


# Atopic Dermatitis

- Atopic dermatitis (AD) is a chronically relapsing skin disease that occurs most commonly during early infancy and childhood.
- It is frequently associated with elevated serum IgE levels and a personal or family history of AD, allergic rhinitis, and/or asthma.
- prevalence in children of 10 to 20 percent in the United States .
- The prevalence of AD in adults is approximately 1 to 3 percent.
- There is also a female preponderance for AD, with an overall female/male ratio of 1.3:1.

# CLINICAL MANIFESTATIONS

- AD typically begins during infancy.
- Approximately 50 percent of patients develop this illness by the first year of life and an additional 30 percent between the ages of 1 and 5 years.
- Nearly 80 percent of patients with AD eventually develop allergic rhinitis or asthma later in childhood.
- Intense pruritus and cutaneous reactivity are cardinal features of AD.
- Its consequences are scratching, prurigo papules , lichenification , and eczematous skin lesions.

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- Infantile phase ( 0-2 years)
  - Majority start within 6 m and onset around 3 m most common but earlier onset unusual.
  - Face, scalp, extensors,napkin area rarely affected.
  - Childhood phase ( 2-12 years)
  - Flexural involvement ,antecubital and popliteal.
  - Adolescent phase( 12-18 years)
  - Flexural and upper trunk and eyelids.



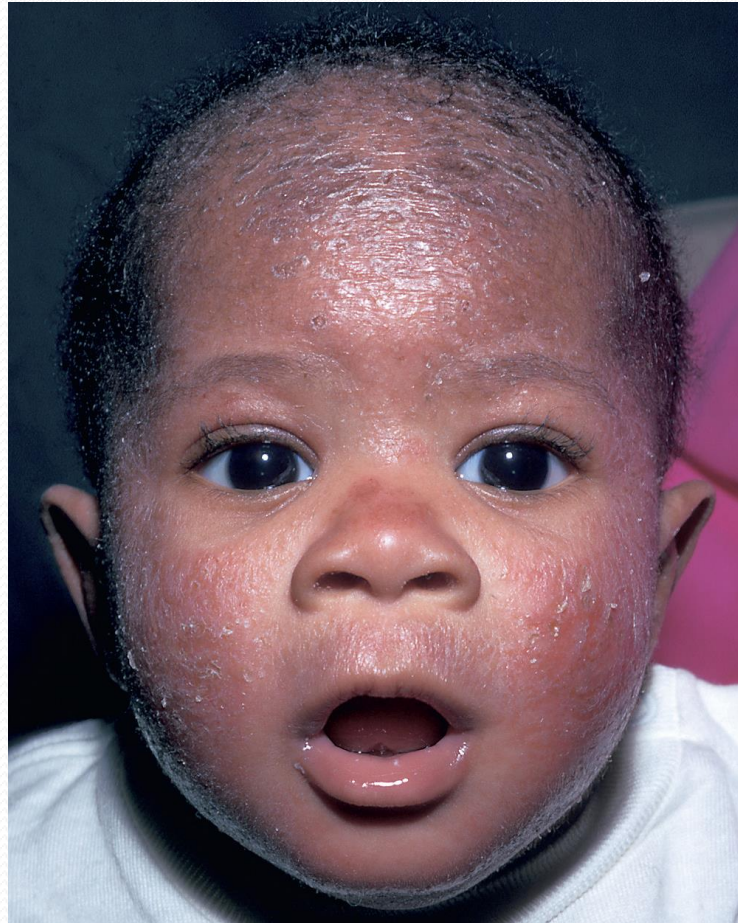
# DIAGNOSIS

- Major criteria-all required
- Pruritus
- Typical morphology and distribution of rash.
- Common findings ( at least two )
  - Personal or family history of atopy
  - Immediate skin test reactivity
  - White dermographism
  - Anterior subcapsular cataracts
- Associated findings ( at least four )
- Ichthyosis'xerosis'hyperlinear palms'pityriasis alba'facial pallor'infraorbital darkening'dennie-morgan folds'keratoconus'hand dermatitis'repeated cutaneous infection.





















