ACE CLINICAL GUIDELINE

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Mild and moderate atopic dermatitis (eczema)

A journey from flare to care

Objective	Scope	Target audience
To optimise management and achieve disease control in mild and moderate atopic dermatitis (AD)	Diagnosis, severity assessment and management of AD, including topical and non-pharmacological interventions in adults and children	This clinical guideline is relevant to all healthcare professionals caring for patients with AD, especially those providing primary or generalist care

Atopic dermatitis (AD) is a common **chronic inflammatory skin condition** characterised by dry skin with recurrent episodes of itching and inflammation. It arises from a complex interplay of genetic predisposition, immune dysfunction and environmental factors, leading to impaired skin barrier function and heightened inflammatory responses.[1] **The clinical course of AD typically alternates between remission and relapses** with symptoms ranging from mild dryness and irritation to severe itch, inflammation and lichenification. In Singapore, AD affects approximately 13.1% of people with higher rates in children (20.6%) than adults (11.1%).[2] A substantial proportion of children with AD will continue to have the condition throughout their lives.

This ACE Clinical Guideline (ACG) provides evidence-based recommendations for managing **mild and moderate AD** to prevent disease progression to more severe cases that require specialist intervention (Figure 1). With appropriate therapeutic interventions and comprehensive care routines, many patients can achieve disease control and maintain healthy, flare-free skin. The focus of this ACG is **primary care settings** where there are numerous opportunities to provide impactful counselling and early interventions that can significantly improve patient outcomes and quality of life.

Statement of Intent
This ACE Clinical Guideline (ACG) provides concise, evidence-based recommendations and serves as a common starting point nationally for clinical decision-making. It is underpinned by a wide array of considerations contextualised to Singapore, based on best available evidence at the time of development. The ACG is not exhaustive of the subject matter and does not replace clinical judgement. The recommendations in the ACG are not mandatory, and the responsibility for making decisions appropriate to the circumstances of the individual patient remains at all times with the healthcare professional.

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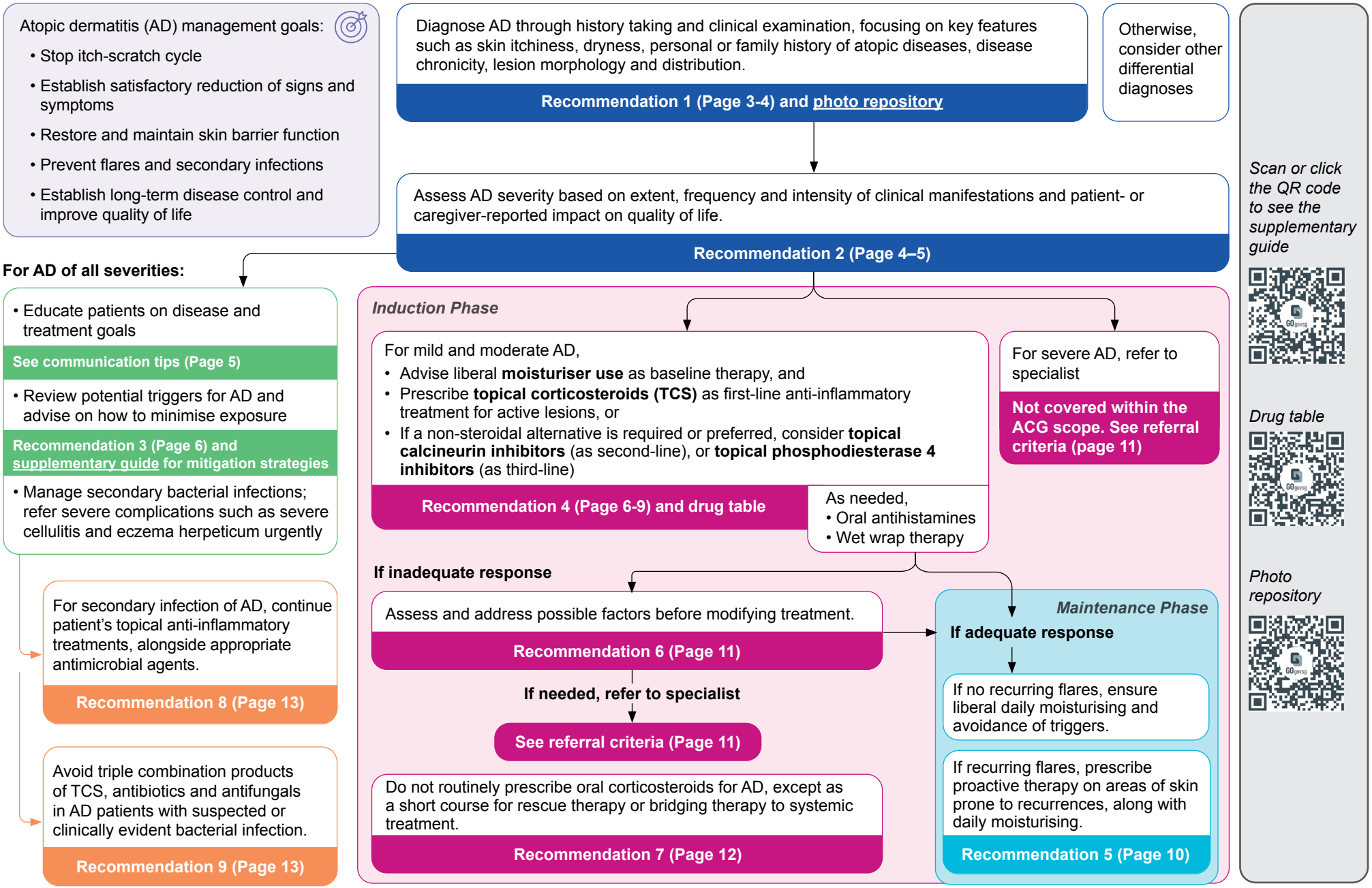
Figure 1. Approach to atopic dermatitis management – at a glance



Diagnosis and severity assessment

Recommendation 1 **Diagnose AD through history taking and clinical examination, focusing on key features such as skin itchiness, dryness, personal or family history of atopic diseases, disease chronicity, lesion morphology and distribution.**

AD is a clinical diagnosis and it relies on recognising key clinical features through careful history taking and physical examination.

