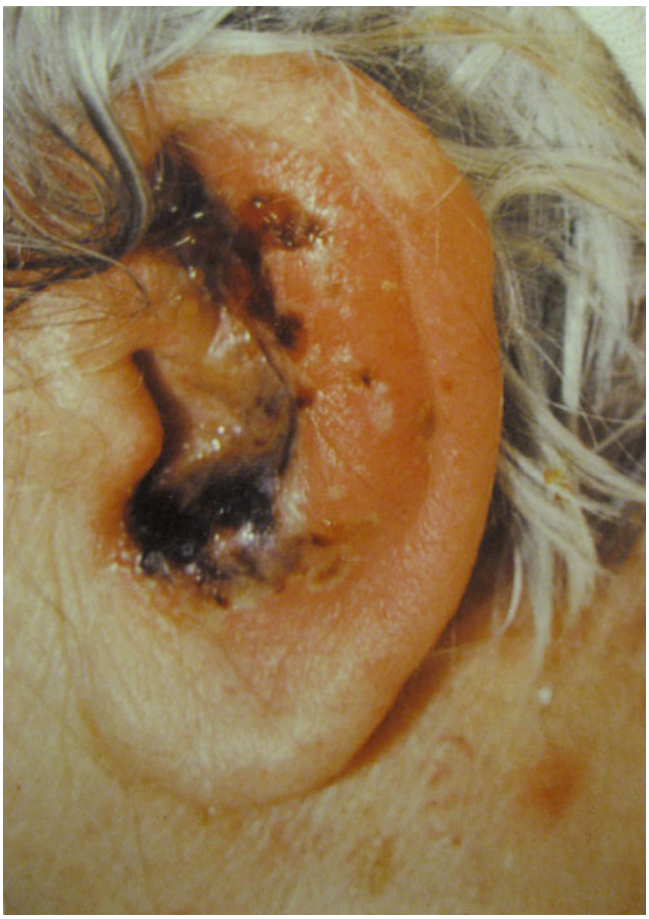
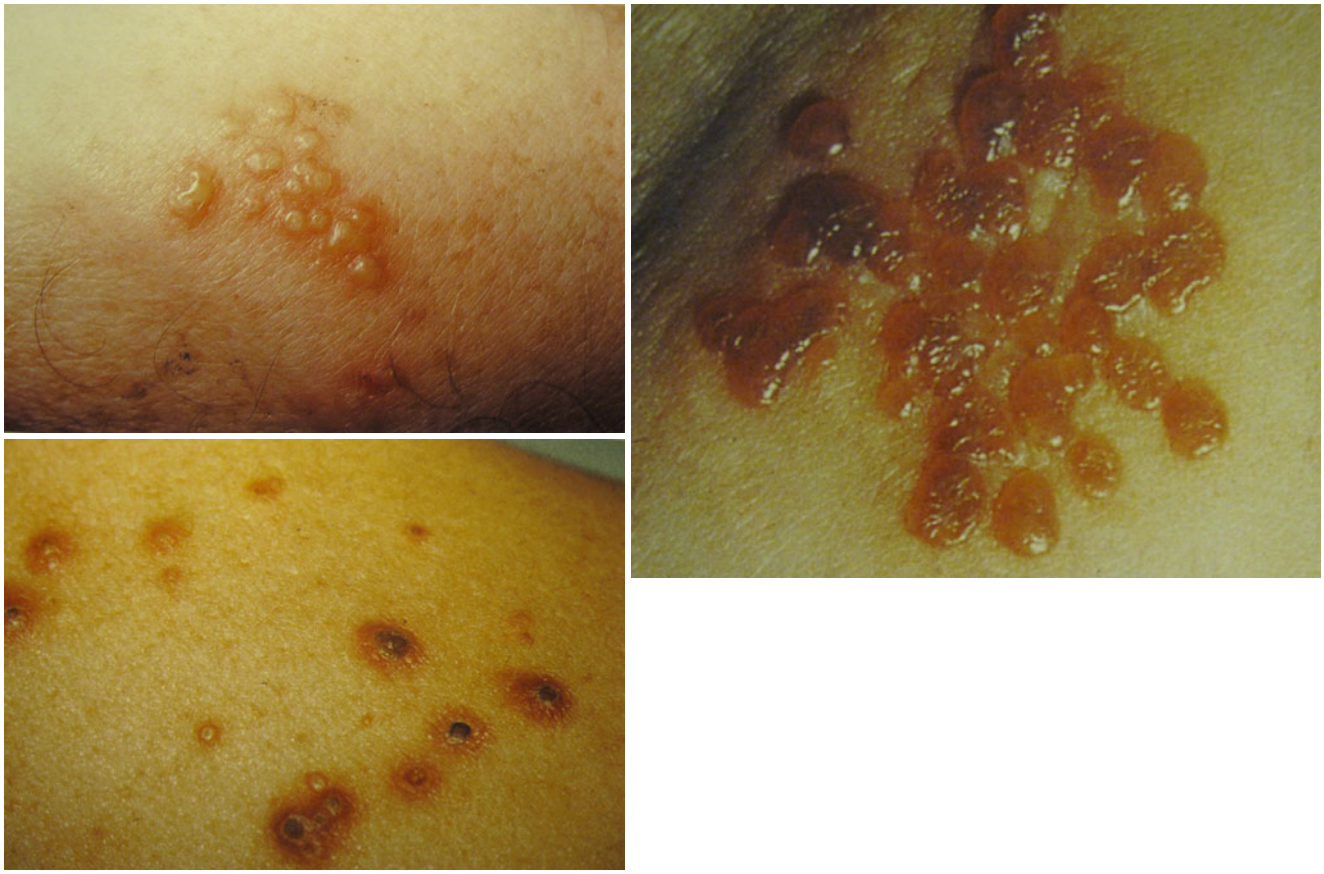


Fig. 22.9 Ramsay-Hunt syndrome involves the facial nerve (cranial nerve VII) and the auditory nerve (cranial nerve VIII). It may cause facial paralysis, vertigo, hearing loss or hypersensitivity, and even deafness



Figs. 22.10, 22.11, and 22.12 Pustular and hemorrhagic changes occur in vesicles in older lesions of zoster

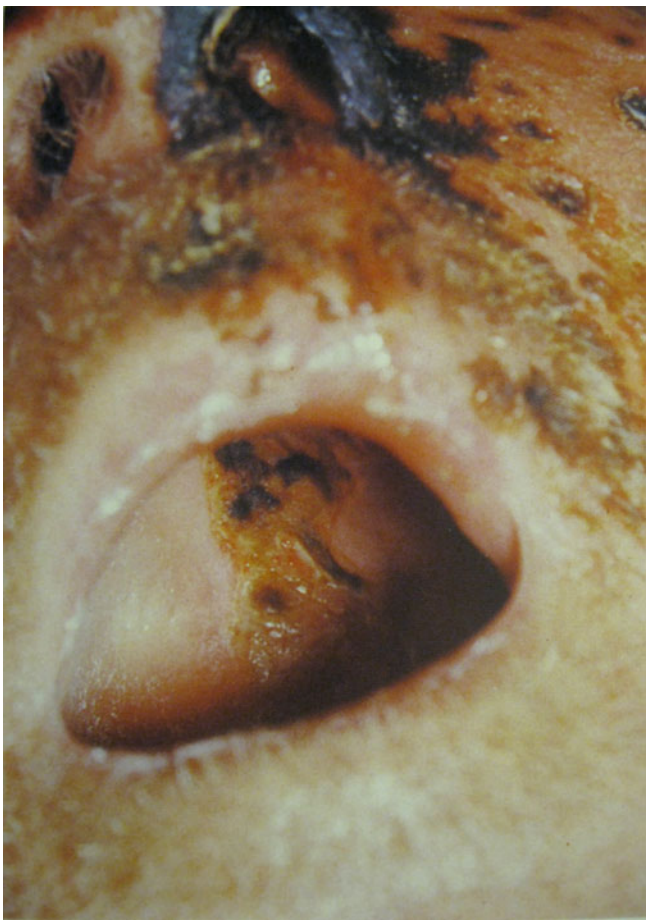


Fig. 22.13 Gangrenous necrotic zoster involving the palate in a leukemia patient

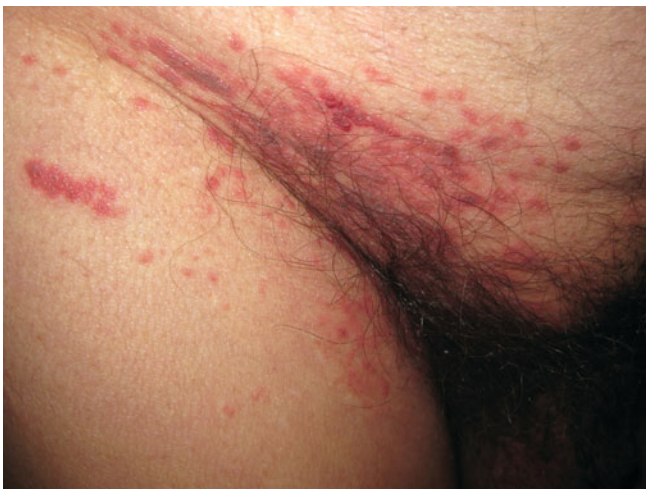


Fig. 22.14 Herpes zoster in inguinal region

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Scabies and Lice

23

Scabies

Synonyms

Synonyms for scabies included 7-year itch and nursing home dermatitis.

Clinical Description

Linear burrows, crusted excoriations, and erythematous papulovesicles typically occur on finger webs, volar wrists, genitals, breasts, and buttocks. Crusted, keratotic, or "Norwegian" scabies is a severe, generalized form that clinically resembles atopic dermatitis in immunocomprised patients; lesions consist of diffuse scaling, erythema, and nail changes, including subungual hyperkeratosis. Persistent postscabetic nodules occur as red-brown domed papules and nodules that resemble those of cutaneous lymphoma.

Etiology and Pathophysiology

Sarcoptes scabiei mite infestation results in an allergic reaction after an incubation period of up to 6 weeks. Symptoms of reinfestation develop more quickly, within 24–48 h. This variable time course complicates the diagnosis of asymptomatic carriers, so all close contacts should be treated simultaneously.

During the last 20 years, the number of patients infected by scabies has been increasing. Scabies has caused major problems in nursing homes, particularly in debilitated patients. The risk factors for infection with scabies among nursing homes include age of the institution (more than 30 years), size of the institution (more than 120 beds), and the ratio of beds to health care workers (more than 10:1) [1–6].

Histopathology

Burrows demonstrate an intraepidermal tunnel containing female mites, ova, feces, and/or immature mites. Papules show a perivascular lymphohistiocytic infiltrate with eosinophils, Occasionally, the activated lymphocytes and clinical presentation of postscabetic nodules closely resemble those of cutaneous lymphoma.

Diagnosis

Skin scraping (full-thickness epidermal shave biopsy coated with mineral oil) of several burrows may reveal female mites, ova, or fecal pellets. The areola in females and genitals in males are common locations of infestations.

Differential Diagnosis

The differential diagnosis of scabies includes the following:

1. Other types of arthropod bites-mosquitoes, lice, fleas, spiders
2. Urticaria, dermatitis herpetiformis, pediculosis, delusions of parasitosis, metabolic pruritus, impetigo, ecthyma, furunculosis, Darier's disease, prurigo nodularis, vasculitis, seborrheic dermatitis, contact dermatitis, papular urticaria, impetigo, recurrent pyoderma, drug adverse reaction

Therapy

1. Ivermectin, repeated in 1 week
2. Permethrin 5 % cream is applied and left on for 8–12 h and repeated in 1 week.

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3. Precipitated sulfur, 2–5 % in petrolatum can be used for infants, pregnant women, and elderly patients [7–9].

Prognosis

The prognosis is good if the epidemic can be eradicated by simultaneous treatment of all close contacts, including medical personnel. Recurrences can occur. Exposure does not prevent the patient from becoming reinfested.

Lice

Synonyms

Synonyms for lice infestation include pediculosis capitis (head lice), pediculosis corporis, or "Vagabond's disease" (body lice), and pediculosis pubis (crab lice, pubic lice).

Clinical Description

1. Pediculosis capitis-mobile, elongate louse and immobile nits (eggs) attach to scalp hair. Head lice affect those at all levels of society and most ethnic groups, but are rare in North American blacks.
2. Pediculosis corporis-punctate inflammatory papules and/or blue macules (maculae ceruleae) occur at sites of bites. Elongate lice or nits are found in seams of clothing.
3. Pediculosis pubis-mobile, round, squat lice and immobile nits attach to pubic hairs. Rarely, eyelash and axillary hairs are also involved.

Etiology and Pathogenesis

Three species of lice affect humans: Pediculus humanus capitis (head louse). P. humanus corporis (body or clothing louse), and Phthirus pubis (crab louse).

1. P. capitis may be acquired by fomite transfer with infestation of lice on caps, hats, and combs.
2. P. corporis is associated with poor personal hygiene.
3. Phthirus pubis is usually venereally (sexually) acquired, but may occasionally be transferred by infested fomites (clothing or furniture).

Diagnosis

Lice and/or nits should be sought on the scalp, axillary, or pubic hair, and in clothing seams

Histopathology

Histopathologic investigation is not routinely performed.

Therapy

Treatment of pediculosis capitis includes the following:

1. A synergized pyrethrin is applied to the wet scalp for 10–30 min and repeated in 7–10 days.
2. A synthetic pyrethoid (1 % permethrin cream rinse) is applied after shampooing to towel-dried hair for 10–30 min, and then rinsed.
3. Nits can be loosened with a vinegar rinse or with 8 % formic acid left on for 5–10 min, and followed by fine-toothed combing.
4. Clothing and fomites (e.g., headgear, towels) should be machine-washed, dry-cleaned, or stored in plastic bags in a warm place for 2 weeks to kill lice and nits.
5. Newer treatments include malathion lotion and benzyl alcohol lotion

Pediculosis pubis is treated similarly to P. capitis:

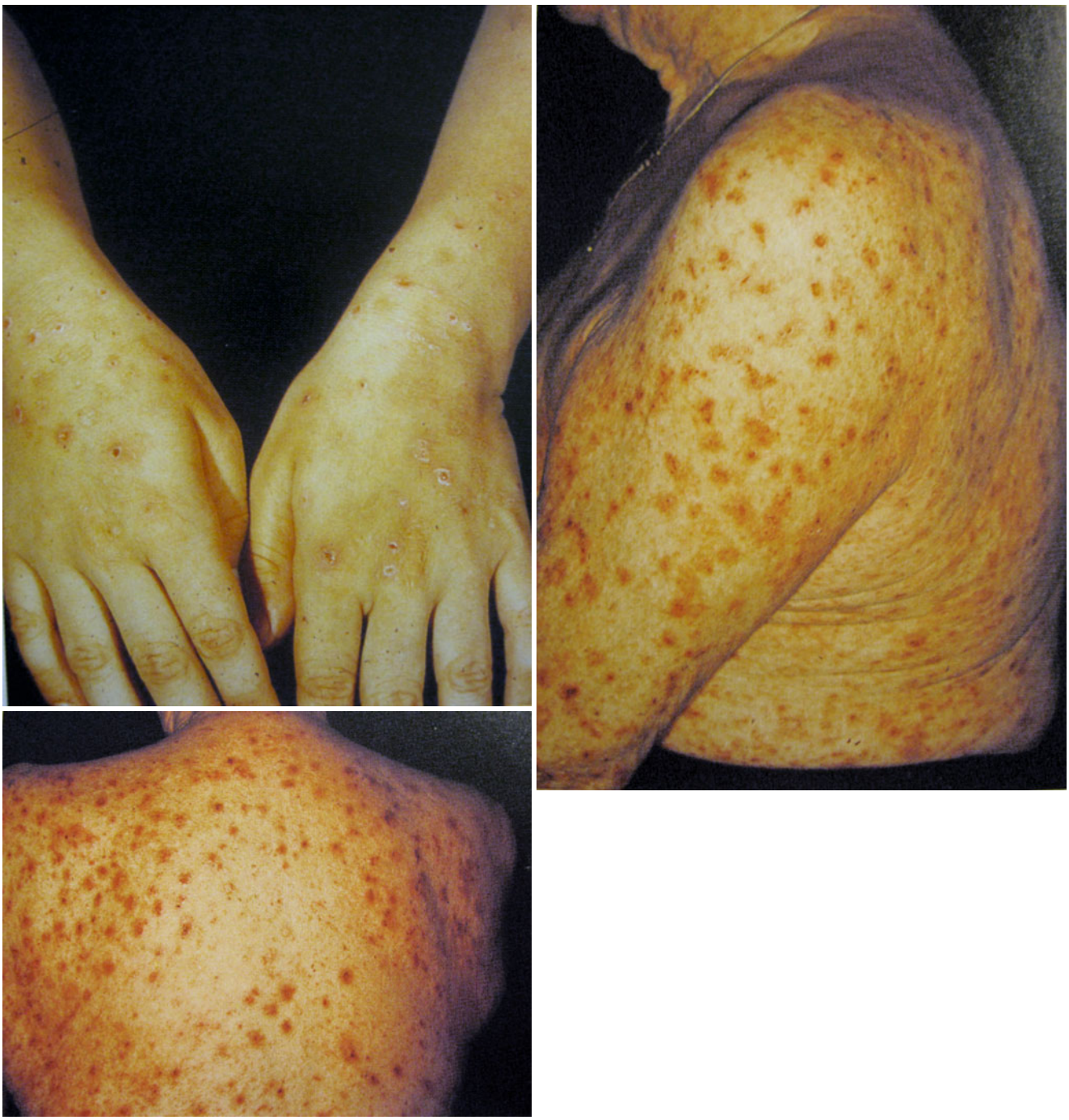
1. A synergized pyrethrin shampoo is applied for 10 min.
2. Involved eyelashes are treated with petrolatum, bid for 8 days, followed by removal of any nits.

Pediculosis corporis treatment includes the following:

1. Proper hygiene, bathing, clean underwear. and good nutrition are essential. Clothes should be dry-cleaned or laundered with boiling water. Special attention should be paid to clothing seams [10, 11].

Prognosis

The prognosis is good to excellent, but recurrences are common and do not prevent the patient from becoming re-infested.



Figs. 23.1, 23.2, and 23.3 Multiple excoriated scabetic papules on arms and trunk

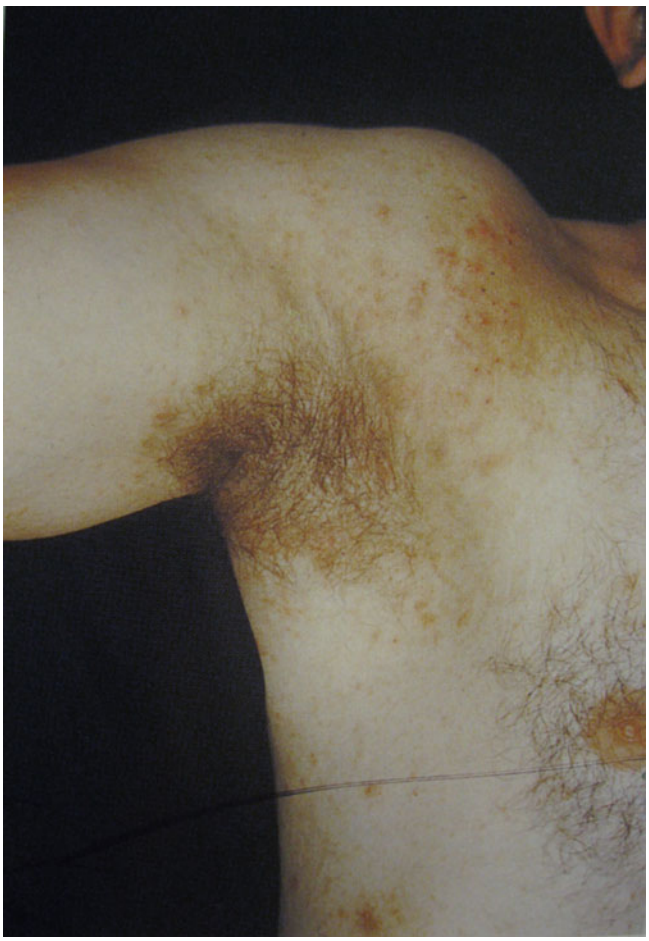


Fig. 23.4 Axillary scabies

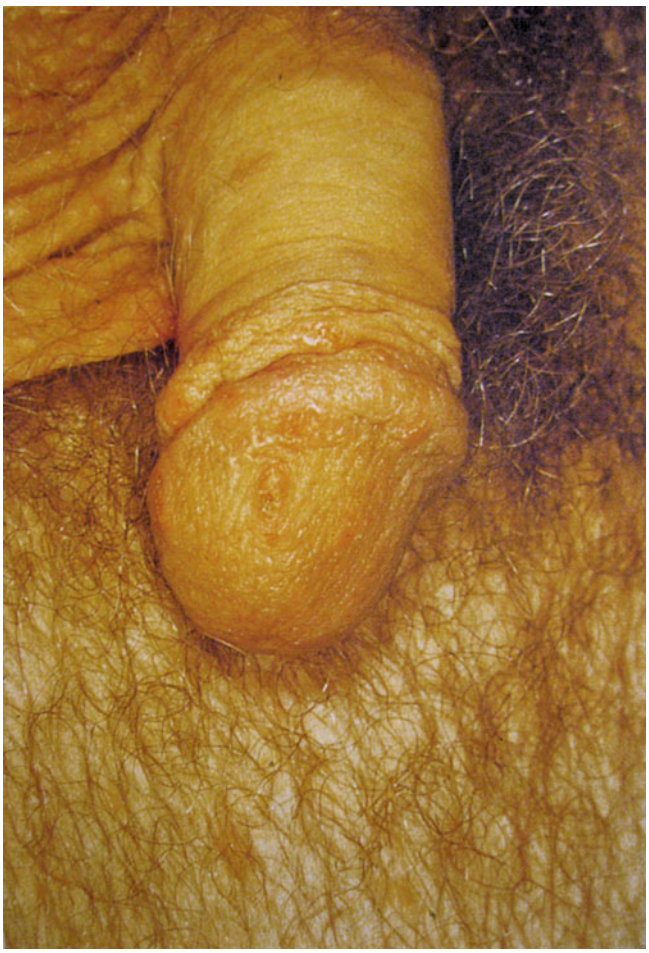
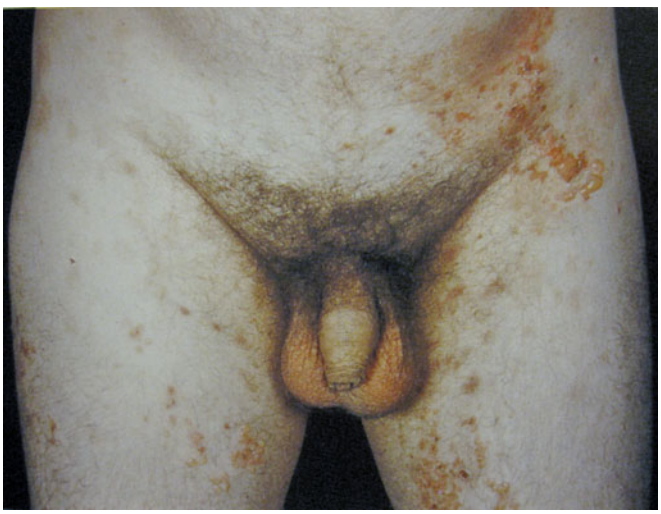
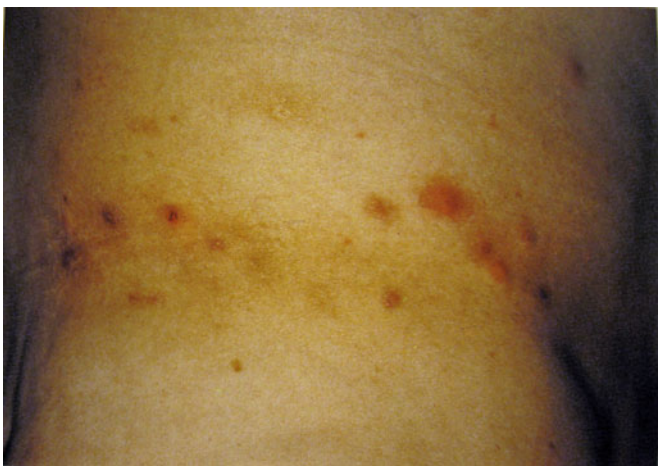
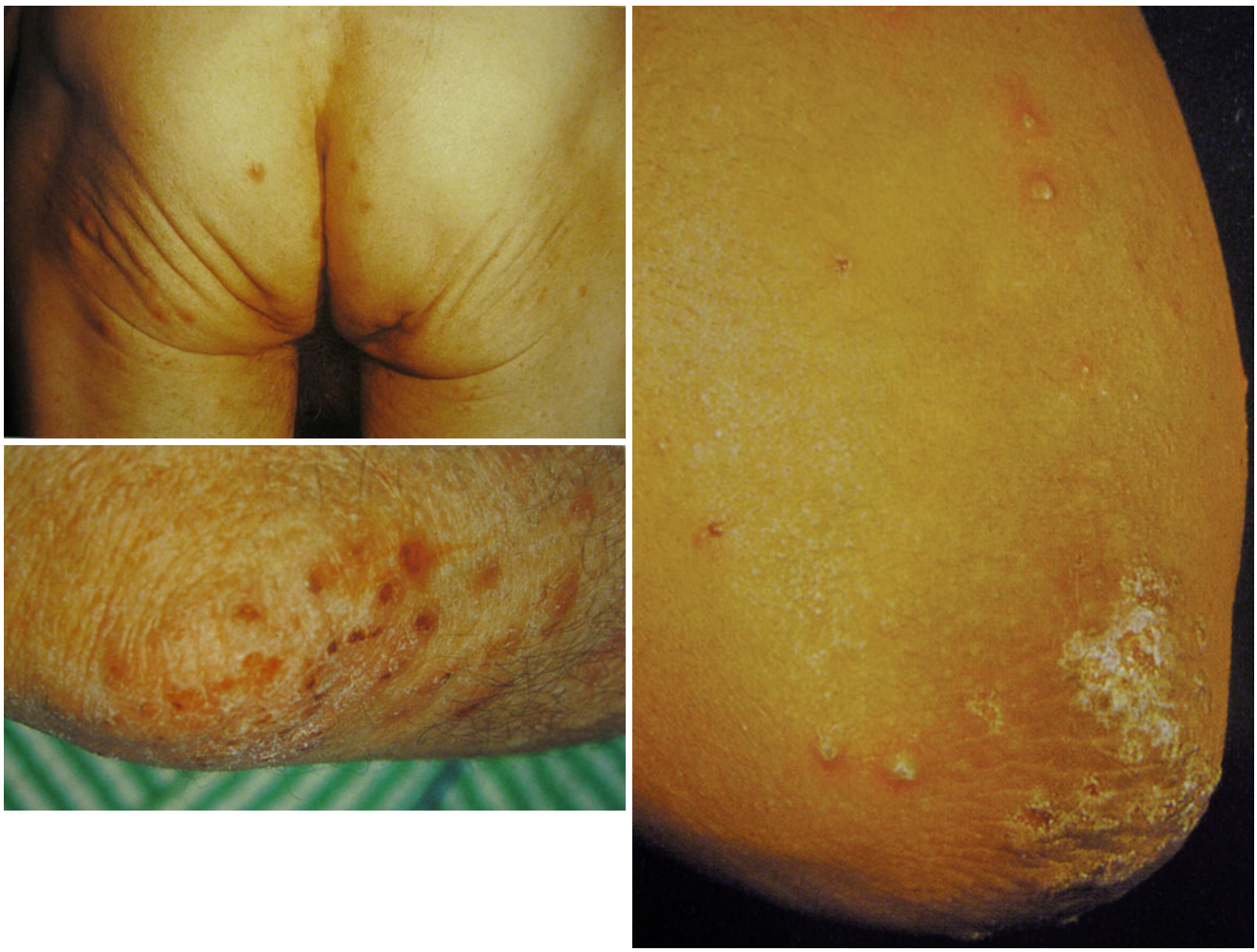


Fig. 23.5 Linear scabetic burrow on glans penis

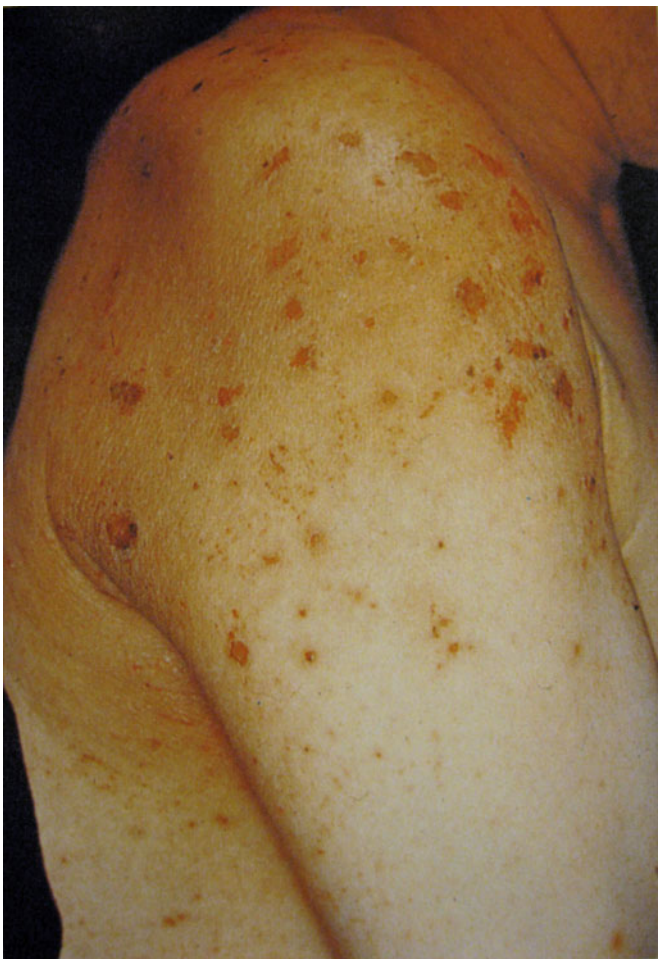


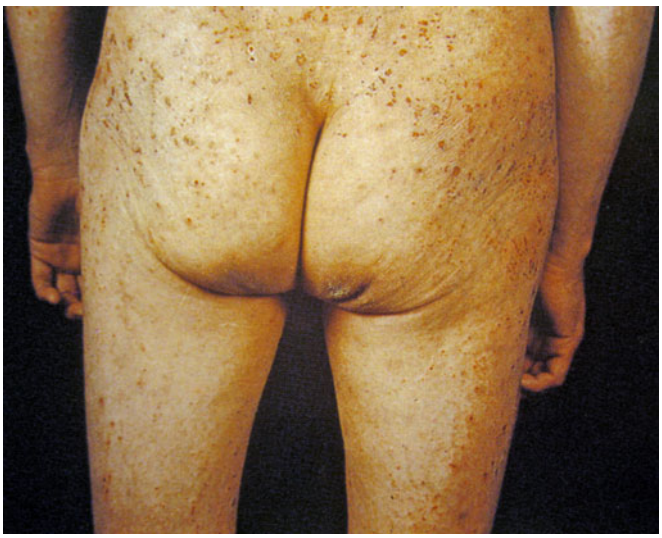


Figs. 23.6 and 23.7 Multiple red-brown scabetic nodules are extremely pruritic



Figs. 23.8, 23.9, and 23.10 Excoriated papulovesicles on sacrum, buttocks, and elbows can be seen in scabies and in dermatitis herpetiformis





Figs. 23.11 and 23.12 Lice bites

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Warts

24

Synonyms

Synonyms for warts include verrucae and human papilloma virus (HPV) infection.

Clinical Description

Warts are most common in children, but can occur at any age. The clinical appearance of warts varies with location [1–7]

1. Flat warts-face, dorsal hands, and legs
2. Filiform (filamentous) warts-neck, lip, eyelid
3. Papular, exophytic, "common" warts-fingers, hands
4. Condylomata acuminata-caullflower-like vegetative red-gray-brown papules on genitals, gluteal cleft
5. Mosaic endophytic warts-sole (plantar) warts.

The Koebner phenomenon refers to the spreading of warts by scratching or other trauma

Etiology

Warts are caused by various human papillomaviruses (over 100 types) [3].

Histopathology

Vacuolated granular cells with basophilic nuclei are histologic changes characteristic of all human papillomavirus infections.

Differential Diagnosis

The differential diagnosis includes the following:

1. Stucco keratoses. These gray-brown-white, flat-topped verrucous papules on the legs and ankles represent a variant of seborrheic keratosis
2. Condylomata lata of secondary syphilis. These are broader (lata) and less pointed (acuminata).
3. Callus (corn, clavus). These may resemble plantar warts, but skin markings are preserved in contrast to warts.
4. Neurotrophic corn. This represents an end-stage callus. It displays punctate, thrombosed capillaries. but has an atrophic, painful center caused by pressure-induced compromise of blood supply and subsequent epidermal thinning with loss of skin markings.

Therapy

The elderly patient must be carefully checked for adequate blood supply and nerve function. Elderly patients do not have normal protective pain warning systems. Familiarity with all forms of treatment is especially important in the management of the elderly patient.

The human papilloma virus (HPV) vaccine prevents infection with certain species of human papillomavirus associated with the development of cervical cancer and genital warts. Two HPV vaccines are currently on the market [8, 9].

Note that no uniformly successful therapy exists; no oral medication or vaccine is currently available to suppress or prevent common warts. The following may be tried [10–14]:

1. Surgery-paring, extraction, laser ablation. The patient should be cautioned regarding possible scarring, recurrence, and discomfort.
2. Chemicals-keratolytics, lactic and salicylic acid solutions, gels, plasters, pads.
3. Antimitotics-topical podophylline tincture, intralesional bleomycin. These are administered by the physician in the office. Podofilox 0.5 % topical solution has been approved for use by the patient on genital warts. This prescription product is applied twice daily for three consecutive days, followed by 4 days of no treatment until no visible genital wart remains, up to a total of 4 weeks.
4. Vesicants-cantharidin is highly caustic but can be effective.

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5. Immunotherapy-oral cimetidine boosts T-cell function; topical DNCB (dinitrochlorobenzene) and squaric acid dibutyl ester induce contact sensitization. Imiquimod may also be used [10].
6. Colposcopy [11] followed by laser eradication of subclinical genital warts
7. Liquid nitrogen cryotherapy is simple and effective, but it is also painful and can produce a ring of warts around the treatment site
8. Intralesional interferon [14] has been used for recalcitrant genital warts [16].

Prognosis

Human papilloma virus has been determined to cause cervical dysplasia and cancer in women and bowenoid papulosis and squamous cell cancer of the penis in men. Many warts resolve spontaneously in 1–2 years. Some are stubbornly recurrent, especially periungual and plantar warts, and should be biopsied to rule out verrucous squamous cell carcinoma.

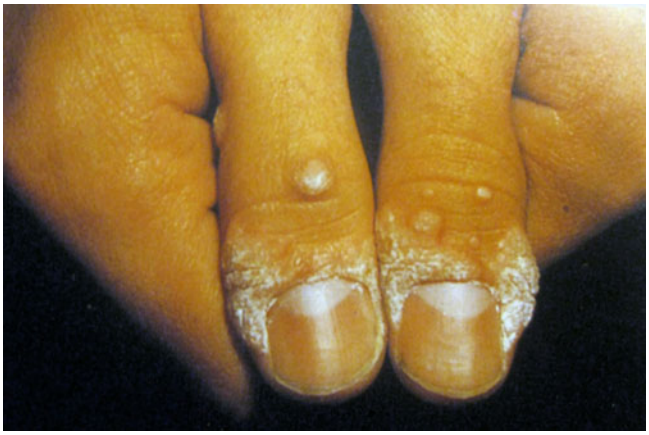


Fig. 24.1 Periungual warts may invade beneath the nails

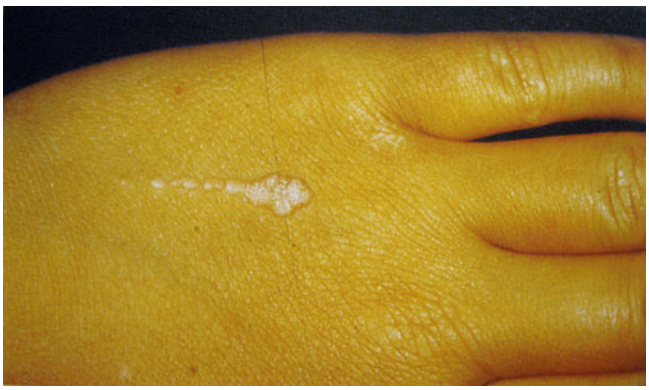
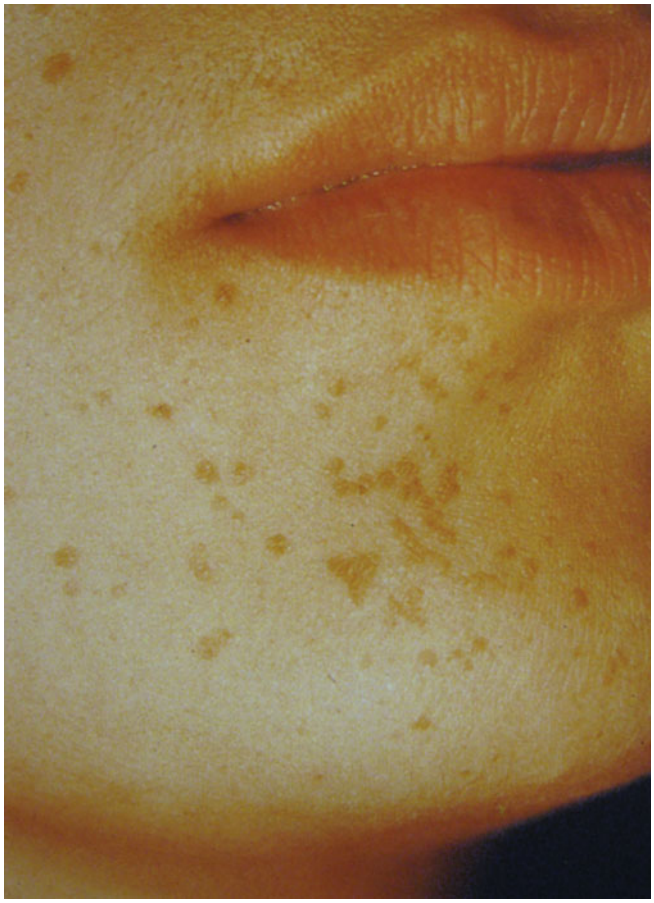
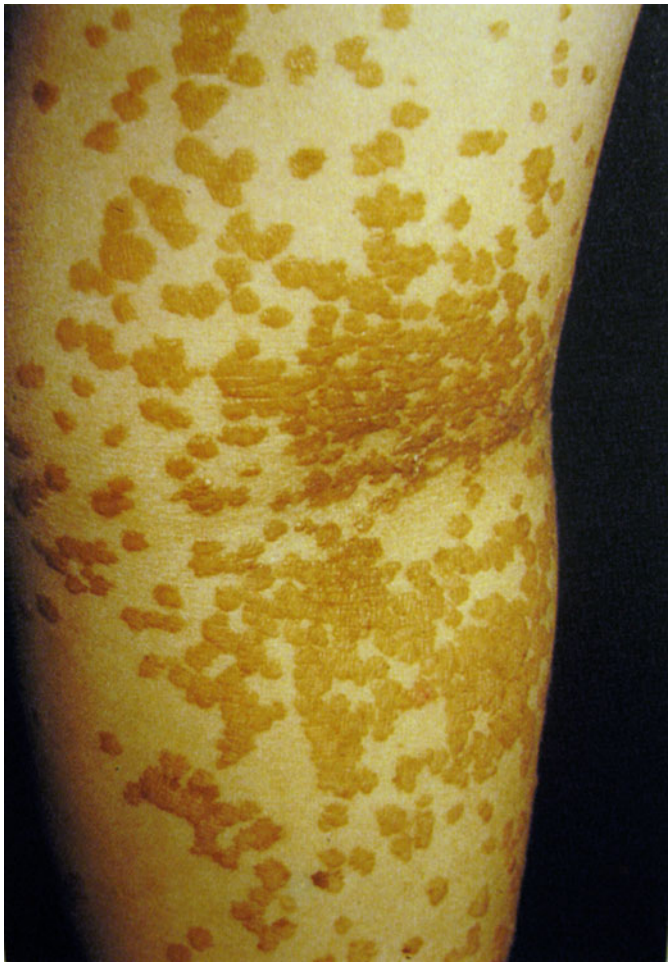


Fig. 24.2 Flat warts spread by scratching (Koebner phenomenon)





Figs. 24.3 and 24.4 Multiple facial flat warts are often spread by shaving

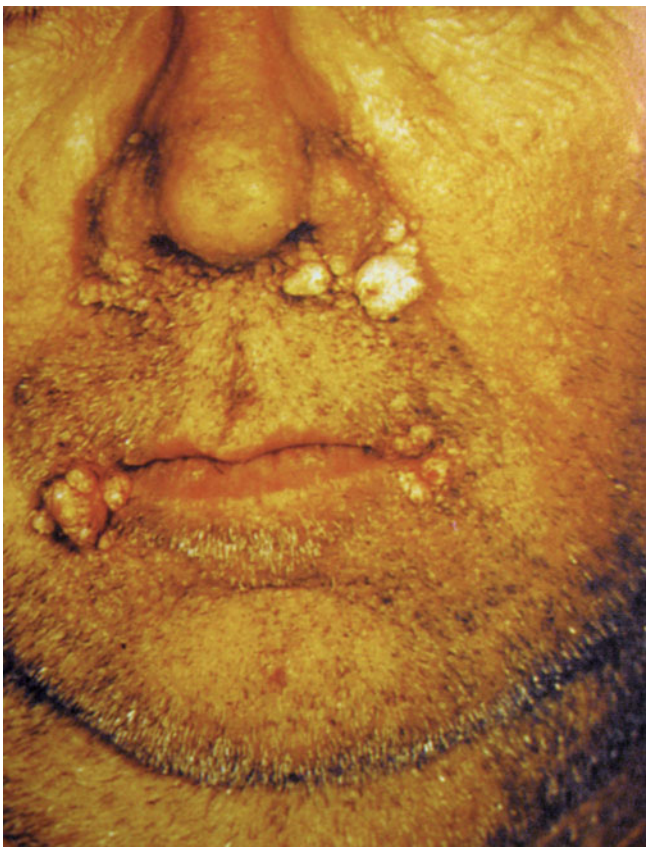
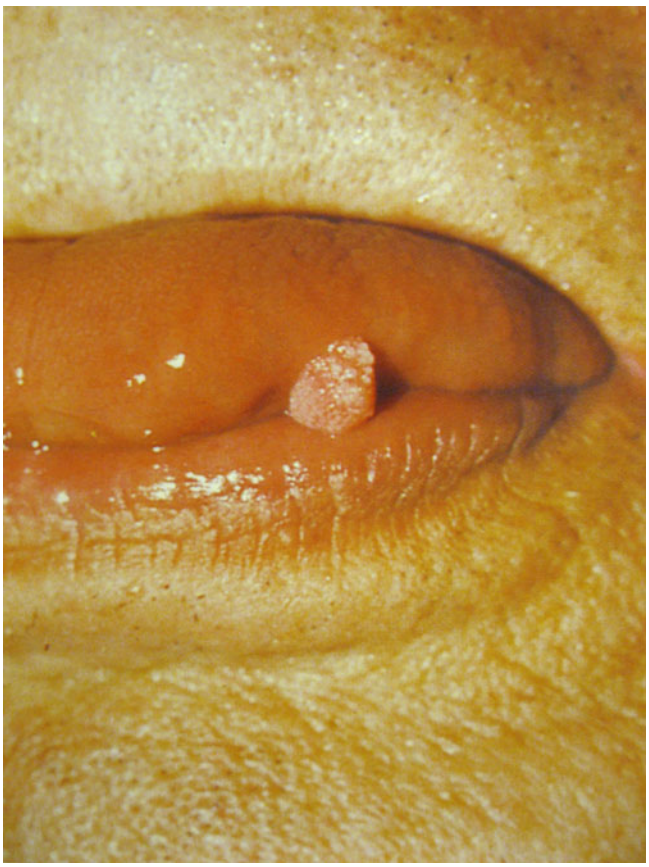
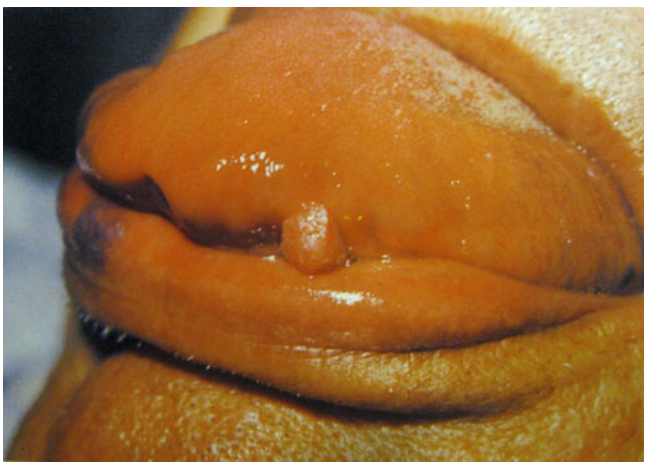


Fig. 24.5 large, atypical facial warts in an immunocompromised leukemic





Figs. 24.6 and 24.7 Filiform (filamentous) wart on lower lip

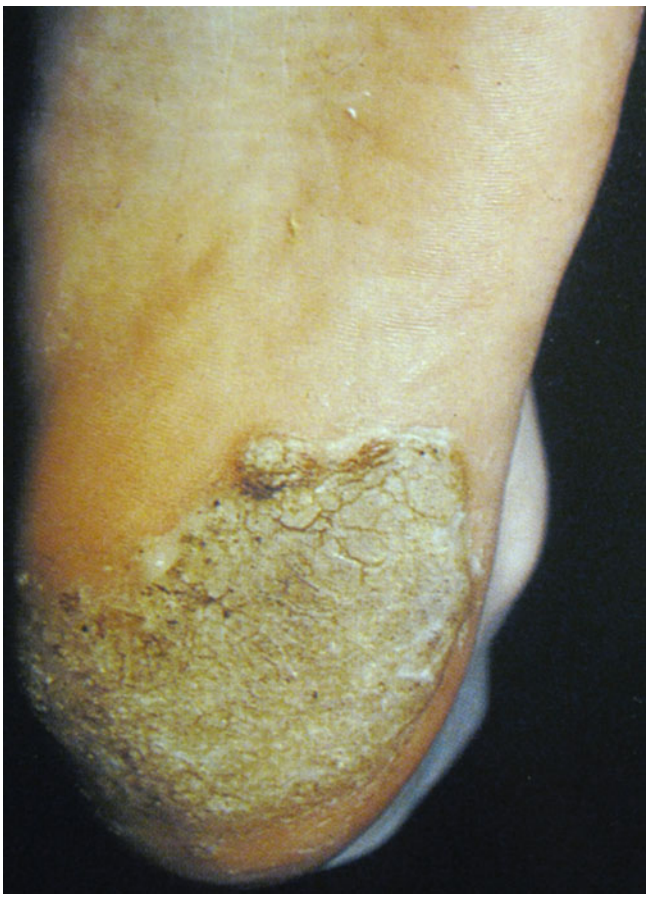
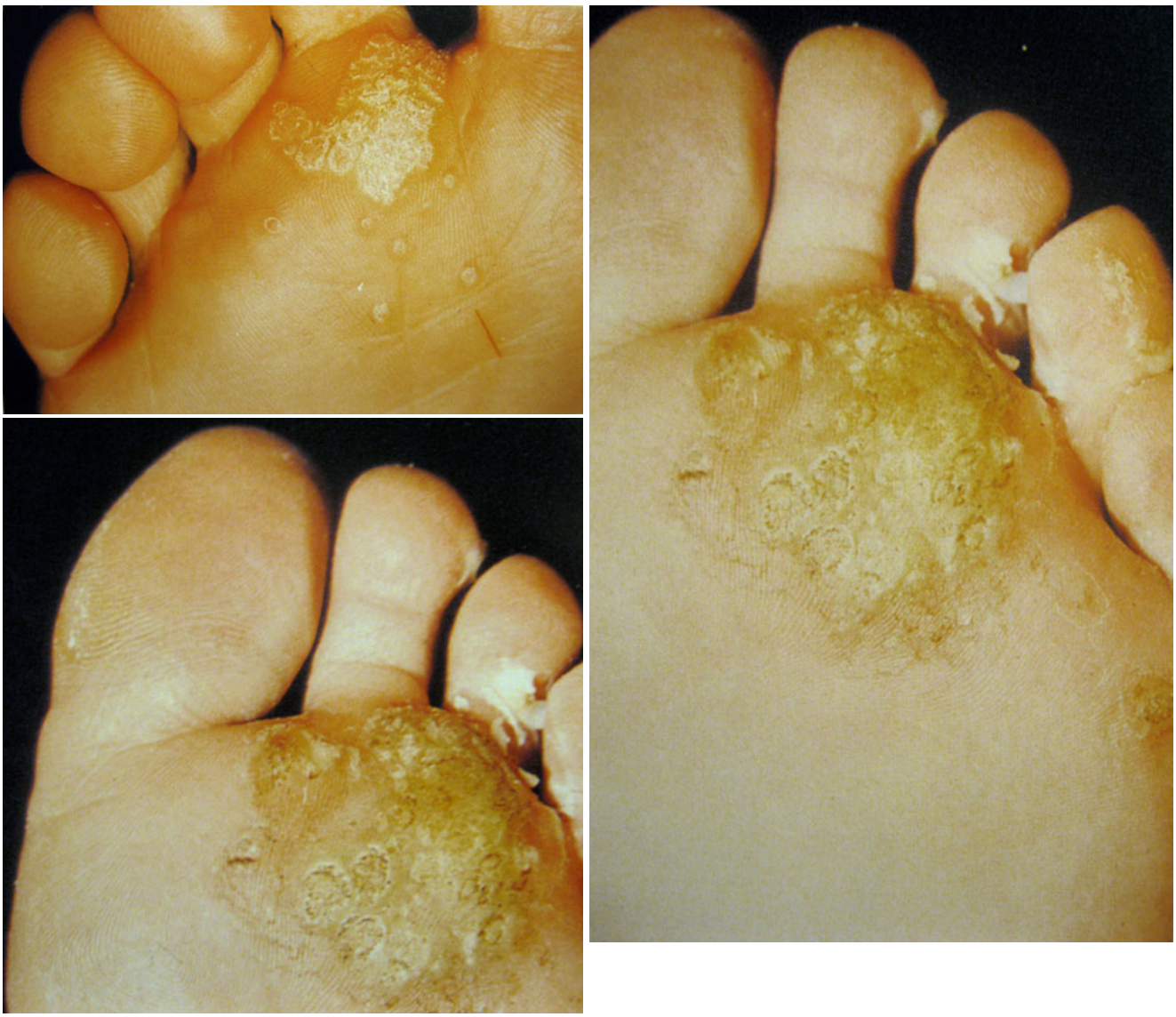
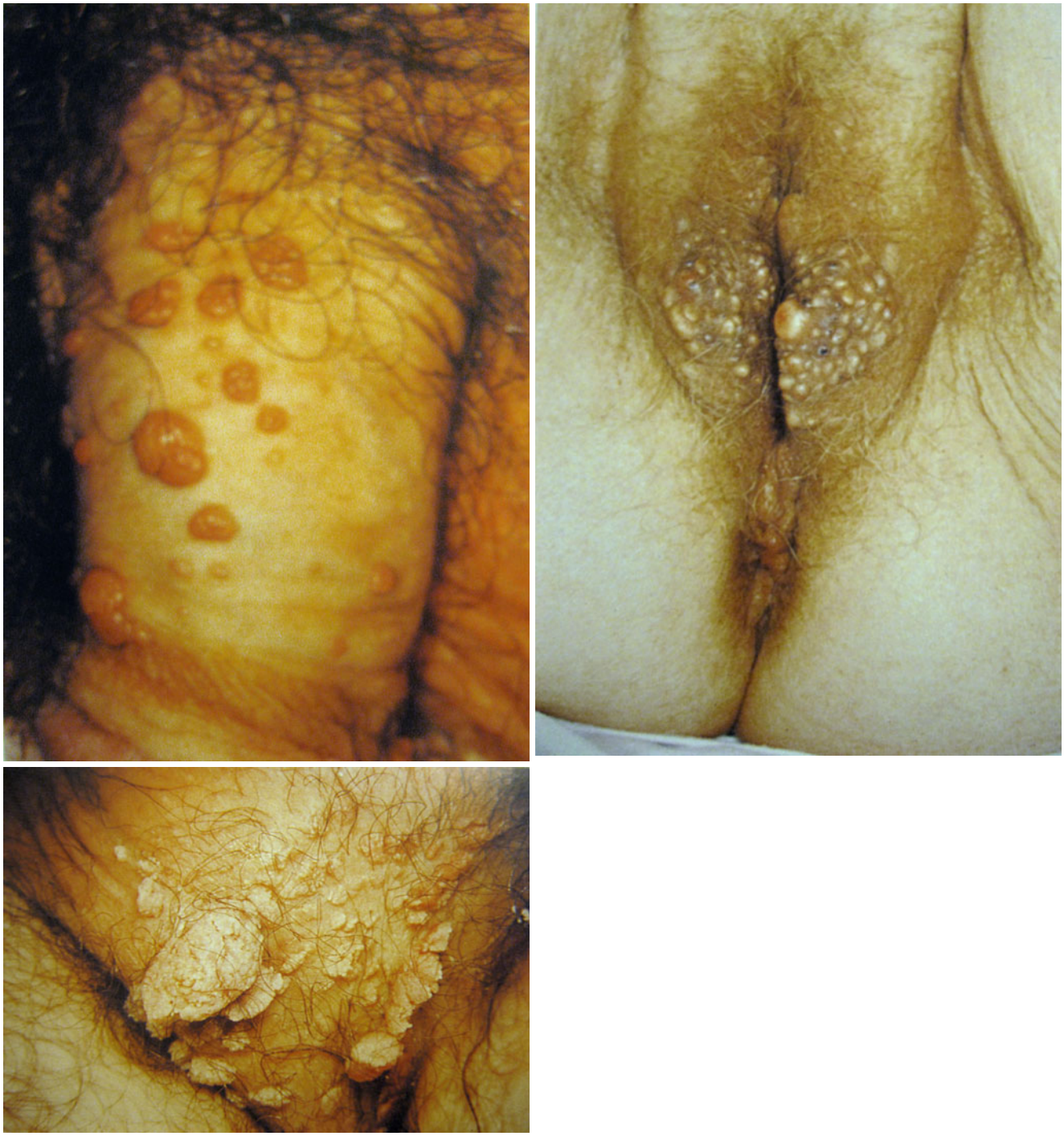


Fig. 24.8 Mosaic plantar wart



Figs. 24.9, 24.10, and 24.11 Mosaic plantar warts



Figs. 24.12, 24.13, and 24.14 Multiple condylomata acuminata (genital or venereal warts)

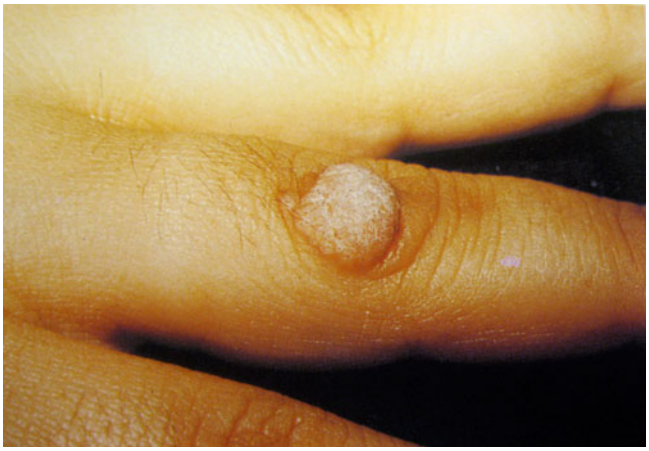


Fig. 24.15 Common single wart on finger. Compare with Fig 24.16

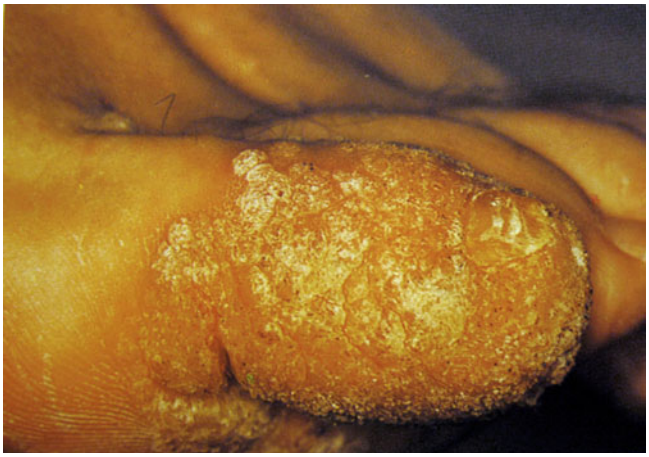


Fig. 24.17 Aggressive, recalcitrant "wart" on toe should be biopsied to rule out squamous cell carcinoma

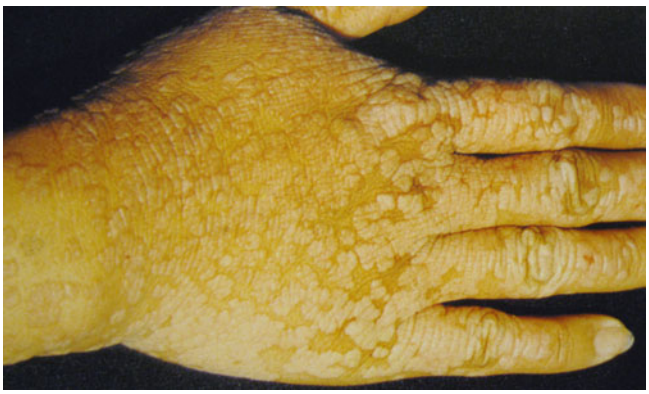


Fig. 24.16 Multiple hand warts in an immunosuppressed patient. Dinitrochlorobenzene topical immunotherapy and oral cimetidine are useful treatments

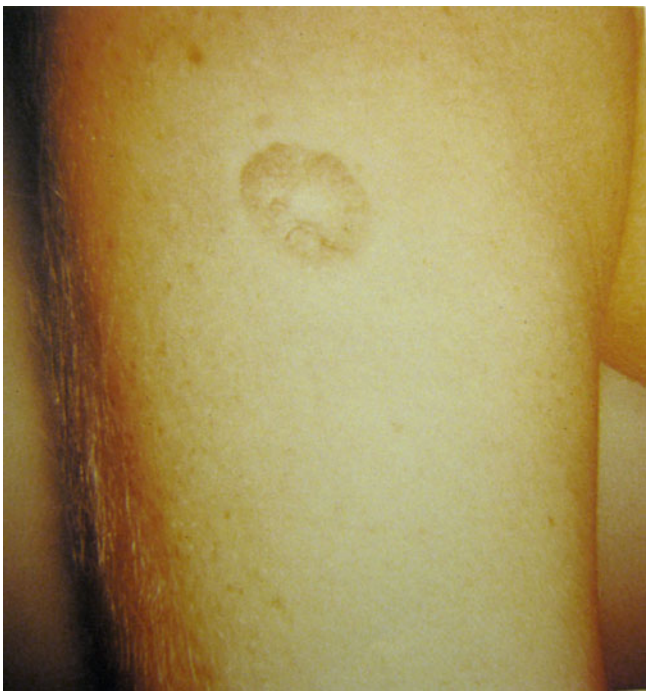


Fig. 24.18 Ring of warts followed liquid nitrogen cryotherapy that induced a blister filled with wart virus. This possible complication must be discussed before considering cryotherapy

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Part IV

Skin Signs in Systemic Disease

Acrochordons

25

Synonyms

Synonyms for acrochordons include fibroepithelial polyps, skin tags, and fibroma molle.

Clinical Description

Acrochordons are soft, flesh -colored or brown filiform or dome-shaped pedunculated papules, occasionally fleshy nodules, that occur in intertriginous areas of the neck, axillae, groin, and inframammary creases. They measure from 0.5 to 6 mm, but can be as large as 2 cm. Acutely thrombosed acrochordons present as suddenly changing, painful red or black papules

Etiology and Pathophysiology

Acrochordons are seen in obese patients, in association with endocrinopathies such as acanthosis nigricans and acromegaly, metabolic syndrome, in sites of skin friction and trauma, and with increasing incidence in the aged. Patients with multiple tags may have associated colonic polyps [1–5] and should be evaluated for colon cancer. A serum growth hormone-like factor may be responsible for both [6]. In certain cases, a metabolic profile should be checked [7–10].

Histopathology

Histopathologic investigation reveals epidermal and dermal papillomatosis with loose vascular and connective tissue. Some tags display nevomelanocytic cells and are histologically melanocytic nevi.

Differential Diagnosis

The differential diagnosis includes filiform warts, exophytic melanocytic nevi, and small seborrheic keratoses.

Therapy

No treatment is necessary, except for cosmesis and/or convenience. Surgical removal is easily accomplished by snip excision or light cautery. Colonoscopy may be advisable, especially if a family history of colon cancer exists.

Prognosis

Lesions persist and may slowly enlarge. Continued trauma can lead to painful thrombosis.

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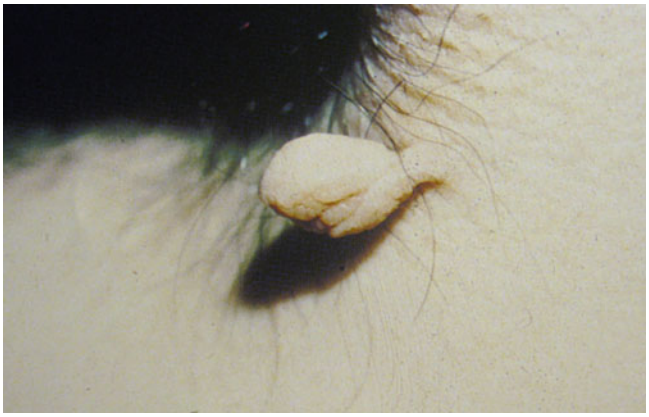


Fig. 25.1 Typical appearance of a fleshy pedunculated ("on a stalk") acrochordon or skin tag

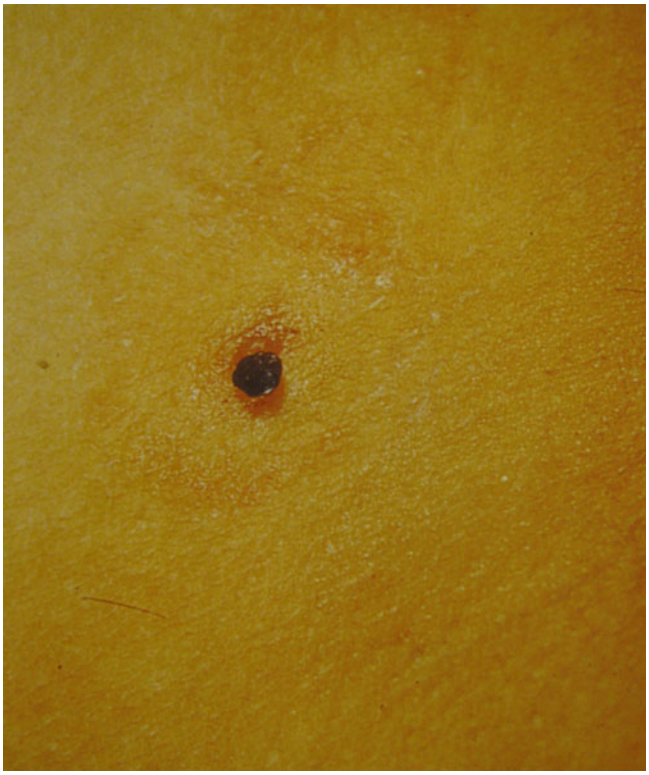
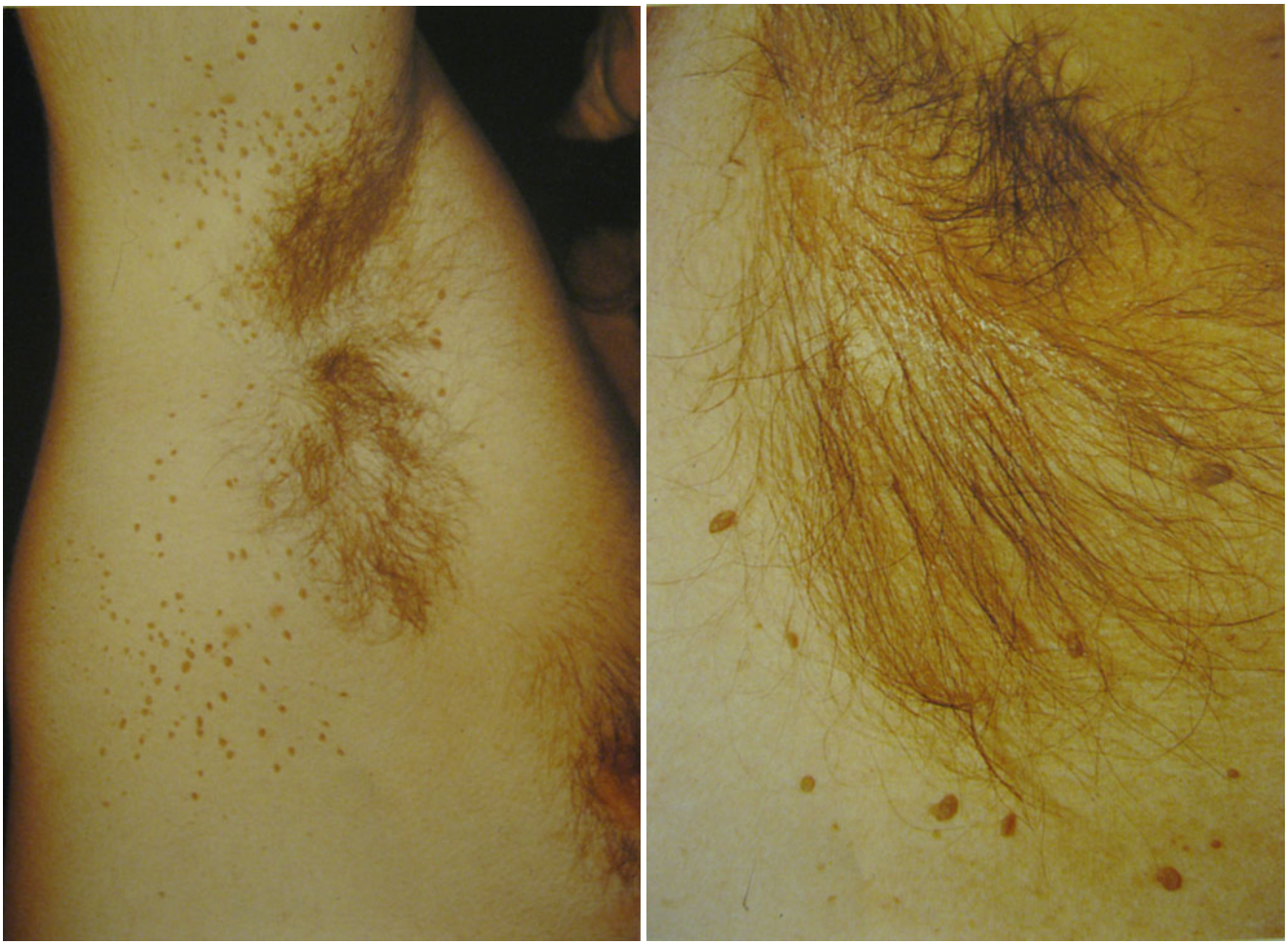
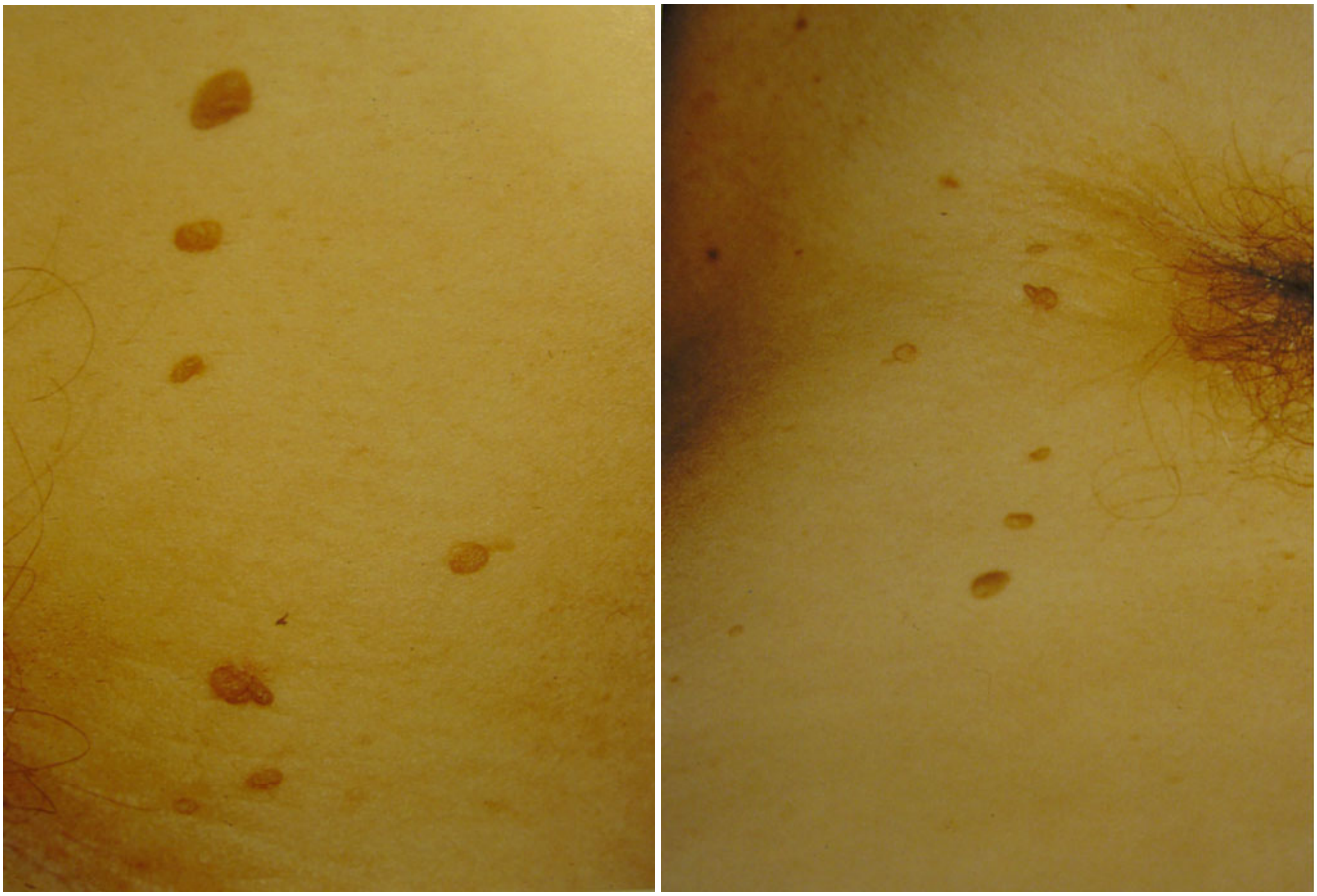


Fig. 25.2 Thrombosed acrochordon may resemble a pyogenic granuloma. Patients frequently bandage these lesions



Figs. 25.3 and 25.4 Multiple axillary tags. This patient should be questioned about any personal and family history of colonic polyps or colon cancer



Figs. 25.5 and 25.6 These skin tags resemble small seborrheic keratoses

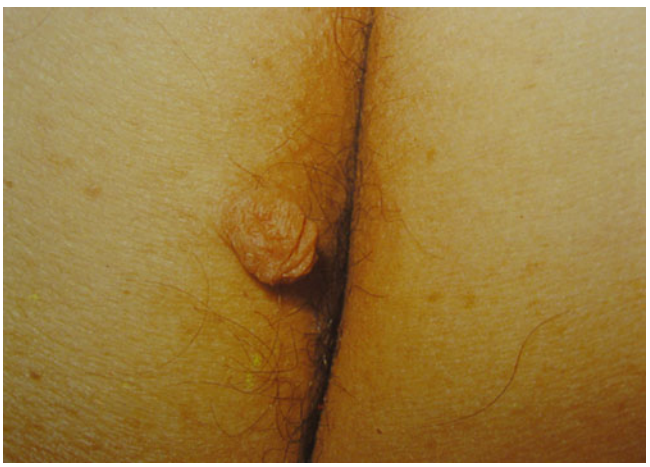


Fig. 25.7 Large. soft fleshy tag: fibroma molle (compare-fibroma durum refers to dermatofibroma)

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Cutaneous Metastases

26

Metastases to the skin from internal malignancy provide important diagnostic clues. They may represent the first manifestation of an undiscovered internal cancer, or may be the first indication of metastasis of a treated cancer [1].