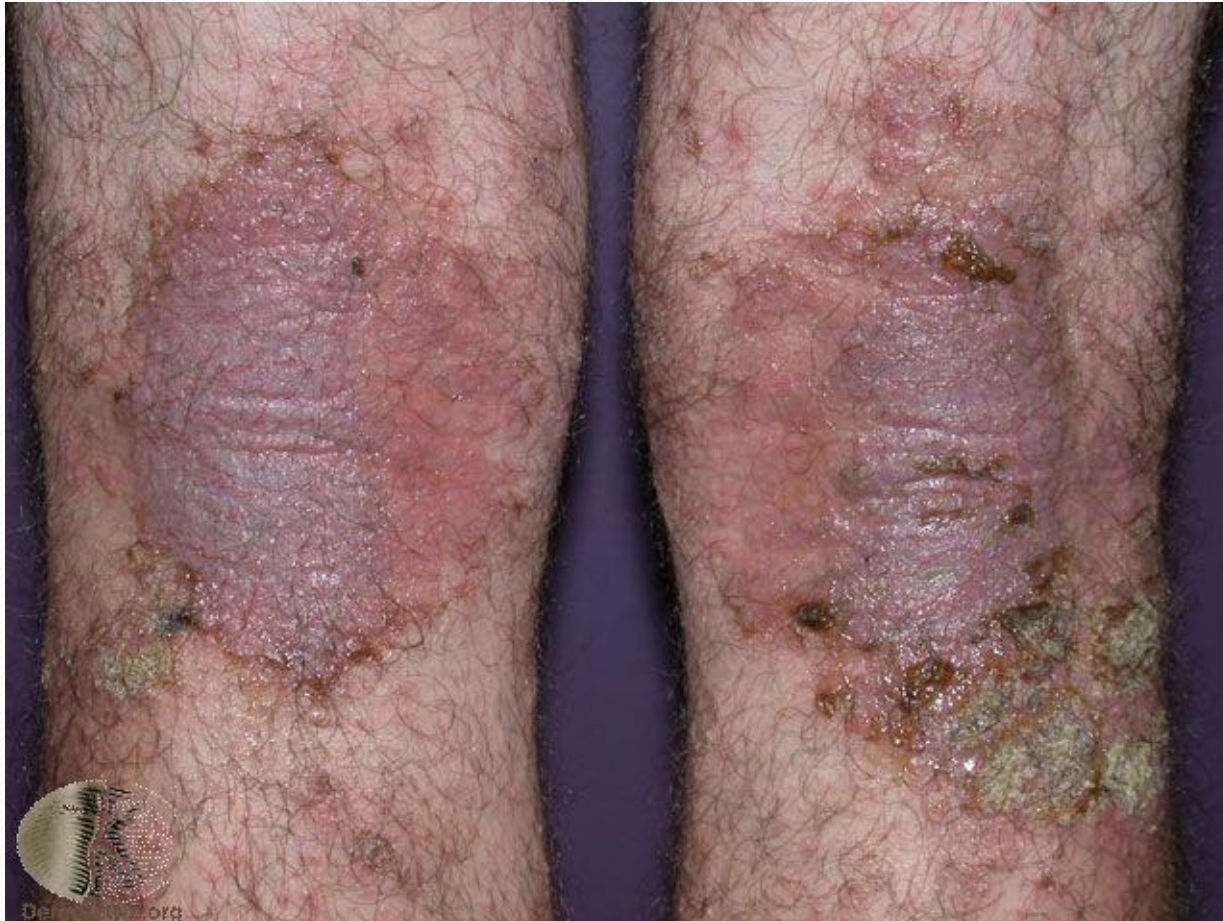
































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# Complications

- **OCULAR PROBLEMS** Eyelid dermatitis and chronic blepharitis are commonly associated with AD and may result in visual impairment from corneal scarring.
- Atopic keratoconjunctivitis is usually bilateral and can have disabling symptoms that include itching, burning, tearing, and copious mucoid discharge.
- Keratoconus is a conical deformity of the cornea believed to result from chronic rubbing of the eyes in patients with AD and allergic rhinitis.
- Cataracts were reported in the early literature to occur in up to 21 percent of patients with severe AD.

# INFECTIONS

- The most serious viral infection is herpes simplex, which can affect patients of all ages, resulting in Kaposi's varicelliform eruption or eczema herpeticum.
- After an incubation period of 5 to 12 days, multiple, itchy, vesiculopustular lesions erupt in a disseminated pattern; vesicular lesions are umbilicated, tend to crop, and often become hemorrhagic and crusted. Punched out and extremely painful erosions result. These lesions may coalesce to large, denuded and bleeding areas that can extend over the entire body.