History taking should focus on the two most prominent symptoms: **skin itch** (present in 94% of patients) and **dryness** (73% of patients). Explore **personal or family history of atopic diseases** (e.g. asthma, allergic rhinitis and allergic conjunctivitis) which are present in 53% of people with AD.[5,6] Identifying individual triggers is important during history taking, as these can vary significantly between patients and may include environmental factors, allergens, stress, or specific irritants (refer to Recommendation 3). Clinicians can **establish chronicity** by checking age of onset, frequency and duration of previous episodes (if any), their specific characteristics and triggers, followed by examining skin for any signs of long-term inflammation (see 'chronic lesions' below).

Are blood tests (e.g. sIgE), routine food allergy tests, and skin biopsies necessary for AD diagnosis?

- Blood tests for serum Immunoglobulin E (sIgE) are elevated in a wide variety of allergic conditions and even in non-atopic conditions. Hence, elevated sIgE does not confirm AD.[3, 4]
- Food allergies are more common in the paediatric patient populations. However, the clinical utility of routine food allergy testing is limited by high false positive rates and the need for complex interpretation (see Supplementary guide).
- Skin biopsies are reserved for uncertain cases as they offer little diagnostic value in routine AD cases.[4]

Morphology and distribution of AD lesions

The morphology of AD varies with disease chronicity:

- **Acute lesions**: typically present as erythema in lighter skin tones, and may appear violaceous, grey or brown in darker skin tones.[1] Lesions may manifest as papules, vesiculopapules and plaques, often accompanied by exudates or crusting.[7]
- **Chronic lesions** (developed from persistent scratching): may present as lichenified plaques or prurigo nodules.[7] After lesions have healed, patients may present with post-inflammatory hyper- or hypopigmentation.[6,8]

Scan or click the QR code to access

Photo repository on common presentation of AD in different skin tones

Supplementary guide on validated diagnostic criteria, severity assessment tools and mitigation strategies for common AD triggers

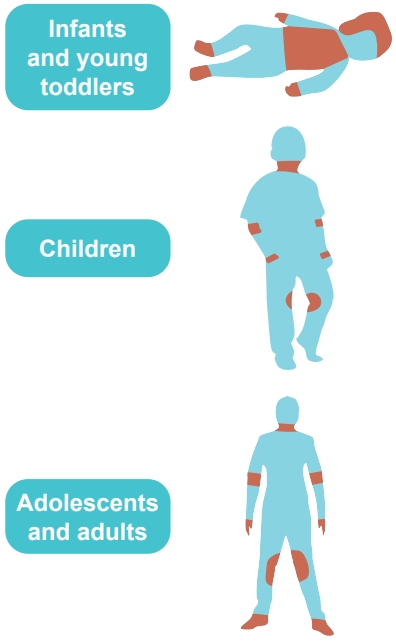
The distribution of lesions typically follows an **age-related** and **symmetrical pattern** (Figure 2), though there may be some overlap between age groups:

- Infants and young toddlers (under 2 years): scalp and face, commonly spreading to trunk and extremities (often extensor involvement).[6,7]
- Children (2–12 years): neck and flexural areas, with higher prevalence in periorbital and auricular areas, and the ventral aspects of wrists (less cheeks, chin or forehead involvement than infants).[5-7]
- Adolescents (13–17 years) and adults: predominantly flexural areas in addition to face, neck and distal extremities. Distribution may be less typical and appear in localised areas like nipples, hands, or feet.[5,6]

It is important to note that, AD may present differently in Asian patients compared to non-Asians, including **more psoriasiform variants (presenting with clearer demarcation, more prominent scaling and lichenification)**.[8–10] They also show increased prevalence of **prurigo nodularis** and follicular prominence[11], with **greater involvement of truncal, extensor, scalp and auricular areas**.[5]

Several conditions can mimic AD and require careful differentiation, including irritant and allergic contact dermatitis, psoriasis, seborrheic dermatitis, scabies or dermatophyte infections.[1]

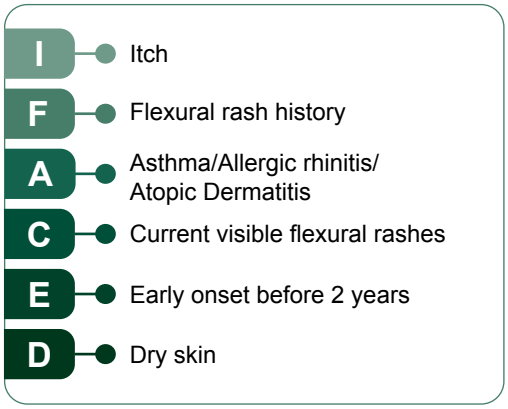
Figure 2. Age-related distribution of AD lesions diagram



Diagnostic criteria

The clinical manifestations of AD can vary widely due to factors such as patient age, disease severity and chronicity, making it difficult to adopt a single set of diagnostic criteria as gold standard in routine clinical practice. Among the most validated and widely cited are the United Kingdom Working Party (UKWP)[12–14] and Hanifin and Rajka[15] criteria, though both have limitations as detailed in Supplementary guide and should be used in conjunction with comprehensive assessment. Given some practical utility of the UKWP criteria in primary settings, this guideline has developed the I-FACED acronym (Figure 3) to support recall and facilitate application in clinical practice.