Clinical Description

The clinical appearance of the metastasis is often not diagnostic. Metastases are usually mobile, dermal or subcutaneous papules or nodules. They may be skin-colored, red, violet, or pigmented and may be single or multiple. They are often firm or hard.

Carcinomas of the breast and mouth metastasize by lymphatic invasion; the location of these cutaneous metastases usually reflects the site of the underlying tumor. Intra-abdominal tumors, such as tumors of the colon, stomach, or pancreas, sometimes metastasize to the umbilicus as the so-called Sister Mary Joseph's nodule. Most tumors invade vascular structures, however, and spread hematogenously to sites far beyond the primary tumor (e.g. renal clear cell carcinoma may present as a scalp nodule).

Four clinical types of cutaneous metastases occur in carcinoma of the breast from lymphatic invasion: (1) inflammatory carcinoma; (2) telangiectatic carcinoma; (3) nodular carcinoma; and (4) carcinoma en cuirasse ("cancer in breastplate," also known as scirrhous carcinoma). In a fifth type, hematogenous spread results in scalp nodules and/or alopecia neoplastica [2–5].

Etiology and Pathogenesis

Cutaneous metastases are rare. In one review of 2,298 patients reported to have died of internal cancer, only 2.7 % had cutaneous metastases. The incidence of cutaneous metastases correlates well with the incidence of the primary malignancy. A review of 724 patients in 1972 gave the following results for the origin of the primary tumor in patients with cutaneous metastases. In decreasing order of frequency, for men, these included lung, colon, melanoma, oral cavity, kidney, and stomach; for women, these included breast, colon, lung, ovary, and melanoma. Lung cancer has now become the most frequent cause of cancer death in women as well as in men [6–10].

Diagnosis and Histopathology

Histologic diagnosis is essential, but histology alone is not always sufficient to identify the tissue origin of the cutaneous metastasis. Special stains and immunoperoxidase techniques are often necessary to distinguish among the various adenocarcinomas.

In four types of carcinoma, the histologic characteristics of the metastasis allow identification of the primary tumor. These include carcinoma of the gastrointestinal tract, kidney, and liver, and choriocarcinoma. Metastatic carcinoma of the gastrointestinal tract shows mucin-containing cells in glandular patterns or in irregular collections as "signet ring cells." Metastatic carcinoma of the kidney shows pale, clear cells with a richly vascular stroma. Metastatic carcinoma of the liver shows hepatocellular carcinoma and/or cholangiocarcinorna: the presence of bile-containing acinar structures is diagnostic. Choriocarcinoma shows two types of cells that arise from the fetal trophoblast; elevated urinary levels of chorionic gonadotropin confirm the diagnosis.

Metastatic carcinoma of the breast shows clusters, cords, and/or single, atypical. adenocarcinoma cells in dilated lymphatic vessels. Metastatic carcinoma en cuirasse (scirrhous carcinoma) shows only a few tumor cells embedded between collagen bundles in single-row lines, termed "Indian filing."

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Hematogenous metastases may cause atrophy of the hair follicles as a result of fibrosis, clinically presenting as alopecia neoplastica.

Differential Diagnosis

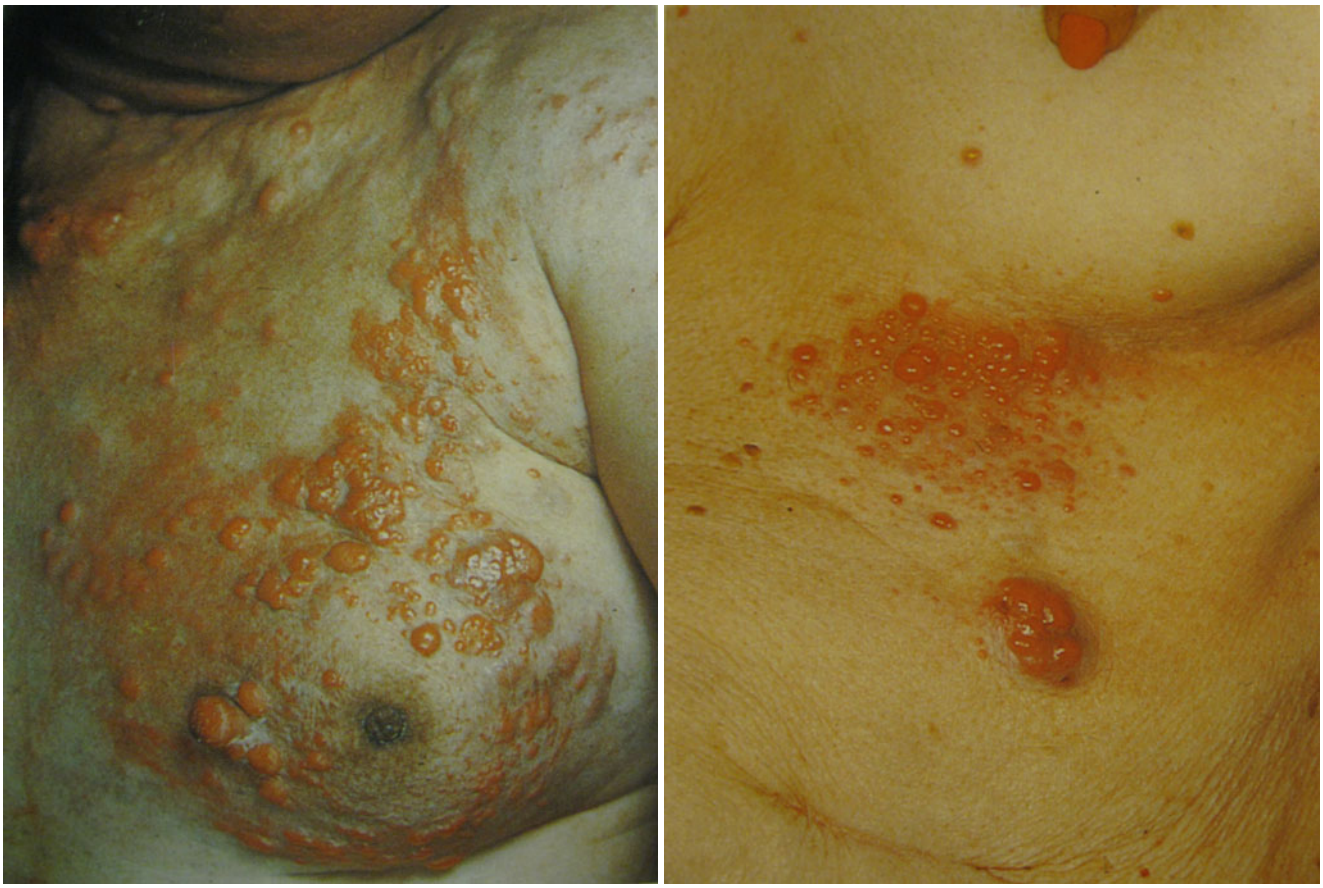
Metastatic tumor nodules can resemble epidermoid cysts, lipomas, neurofibromas, adnexal tumors, polyps, pyogenic granulomas, Kaposi's sarcoma, or lymphoma. Alopecia neoplastica can simulate other forms of scarring alopecia such as discoid lupus erythematosus, lichen planus of the scalp (lichen planopilaris), aplasia cutis, and alopecia mucinosa (mucinous infiltration of the follicles, occasionally seen in mycosis fungoides).

Therapy

Therapy must be aimed at finding and treating the primary underlying tumor. Palliative treatments for multiple cutaneous metastases include radiation therapy, chemotherapy, and immunotherapies (monoclonal antibodies).

Prognosis

The prognosis depends on the type of primary tumor but, once metastases develop, the outlook for survival is grim.



Figs. 26.1 and 26.2 Cutaneous metastases from the lymphatic spread of breast adenocarcinoma typically involve the anterior chest wall

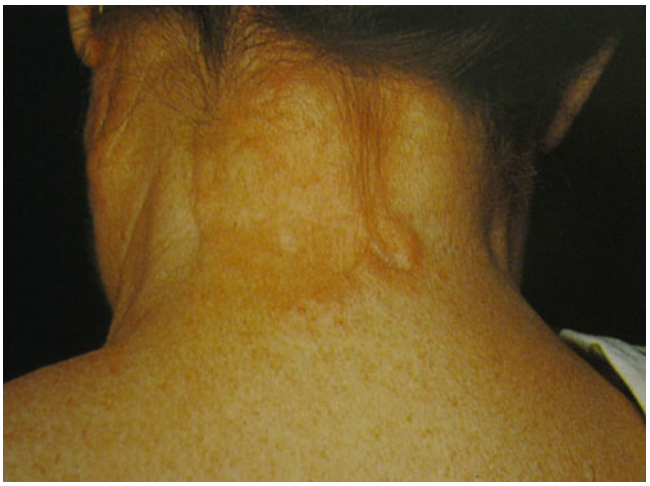


Fig. 26.3 The less common pattern of hematogenous spread of breast cancer results in distant metastasis-in this case, the posterior neck

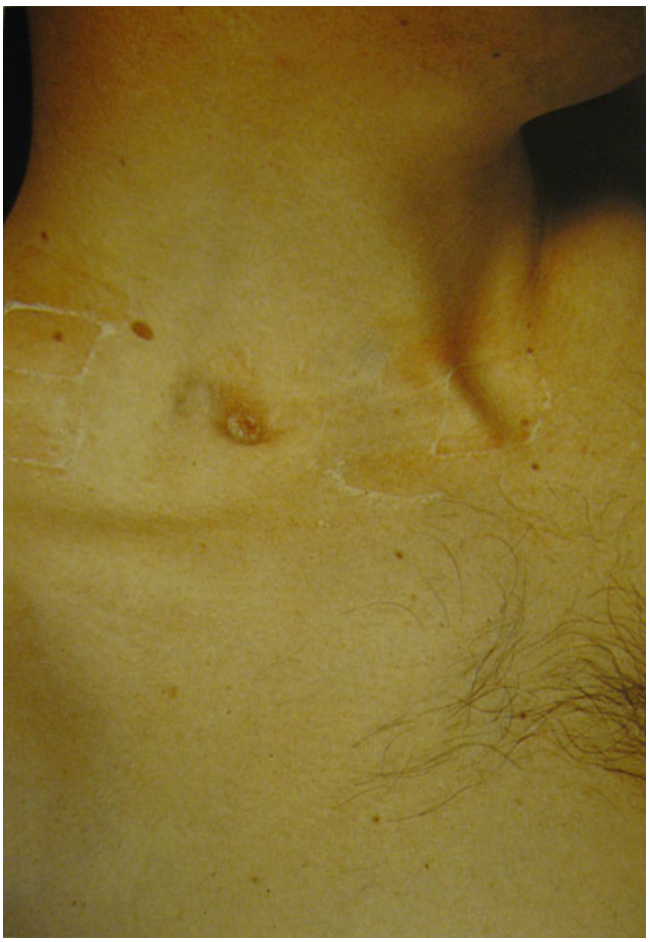
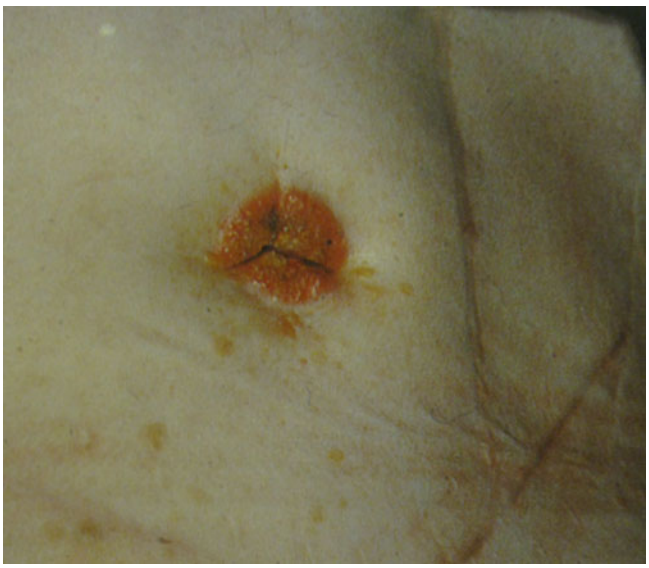
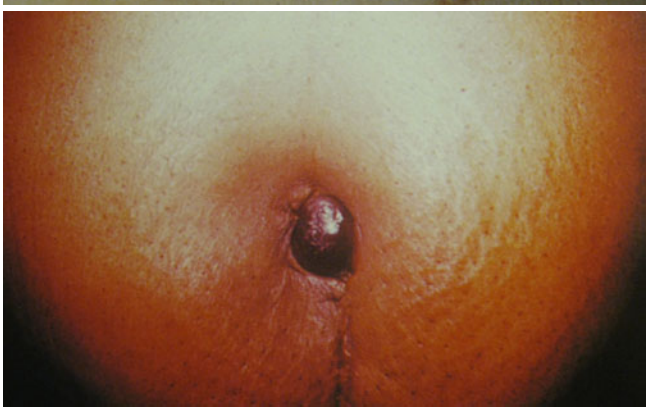


Fig. 26.4 Local extension of carcinoma from the lung to the clavicular region. Bronchogenic carcinoma is now the most common cause of cancer death in men and women





Figs. 26.5 and 26.6 Sister Mary Joseph's nodule: an intra-abdominal tumor, usually of the colon, stomach, or pancreas, metastasizes to the umbilicus

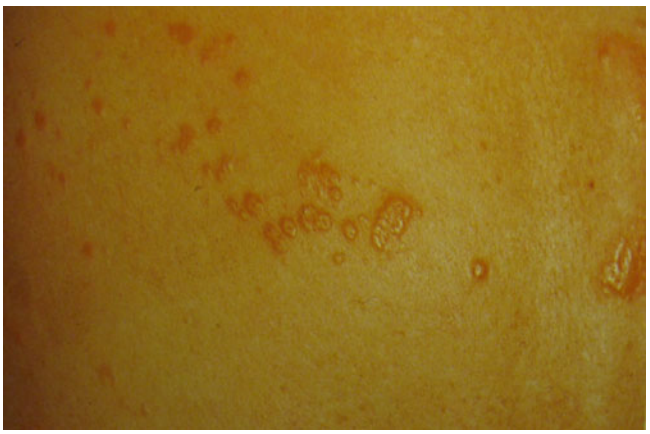


Fig. 26.7 Metastatic carcinoma of the stomach has spread to the back

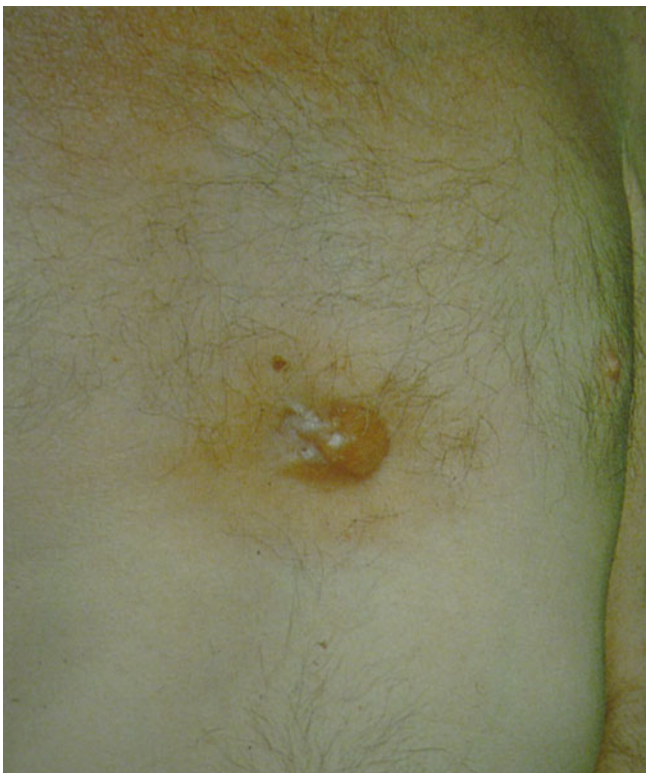
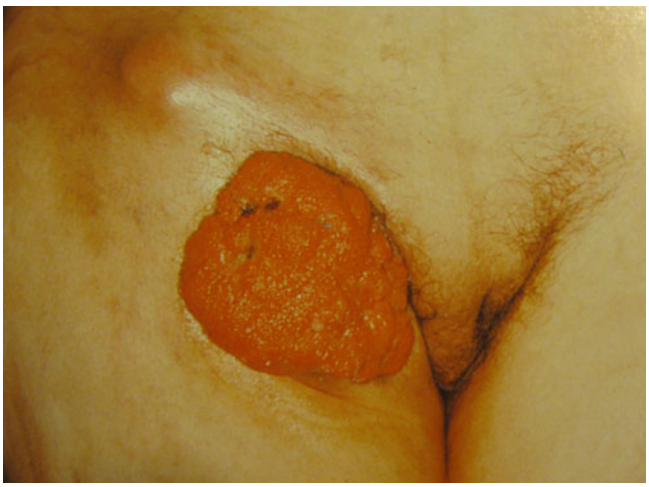
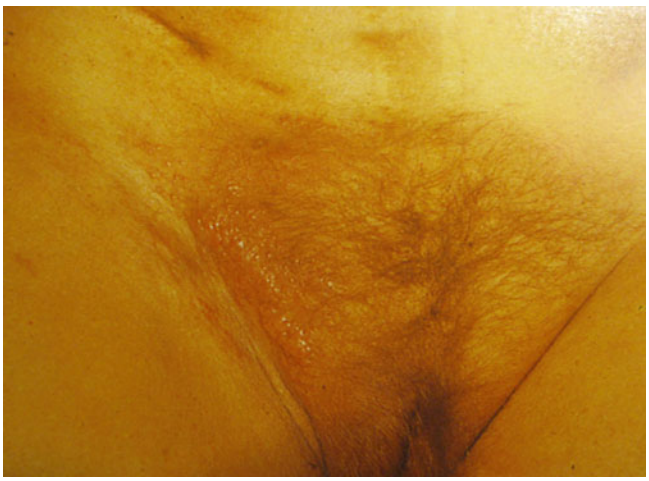


Fig. 26.8 Metastatic rectal cancer has spread hematogenously to the anterior chest wall





Figs. 26.9 and 26.10 Carcinoma of the vulva (Fig. 26.9) and uterine cervix (Fig. 26.10) have metastasized to the groin

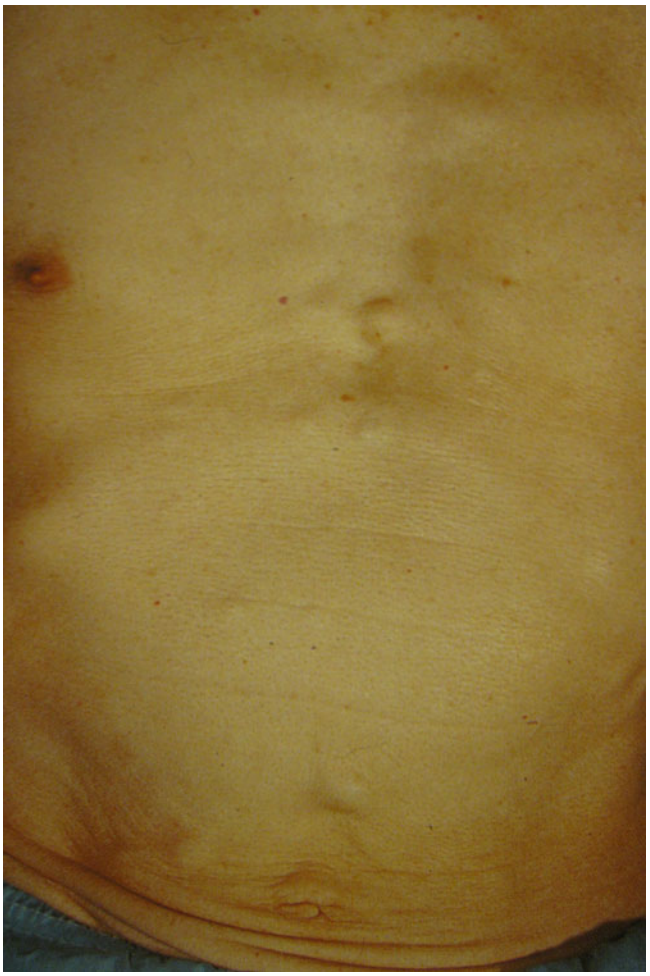


Fig. 26.11 Hematogenous metastasis of prostatic adenocarcinoma appears as firm, subcutaneous nodules on the chest and abdomen

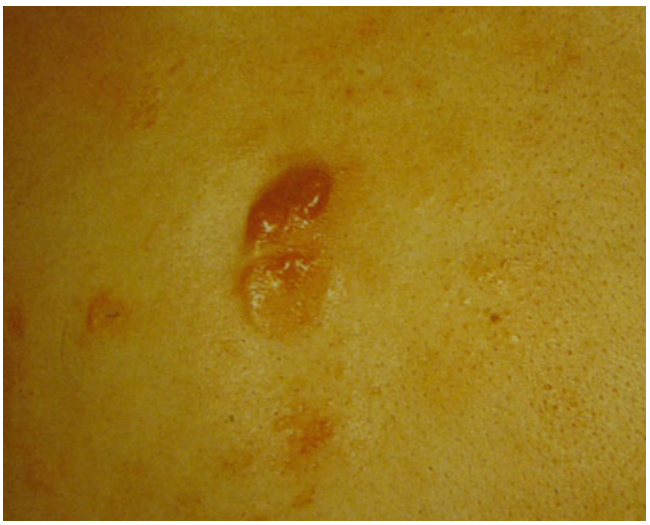


Fig. 26.12 Hematogenous spread of renal cfear cell carcinoma localized on the back

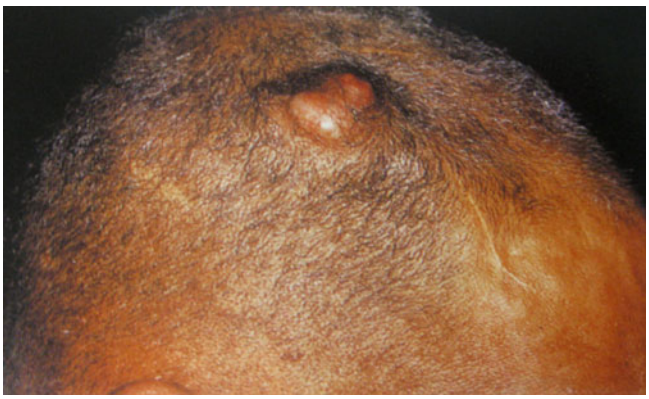


Fig. 26.13 Carcinoma of the tonsils has spread hematogenously, resulting in a rock-hard scalp nodule

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Xanthomas and Xanthelasma

27

Synonyms

Xanthoma subtypes include eruptive, planar, tuberous, and tendinous xanthomas.

Clinical Description

Xanthelasmata appears clinically as soft periorbital, yellow-tan, domed papules and plaques Xanthomas are yellow-tan-orange papules, plaques, and patches, often appearing on the palms and elbows and along tendons.

Etiology and Pathophysiology

More than 50 % of patients affected with xanthelasma have normal serum cholesterol levels. Patients with xanthomas show overproduction and dermal deposition of various lipoproteins. Eruptive xanthomas can occur in those with severe, uncontrolled diabetes or other conditions associated with a sudden elevation of triglyceride levels [1–7].

Histopathology

Formalin-fixed frozen sections must be used for histopathologic investigation because lipids are extracted by the xylene used during the automated analysis procedure used for specimen processing. Foamy, vacuolated, lipid-laden histiocytes replace much of the dermis. Fat stains distinguish xanthomas from nonlipid disorders.

Differential Diagnosis

Clinically, small xanthelasmata may resemble syringomas (eccrine sweat gland cysts) and milia (tiny epidermal cysts). Histologically, lepromatous leprosy (positive Fite stain) also consists of foamy macrophages.

Therapy

The treatment of xanthelasma includes excision, trichloroacetic acid peel, liquid nitrogen cryotherapy, and CO_2 laser vaporization [8]. The treatment of xanthomas involves correction of the underlying lipid disorder and/or diabetes, when present.

Prognosis

Xanthelasmata are persistent and stable. Xanthomas may resolve rapidly with correction of the metabolic disorder (eruptive xanthomas), or may resolve slowly.

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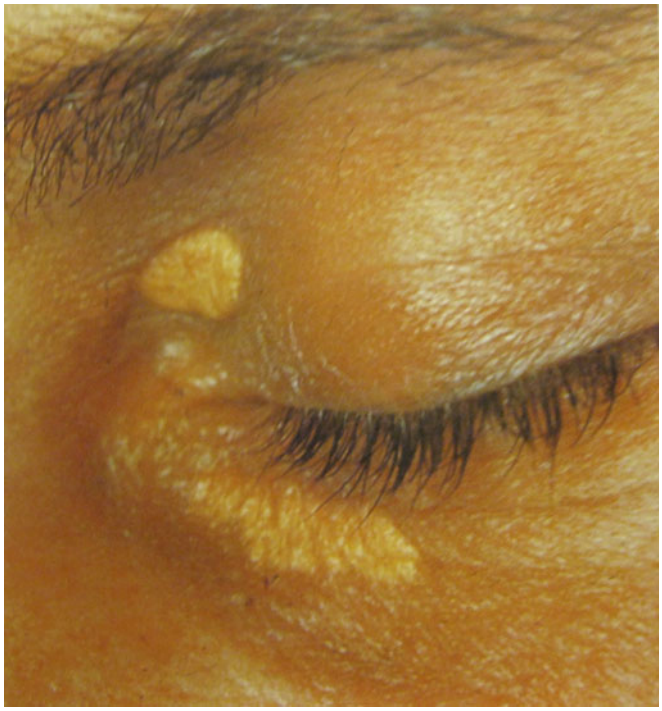
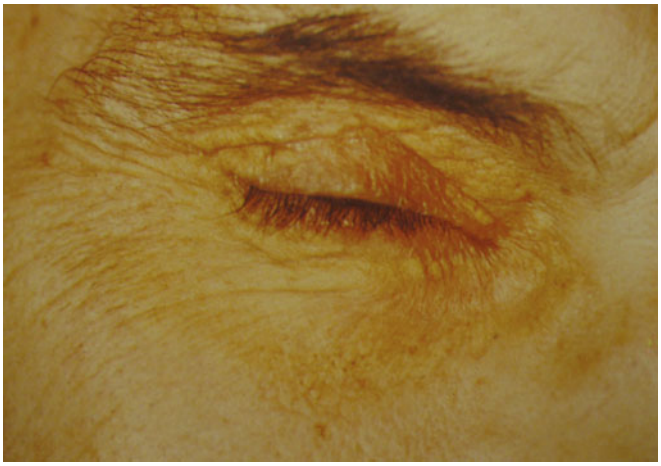
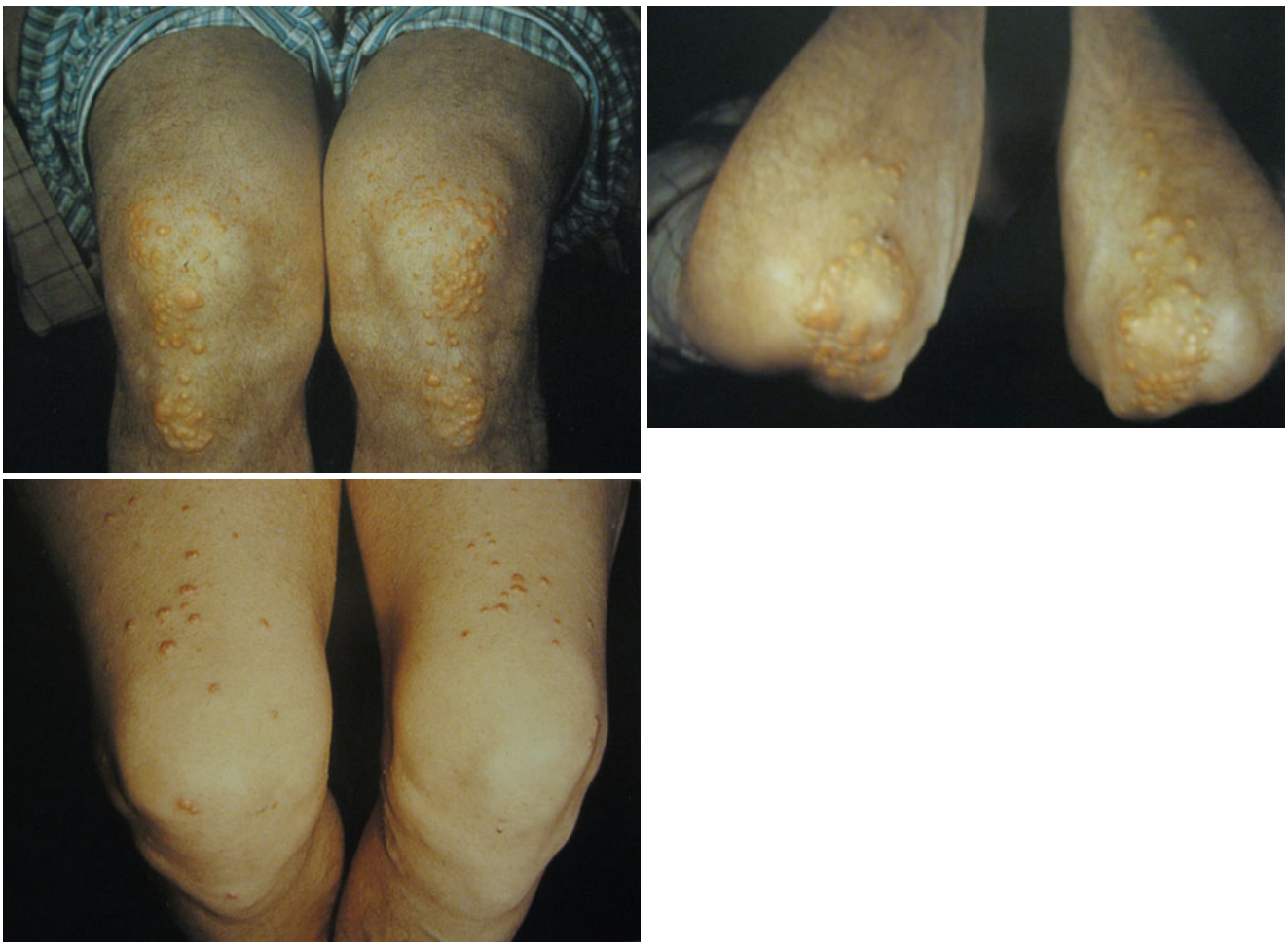
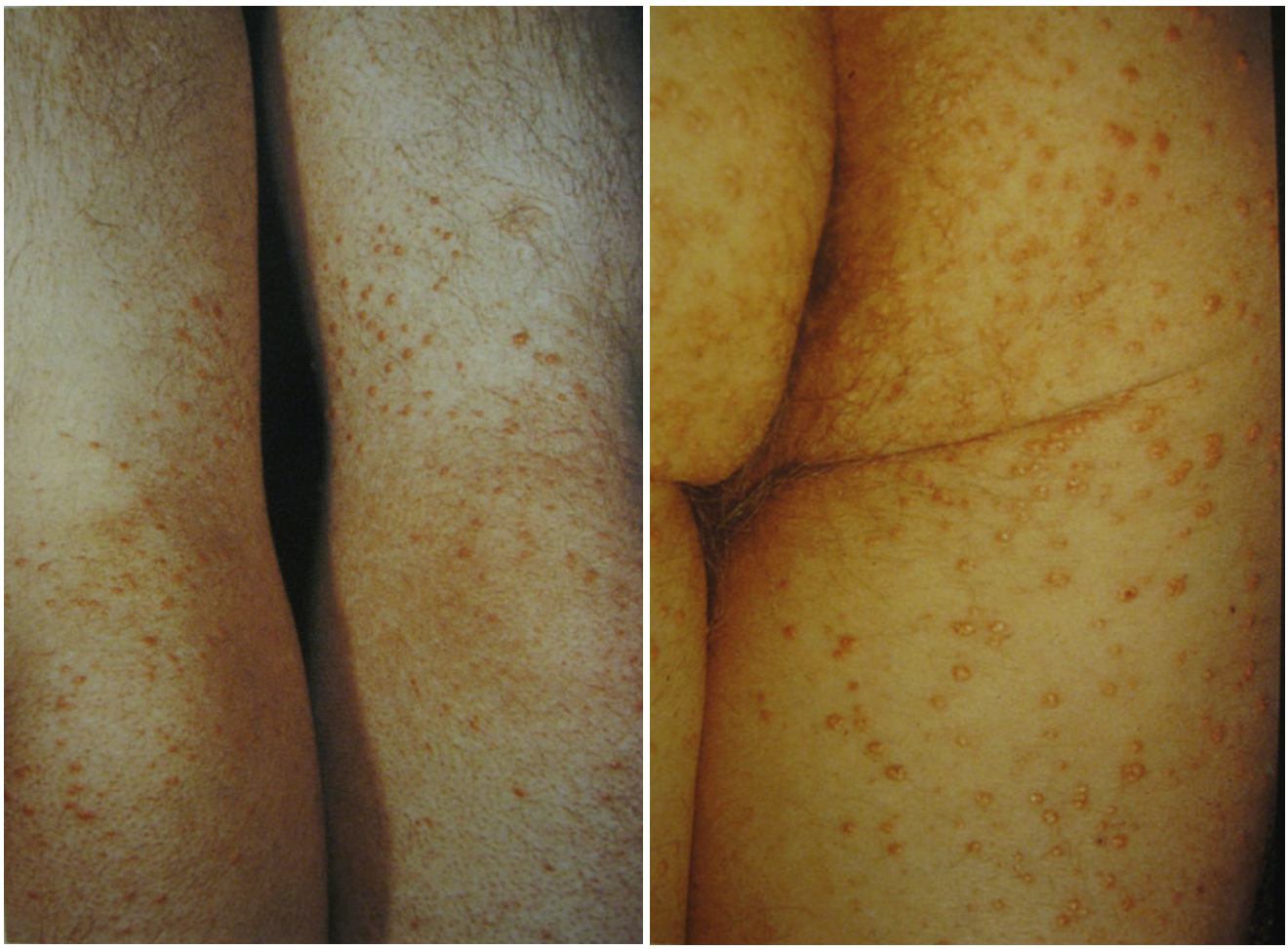




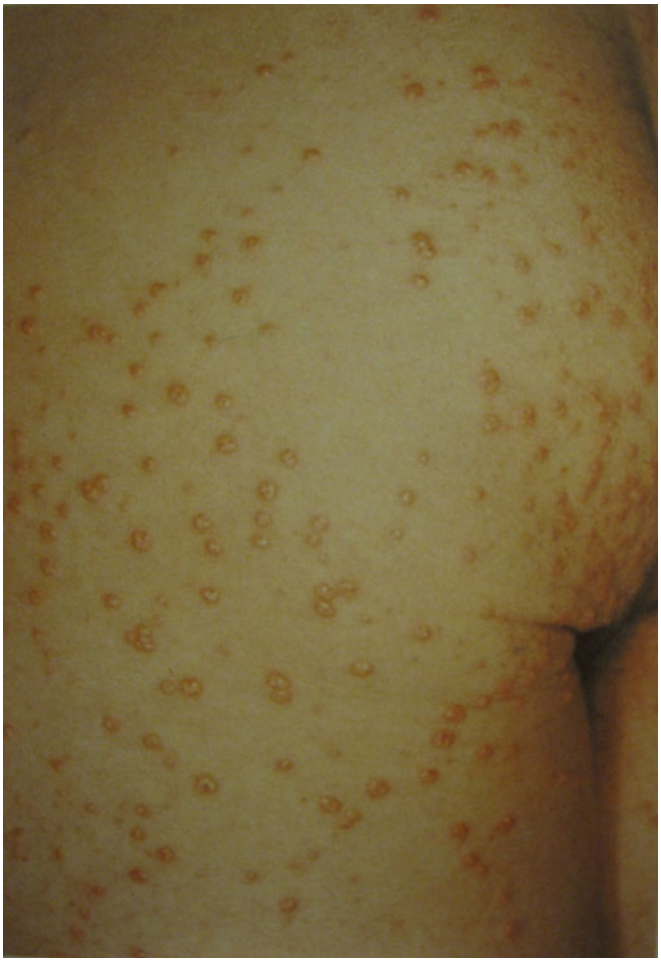
Figs. 27.1 and 27.2 Typical xanthelasmata are smooth, soft, yellow-orange, dome-shaped papules and plaques around the eyes. Most patient have *normal* serum *cho/esterol/eve/s*

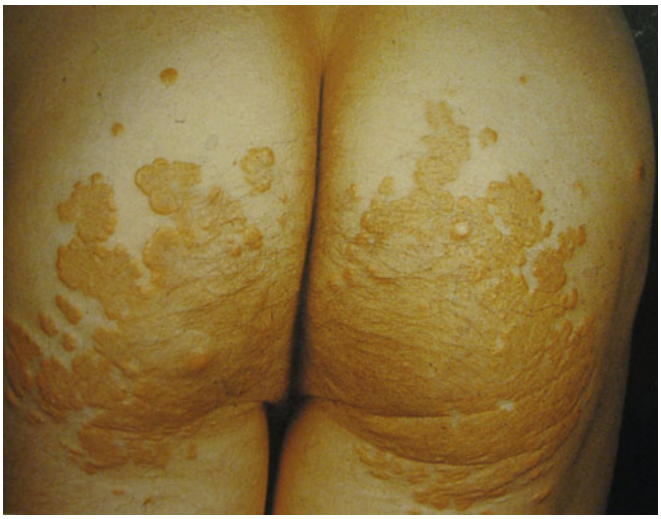


Figs. 27.3, 27.4, and 27.5 Eruptive xanthomas occur in patients with hypertriglyceridemia, often with uncontrolled diabetes mellitus. These yellow-pink papules may wax and wane *quickly, along* with serum triglyceride levels

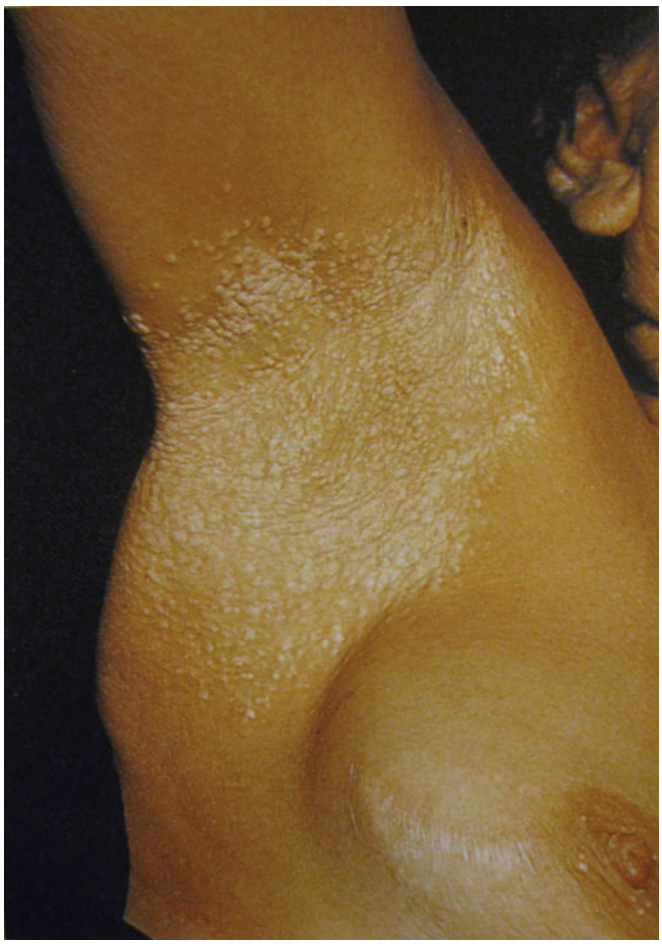


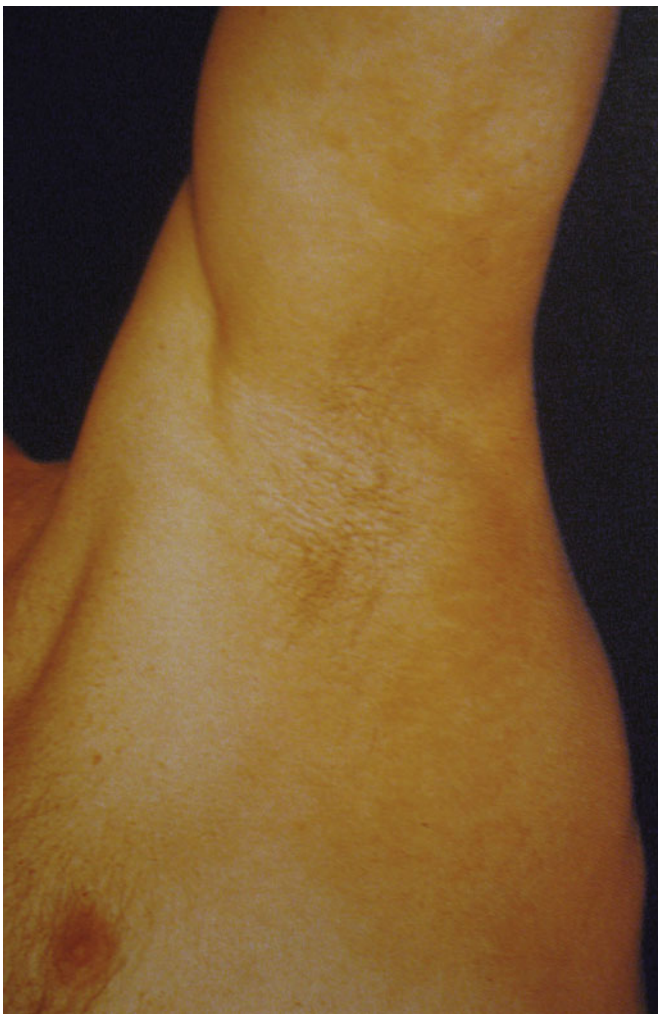
Figs. 27.6, 27.7, 27.8, and 27.9 Eruptive xanthomas occur in patients with hypertriglyceridemia, often with uncontrolled diabetes mellitus. These yellow-pink papules may wax and wane quickly, along with serum triglyceride levels





Figs. 27.6, 27.7, 27.8, and 27.9 (continued)





Figs. 27.10 and 27.11 Axillary xanthomas associated with multiple myeloma

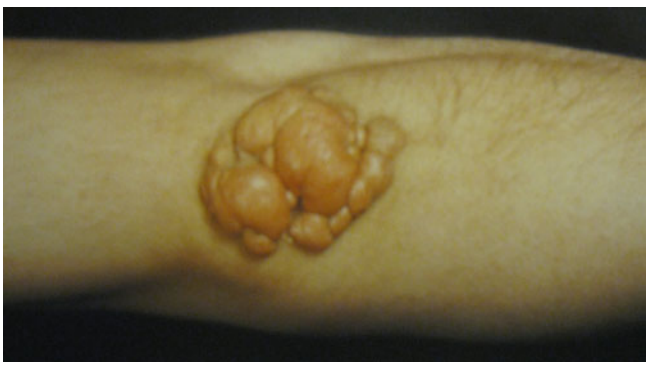


Fig. 27.12 Tendinous xanthomas over the elbow

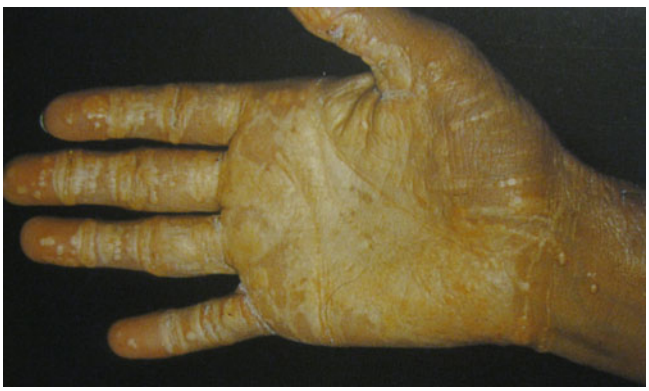


Fig. 27.13 Palmar xanthomas

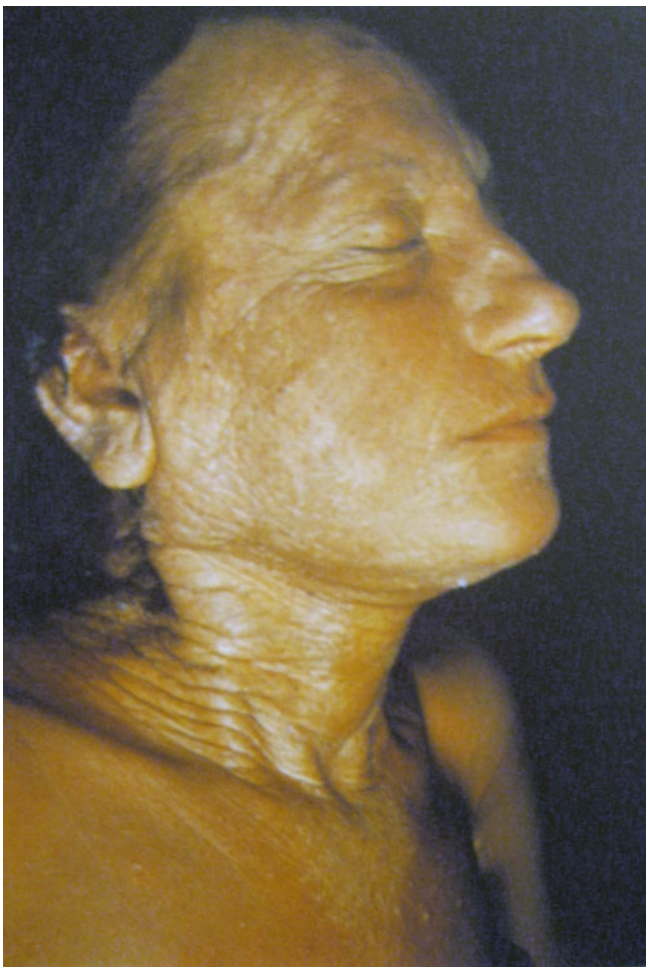


Fig. 27.14 Facial xanthomas in a patient with biliary cirrhosis

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Part V

Regional Dermatoses

Intertrigo

28

Clinical Description

Intertrigo means "inflammation of body folds": erythema, maceration, fissures, and even erosions in body folds. Intertriginous areas include the axilla, inframammary fold, groin, gluteal crease, and digital web spaces. In supine, bedridden, elderly patients, the back also becomes an intertriginous area [1].

Etiology and Pathophysiology

Several diverse causes, often multiple, can be present in an individual patient:

1. Often, a mixture of fungal and bacterial infections occurs. Fungal infections can be produced by Candida or dermatophytes (see Chap. 20). Bacterial infections can be produced by Corynebacterium minutissimum (erythrasma), staphylococcus, streptococcus, or gram-negative bacteria (especially Pseudomonas aeruginosa). In gram-negative bacterial infection of the toe webs, dermatophytes produce penicillin-like substances that kill gram-positive bacteria, leaving gram-negative bacteria that produce irritating chemicals. These chemicals cause maceration, inflammation, vesiculation, and a rapid epidermal turnover rate that results in shedding of dermatophytes. Therefore, KOH, microscopic examination, and fungal culture results are often deceptively negative. Therapy requires a fungicide as well as an antibiotic.
2. Environmental factors include chafing from clothes, chemical irritants, soaps, lotions, medications (see Chap. 2), and overcleansing.
3. Heat, moisture, excessive body folds (obesity) [2], and urinary and fecal incontinence can produce intertrigo.
4. Primary dermatoses include seborrheic dermatitis, psoriasis, and Hailey-Hailey disease (benign familial pemphigus).

Differential Diagnosis

The differential diagnosis includes the following:

1. Pure tinea--more scale, positive KOH test, scrotal sparing, dry.
2. Pure candida--beefy red, satellite pustules, positive KOH test, scrotal involvement.
3. Lichen sclerosus et atrophicus--atrophy, erythema, and leukokeratosis with secondary erosion in female vulva. This responds to topical testosterone dipropionate cream. In men, penile involvement is called balanitis xerotica obliterans. In both there is an increased risk of developing squamous cell carcinoma.

Therapy

1. Therapy includes elimination and treatment of causative factors.
2. The patient should keep body folds cool and dry, wear cotton undergarments, and use a warm air blow dryer after bathing or showering.
3. Protective zinc oxide ointment may be used.
4. A topical antifungal alternating with a mild corticosteroid can be tried, but the patient should be warned of the possibility of atrophy.
5. Compresses with aluminum subacetate (Burow's or Domeboro solution) or vinegar in tap water may be used.
6. Powders (Zeasorb AF-antifungal) can be helpful.
7. Castellani's paint may be applied to the affected area [3, 4]

Prognosis

The prognosis is fair, but the recurrence rate is high, especially in obese, bedridden, and/or incontinent patients.

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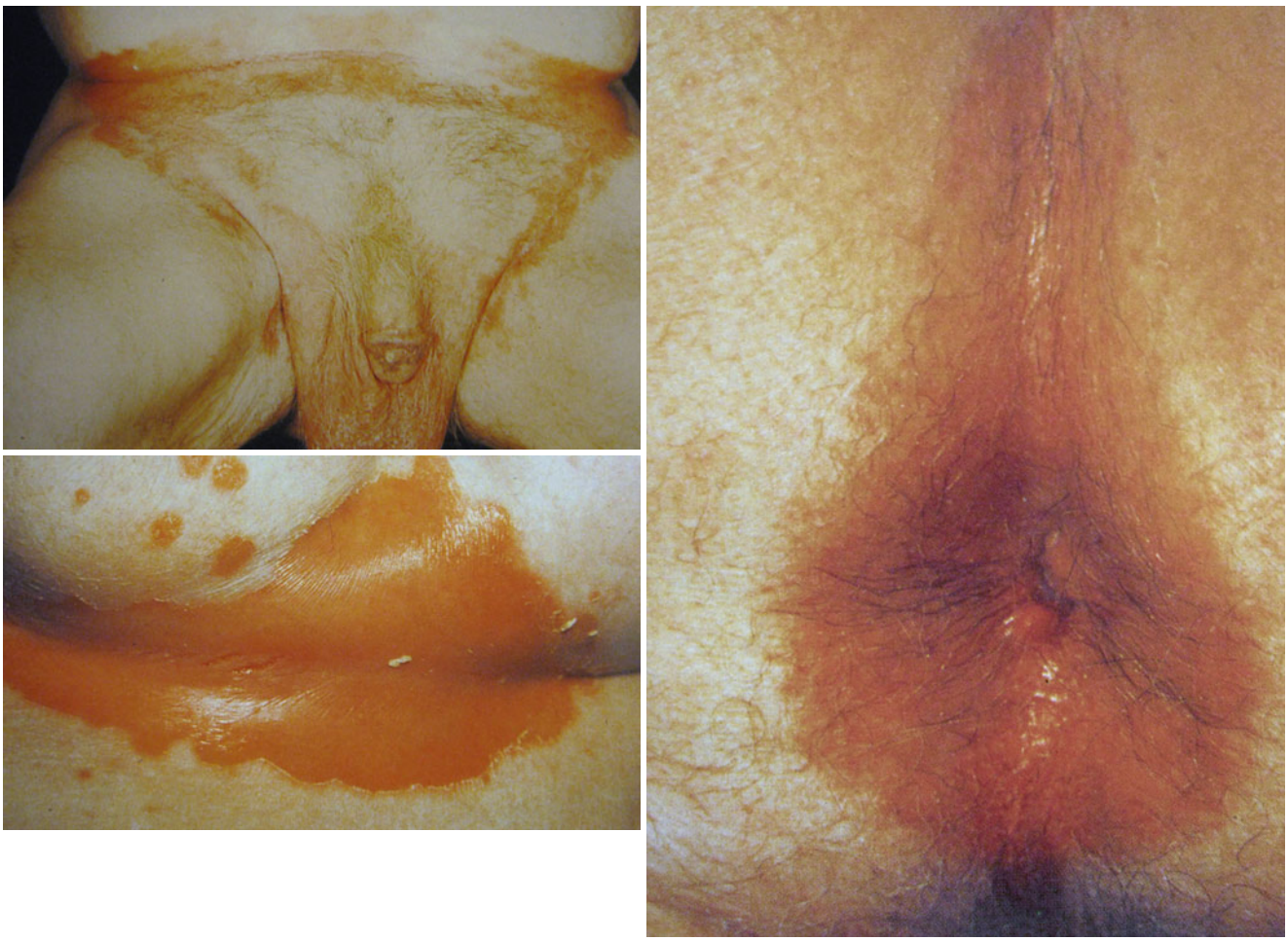

Figs. 28.1, 28.2, and 28.3 Typical presentation of intertrigo shows inflamed, macerated, even eroded skin of body folds: groin, abdomen, inframammary fold, and gluteal cleft

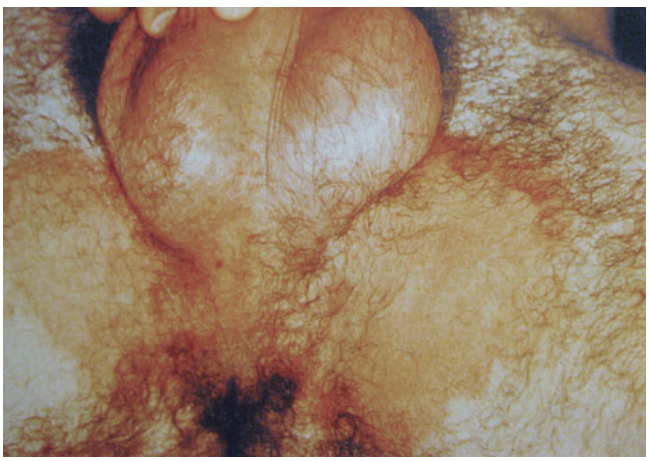

Fig. 28.4 Intertrigo caused by Candida infection

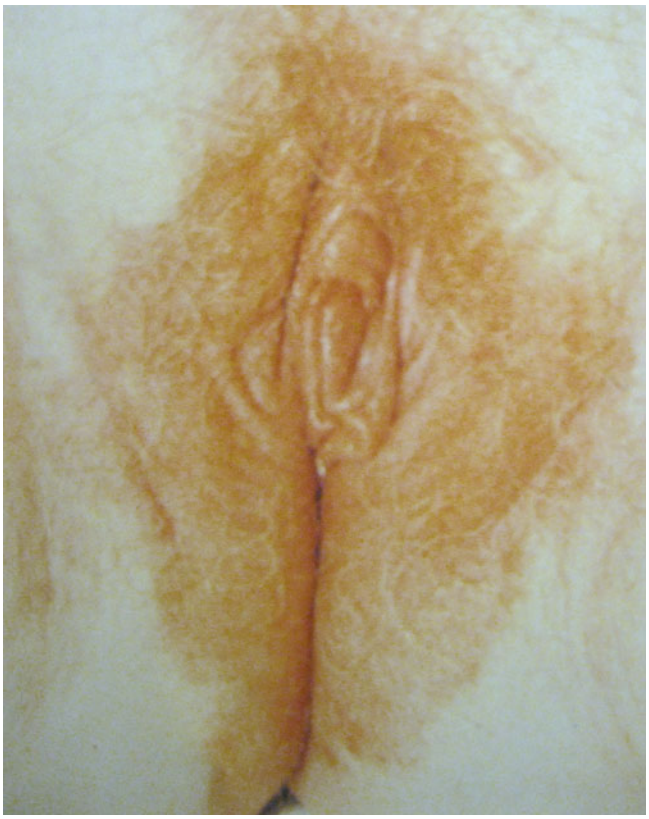

Fig. 28.5 "Dry" dermatophytosis of the groin

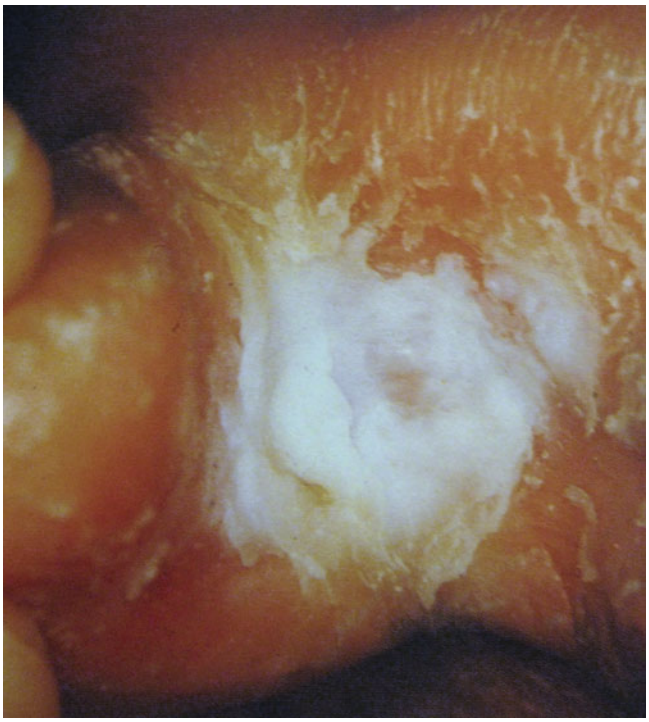

Fig. 28.6 Gram-negative toe web infection (see text for details)

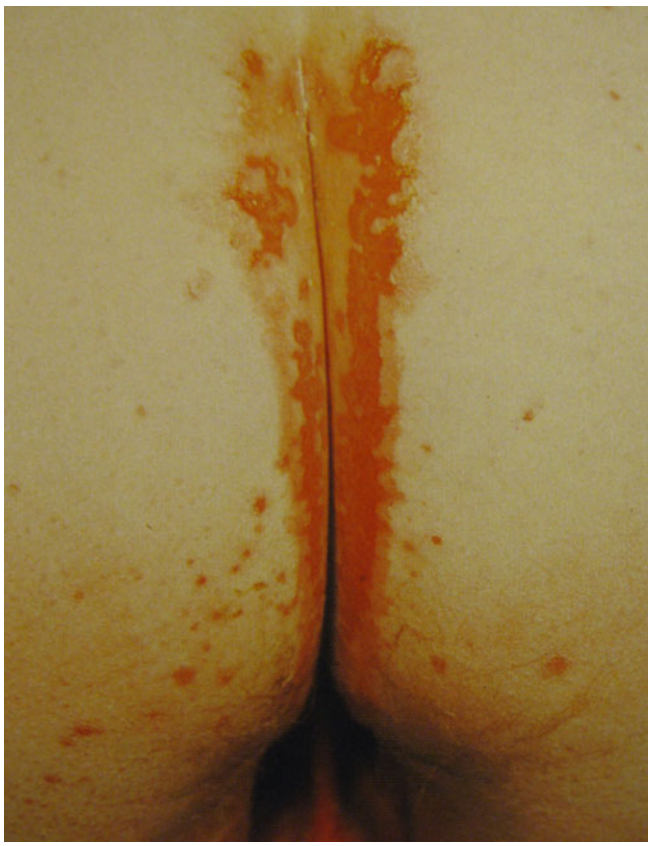

Fig. 28.7 Herpes simplex viral infection of the gluteal crease

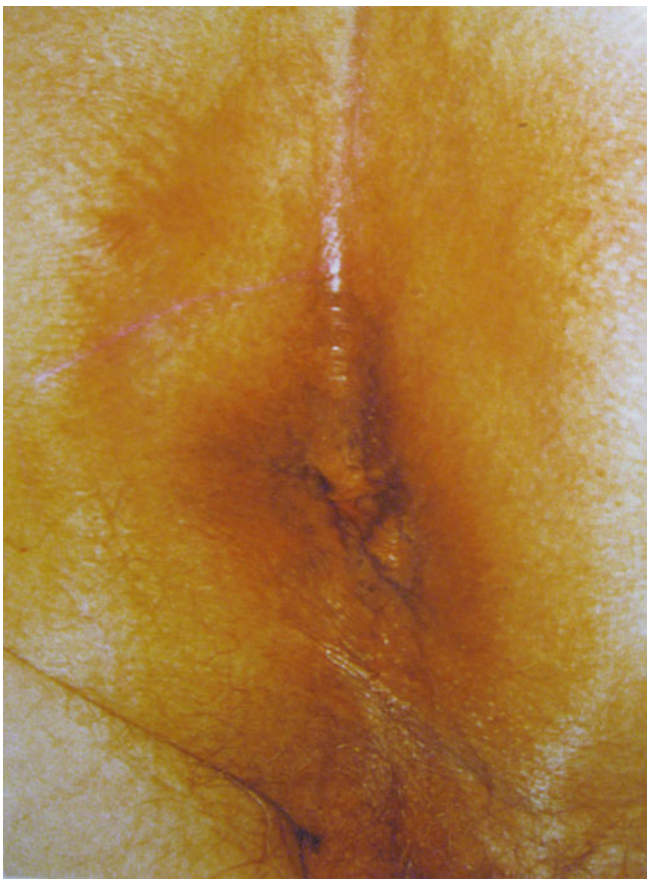

Fig. 28.8 Steroid atrophy from inappropriately prescribed long-term potent corticosteroid applied for relief of itching hemorrhoids

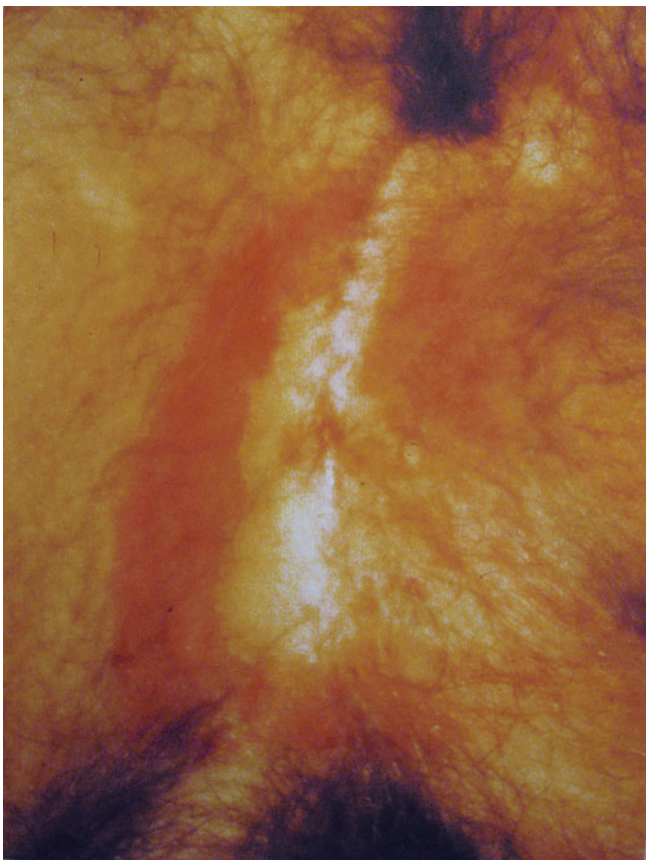

Fig. 28.9 Close-up view of intertrigo of the groin

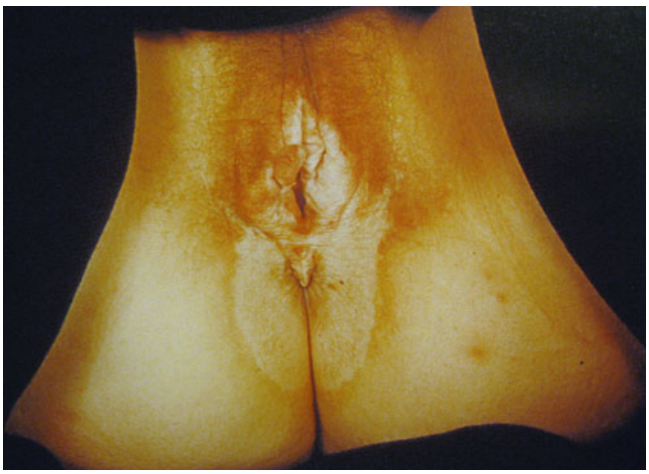

Fig. 28.10 Lichen sclerosus atrophicus of the groin simulates intertrigo. In women, this usually occurs on the vulva shaped as a keyhole or figure B. In men it occurs on the glans penis, called balanitis xerotica obliterans

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Leg Ulcers

29

Clinical Description

Leg ulcers differ in location depending on their cause [1–5].