Superficial fungal infections are also more common in atopic individuals and may contribute to the exacerbation of AD. Patients with AD have an increased prevalence of *Trichophyton rubrum* infections compared to nonatopic controls.

- As discussed earlier, *S. aureus* is found in more than 90 percent of AD skin lesions. Honey-colored crusting, folliculitis, and pyoderma are indicators of secondary bacterial skin infection, usually due to *S. aureus* that requires antibiotic therapy.














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- **HAND DERMATITIS** Patients with AD often develop a nonspecific, irritant hand dermatitis.
  - It is frequently aggravated by repeated wetting and by washing of the hands with harsh soaps, detergents, and disinfectants.
  - Atopic individuals with occupations involving wet work are prone to develop an intractable hand dermatitis in the occupational setting. This is a common cause of occupational disability.

- **EXFOLIATIVE DERMATITIS** Patients with extensive skin involvement may develop exfoliative dermatitis.
- This is associated with generalized redness, scaling, weeping, crusting, systemic toxicity, lymphadenopathy, and fever .
- Although this complication is rare, it is potentially life-threatening.
- It is usually due to superinfection, for example, with toxin-producing *S. aureus* or herpes simplex infection, continued irritation of the skin, or inappropriate therapy.
- In some cases, the withdrawal of systemic glucocorticoids used to control severe AD may be a precipitating factor for exfoliative erythroderma.















Infiltrated, erythematous facial lateral  
thinning of eyebrows and infraocular  
(Morgan's) fold






# DIFFERENTIAL DIAGNOSIS

- Of the major features, pruritus and chronic or remitting eczematous dermatitis with typical morphology and distribution are essential for diagnosis.
- lists a number of inflammatory skin diseases, immunodeficiencies, skin malignancies, genetic disorders, infectious diseases, and infestations that share symptoms and signs with AD.

- Infants presenting in the first year of life with failure to thrive, diarrhea, a generalized scaling erythematous rash, and recurrent cutaneous and/or systemic infections should be evaluated for severe combined immunodeficiency syndrome.
- Wiskott-Aldrich syndrome is an X-linked recessive disorder characterized by cutaneous findings almost indistinguishable from AD. It is associated with thrombocytopenia, variable abnormalities in humoral and cellular immunity, and recurrent severe bacterial infections.
- Hyperimmunoglobulin-E syndrome is characterized by markedly elevated serum IgE levels, defective T cell function, recurrent deep-seated bacterial infections, including cutaneous abscesses due to *S. aureus* and/or pruritic skin disease due to *S. aureus* pustulosis, or by recalcitrant dermatophytosis.

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- It is important to recognize that an adult who presents with an eczematous dermatitis with no history of childhood eczema, respiratory allergy, or atopic family history may have allergic contact dermatitis .
  - A contact allergen should be considered in any patient whose AD does not respond to appropriate therapy.
  - Of note, topical glucocorticoid contact allergy has been reported increasingly in patients with chronic dermatitis on topical corticosteroid therapy.
  - Cutaneous T cell lymphoma must be ruled out in any adult presenting with chronic dermatitis poorly responsive to topical glucocorticoid therapy.














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- Eczematous dermatitis has been also reported with HIV as well as with a variety of infestations such as scabies.
  - Other conditions that can be confused with AD include psoriasis, ichthyoses, and seborrheic dermatitis.







# Keratolysis exfoliativa.



# Irritant hand dermatitis





# Nummular eczema.





















# TREATMENT AND PROGNOSIS

- *Topical corticosteroids*
- Topical corticosteroids represent first-line pharmacologic therapy for AD.
- These agents have anti-inflammatory antiproliferative, immunosuppressive, and vasoconstrictive actions, with effects on cutaneous T cells, macrophages, and dendritic cells.



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- Newest topical corticosteroid
  - Budesonide
  - Mometasone furoate
  - Prednicarbate topical cream
  - Fluticasone propionate
  - Have higher anti inflammatory effect.
  - Good copilance- once daily application
  - Weak atrophogenicity.


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- *Crisaborole*
  - Crisaborole 2% ointment is a phosphodiesterase-4 (PDE-4) inhibitor that is FDA-approved for the treatment of mild to moderate AD in patients  $\geq 2$  years of age.
  - The most common side effect is stinging or burning in the area of application.