Figure 3. I-FACED acronym for UKWP criteria



Recommendation 2 **Assess AD severity based on extent, frequency and intensity of clinical manifestations, and patient- or caregiver-reported impact on quality of life.**

AD severity can be classified using criteria adapted from NICE guidelines[16] (Table 1) and comprehensively assessed using a practical checklist derived from validated scoring tools such as EASI, SCORAD and DLQI (Figure 4). Although these tools allow detailed assessment, time constraints and patient burden may limit their routine use. Therefore, they are positioned as optional tools (Supplementary guide) that may be used for detailed assessment when necessary.

Table 1. Adaptation from NICE severity definition

	Skin and physical severity	Impact on quality of life
Mild	Areas of dry skin, infrequent itching (with or without small areas of inflammation)	**Little impact** on everyday activities, sleep and psychosocial wellbeing
Moderate	Areas of dry skin, frequent itching, inflammation (with or without excoriation and localised skin thickening)	**Moderate impact** on everyday activities and psychosocial wellbeing, frequently disturbed sleep
Severe	**Widespread areas** of dry skin, persistent itching, inflammation (with or without excoriation, extensive skin thickening, bleeding, oozing, cracking and alteration of pigmentation)	**Severe limitation** of everyday activities and psychosocial functioning, nightly loss of sleep

Figure 4. Practical checklist for assessing AD severity

Clinical features

- ☐ Distribution and extent of area involved
- ☐ Erythema, or pigmentary changes (in darker skin tones)
- ☐ Oedema/papulation
- ☐ Excoriation
- ☐ Oozing/crusting
- ☐ Lichenification
- ☐ Dryness

Symptoms

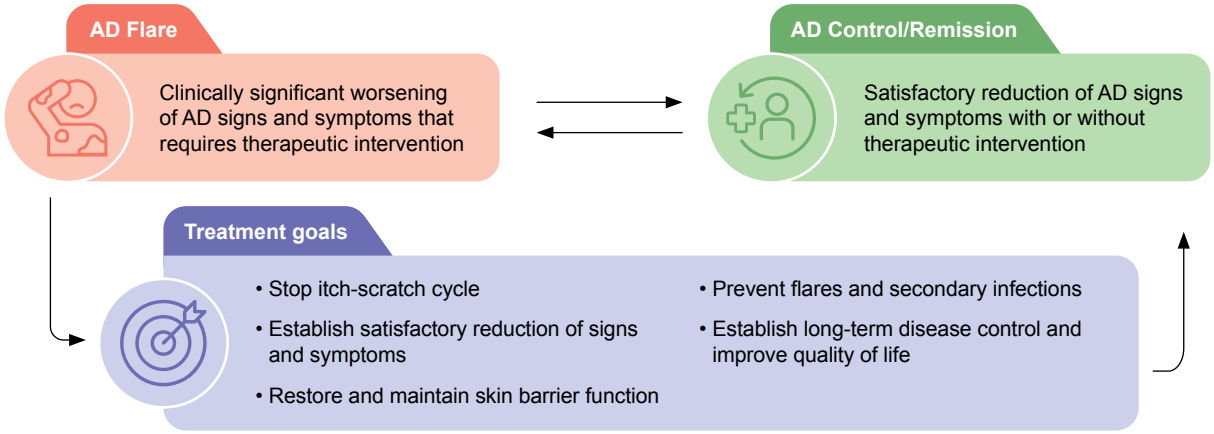
- ☐ Itch intensity and frequency
- ☐ Sleeplessness (quality, frequency of night waking)

Impact on quality of life

- ☐ Negative impact on daily activities, school/work, social, emotional wellbeing
- ☐ Negative family impact (if any)

Once severity is assessed, the goals of treatment are to control flares and to maintain remission (Figure 5).

Figure 5. From flare to control through specific treatment goals



Use clear and empathetic communication when explaining the disease course and management. Setting realistic expectations helps build trust and encourages adherence to treatment plans which improves quality of life for both patients and their caregivers.

Communication tips to educate patients and caregivers

- Introduce AD as a **chronic disease** that is manageable with consistent care
  - **Disease severity and triggers can vary between individuals and over time**
  - The aim of treatment is to **control flares and maintain remission**, as opposed to providing a permanent cure
- Stress **daily skin care**, with regular liberal use of moisturisers, even when skin appears clear
- Learn what may **trigger** a flare and avoid them when feasible
- Stop the itch-scratch cycle (Figure 6) which worsens inflammation and delays healing
  - Manage scratching by keeping nails short and clean. If possible, wear cotton gloves at night, and rub or cool AD lesions rather than scratching to relieve itch[6]
- **Recognise AD flares** and respond using appropriate topical anti-inflammatory treatments while maintaining daily skin care
  - Explain the role of topical anti-inflammatory treatments, including application techniques (e.g. fingertip unit method for topical corticosteroids)
  - Address common fears (see notepad on ‘Addressing steroid phobia’) and assure safety when used correctly
  - Stress the need for treatment adherence
- Seek timely treatment for **signs of secondary infections** such as increased pus, pain, fever, rapidly spreading redness or blistering lesions
- Return if they experience worsening symptoms, frequent flares, treatment side effects or signs of infection

Figure 6. Itch-scratch cycle

Scan or click the QR code to access patient education leaflets on AD

For adults

For caregivers of young children

For common questions on AD

Avoidance of triggers

Recommendation 3 **Review potential triggers for AD and advise on how to minimise exposure.**

Provide tailored counselling to address factors that may trigger or worsen AD. Complete elimination may be feasible for some triggers (e.g. contact allergy), but impractical for others like dust or pollutants, which can typically be minimised rather than avoided entirely. Suspected food allergies require careful evaluation before dietary modifications, to avoid nutritional deficiencies from unnecessary food elimination. Common triggers are outlined below (Figure 7). Refer to Supplementary guide for mitigation strategies.