Venous Stasis (Gravitational) Ulcers. These typically occur on the medial malleoli (ankles) and legs, with concomitant stasis dermatitis-red-brown macules, papules, patches associated with varicose veins, cyanosis, dependent edema, and pain.

Hypertensive (Arterial) Ulcers. These usually present as clean, "punched-out" ulcers on the lateral malleoli and legs, with concomitant shiny skin, hair loss, pallor, and decreased pulse. Pain is relieved by dependency but exacerbated by leg elevation, in contrast to venous stasis ulcers.

Atrophie Blanche. This is a segmented, hyalinizing, vasculitis that shows porcelain white reticular scarring and atrophy, usually on the lower third of the calf, ankle, and dorsal fool.

Infectious Ulcers. These may occur anywhere, or at an inoculation site.

Neoplastic Ulcers. These often occur at the site of a previous draining ulcer or burn scar (Marjolin's ulcer with squamous cell carcinoma).

Inflammatory Ulcers. Rheumatoid vasculitis accompanies joint changes; pyoderma gangrenosum is commonly associated with inflammatory bowel disease.

Traumatic or Factitial Ulcers. These often display bizarre, irregular shapes, and can occur wherever the patient can reach.

Etiology and Pathophysiology

Venous Stasis (Gravitational) Ulcers. Tissue hypoxemia is caused by venous stasis and/or perivenous fibrosis that prevents diffusion of oxygen and other nutrients.

Arterial Ulcers. Tissue hypoxemia is secondary to arteriosclerosis.

Atrophie Blanche. This is a thrombotic vasculopathy.

Infectious Ulcers. Various bacterial, fungal (chromoblastomycosis), treponemal, and parasitic infections and rarely, viral (herpetic) infections can produce ulcers.

Neoplastic Ulcers. These can result from squamous cell carcinoma, mycosis fungoides, basal cell carcinoma, and keratoacanthoma.

Inflammatory Ulcers. These can be produced by vasculitis of any cause, especially rheumatoid arthritis, diabetes (ulcerative necrobiosis lipoidica diabeticorum), panniculitis, and pyoderma gangrenosum.

Traumatic or Factitial Ulcers. These can occur from injecting drugs under the skin ("skin popping") or from underlying depression, resulting in deep excoriations.

Histopathology

Histopathologic investigation reveals the following:

1. Venous ulcers-perivascular fibrosis, hemosiderin deposits in dermis
2. Arterial ulcers-arteriosclerotic changes in arterioles
3. Atrophie blanche-"vasculopathy," often with fibrin thrombi but without leukocytoclastic vasculitis
4. Infectious ulcers-wide variety of histopathology; special stains for fungi, bacteria may be useful
5. Neoplastic ulcers-results vary with tumor type
6. Inflammatory ulcers-leukocytoclastic vasculitis, panniculitis
7. Traumatic ulcers-dermal ulceration with parakeratotic scale or crust, bacterial colonization, and acute and/or chronic dermal inflammation

Therapy

The underlying cause must be treated or eliminated:

1. Venous stasis ulcers-soaks, antibiotics, leg elevation, compression stockings, Unna's boot (zinc oxide, glycerin, and gauze bandage).Vein stripping may be necessary

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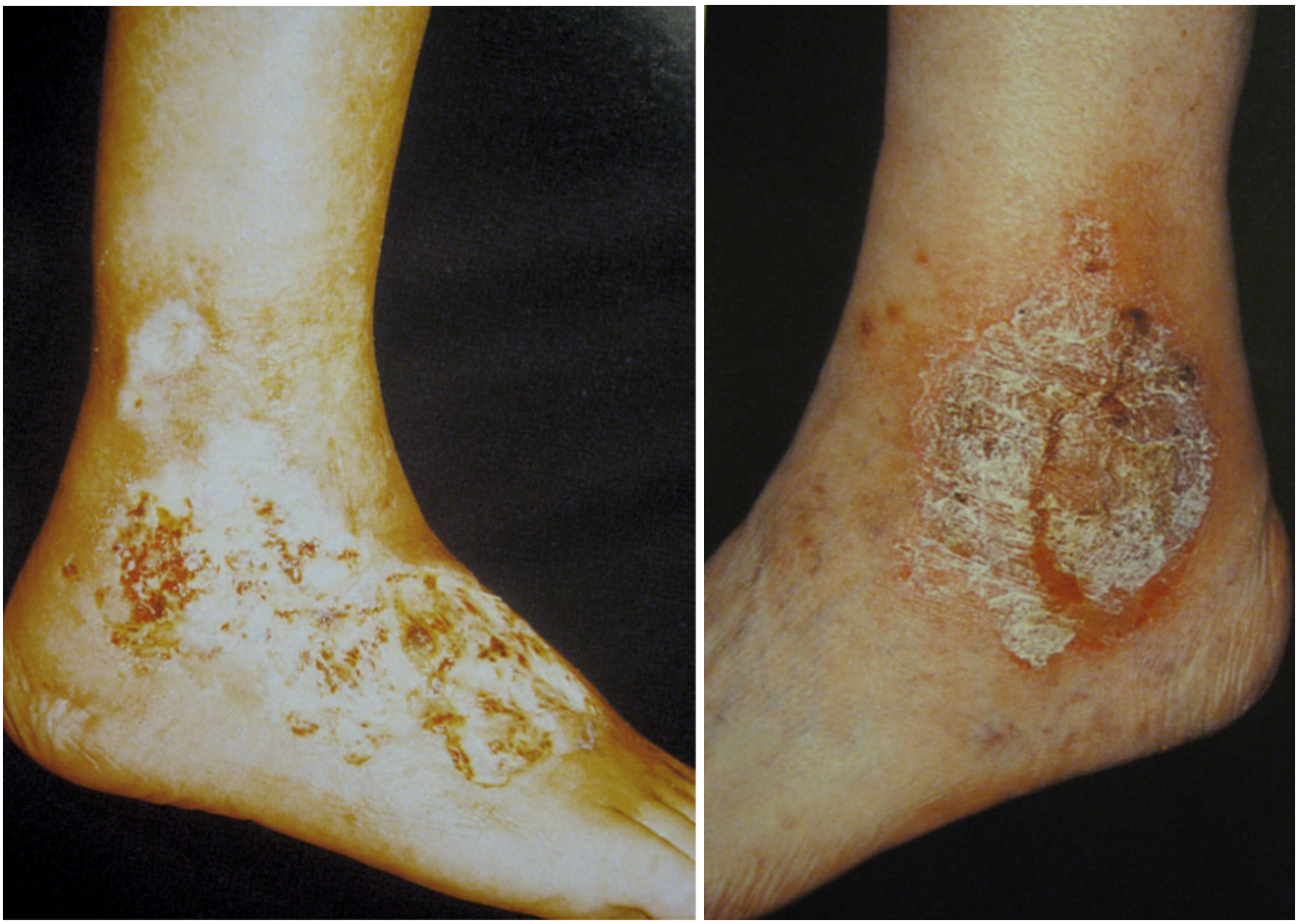
later. Pentoxifylline 400 mg orally bid, enhances erythrocyte flexibility.
2. Arterial ulcers-control hypertension, femoral bypass grafts, vasodilators (e.g., nifedipine). Avoid leg elevation.
3. Atrophie blanche--steroids; pentoxifylline 400 mg orally bid; aspirin (low dose, 325 mg daily) or dipyridamole (50 mg orally tid).
4. Infectious ulcers-appropriate antimicrobials, occasionally excision (chromoblastomycosis)
5. Neoplastic ulcers-excision, radiation therapy, or chemotherapy (mycosis fungoides)
6. Inflammatory ulcers-treat underlying cause of vasculitis, if possible. Systemic steroids may be necessary to suppress a generalized, noninfectious systemic vasculitis, especially if hematuria and/or melena are present.
7. Traumatic ulcers-protection with Unna's boot or plaster cast. Patients with factitial ulcers, however, may continue to traumatize the ulcer with knives or wires inserted under the cast [6–8].



Figs. 29.1 and 29.2 Hypertensive, arteriosclerotic ulcers typically *affect* the *lateral* ankle

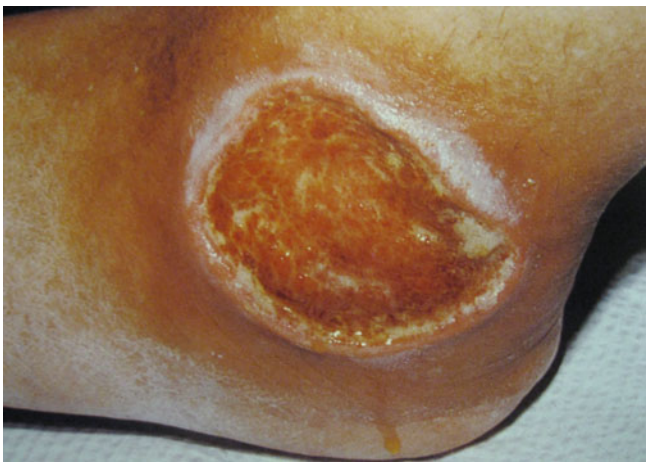


Fig. 29.3 Hypertensive, arteriosclerotic ulcers typically affect the lateral ankle

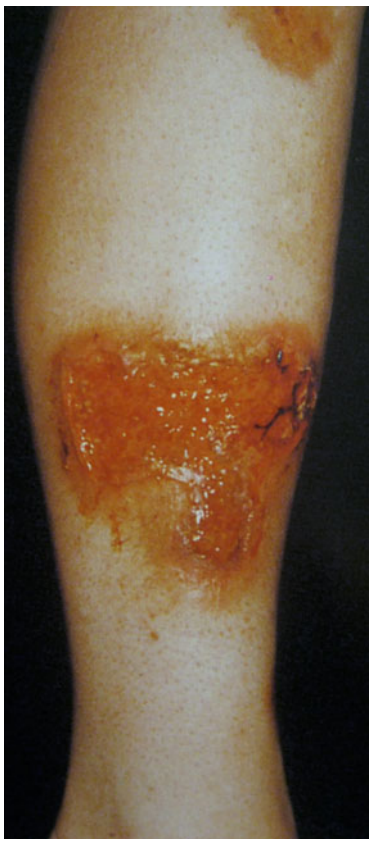


Fig. 29.4 Diabetic leg ulcer is a subset of arteriosclerotic ulcer

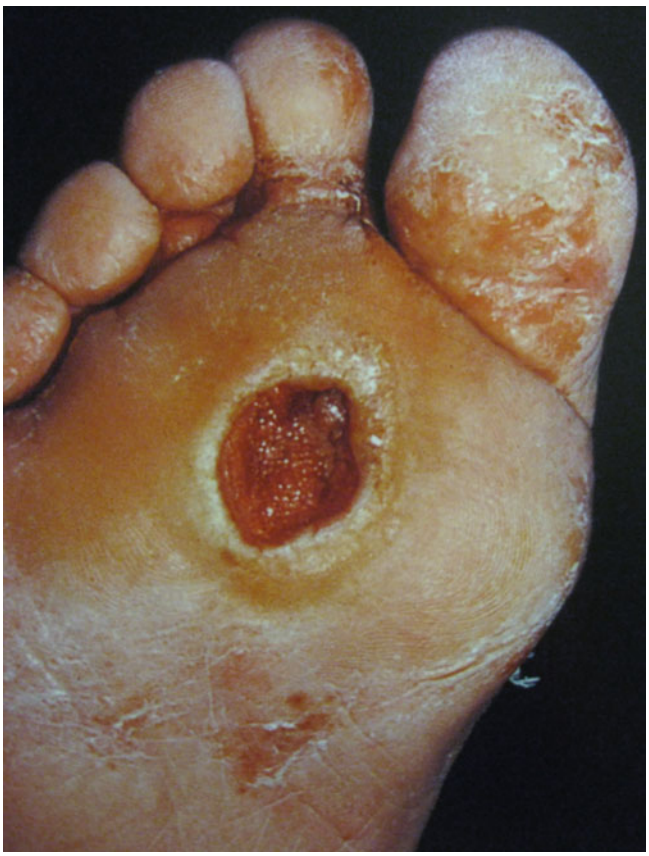


Fig. 29.5 Mal perforans occurs in diabetics whose lack of sensation predisposes to traumatic and vascular ulceration of the sole

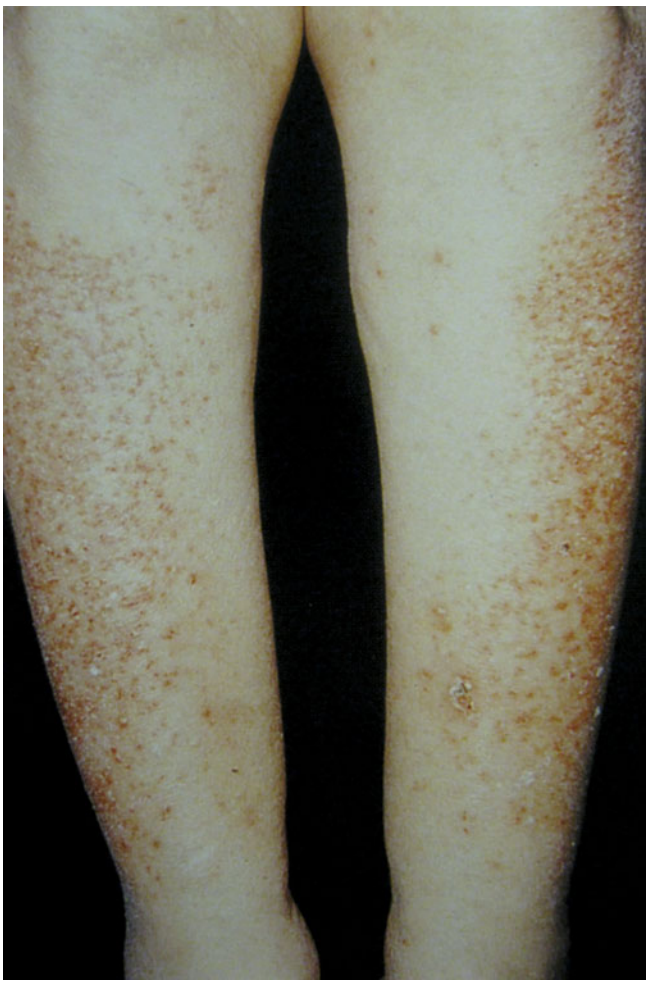


Fig. 29.6 Early venous stasis dermatitis of the legs results from tissue hypoxemia, extravasation of red blood cells, and iron Ihemosiderin) deposition in the dermis. The iron induces cutaneous hyperpigmentation. The affected area is at increased risk of developing contact *dermatitis* to topical medications such as neomycin, aloe vera, vitamin E, and benzocaine

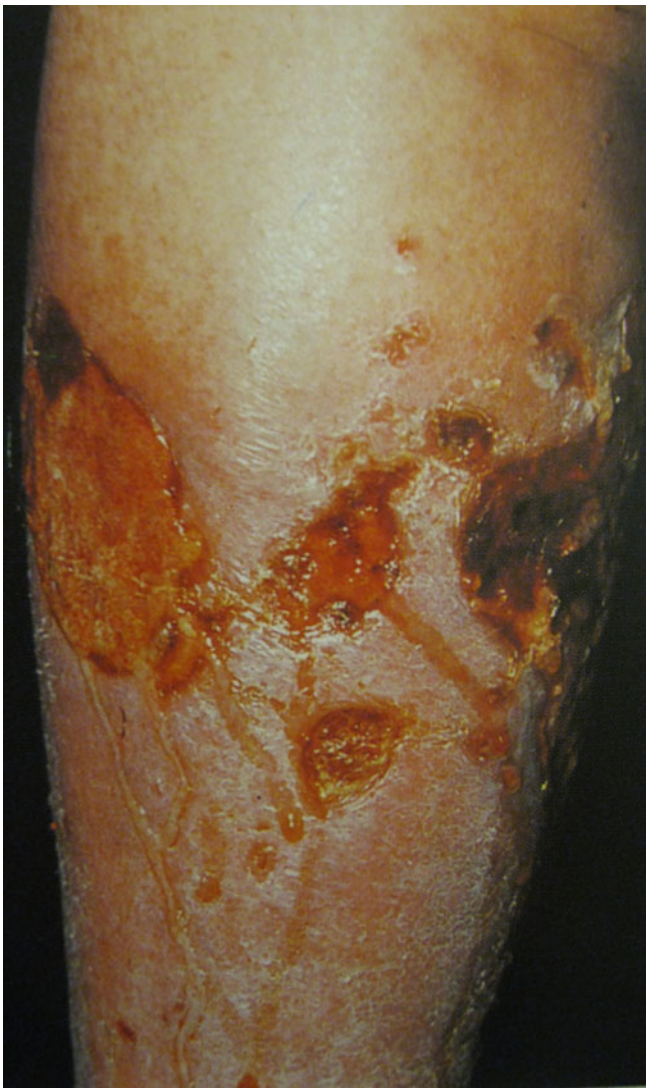


Fig. 29.7 Advanced *stasis* dermatitis showing brawny or woody induration and non pitting edema, ulcers, oozing, and secondary infection

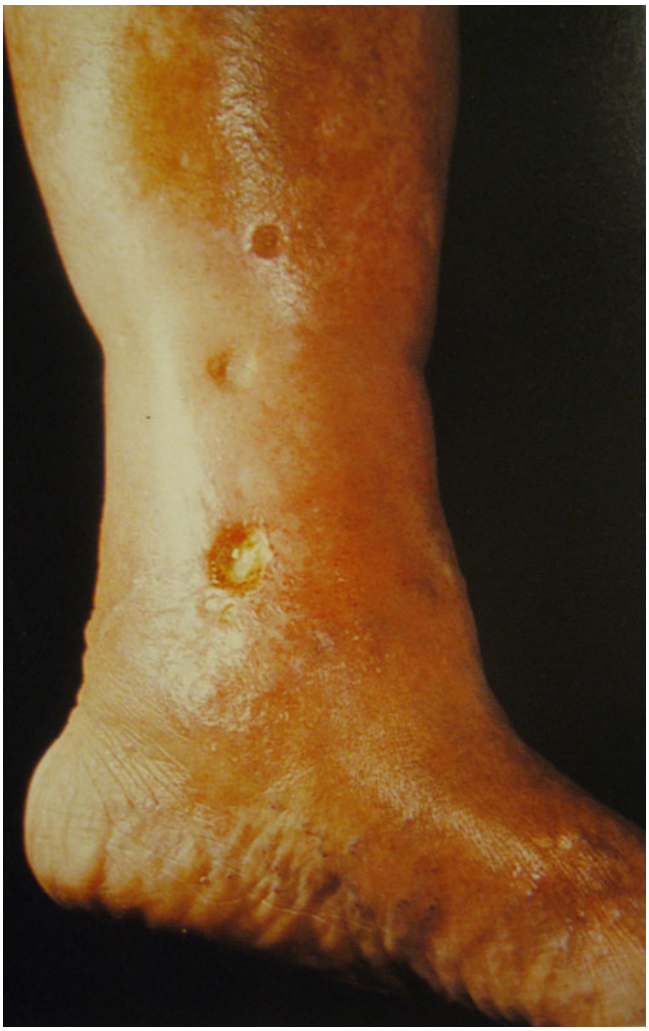


Fig. 29.8 "Punched-out" leg ulcers and severe stasis dermatitis. Note narrowing above the ankle caused by concentric fibrosis and brawny edema superiorly

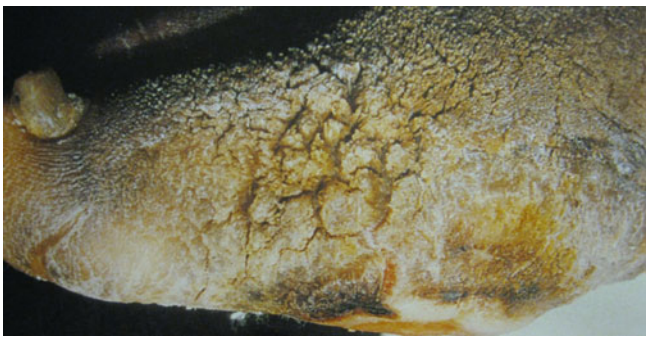
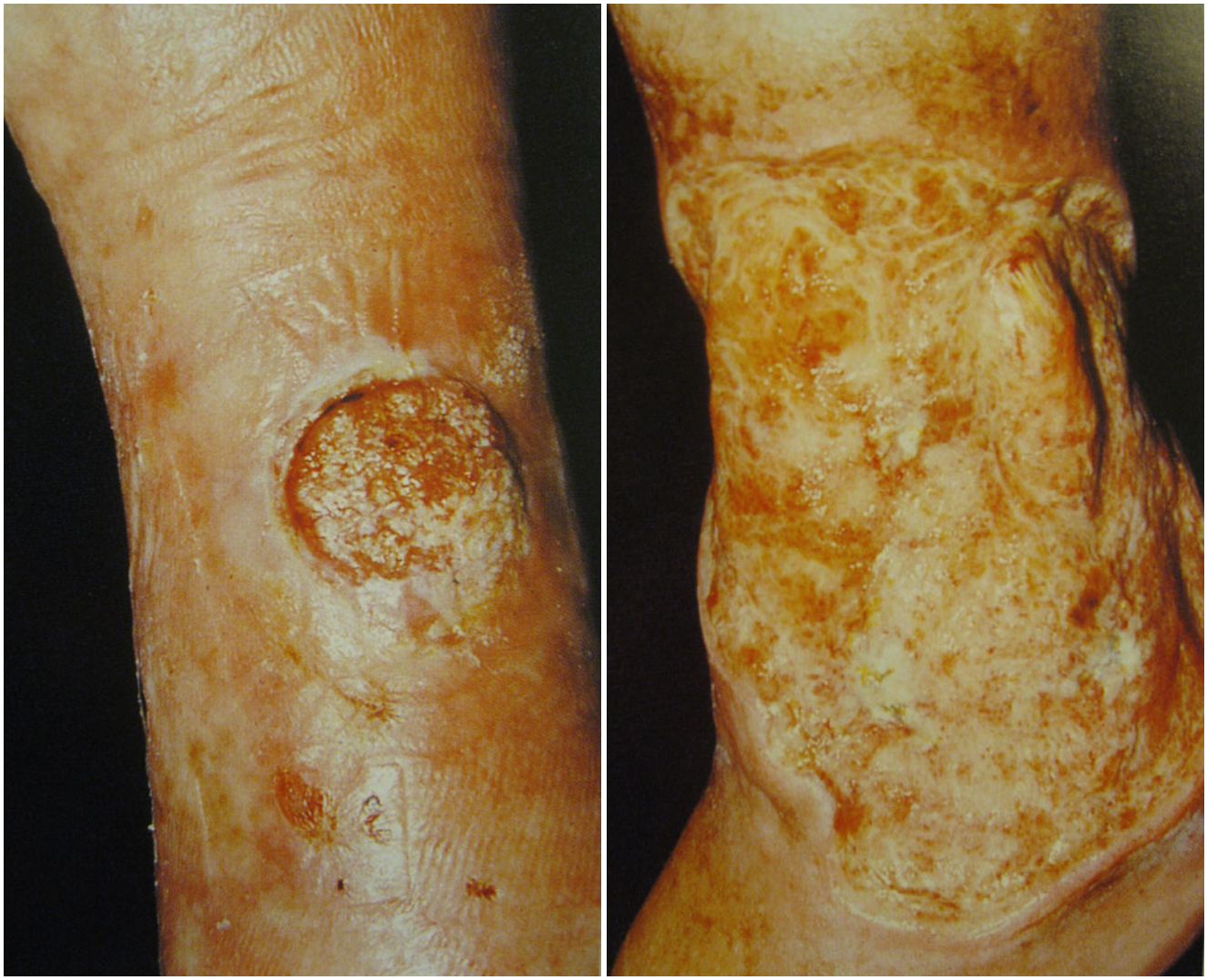


Fig. 29.9 Lymphostasis verrucosa cutis (elephantiasis nostras) is a complication of long-standing venous hypertension and edema. Verrucous vegetations and brush-like filiform keratotic projections carpet the *soles*. Lymphangiosarcoma is another possible long-term complication of venous and lymphatic obstruction and stasis



Figs. 29.10 and 29.11 Rheumatoid arthritis. Vasculitic ulcers occur independently of venous stasis, and can invade muscle, fascia, and even bone. This is a serious although relatively uncommon complication. Corticosteroids and other immunosuppressive therapies are mandatory to control the arthritis, but their use can delay wound healing

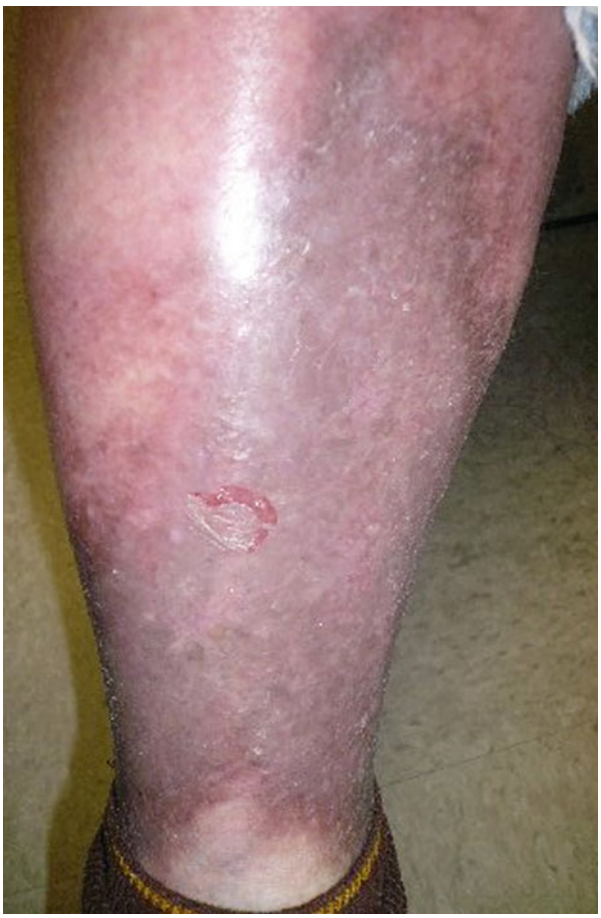


Fig. 29.12 Severe stasis dermatitis with extensive hemosiderin deposits

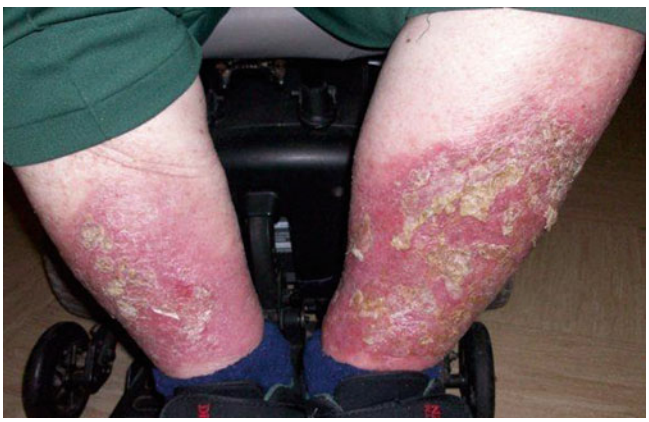


Fig. 29.13 Stasis dermatitis and cellulitis of lower extremities

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Pressure Ulcers

30

Synonyms

Synonyms include decubitus ulcers, pressure sores, and bedsores.

Clinical Description

Pressure ulcers occur over sites of gravitational pressure, most commonly the sacrum, heels, ischia, and femoral trochanters. Any site of chronic pressure may be affected [1–3].

Etiology and Pathophysiology

Extrinsic factors include pressure (constant pressure greater than 32 mmHg, the average capillary pressure at its arterial inflow), shearing, and frictional forces. Intrinsic factors include susceptibility to tissue breakdown from impaired mobility, protein malnutrition, anemia, loss of subcutaneous fat cushion, incontinence, and infection.

Diagnosis and Differential Diagnosis

The clinical situation makes the diagnosis of pressure ulcers straightforward. Other causes of lower extremity ulcers are discussed in Chap. 29. Bacterial cultures showing greater than 100,000 organisms per gram of tissue denote true infection rather than contamination and require antibiotic therapy.

Histopathology

Histopathologic investigation is usually not performed.

Treatment involves various measures:

1. Prevention is extremely important. Good nursing care is essential, with frequent repositioning of the patient's bony prominences, adequate cushioning with wool or foam padding, use of wheelchairs with adjustable, inflatable cushions, special airflow or sand-and-airflow beds, diversion of catheters to prevent tissue breakdown and contamination, and adequate nutrition
2. Negative-pressure wound therapy (NPWT) is a therapeutic technique in wound management that increased dramatically over the 1990s and 2000s. The therapy involves the controlled application of sub-atmospheric pressure to the local wound environment using a sealed wound dressing connected to a vacuum pump to promote healing in acute or chronic wounds and enhance healing of first and second degree burns [4–8].
3. Surgery involves myocutaneous flaps after wound debridement.
4. 4 Antibiotics may be necessary, especially if osteomyelitis develops.

Prognosis

The outlook depends on the patient's potential for remobilization and/or avoidance of constant pressure.

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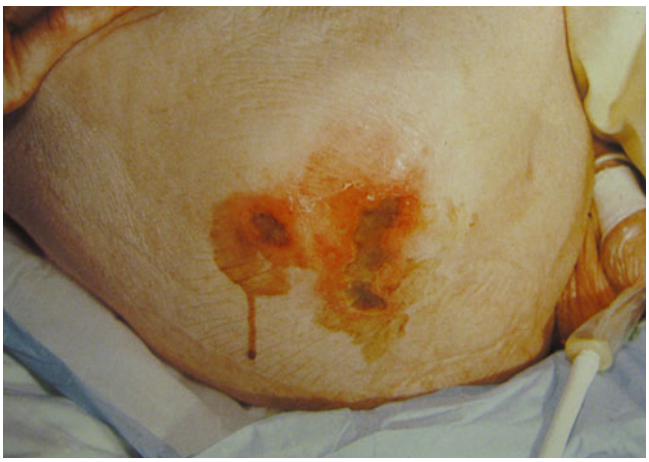
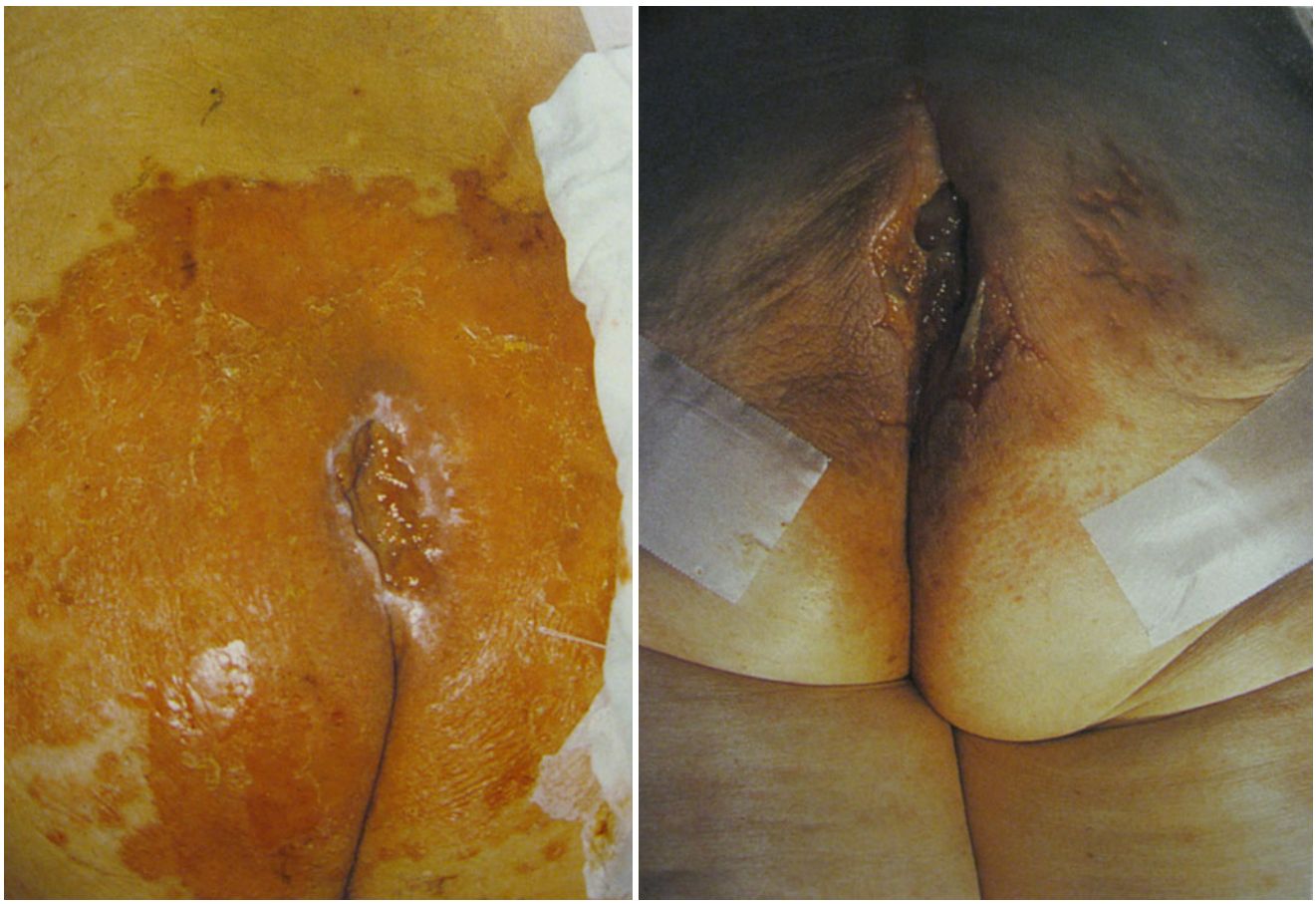


Fig. 30.1 Early pressure ulcer showing erythema, erosion, and crusting. Drainage devices such as catheters must be diverted from pressure points to prevent their contributing to ulcer development



Figs. 30.2 and 30.3 Extensive erosion with central ulcer over the sacrum

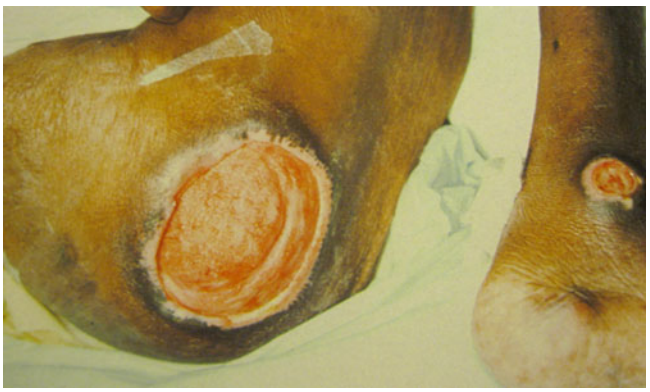


Fig. 30.4 Advanced pressure ulcers of the hip and ankle in a cachectic cancer patient on chemotherapy

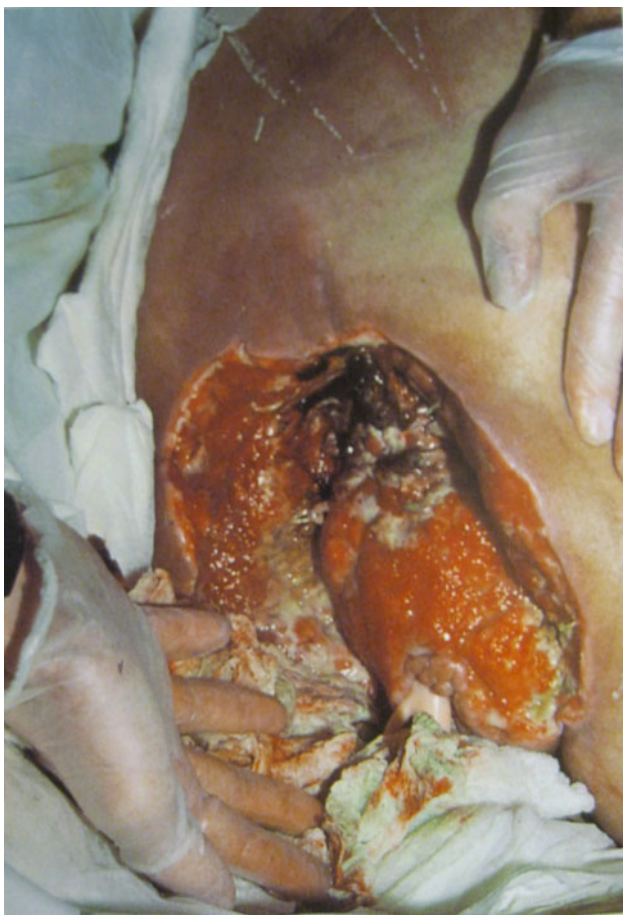


Fig. 30.5 Progression of patient shown in Fig. 30.2, The ulcer has progressed to involve muscle and bone. Osteomyelitis is a life-threatening complication

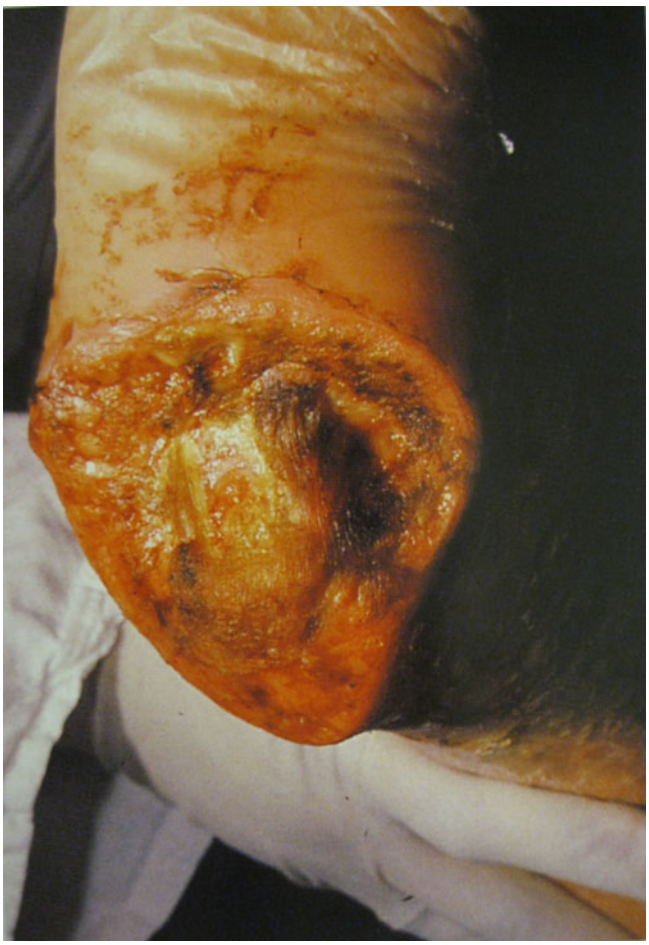


Fig. 30.6 Deep pressure ulcer on the heel of a bedridden patient

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Part VI

Benign Tumors

Chondrodermatitis

31

Synonym

Chondrodermatitis is also known as chondrodermatitis nodularis helicis chronicus (CNHC, CNH).

Clinical Description

A painful or tender pink, firm papule, often ulcerated or crusted, is found on the helix or antihelix of the ear. Rarely, chondrodermatitis can be multiple [1, 2].

Etiology and Pathophysiology

Chondrodermatitis results from the degeneration of epidermis, dermis, and/or cartilage from chronic sun exposure, pressure, trauma with scarring and binding of the dermis to the perichondrium, or other physical trauma (cold injury).

Histopathology

Chondrodermatitis shows irregular acanthosis surrounding a central ulcer filled with degenerated, homogenous dermal collagen, granulation tissue at the periphery, thickened perichondrium, and variable cartilaginous degeneration with focal calcification and, rarely, ossification. Cellular atypia is absent, but pseudo-epitheliomatous hyperplasia (PEH) of overlying epidermis is common and otherwise resembles squamous cell carcinoma [3].

Differential Diagnosis

Clinically, squamous cell carcinoma or keratoacanthoma is not so exquisitely tender. Biopsy is necessary to exclude squamous cell carcinoma.

Therapy

1. Surgical excision must include involved cartilage.
2. Intralesional steroids (triamcinolone acetonide, 40 mg/ml) and liquid nitrogen cryotherapy have limited efficacy.
3. For patients who cannot tolerate surgery, sleep easy CNH Pillow has a hole in the center to allow the ear to rest on air while the neck and shoulders are supported by the contoured cushion.

Prognosis

This is a benign but painful lesion, with no tendency for spontaneous involution.

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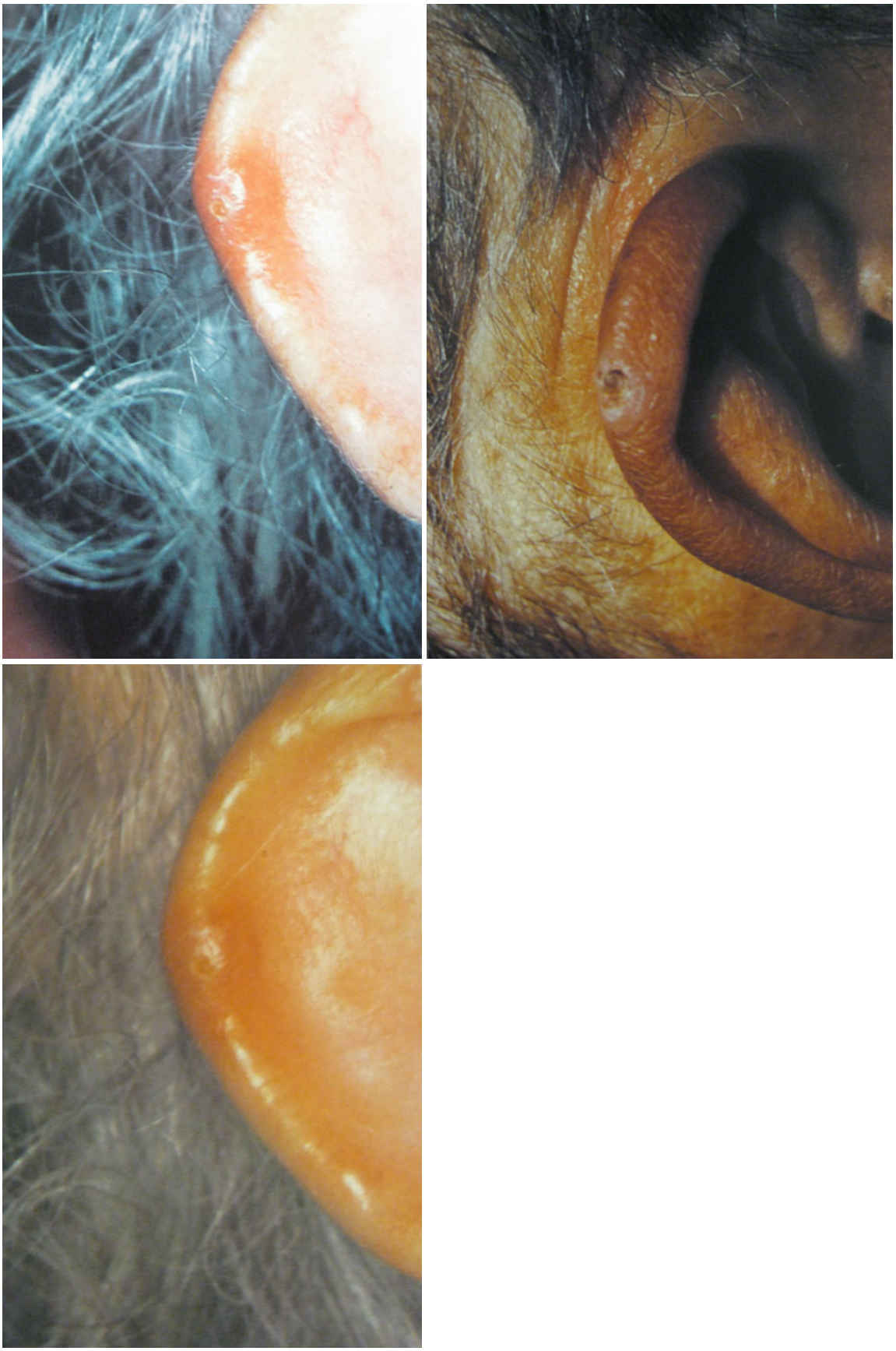

Figs. 31.1, 31.2, and 31.3 Typical presentation of chondrodermatitis nodularis helicis chronicus is an exquisitely tender, painful, eroded or ulcerated papule on the helical rim. The condition can develop elsewhere on the ear from constant mechanical pressure, trauma, or congenital deformity

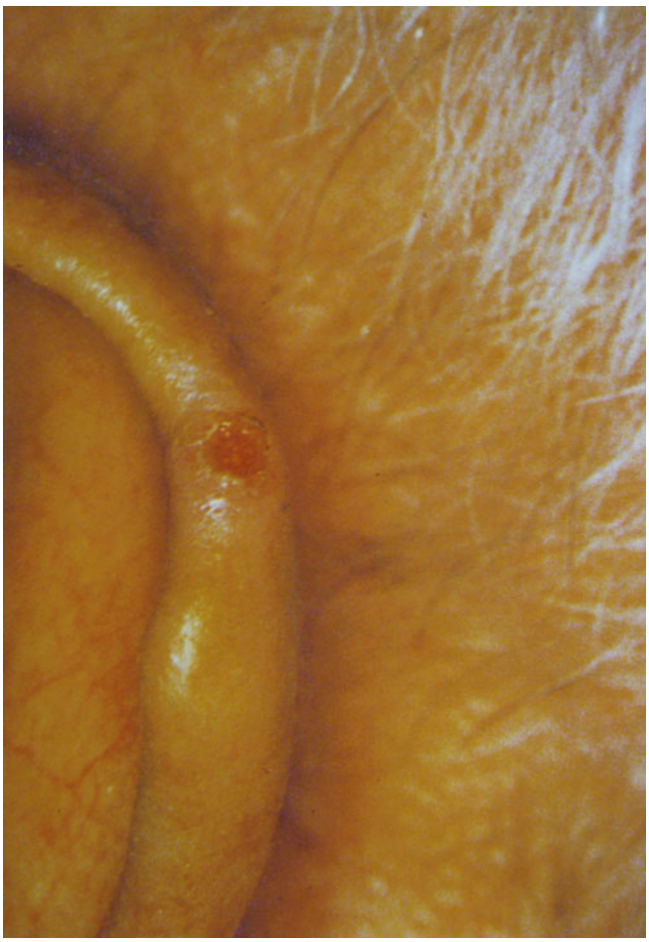

Fig. 31.4 Chondrodermatitis papules may be multiple. Note the development of a second papule inferior to the original chondrodermatitls

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Cysts

32

Clinical Description

Various types of cysts can occur:
1. Epidermal cysts-commonly (erroneously) called "sebaceous" cysts
2. Pilar cysts-wens, atheromas
3. Steatocysts-true sebaceous cysts

Epidermal cysts occur on hair-bearing skin and often demonstrate a surface pore [1]. Pilar cysts occur most commonly on the scalp. Steatocysts occur most commonly on the chest, scrotum, and labia majora as grouped papules or nodules, occasionally draining a yellow, oily, translucent, foul-smelling liquid.

Etiology and Pathophysiology

Most epidermal cysts originate from the follicular infundibulum (upper third). True epidermal inclusion cysts, resulting from traumatic implantation or inclusion of epidermis into dermis, are rare. All three types of cysts may occur sporadically or as an inherited tendency, particularly autosomal dominant steatocystoma multiplex and autosomal dominant Gardner's syndrome. The latter is associated with colonic and ovarian malignancies.

Histopathology

Histopathologic investigation reveals the following:
1. Epidermal cyst--cyst wall consists of stratified squamous epithelium containing a granular layer surrounding lamellated orthokeratin.
2. Pilar cyst--cyst wall consists of trichilemmal epithelium without a granular layer surrounding compact keratohyalin. Mixed epidermal-pilar cysts can also occur.
3. Steatocyst--cyst wall consists of stratified squamous epithelium with corrugated, eosinophilic cuticle containing compressed sebaceous glands and oily sebaceous material centrally.

Differential Diagnosis

Differential diagnosis includes the following:
1. Epidermal cyst--lipomas usually are less discrete, more lobular, and lack a central pore. Dermoid cysts occur in the periorbital region or in anatomic cleavage planes; they contain mature epidermal appendages, including hair and sebaceous glands. Pilonidal cysts occur in the gluteal cleft.
2. Pilar cyst-cylindroma ("turban tumor") is usually multiple and can occur in association with facial trichoepitheliomas. Pilomatrixoma (calcifying epithelioma of Malherbe) is a blue-gray, hard nodule.
3. Steatocystoma-scrotal epidermal cysts are often calcified; eruptive vellus hair cysts occur on the trunk as smaller, keratotic papules containing vellus hairs rather than sebaceous glands [2–4].

Therapy

1. Excision. For small cysts, punch biopsy with evacuation of cyst contents and sac, if possible, results in minimal scarring.
2. Incision and drainage with curettage. This must be performed on clinically infected, fluctuant cysts [5].

Prognosis

Rarely, malignant degeneration to squamous cell carcinoma has been reported in epidermal cysts.

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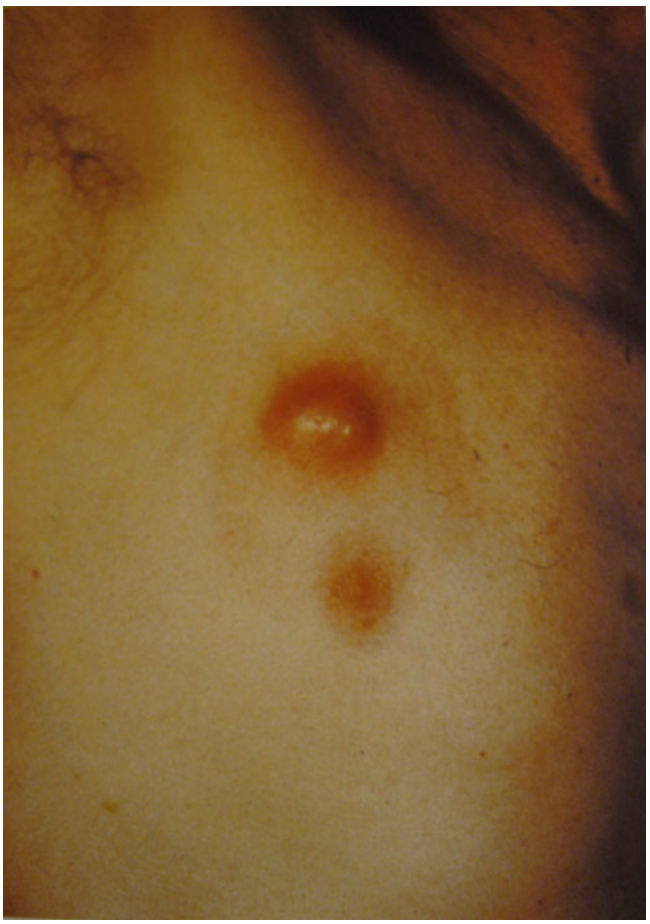

Fig. 32.1 Abscessed epidermal cyst

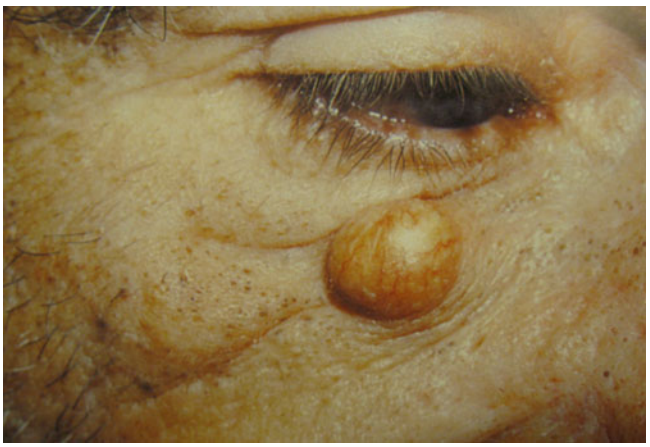

Fig. 32.2 Large epidermal cyst in a patient with Favre-Racouchot syndrome (nodular elastosis with cysts and comedones). Note open comedones flanking the cyst

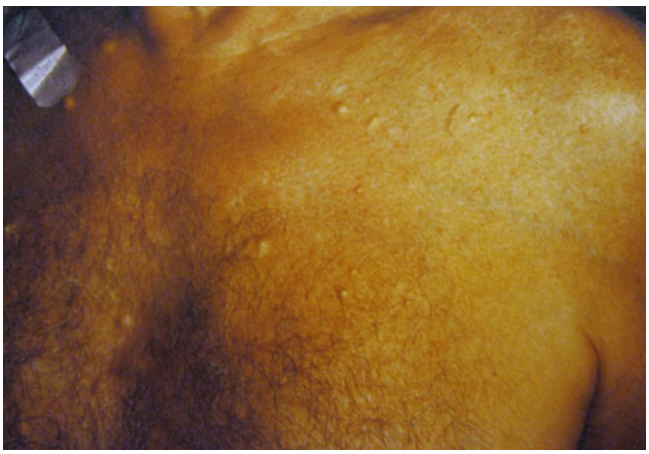

Fig. 32.3 Steatocystoma multiplex is an autosomal dominant condition with multiple yellow cysts containing oily, foul-smelling fluid

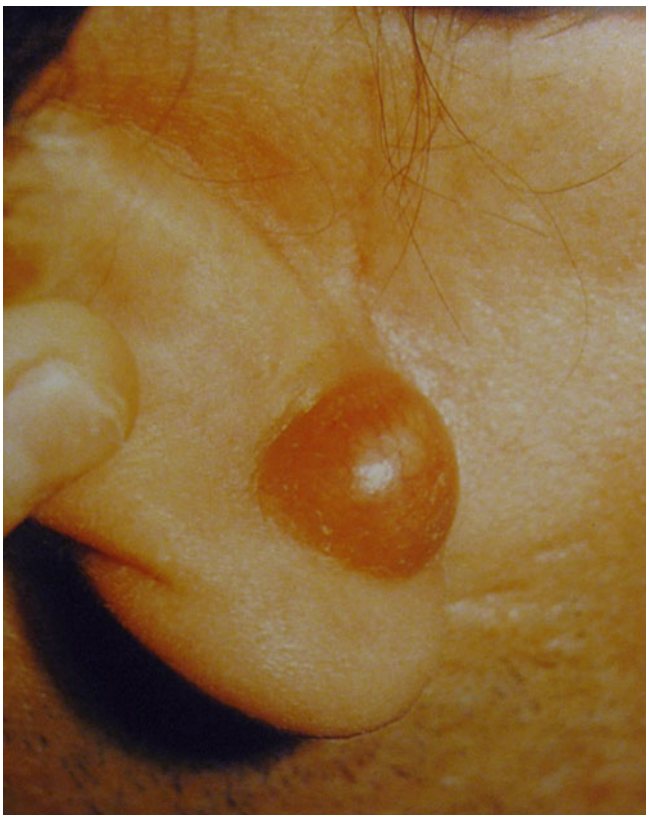

Fig. 32.4 Pilar cyst (wen) is most common on the scalp, here shown on the ear

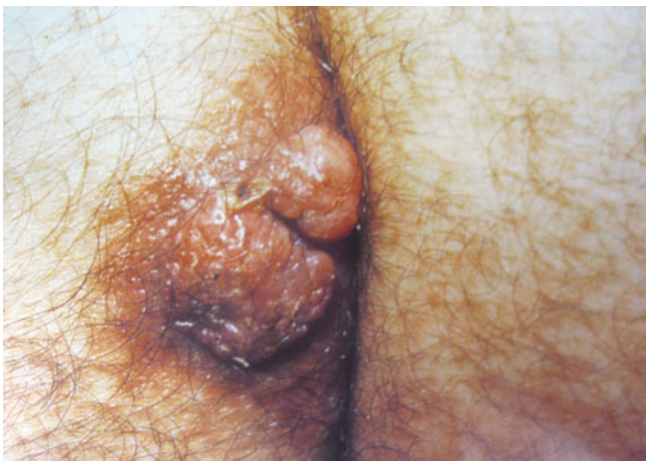

Fig. 32.5 Pilonidal [literally, "hair nest") cyst on the buttocks begins as an ingrown hair leading to foreign body reaction and even sinus tract formation. This condition is more common in hairy individuals. Therapy is surgical marsupialization or complete excision

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Digital Myxoid Cyst and Ganglion

33

Synonyms

Synonyms include mucinous cyst and synovial cyst.

Clinical Description

A digital myxoid cyst is a translucent, blue, cystic papule on the digits, especially around the dorsal distal interphalangeal joint or phalanx. A ganglion is a subcutaneous cystic nodule that moves with the attached tendon [1, 2].

Etiology and Pathophysiology

Two subtypes of digital myxoid cysts are seen:

1. Traumatic herniation of synovial sac, with extrusion of mucin into dermis
2. Overproduction of mucin (hyaluronic acid) by fibroblasts, with underproduction of collagen (focal mucinosis)

A ganglion involves herniation of the synovial lining of a tendon or joint [3].

Histopathology

Mucinous deposits occur in multiple clefts or coalesce into a large cystic mass. No cyst lining is seen in the dermis but a stalk may connect to the joint space.

Differential Diagnosis

Mucinous cyst and ganglion have a distinctive clinical appearance. Eccrine and apocrine hidrocystomas are much smaller and are located periorbitally. Giant cell tumor of the tendon sheath and exostosis are solid, not cystic.

Therapy

1. Drainage of mucin followed by injection with triamcinolone acetonide, 4–10 mg/ml. and compression
2. Hand surgery with exploration of joint or tendon and excision of cystic pedicle in refractory cases [4, 5]

Prognosis

A digital myxoid cyst or ganglion is stable, with little tendency for spontaneous resolution and no tendency for malignant degeneration.

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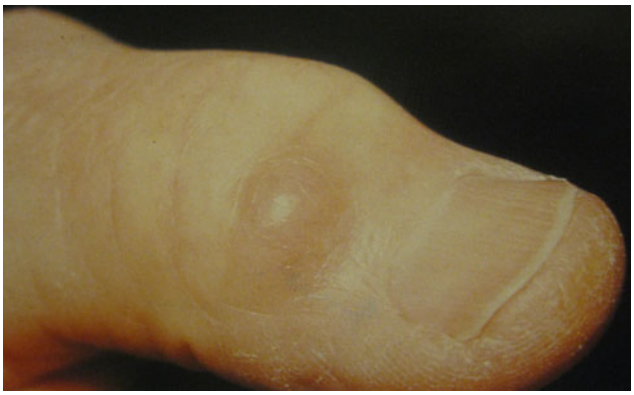

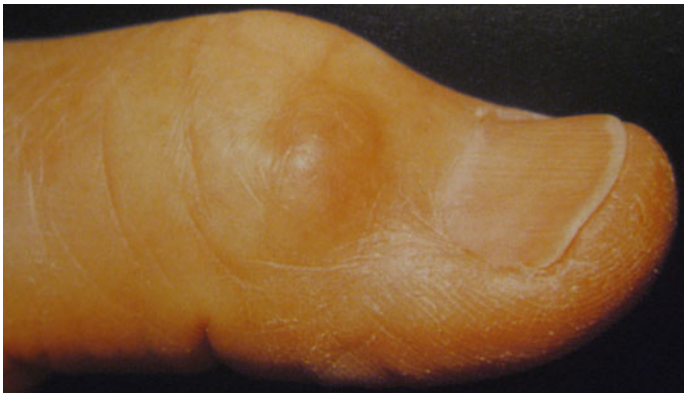

Figs. 33.1 and 33.2 Digital mucinous cyst. This is a firm, translucent, blue cystic papule overlying the distal interphalangeal joint

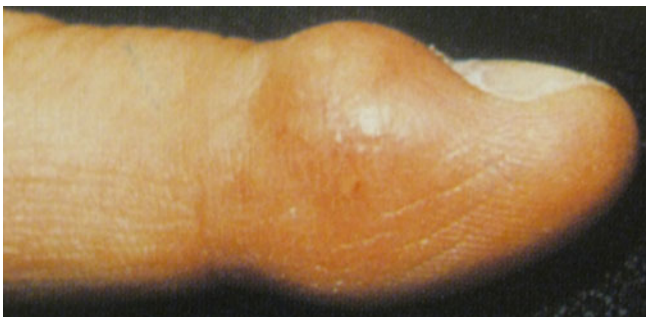

Fig. 33.3 Giant cell tumor of the tendon sheath. This is a firm nodule that usually occurs at the distal phalanx. It can be confused with a digital mucinous cyst

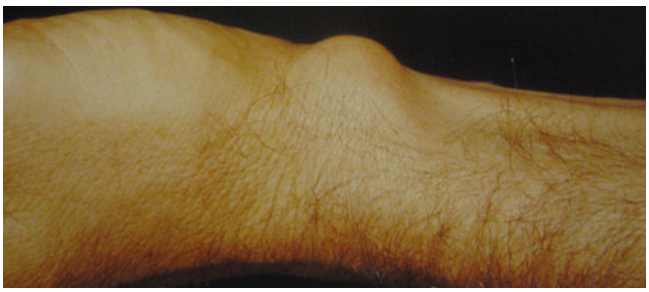

Fig. 33.5 Ganglion of the volar wrist. These lesions have been treated by smashing with the Bible, although surgical techniques have lower recurrence rates

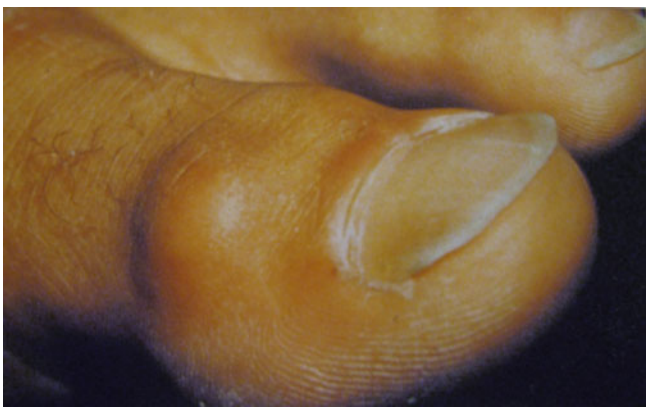

Fig. 33.4 Exostosis. This is a bony overgrowth produced by trauma, with reactive hyperplasia of the periosteum and underlying bone

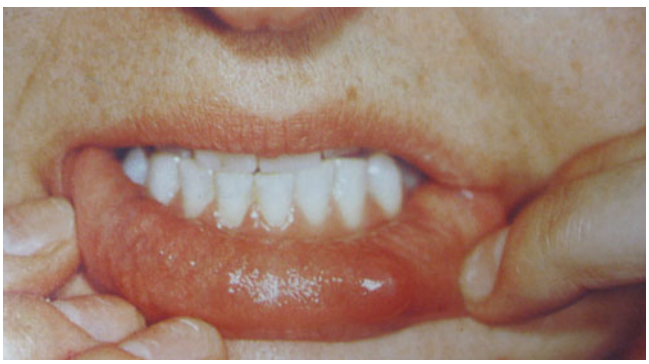

Fig. 33.6 Mucocele of inner lip. This results from a blocked salivary gland, with retention of mucus

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Fibrous Papule

34

Synonyms

Fibrous papule is also known as perinasal papule and perinasal fibroma.

Clinical Description

Fibrous papule is a firm, smooth, dome-shaped, skin-colored, yellow, red, or pigmented 1–4-mm papule on the nose or perinasal skin. It is clinically significant because of its resemblance to a small basal cell carcinoma [1].

Etiology and Pathophysiology

Fibrous papule has been proposed to represent an angiofibroma, regressing dermal nevus, or histiocytoma. Immunoperoxidase studies to elucidate the precise cellular origin have yielded conflicting results. Fibrous papule might represent a final common end stage of several different lesions.

Histopathology

Fibrous papule may show the following: features of an angiofibroma (spindle-shaped, plump, stellate, dermal fibroblasts, vascular dilatation, numerous vellus hair follicles with concentric coarse collagen fibers, occasional dermal melanophages, and increased basal melanocytes); a fibrosing melanocytic nevus; or a perifollicular fibroma (coarse perifollicular collagen and fibroblasts) [2–5].

Differential Diagnosis

Differential diagnosis of central facial papules includes the following: basal cell carcinoma, dermal melanocytic nevus, milia (tiny epidermal cysts), angiofibromas of tuberous sclerosis (multiple, in younger patients), sebaceous gland hyperplasia, syringomas (benign eccrine sweat gland cysts), trichoepithelioma, sarcoidosis, granulomas of papular rosacea, trichilemmomas of Cowden (multiple hamartoma syndrome), and solar elastomas.

Therapy

No treatment is necessary. Shave biopsy is sufficient for cosmetic treatment and to rule out basal cell carcinoma

Prognosis

The prognosis is good. This benign lesion sometimes undergoes spontaneous involution.

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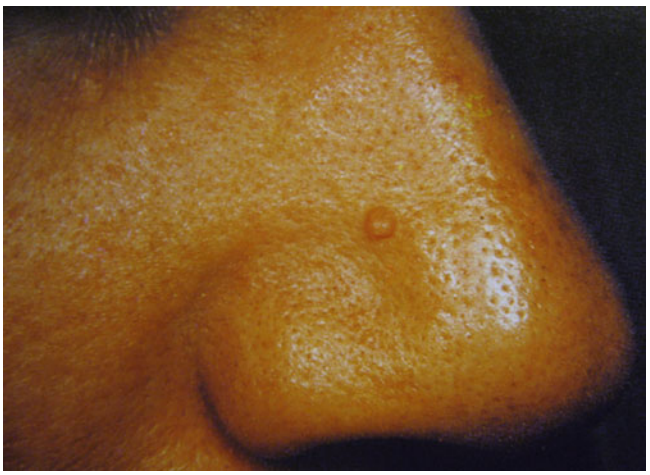

Fig. 34.1 This biopsy-proven fibrous papule of the nose tip resembles a small basal cell carcinoma, sebaceous hyperplasia, or solar elastoma

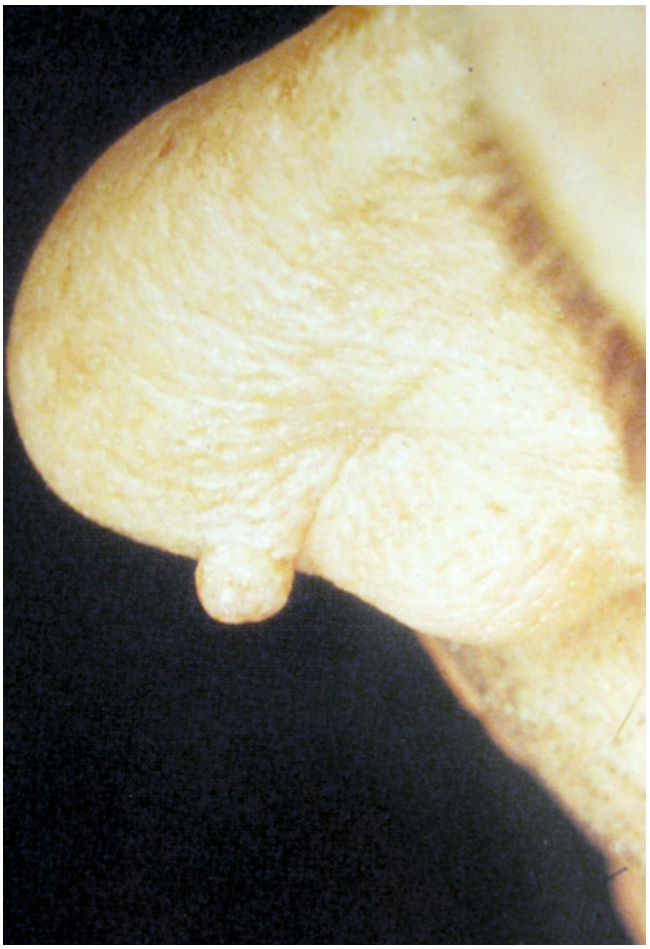

Fig. 34.2 This fibrous papule of the nasal rim resembles a pedunculated dermal nevus, acrochordon, or molluscum contagiosum

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Seborrheic Keratoses

35

Entities and Synonyms

These include the following:
1. Seborrheic keratoses-"senile" warty keratoses
2. Benign lichenoid keratosis-lichen planus-like keratosis
3. Flegel's keratosis-hyperkeratosis lenticularis of Flegel

Clinical Description

Seborrheic keratosis is a brown-gray, verrucous papule or plaque most common on the trunk. Stucco keratoses are smaller, gray-white, warty papules on the legs and ankles. Benign lichenoid keratosis is a pink-red, smooth, flat-topped or domed papule, often pruritic. Flegel's keratosis is a flat-topped, hyperkeratotic papule, 1–5 mm in diameter, on the dorsal feet and lower legs [1, 2].