- **CUTANEOUS HYDRATION**
- **TOPICAL GLUCOCORTICOID TREATMENT**
- **Topical Immunomodulators**
- ***Tacrolimus***
- Tacrolimus inhibits the activation of a number of key cells involved in AD including T cells, Langerhans cells, mast cells, and keratinocytes.
- ***Pimecrolimus***
- **INFECTIOUS AGENTS**
- **Phototherapy**
- Ultraviolet B (311 nm), UVA-1 (340 to 400 nm), and combined UVAB phototherapy can be useful adjuncts in the treatment of AD.

- *Dupilumab*
- Dupilumab is a human monoclonal antibody that targets the IL-4.
- Dupilumab is administered via subcutaneous injection of 600 mg initially and then 300 mg every two week, and it can be used with or without concurrent topical corticosteroid treatment.

It has a favorable side effect profile, with injection site reactions and conjunctivitis each occurring in ~10% of patients.





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- Dupilumab is FDA-approved for the treatment of adults with moderate to severe atopic dermatitis that is not adequately controlled with topical therapy.

Risk of skin infection eczema herpeticum.

Laboratory monitoring not required.

Suggest first line systemic treatment for adult severe AD.

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- *Omalizumab*
  - The anti-IgE monoclonal antibody omalizumab is FDA-approved for chronic idiopathic urticaria in patients  $\geq 12$  years of age and for asthma in patients  $\geq 6$  years of age.
  - It is administered every 2–4 weeks via subcutaneous injection, and potential side effects include a risk of anaphylaxis.

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- Although improvement of AD with omalizumab therapy has been described in uncontrolled series, a small randomized controlled trial in adults with AD did not demonstrate clinical improvement, despite reduction in IgE levels.

# Tralokinumab

- Anti iL-13
- Adult sever AD
- Adverse effect viral upper respiratory infection.
- 600mg once followed by 300 mg once ewery other week.




# Jak inhibitors

- Abrocitinib
- Adult AD
- 100mg once daily
- Upadacitinib
- 15mg daily
- Adverse effect
- Acne, headache, nasopharyngitis


# Third-line therapies

- **ANTIMETABOLITES** Mycophenolate mofetil (MMF).
- Methotrexate.
- Azathioprine .
- cyclosporine
- **EXTRACORPOREAL PHOTOPHERESIS**

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- Following predictive factors correlate with a poor prognosis for AD:  
widespread AD in childhood;  
associated allergic rhinitis and asthma;  
family history of AD in parents or siblings; early age at onset of AD; being an only child; and very high serum IgE levels .


# Ultraviolet Radiation




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- It is usually subdivided, rather arbitrarily, into UVC (200–290 nm), UVB (290–315 nm), and UVA (315–400 nm).
  - The sun emits UV radiation as part of an electromagnetic spectrum.
  - More than 95% of the sun's UV radiation that reaches the earth's surface is UVA.
  - Practically all of the UVC, and much of the UVB, are absorbed by the oxygen and ozone in the earth's atmosphere, so that UV radiation below 290 nm is virtually undetectable at ground level.


# *Polymorphous light eruption*

- The most common photodermatosis.
- Action spectra: UVB, UVA, and rarely visible light.
- Attacks are intermittent and follow minutes to hours (rarely days) of exposure of
- the skin to sunlight or artificial UVR.
- Non-scarring, pruritic, erythematous papules, papulovesicles, vesicles, or plaques then develop hours later (occasionally within minutes).
- PMLE appears to represent a delayed-type hypersensitivity (DTH) response to as-yet-undefined, endogenous cutaneous, *photoinduced* antigens.

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- Women are affected slightly more often than men, with the second and third decades being the most common times of onset.
  - Susceptibility to PMLE appears to be genetic, with up to 70% of the population having a propensity for developing this condition.

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- PMLE occurs most commonly during the spring and early summer, following minutes to hours (occasionally days) of sun exposure.
  - The eruption develops hours (occasionally minutes) after UVR exposure; it then fades over one to several days or occasionally weeks if the exposure is ongoing.
  - However, the likelihood of occurrence often diminishes or ceases over the summer or a lengthy sunny vacation, presumably due to the development of immunologic tolerance, sometimes called “hardening.”



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- Typically, some, but virtually never all, of the exposed skin is affected.
  - Lesions are usually symmetrically distributed in a patchy fashion. Areas that are normally continuously exposed to sunlight (e.g. face) are often spared, although not always.
  - Areas commonly affected are the neck, outer aspects of the arms, and dorsal hands, but there may be more widespread involvement of sun-exposed sites .