Figure 7. Common triggers

Environment factors

- Extreme hot, cold or dry weather conditions
- House dust mites
- Insect bites
- Pet dander
- Pollutants (e.g. haze)

Personal or Lifestyle factors

- Stress
- Rough fabrics
- Smoking
- Skin infection
- Chemical irritants
- Intercurrent illness
- Food allergy

Pharmacotherapy for mild and moderate AD

Recommendation 4 **For patients with mild or moderate AD, advise liberal moisturiser use as baseline therapy, and**

- **Prescribe topical corticosteroids as first-line anti-inflammatory treatment for active lesions**
- **If a non-steroidal alternative is required or preferred, consider topical calcineurin inhibitors (as second- line) or topical phosphodiesterase 4 inhibitors (as third-line).**

Moisturisers as baseline therapy

Moisturisers are the cornerstone of AD management and recommended for all patients with AD, regardless of disease severity. They are generally well-tolerated, safe and provide multiple benefits such as preventing skin dryness and restoring skin barrier, thereby reducing AD signs and symptoms and decreasing flare frequency.[17]

Moisturiser prescribing essentials

Selection and Quantity

- ✓ Choose fragrance-free, hypoallergenic formulations[1]
- ✓ Match formulation choice to needs, climate and personal preference (e.g. lighter lotions or creams for daytime use and thicker creams or ointments for night or colder environment)
- ✓ Consider cost and accessibility as it affects long-term adherence
- ✓ Ensure adequate quantities as under-prescription often results in insufficient use (estimated weekly requirements: at least 125g for infants, 250g for children and 500g for adults)[18]

Application technique and frequency

- ✓ Apply liberally multiple times daily (e.g. at least twice daily), continuing during remissions, with increased frequency during active flares[4]
- ✓ After a lukewarm shower, gently pat skin dry and apply within minutes to lock in hydration[17,19]
- ✓ Can be applied before or after topical anti-inflammatory treatments, with a 5-10 minute interval between applications to maximise absorption and reduce dilution (unless package insert specifies otherwise)
- ✓ Although more research is needed to confirm optimal waiting intervals between application,[1] prioritise feasibility and patient compliance since waiting can be difficult for younger children
- ✓ When using multiple products, apply in order of consistency: gels, lotions, creams, then ointments

Topical anti-inflammatory treatment for active AD lesions

For active AD lesions or flares, prescribe a suitable topical anti-inflammatory **in addition** to moisturisers. **Topical corticosteroids (TCS) remain the first-line treatment** due to their strong and well-established efficacy. The choice of TCS (vehicle and potency), should be tailored to the anatomic site and clinical severity (Table 2), balancing therapeutic benefit with potential local side effects (skin atrophy, striae, telangiectasia).[6] Risks of local side effects increases with higher potency TCS, application to thin-skinned areas, occlusion, or long-term continuous use.

Topical calcineurin inhibitors (TCIs) (e.g. pimecrolimus and tacrolimus) are alternatives if non-TCS options are preferred. The topical anti-inflammatory potency of TCIs is broadly comparable to low- and moderate-potency TCS[20,21] (see drug table for more details). TCIs are generally well tolerated except for a transient burning or stinging sensation upon application. Despite FDA black box warnings based on high doses of systemic calcineurin inhibitors in animal studies, long-term studies have found no evidence for increased cancer risk in humans.[22,23]

Topical phosphodiesterase-4 inhibitors (PDE4i), such as crisaborole, are a newer class of non-TCS anti-inflammatory therapy. They work by down-regulating inflammatory cytokine production and reducing skin inflammation, and are effective in reducing AD signs and symptoms compared to placebo.[24] Recent head-to-head trials showed moderate potency TCS had greater efficacy in reducing AD signs and symptoms compared to crisaborole,[25] while differences between crisaborole and TCIs were not statistically significant[25–29] (see drug table and Evidence-to-Recommendation summary of findings). **Given the limited evidence on PDE4i efficacy versus TCS and TCIs; similar tolerability issues and cost concerns; and it being less accessible in primary care settings, consider PDE4i when conventional treatments (TCS or TCIs) are unsuitable or not tolerated**. Topical PDE4i are generally well tolerated except for a transient burning or stinging sensation.

Scan or click the QR code to access drug table of topical anti-inflammatories

Table 2. Factors to consider when choosing topical anti-inflammatory treatments

	TCS	TCIs*	Topical PDE4i
Age limit	No age limit	Pimecrolimus 1% cream: ≥3 months Tacrolimus 0.03% ointment: ≥2 years; 0.1% ointment: ≥16 years	Crisaborole 2% ointment: ≥3 months
Sensitive skin areas (face, neck, flexures, groin)	Use low potency for shortest possible duration	Suitable	Suitable
If patients present with fear or concerns about TCS side effects	Address misconceptions and emphasise safety of appropriate use (see notepad on 'Addressing steroid phobia')	Suitable if steroid phobia cannot be resolved	Suitable if steroid phobia cannot be resolved
Product formulation	Various formulations (solution, lotion, cream, ointment); in tubes and tubs	Cream/ointment; smaller tubes	Ointment; smaller tubes
Cost/availability	Low cost, generics widely available	Higher cost, less widely available	Higher cost, not widely available

*Individual medications in the TCIs class are listed according to age limit

Scan or click the QR code to access HealthHub content for patient education leaflets on application technique

On moisturisers *On TCS* *On TCI*

While disease severity, patient age and anatomical site (Table 3) are useful for guiding treatment selection, clinicians should also consider other patient factors, treatment history, cost and availability. Clinicians may refer to the comparative efficacy drug table in conjunction with these recommendations to inform treatment decisions.