Etiology and Pathophysiology

Seborrheic keratosis has a hereditary predisposition and may develop from solar lentigo. The sudden onset of multiple, pruritic, seborrheic keratoses (the Leser-Trelat sign) and linear seborrheic keratoses may signal underlying malignancy.

Benign lichenoid keratosis represents an inflamed seborrheic keratosis.

Flegel's keratosis has autosomal dominant inheritance in some cases.

Histopathology

Histopathologic investigation reveals the following:
1. Seborrheic keratosis-verrucous acanthosia, hyperkeratosis, horn pseudocysts, horizontal epidermal base
2. Benign lichenoid keratosis-as above, with lichenoid, lymphohistiocytic, high-hugging infiltrate, parakeratosis, occasional eosinophils
3. Flegel's keratosis-hyperkeratosis, flattened epidermis with thin or absent granular cell layer centrally, "church spire" epidermal papillomatosis peripherally, dermal band-like lymphocytic infiltrate

Differential Diagnosis

The differential diagnosis includes the following:
1. Seborrheic keratosis-nevi, melanoma, verrucae
2. Benign lichenoid keratosis-basal cell carcinoma, lichen planus, dermal nevus
3. Flegel's keratosis-lentigines, early seborrheic keratoses, parakeratosis

Prognosis

The prognosis is for seborrheic keratosis and Flegel's keratosis is stable. Benign lichenoid keratosis often resolves spontaneously.

Therapy

No treatment is necessary unless the lesion is cosmetically bothersome or diagnostically questionable:
1. Surgery-superficial tangential shave excision provides material for microscopic confirmation, which is mandatory for clinically atypical or suspicious lesions.
2. Chemical peeling agents-trichloroacetic acid, tretinoin, ammonium lactate, or an alpha-hydroxy add, can be used.
3. Delicate electrocautery, CO_2 laser vaporization, or cryotherapy can be carried out when the diagnosis is certain.

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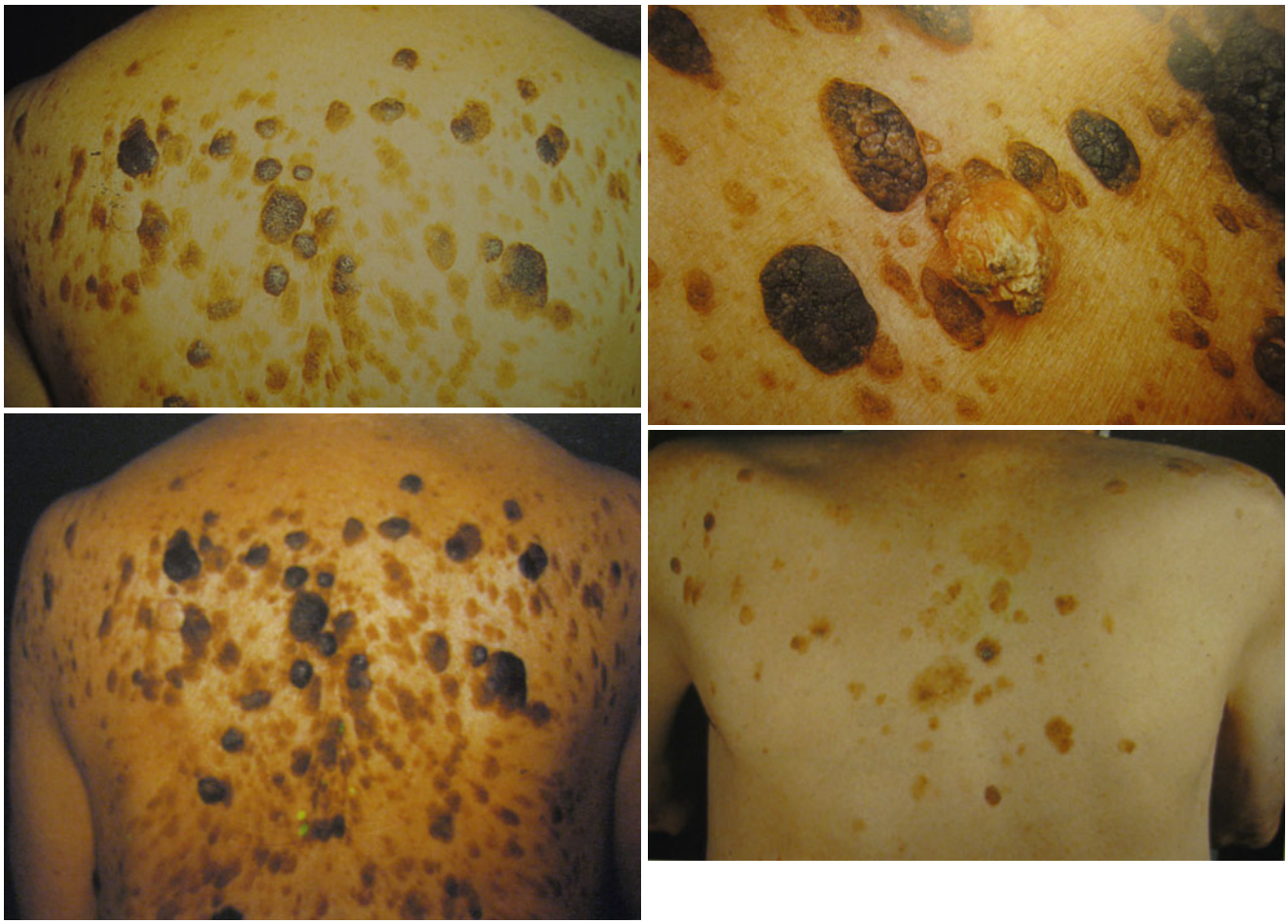
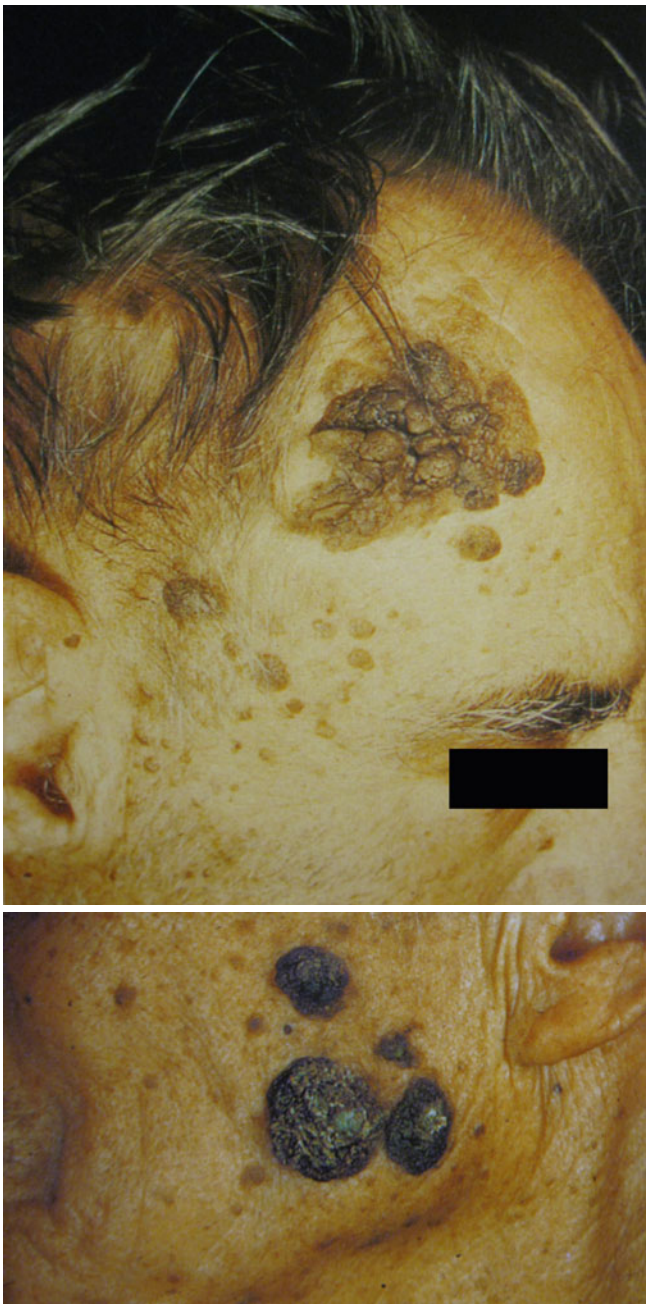


Figs. 35.1, 35.2, 35.3, and 35.4 Multiple, brown-black, seborrheic keratoses. The central red keratotic nodule in Fig 35.2 is a keratoacanthoma

Fig. 35.5 Close-up view of a seborrheic keratosis showing "stuck on" appearance, with surface invaginations filled with keratotic plugs



Figs. 35.6 and 35.7 Seborrheic keratoses of the face-a cosmetic blight

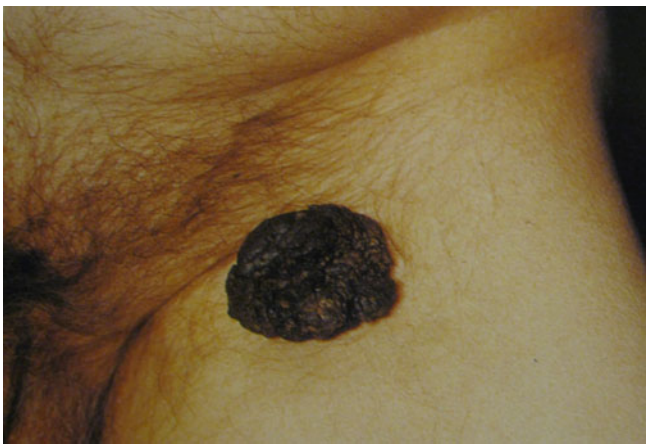


Fig. 35.8 Giant seborrheic keratosis of the groin is a benign simulator of melanoma. The lesion can become irritated by maceration and rubbing against skin folds, so it should be removed

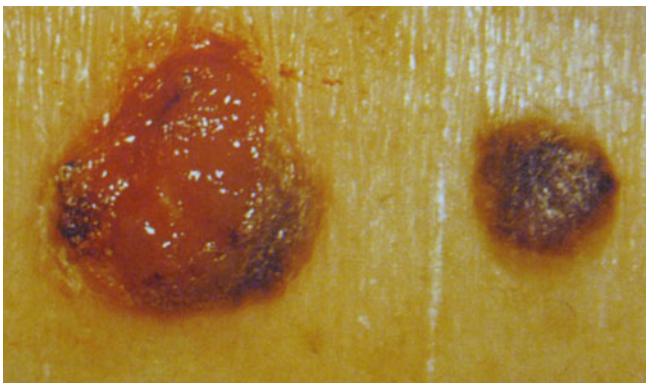


Fig. 35.9 A rare complication. Infection developed in the seborrheic keratosis that the patient tried to scratch off

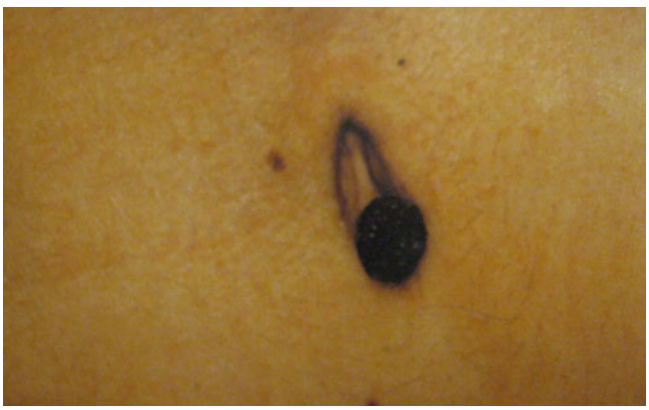
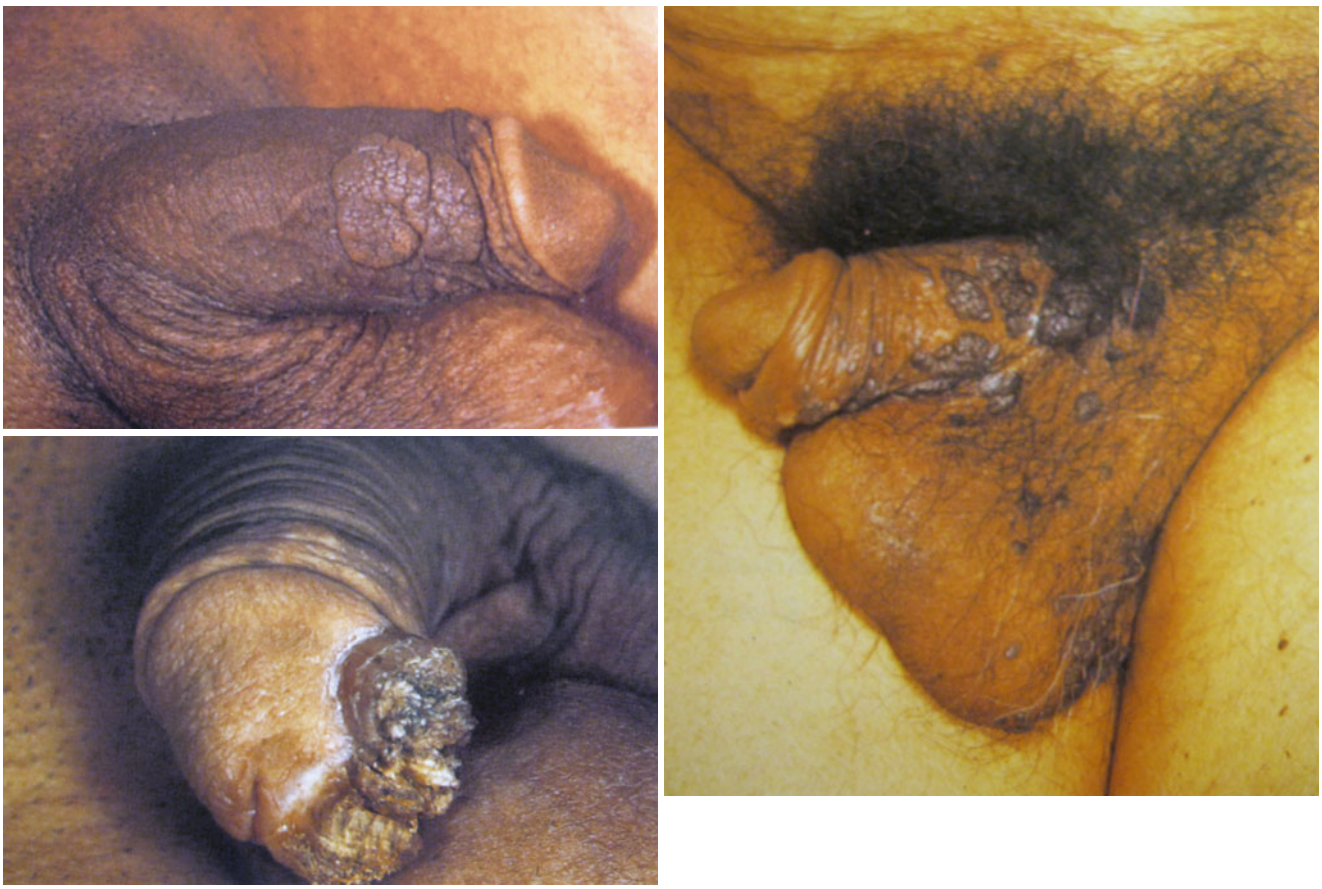


Fig. 35.10 Seborrheic keratosis in the umbilicus. Differential diagnosis includes Sister Mary Joseph's nodule, which is a cutaneous metastasis from an intra-abdominal carcinoma, usually of the colon, stomach, or pancreas (*see* Chap. 26)



Figs. 35.11, 35.12, and 35.13 Seborrheic keratoses of the penis simulate genital warts (Figs. 35.11 and 35.12) or even squamous cell carcinoma (Fig. 35.13)

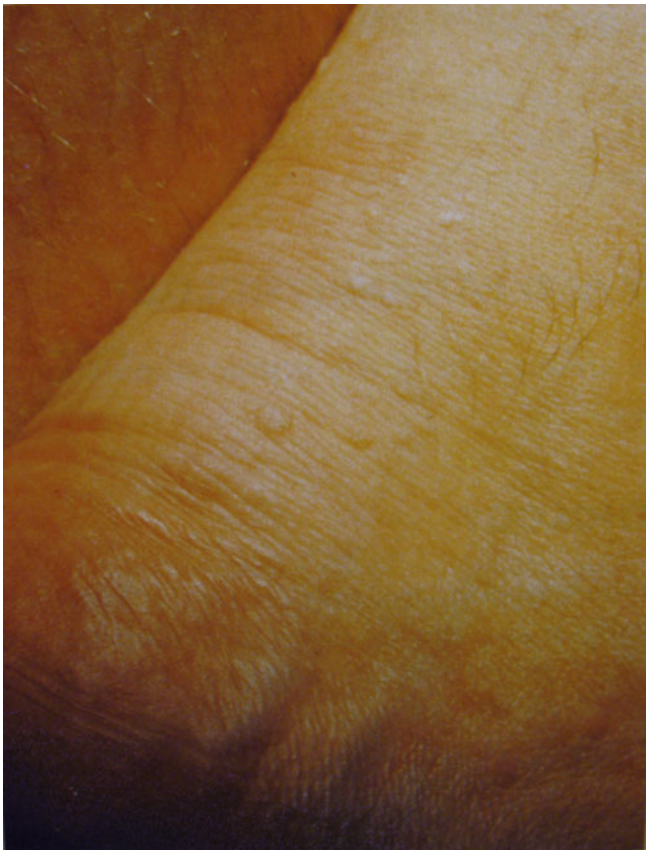


Fig. 35.14 Stuccokeratoses are small, flat-topped, gray-white seborrheic keratoses that clinically resemble flat warts (verrucae plana), but usually occur on the lower legs or feet

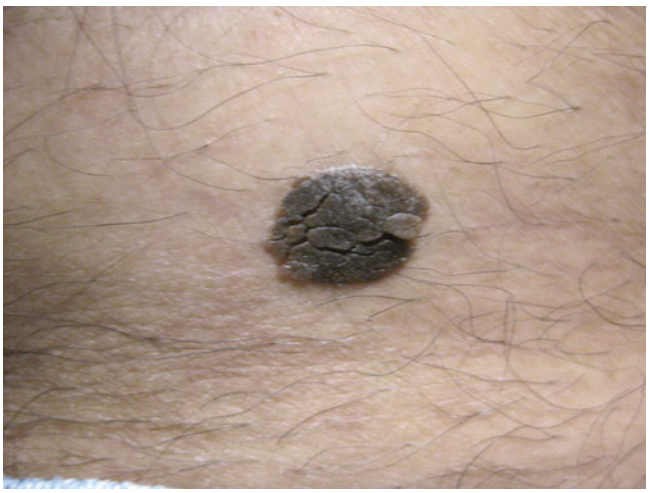


Fig. 35.15 Classic appearance of seborrheic keratosis

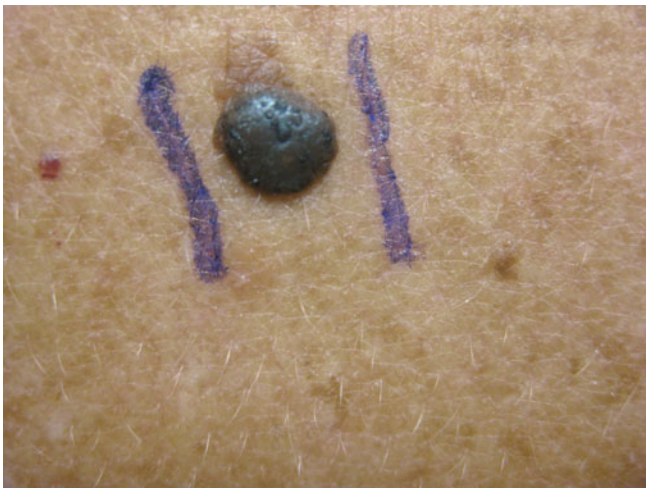


Fig. 35.16 66-year-old white female presented with a growth on her mid superior back. Examination showed an oval tan lesion with dome shaped dark brown lesion attached. The wall of the dome contained multiple micronodules

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Lentigines

36

Synonyms

Synonyms for lentigines include "liver" or "age" spots, lentigo senilis, and solar lentigo.

Clinical Description

Lentigines are smooth, brown macules and patches that are concentrated on the sun-exposed face, arms, and dorsal hands [1, 2].

Etiology and Pathophysiology

Lentigines are a result of excessive melanin pigment due to solar (actinic) stimulation of melanocytes.

Histopathology

Lentigo shows hyperpigmentation of the basal cell layer, especially at the tips of elongated, club-shaped, epidermal rete ridges, with variable melanocytic hyperplasia.

Differential Diagnosis

Freckles (ephelides) and junctional nevi occur in younger patients; freckles fade in winter, whereas lentigines persist. Pigmented actinic keratosis and seborrheic keratosis have a rough surface. Large lentigines may degenerate into lentigo maligna, and then into lentigo maligna melanoma.

Therapy

Treatment includes the following:

1. Sun avoidance, avoid tanning parlors [3–5]
2. Regular use of sunscreens
3. Bleaching creams and solutions, such as hydroquinone
4. Light chemical peel with trichloroacetic acid, tretinoin
5. Liquid nitrogen cryotherapy and laser ablation (note that permanent hypopigmentation and/or hyperpigmentation can occur)

Prognosis

Lentigines are generally benign. Lentigo may, however, develop into a reticulated seborrheic keratosis.

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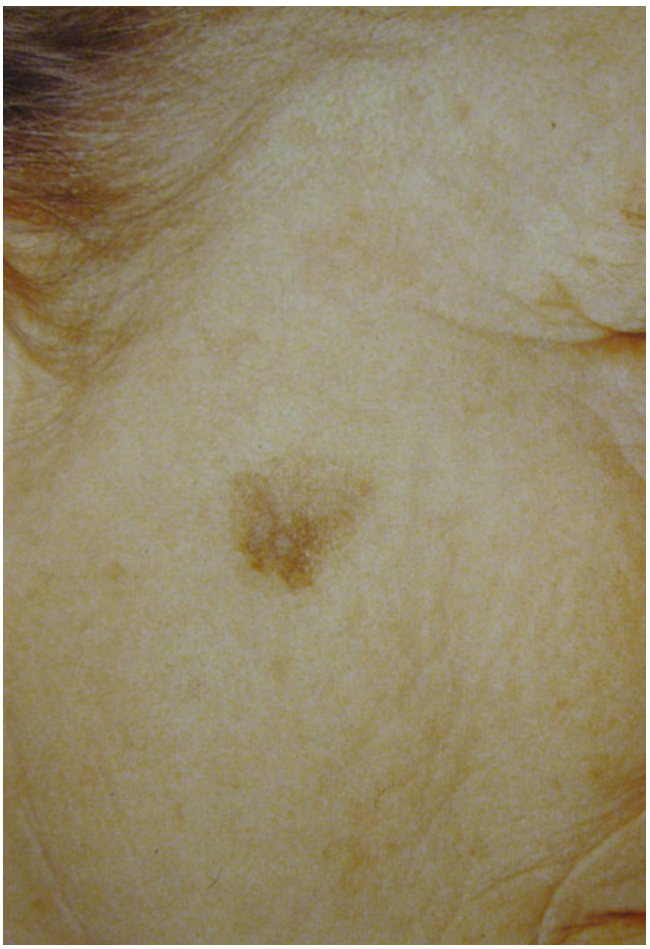

Fig. 36.1 Solar ("senile") lentigo is typically a uniform brown macule or patch. This cheek lesion shows two shades of brown and developed a papular component inferiorly. Biopsy to rule out lentigo maligna or lentigo maligna melanoma showed a benign, seborrheic keratosis instead

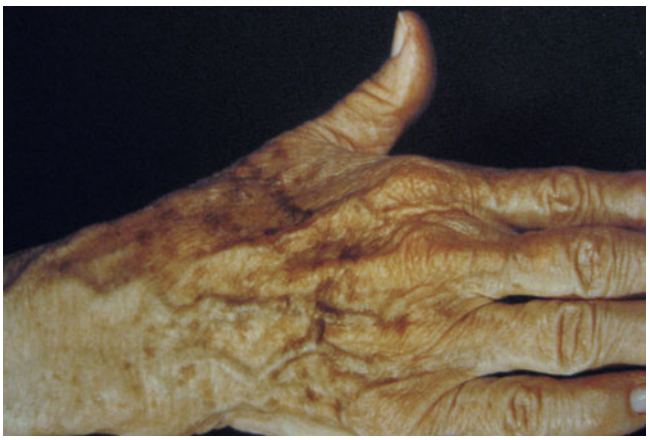

Fig. 36.2 Solar lentigines of the hands are commonly (erroneously) termed "age" or "liver" spots. They are age-related but are a result of chronic sun exposure, and have nothing to do with the liver

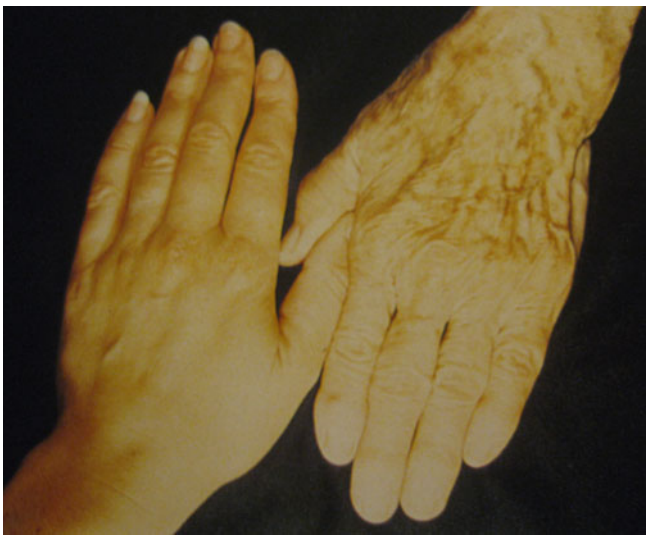

Fig. 36.3 Compare this young woman's plump, unblemished hand with the photoaged, atrophic, pigmented hand of her grandmother. Gloves and sunscreens can prevent some of these changes

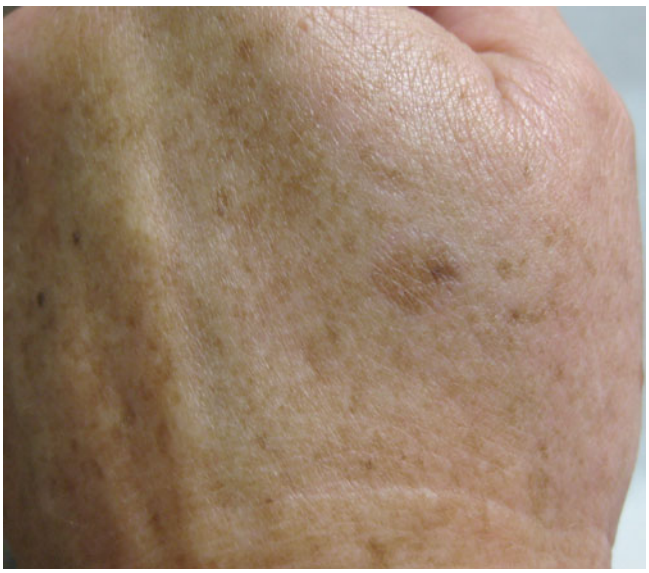

Fig. 36.4 64-year-old white female presents with a slow growing spot on the back of the left hand. Examination shows a well demarcated macule with a uniform tan appearance

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Benign Vascular Lesions

37

Clinical Conditions

Benign vascular lesions include various clinical conditions:

1. Cherry angiomas (De Morgan's spots) are bright red papules, 1–5 mm in diameter. These capillary hemangiomas occur with advancing age, independent of sun exposure
2. Actinic, senile, traumatic, or Bateman's purpura appears as purple, nonblanching patches (ecchymoses), 1–5 cm in diameter that develop on thin, usually sun-damaged, skin of the dorsal forearms and hands. Minimal trauma is required to produce these ecchymoses; trauma may occur while the patient is sleeping. Patients usually deny or disbelieve that trauma is responsible. Ecchymoses resolve more slowly than typical bruises and may heal with white, stellate pseudo scars caused by tearing of the dermis.
3. Telangiectases, or "spider veins" of the face and legs, are bright red, blue, or purple blanchable, dilated capillaries and postcapillary venules. "Spider nevi" are angiomas with a dilated pulsatile center and several thin branches that radiate outward.
4. Venous lakes are blue-purple, nonblanching, compressible, collapsible papules or macules, 1–5 mm in diameter. that typically occur on the lower lip or helical rim of the ear. They represent dilated postcapillary venules.
5. Angiokeratomas (Fordyce lesions) are 1–4-mm blue-black papules, 1–4 mm in diameter, that commonly occur on the scrotum but may extend to involve the penis, lower abdomen, and thighs [1, 2].

Etiology

All these benign vascular lesions increase in frequency with age:

1. Cherry angiomas and angiokeratornas occur on sun-protected and on sun-exposed sites.
2. Actinic purpura is a traumatic ecchymosis, primarily occurring on sun-damaged skin.
3. Spider veins of the leg are related to increased intravascular pressure; incompetent perforating veins connect the deep and superficial venous systems. Spider veins and spider nevi of the face are partly familial (genetic) and are exacerbated by facial flushing from heat, spicy foods, hot beverages, alcohol, sun exposure, estrogens, and liver disease.
4. Venous lakes are produced by dilated, thin-walled venules in a relatively acellular dermis. Trauma may play a role.
5. Angiokeratomas are dilated veins, sometimes with a hyperkeratotic epidermis related to increased venous pressure [3–6].

Diagnosis and Differential Diagnosis

The distinctive clinical appearance usually obviates the need for biopsy. Thrombosed hemangiomas and angiokeratomas may appear black and require biopsy to exclude melanoma. Cutaneous angiosarcoma may resemble actinic purpura but occurs preferentially on the face and scalp, rather than on the forearms and hands, and fails to resolve.

Therapy

Therapy is primarily for cosmetic benefit, although spider veins of the legs may progress to frank varicose veins that require vein stripping.

1. Cherry hemangiomas, venous lakes, angiokeratomas, and spider telangiectases of the face respond to light electrodesiccation or argon laser therapy.
2. Actinic purpura has no satisfactory therapy. Creams containing vitamin K (phytadione) and vitamin C (ascorbic acid) have shown occasional success.

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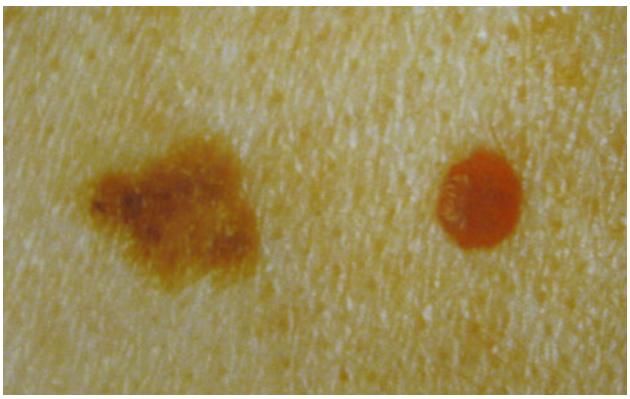
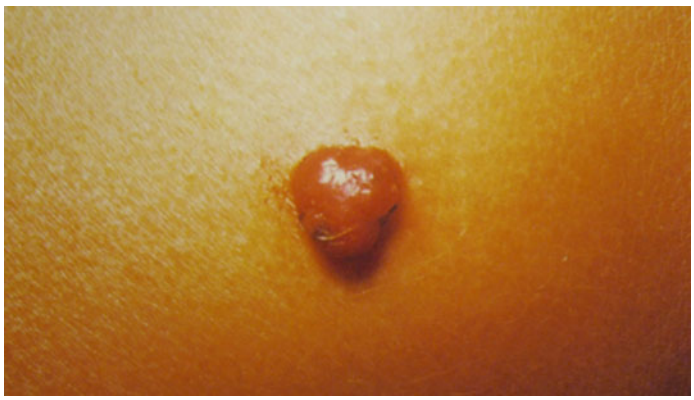
3. Vitamin C with bioflavinoids and zinc have been advised for patients whose diets are deficient in these wound-healing nutrients. Scurvy (vitamin C deficiency) produces perifollicular purpura. Patients must wear protective long-sleeved shirts or blouses and gloves to minimize trauma. Cosmetic concealers can be applied to camouflage unsightly ecchymoses.
4. Spider veins of the leg are usually treated with injectable sclerosing agents (hypertonic saline, polidocanol, aethoxysklerol) and compression. A thorough evaluation for and treatment of incompetent perforating vessels will lessen recurrence [7–9].

Prognosis

These lesions are benign, with no tendency for malignant degeneration. Spider veins of the legs may recur or progress to painful varicosities.





Figs. 37.1 and 37.2 Red "cherry" or capillary hemangioma adjacent to a pigmented melanocytic nevus

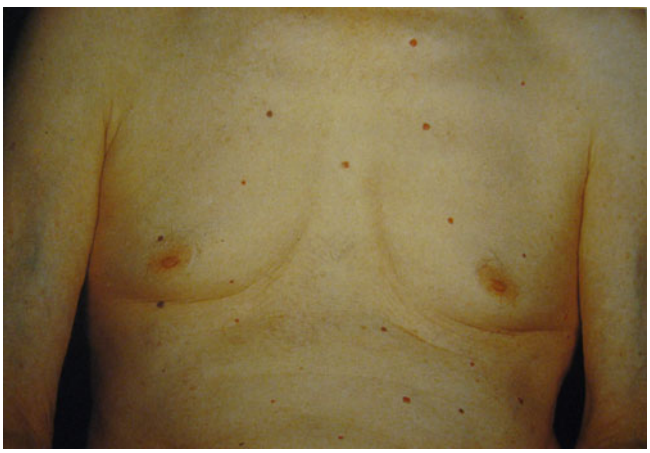
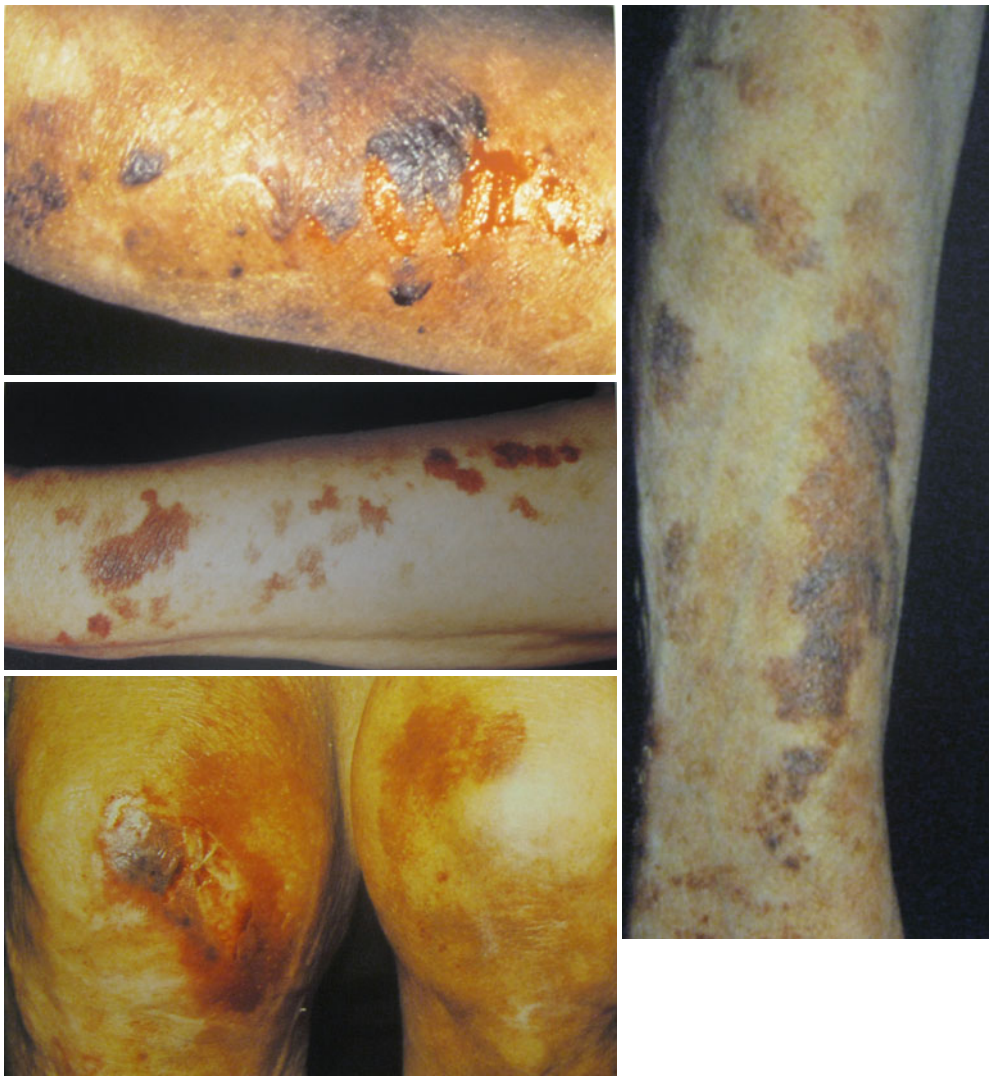


Fig. 37.3 Multiple capillary hemangiomas on the trunk occur with advancing age (De Morgan's spots)



Figs. 37.4, 37.5, 37.6, and 37.7 Actinic, traumatic, or Bateman's purpura occurs commonly on the arms and dorsal hands. It represents bruising resulting from fragile vessels, attenuated dermis, and atrophic subcutaneous fatty cushion

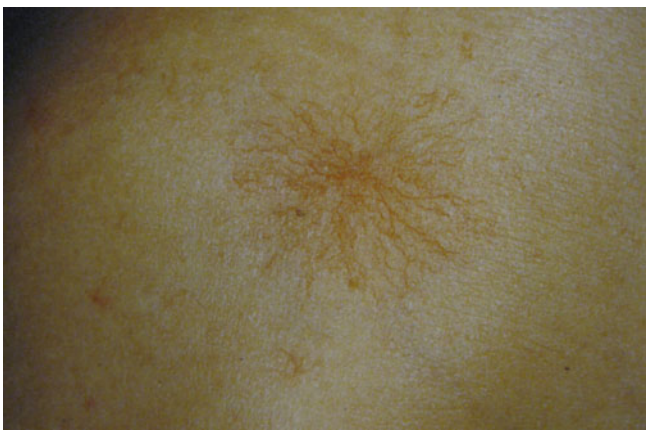
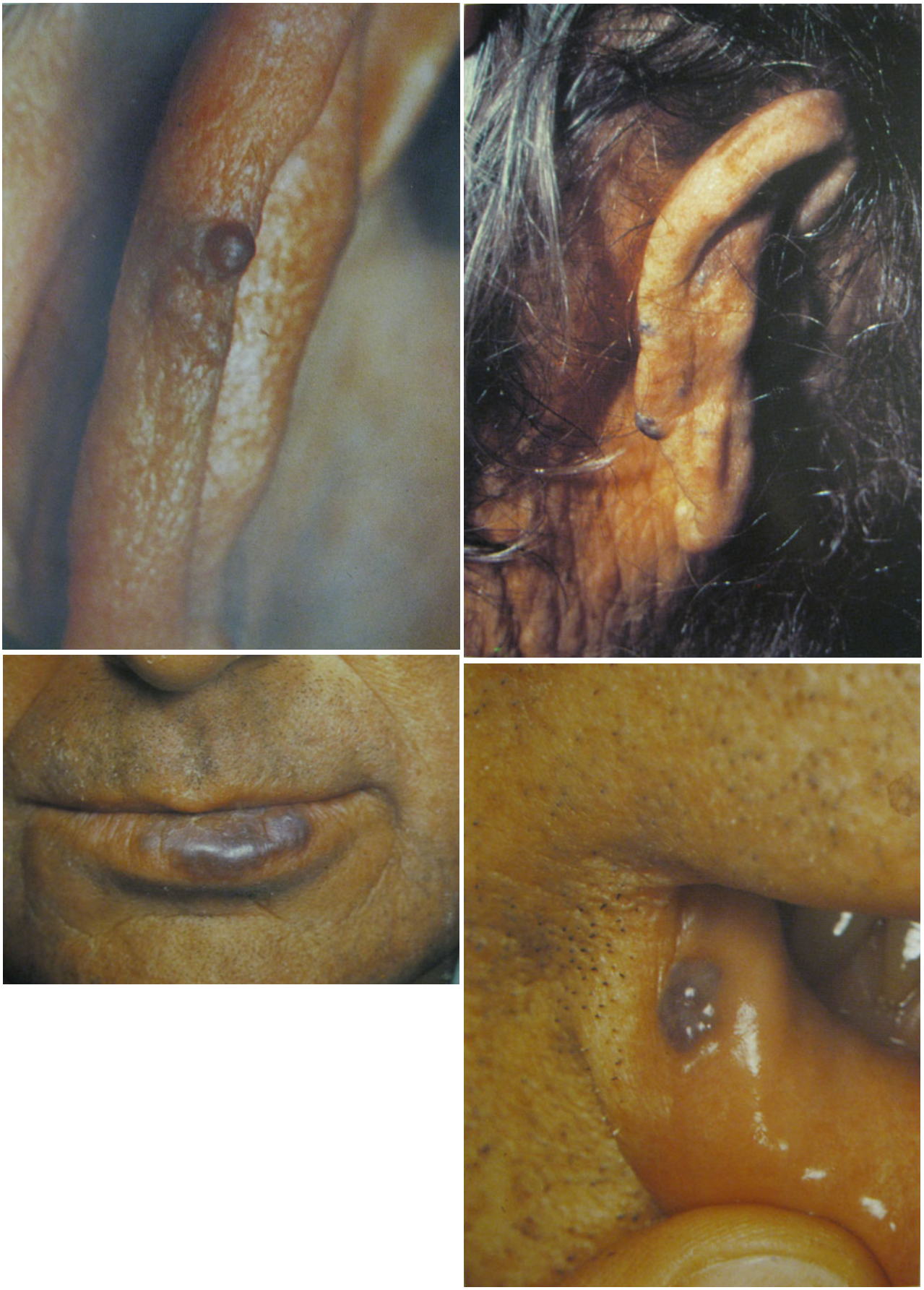


Fig. 37.8 Spider hemangioma (nevus araneus) demonstrates a central red papule with fine, radiating vessels. Its resemblance to a spider explains the common term "spider veins"



Figs. 37.9, 37.10, 37.11, and 37.12 Venous lakes of the ear (Figs. 37.9 and 37.10) and lip (Figs. 37.11 and 37.12) are soft, compressible, blue papules. Treatment, if desired, is electrocautery

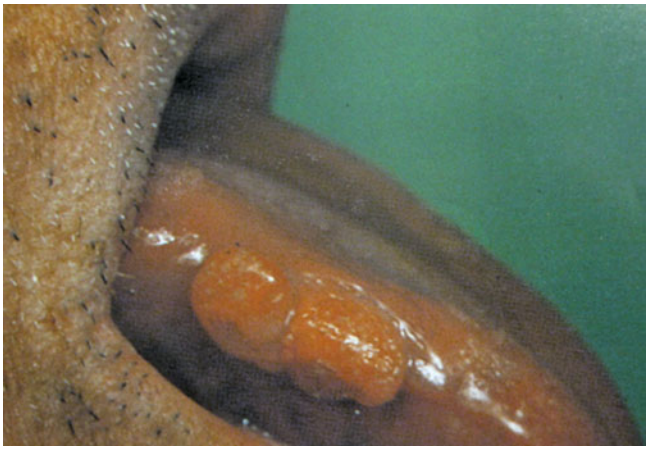


Fig. 37.13 Benign hemangioma of the tongue was suspected of being a squamous *cell* carcinoma

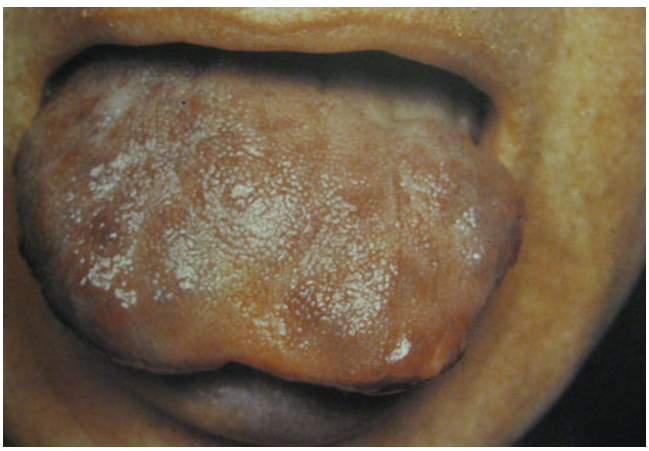


Fig. 37.14 Diffuse hemangioma of the tongue. Multiple hemangiomas suggest the possibility of rare derrnatoloqic syndromes, including blue rubber bleb "nevus" (tender hemangiomas) syndrome

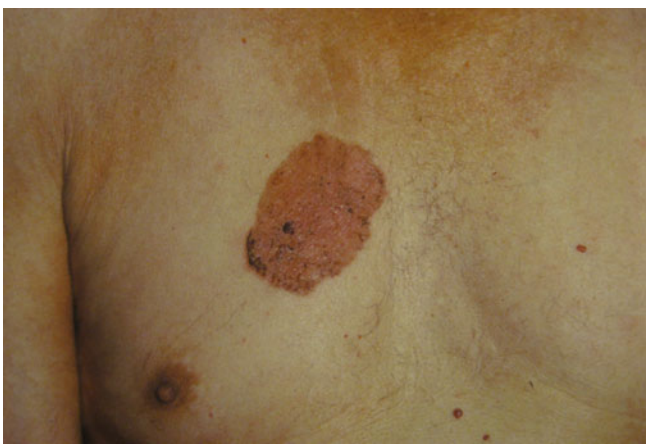
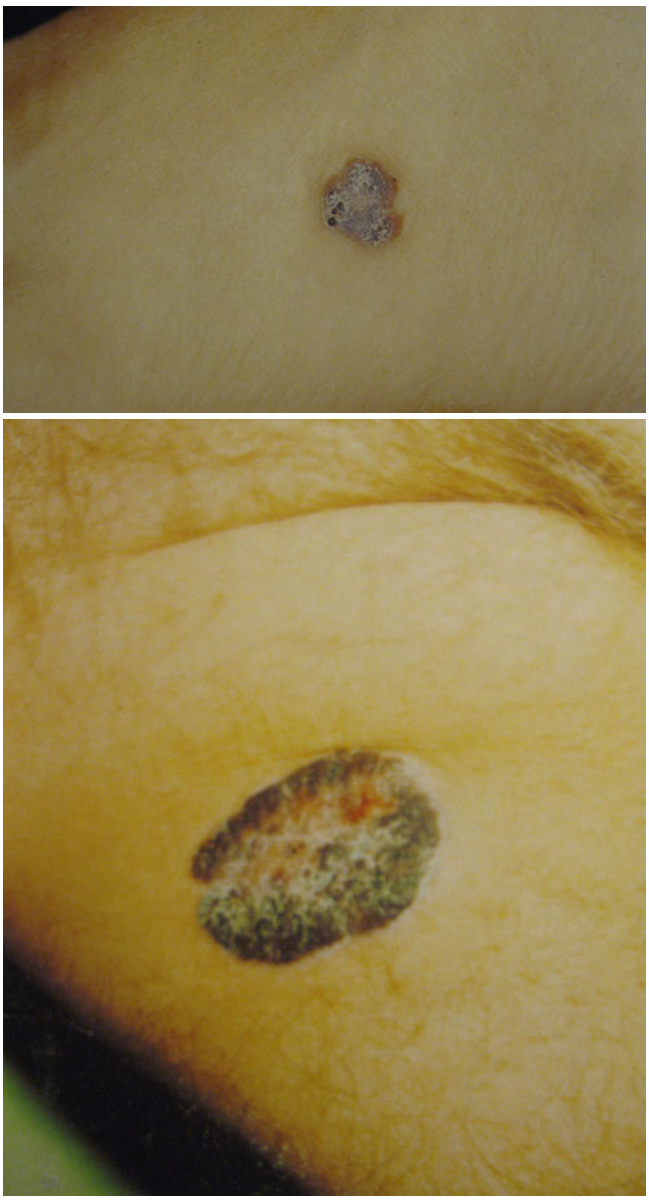


Fig. 37.15 Angiokeratoma plaque simulates superficial basal cell carcinoma or Bowen's squamous cell carcinoma-in-situ



Figs. 37.16 and 31.17 Angiokeratomas of the foot and thigh simulate malignant melanoma. Biopsy is essential and diagnostic

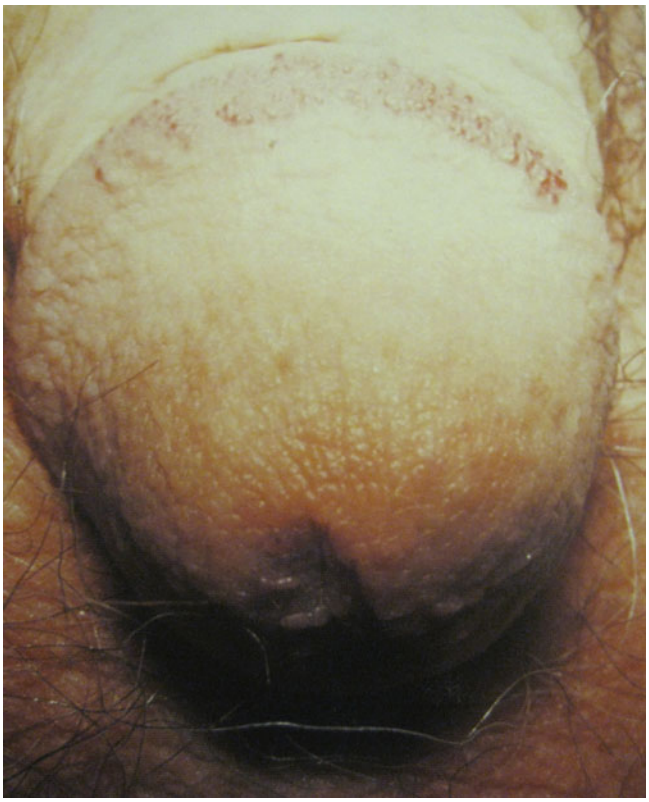


Fig. 37.18 Angiokeratomas of the corona of the glans penis. Angiokeratomas of the scrotum are called Fordyce angiol<eratomas

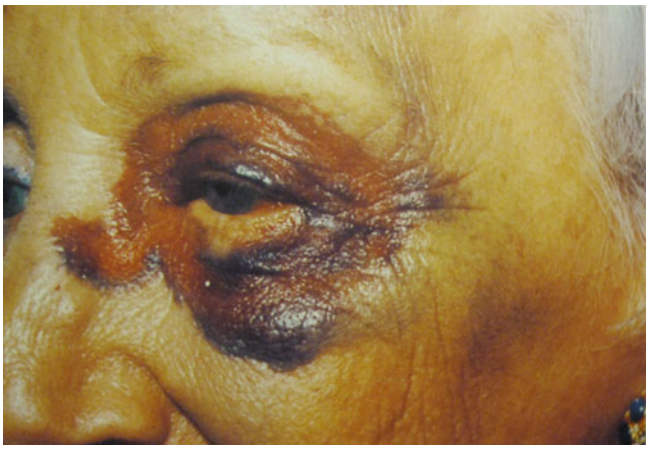


Fig. 37.19 Traumatic bruise of thin, elderly, orbital skin. If the lesion persists for more than a month, angiosarcoma must be suspected

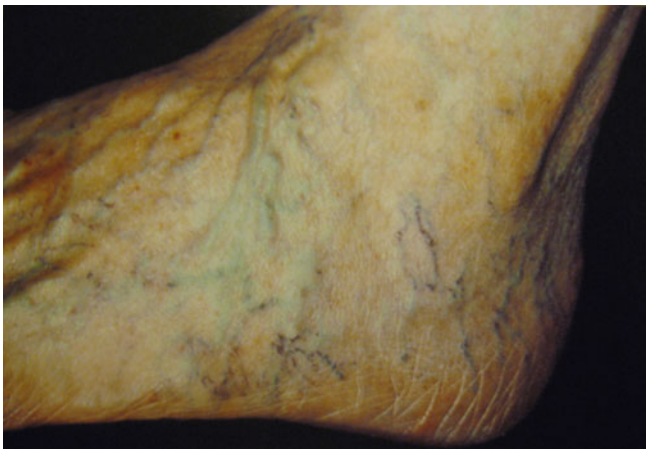
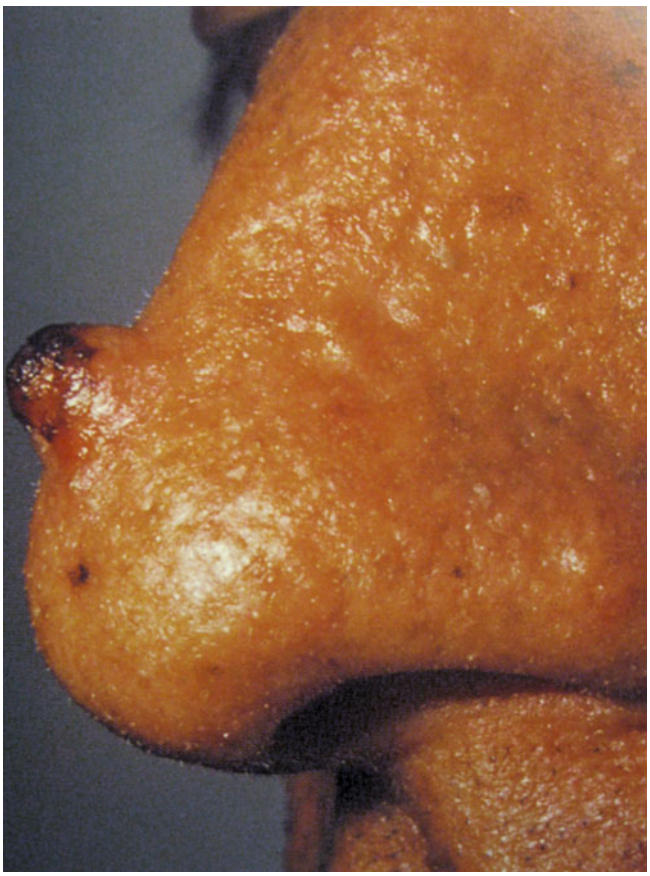
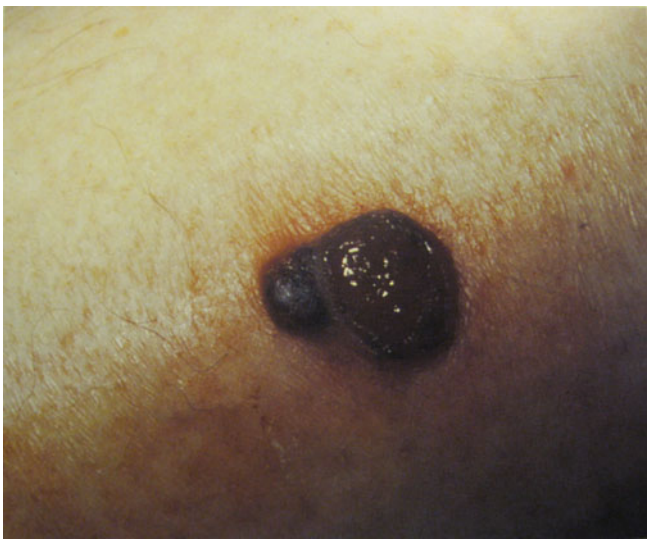


Fig. 37.20 Elderly foot shows large, distended varicose veins and smaller, superficial "spider veins"





Figs. 37.21 and 37.22 Pyogenic granuloma is a rapidly qrowinq, often eroded capillary hemangioma. Differential diagnosis includes Kaposi's sarcoma of AIDS and amelanotic melanoma. Biopsy is essential

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Part VII

Premalignant and Malignant Tumors

Actinic Keratoses

38

Synonyms

Actinic keratoses are also called solar or "senile" keratoses and AKs.

Clinical Description

Actinic keratoses are red-brown, rough, scaly, macules or patches, usually asymptomatic but sometimes tender, on sun-exposed skin, particularly the face, arms, dorsal hands, and bald scalp

Etiology and Pathophysiology

Actinic keratoses result from chronic sun exposure and genetic predisposition, particularly in Celtic, red-haired, or blond, blue-eyed patients [1–6].

Histopathology

The lower third of the epidermis shows atypical squamous keratinocytes with mitoses. Hyperparakeratosis spares adnexal ostia (hair follicles, eccrine sweat glands), resulting in alternating pink (orthokeratotic) and blue (parakeratotic) columns.

Differential Diagnosis

Actinic keratoses are usually easy to diagnose clinically. Biopsy may be useful to confirm the diagnosis or to rule out incipient squamous cell carcinoma. Differential diagnosis also includes solar lentigines (no scale or erythema), seborrheic keratoses, flat warts, and superficial, multicentric, basal cell carcinomas.

Therapy

Treatment includes various measures:

1. Topical chemotherapy with 5-fluorouracil cream or solution, imiquimod, tretinoin, alpha-hydroxy acids, ingenol mebutate gel, and blue light photodynamic therapy.
2. Cryotherapy (liquid nitrogen)
3. Electrodesiccation, curettage, and cautery for deeper, thicker lesions
4. Shave excision of thick lesions to exclude squamous cell carcinoma
5. Dermabrasion for extensive scalp involvement
6. Prevention with sunscreens, protective clothing, hats, and sun avoidance [7–12]

Prognosis

This premalignant precursor lesion to squamous cell carcinoma has a low (0.1–10 %) incidence of malignant progression. Spontaneous regression has been reported, and may be encouraged by the use of sunscreens.

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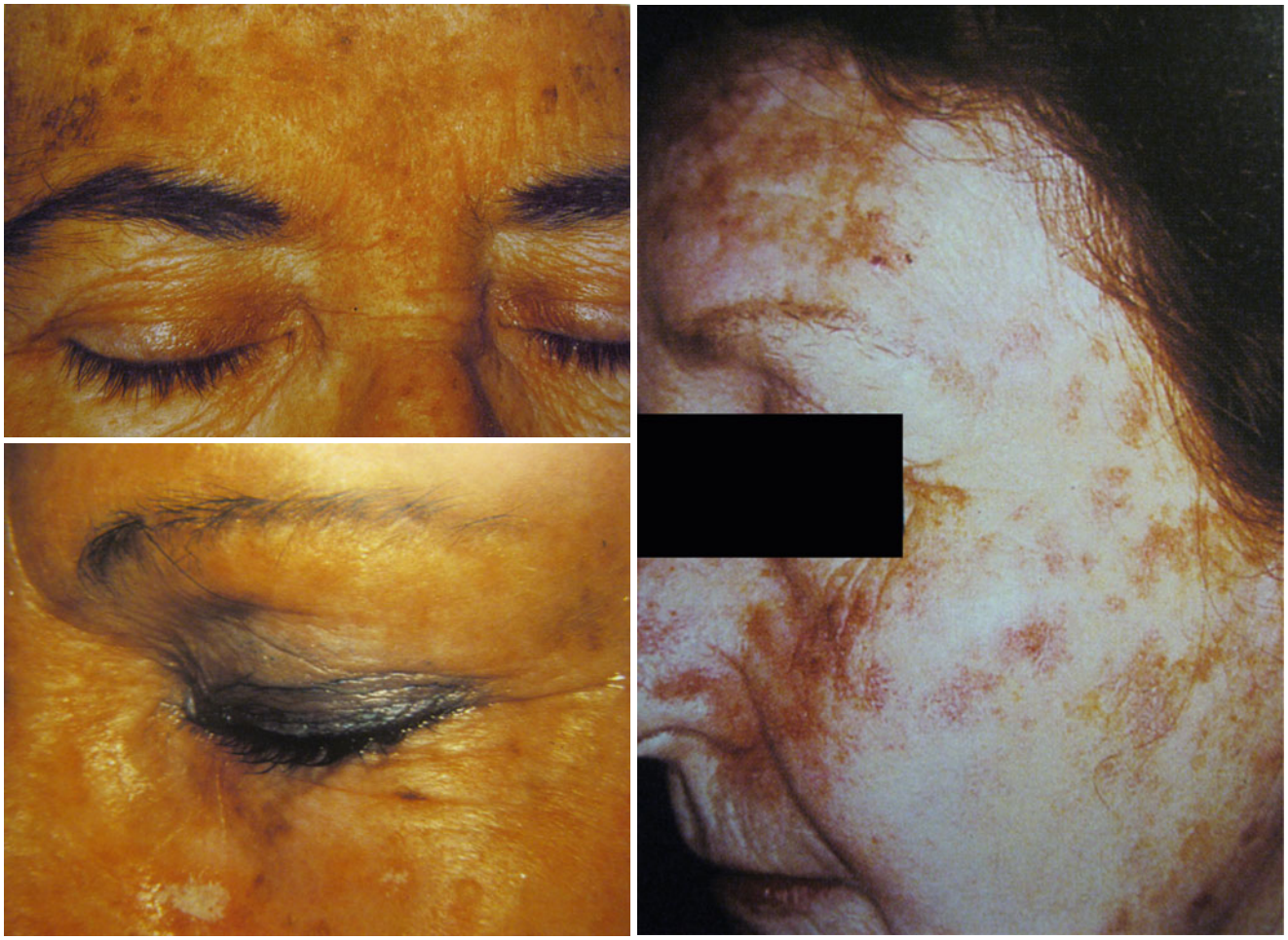
DOI 10.1007/978-1-4471-4579-0_38, © Springer-Verlag London 2014



Figs. 38.1, 38.2, and 38.3 Typical actinic keratoses of the forehead and nasal bridge are brown or reddish brown scaly macules, patches, or papules. The white area in Fig. 38.2 is the site of trichloroacetic acid application

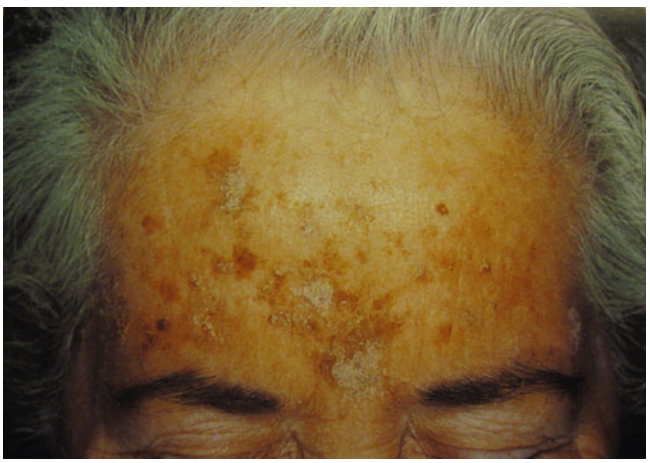
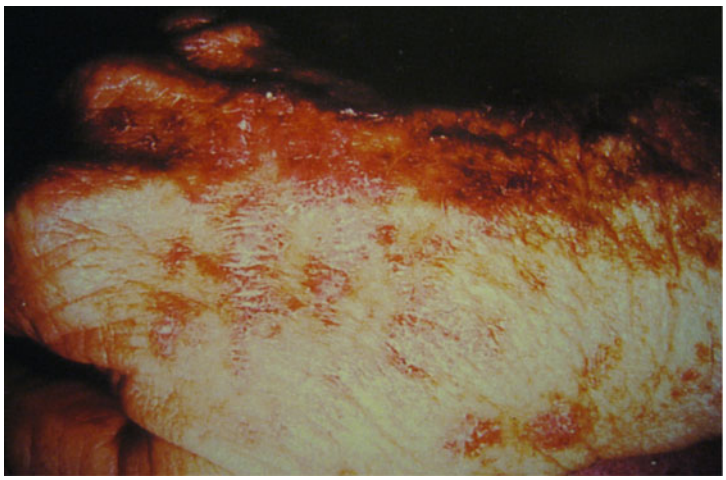
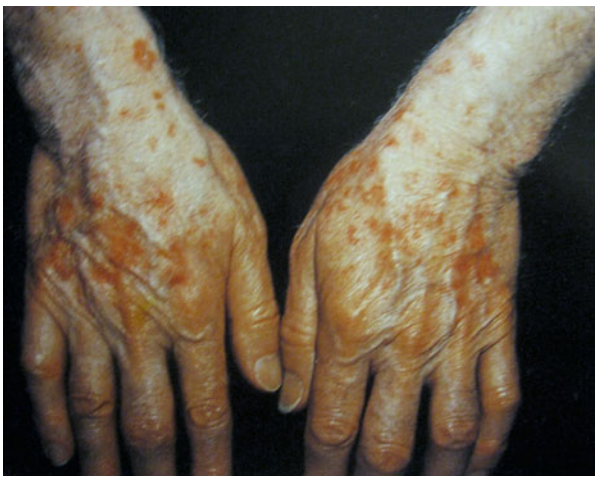


Fig. 38.4 Hypertrophic actinic keratoses of the forehead. For these thicker lesions, pretreatment with tretinoin (retinoic acid, Retin-A) or other keratolytics may improve the efficacy of topical chemotherapy, such as 5-fluorouracil or trichloroacetic acid. Uquid nitrogen cryotherapy is less effective on thick lesions; the additional cryospray required to penetrate the horn may cause blisters and permanent depigmentation





Figs. 38.5 and 38.6 Actinic keratoses on the hands. Keratoses turn bright red during 5-fluorouracil topical chemotherapy (Fig. 38.6). Note the sparing of unaffected, normal skin

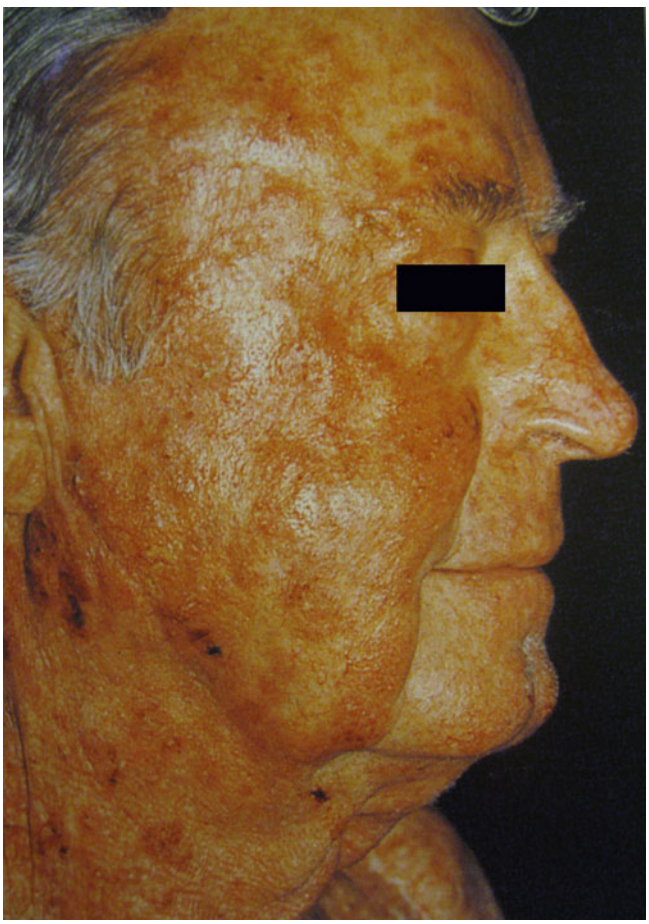
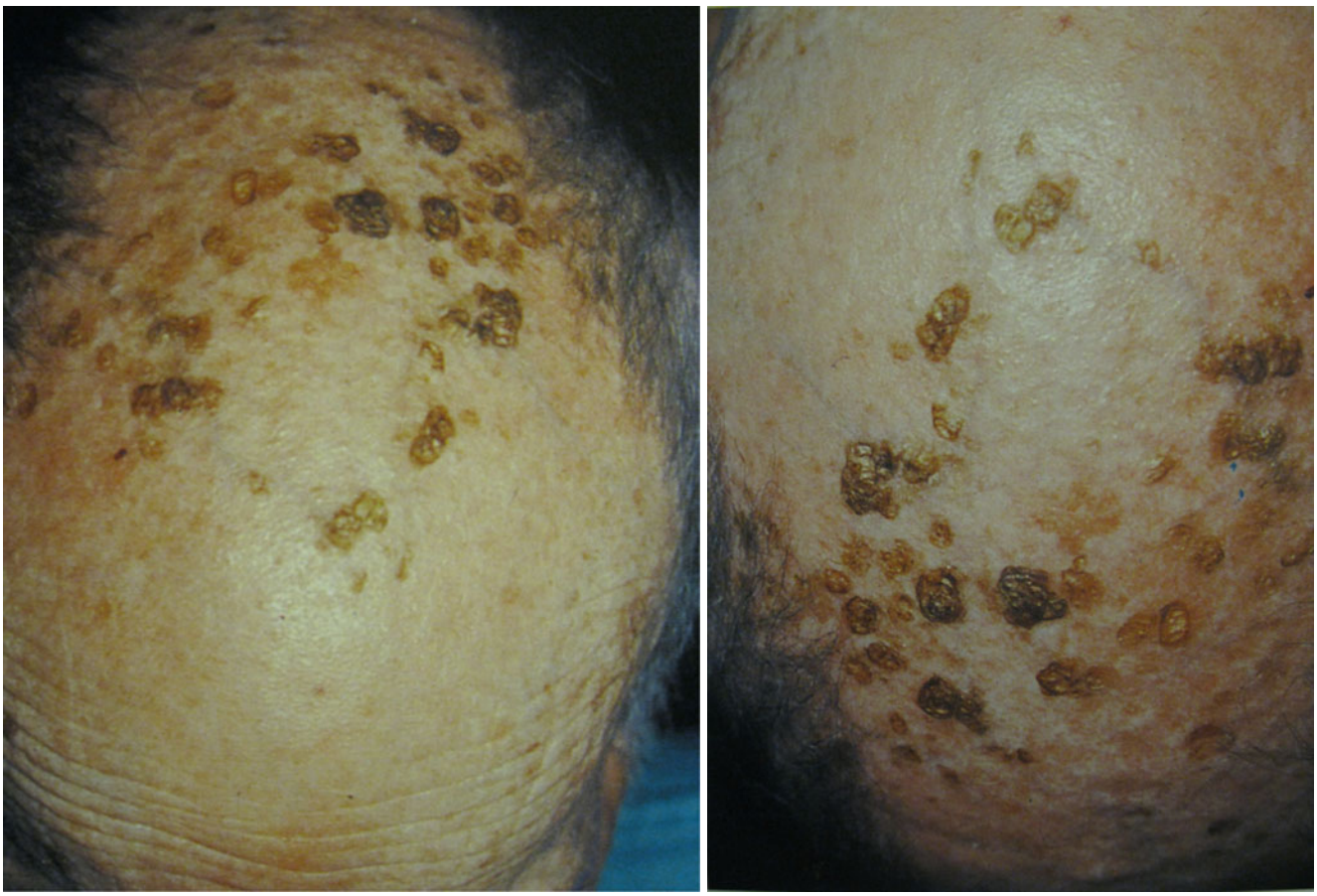


Fig. 38.7 Chronically sun-exposed patient of Celtic ancestry with multiple actinic keratoses and basal cell carcinomas



Figs. 38.8 and 38.9 Crusted, hypertrophic, actinic keratoses of the scalp

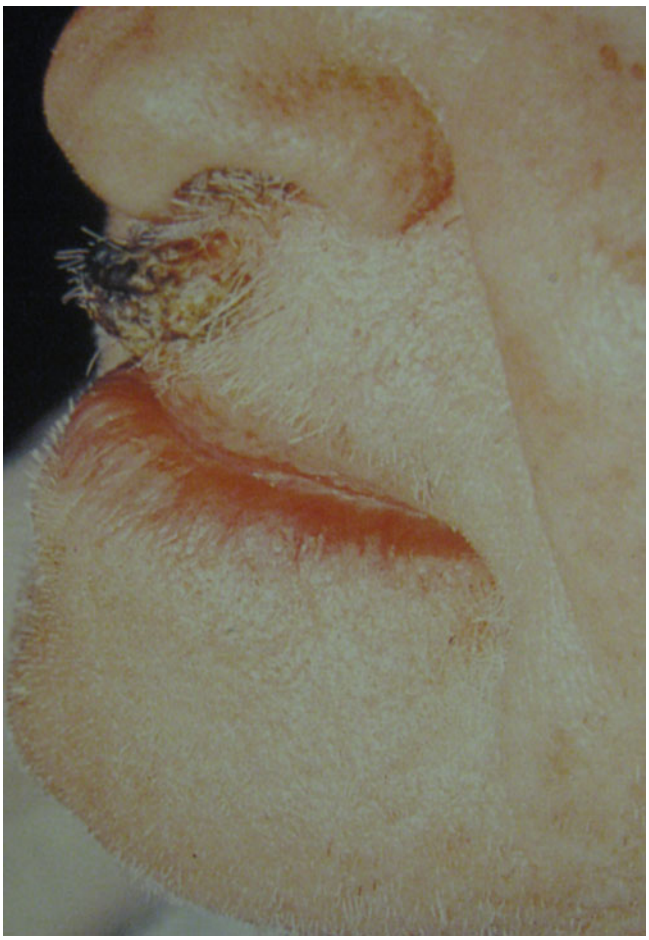
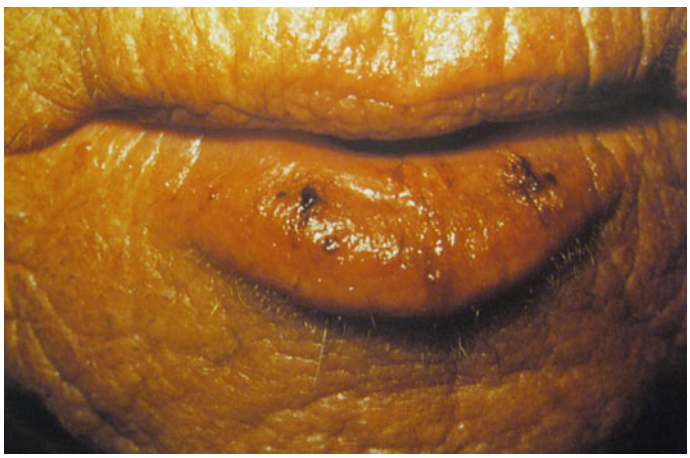
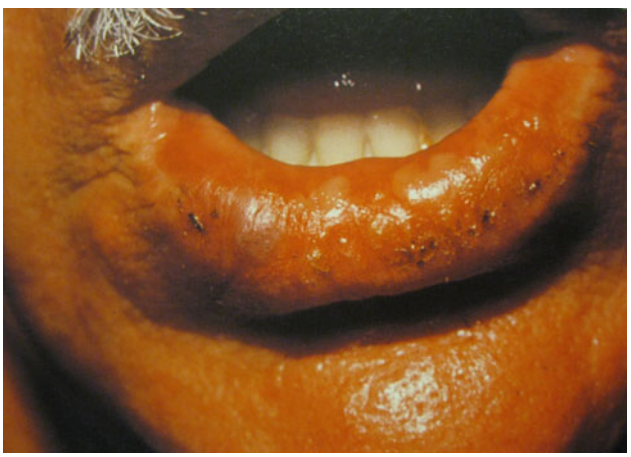


Fig. 38.10 Cutaneous horn of the upper lip. Underlying this horn may be an actinic keratosis, squamous cell carcinoma, keratoacanthoma, or wart. Biopsy sufficiently deep to include full-thickness epidermis is essential





Figs. 38.11 and 38.12 Two patients with actinic chelitis before (Fig. 38.11) and during (Fig. 38.12) topical 5-fluorouracil chemotherapy. The medication easily penetrates the vermilion of the lip. The patient should be cautioned regarding sun-exposure and overzealous treatment. Alternative therapies include a lip peel and mucosal advancement

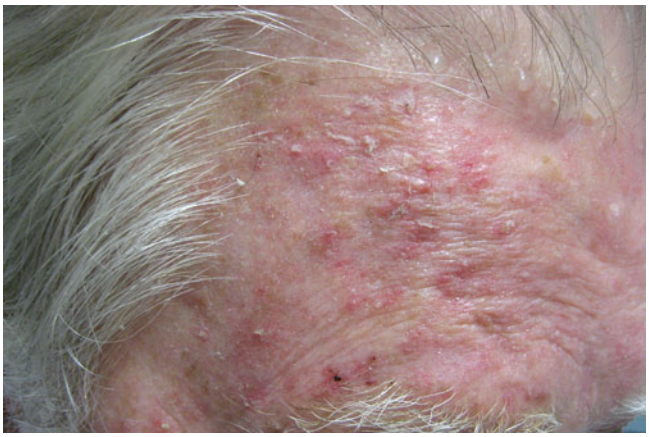


Fig. 38.13 Extensive actinic keratoses on forehead

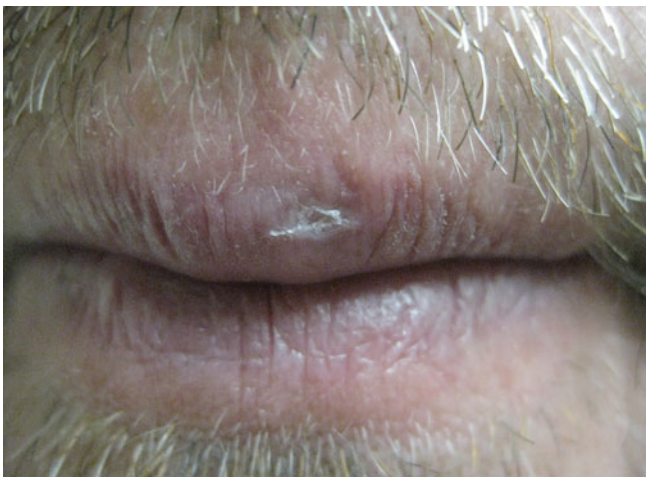


Fig. 38.14 Hypertrophic actinic keratosis on the upper lip

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Angiosarcoma

39

Synonym

Angiosarcoma is also called a "malignant bruise."

