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An Overview of Psoriasis and Atopic Dermatitis

Clinical presentation, differential diagnosis, and management of disease

Prevalence, overlapping features, and pathogenesis of psoriasis (PSO) and atopic dermatitis (AD)[1]

PSO and AD are inflammatory skin diseases with distinct clinical features[1,2]

PSO[1,3,4]

- Prevalence
  - 2% in Caucasians
  - 0.2% in Asians
- Occurs on the scalp and skin surface outside of joints
- Peak onset is between 20 and 30 years of age
- Patients with PSO continue to experience the clinical symptoms throughout life

AD[1,5,6]

- Prevalence of AD in children: 20%
- Occurrence of AD varies with age
  - During infancy – face and skin surface outside of joints
  - In adolescents and adults – skin surface inside of joints and hands
- AD initially occurs during early childhood and improves around adolescence

Overlapping features of PSO and AD[1]

Both PSO and AD share genetic profiles, immune pathways, and pathologic changes

Genetic profiles[3,4,6–9]

PSO

- HLA-Cw*0602 (on PSORS1 6p21) is a common susceptibility gene locus

AD

- Null mutation of *filaggrin* (*FLG*) gene is a strong genetic risk factor
- *FLG* mutation variants increase the risk of developing PSO in Taiwanese and Chinese populations

5q31.1-q33.1 is a shared chromosome for both AD and PSO

Immunopathogenesis[1]

PSO

- Genetic factors and environmental triggers together induce PSO
- A cascade of events involving:
  - T helper 17 (Th17) cells
  - Interleukin (IL)-23 and IL-17
  - Tumour necrosis factor alpha (TNF-α)

AD

- Causes include:
  - Genetic factors
  - Epidermal barrier defects
  - Immune dysregulation
  - Microbiome imbalance
- Pathogenesis involves:
  - Th2 pathway
  - IL-4, IL-5, IL-13, IL-31, and IL-33
  - Thymic stromal lymphopoietin

Both PSO and AD involve Th1, Th17, and Th22 cells

In children, PSO and AD can occur as overlapping conditions or with very similar presentations

Subtypes of overlapping PSO and AD[1]

- PSO with AD features (nummular PSO, erythrodermic PSO)
- AD with PSO features (Asian AD)
- Coexisting AD and PSO (PSO dermatitis)
- Development of AD-like dermatitis during PSO or AD treatment (TNF-αi, IL-12/23i, IL-17i, IL-23i, IL-4/13i)
- Development of PSO during AD treatment (dupilumab)

Impact of skin diseases on quality of life (QoL) and mental well-being[2]

- Skin diseases can affect a person's self-confidence and reduce their willingness to socialise
- QoL can be affected due to the abnormal appearance of the skin leading to personal and psychological dysfunction

Metabolic and neurological comorbidities in Asian patients with PSO and AD[10]

Asian patients with PSO are more likely to develop:

- Obesity
- Hypertension
- Diabetes
- Chronic kidney disease
- Parkinson's disease

Asian patients with PSO are susceptible to metabolic and neurologic comorbidities and need to be monitored appropriately[10]

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PSO – clinical presentation, differential diagnosis, diagnostic criteria, and complications

Plaque PSO[11]

- Accounts for >80% of PSO cases
- Defined by erythematous scaly patches
- Plaques are more common on extensor surfaces
- Can also affect the intertriginous areas like palms, nails, and soles

Clinical presentation of PSO[11]

- Clinical characteristics depend on the variant
- PSO variants – plaque PSO, erythrodermic PSO, guttate PSO, and pustular PSO
- Variants of PSO follow 3 key clinical features: erythema, thickening, and scales

Plaque PSO can negatively affect the QoL when it involves the face and palms

Differential diagnoses of PSO[11]

- PSO lesions have characteristic clear demarcation on the lesion edge
- Diagnosis involves family history of psoriatic diseases, comprehensive skin and nail examination
- Evaluation of morphology and spread of PSO lesions
- A skin biopsy may be required for atypical cases

Treatment of PSO[11]

The overall treatment approach for PSO begins with evaluation for psoriatic arthritis

Presence of active psoriatic arthritis can affect treatment choices

Treatment options for patients with mild PSO

- Topical corticosteroids
- Vitamin D analogues
- Calcineurin inhibitors
- Keratolytics
- Targeted phototherapy

Comorbidities associated with PSO[11]

~33% of patients with PSO develop psoriatic arthritis

Psoriatic arthritis is characterised by pain, stiffness, and swelling of joints and can lead to debilitating joint disorders