Table 3. Initial topical treatment selection guidance

Age	Topical anti-inflammatory	Site	Mild AD	Moderate AD	Severe AD
Adult (>12 years)	TCS (First-line)	Body	Low to moderate potency	Moderate to high potency	High to ultra-high potency[c]
		Thin skin[a]	Low potency for shortest duration possible		
	Non-TCS[b] (Only if TCS unsuitable)	Any site	Pim / PDE4i	Pim / Tac 0.03 / Tac 0.1 / PDE4i	Tac 0.1 (≥16 years)
Children (≤12 years)	TCS (First-line)	Body	Low potency	Moderate potency	High potency[c]
		Thin skin[a]	Low potency for shortest duration possible		
	Non-TCS[b] (Only if TCS unsuitable)	Any site	Pim / Tac 0.03 / PDE4i		

Mod, moderate; PDE4i, topical phosphodiesterase 4 inhibitor which is currently only crisaborole 2% ointment; Pim, pimecrolimus 1% cream; Tac 0.03, tacrolimus 0.03% ointment; Tac 0.1, tacrolimus 0.1% ointment; TCS, topical corticosteroids

[a] Thin skin areas include the periorbital region, face, scalp, neck, flexures, and groin.

[b] Minimum approved ages for pimecrolimus 1% cream is ≥ 3months; tacrolimus 0.03% ointment is ≥2 years; 0.1% ointment is ≥16 years; crisaborole 2% ointment: ≥3 months. Individual medications in the TCIs class are listed alphabetically, not in order of preference.

[c] While severe AD may initially require the higher potency TCS, the concurrent use of systemic therapies or phototherapy may permit the stepping down to lower potencies as adjunctive therapy, under specialist advice.

Addressing steroid phobia

A local qualitative study revealed that most patients with steroid phobia placed great value on their own experiences and expressed the need to be emotionally validated and understood. Many reported deteriorating doctor-patient relationships when their concerns were dismissed.[30]

Talking points when addressing patients' steroid phobia:

- **Validate concerns/perspectives** and acknowledge their lived experiences; avoid giving perfunctory reassurances without further explanation[30]
- **Manage expectations**, including concerns about 'lack of sustained improvement' with TCS use, by explaining AD is a relapsing, chronic condition that will require consistent ongoing management as it cannot be cured[30]
- **Build rapport over time** as repeated, empathetic conversations are often more effective than one-time education sessions (e.g. when patients refuse TCS, healthcare workers should try to preserve opportunities for future engagement and prevent patients from completely withdrawing from treatment)[30]
- **Reassure patients** that treatment involves a personalised approach (e.g. using the most appropriate potency and duration under close monitoring)
- **Distinguish** between TCS and oral corticosteroids (OCS) use as patients may have misconceptions that they are equivalent. Reassure patients that the systemic absorption of TCS is much lower than OCS, and TCS is generally safe for use under medical supervision. Contrast this to OCS where the potential for adverse effects with indiscriminate use is higher and indications for use are much more limited (see Recommendation 7).

If steroid phobia persists despite repeated counselling efforts by primary care professionals, consider referring to a dermatologist, particularly in patients with moderate or severe AD.

Topical JAK inhibitors

Topical JAK inhibitors such as ruxolitinib are a novel class of non-steroidal topical anti-inflammatory with demonstrated efficacy in reducing AD severity.[20] Common side effects include nasopharyngitis and upper respiratory tract infections.[31] All JAK inhibitors carry a black box warning due to associations with increased risk of cancer, thromboembolism, adverse cardiovascular events and serious infections. Safety data is available up to 52 weeks, but long-term data beyond this period remains limited.[1,31,32] The medication is currently not registered in Singapore for use in AD.

While topical anti-inflammatories are the main treatment options for AD, **other adjunctive therapies exist**, although the evidence supporting their effectiveness is generally limited.

Oral antihistamines

Oral antihistamines have limited effectiveness as AD-related itch is predominantly driven by non-histamine mediated pathways.[33] Studies demonstrate weak antipruritic effects of oral antihistamines (chlorpheniramine, cetirizine, levocetirizine, loratadine, fexofenadine, and hydroxyzine) for AD.[34–36] However, they may provide benefits as adjuvant therapy in concurrent urticaria or allergic rhinitis (where histamine plays a key role). Additionally, first-generation antihistamines may be more useful than second-generation antihistamines for patients experiencing itch-induced sleep disturbances due to their more sedating properties, though this benefit must be weighed against potential anticholinergic side effects including drowsiness, dry skin and mouth, urinary retention and increased fall risk.[34]

Wet wrap therapy

Wet wrap therapy (WWT) may be considered for patients with moderate or severe AD refractory to standard application of topical treatments. While evidence is limited, studies of WWT suggest some efficacy in improving AD severity.[1,37,38] Moisturisers or TCS are suitable topical agents for use with WWT (Table 4). Safety data for the use of TCIs and topical PDE4 inhibitors under occlusion with WWT remains limited, therefore specialist dermatological advice is essential before using these agents in WWT.[1,16] Commonly recommended duration ranges from one hour to maximum overnight.[1,17]

Scan or click the QR code for WWT patient leaflets

Table 4. Considerations for implementation of WWT

Assessing patients' suitability for WWT	Suitable for: • Very dry skin • More severe AD • Limbs heavily scratched at night • Flares not controlled by standard application of topical treatment	Less suitable for: • Lesions in sensitive areas • Lesions in areas that are hard to wrap • Patients or caregivers unable to adhere to treatment regimen • Patients or caregivers unable to understand wrapping techniques Avoid use in: • Excessively 'weepy' lesions or if there are other signs of secondary infections (could be considered after appropriate management of infection with close monitoring)
Potential adverse effects	Generally well-tolerated with mild local adverse effects like skin maceration, folliculitis or other skin infections.[1,38] Though TCS efficacy increases under occlusion, studies indicated no significantly higher risk of steroid-related systemic adverse effects.[38]	
Resources required	Additional time and nursing support to educate patients or caregivers on proper wrapping techniques and monitoring of adverse effects	