Clinical Description

Cutaneous angiosarcoma presents as persistent, red-black patches and plaques on the face, especially the scalp. The clinical significance is that a banal-appearing, persistent bruise or ecchymosis can be fatal [1–4].

Etiology and Pathophysiology

Angiosarcoma is a multicentric, malignant, vascular tumor of unknown cause.

Histopathology

Histopathologic investigation reveals blood-filled irregularly anastomosing vascular slits and spaces that are lined by atypical vascular endothelia

Differential Diagnosis

Early biopsy of suspected cutaneous angiosarcoma is essential because of rapid, early metastasis in the elderly, Hemangiomas and telangiectases, including port wine stain, appear much earlier in life. Ecchymoses resolve spontaneously,

Therapy

Treatment includes the following:

1. Extensive radiation therapy beyond the clinical borders of the tumor.
2. Surgery is less effective, because the tumor is multicentric.

Prognosis

The prognosis is poor. Angiosarcoma is often fatal, even with aggressive radiation therapy.

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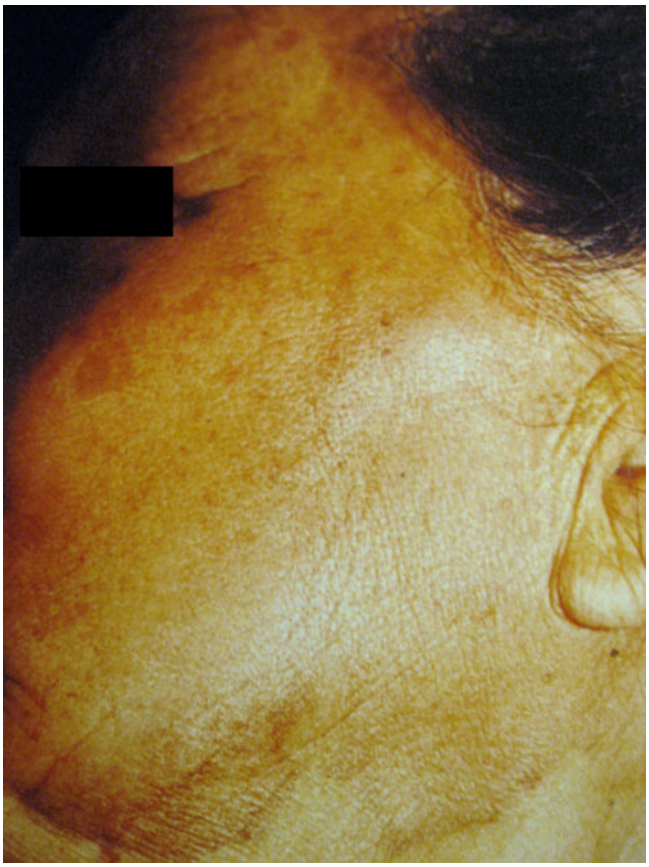
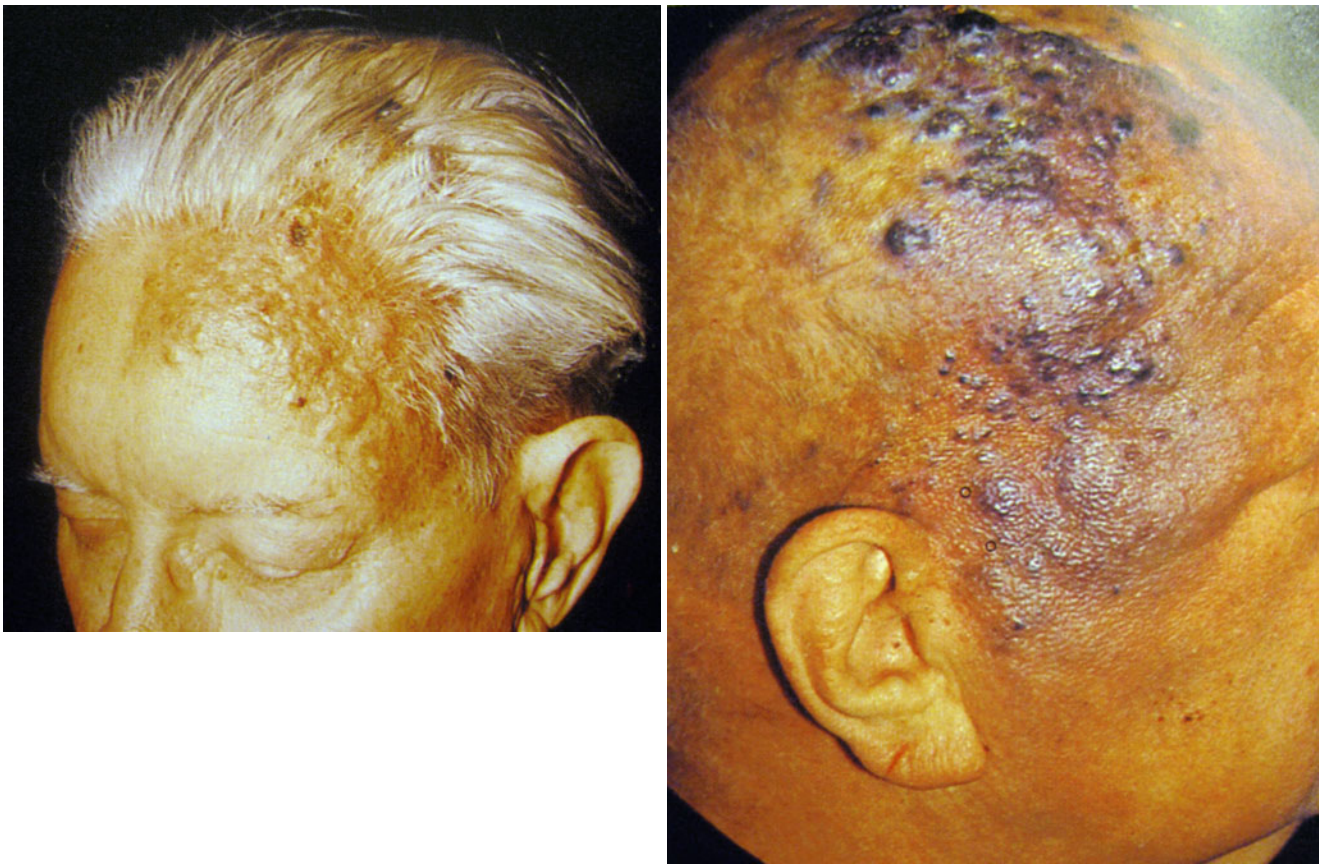


Fig. 39.1 Cutaneous angiosarcoma appears as a nonresolving ecchymotic patch, typically on the face, giving the appearance of a "malignant" bruise



Figs. 39.2 and 39.3 Cutaneous angiosarcoma of the forehead and scalp shows a papular-nodular component. This is a multicentric, rapidly metastatic tumor

Fig. 39.4 Ulcerative nodular angiosarcoma of the scalp

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Basal Cell Carcinoma

40

Synonyms

Basal cell carcinoma (BCC) is also called "rodent ulcer" and basal cell epithelioma (BCE).

Clinical Types and Description

Four major clinical types are seen:

1. Nodular. This is a pink, translucent, waxy papule or nodule, with a raised, rolled border, central depression, and surface telangiectasia or ulceration. It is the most common form, particularly on the face [1, 2]. The colorful term "rodent ulcer" refers to the clinical appearance of a rat's gnawing a hole in the skin
2. Superficial multicentric. This red scaly patch, most often occurring on the trunk or extremities, clinically resembles a patch of tinea (ringworm), eczema, or Bowen's disease
3. Sclerosing or morpheaform. This white, indurated patch clinically resembles scar tissue or morphea (localized scleroderma)
4. Pigmented varieties. Some of these, particularly the nodular and superficial types, clinically resemble melanoma

Etiology and Pathophysiology

Basal cell carcinoma develops from chronic sun exposure in patients with fair skin and/or a genetic predisposition [1–5]. Nevoid basal cell carcinoma syndrome presents early in life with multiple basal cell carcinomas, palmar pits, jaw cysts, and autosomal dominant inheritance.

Histopathology

Basal cell carcinoma is characterized by nests and lobules of hyperchromatic, small, basaloid cells with peripherally aligned (palisaded) nuclei, mucinous retraction spaces, possible sebaceous, follicular, or glandular differentiation, and variable degrees of infiltration and invasiveness [6–13].

Differential Diagnosis

Differential diagnosis includes the following:

1. Nodular BCC-dermal nevus, trichoepithelioma, fibrous papule, angiofibroma, sebaceous hyperplasia, solar elastorna, squamous cell carcinoma
2. Superficial BCC-tinea, eczema, Bowen's disease
3. Sclerosing BCC-scar, keloid, morphea (localized scleroderma)
4. Pigmented BCC-melanoma, seborrheic keratosis, nevus

Biopsy is mandatory. Histology is usually diagnostic.

Therapy

Treatment includes the following [14–33]:

1. Surgery involves complete excision including Mohs excision (microscopically controlled excision) of recurrent primary lesions, sclerotic morpheaforrn lesions, andlor lesions located in anatomicaliy critical areas such as the ocular canthi, nasal alae, periorbital and periocular areas. Shave excision followed by electrodesiccation, curettage, and cautery can be used for superficial nonsclerosing lesions.

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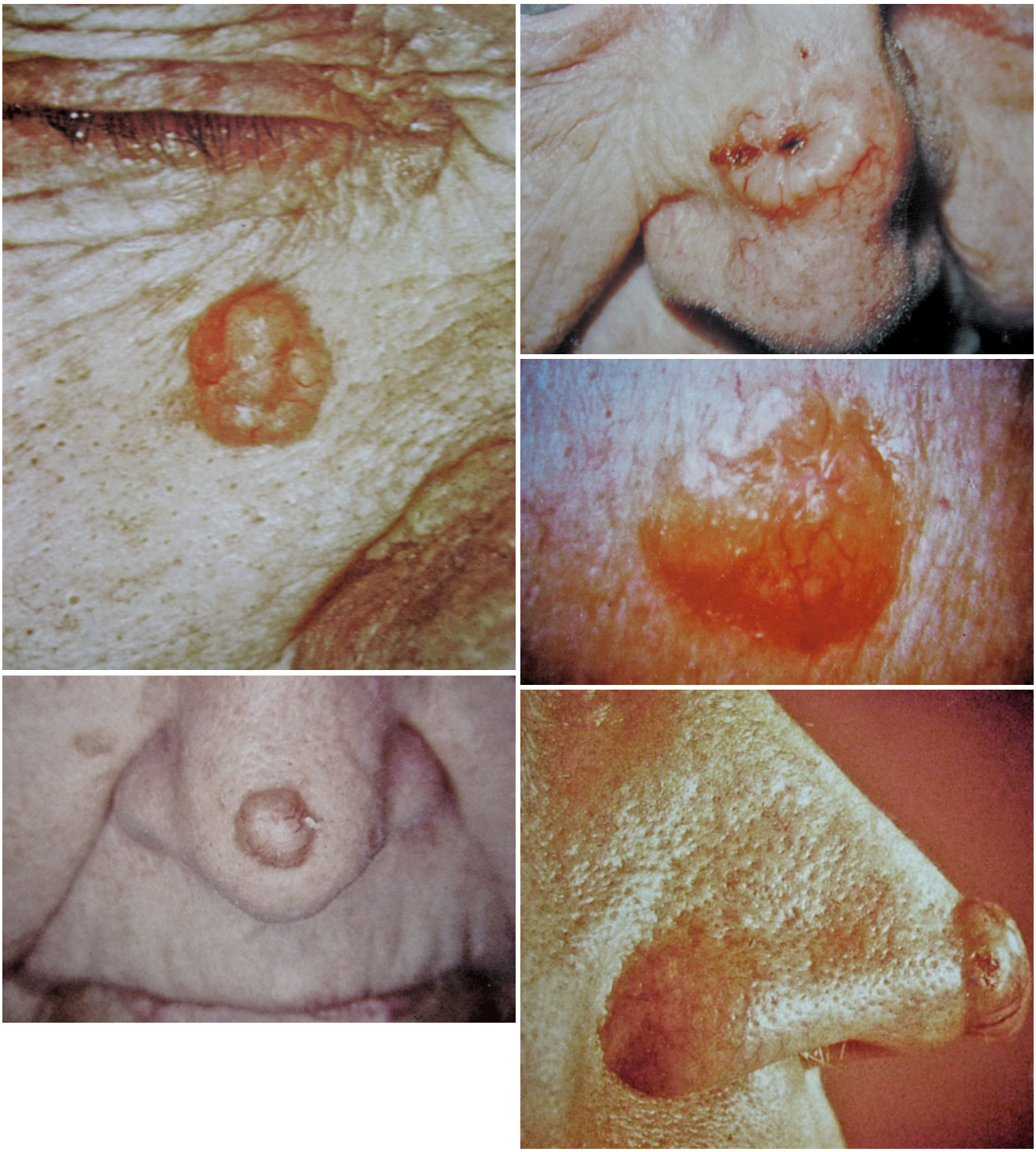
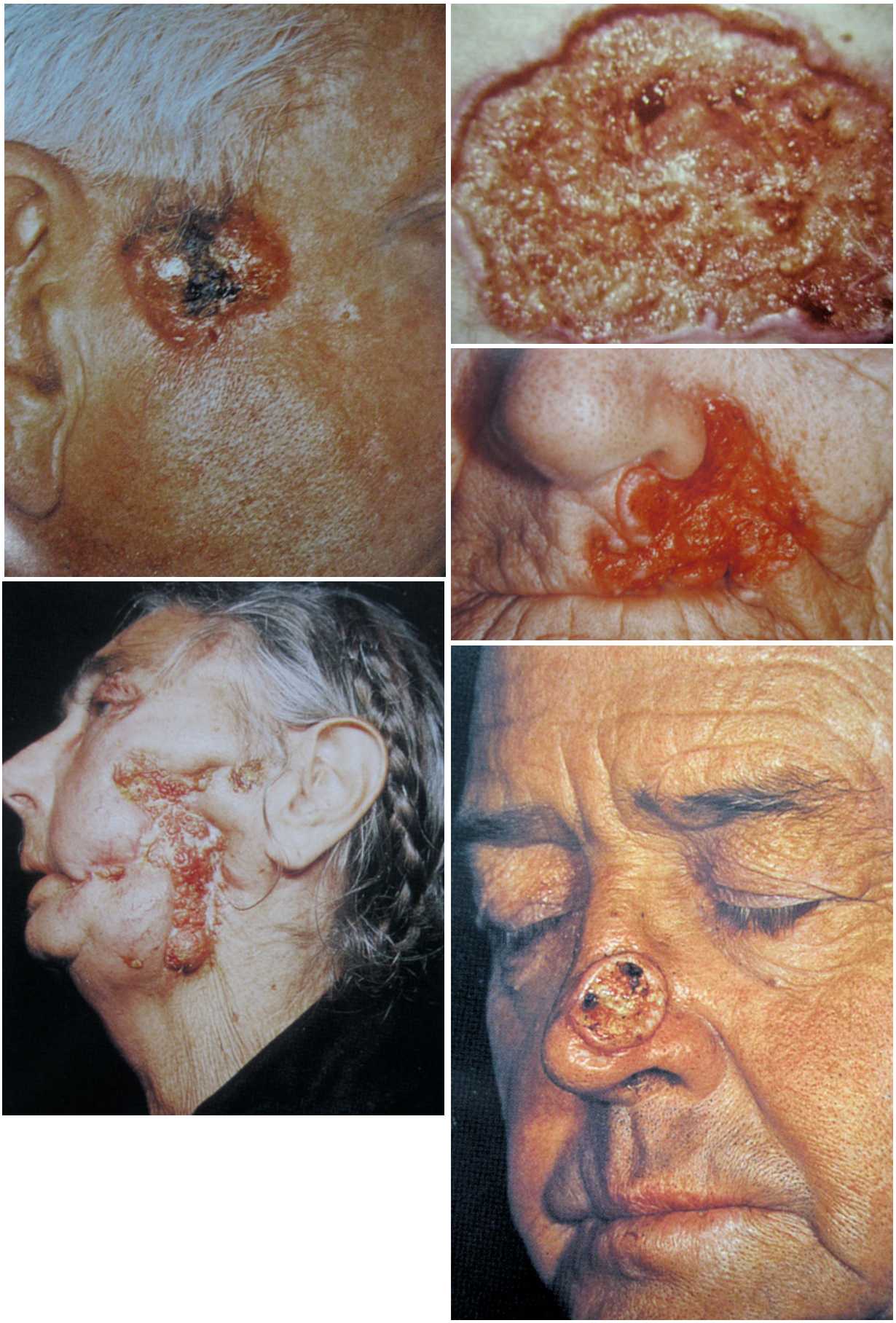
2. Radiotherapy is reserved for older patients who cannot tolerate surgery, but it is contraindicated for those with radiation-induced basal cell carcinomas.
3. Chemoprevention is possible with retinoids (e.g., isotretinoin) for multiple BCCs, particularly patients with nevoid basal cell carcinoma syndrome.
4. Deep cryotherapy can be useful for anticoagulated patients or others who cannot undergo surgery.
5. Other treatments include intralesional interferon, imiquimod, topical photodynamic therapy

Prognosis

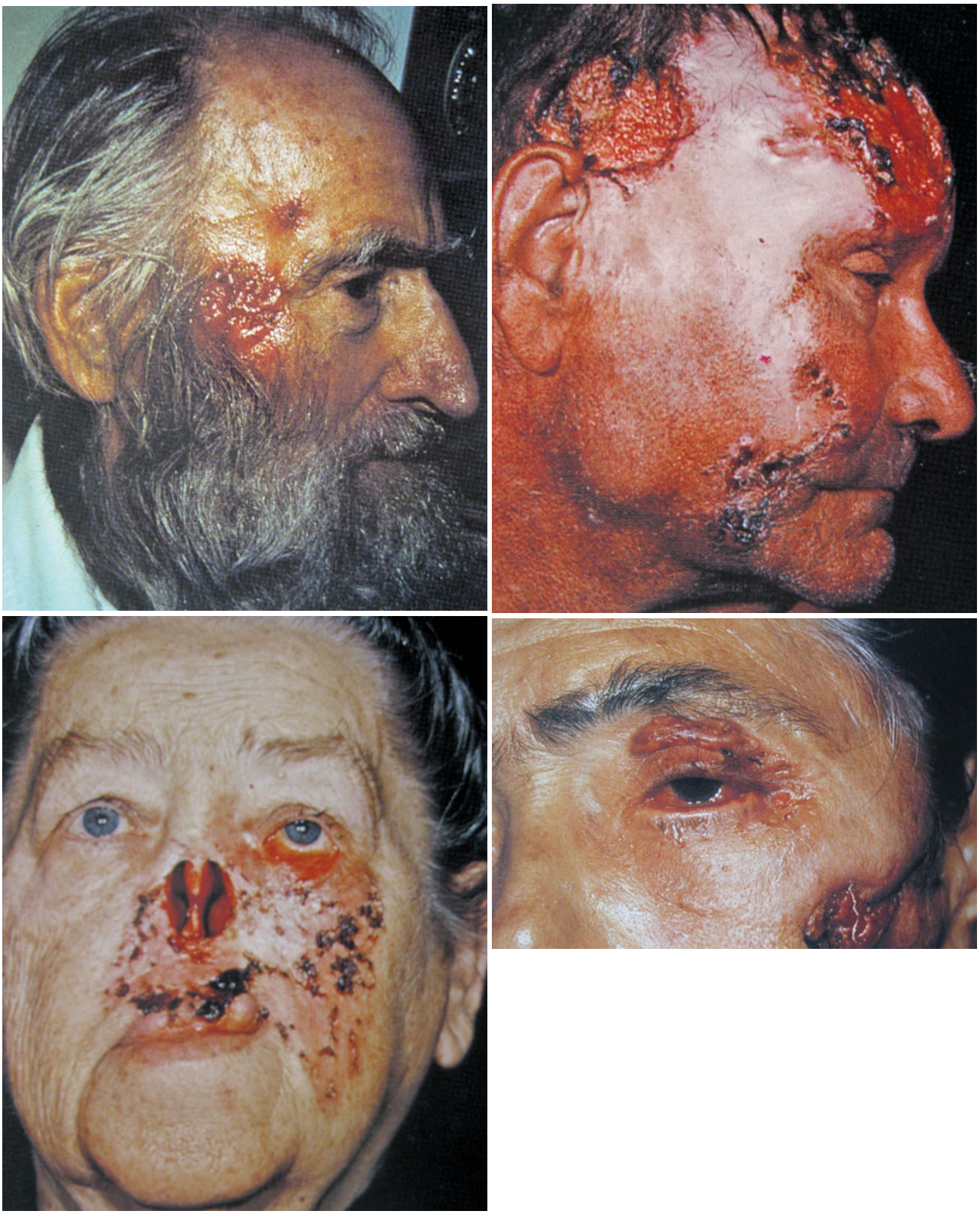
Basal cell carcinoma, the most common and least dangerous cancer, has a good prognosis. Metastases are rare, but do occur [34].



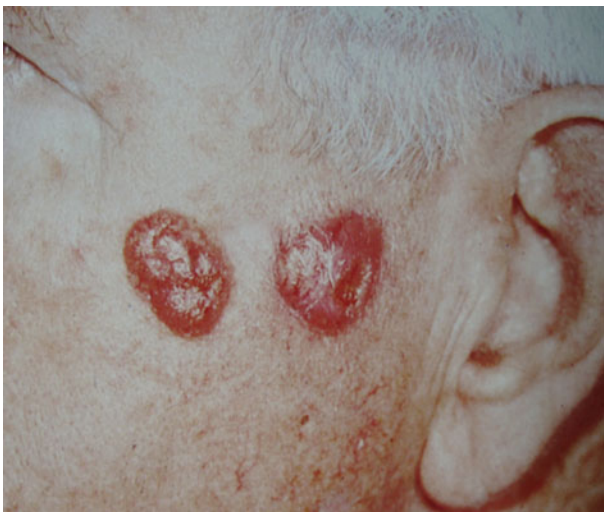
Figs. 40.1, 40.2, 40.3, 40.4, and 40.5 Classic nodular basal cell carcinoma shows a pink, pearly, waxy papute with central depression and telangiectasia

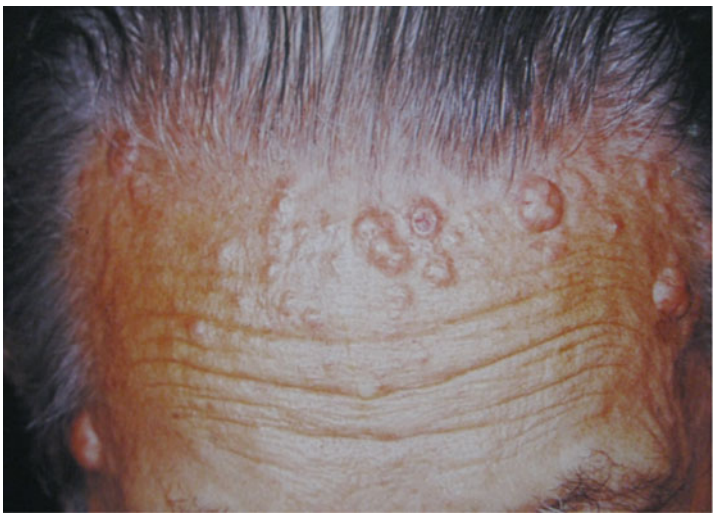


Figs. 40.6, 40.7, 40.8, 40.9, 40.10, 40.11, 40.12, 40.13, and 40.14 Large, centrally eroded "rodent ulcers"

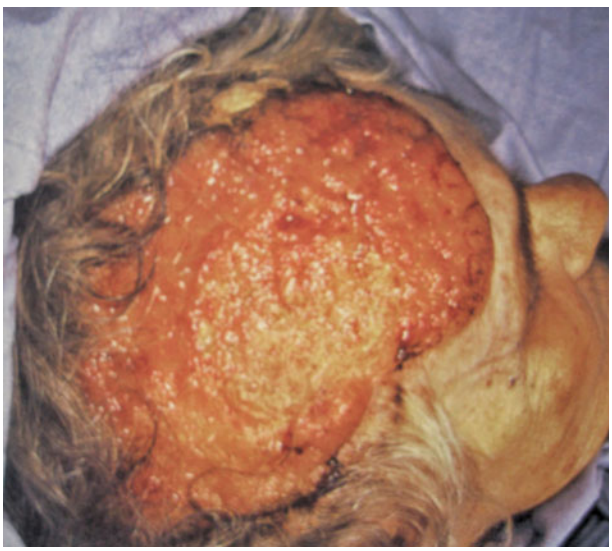


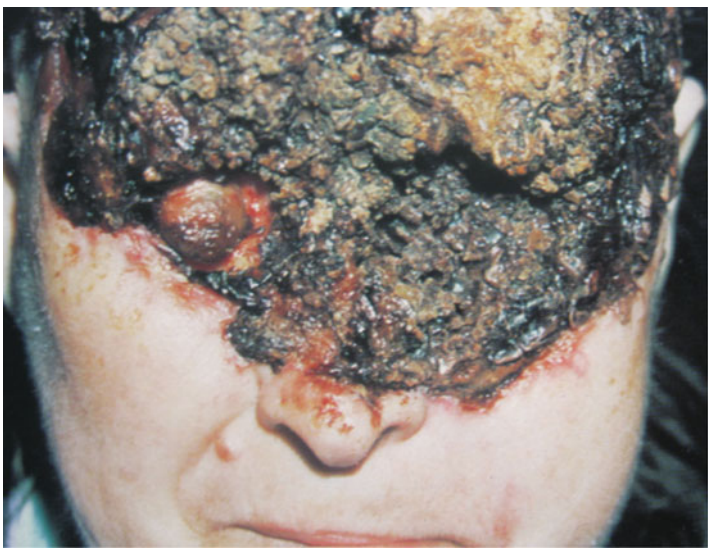
Figs. 40.6, 40.7, 40.8, 40.9, 40.10, 40.11, 40.12, 40.13, and 40.14 (continued)

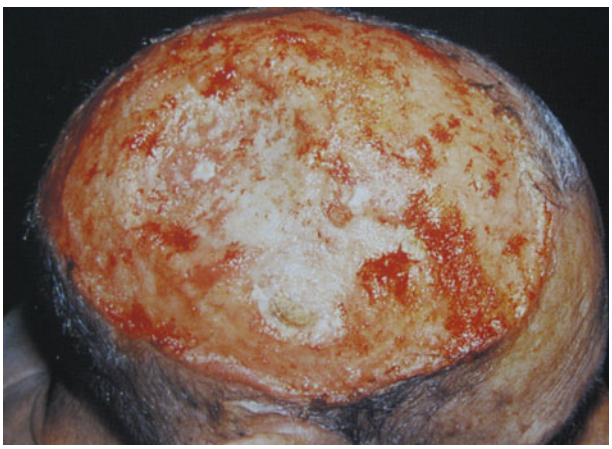




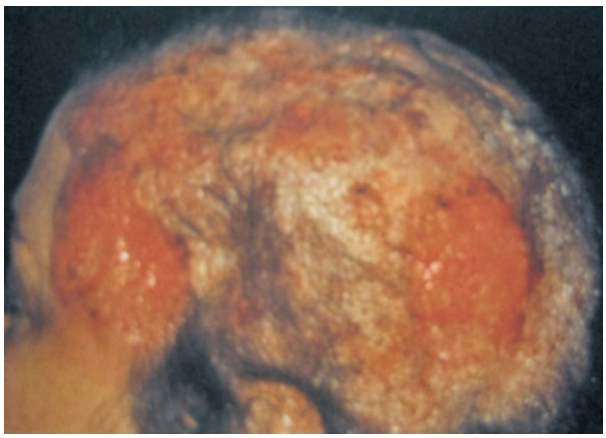
Figs. 40.15 and 40.16 Basal cell carcinomas are frequently multiple

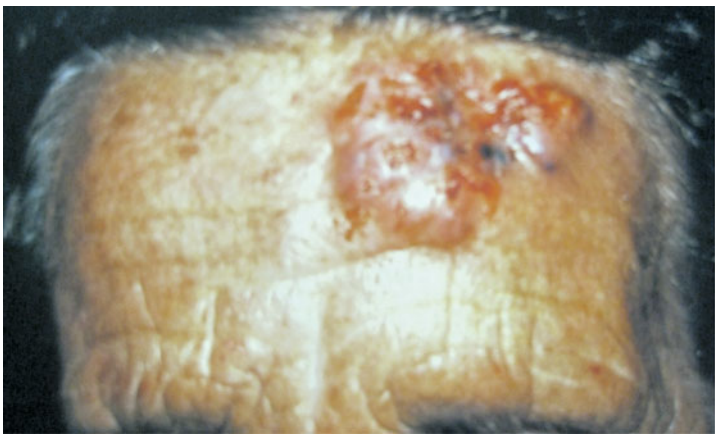






Figs. 40.17, 40.18, and 40.19 Basal cell carcinoma metastases to the brain after invading the bony skull





Figs. 40.20 and 40.21 These gigantic, long-neglected, basal cell carcinomas were painless

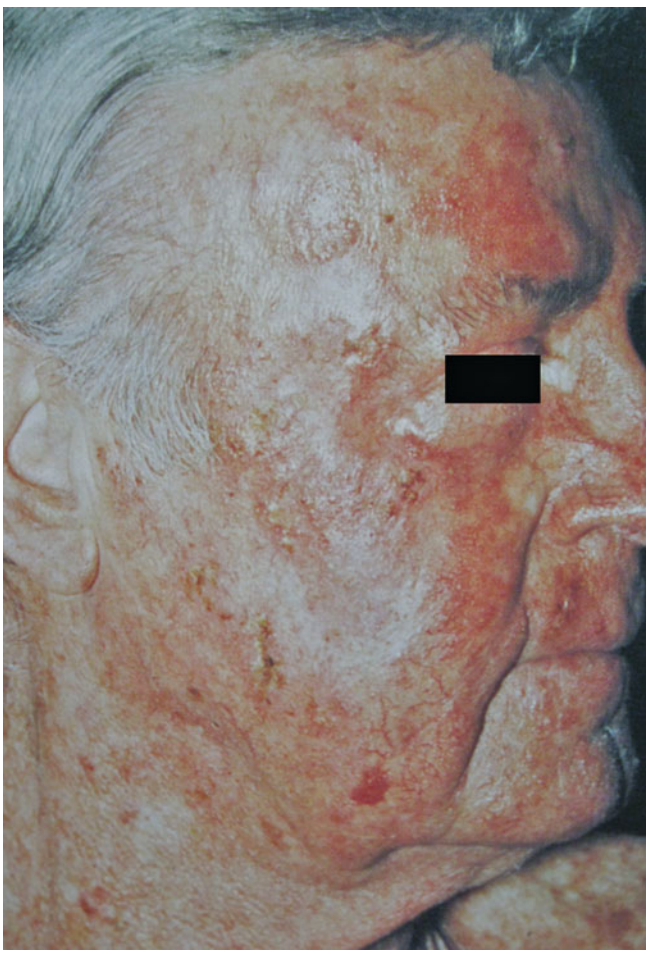


Fig. 40.22 Multiple superficial and nodular basal cell carcinomas developed on the face of this Celtic farmer who worked outdoors all his life

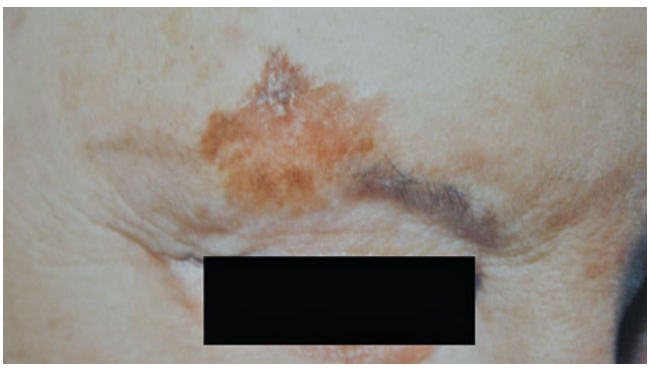
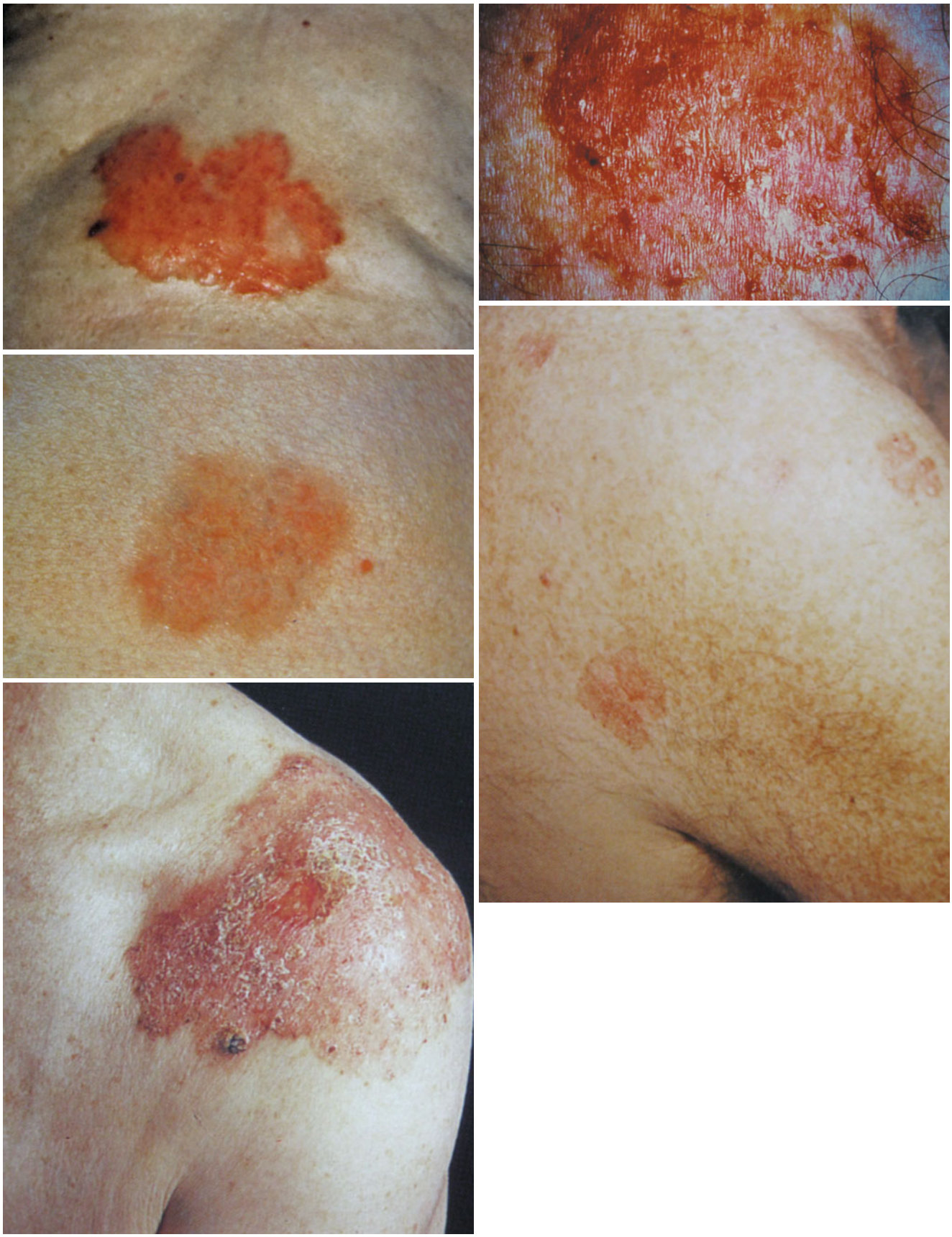
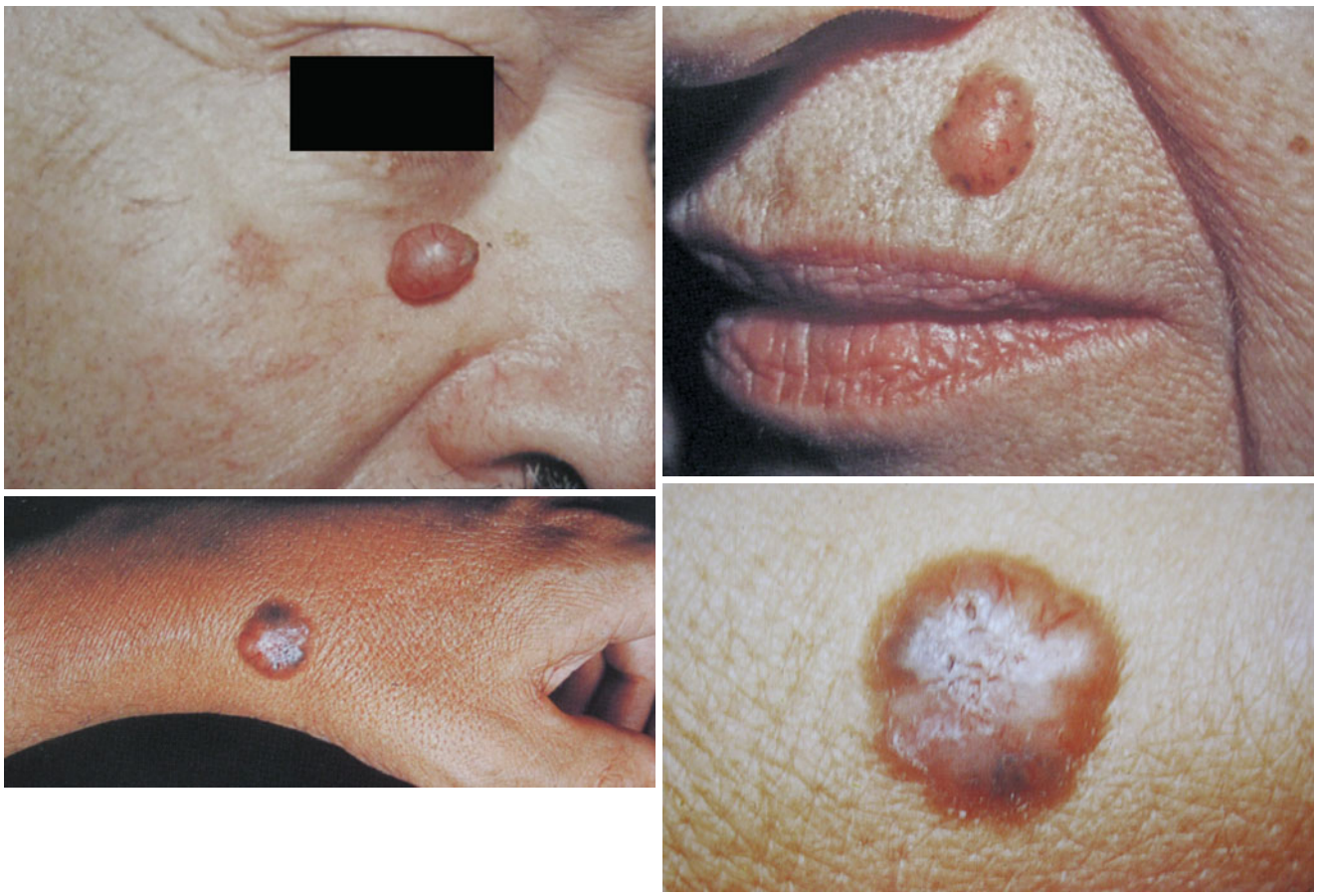


Fig. 40.23 Pigmented superficial basal cell carcinoma obliterates part of the eyebrow. Clinical differential diagnosis includes a lentigo maligna and lentigo maligna melanoma



Figs. 40.24, 40.25, 40.26, 40.27, and 40.28 Superficial basal cell carcinoma clinically resembles eczema, tinea, psoriasis, and Bowen's disease



Figs. 40.29, 40.30, 40.31, and 40.32 Typical nodular basal cell carcinomas show a pearly nodule with telangiectasia and sometimes spotty pigmentation

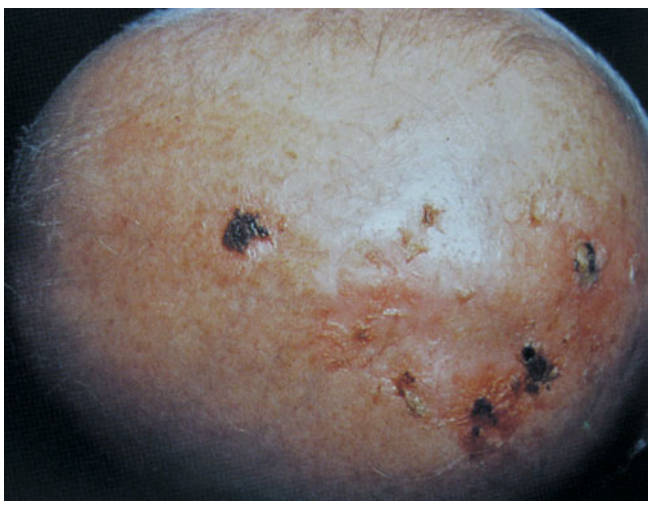


Fig. 40.33 Basosquamous cell carcinoma shows histologic features of both basal and squamous cell carcinomas. It is more aggressive than a typical basal cell carcinoma

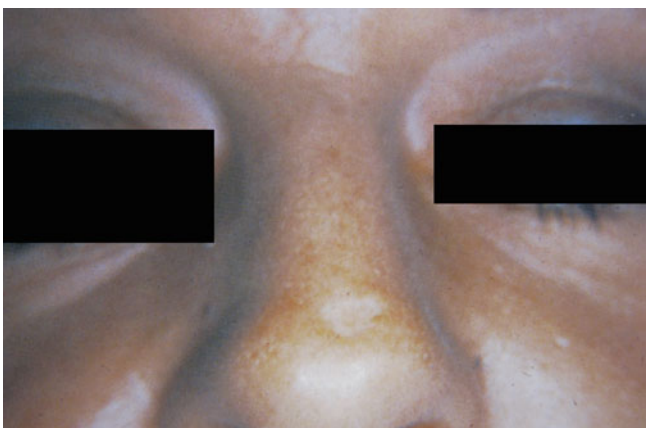
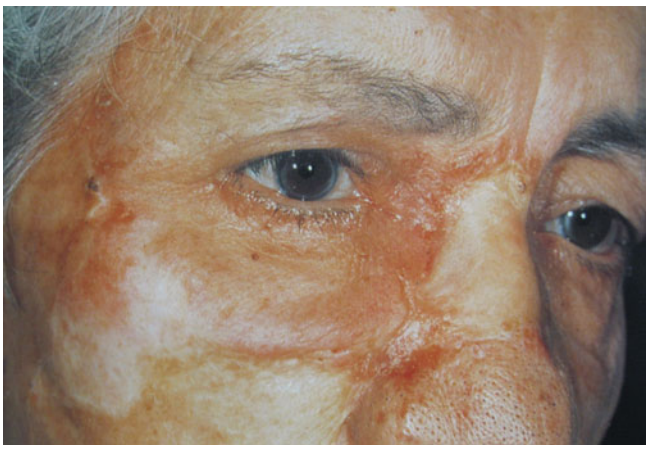
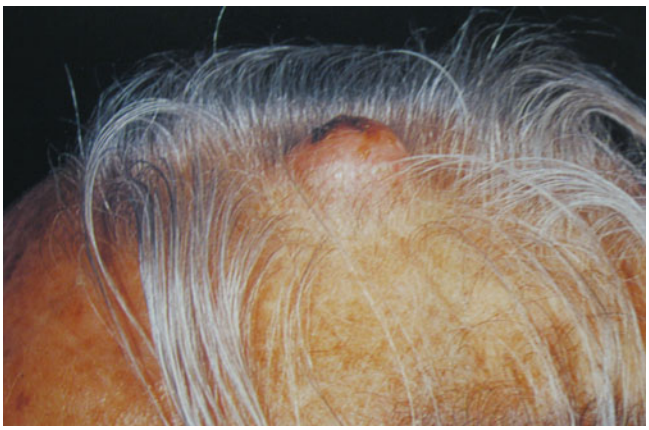


Fig. 40.34 Sclerosing basal cell carcinoma clinically resembles a depressed, white scar or morphea (localized scleroderma)



Figs. 40.35 Recurrent basal cell carcinoma developed within the site of skin grafting



Figs. 40.36 Recurrent basal cell carcinoma developed on top of the scalp

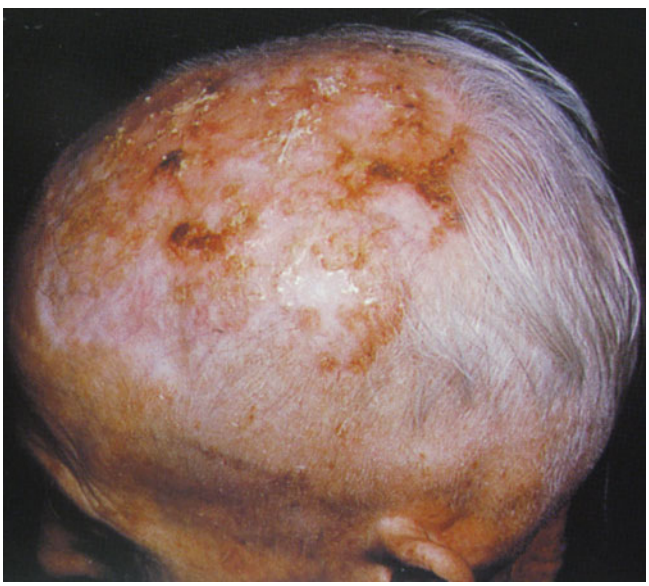


Fig. 40.37 This diffusely sun-damaged scalp is covered with basal cell carcinomas

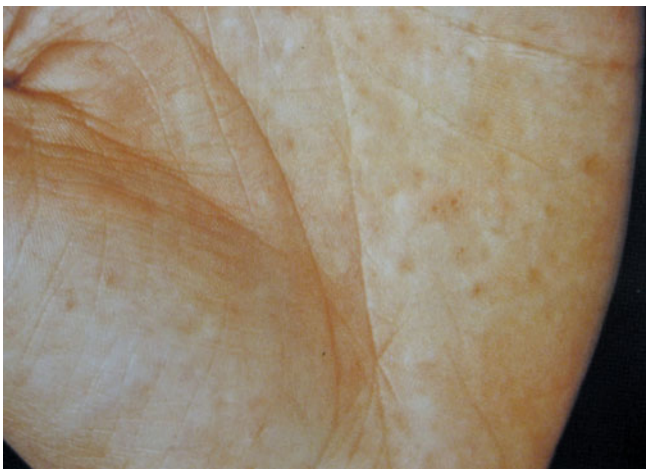


Fig. 40.38 Palmar pitting in a patient with multiple basal cell carcinomas suggests the nevoid basal cell carcinoma syndrome, an autosomal dominant disorder. Systemic retinoids, such as isotretinoin (Accutane), can be helpful in preventing the development of new tumors

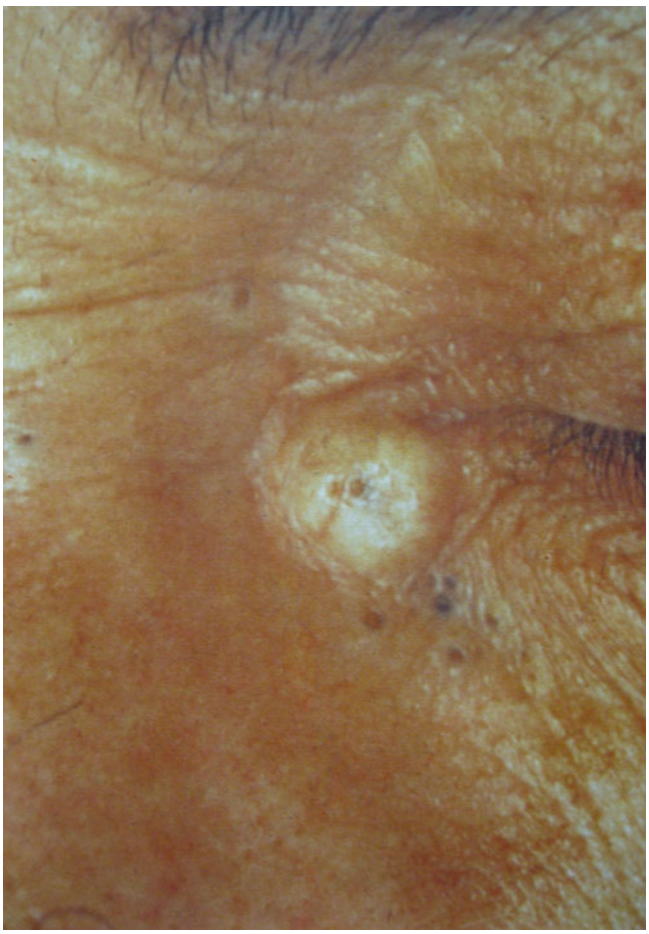


Fig. 40.39 Basal cell carcinoma of inner canthus clinically simulates an epidermal inclusion cyst

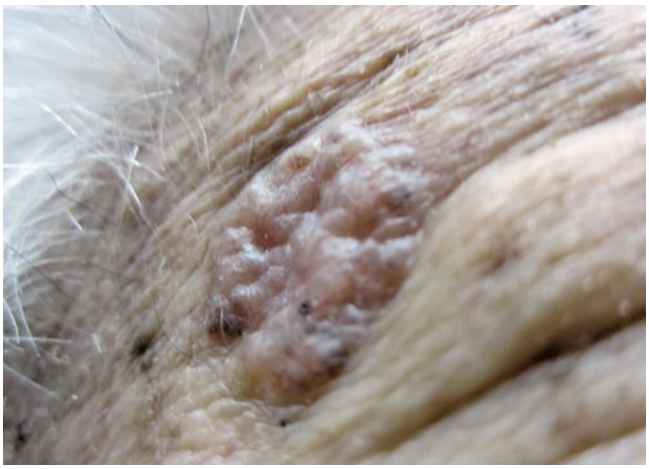


Fig. 40.40 Large basal cell cancer on right forehead

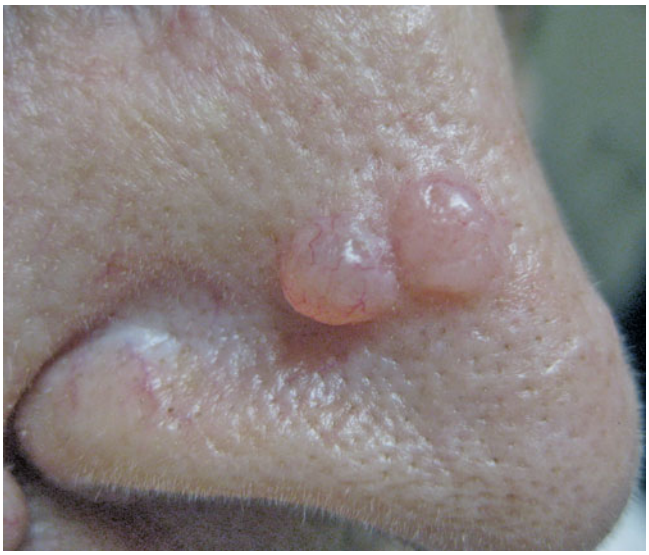


Fig. 40.41 73-year-old white female presented with a 3-month growth on the right ala. Examination showed two adjacent flesh/light pink colored, dome shaped papules. These papules are well defined and feel firm and are immobile. Their wall was smooth and prominent telangectasias are found on the surface

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Dermatofibroma and Dermatofibrosarcoma Protuberans

41

Synonyms

Dermatofibroma (DF) is also called nodular subepidermal fibrosis, benign fibrous histiocytoma, sclerosing hemangioma, and fibroma durum. Dermatofibrosarcoma protuberans (DFSP) is also called Bednar's tumor (pigmented DFSP).

Clinical Description

DF is a red-brown, smooth, hard, domed papule or nodule, usually occurring on the arms or legs. A positive "dimple" or indentation sign can be exhibited with lateral compression. DFSP is a pink or flesh-colored nodule or lumpy keloidal plaque, often on the upper trunk.

Etiology and Pathophysiology

DF is caused by a penetrating injury, such as an insect bite or a nick from shaving. It may represent an inflammatory rather than a neoplastic process. DFSP is a malignant fibroblastic-histiocytic tumor of unknown cause.

Histopathology

DF demonstrates epidermal basaloid hyperplasia, often resembling basal cell carcinoma [1], hyperpigmentation of the basal layer, a cellular dermal tumor composed of fibroblasts arranged in a circular or curlicue pattern around collagen bundles (trapped collagen) and blood vessels (sclerosing hemangioma type), with hemosiderin deposits. DFSP is a densely cellular tumor involving the dermis and often the subcutaneous fat [2]. It is composed of fibrohistiocytic cells (probably fibroblasts) arranged in a storiform (mat-like), cartwheel, whorl, or radial pattern around hubs of nonpolarizable collagen [3] in contrast to normal polarizable stromal collagen. Scattered mitoses, giant cells, and hemosiderin deposits are seen [4, 5].

Differential Diagnosis

The differential diagnosis of DF includes melanocytic nevi (less firm), melanoma, and pigmented basal cell carcinoma. That of DFSP includes keloid (clinically), fibrosarcoma, and large dermatofibroma (histologically).

Therapy

No treatment is needed for DF. Early lesions may resolve with cryotherapy or low-dose intralesional corticosteroids (triamcinolone acetonide 4–10 mg/ml). Older lesions are permanent. Patients should be warned that excision for cosmesis or convenience may leave a worse scar, especially on the legs. The treatment of DFSP involves complete excision. Mohs surgery has been successfully used [6, 7].

Prognosis

DF is stable and benign, but DFSP occasionally metastasizes, and is considered to be a low-grade fibrosarcoma.