Utilisation of topical corticosteroids and therapeutic drugs can improve the skin condition and contribute to a better QoL[11]

Clinical presentation, differential diagnosis, and management of AD

AD[12]

Characterised by the presence of eczema, intense pruritus, and dry skin

Eczema and pruritis are a result of altered barrier function of the skin and dysfunction of the immune system

Clinical presentation and differential diagnosis of AD[13,14]

- Pruritus or itching is a common symptom
- Repeated scratch-itch cycles can lead to poor QoL
- Depending on the patient's age, symptoms can vary

- Clinical diagnosis of AD involves no definitive laboratory test
- ~80% of patients are diagnosed and treated in primary care settings

Diagnostic criteria for AD by the American Academy of Dermatology[13,15]

Essential features: must be present for diagnosis

- Chronic or relapsing history
- Eczema
- Pruritus
- Typical morphology and age-specific patterns

Important features: support the diagnosis (observed in most cases)

- Atopy (personal or family history)
- Early age at onset
- Immunoglobulin E reactivity
- Xerosis

Associated features: suggestive of the diagnosis but non-specific

- Atypical vascular responses
- Keratosis pilaris, hyperlinear palms, pityriasis alba, or ichthyosis
- Ocular or periorbital changes
- Lichenification, perifollicular accentuation, or prurigo lesions

Complications associated with AD[13]

- Infections are common in patients with AD due to the disruption of the epidermis
- *Staphylococcus aureus* and beta-haemolytic *Streptococcus* bacterial infections on skin
- Post-inflammatory scarring and lichenification due to uncontrolled scratch-itch cycles

Management of AD[13,16–20]

- Education and awareness among patients and family members can reduce disease severity and improve QoL
- Emollients (moisturisers)
  - Application of emollients can be beneficial
  - Retain moisture, decrease disease severity, and prolong the interval between flare-ups
  - Significantly reduce the need for prescription drugs to treat AD
- Bathing practices
  - Hydrate and cleanse the skin
  - Remove scales, bacteria, irritants, and allergens
  - Bathing with lukewarm water once daily is recommended

Clinically diagnosing AD using the diagnostic criteria by the American Academy of Dermatology is vital to enhancing the treatment outcomes

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Treatment approaches, therapeutic drugs, and biologics for PSO and AD

For PSO[11]:

Topical corticosteroids
- Main form of therapy for patients with mild PSO
- Possess antiproliferative, anti-inflammatory, and locally vasoconstrictive effects
- Treatment with topical corticosteroids necessitates appropriate dosage of medication and patient adherence
- During active PSO, they can be used twice daily

Topical Vitamin D analogues
- Block keratinocyte proliferation
- Calcitriol in combination with calcipotriol or calcipotriene

Topical calcineurin inhibitors
- Block T cell activation by inhibiting the synthesis of IL-2 and interferon gamma
- Tacrolimus and pimecrolimus

Topical keratolytics
- Include tazarotene and salicylic acid
- Topical tazarotene, a retinoid, inhibits the proliferation of keratinocytes and breaks down the thick scales of the plaque

Targeted phototherapy
- Phototherapy or light therapy, involves exposure to specific wavelengths of light
- Used to treat patients with plaque PSO

For AD[12]:

Topical treatment of AD involves topical corticosteroids and topical calcineurin inhibitors (pimecrolimus and tacrolimus)

Topical inhibitors of the Janus kinase (JAK)/STAT pathway
- Hold significant potential for the treatment of AD
- Ruxolitinib – JAK1/JAK2 inhibitor
- Delgocitinib – JAK inhibitor

Topical inhibitor of phosphodiesterase-4 (PDE-4)
- Crisaborole 2% is a PDE-4 inhibitor approved for treating mild-to-moderate AD

Aryl hydrocarbon receptor agonists
- Tapinarof suppresses IL-17 and IL-22

Transient receptor potential vanilloid 1 (TRPV1) antagonists
- TRPV1 can regulate pruritus, epidermal barrier function, and inflammation
- Asivatrep, a promising antagonist of TRPV1, is in a phase 2b trial

Biologics for the treatment of PSO and AD

4 classes of biologics are available to treat PSO[11]
- TNF inhibitors
- IL-12/23 inhibitors
- IL-17 inhibitors
- IL-23 inhibitors

Biologics in the treatment of AD[12]
- Dupilumab inhibits IL-4 and IL-13 signalling pathways
- Tralokinumab and lebrikizumab are IL-13 inhibitors

Key takeaways

Timely and accurate diagnosis of PSO and AD is vital for improving patient outcomes

With increased patient adherence, currently available therapies can enhance the overall QoL

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