Special populations

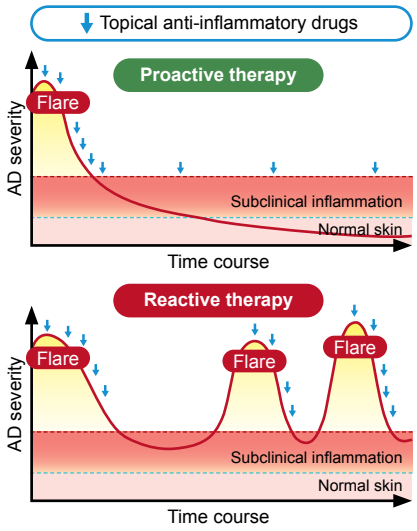
Adolescents (13–17 years)	• Management is similar to that for adults, but give additional consideration to the psychosocial impact, as poor cosmesis due to AD can greatly diminish adolescents' self-esteem[6, 39]	• Adherence is often poor[6] due to increasing autonomy in self-management,[40] thus shared decision-making and education are essential to improve adherence and establish lifelong self-management skills[39, 40]
Pregnant/ breastfeeding individuals	• **Moisturisers** are a key treatment for all pregnant or breastfeeding individuals with AD • **TCS** are generally safe in this group, when used appropriately due to limited systemic absorption of TCS, [1,7,19] but avoid application to nipple and areolar areas immediately before feeding and clean breasts before breastfeeding[41] • **TCIs** can be used with the same breastfeeding precautions as TCS, since studies suggest little to no systemic absorption by the mother[1,19]	• **Topical PDE4 inhibitors** should be avoided due to limited safety data, unless safer alternatives are inadequate[26] • **Antihistamines** have limited effectiveness for AD-related itch but may be considered for symptom relief, when indicated and benefits outweigh risks, particularly for concurrent allergic conditions or sleep disturbances[41] • Long-term **oral corticosteroids** should not be used due to increased complications including gestational diabetes. Only short courses should be used (see Recommendation 7).[19] • **General principle**: Use the lowest effective dose for the shortest duration necessary.

Recommendation 5

For patients with recurring AD flares (e.g. 2–3 flares/month), prescribe proactive therapy of topical anti-inflammatory treatments to areas of skin prone to flare recurrences.

Proactive therapy is a prevention-focused strategy that reduces flare frequency and maintains long-term control by **continuing intermittent topical anti-inflammatory treatment** on recurrently relapsing skin areas, **even after initial AD flare has been controlled**. Studies of patients with moderate-severe AD and recurring flares showed that **proactive therapy significantly reduced relapse risk with generally low risk of adverse effects, compared to reactive treatment** (Figure 8).[28,37,42] Based on expert experience, patients with recurring AD flares (2–3 flares per month) affecting the same specific locations, regardless of overall disease severity, will likely benefit from proactive therapy.

Figure 8. Disease control patterns with proactive versus reactive therapy



Factors to consider when prescribing proactive therapy (Figure 9):

Patient suitability assessment:

- Unsuitable for patients with known adherence issues, steroid phobia (if using TCS), pre-existing adverse effects even with reactive therapy

Patient education:

- Adherence to prescribed proactive therapy regimen
- Flare management - If AD flares occur, increase to daily application until clear, then resume proactive regimen
- Seek medical advice for persistent/unexplained recurring flares

Monitoring and assessment:

- Review every 3–6 months for proactive therapy appropriateness. Consider monthly monitoring if adherence or adverse effects concerns exist
- Assess for efficacy (flare frequency and severity), adverse effects, and patient's adherence at each visit

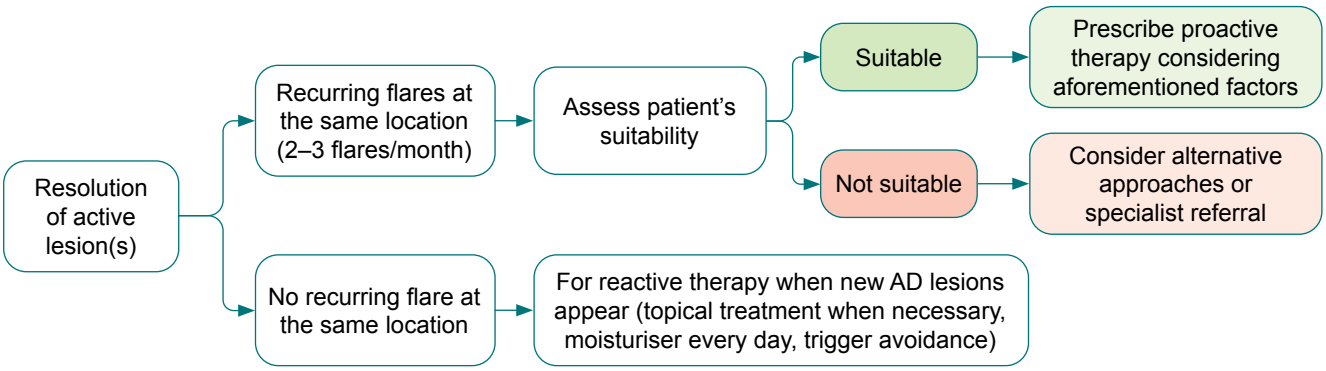
Proactive treatment regimen:

- Select appropriate topical anti-inflammatory based on medication history, AD severity and location
- If TCS or TCI are chosen, apply **two times a week**[1]
- If topical PDE4 inhibitor is chosen, **apply once daily**[43] (currently limited evidence for twice weekly application[44])
- Continue daily moisturiser use

Discontinuation of proactive therapy:

- No further flares in treated areas for 3–6 months
- If significant adverse effects develop during proactive therapy
- Patient preference

Figure 9. Flowchart to approach proactive therapy



For patients with moderate or severe AD refractory to topical treatment, phototherapy and systemic options including oral conventional immunosuppressants, oral JAK inhibitors, and monoclonal antibodies have been shown to improve AD severity (no treatment hierarchy implied).[45, 46] Patients requiring such therapies should be referred to specialists treating severe AD, such as dermatologists, allergists or relevant specialists.