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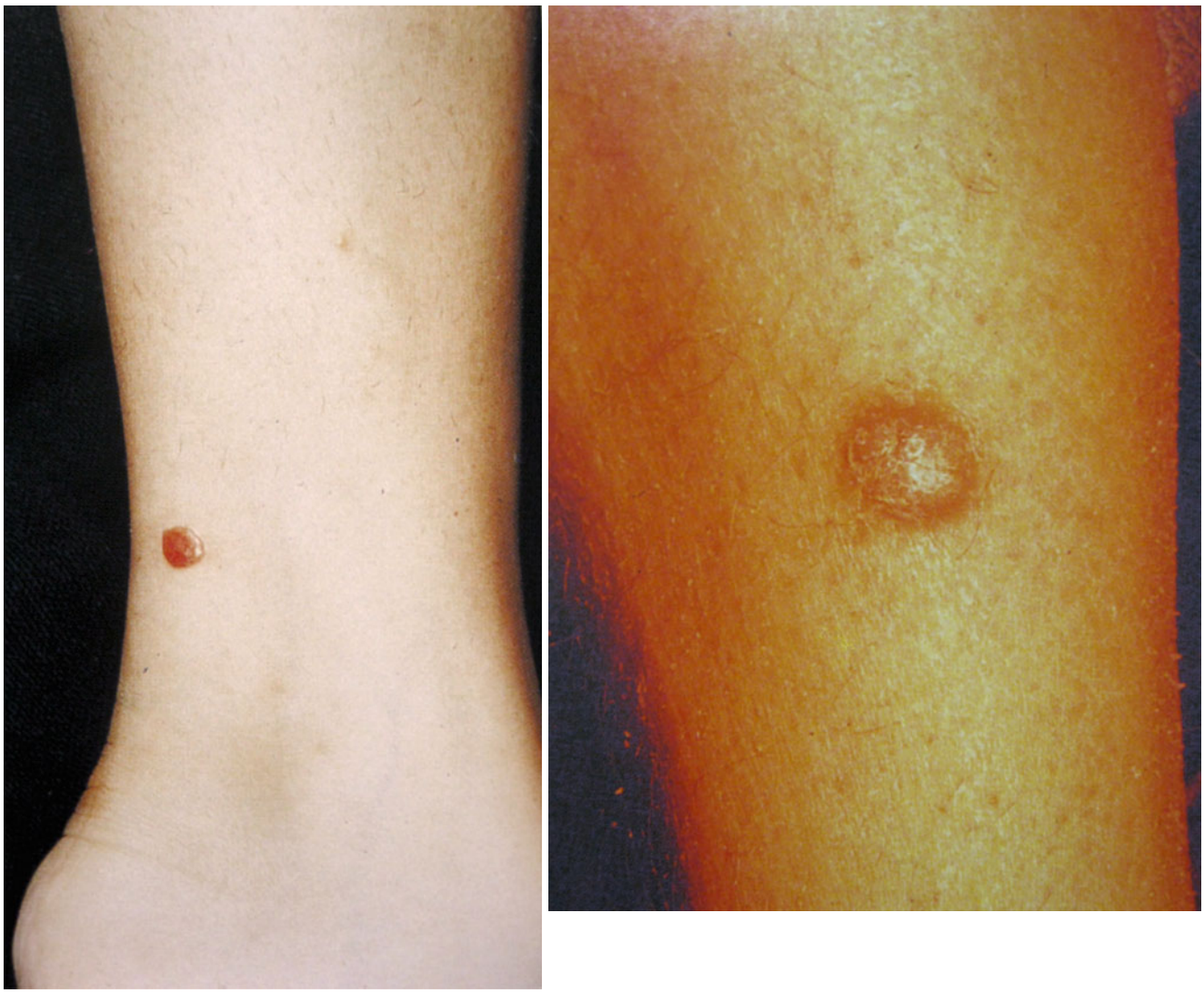


Figs. 41.1 and 41.2 Typical dermatofibromas on the leg occur after a puncture wound, such as an insect bite or a shaving nick

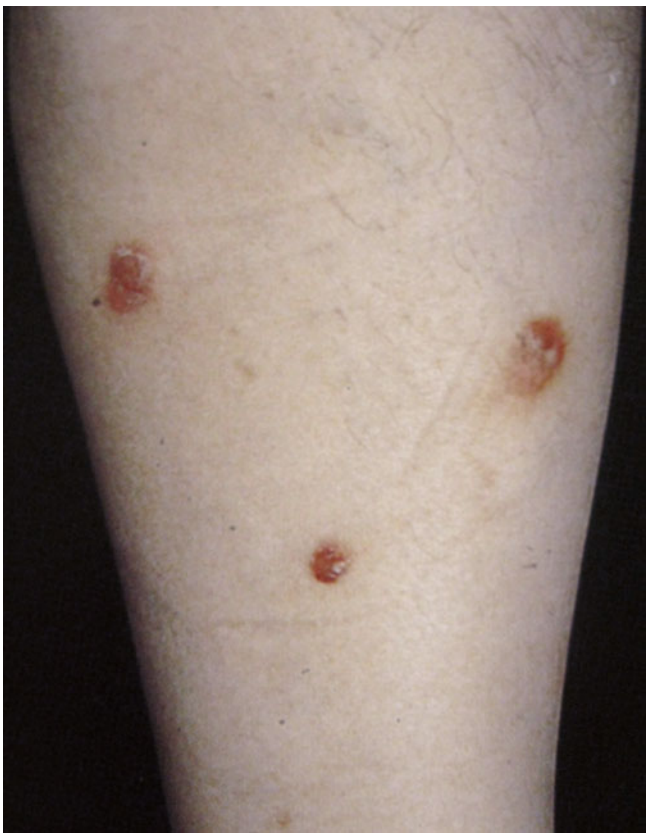


Fig. 41.3 Multiple dermatofibromas have been associated with systemic lupus erythematosus

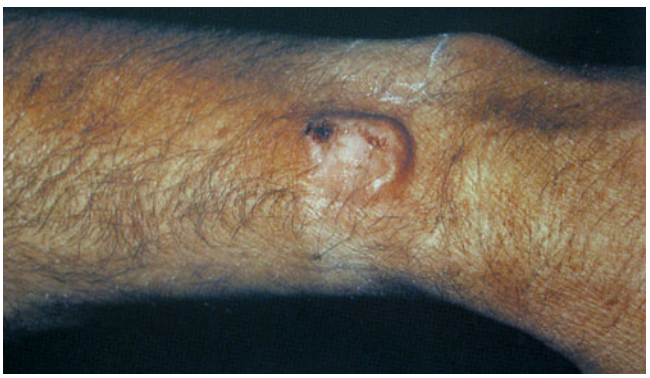


Fig. 41.4 Large dermatofibromas developed beneath a watchband

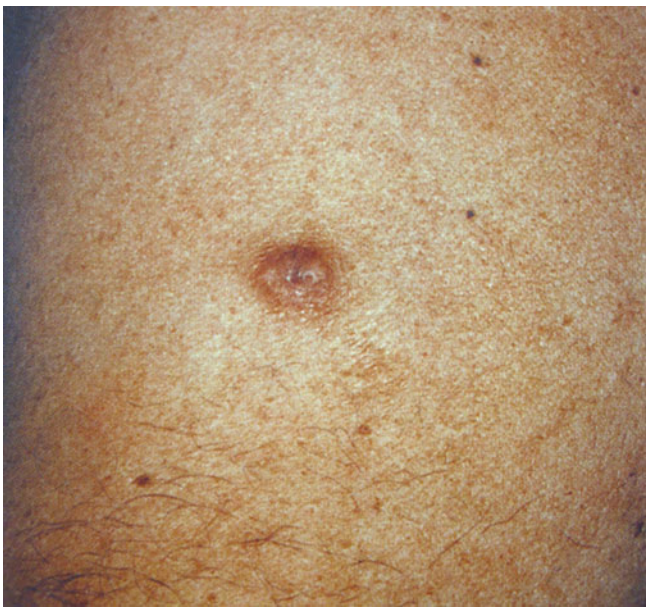
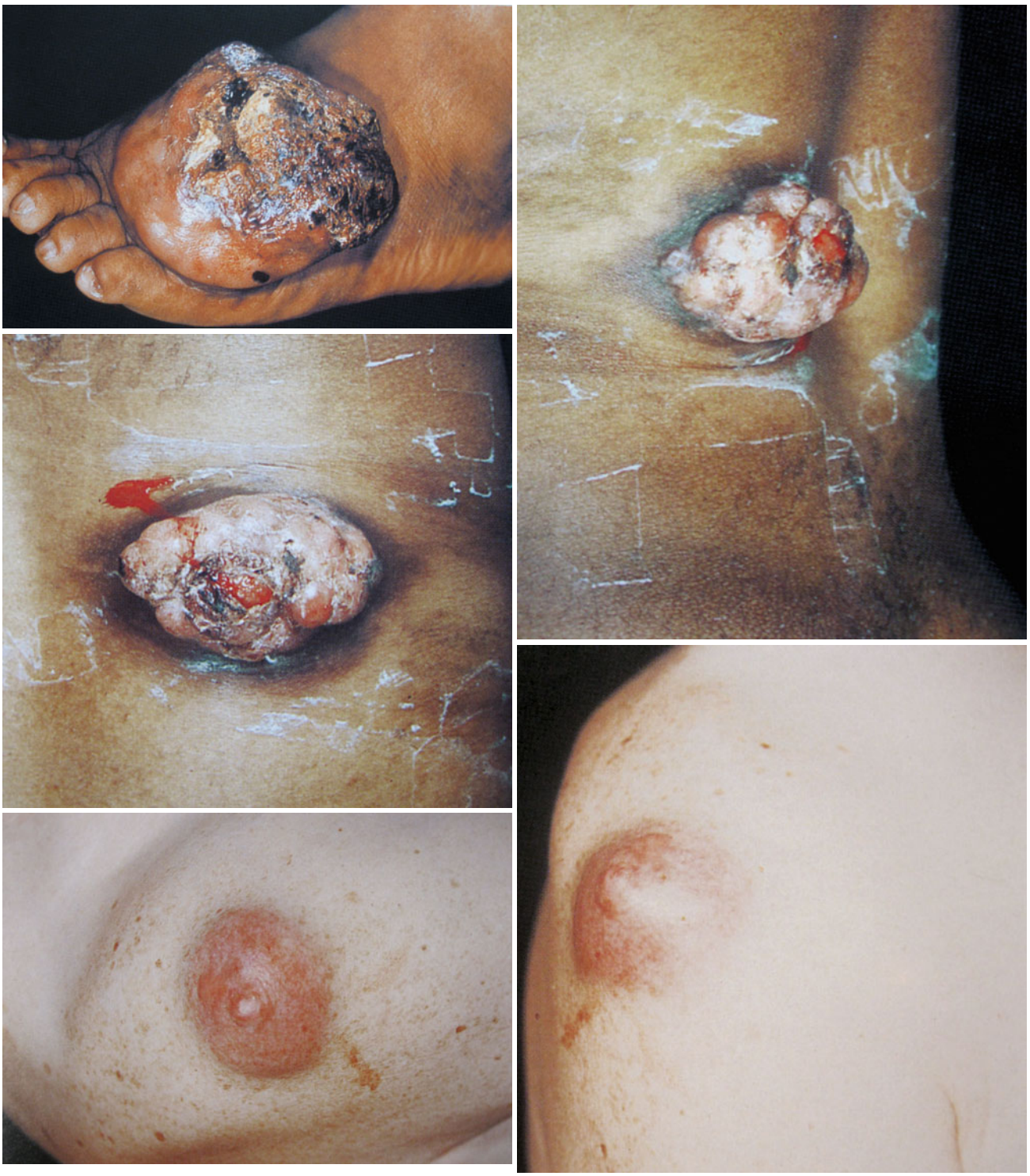


Fig. 41.5 Large dermatofibromas developed at a vaccination site on the upper arm



Figs. 41.6, 41.7, 41.8, 41.9, and 41.10 Dermatofibrosarcoma protuberans (DFSP): large fungating tumors on the foot (Fig. 41.6), back (Figs. 41.7 and 41.8), and typical keloidal nodule on the shoulder (Figs. 41.9 and 41.10)

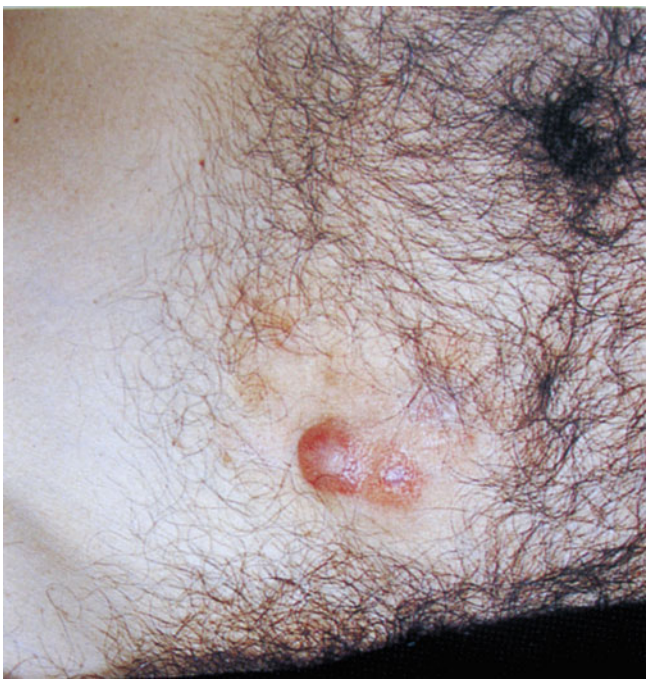


Fig. 41.11 Dermatofibrosarcoma protuberans in the groin represents metastases from the primary lesion on the back

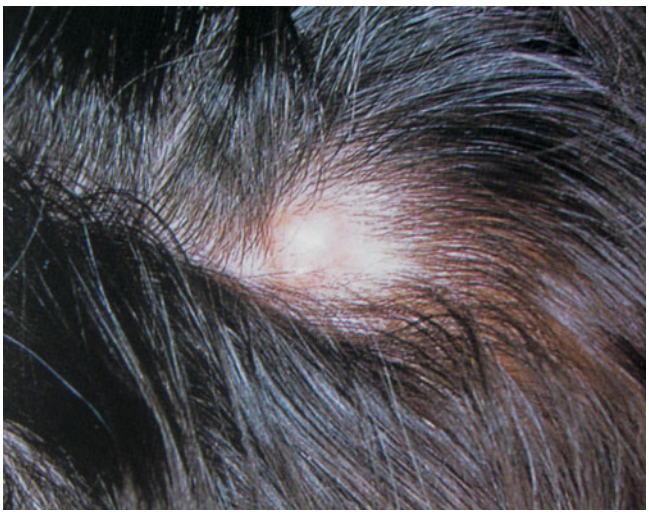


Fig. 41.12 Dermatofibrosarcoma protuberans in the scalp represents metastases from the primary lesion on the back

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Intraepidermal Neoplasia

42

Definitions

Bowen's disease is a squamous cell carcinoma-in-situ. Paget's disease and extramammary Paget's disease are adenocarcinomas-in-situ.

Clinical Description

Bowen's disease presents as one or multiple red, scaly, eczematous or psoriasiform papules, patches, or plaques that may be associated with arsenic ingestion and arsenical keratoses. Arsenical keratoses on the palms and soles may resemble small calluses but are not restricted to points of pressure.

Paget's disease is usually a single, red, scaly, often exudative patch or plaque that extends from the nipple and areola onto adjacent skin. Extramammary Paget's disease is a red, scaly, or macerated (especially when intertriginous) patch or plaque. It commonly occurs in the axilla, groin, scrotum, vulva, or gluteal areas, all sites of apocrine glands.

Etiology and Pathophysiology

Bowen's disease is associated with excessive sun or radiation exposure, or with arsenic ingestion. Patients with prior arsenic exposure warrant a work-up for possible underlying malignancy. The risk of internal malignancy in Bowen's disease without arsenic ingestion is still controversial [1–4]. Bowen's disease is clinically distinct from bowenoid papulosis of the penis, a human papilloma virus-associated intraepidermal carcinoma.

Paget's disease is a manifestation of underlying early intraductal adenocarcinoma of the breast.

Extramammary Paget's disease may represent a cutaneous manifestation of underlying adenocarcinoma of apocrine glands (e.g., rectal carcinoma).

Diagnosis

The toluidine blue test identifies areas of intraepidermal squamous carcinoma [5, 6]. Application of the metachromatic nuclear stain, 1 % toluidine blue, may aid in the recognition of the lesion and its margins. The erythroplastic (neoplastic) areas stain blue, whereas simple erythema remains unstained.

Histopathology

Bowen's disease shows a psoriasiform, regularly acanthotic epidermis with full-thickness squamous cell atypia, mitoses, and malignant dyskeratosis. Origin in, and involvement of hair follicles distinguishes Bowen's disease from bowenoid actinic keratosis. Paget's and extra mammary Paget's disease show clear, round, large Paget cells that involve the hair follicles and the entire epidermis, with sparing and compression of the unaffected basal cell layer.

Differential Diagnosis

The differential diagnosis includes psoriasis, eczema, superficial basal cell carcinoma, and tinea.

Therapy

Treatment involves the following [7–14]:

1. Excision should be carried down to the level of the hair follicle, the origin of Bowen's disease.
2. Underlying carcinoma must be ruled out. Keratoses on the palms, sales, and trunk suggest prior arsenic ingestion.

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Prognosis

Bowen's disease can progress to squamous cell carcinoma, which may metastasize. All three conditions described may portend an underlying carcinoma whose prognosis overshadows that of the cutaneous lesion.

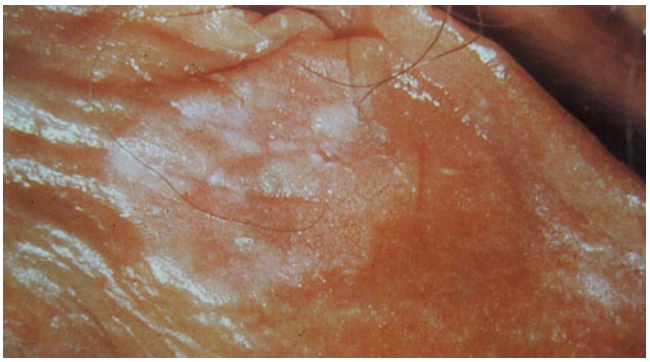


Fig. 42.1 Bowen's disease (squamous cell carcinoma in-situ) of the labia majora can easily be mistaken for a patch of eczema, psoriasis. seborrhea, tinea, or superficial basal cell carcinoma

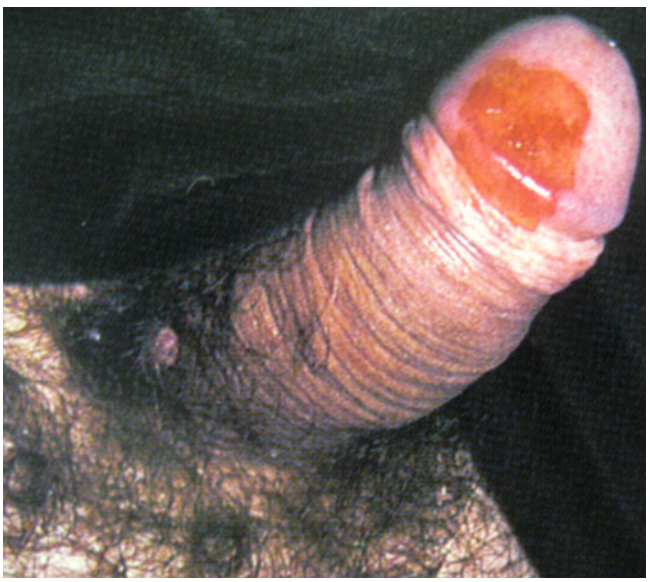
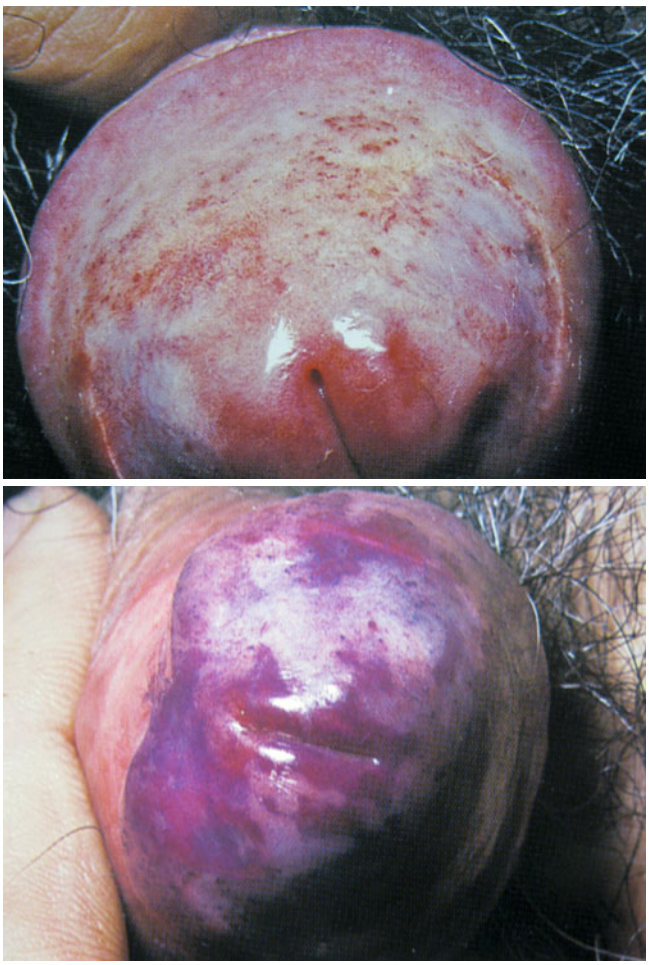


Fig. 42.2 Squamous cell carcinoma-in-situ of the glans penis, known as erythroplasia of Queyrat, shows a sharply demarcated, bright red, velvety plaque that is morphologically similar to the benign plasma cell balanitis of Zoon



Figs. 42.3 and 42.4 Toluidine blue test (before and after) identifies areas of intraepidermal squamous cell carcinoma

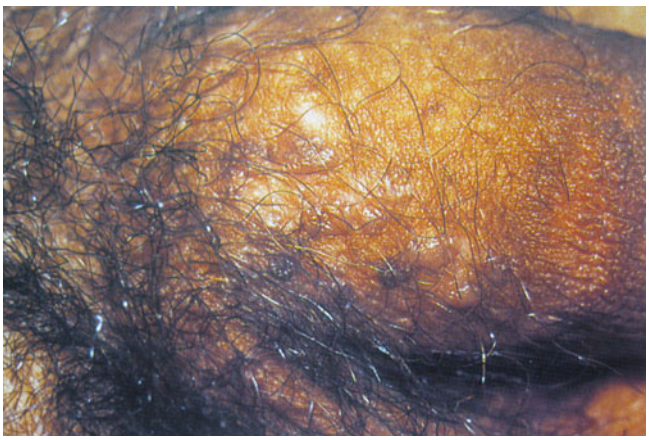


Fig. 42.5 Bowenoid papulosis of the penile shaft clinically resembles genital warts (condylomata acuminata), but histologically is a papular form of Bowen's disease. Similar histologic changes, however, can be seen in genital warts treated repeatedly with podophyllin. Human papillamavirus immunophenotyping can help distinguish these conditions

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Kaposi's Sarcoma

43

Synonyms

Synonyms include multiple idiopathic hemorrhagic sarcoma and "classic" Kaposi's sarcoma (KS).

Clinical Description

Kaposi's sarcoma presents as blue, red, or brown patches, papules, plaques, and nodules on the lower legs and feet, with ulceration and edema. KS most often occurs in elderly men of Mediterranean origin [1].

Etiology and Pathophysiology

Kaposi's sarcoma is a multifocal vascular, endothelial tumor related to immunosuppression; approximately 10 % of patients eventually develop lymphoma or leukemia. The HIV I (human immunodeficiency virus, formerly called HTLV III) retrovirus has been postulated for AIDS-related and African Kaposi's sarcoma [2, 3]; another infectious agent may explain Kaposi's sarcoma in HIV negative patients [2, 4–9].

Histopathology

Early patch stage lesions show irregularly dilated, jagged, anastomosing, thin-walled vascular slits containing erythrocytes. Plasma cells are frequent. Later plaque and nodular stage lesions show vascular proliferations surrounded by spindle cells that extend irregularly in various directions [8].

Differential Diagnosis

The clinical differential diagnosis includes severe varicose veins and stasis dermatitis. Histologically, fibrosarcoma, dermatofibroma (sclerosing hemangioma type), and pyogenic granuloma can resemble the nodules of Kaposi's sarcoma.

Therapy

Treatment includes the following [10–13]:

1. Radiation therapy
2. Excision of a solitary lesion reported to be curative8 (but this is a *multifocal* vascular neoplasm)
3. Chemotherapy with vinblastine and vincristine
4. Cryotherapy for small lesions of AIDS-related Kaposi's sarcoma
5. Imiquimod, Thalidomide

Prognosis

About 10 % of patients with classic Kaposi's sarcoma eventually develop lymphoma or leukemia. Classic Kaposi's sarcoma usually has an indolent course, but fulminant cases, more commonly seen in AIDS and African KS, have been reported.

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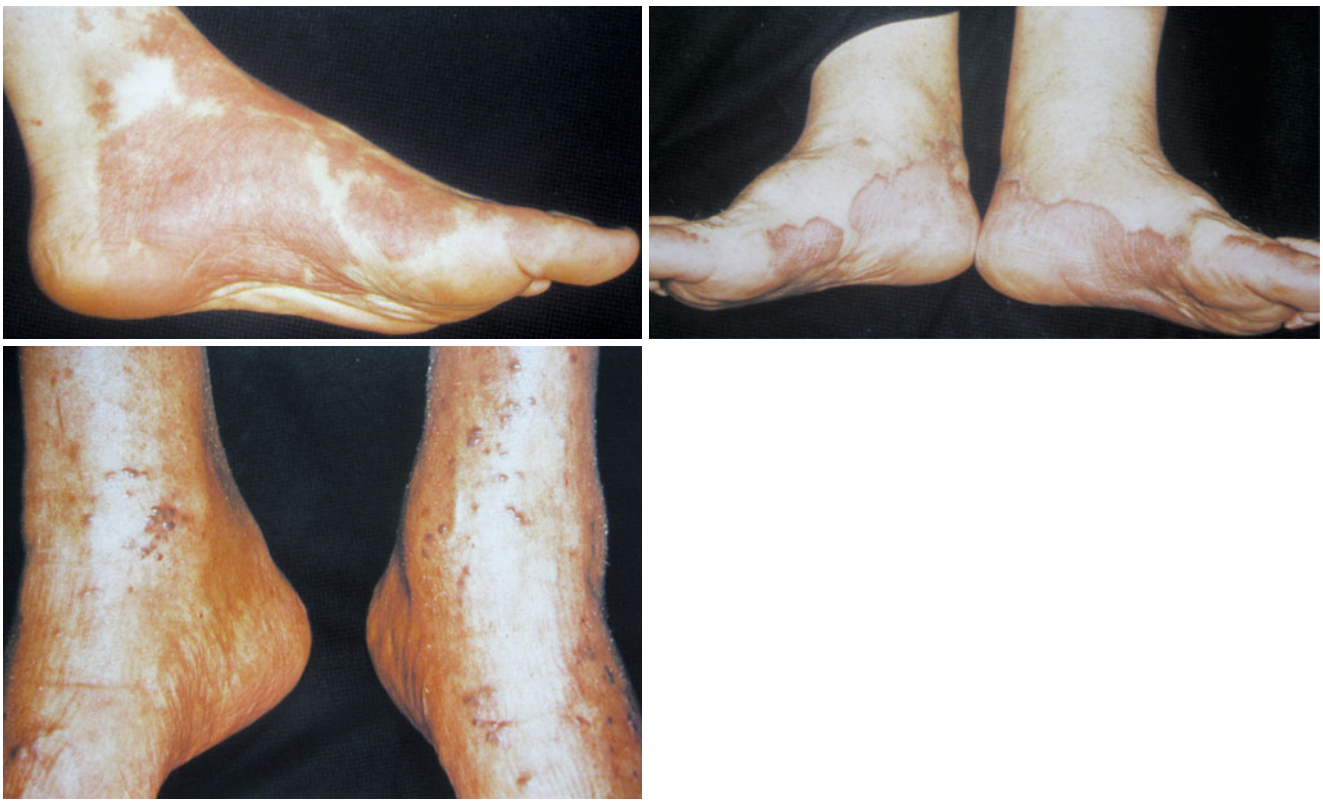

Figs. 43.1, 43.2, and 43.3 Classic Kaposi's sarcoma of the feet shows non-blanching blue-purple patches, papules, and plaques. Involvement of the soles distinguishes it from stasis-varicose dermatitis

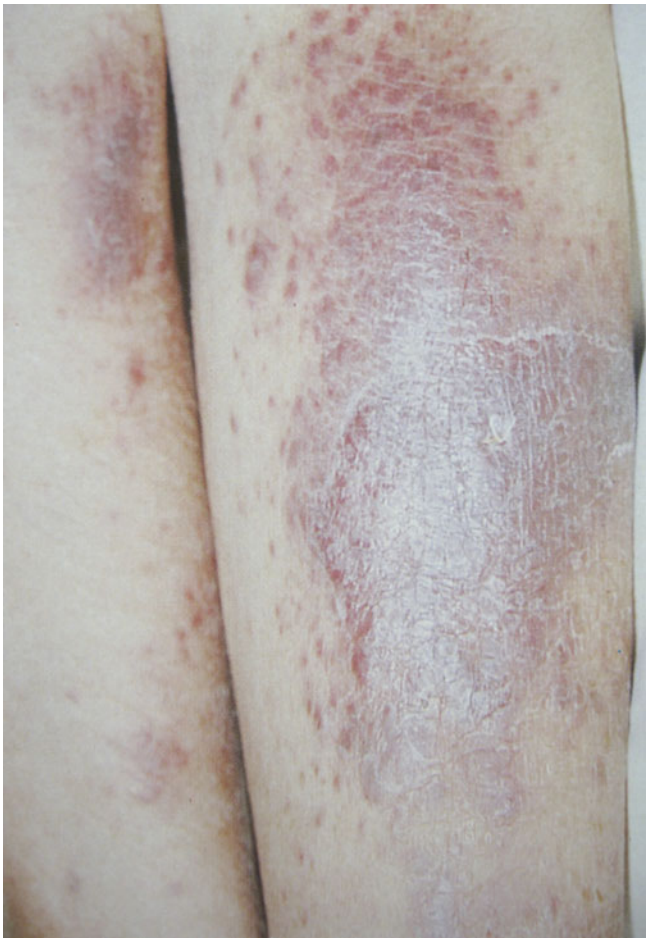

Fig. 43.4 Classic Kaposi's sarcoma of the legs resembles stasis dermatitis, but varicose veins are absent

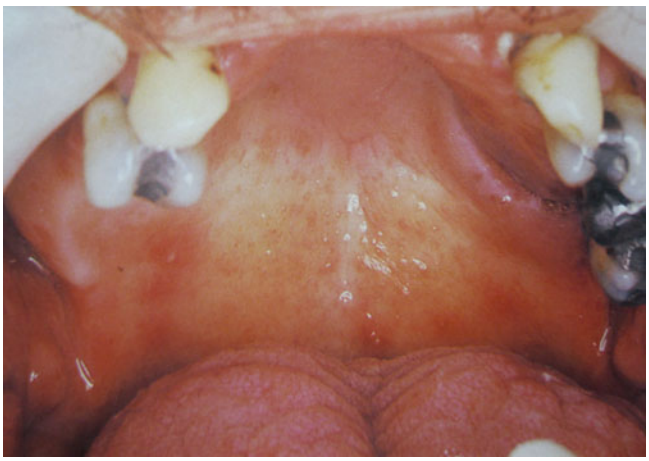

Fig. 43.5 Kaposi's sarcoma of the palate was detected by dental examination in an elderly homosexual male, Oral lesions, particularly on the palate, are more common in homosexual patients with AIDS than in patients who acquire the HIV-I (human immunodeficiency virus) by blood transfusion or intravenous drug usage. A different or additional infectious organism may be responsible

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Keratoacanthoma

44

Synonyms

Keratoacanthoma (KA) is also called "self-healing" squamous cell carcinoma and molluscum sebaceum.

Clinical Description

Keratoacanthoma is a crateriform or volcano-like hard, red papule or nodule with a dense central keratotic plug. KA occurs most commonly on hair-bearing, sun-exposed skin of the elderly. KA may develop and resolve within a few weeks to months (in contrast to squamous cell carcinoma). Two rare syndromes of multiple KAs are the self-healing or familial Ferguson-Smith type, which occurs in younger patients, with 10–20 typical KAs involving even the palms and soles, and the eruptive or Gryzbowski type, which affects adults, with hundreds of smaller, follicular KAs, even on mucosal surfaces.

Etiology and Pathophysiology

The cause of KA is unknown. Sun exposure, radiation, chemical carcinogens, and viruses have all been suggested as predisposing or causative factors [1–4]. Many dermatopathologists consider KA to be a low-grade squamous cell carcinoma.

Histopathology

A complete excisional biopsy is often necessary to distinguish KA from squamous cell carcinoma. Crateriform architecture, glassy eosinophilic keratinocytes, and a mixed inflammatory infiltrate with eosinophils may help distinguish KA from squamous cell carcinoma; both show irregular acanthosis, mitoses, and dyskeratosis.

Differential Diagnosis

The differential diagnosis includes squamous cell carcinoma, basal cell carcinoma, occasionally deep fungal infections and other granulomatous processes [5–9].

Therapy

Complete surgical excision or saucerization, followed by electrodesiccation, curettage, and cautery, is preferred unless the lesions are multiple or the patient cannot tolerate surgery. Alternative therapies include the following:

1. Intralesional or topical 5-fluorouracil and bleomycin
2. Radiation
3. Isotretinoin 1.5 mg/kg/day for multiple KAs

Prognosis

Spontaneous involution may occur in 4–6 weeks.

Satellite nodules of KA may develop after biopsy and even after thermal burns. Because of the difficulty of distinguishing keratoacanthoma from squamous cell carcinoma, it is probably wise to regard KA as a low-grade form of squamous cell carcinoma. Reports of metastasis of clinically "classic" KAs (which may have been misdiagnosed squamous cell carcinomas) further support this recommendation.

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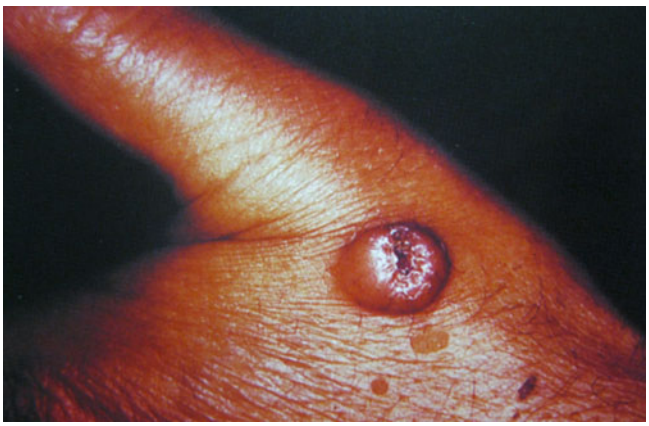
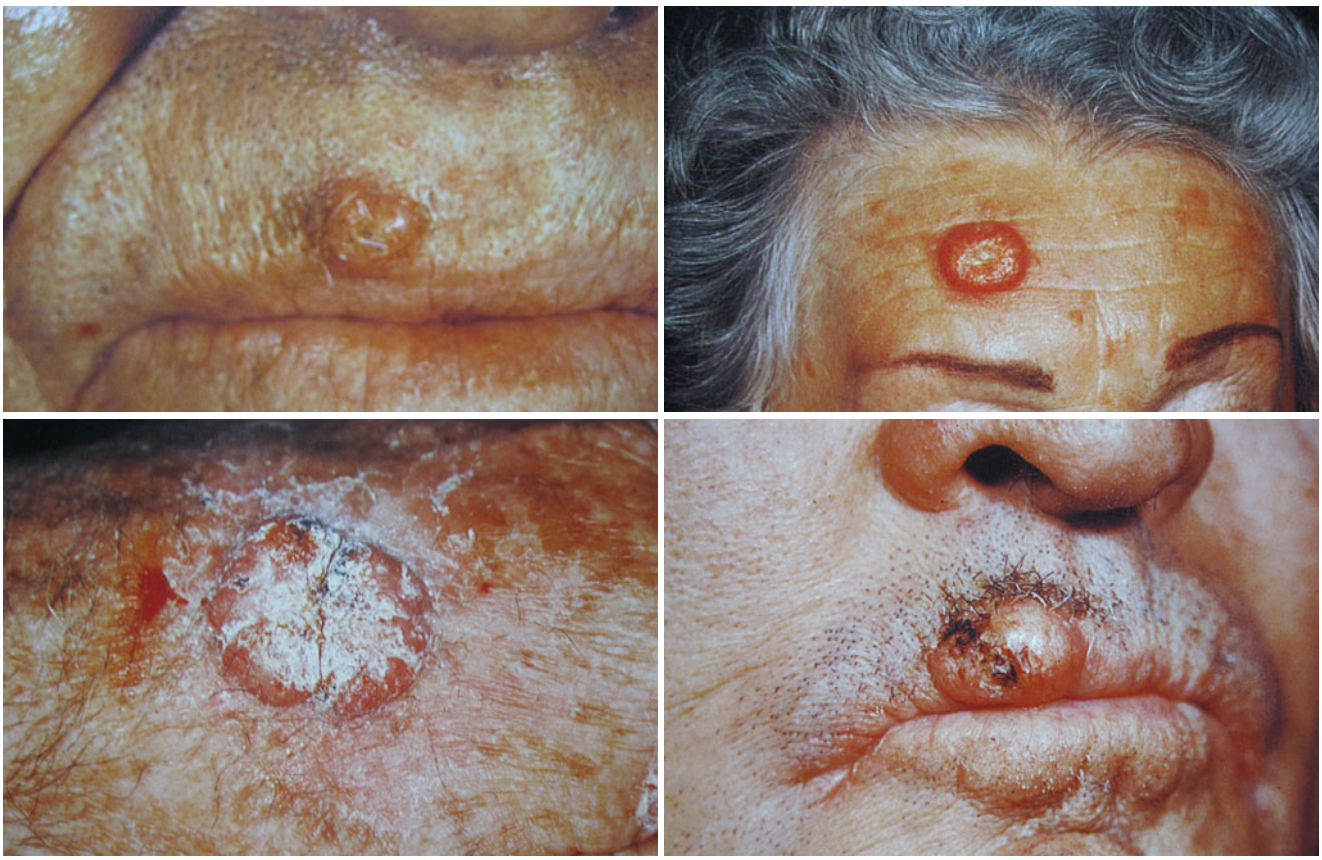
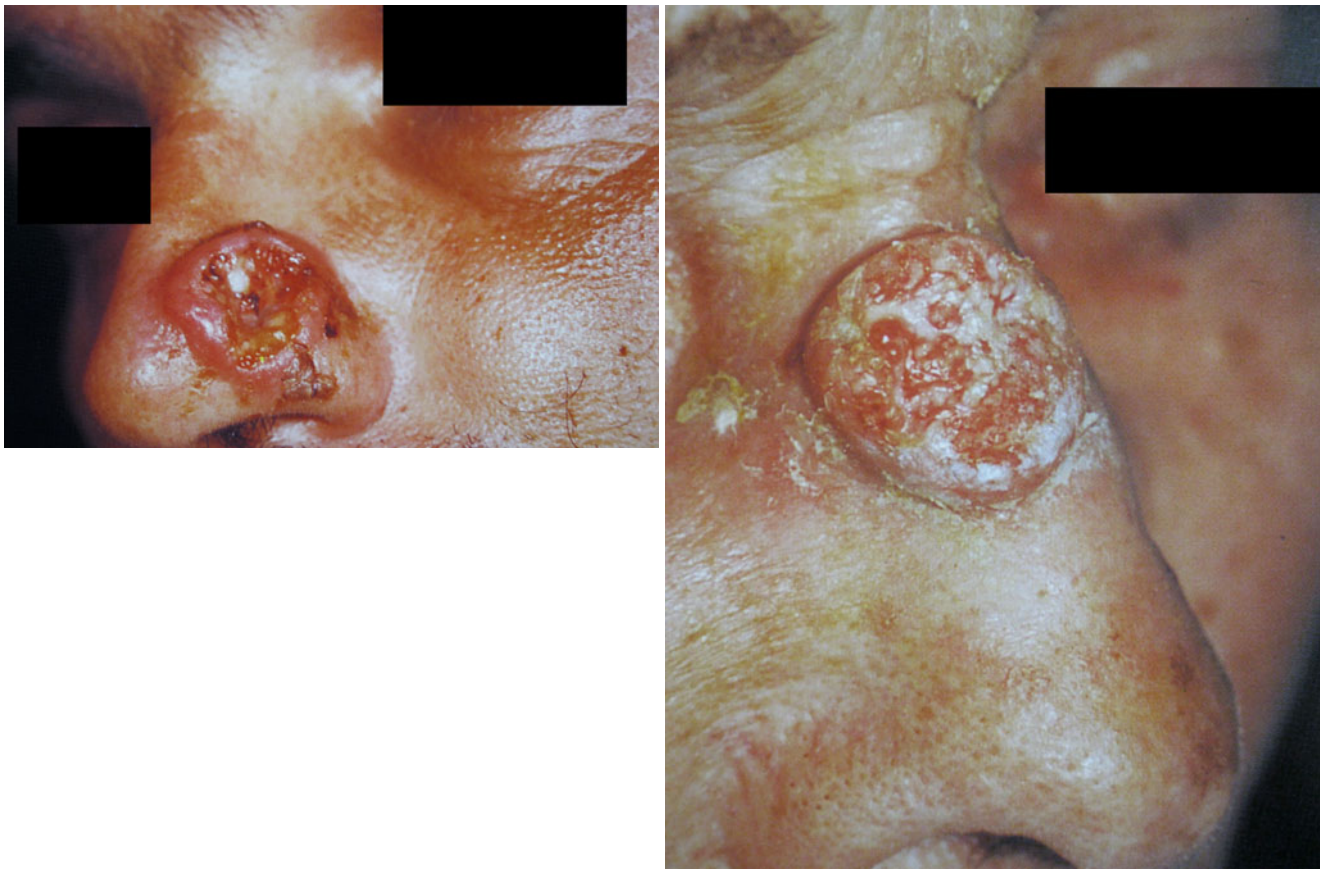


Fig. 44.1 Classic keratoacanthoma shows a dome-shaped, erythematous nodule with a central, dark, keratotic plug, reminiscent of a volcano



Figs. 44.2, 44.3, 44.4, and 44.5 Multiple examples of typical solitary keratoacanthomas



Figs. 44.6 and 44.7 Large, ulcerative keratoacanthomas of the nose clinically resemble basal cell carcinomas

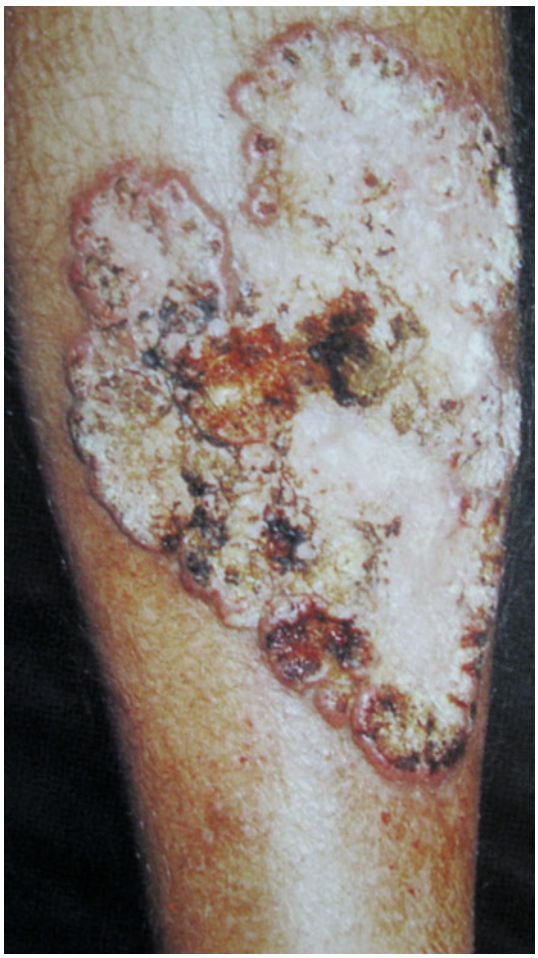


Fig. 44.8 Giant keratoacanthoma clinically *resembles* deep *fungus* infection and other *infectious granulomas*

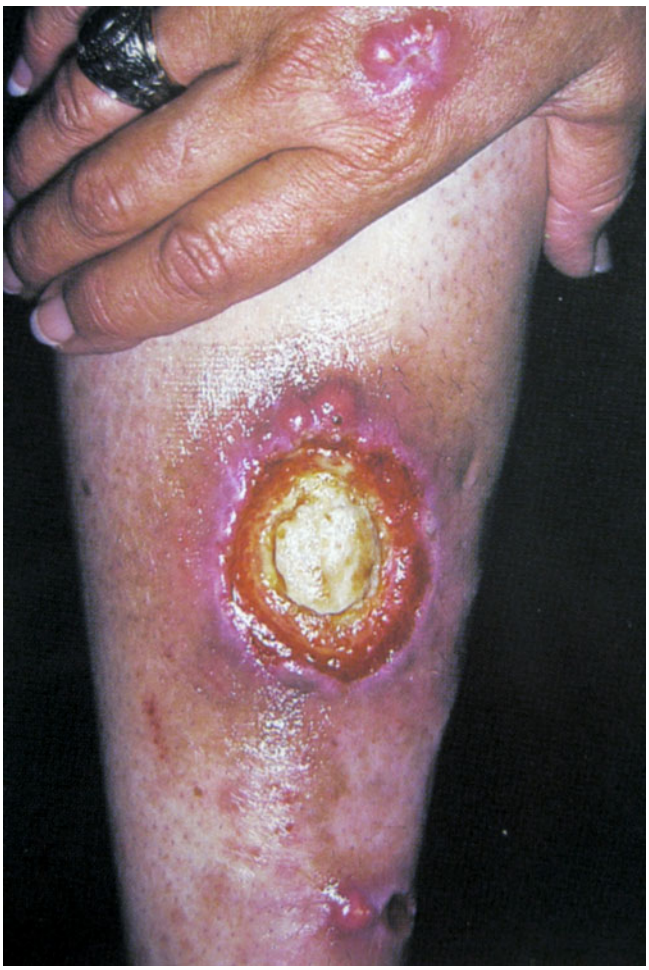


Fig. 44.9 Multiple keratoacanthomas in a patient with Ferguson-Smith syndrome (Courtesy of Dr. S. L. Stedinger)

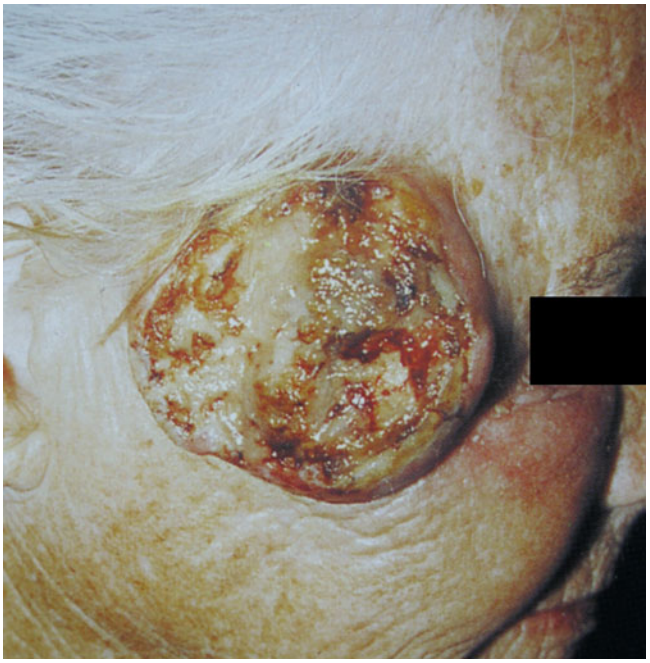


Fig. 44.10 Giant mutilating keratoacanthoma of the cheek may invade and *metastasize just* like a squamous cell carcinoma. Complete removal of keratoacanthomas at an early stage can prevent this tragedy. Satellite nodules may develop around the initial growth. Patients must be warned of this possibility

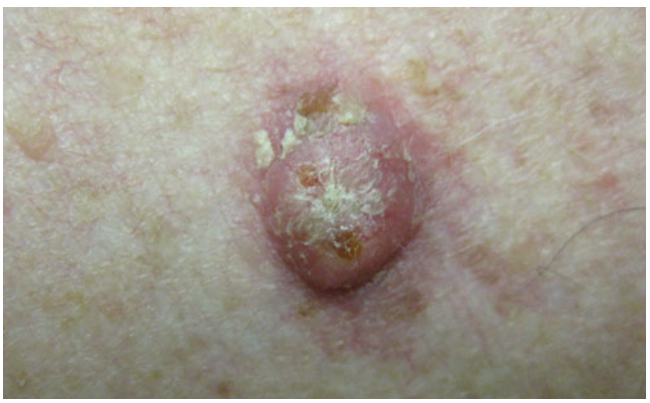


Fig. 44.11 A 64-year-old white male with history of multiple skin cancers presented with a 6 week growth on the right upper chest. Examination showed a firm, round, dome shaped, fixed lesion. It had a light pink color and was covered by irregular yellow-white scale on the top

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Lentigo Maligna

45

Synonyms

Lentigo maligna is also known as Hutchinson's melanotic freckle, melanosis circumscripta preblastomatosa of Dubreuilh, and melanoma-in-situ [1–4].

Clinical Description

Lentigo maligna appears as a brown-black macule or patch with irregular borders and uneven pigmentation on sun-damaged, elderly skin, especially the face [5].

Etiology and Pathophysiology

Chronic sun exposure causes malignant degeneration of melanocytes.

Histopathology

This melanoma-in-situ shows epidermal atrophy, with loss of rete ridges, lentiginous or continuous horizontal proliferation of atypical, spindle-shaped melanocytes along the basal cell layer and down hair follicles, hyperpigmentation, and dermal solar elastosis [6].

Differential Diagnosis

The differential diagnosis includes the following:

1. Solar lentigo-even pigmentation, smaller
2. Lentigo maligna melanoma-papular component is present clinically, with dermal invasion of atypical melanocytes histologically [7]

Therapy

Treatment involves excision, including sequential, staged excision for large lesions (to minimize scar length) and Mohs microscopically controlled surgery for lesions near critical anatomic areas (e.g., eyes, ears, nose).

Note that nonsurgical therapies, such as liquid nitrogen cryotherapy, trichloroacetic acid chemical peels, and hydroquinone bleaching creams, may remove the pigment but not the atypical melanocytes, delaying diagnosis and proper surgical therapy [8–13].

Prognosis

Lentigo maligna is the premalignant precursor lesion to lentigo maligna melanoma. Malignant progression occurs in about one-third of lesions after 10–15 years.

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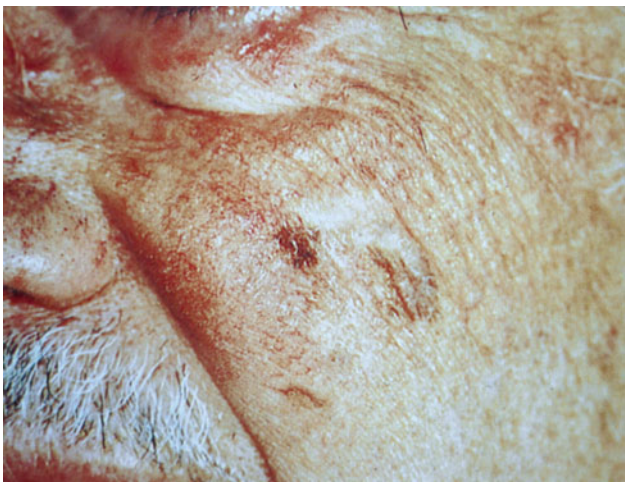

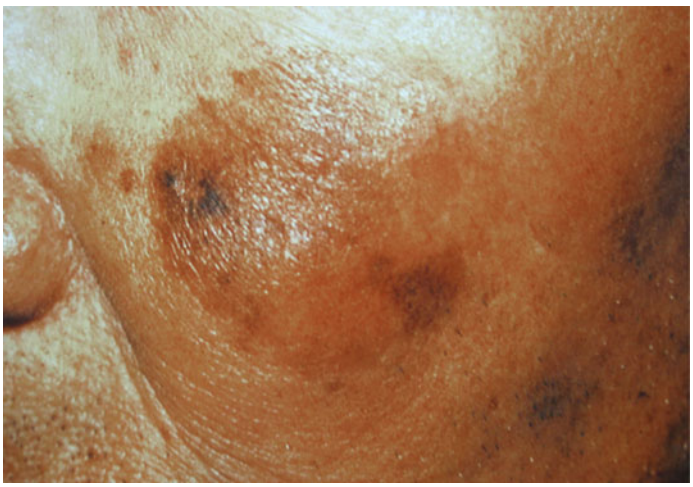

Figs. 45.1 and 45.2 Lentigo maligna typically presents as a large patch on the cheek, with different shades of *brown* and sometimes *white* and/or *black* areas

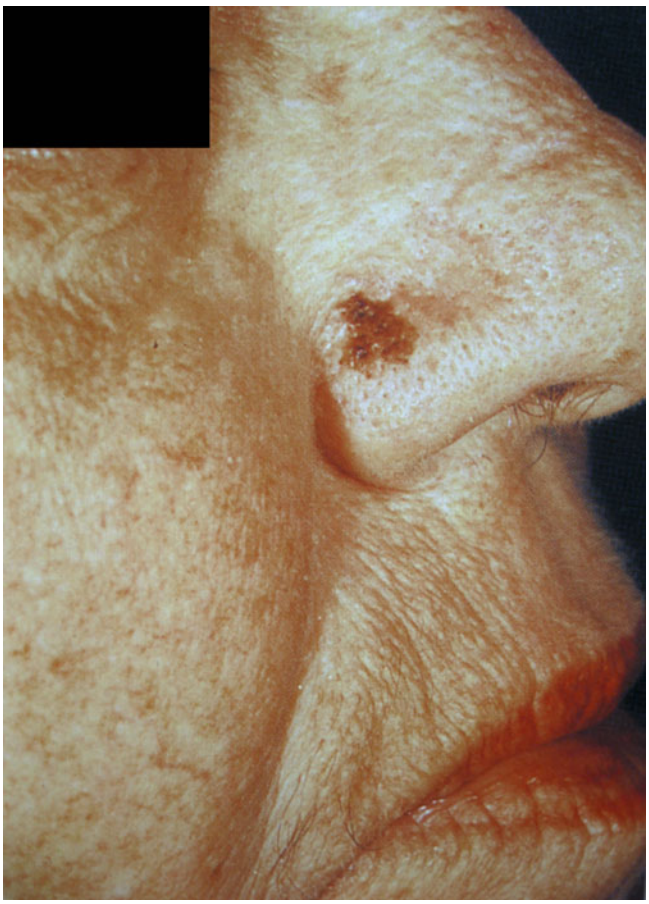

Fig. 45.3 This lentigo maligna on the nose could easily be mistaken for a pigmented basal cell carcinoma. Histologic investigation is diagnostic and mandatory. Cryosurgery, chemical peels, bleaches, and even electrodesiccation and curettage may not completely eradicate the atypical melanocytes of this melanoma precursor

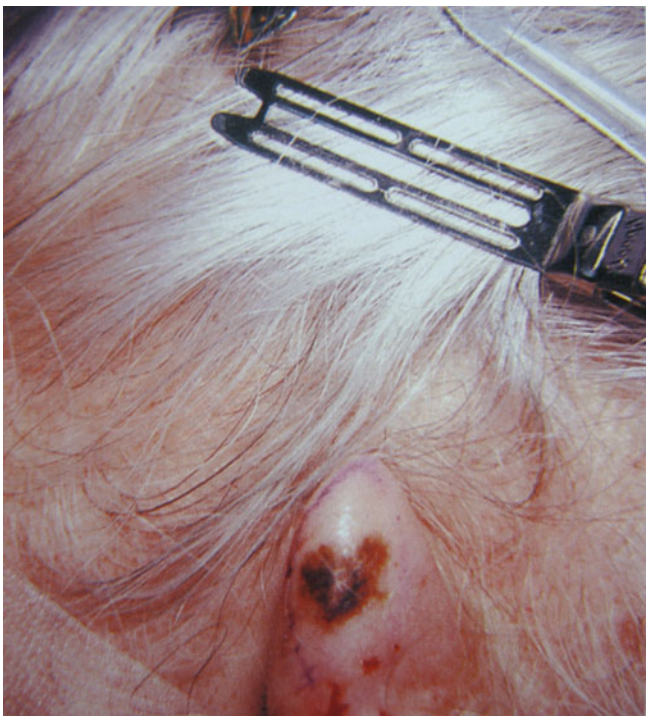

Fig. 45.4 Lentigo maligna on the helical rim

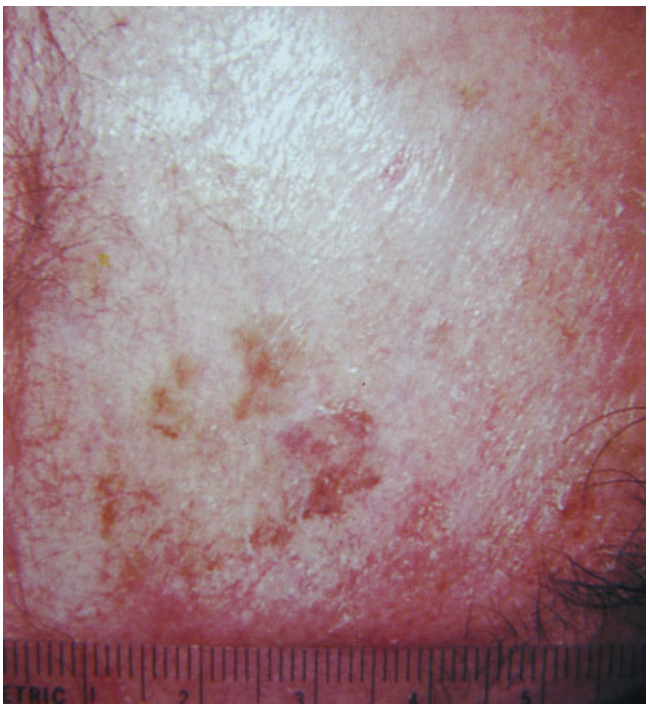

Fig. 45.5 Lentigo maligna on the forehead

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Cutaneous Lymphoma (Excluding Mycosis Fungoides) and Pseudolymphoma

46

Synonyms

Entities include the following:

1. Specific eruptions of leukemia and nonmycosis fungoides lymphomas, such as Hodgkin's disease. Chloroma (granulocytic sarcoma) represents cutaneous acute or chronic myelogenous (granulocytic) leukemia; the greenish color of freshly cut tissue is caused by the presence of large amounts of myeloperoxidase.
2. Lymphocytic infiltration of Jessner-Kanof.
3. Other "pseudolymphomas": chronic insect bite reaction; postscabetic nodules.

Mycosis fungoides (cutaneous T-cell lymphoma) is discussed in Chap. 47.

Clinical Description

Cutaneous lymphoma and pseudolymphoma are characterized by red-brown or violet-plum dermal plaques or nodules with patulous (dilated) follicular orifices (peau d'orange or orange peel appearance).

Etiology and Pathophysiology

Specific eruptions include the following:

1. Dermal infiltration by leukemic or lymphoma cells can occur.
2. Jessner-Kanof lymphocytic infiltrate may represent the earliest manifestation of discoid lupus erythematosus (negative antinuclear antibody, negative direct immunofluorescence), plaque-type PMLE (polymorphous light eruption) lymphocytoma cutis, or lymphocytic lymphoma (the "five Ls").'

"Pseudolymphomas" include the following:

1. A persistent reaction to insect or scabies body parts, toxins, ova, and feces, is seen.
2. Nonspecific eruptions of cutaneous lymphoma include morbilliform rashes, urticaria, ichthyosis, erythema multiforme, and annular erythemas. These can be seen in association with cutaneous lymphomas, but are usually due to other causes (see Chaps. 6 and 18) [1–7].

Histopathology

Histopathologic investigation reveals the following:

1. Specific leukemic and lymphomatous infiltrates are characterized by a Grenz zone (sparing of the uppermost dermis) with a "bottom-heavy," diffuse, monomorphous dermal infiltrate. Cells may be normal or atypical lymphohistiocytic cells. The dermal infiltrate is usually not diagnostic of the primary leukemia or lymphoma. In cutaneous Hodgkin's disease, however, pathognomonic Reed-Sternberg (owl's eye) cells may be seen
2. Jessner's lymphocytic infiltration shows a normal or flattened epidermis containing well-circumscribed periadnexal and perivascular nodules of lymphoid cells, with fewer histiocytes and plasma cells than seen in discoid lupus erythematosus, but without the lichenoid infiltrate or epidermal changes seen in discoid lupus erythematosus,
3. Pseudolymphomas display a scattered, dense, chronic inflammatory infiltrate, with eosinophils, atypical mononuclear cells resembling those of lymphoma, thickened vessels, and occasional vasculitis. Insect parts are rarely identified,
4. Nonspecific eruptions show the histopathology of urticaria, erythema multiforrne, ichthyosis, or annular erythema without infiltration of leukemia or lymphoma cells.

Differential Diagnosis

The conditions described above can be clinically indistinguishable, Histology is often but not always useful. Immunophenotyping of B- and T-cells using the monoclonal

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antibody immunoperoxidase technique may be helpful. Monomorphic infiltrates are frequently malignant, whereas polymorphic infiltrates are more often benign.

Therapy

Treatment involves the following [8, 9]:

1. Specific leukemic and lymphomatous infiltrates require identification and treatment of the underlying leukemia or lymphoma. Granulocytic sarcoma often presages the fatal progression of acute or chronic myelogenous leukemia to acute blast crisis. Radiation is usually preferred to surgery, because the lesions may extend beyond their clinically apparent borders.
2. For suspected cases of Jessner's, rule out lupus erythematosus with antinuclear antibody, direct immunofluorescence, light testing, and sun avoidance for polymorphous light eruption. Identify and treat incipient leukemia or lymphoma.
3. For the other psuedolymphomas, intralesional or topical steroids and topical tar help relieve itch. Scabetic nodules occur and persist, despite adequate scabies treatment (see Chap. 23).

Prognosis

For specific leukemic and lymphomatous infiltrates, the prognosis depends on the underlying hematologic malignancy. Jessner's may eventually evolve into one of the other conditions comprising the "five Ls." The other pseudolymphomas are stubbornly persistent and pruritic, but are not life-threatening.

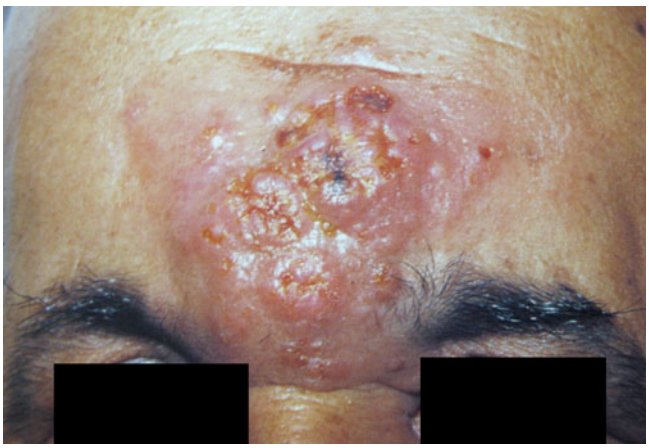

Fig. 46.1 Cutaneous lymphoma presents as a pink nodular plaque. This clinical appearance is nonspecific. Biopsy is essential to rule out tumors and infections

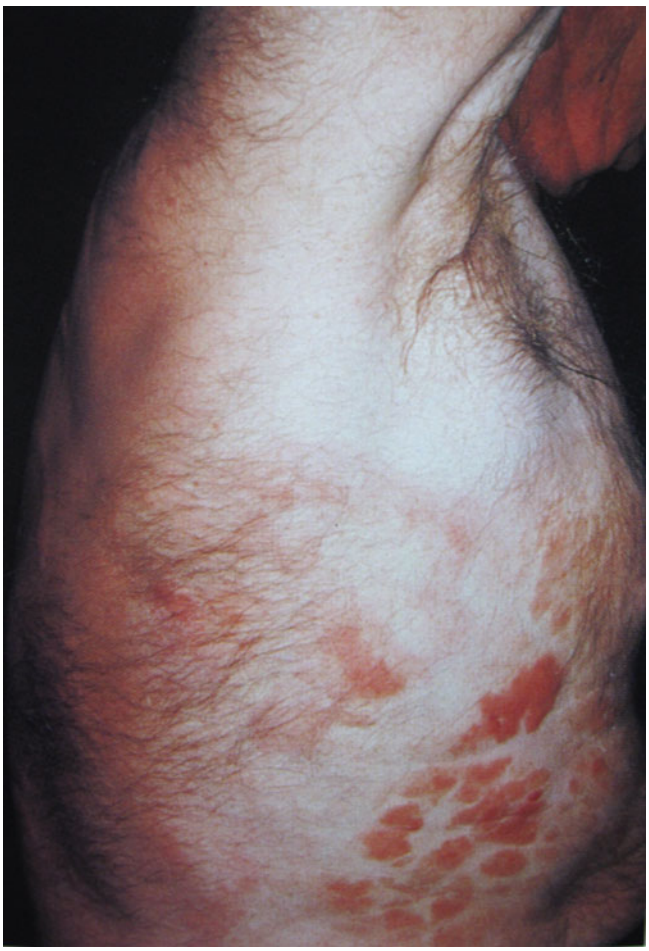

Fig. 46.2 Multiple deep pink plaques in disseminated cutaneous lymphoma another nonspecific morphology

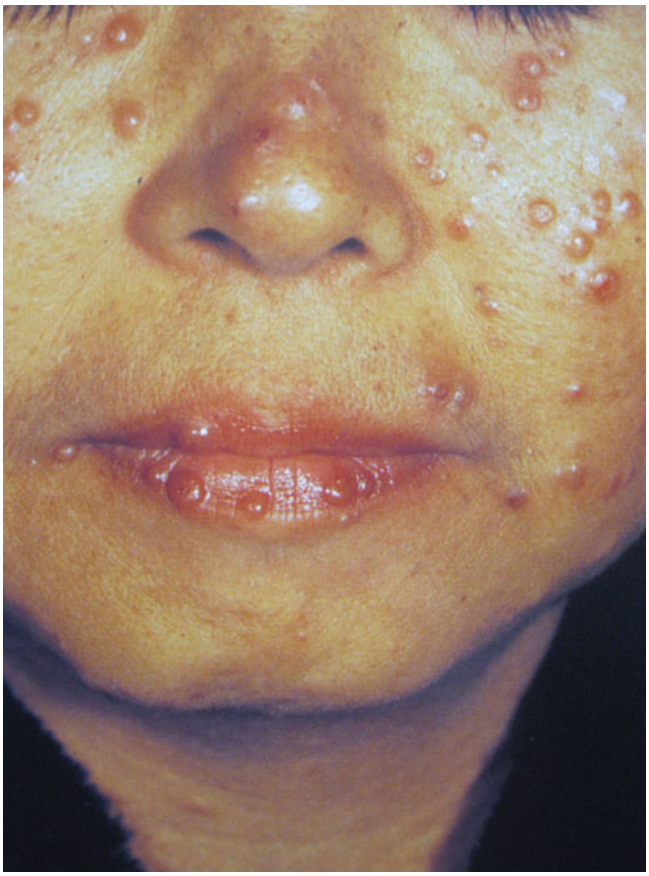

Fig. 46.3 Multiple facial papules require biopsy to diagnose cutaneous lymphoma. These lymphoma papules involve the vermilion of the lips

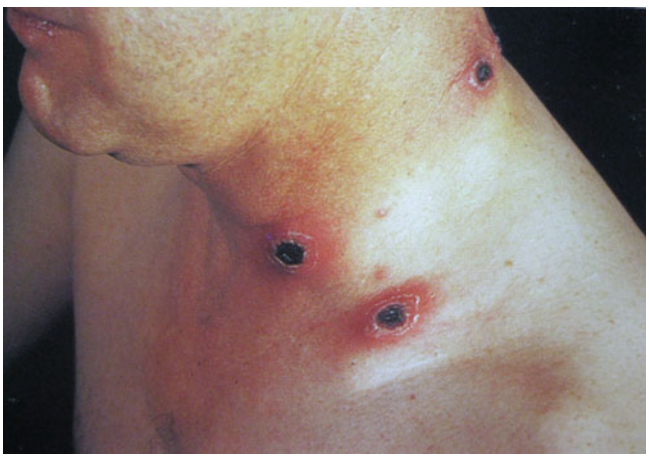

Fig. 46.4 Crusted, erythematous, grouped nodules on the neck simulate an arthropod bite reaction in a patient with underlying lymphoma

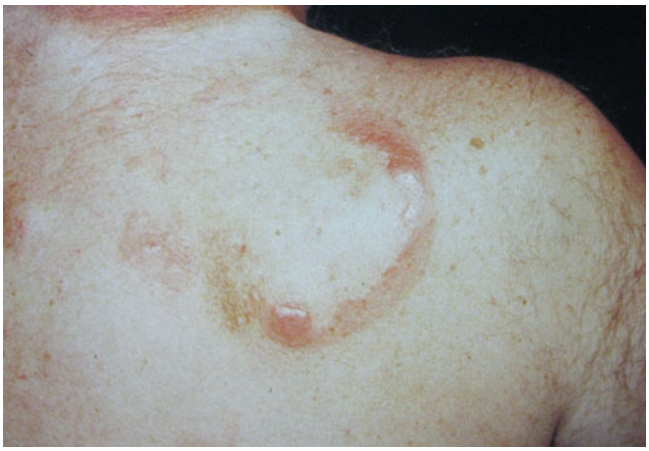

Fig. 46.5 Geographic, arcuate, dermal plaque of cutaneous lymphoma resembles tinea, urticarial, granuloma annulare, and erythema annulare centrifugum

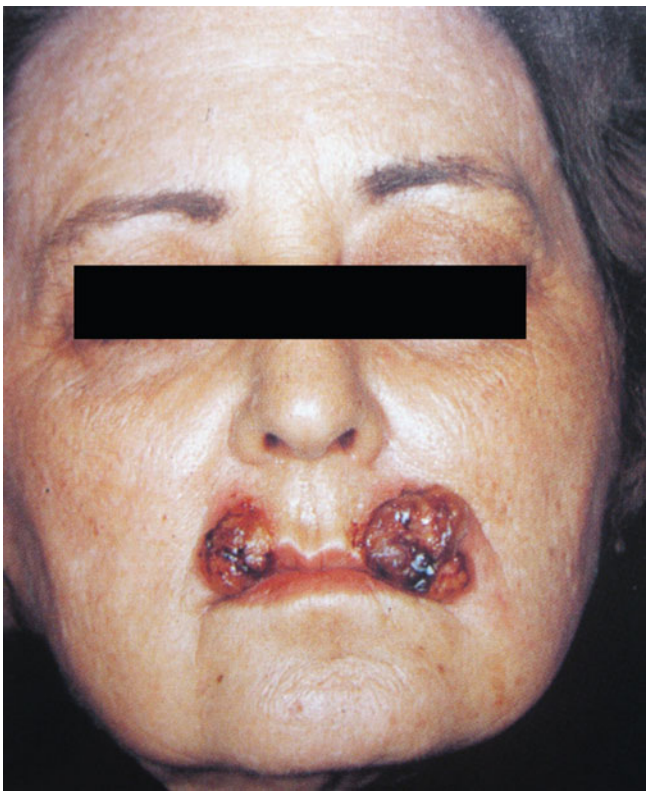

Fig. 46.6 Crusted lymphoma nodules involving the upper lip

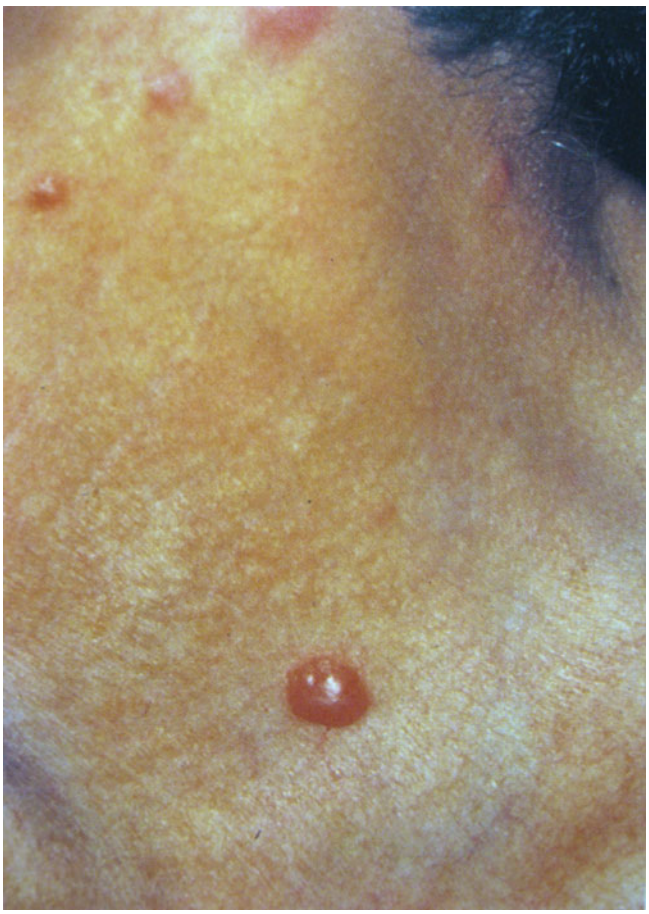

Fig. 46.7 Pseudolymphoma resulting in an insect bite reaction in a healthy patient. Compare with Fig. 46.4. One is crusted and the other is not but both are red nodules

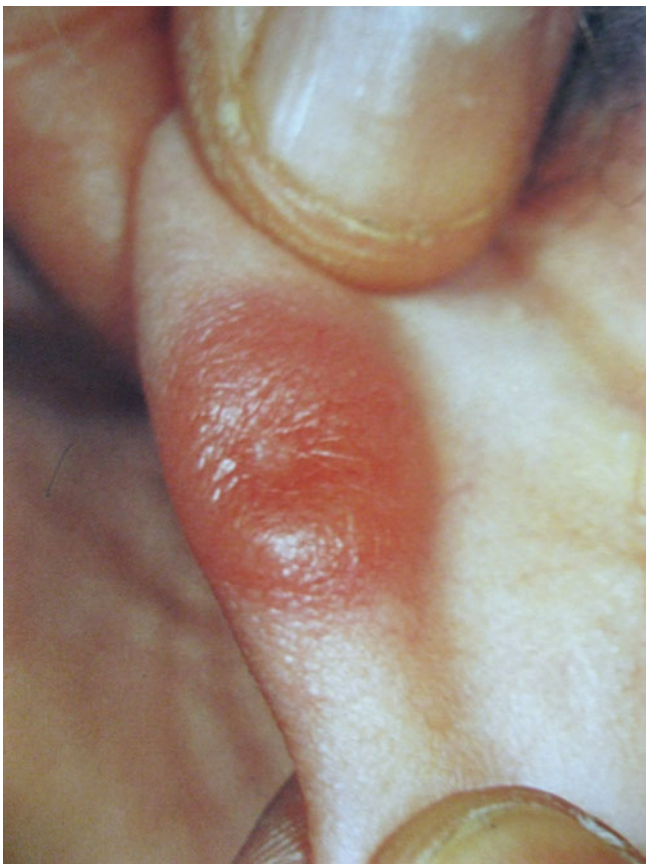

Fig. 46.8 Pseudolymphoma of the posterior ear (helix) resembles a basal cell carcinoma and angiolymphoid hyperplasia with eosinophilia (Kimura's disease)

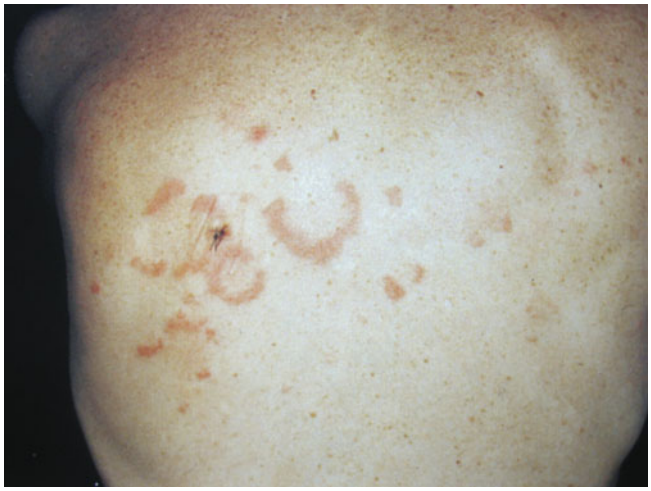

Fig. 46.9 Benign "reactive" lymphoid hyperplasia clinically resembles the lymphoma depicted in Fig. 46.5. Biopsy is mandatory. Immunophenotyping of the cellular infiltrate is often helpful; a monomorphous infiltrate is more likely to represent a malignant clone of cutaneous lymphoma

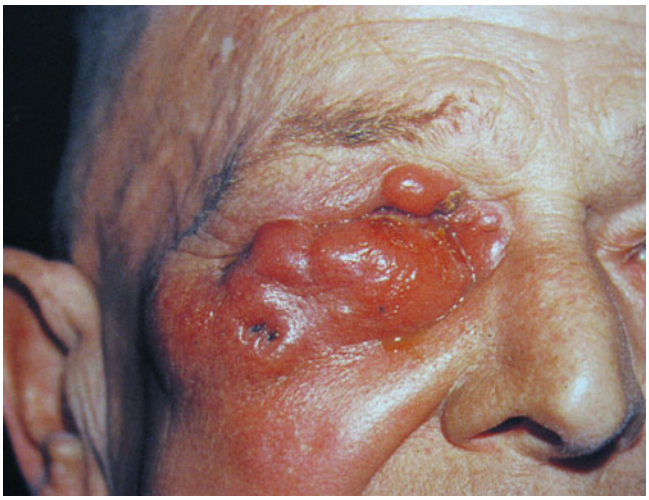

Fig. 46.10 Lymphosarcoma has invaded and obliterated the orbit

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Mycosis Fungoides (Cutaneous T-Cell Lymphoma)

47

Mycosis fungoides is the most common form of cutaneous T-cell lymphoma. In the western population there are around 0.3 cases of Sezary syndrome per 100,000 people. Sézary disease is more common in males with a ratio of 2:1, and the mean age of diagnosis is between 55 and 60 years of age.