- Clinically, mildly to markedly pruritic, grouped, erythematous or skin-colored papules of varying sizes, sometimes coalescing into large, smooth or unevenly surfaced plaques, are seen.
- Vesicles, bullae, papulovesicles, and confluent edematous swelling are additional manifestations.
- Rarely patients develop fever, general malaise, headache, and nausea.
- PMLE may be lifelong; however, in a 32-year follow-up of 94 patients, the disease improved or resolved in 58% over a period of 16 years.









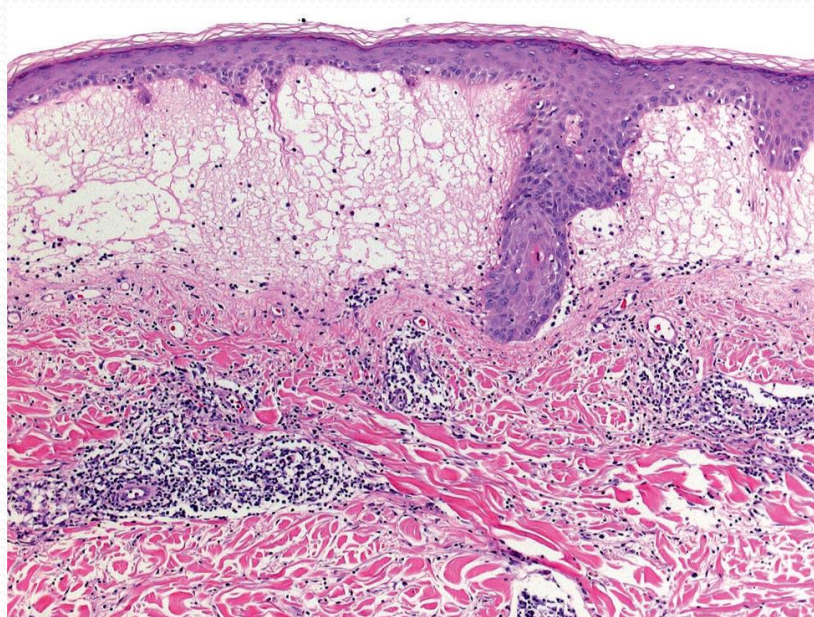








- Pathology
- There is variable epidermal spongiosis and a superficial and deep, perivascular and periappendageal, lymphohistiocytic dermal infiltrate, often with scattered eosinophils and neutrophils.
- Significant papillary dermal edema occurs commonly.
- Differential diagnosis
- PMLE can often be distinguished from lupus erythematosus (LE), photoaggravated dermatoses (e.g. atopic dermatitis, seborrheic dermatitis), solar urticaria, and rarely, erythropoietic protoporphyria (EPP) by the natural history and clinical appearance of the cutaneous lesions.




- Treatment
- PMLE in its milder forms may respond to photoprotection, including the use of broad-spectrum, high SPF sunscreens and physical barriers.
- In patients with more severe disease, hardening via prophylactic, two-to-three times weekly sessions of NB-UVB, usually for 15 sessions in the spring, may be effective for several months.
- Oral prednisone (0.5–1 mg/kg)
- Hydroxychloroquine
- For symptomatic eruptions, topical corticosteroids and oral prednisone can be prescribed.

# *Actinic prurigo*

- cheilitis and conjunctivitis.
- Actinic prurigo is a fairly uncommon, sunlight-induced, pruritic and crusted, papular or nodular eruption involving uncovered and, to a lesser extent, covered skin. UVB or UVA.
- There is also a strong association of actinic prurigo with HLA-DR<sub>4</sub>
- Actinic prurigo usually appears during childhood and is more frequently seen in girls. It often resolves by adolescence, but may




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- Erythematous papules or nodules, sometimes with hemorrhagic crusts due to excoriation, are present in sun-exposed sites, especially the face (including the nose) and the distal limbs.
  - pitted scars may appear as facial papules heal.
  - In patients from the UK, involvement of covered sites, particularly the buttocks, has been well described .
  - Cheilitis and conjunctivitis are common, with cheilitis often being the only clinical manifestation.