Role of complementary therapies

Topical anti-inflammatory treatments remain the mainstay of AD management. Clinicians might be asked about the efficacy of complementary therapies such as probiotics[47–49] and polynucleotide injections.[50] These therapies currently have limited evidence for routine use in AD treatment. They may incur additional cost without established clinical benefit in AD management and should not replace established topical therapies. Patients wishing to proceed with these complementary therapies should be counselled adequately about the additional costs and limited evidence of clinical benefit.

Recommendation 6 **For patients with inadequate treatment response, assess and address possible factors before modifying treatment.**

When evaluating inadequate treatment response, defined as a lack of satisfactory improvement in AD signs, symptoms or impact on quality of life after 2 weeks, clinicians should systematically consider different factors (see below) before modifying treatment.

Confirm diagnosis and exclude complicating factors	Assess treatment-related factors	Assess patient-related factors
• Reassess diagnosis to rule out mimickers (e.g. contact dermatitis, psoriasis) • Exclude and treat secondary infections (e.g. *S. aureus*/ eczema herpeticum) • Review trigger factors	Assess: • Whether potency, formulation, frequency, and duration of topical treatment was prescribed appropriately for severity and site of AD lesion(s) • Adverse effects • Intolerance (patient-reported discomfort or inability to tolerate treatment)	Assess: • Adherence of prescribed treatment • Moisturiser usage • Application technique (see page 7 for patient education materials) • Steroid phobia: address if present (see section on 'Addressing steroid phobia')

Most patients with mild and moderate AD can be effectively managed in primary care.

Consider **specialist referral** for the following situations:

- Uncertain diagnosis (including suspected allergic contact dermatitis with unidentifiable allergen requiring patch testing)
- Unsatisfactory response to topical treatments in primary care such as:
  - Flare persisting for more than 2 to 4 weeks after appropriate treatment
  - Flares still occurring frequently after 3–6 months of proactive therapy
  - Significant adverse effects or poor tolerance to topical treatment(s)
- Extensive (e.g. >10%) body surface area affected
- Significant impact on quality of life (including social or psychological problems)
- Severe complications such as skin infections requiring intravenous antibiotics and antivirals (e.g. severe cellulitis, eczema herpeticum), and erythrodermic flare – **refer to emergency department**

Recommendation 7

Do not routinely give oral corticosteroids for AD, except as a short course for:
- **Rescue therapy for acute, severe flares, or**
- **Bridging therapy to systemic treatment**

Oral corticosteroids (OCS) are not recommended for routine use in the mild and moderate management of AD. Although they provide rapid symptom relief, **benefits are short-lived**, and **disease activity typically rebounds after discontinuation**.[1,19,51] Long-term or repeated use carries significant risks such as infection, hypertension, glucose intolerance, weight gain, gastritis, reduced bone mineral density, and adrenal suppression.[52] Reliance may also delay the initiation of more appropriate and effective long-term treatment.

OCS is strongly discouraged in children due to the additional risk of growth retardation.[52,53]

Short courses of OCS may be considered for rescue therapy during acute, severe flares - defined as ***episodes with widespread inflammation, persistent itching and significant impairment of quality of life*** *(following NICE severity definition)* - or as bridging therapy while initiating slower-acting systemic treatments.[51,54] **When prescribed, clinicians must communicate temporary benefits and risks through shared decision-making, emphasising adherence to prescribed duration and need for effective long-term alternatives.**

Practical prescribing considerations for OCS

- Always taper the OCS dose to prevent rebound flares whilst ensuring concurrent optimisation of appropriate topical treatments. Start OCS, e.g. oral prednisolone at **0.5–1 mg/kg/day**[19] and **reduce gradually every 3–5 days over a total duration of 7–14 days**.[54] An example prescription for a 50–60 kg adult would be 30 mg OD for 3 days, 20 mg OD for 3 days, 10 mg OD for 3 days, 5 mg OD for 4 days, then stop.
- Exercise caution through closer monitoring in patients with **comorbidities such as diabetes** (OCS may worsen hyperglycaemia), **hypertension**, **cardiovascular disease or advanced chronic kidney disease** (for OCS-induced fluid retention and elevated blood pressure)
- Instruct patients to take it after food or prescribing a proton pump inhibitor (e.g. omeprazole) or H2 receptor antagonist (e.g. famotidine) to reduce risk of gastric irritation

OCS, oral corticosteroids; OD, once daily

Management of AD with secondary bacterial infection

Patients with AD are predisposed to skin infections due to multiple factors such as skin barrier. skin barrier disruption, inflammation, bacterial colonisation and dysbiosis of skin flora.[55] Scratching also disrupts the skin barrier and may allow entry of pathogens. It may be challenging to distinguish between an AD flare and infected AD as both can present with erythema, weeping, and worsening skin symptoms.[56]

Recognising AD with secondary bacterial infection

AD with secondary bacterial infection typically presents as lesions with **honey-coloured crusting, oozing or weeping, pain, failure to respond to standard AD therapy or rapidly worsening AD**.[56] Systemic signs such as **fever and malaise** may indicate more severe infection.[56] Most cases can be managed similarly to **impetigo**. Less commonly, secondary **cellulitis or skin abscesses** may develop and require more intensive treatment.[55]

Scan or click the QR code to access photo repository on secondary infection of AD

Bacterial infection in AD is mostly commonly caused by **Staphylococcus aureus** (*S. aureus*), followed by **Streptococcus pyogenes** (*S. pyogenes*).[56] **Skin swabs are not necessary** in most cases, as bacterial colonisation is common in AD patients regardless of infection status,[57] and routine swabbing of AD patients with suspected infection could lead to inappropriate antibiotic prescribing.[56] If infection worsens, consider a skin swab to check for other causative organisms or resistant strains.[19,56]

Recommendation 8

For secondary infection of AD, continue patient's topical anti-inflammatory treatments alongside appropriate antimicrobial agents.