Clinical Description

The curious name "mycosis fungoides" refers to the initial clinical description of mushroom-like tumors evolving from a desquamating rash. Four clinical stages are seen:

1. Patch stage-persistent, pruritic, red, pink, or brown patches, with or without scale
2. Plaque stage-persistent, pruritic, red, pink, or brown plaques
3. Tumor stage-persistent red, brown, or violet papules, nodules, and/or tumors (d' emblee type refers to sudden appearance of tumors without previous patches or plaques)
4. Sezary syndrome-exfoliative erythroderma with numerous, bloodborne Sezary cells (convoluted T lymphocytes) [1–6]

Etiology

The cause of mycosis fungoides is still unknown.

Theories include chronic, low-grade contact dermatitis and/or retrovirus (HIV III, HTL V I) infection. (HIV III, also called HTLV I, is different from the AIDS virus, which is HlV I, and is also called HTLV III). The result is a malignant clone of helper T cells [7–10].

Histopathology

Cutaneous T-cell lymphoma shows a lichenoid (band-like) lymphocytic infiltrate with Pautrier microabscesses consisting of collections of atypical cerebriform or hyperconvoluted T lymphocytes in the epidermis, with no or at most minimal spongiosis and a mixed lymphohistiocytic perivascular dermal infiltrate, with variable eosinophils and plasma cells. The lack of spongiosis is one clue to distinguishing cutaneous T-cell lymphoma from eczematous diseases.

Differential Diagnosis

The differential diagnosis includes the following:

1. Patch stage-eczema, tinea, pityriasis rosea, pityriasis lichenoides chronica, secondary syphilis, other papulosquamous disorders
2. Plaque stage-psoriasis, parapsoriasis en plaques (large plaque parapsoriasis may represent a precursor lesion to mycosis fungoides)
3. Tumor stage-squamous cell carcinoma (usually single, not multiple), other lymphomas (cutaneous nodules of Hodgkin's disease and leukemic infiltrates), postscabetic nodules, Kaposi's sarcoma
4. Sezary syndrome-other causes of exfoliative erythroderma, including psoriasis, generalized eczema, drug eruptions, tinea, erythema multiforme (toxic epidermal necrolysis)

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Therapy

Treatment includes the following:

1. Electron beam therapy, orthovoltage radiotherapy
2. Topical chemotherapy-topical nitrogen mustard (mechlorethamine), topical carmustine (BCNU, bischloroethyl nitrosurea)
3. Systemic chemotherapy-methotrexate, cyclosporine
4. Photochemotherapy–PUVA (psoralen plus UV A light)
5. Extracorporeal photochemotherapy-plasmapheresis and PUVA
6. Vorinostat is a second-line drug for cutaneous T-cell lymphoma. Treatments are often used in combination with phototherapy and chemotherapy

No single treatment type has revealed clear-cut benefits in comparison to others, and treatment for all cases remains problematic.

Prognosis

The prognosis is poor, with a gradual but inexorable progression from patch to plaque to tumor stage, which may take over 20 years. Patients often succumb to other diseases, infections, or complications of therapy for mycosis fungoides. Patients with Sézary disease have a median survival of 5 years.

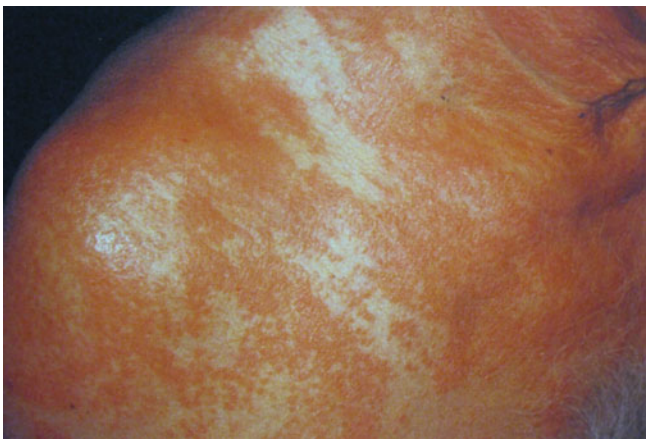


Fig. 47.1 Patch stage of mycosis fungoides. This may be treated as "eczema" for 10–20 years before the correct diagnosis is made by biopsy

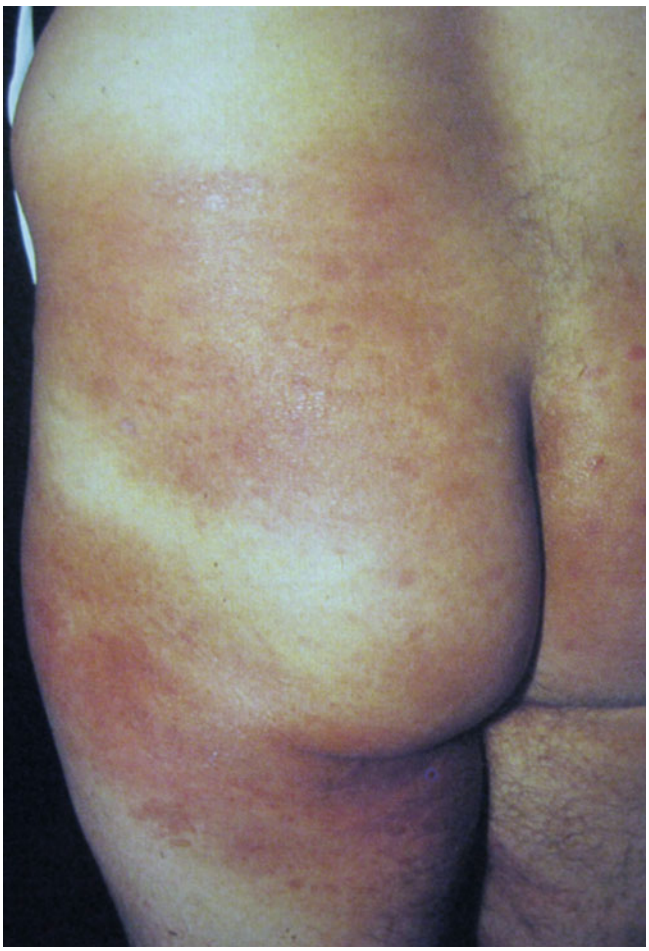
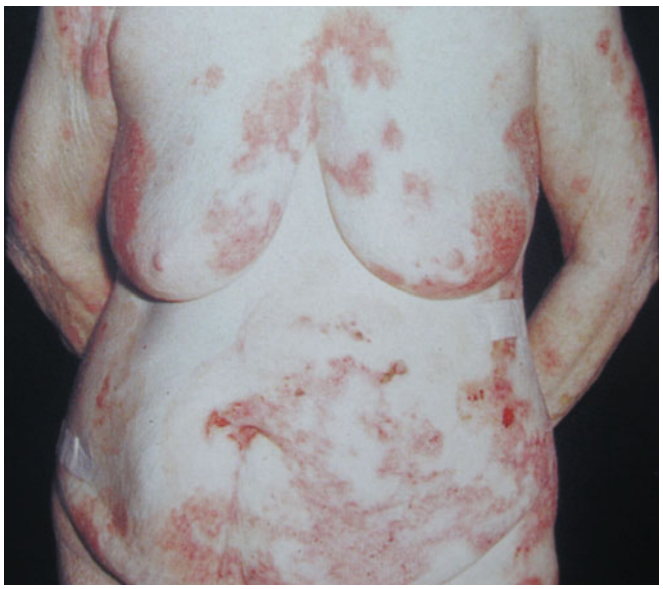
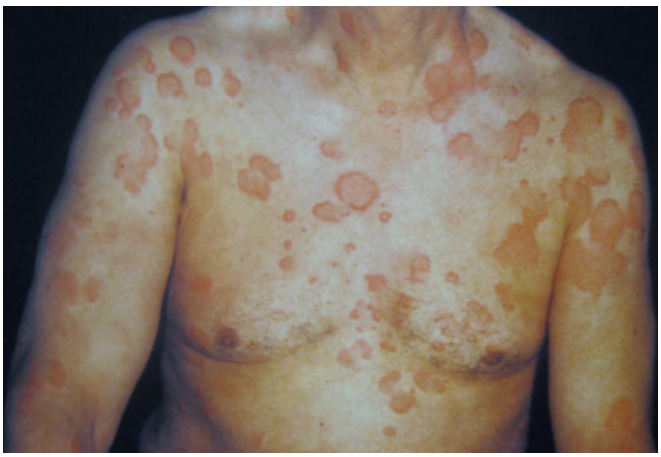
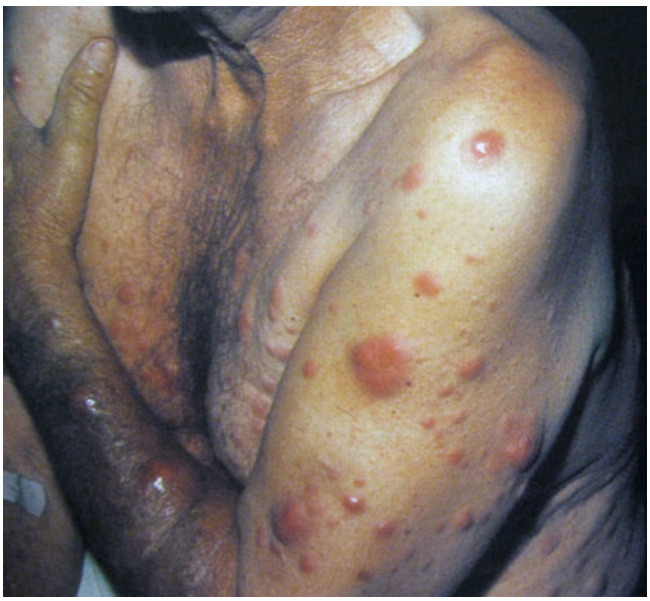


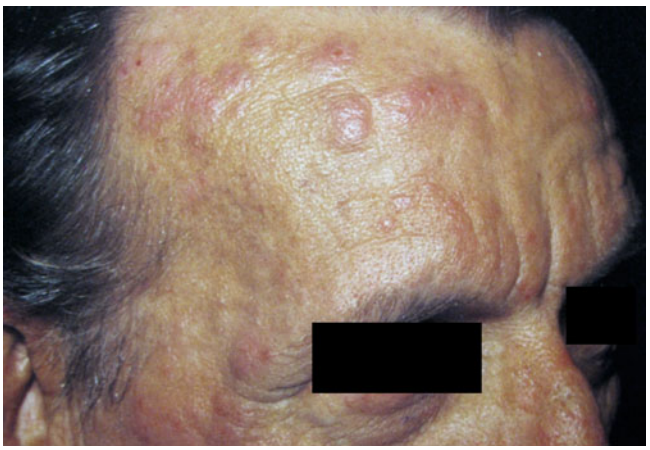
Fig. 47.2 Large plaque parapsoriasis of the thighs and buttocks shows histologic changes of mycosis fungoides

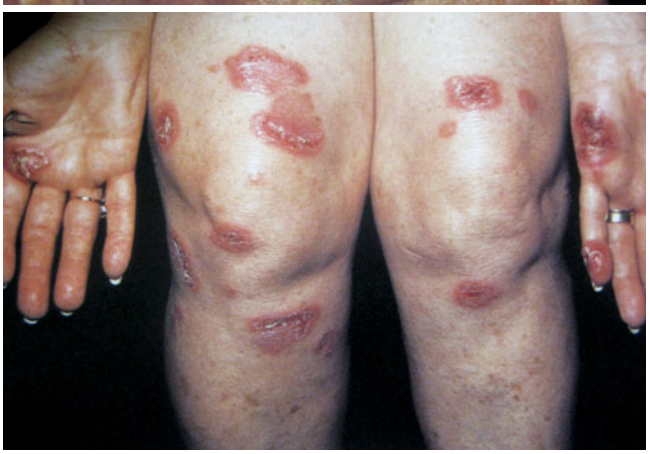




Figs. 47.3 and 47.4 Widespread plaque stage of mycosis fungoides







Figs. 47.5, 47.6, and 47.7 Tumor or nodular stage of mycosis fungoides

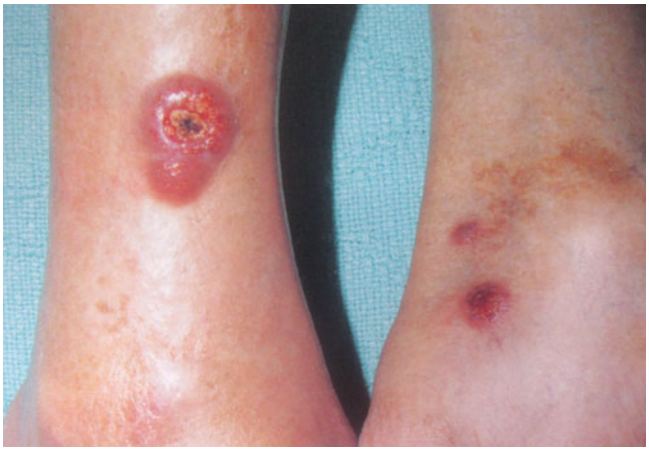


Fig. 47.8 Mycosis fungoides presenting as ulcerated nodules on the legs

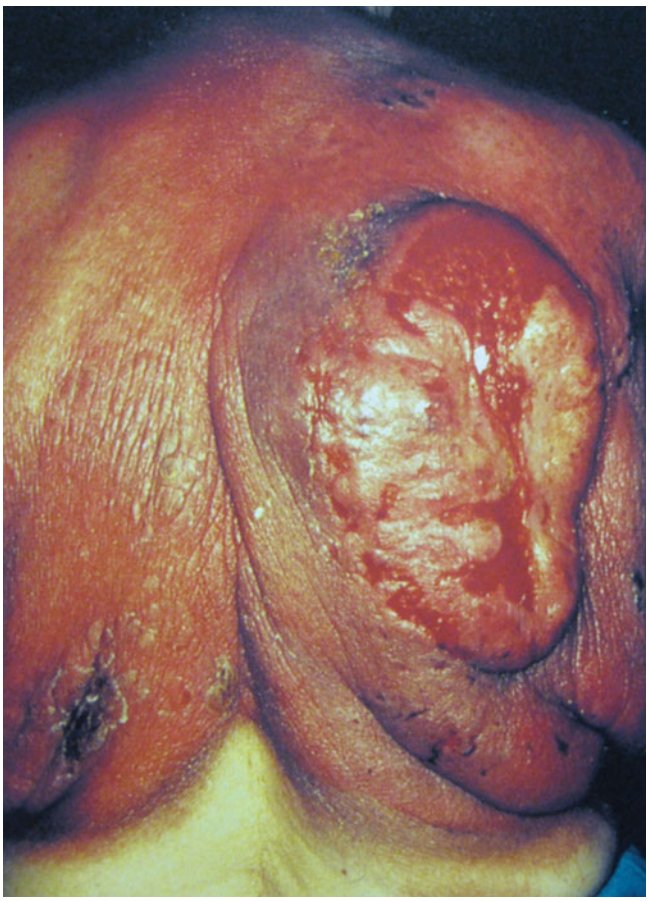


Fig. 47.9 Gigantic fungating tumor of mycosis fungoides. Differential diagnosis includes dermatofibrosarcoma protuberans

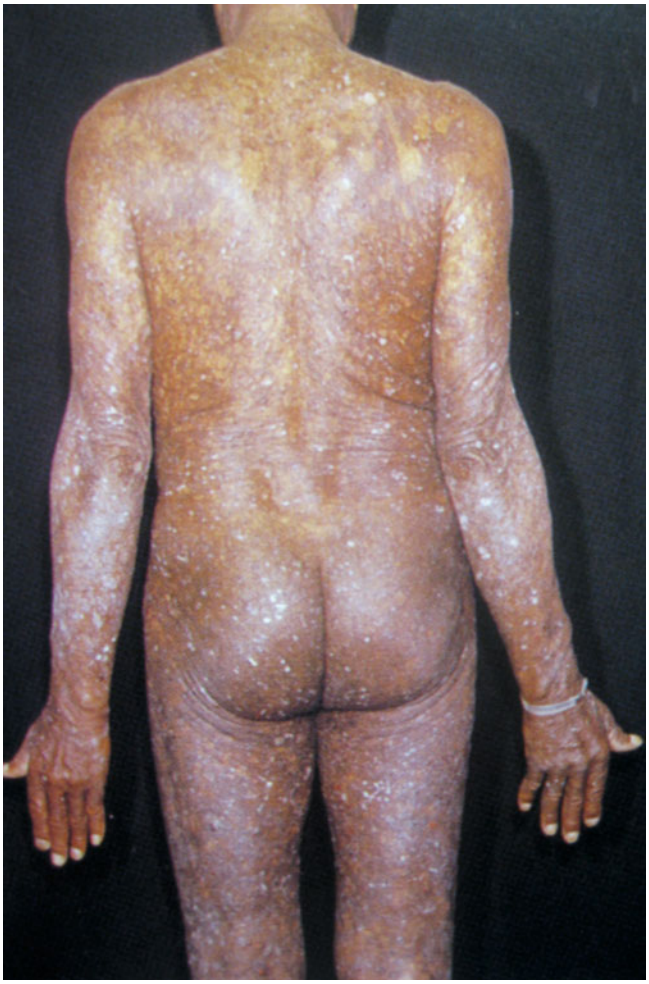


Fig. 47.10 Sezary syndrome is the blood borne erythrodermic form of mycosis *fungoides*

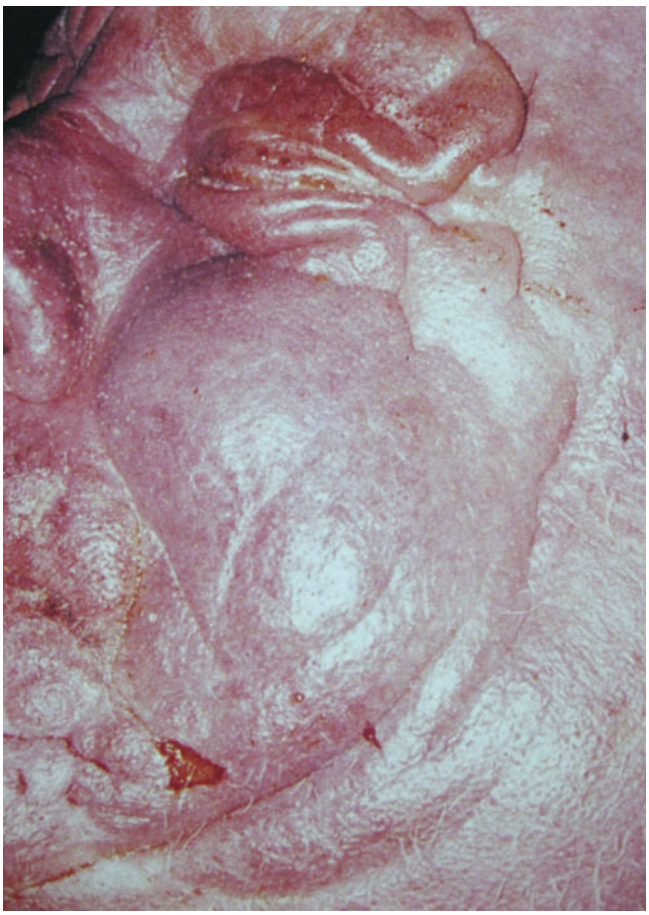
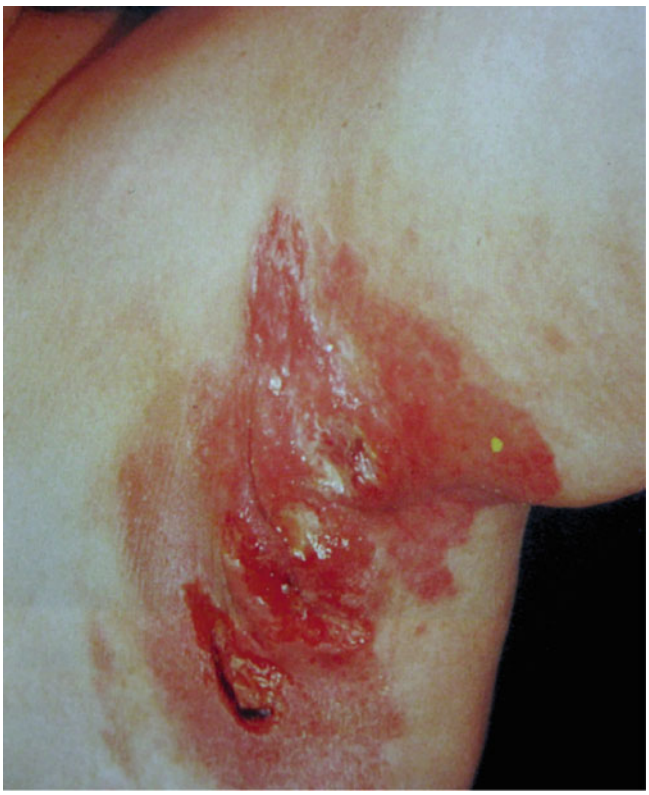
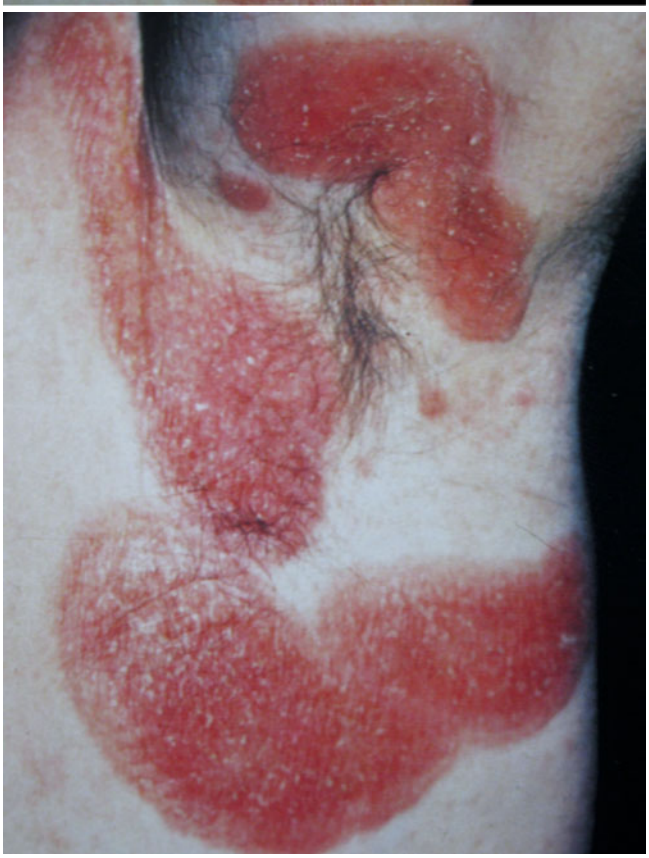
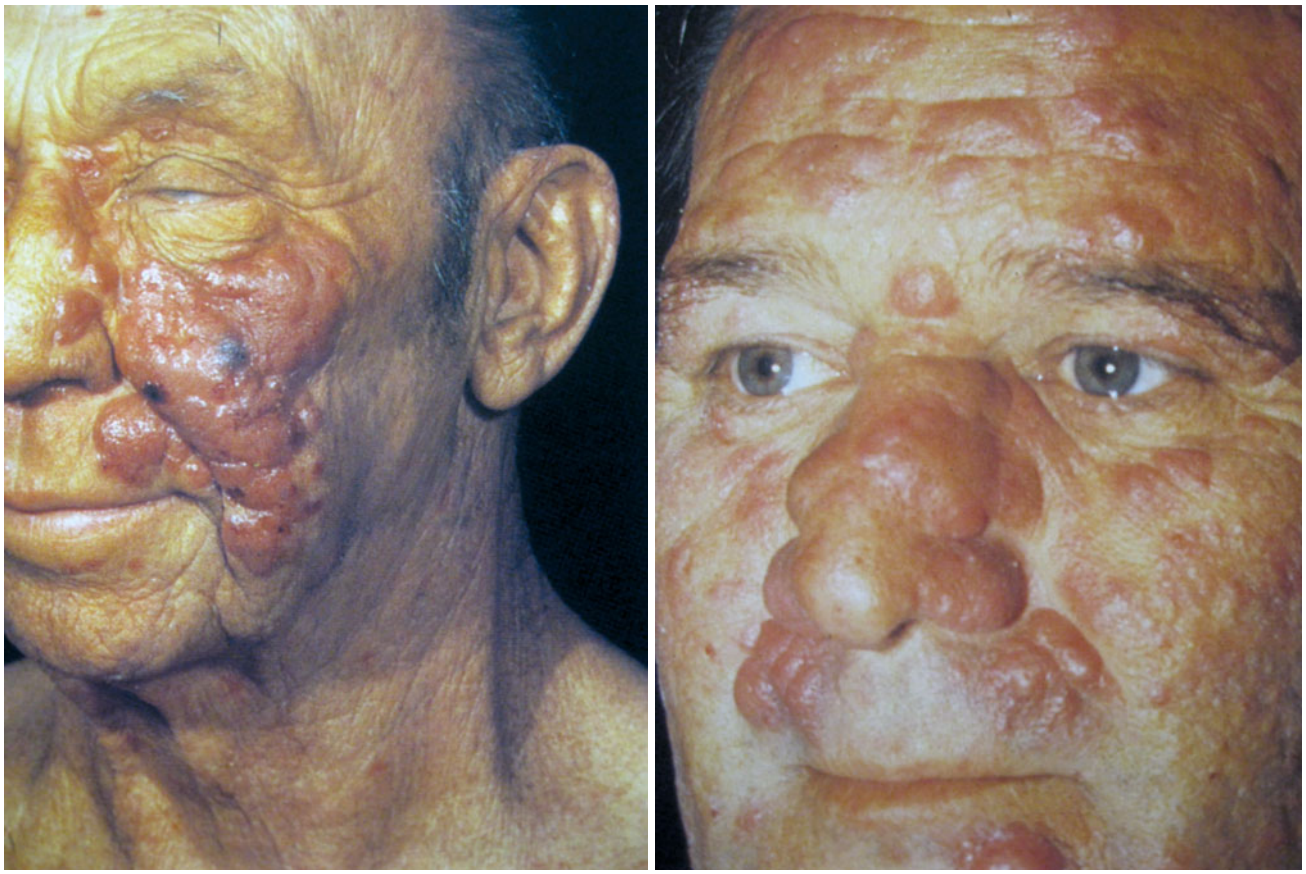


Fig. 47.11 Leonine (lion-like) face results from diffuse cutaneous infiltration by lymphoma in a patient with Sezary syndrome (Courtesy of Dr. R. Kanas)





Figs. 47.12 and 47.13 Mycosis fungoides of the axilla



Figs. 47.14 and 47.15 Mycosis fungoides of the face

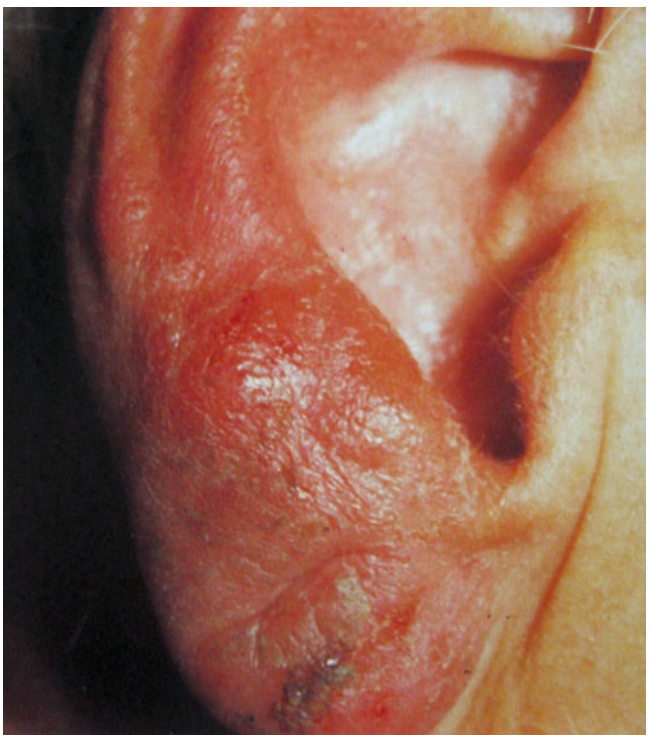


Fig. 47.16 Infiltration of the earlobe by mycosis fungoides is fairly common

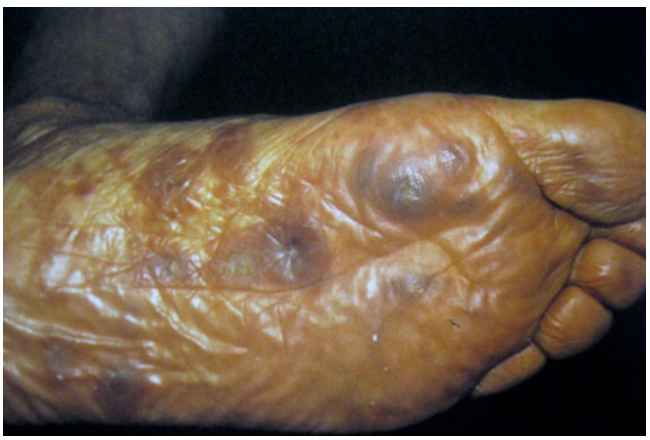


Fig. 47.17 Mycosis fungoides of the sole

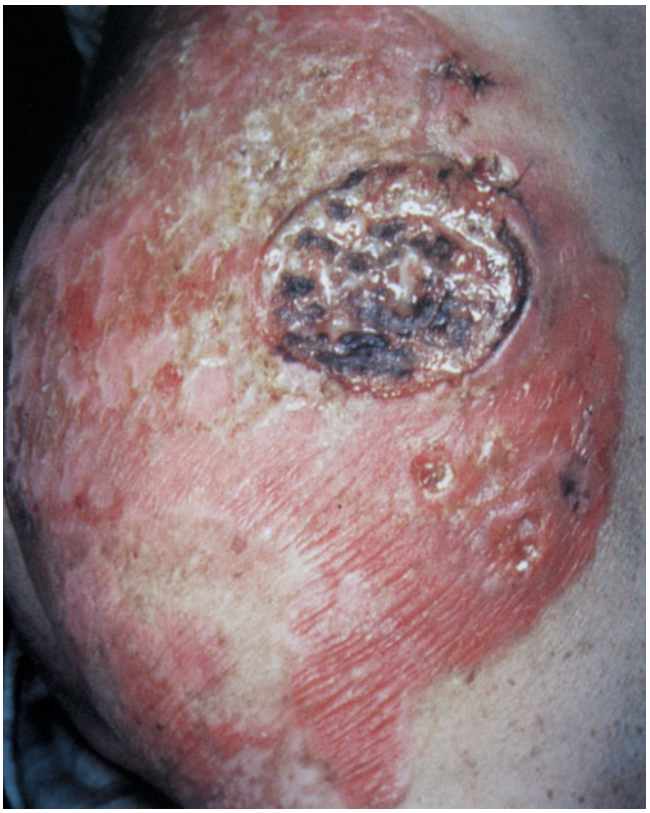


Fig. 47.18 D'ernblee type of mycosis fungoides-sudden appearance of tumors

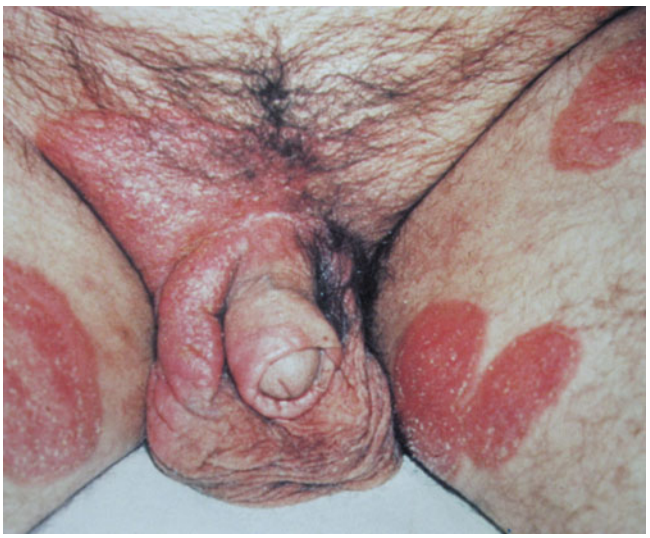


Fig. 47.19 Mycosis fungoides of the groin was mistakenly treated for years as tinea cruris (ringworm)

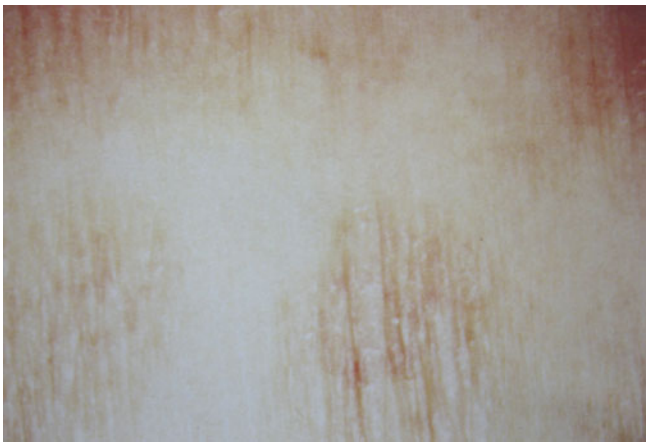


Fig. 47.20 Poikiloderma atrophicans vasculare. Atrophic, erythematous patches resemble those of eczema, tinea, and psoriasis. This form of parapsoriasis occasionally progresses to mycosis fungoides

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Melanoma

48

Subtypes

Subtypes of melanoma include the following:

1. Lentigo maligna-premalignant Hutchinson's freckle, or intraepidermal melanoma-in-situ
2. Lentigo maligna melanoma (LMM)-most common in elderly
3. Superficial spreading malignant melanoma (SSMM)-most common melanoma in all age groups, even the elderly; has a radial growth phase
4. Nodular malignant melanoma (NMM)-has a rapid vertical growth phase
5. Acral-lentiginous malignant melanoma (ALMM)-occurs on palms and soles; least common type of melanoma overall, but most common type in blacks

Clinical Description

Pigmented macules, papules, nodules, patches, or tumors with any of the ABCD warning signs of melanoma may be noted: asymmetry, border irregularity, color variability, diameter over 6 mm. Note that amelanotic melanomas can also occur [1, 2].

Etiology and Pathophysiology

The malignant degeneration of melanocytic lesions is influenced by genetic predisposition, sun exposure, hormonal factors, possibly other environmental factors [3–6]. A flow chart of risk factors for cutaneous malignant melanoma appears in Table 48.1

Histopathology

All types of melanoma show dermal invasion of atypical melanocytes. Superficial spreading malignant melanoma often shows pagetoid epidermal involvement. Lentigo maligna melanoma shows atrophic epidermis, atypical spindle-shaped melanocytes irregularly arranged along the dermoepidermal junction and invading the dermis, prominent solar elastosis, and variable degrees of pigmentation.

Differential Diagnosis

The differential diagnosis includes seborrheic keratosis, nevomelanocytic nevi, and lentigo maligna (melanoma-in-situ).

Therapy

Treatment involves excision with "adequate" (controversial-1-to- 3 -cm) borders for marginal clearance. Chemotherapy, immunotherapy (monoclonal antibodies), and vaccine trials for metastatic melanoma are being actively investigated [8–10].

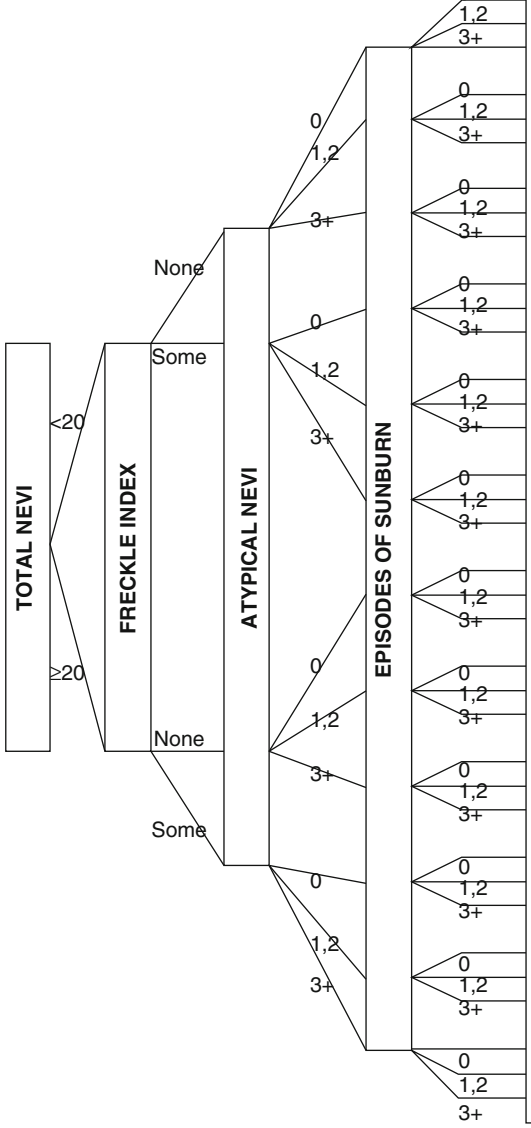
Prognosis

The prognosis depends primarily on the lesion depth, as denoted by the Breslow tumor thickness in mm and by Clark's level of skin invasion.

Other unfavorable clinical prognostic factors include the following: male sex, advancing age, larger lesion diameter, ulceration. A previous history of melanoma is the single greatest risk factor for the development of another melanoma. Other unfavorable histologic prognostic factors include the following: tumor type (rapid vertical growth phase [nodular] is worse than radial growth phase [superficial spreading]), high mitotic index (increased number of mitoses), vascular or lymphatic invasion, and low degree of host inflammatory reaction.

R.A. Norman, E.M. Young, Jr, *Atlas of Geriatric Dermatology*,
DOI 10.1007/978-1-4471-4579-0_48, © Springer-Verlag London 2014

Table 48.1 Flowchart of risk factors for cutaneous malignant melanoma



	MEN			WOMEN		
	Relative Risk (95% CI)		Risk Group	Relative Risk (95% CI)		Risk Group
0	1.0	(1.0)	1	1.0	(1.0)	1
1,2	2.5	(1.4, 4.6)	1	1.5	(0.96, 2.2)	1
3+	6.3	(1.9, 2 1.0)	1	2.1	(0.91, 5.0)	1
0	1.6	(0.6,3.9)	1	2.1	(1.2, 3.6)	1
1,2	3.9	(1.4,11.4)	1	3.1	(1.6, 6. 1)	1
3+	9.9	(2.3, 43.0)	2	4.5	(1.6, 12.0)	1
0	2.5	(0.4, 15.0)	1	6.6	(1.5, 13.0)	1
1,2	6.2	(1 .0, 40 .0)	1	6.5	(2 .0, 21.0)	2
3+	15. 6	(1.9, 130.0)	1	9.5	(2.4, 38 .0)	2
0	3.7	(1.6, 9.0)	1	3.1	(1.7, 5.6)	1
1,2	9.7	(3.2, 28.0)	2	4.5	(2.3, 9.1)	2
3+	23 .6	(5.1, 110.0)	3	4.6	(2.5, 18.0)	2
0	5.9	(1.6, 22.0)	1	6.5	(2.9, 15.0)	2
1,2	14.8	(3.4, 63.0)	2	9.6	(4.0, 23.0)	2
3+	37 .1	(6.2, 223.0)	3	14.0	(4.5, 43.0)	2
0	9.2	(1.1. 75 .0)	1	13.8	(4.0, 47.0)	2
1,2	23 .2	(2.7, 202 .0)	2	20.2	(5.6, 73 .0)	3
3+	58.2	(5.3, 637.0)	3	29.6	(6.8, 128.0)	3
0	10.1	(2.7, 38.0)	2	5.9	(2.7, 13.0)	2
1,2	25.4	(5.2, 123 .0)	3	8.6	(3.6, 21.0)	2
3+	63.7	(8.7, 469 .0)	3	12.5	(4.0, 39.0)	2
0	15.8	(3.6, 71.0)	2	12.3	(5.0, 30.0)	3
1,2	39.8	(7.2, 219.0)	3	18.1	(6.7, 49.0)	3
3+	100.0	(12.6, 797.0)	4	26.4	(7.7, 90.0)	3
0	24. 8	(3.1, 200.0)	2	26.0	(7.4, 92.0)	3
1,2	62.5	(6.8, 578.0)	3	38.1	(10.1, 144.0)	4
3+	157.0	(12.8, 1925)	4	55.8	(12.0, 255 .0)	4
0	37.7	(6.3, 225.0)	3	18.2	(16.1, 54.0)	4
1,2	94.8	(12.9, 696.0)	3	26.6	(18.5, 83.0)	4
3+	238.2	(22.8, 2488.0)	4	38.9	(10.2, 148.0)	4
0	59.2	(8.3, 420)	3	38.3	(11.7, 125.0)	4
1,2	148 .8	(17.6, 1255.0)	4	56.1	(16.0, 193.0)	4
3+	374.0	(32.0, 4336)	4	82.1	(0.9 1, 5.0)	4
0	92.9	(17.8, 1106.0)	3	80.8	(18.0, 356.0)	4
1,2	233.5	(17.3 , 3151.0)	4	118.2	(26.0, 543.0)	4
3+	587.0	(33.7, 10200.0)	4	173.1	(32.0, 925.0)	4

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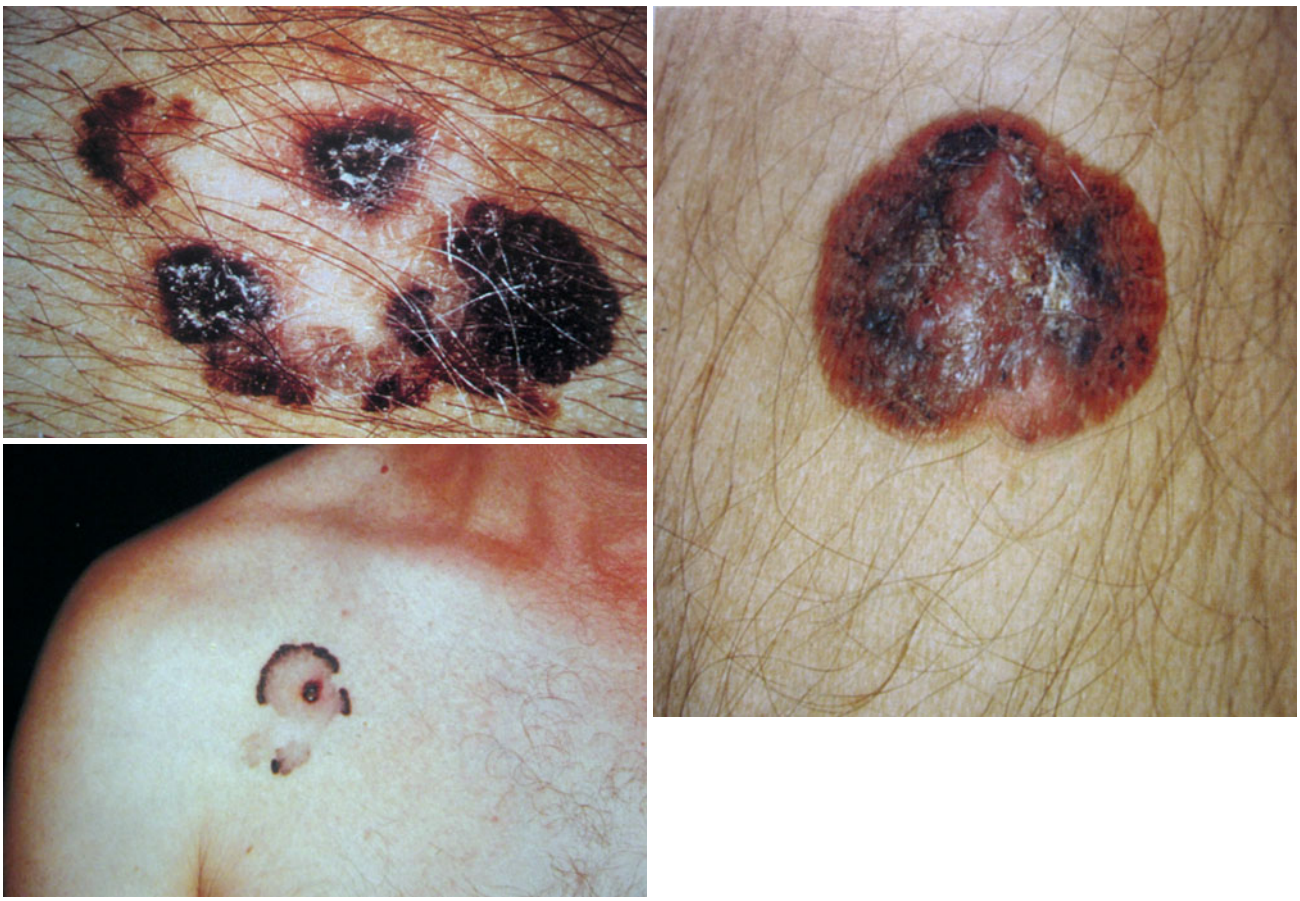
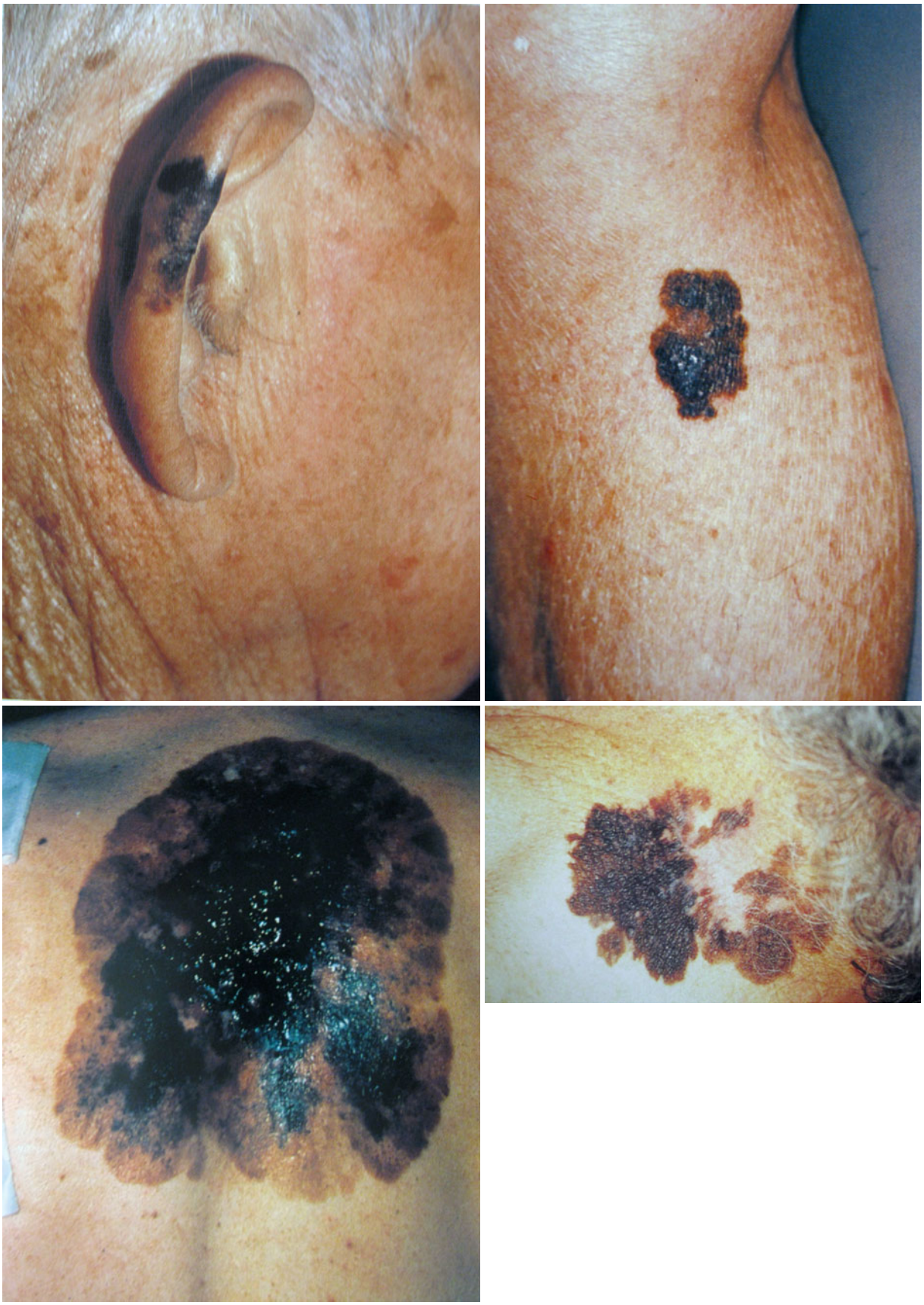
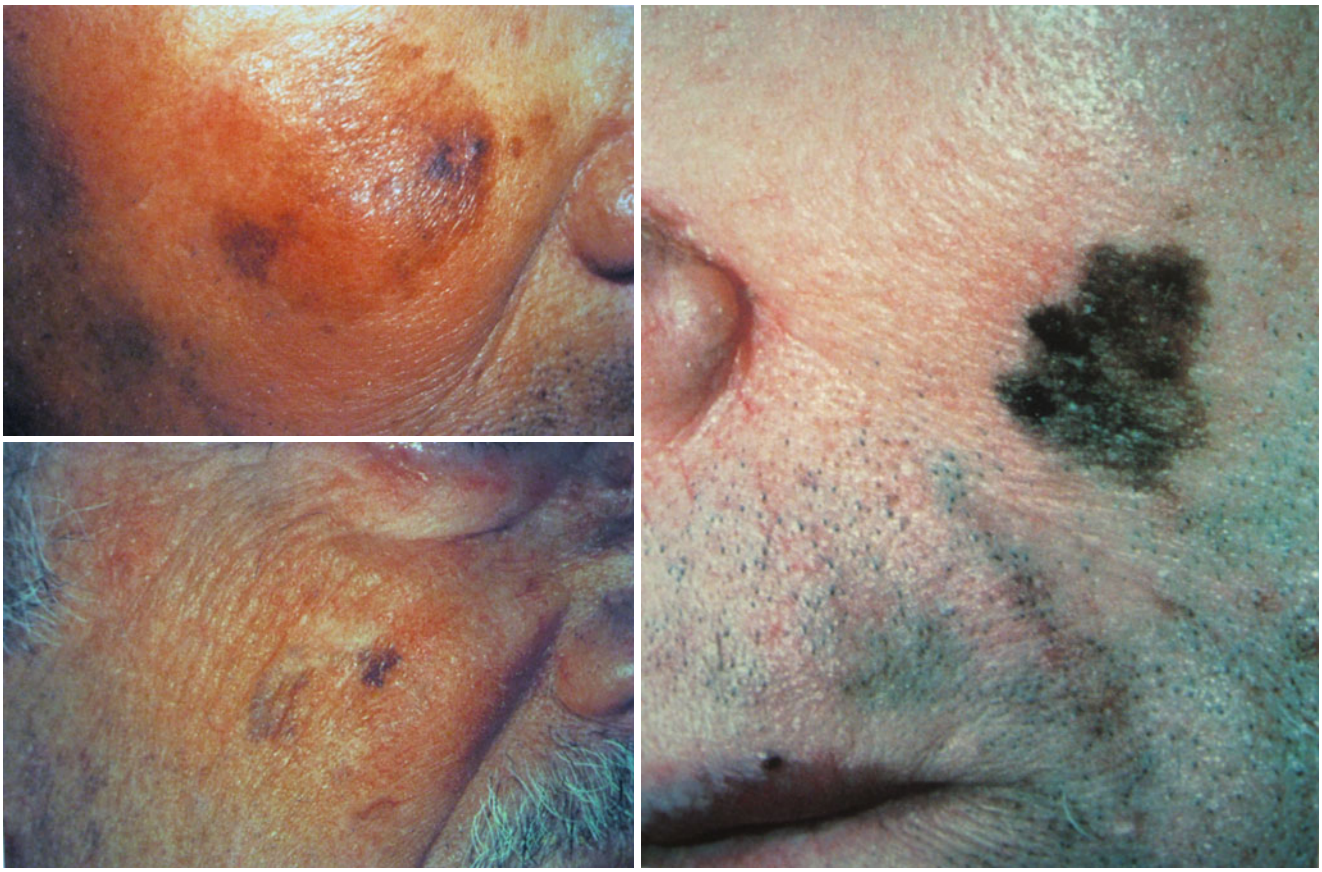
Risk groups: *1* marginally increased risk, *2* increased risk, *3* very increased risk, *4* worryingly high risk. Relative risk coefficients used (for men/women respectively): 10.1/5.9 for total nevi; 3.7/3.1 for freckles; 1.6/2.1 for atypical nevi; and 2.5/1.5 for episodes of sunburn



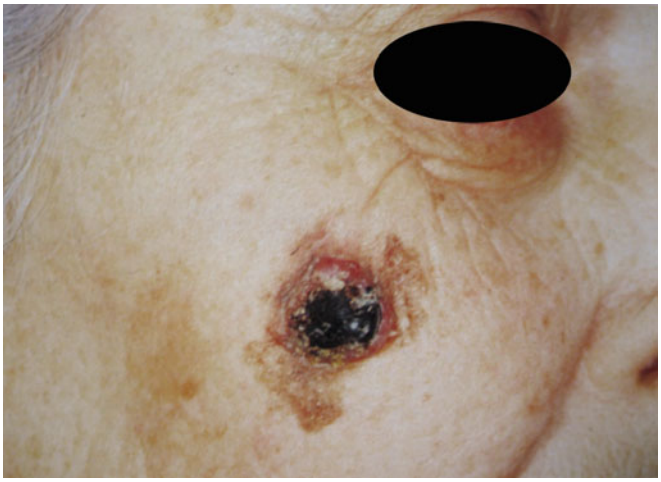
Figs. 48.1, 48.2, 48.3, 48.4, 48.5, 48.6, and 48.7 Superficial spreading *malignant* melanoma is the *most* common type of melanoma in *all* age groups. It can assume giant size (Fig. 48.6). Suspicious pigmented lesions display one or more of the ABCD warning signs: asymmetry, border irregularity, color variation, diameter greater than 6 mm

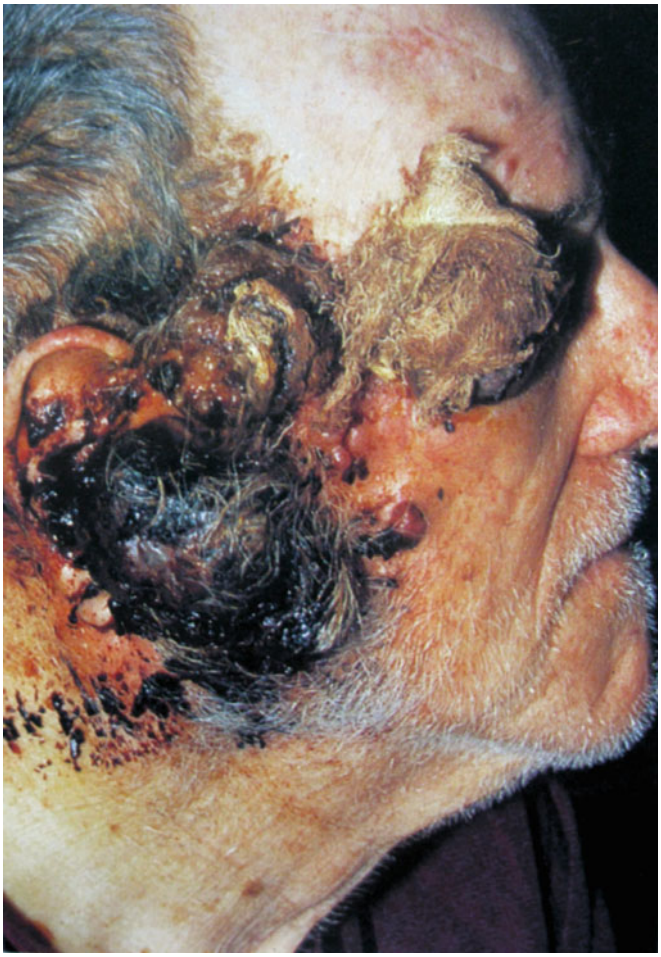


Figs. 48.1, 48.2, 48.3, 48.4, 48.5, 48.6, and 48.7 (continued)

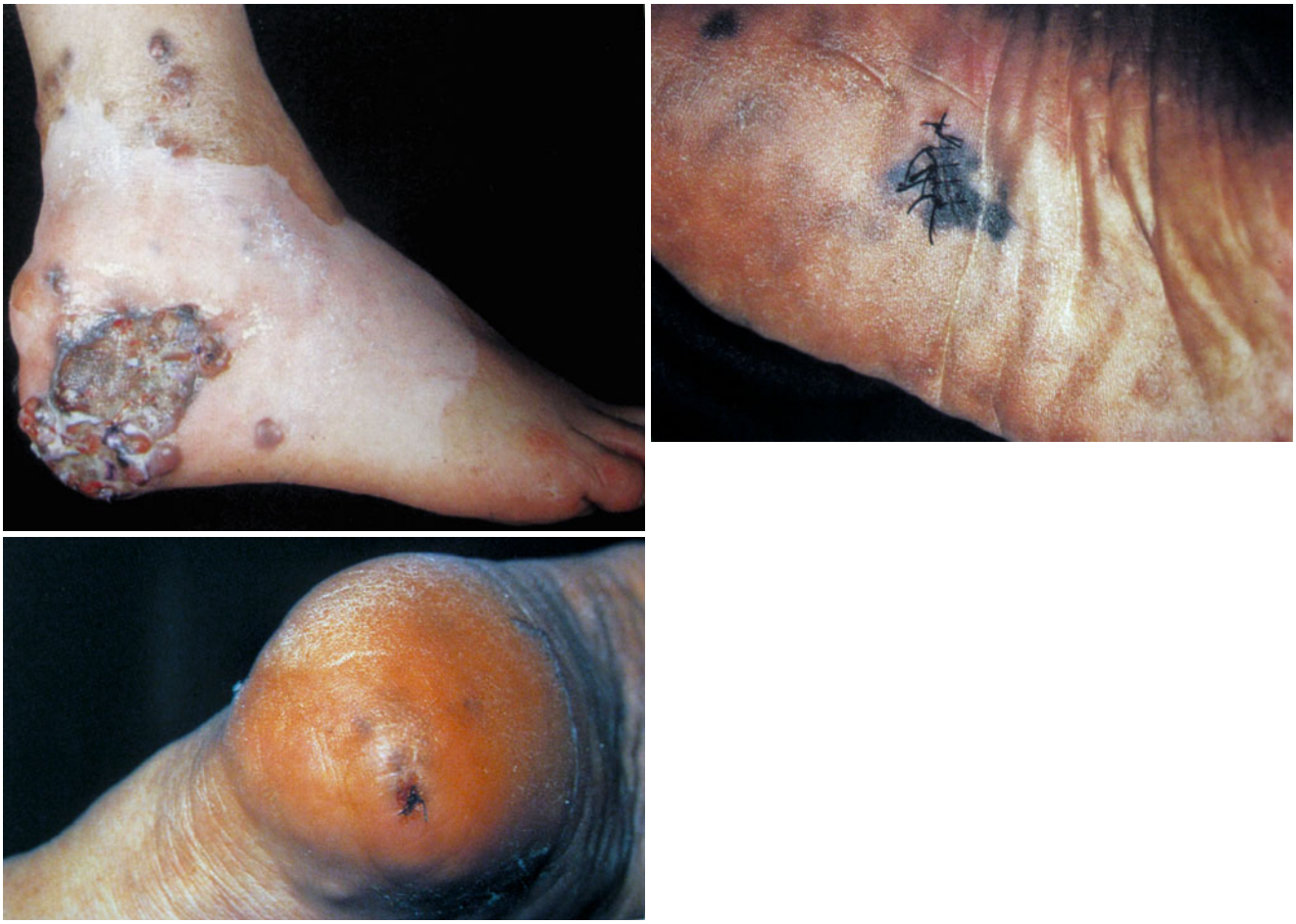


Figs. 48.8, 48.9, and 48.10 Lentigo maligna melanoma evolves from a precursor lesion called lentigo maligna, or Hutchinson's melanotic freckle (See Chap. 45). Lentigo maligna melanoma (Fig. 48.10) is seen almost exclusively in elderly patients and has the *slowest* rate of progression (Fig. 48.10, Courtesy of Dr. Swinyer)





Figs. 48.11 and 48.12 In contrast, nodular melanoma is a rapidly progressive, fatal type. These lesions on the cheek developed at the site of a lentigo maligna that soon developed an *ulcerated* nodule. Metastasis *is* rapid



Figs. 48.13, 48.14, and 48.15 Acral-lentiginous melanoma is the most rapidly fatal type. Note the in-transit satellite metastases ascending the leg (Fig. 48.13)

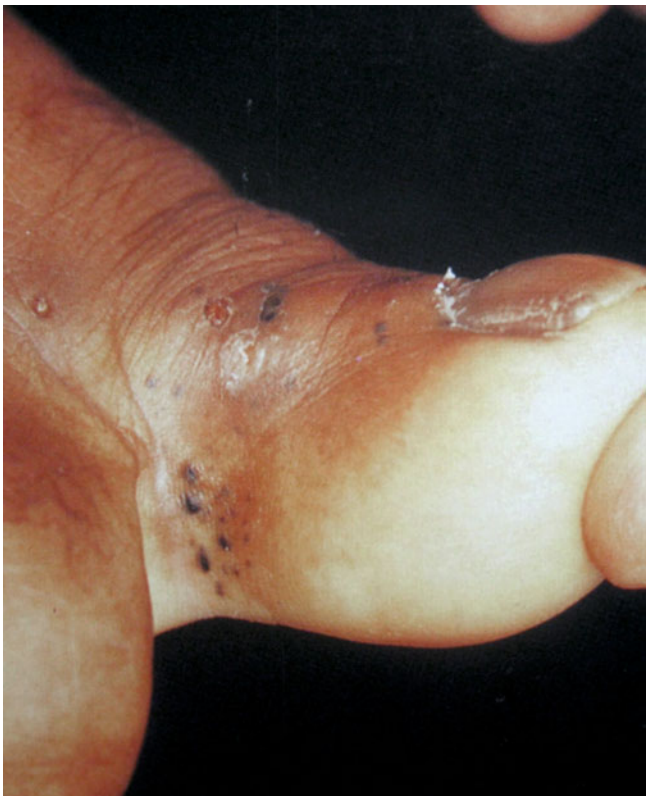
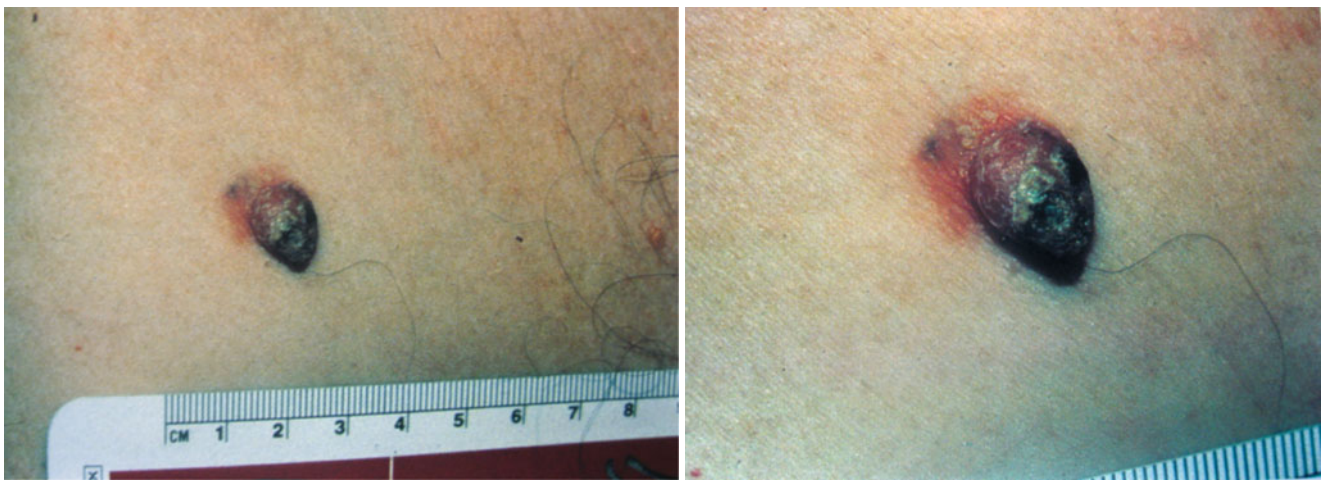