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- Treatment
  - Rigorous photoprotection is helpful.
  - Topical corticosteroids and topical tacrolimus.
  - NB-UVB should be considered next, with protocols similar to those for PMLE.
  - Resistant disease is best treated with oral thalidomide (50–100 mg nightly).
  - However, the risks of teratogenicity and peripheral neuropathy
  - require very careful patient selection and supervision .
  - Other possible systemic therapies include

# *Hydroa vacciniforme*

- The condition typically has its onset during childhood, affecting boys slightly more often than girls and with more severe disease in boys.
- The disorder can resolve during adolescence or early adulthood; rare familial cases have been noted.
- Symmetrical, clustered, pruritic or stinging, erythematous macules develop in a photodistribution, especially on the face and dorsal aspects of the hands, within hours of summer sun exposure.
- Over the next several hours, the macules become tender papules or plaques surmounted by vesicles or bullae (often hemorrhagic), before umbilicating and


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- Healing then occurs over a period of weeks, leaving individual or confluent, sometimes telangiectatic, varioliform scars.
  - Epstein–Barr viral infection has been detected in a number of patients.










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- Differential diagnosis
  - While LE and childhood porphyrias can be excluded by clinicopathologic correlation and laboratory evaluation , herpes simplex is excluded by a negative PCR.
- HV is almost always refractory to treatment.
- Photoprotection.
- Broadband (BB)-UVB, NB-UVB, PUVA,  $\beta$ -carotene, antimalarials, azathioprine, thalidomide, cyclosporine.

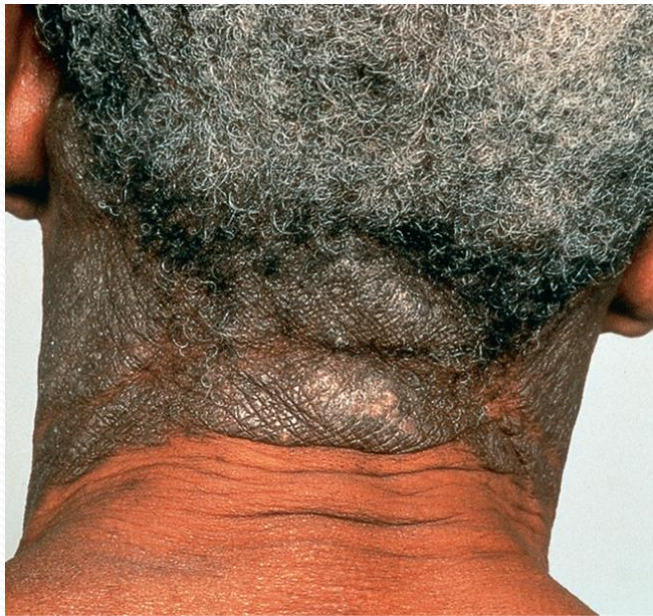
# *Chronic actinic dermatitis*

- Chronic actinic dermatitis is an uncommon, persistent, often disabling dermatosis of uncovered (and to a lesser extent, covered) skin, generally affecting older men, particularly in the summer.
- Summer.
- It is evoked by UVR and occasionally also visible light exposure, and CAD probably represents a contact allergy-like DTH response against endogenous
- photoinduced antigen(s).
- most commonly affecting older men of any race.
- However, familial incidence has not been noted.

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- The disorder may develop in previously normal skin, in patients with a prior history of dermatitis (in particular, photoallergic or allergic contact dermatitis), or rarely following longstanding oral drug photosensitivity or PMLE.
  - The CAD
  - eruption is pruritic, patchy or confluent, and the eczematous lesions can be acute, subacute or chronic in nature; the latter is frequently associated with lichenification.
  - Scattered or widespread, erythematous, shiny, infiltrated, pseudolymphomatous papules or plaques




- Lesions develop primarily within sun-exposed sites, frequently with a sharp cut-off at the lines of clothing.
- sparing in the depths of skin furrows, upper eyelids, finger webs, nasolabial folds, or postauricular areas.
- Occasionally, palmoplantar eczematous changes may also be present, while eyebrow or scalp hairs may be stubbly or sparse from rubbing or scratching.
- Rarely, erythroderma develops in severely affected patients.
- The probability of resolution has been estimated to be 10% over 5 years, 20% over 10 years, and 50% over 15 years.







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- Treatment
  - Strict photoprotection and the avoidance of relevant contact allergens are of primary importance.
  - Topical or intermittent oral corticosteroid therapy along with emollient use is generally needed, and topical tacrolimus.
  - Therapy for refractory disease includes very low-dose PUVA with initial high-dose oral and topical corticosteroid coverage over months, cyclosporine (3.5–5 mg/kg/day), azathioprine or mycophenolate mofetil (1–2 g/day).