For this ACG's management of secondary bacterial infection of AD, antimicrobial agents refer to either antiseptics or antibiotics.

In general, the patient's anti-inflammatory treatments (TCS or TCI) should be continued alongside appropriate antimicrobial agents during episodes of infected AD, including severe viral infections such as eczema herpeticum.[19,56] Both TCS and TCIs have been shown to reduce *S. aureus* colonisation, increase microbial diversity, and reduce scratching by managing the underlying inflammation.[55] Evidence of effectiveness for antimicrobial agents (antibiotics or antiseptics) in combination with topical anti-inflammatories in infected AD remains limited. For topical antibiotics, studies show significant reduction in *S. aureus* isolation rates, but only modest improvements over TCS alone in overall disease signs and symptoms.[58]

Clinicians should weigh the need for antimicrobial treatment against the risk of contributing to antimicrobial resistance. **AD flares can clinically mimic skin infection**,[56] making it difficult to distinguish between inflammatory flares and actual bacterial infections. As such, infections may be incorrectly diagnosed when none is present, resulting in unnecessary antimicrobial prescribing. Clinicians must carefully assess whether an infection is truly present before prescribing antimicrobials.

When the decision to treat as a secondary AD infection has been reached:

- **Topical antiseptics may be used to reduce microbial load**, although resistance may develop with prolonged use. Evidence supporting their use in infected AD is limited and derived from a few small trials.[17, 21, 58] Staphylococcus resistance to antiseptics is emerging (to triclosan, and to a lesser extent to chlorhexidine and octenidine, whilst povidone-iodine has limited evidence of antimicrobial resistance).[59,60] Nevertheless, antiseptic resistance is less common than antibiotic resistance.
- **Topical antibiotics, if indicated, should be used judiciously**. When antibiotics are necessary, select narrow-spectrum agents with appropriate duration (e.g. 5 to 7 days); widespread, prolonged or indiscriminate antibiotic use escalates antibiotic resistance. Global surveillance data indicate only 80% fusidic acid susceptibility and 98% for mupirocin.[61] For mild impetiginised AD, topical fusidic acid may treat *S. aureus* and *S. pyogenes* skin infections, while topical mupirocin should be reserved for confirmed methicillin-resistant *S. aureus* (MRSA) where infection has worsened.[56]
- Dual combination creams (TCS plus suitable topical antimicrobial) may reduce treatment regimen complexity and support patient adherence.
- **Systemic antibiotics** are indicated for severe or widespread infected AD, or when patients are systemically unwell.[56]
- If the response to initial treatment course is suboptimal and symptoms persist, reassess the diagnosis and management plan, and consider escalating care, such as a skin swab to guide targeted antibiotic choice, or referral.[56]

Differences between antiseptics and antibiotics

Antiseptics are topical agents with broad-spectrum activity against bacteria, fungi and some virus through non-specific mechanisms.[36] They have a lower risk of developing resistance due to multiple mechanisms of action.[59] Examples are benzalkonium chloride, clioquinol, chlorhexidine, hydrogen peroxide, povidone-iodine, octenidine and triclosan.

Antibiotics can be delivered in many ways (orally, intravenously, topically, etc) and specifically target bacteria through targeted molecular mechanisms to kill (bactericidal) or inhibit growth (bacteriostatic).[36] This specificity of action makes bacterial resistance development more likely.[59]

Recommendation 9

Avoid triple combination products of TCS, antibiotics and antifungals in AD patients with suspected or clinically evident bacterial infection.

Products containing a combination of TCS, antibiotics and antifungals should be avoided:

Inappropriately high potency TCS: The TCS components in locally available products, such as betamethasone dipropionate 0.05% are typically high-potency formulations. This may be unnecessarily potent for many AD patients, increasing the (avoidable) risk of local side effects such as skin atrophy, especially without proper monitoring.

Limited role of gentamicin: Products containing aminoglycosides like gentamicin, have poor penetration and limited bactericidal activity against *S. aureus* and *S. pyogenes*, without synergistic combinations with beta-lactams.[64,65] As such, they are not suitable as a monotherapy for treating common gram-positive skin infections.

Risk of contact dermatitis: Topical aminoglycosides commonly found in these combinations, such as gentamicin and neomycin, have been shown to cause contact dermatitis in some individuals, potentially worsening the existing skin condition.[64, 65]

Unnecessary antifungal component: Most bacterial infections in AD do not require concurrent antifungal treatment. Their use potentially exposes patients to unnecessary medication, given the limited evidence supporting topical antifungals in AD patients.[66]

References

Click or scan the QR code for the reference list to this clinical guideline

Evidence-to-Recommendation Framework

Click or scan the QR code to view the Evidence-to-Recommendation Framework for the recommendations in this clinical guideline

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The Agency for Care Effectiveness (ACE) was established by the Ministry of Health (Singapore) to drive better decision-making in healthcare by conducting health technology assessments (HTA), publishing healthcare guidelines and providing education. ACE develops ACE Clinical Guidelines (ACGs) to inform specific areas of clinical practice. ACGs are usually reviewed around five years after publication, or earlier, if new evidence emerges that requires substantive changes to the recommendations. To access this ACG online, along with other ACGs published to date, please visit www.ace-hta.gov.sg/healthcare-professionals/ace-repository-for-clinical-guidelines/

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