Fig. 48.16 Acral-lentiginous melanoma lurks *in* the toe web. The entire cutaneous surface must be examined for suspicious pigmented *lesions*



Figs. 48.17 and 48.18 *Amelanotic* melanoma clinically resembles a pyogenic granuloma (capillary hemangioma). Biopsy is imperative

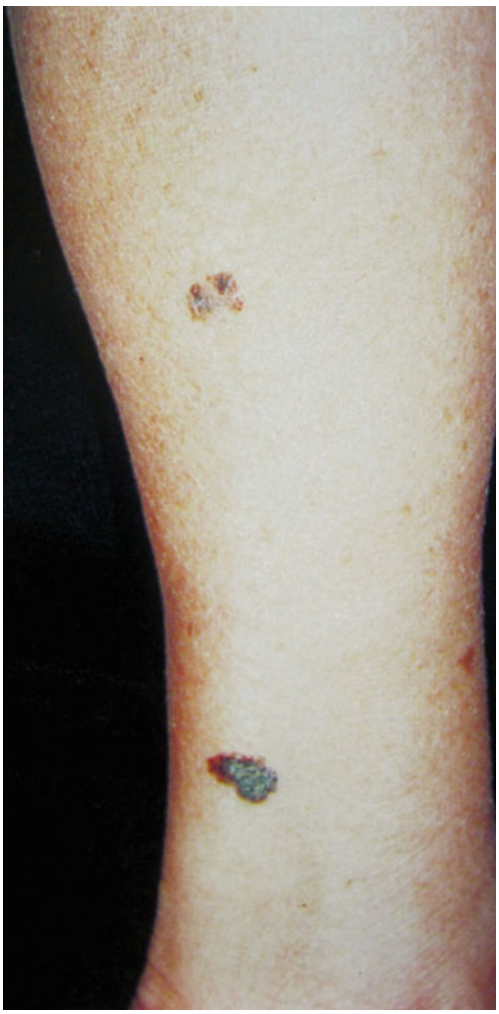
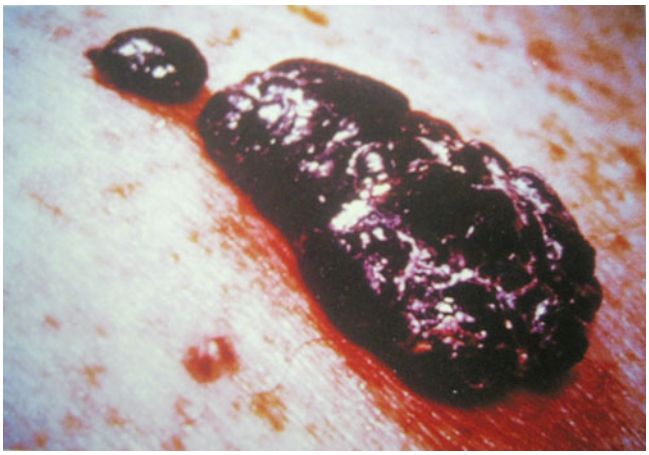
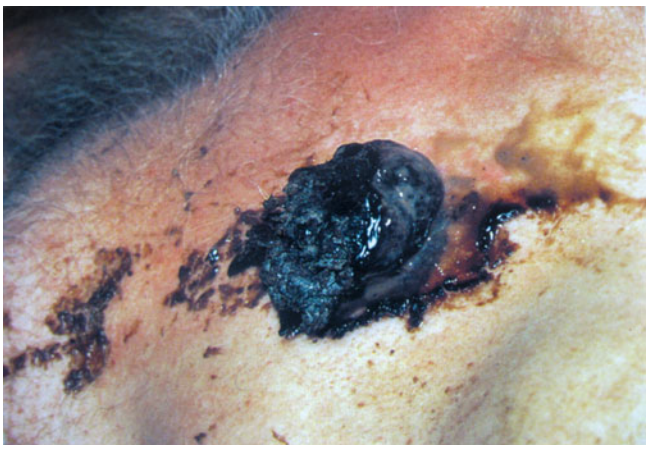


Fig. 48.19 Multiple melanomas. The greatest risk factor for developing melanoma is a history of a previous melanoma





Figs. 48.20 and 48.21 Polypoid nodular melanoma resembles a lump of coal

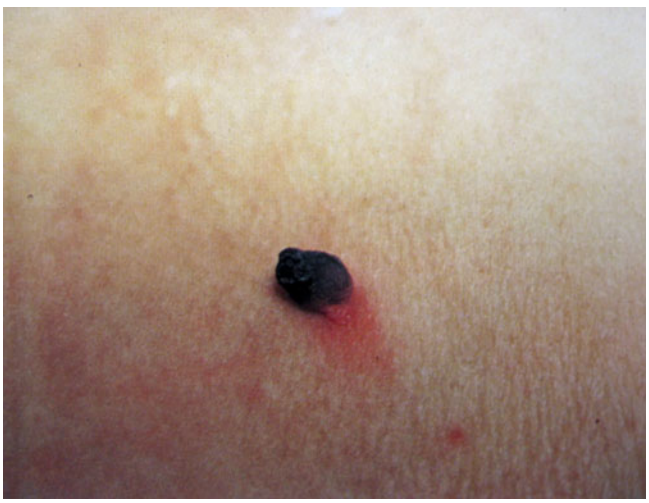
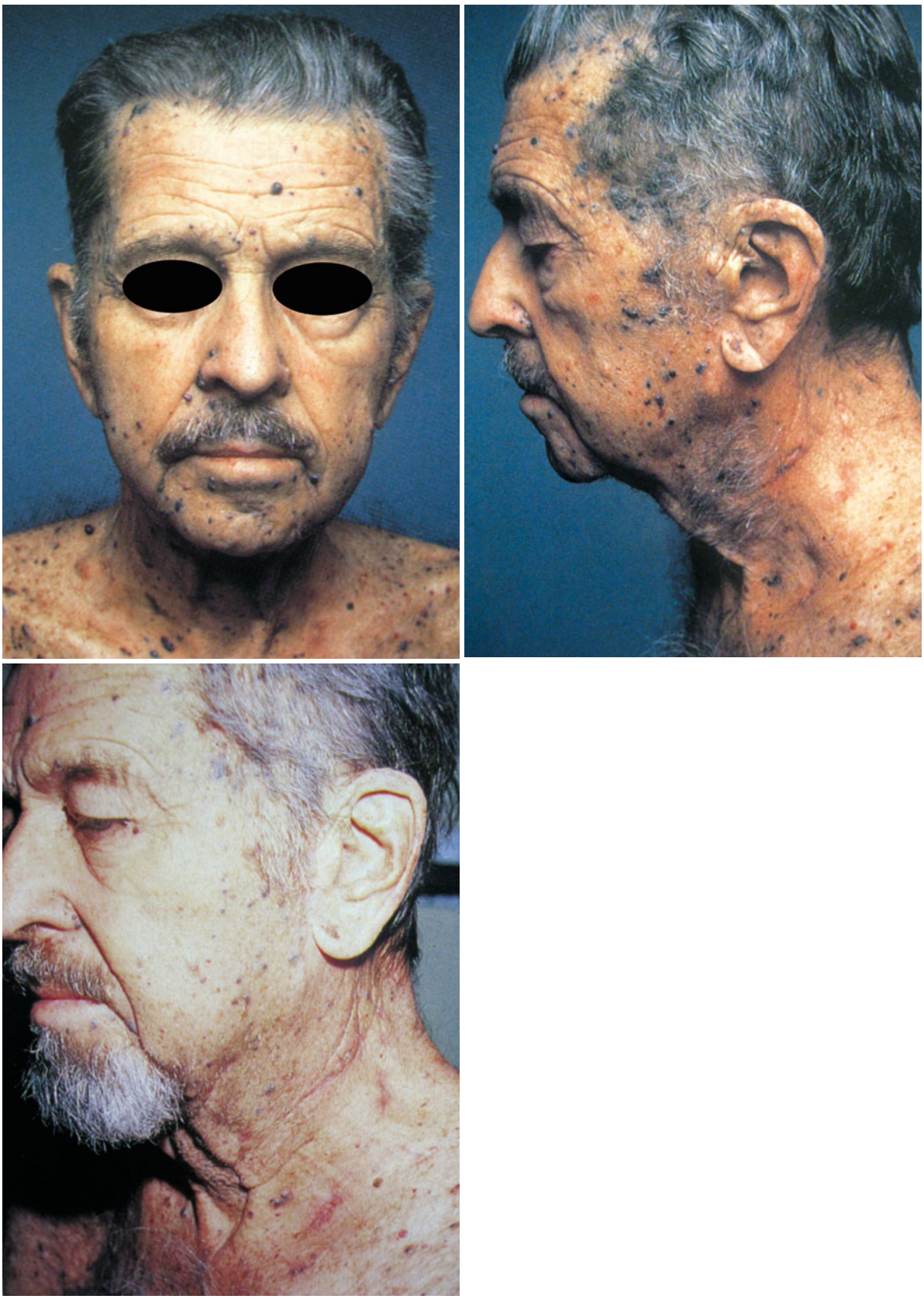
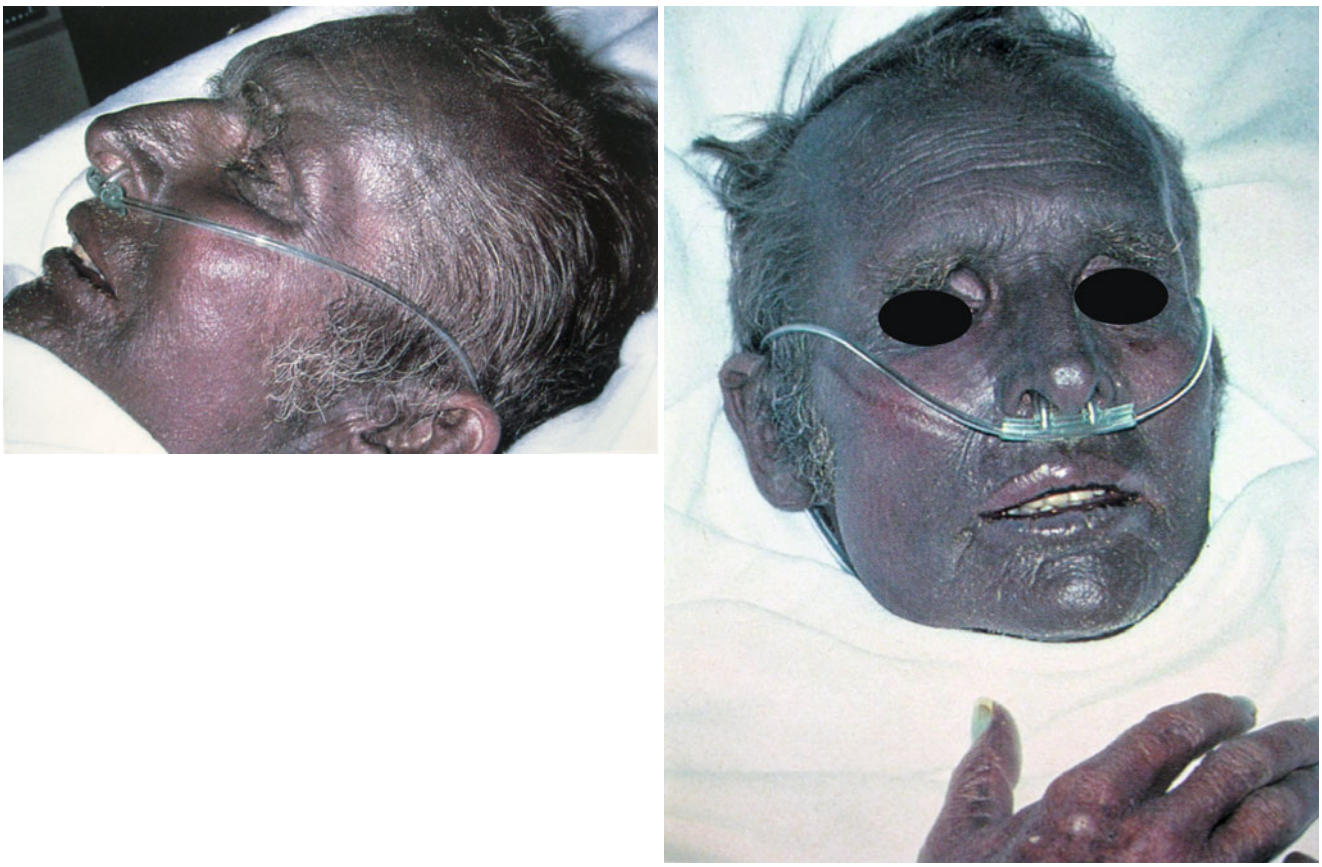


Fig. 48.22 *This* thrombosed acrochordon (skin tag) clinically simulates a nodular melanoma



Figs. 48.23, 48.24, and 48.25 Metastatic malignant melanoma. The blueberry muffin-like appearance is a result of discrete, disseminated nodules of melanoma. Experimental treatment consisted of chemotherapy-tagged anti melanoma antibodies, cloned from this patient's own melanoma *cells*



Figs. 48.26 and 48.27 *Diffuse* blue-gray melanosis involving the lips, ocular sclerae, and hair in a Caucasian with metastatic melanoma. This is an unusual clinical presentation of metastatic melanoma (The patient was previously blond-haired and blue-eyed) (Courtesy of Dr. S. R. Weiss)

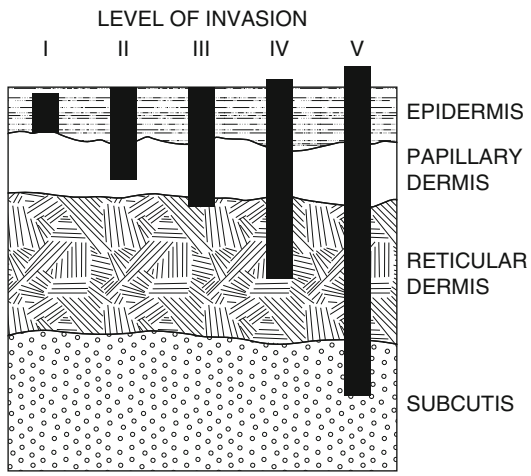


Fig. 48.28 Schematic of Clark's level of invasion

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Nevi

49

Synonyms

Synonyms include moles, melanocytic nevi, and nevocellular nevi.

Clinical Description

Nevi appear as soft, flesh-colored papules, brown macules, or brown, dome-shaped papules on any cutaneous surface, especially the trunk [1].

Etiology and Pathophysiology

"We are born without moles and die without moles." Natural evolution is toward the spontaneous involution of nevi with advancing age. Benign melanocytic tumors appear in childhood as pigmented macules (junctional nevi), progress in adolescence to pigmented papules (compound nevi), and eventually evolve into flesh-colored papules (dermal nevi) before undergoing spontaneous involution. Dysplastic nevus syndrome describes patients who continue to develop irregular nevi throughout adulthood [2–9]. These patients may have a personal and/or family history of melanoma, and are at an increased lifetime risk for developing melanoma.

Histopathology

Nevi show nests or clusters of benign nevus cells (specialized melanocytes) at the dermoepidermal junction (junctional nevus), at both the dermoepidermal junction and intradermally (compound nevus), or only intradermally (dermal nevus) [10].

Differential Diagnosis

Clinically, some cases of fibrous papule of the nose may represent involuting dermal nevi (see Chap. 34). Basal cell carcinoma, trichoepithelioma, and seborrheic keratoses can be confused with nevi [11]. (See section "Differential Diagnosis" in Chap. 34.)

Therapy

1. Biopsy is essential for clinically atypical, cosmetically disturbing, or otherwise bothersome nevi. The ABCD warning signs of melanoma are asymmetry, border irregularity, color variation, and diameter over 6 mm.
2. Sun protection and avoidance are helpful.

Prognosis

The incidence of transformation of acquired nevi into malignant melanoma is less than 1 %. When transformation does occur, however, prognosis is related to the depth of the melanoma (see Chap. 48). Patients with multiple atypical nevi (dysplastic nevus syndrome) must be examined often to prevent and detect the development of melanoma.

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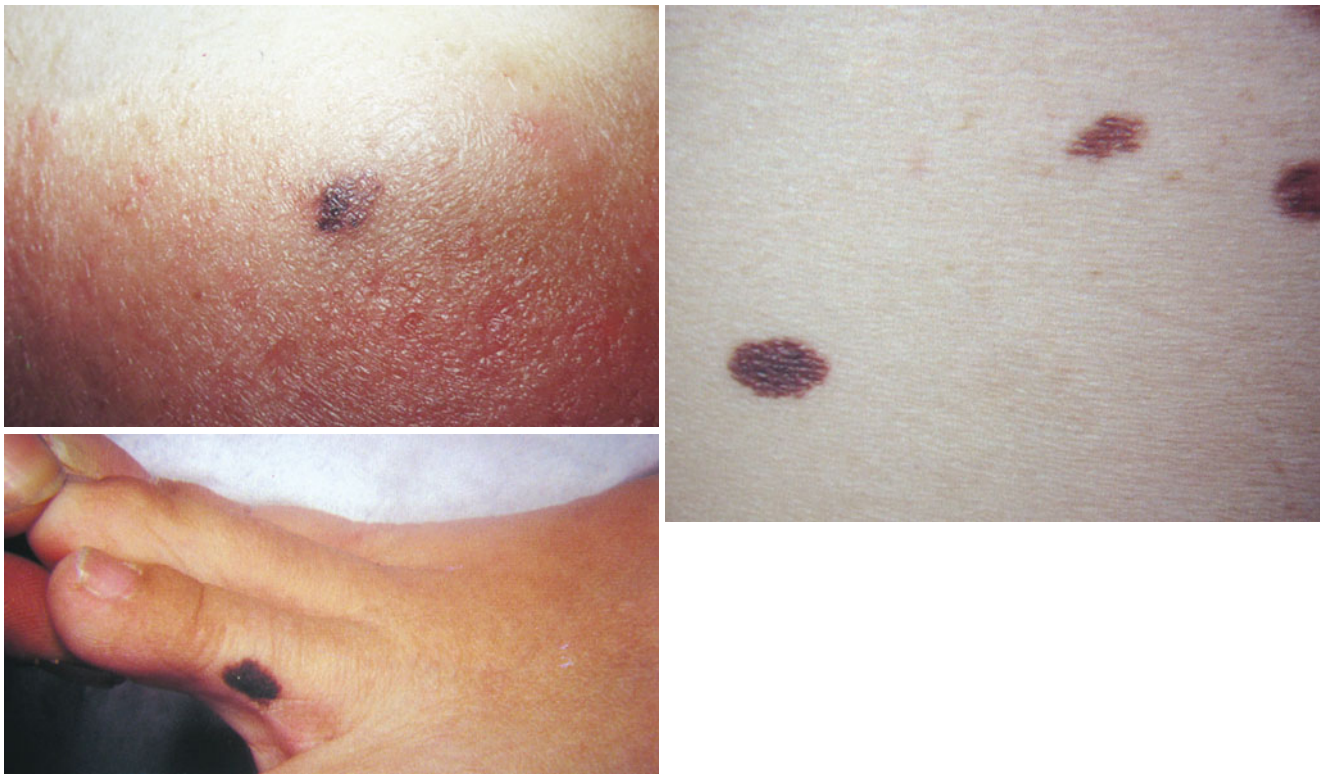


Figs. 49.1, 49.2, and 49.3 Junctional nevi: macular pigmentation on back (Fig. 49.1). Cheek (Fig. 49.2), and interdigital web space (Fig. 49.3)

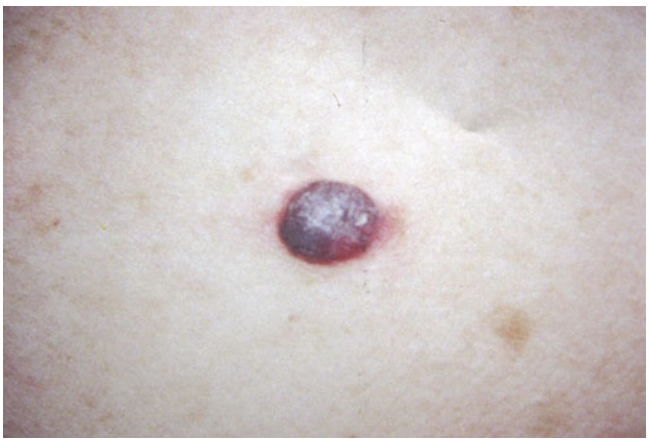


Fig. 49.4 Compound nevus. Elevated brown-gray papule consists of pigmenting melanocytes at the dermal-epidermal junction and in the dermis

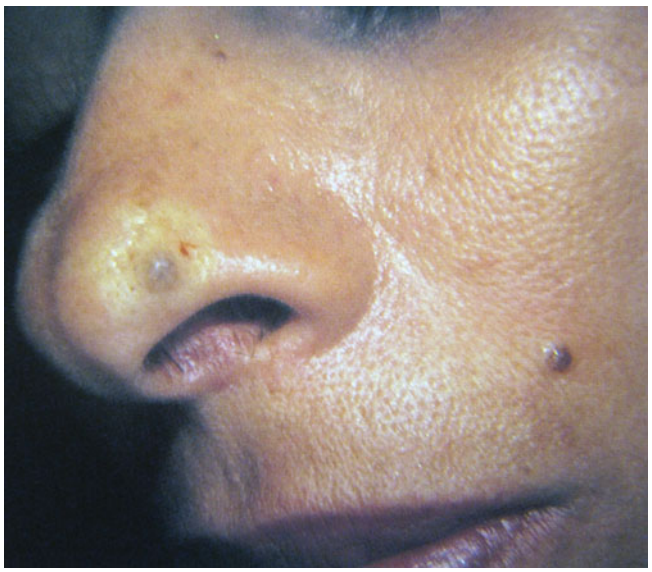


Fig. 49.5 Compound nevi: brown papules on cheek and nose. The nasal lesion has been flattened with shave biopsy and thermal ("hot Wire") cautery. Note the residual gray-brown pigment at the base

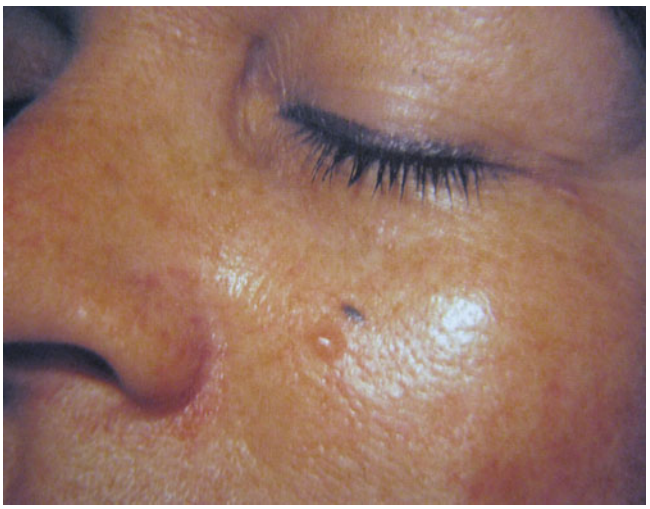


Fig. 49.6 Dermal nevi: flesh-colored papule on cheek. Note the dark junctional nevus superiorly

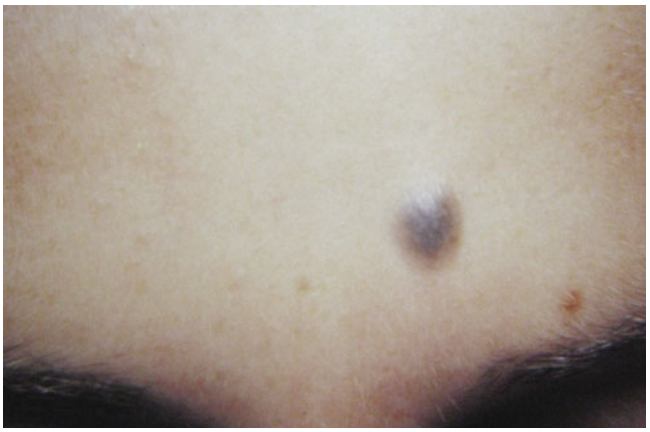


Fig. 49.7 Blue nevus on forehead. Pigmented melanocytes occupy the dermis

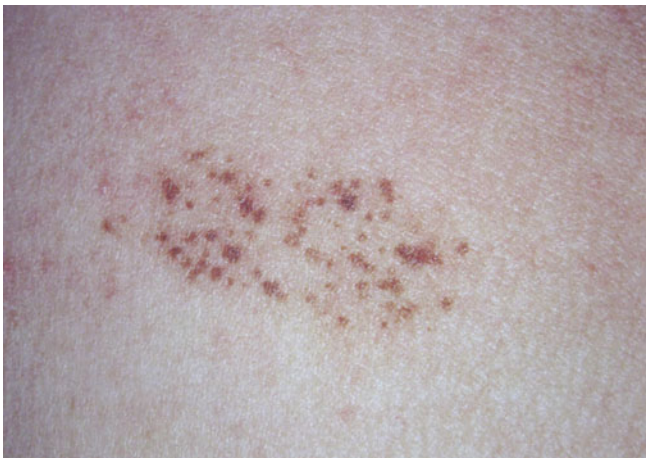
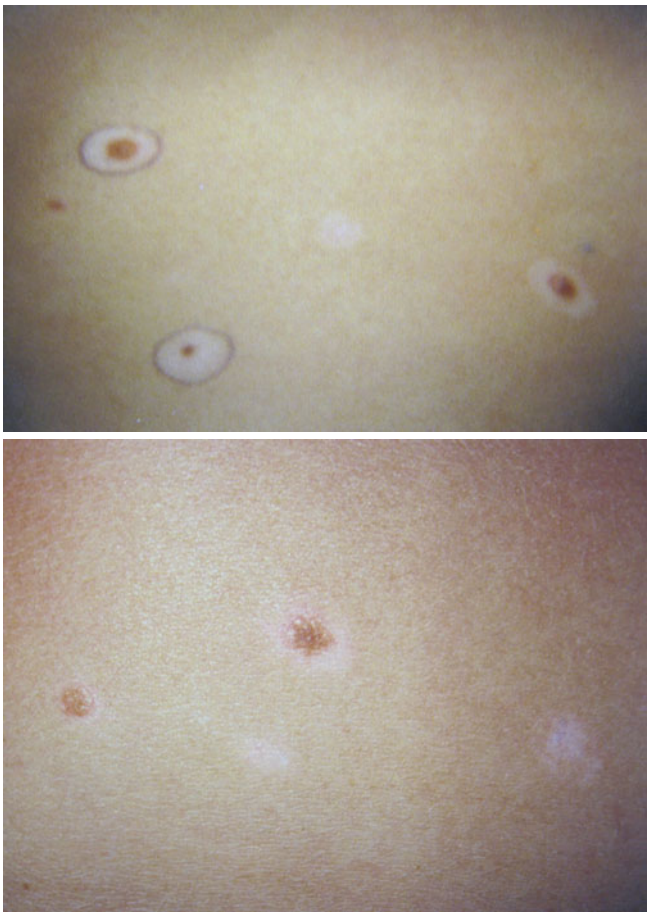


Fig. 49.8 Nevus spilus (speckled lentiginous nevus) consists of a light brown patch (lentigo simplex/peppered with darker macules consisting of junctional nests of nevus cells, dermal nests of nevus cells, and diffuse junctional activity)



Figs. 49.9 and 49.10 Halo nevi. The white halo represents postinftammatory depigmentation. The halo effect may be completely benign or may represent regression of a distant melanoma with subsequent regression of melanocytic nevi

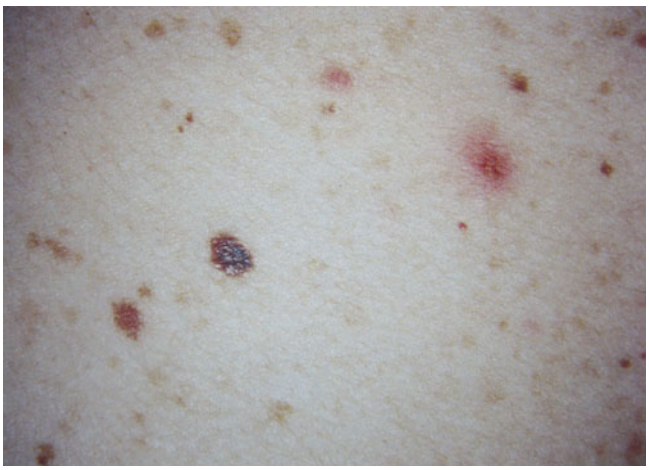


Fig. 49.11 Asymmetry, border irregularity, color variation (*brown*, *black*, *red*, *white*, *blue*), diameter greater than 6 mm (Courtesy of American Academy of Dermatology, 930 N. Meacham Rd., Schaumburg, Il, 60173–4965)

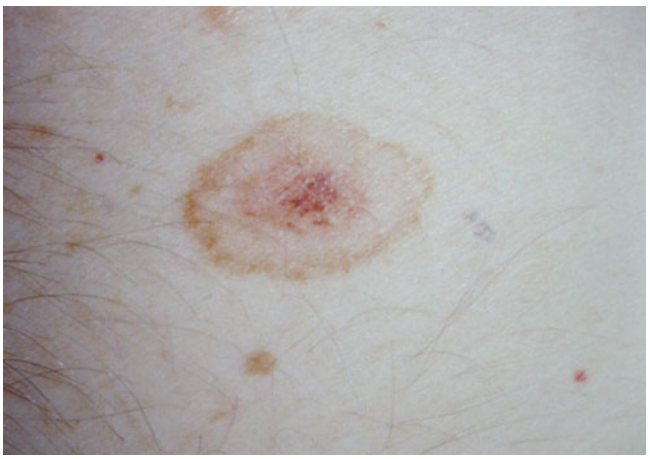


Fig. 49.12 Targetoid or "bull's eye" dysplastic nevus

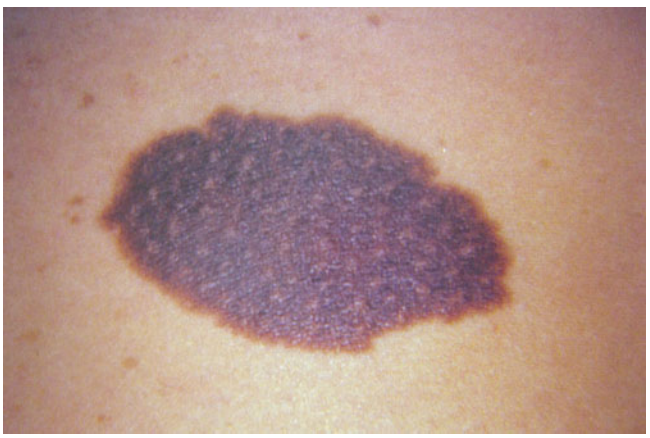


Fig. 49.13 Large congenital melanocytic nevus. Although these nevi have a higher incidence of malignant degeneration before or at puberty, they may occasionally become malignant, even in old age. For this reason, surgical excision prior to puberty is recommended unless such surgery would be disfiguring or would damage adjacent anatomic structures

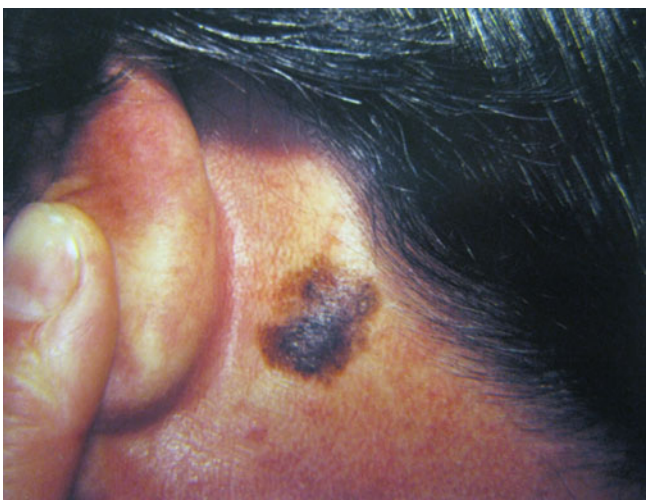


Fig. 49.14 Dysplastic nevus versus melanoma. Excisional biopsy is essential

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Squamous Cell Carcinoma

50

Clinical Description

Squamous cell carcinoma (SCC), also called squamous cell epithelioma (SCE), presents as a red-brown papule, nodule, or tumor, often indurated, with scale or even cutaneous horn. It is often ulcerated. It occurs most commonly on the face, lower lip, or on any sun-exposed area [1–3].

Etiology and Pathophysiology

The major cause of squamous cell carcinoma of the skin is chronic sun or radiation exposure. Squamous cell carcinoma can also develop in chronically draining sinus tracts (Marjolin's ulcer). Chemical carcinogens may be causative factors; Sir Percival Potts described scrotal squamous cell carcinoma resulting from exposure to chimney soot. PUVA (psoralens and UVA light) and immunosuppression (lymphoma, leukemia, AIDS) also play a role. HPV and other viral etiologies have been discovered. Squamous cell carcinomas of the oral mucosa are associated with tobacco and alcohol use [4–9].

Histopathology

Squamous cell carcinoma shows infiltrative lobules of eosinophilic, dyskeratotic, disorganized atypical squamous keratinocytes with mitoses, horn (keratin) pearls, and occasional acantholysis (separation of keratinocytes).

Differential Diagnosis

Basal cell carcinoma usually lacks scale or horn, and is often translucent. Biopsy is mandatory for keratoacanthoma (KA). KA has a crateriform or volcano-like appearance, and more rapid onset and growth. Ulcerated SCC can be mistaken for ulcers of any cause. Recalcitrant "warts" on the hands or feet should be biopsied to exclude verrucous squamous cell carcinoma.

Therapy

Treatment includes the following:

1. Surgery-excision; Mohs surgery (microscopically controlled excision); electrodesiccation, curettage, and cautery (for shallow lesions)
2. Radiation therapy-but contraindicated in those with radiation-induced SCC [10]

Prognosis

SCC on the mucous membranes (lips, genitals, oral mucosa) has a high and early metastatic potential. SCC on sun-exposed skin occasionally metastasizes, and usually does so late in its course [11, 12].

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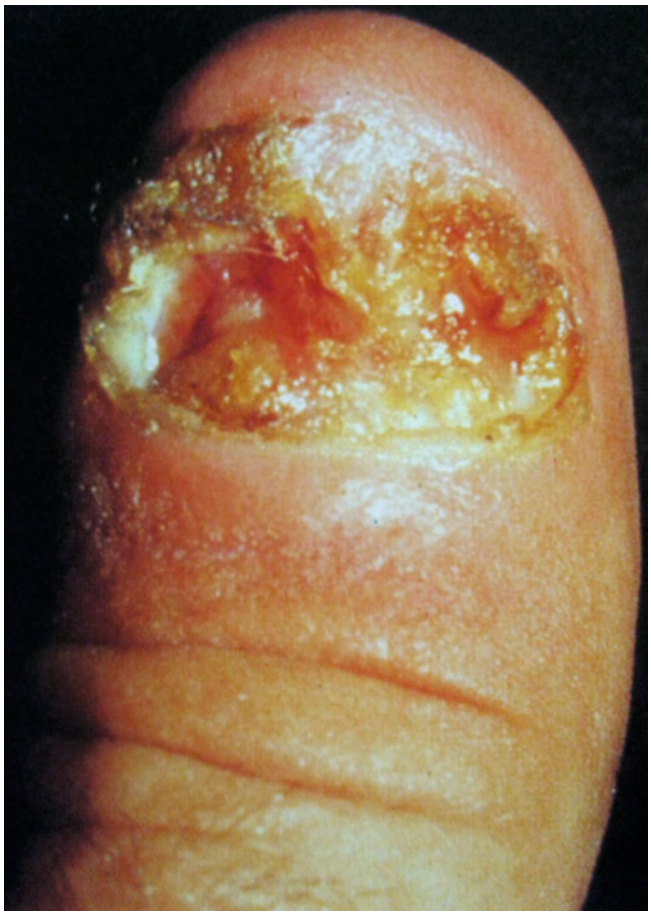


Fig. 50.1 Subungual squamous cell carcinoma has destroyed the nail plate and defied all topical therapies to eradicate a (misdiagnosed) "stubborn wart"

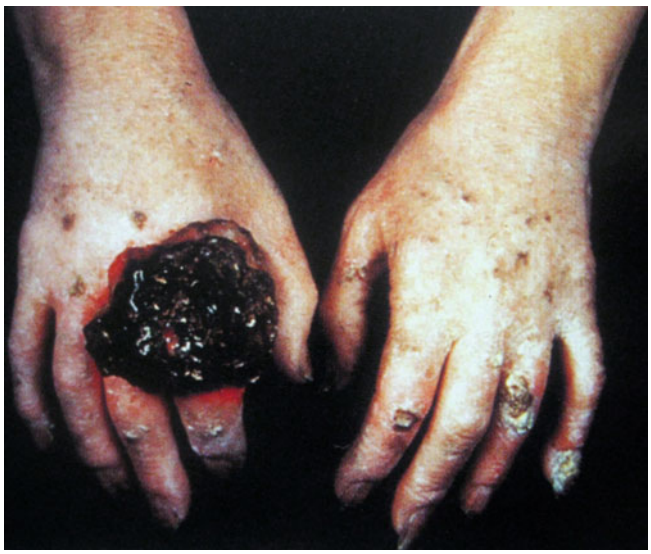


Fig. 50.2 Large, fungating, crusted squamous cell carcinoma and several smaller ones occurred on the hands of a dental technician who failed to practice proper x-ray shielding techniques

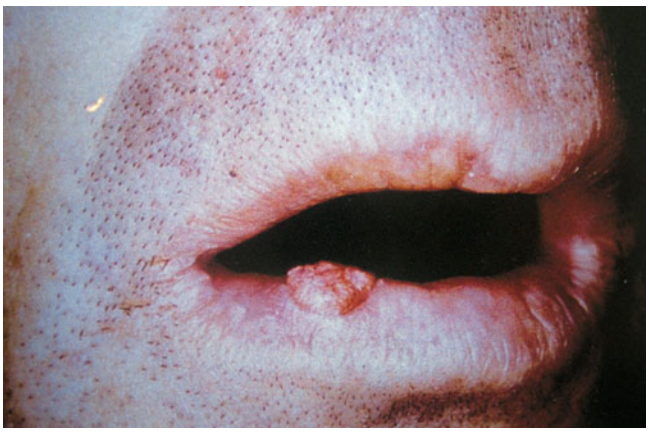


Fig. 50.3 Typical squamous cell carcinoma of the lip. Complete excision is mandatory, because lesions metastasize early

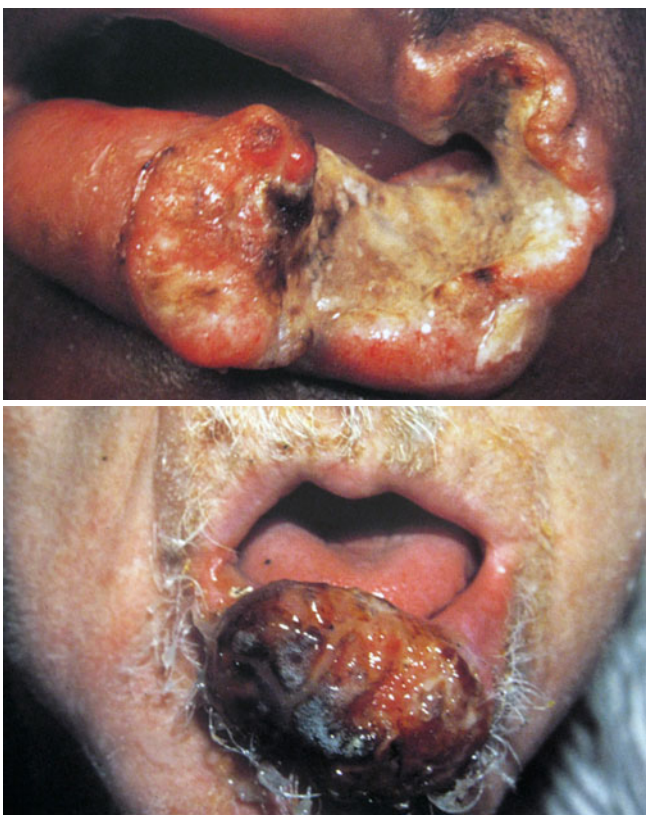


Fig. 50.4 Horrendous squamous cell carcinoma of the lip

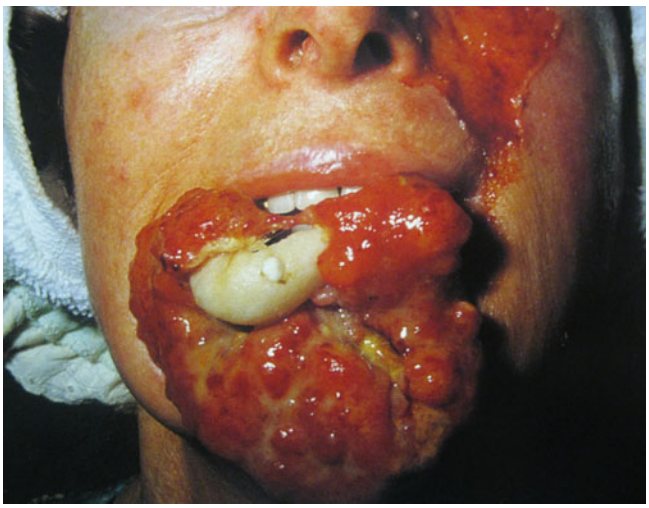
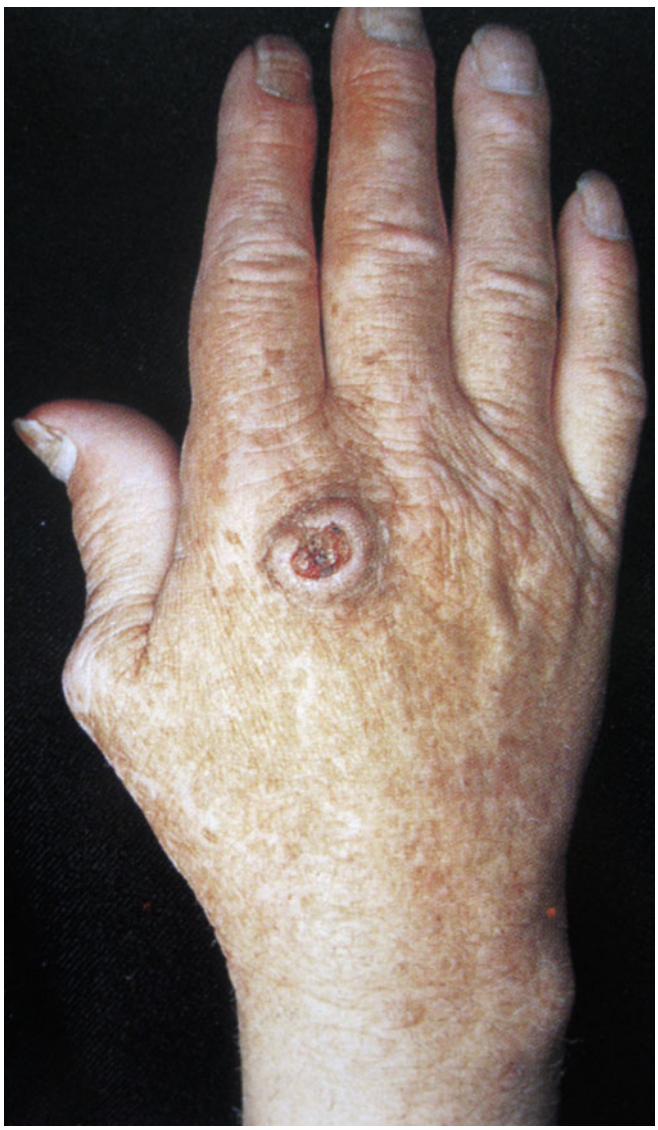
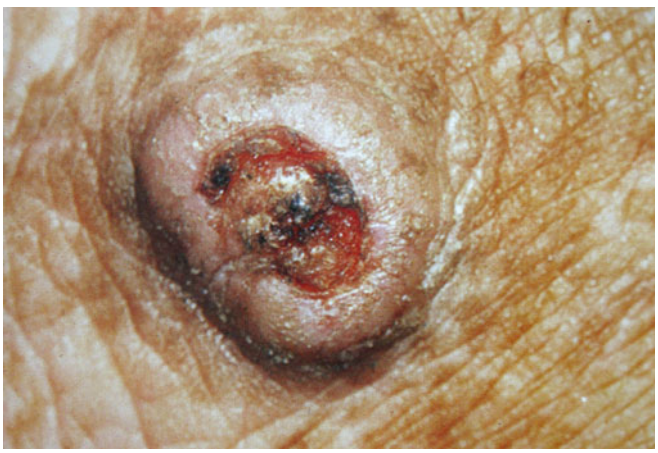


Fig. 50.5 Horrendous squamous cell carcinoma of the lip (Courtesy of Dr. S. L. Stedinger)





Figs. 50.6 and 50.7 Crateriform squamous cell carcinoma of the hand resembles a keratoacanthoma

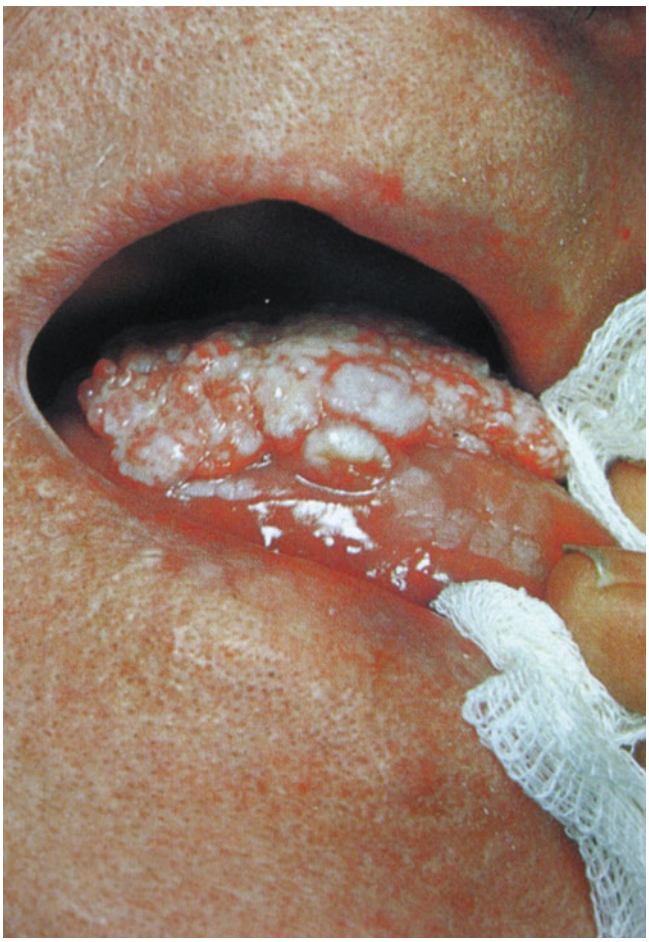


Fig. 50.8 Squamous cell carcinoma of the tongue. Complete dermatologic examination includes inspection of the mouth and oral mucosa

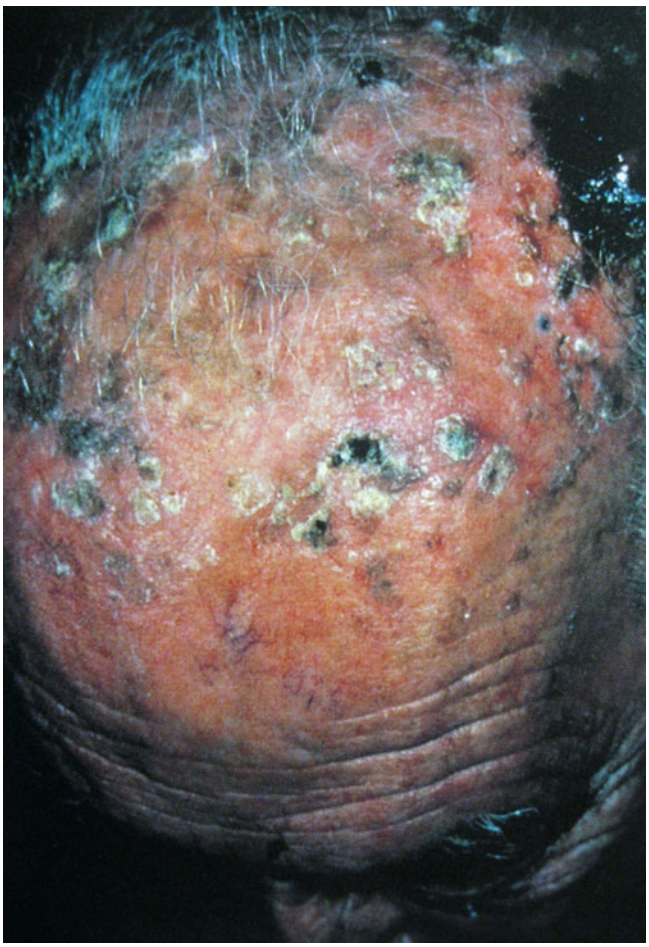


Fig. 50.10 Multiple squamous cell carcinomas of the scalp

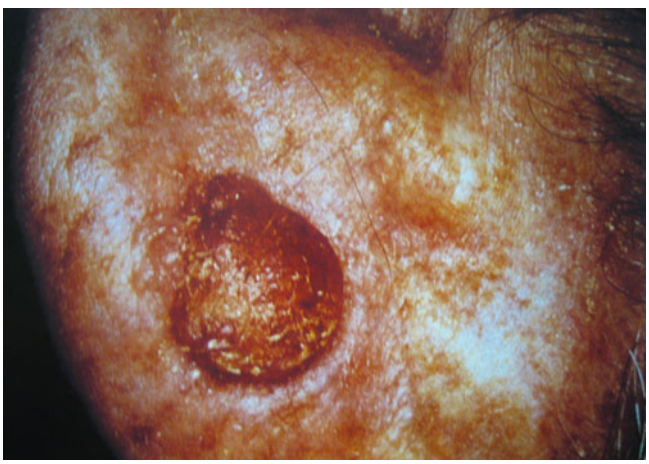


Fig. 50.9 Squamous cell carcinoma on the posterior ear. Sunscreen should also be applied to the backs of the ears

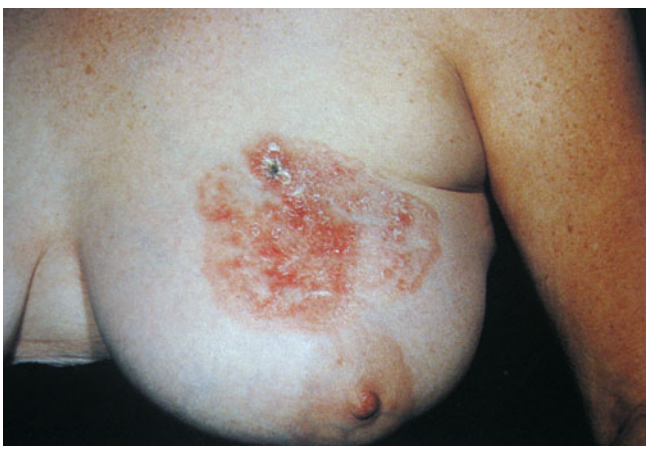


Fig. 50.11 Squamous cell carcinoma of the breast. This is a cutaneous lesion; no underlying adenocarcinoma was detected with mammography

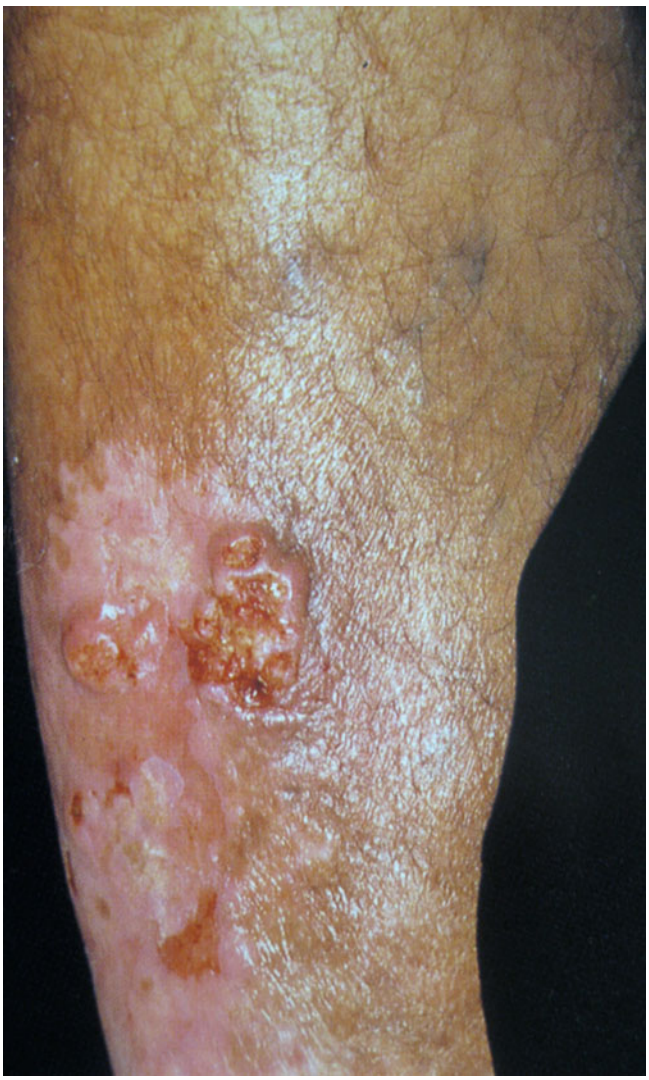


Fig. 50.12 Squamous cell carcinoma of the leg developed at the site of an old burn scar. Marjolin's ulcer refers to ulcerative squamous cell carcinoma arising in burn scars or draining sinus tracts

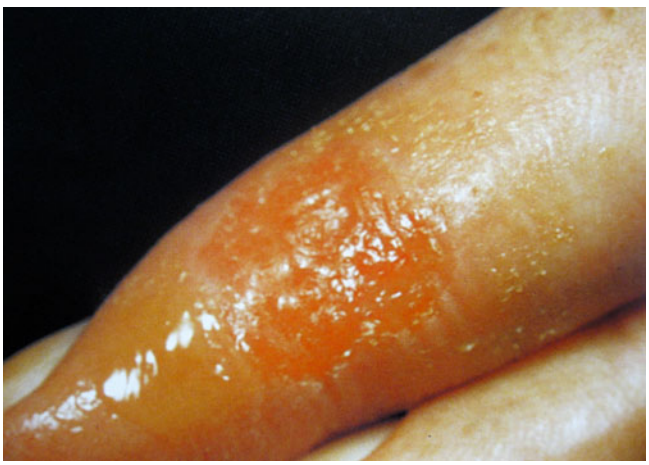


Fig. 50.13 Erosive squamous cell carcinoma arising from Bowen's disease (squamous cell carcinoma-in-situ) on the finger (see Chap. 42)

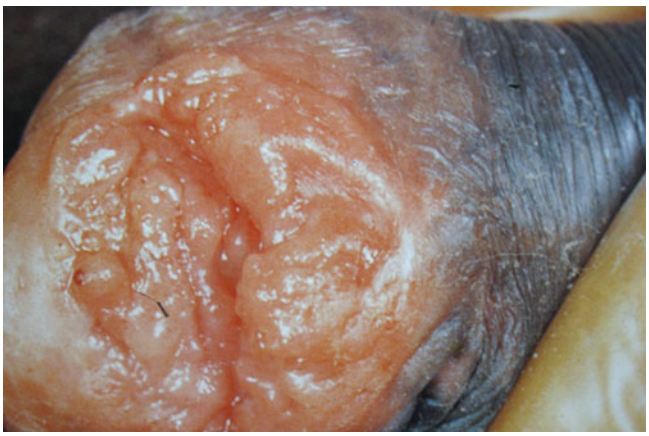


Fig. 50.14 Squamous cell carcinoma of the glans penis

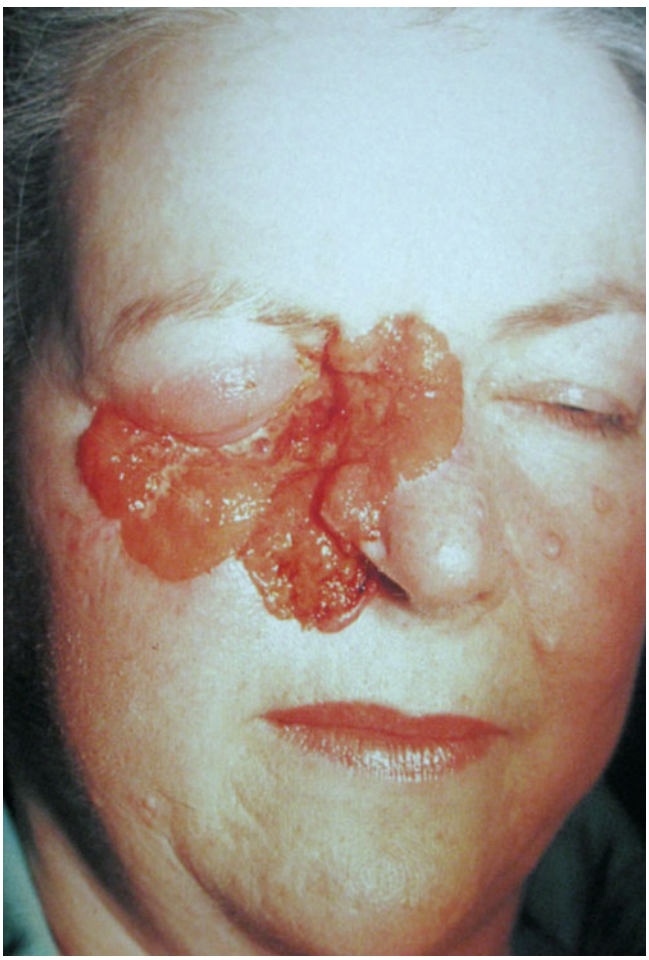


Fig. 50.15 Squamous cell carcinoma of the face has invaded the orbit

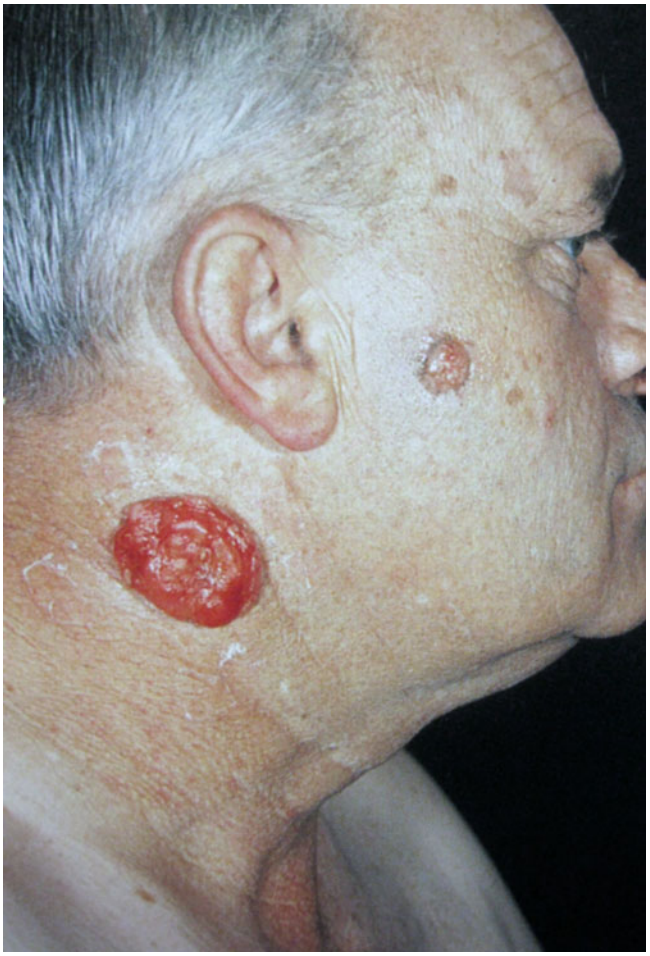


Fig. 50.16 Multiple large, fungating squamous cell carcinomas of the face

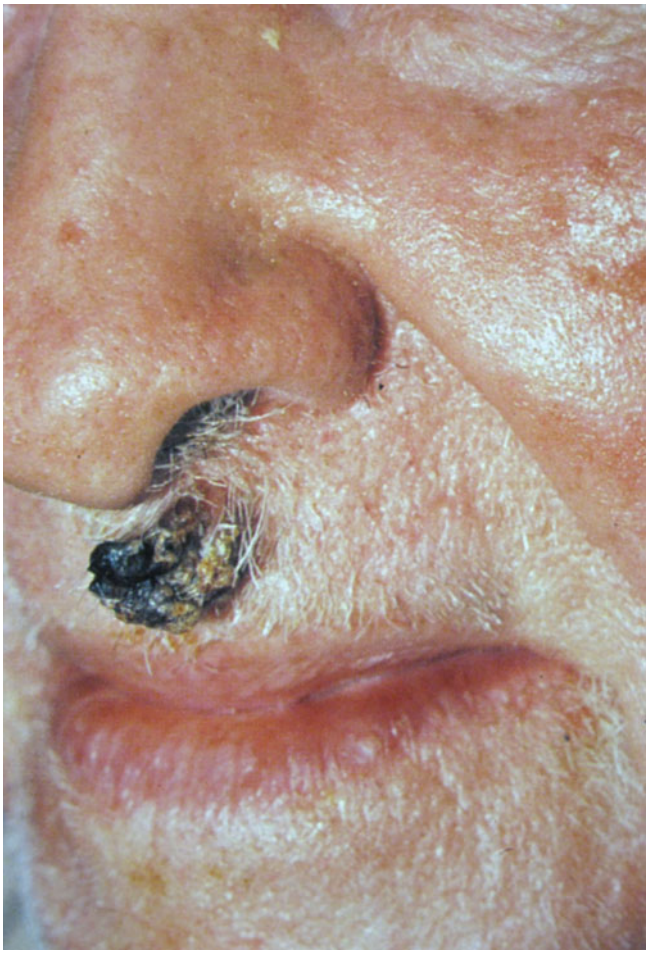


Fig. 50.17 Cutaneous horn of the upper lip. Underlying this horn may be an actinic keratosis, squamous cell carcinoma, keratoacanthoma, or wart. A biopsy sufficiently deep to include full-thickness epidermis is essential

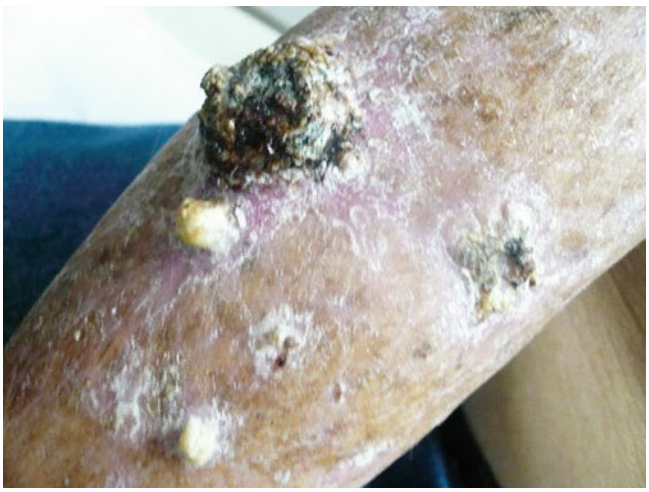


Fig. 50.18 Multiple squamous cell cancers on the arm

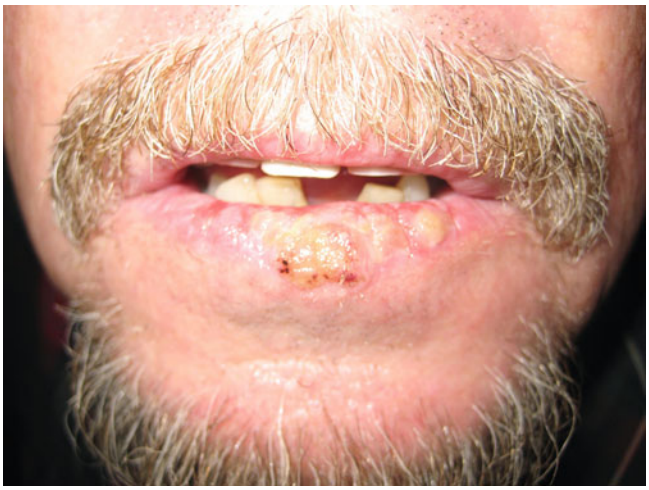


Fig. 50.19 Squamous cell carcinoma of the lip

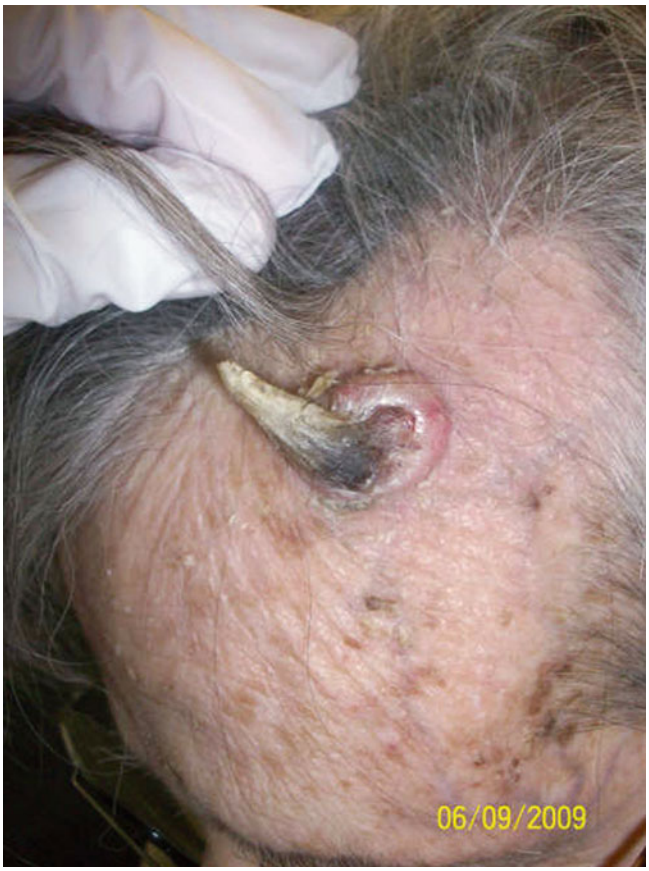


Fig. 50.20 Cutaneous horn arising from a squamous cell carcinoma on forehead

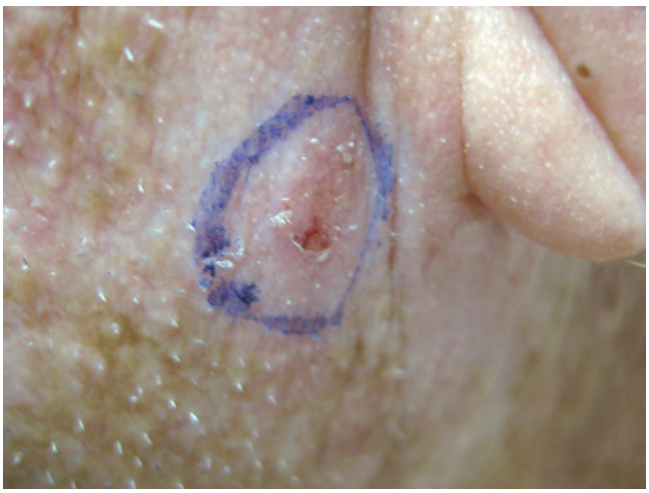


Fig. 50.21 A 79-year-old white male presented with a slightly eroded lesion anterior to the left ear. Examination showed a poorly circumscribed lesion that was covered by scanty white scales

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