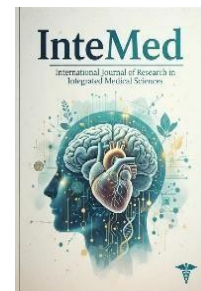




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Review Article

Critical Review of Classical Pathogenesis Disease Biology and Therapeutic Evidence

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Abstract:

Background: *Vicharchika* is described in Ayurveda as a *Kshudra Kushtha* with prominent itching, eruptions, exudation, and altered colour. This symptom pattern often resembles eczematous dermatitis, yet neither term represents a precise translation of the other. Contemporary eczema, particularly atopic dermatitis, includes distinct barrier, immune, microbial, and neural abnormalities.

Objective: To critically relate the classical disease model of *Vicharchika* to current knowledge of eczema and to examine how Ayurvedic and modern treatments can be evaluated without forcing direct biological equivalence between the two systems.

Approach: Classical descriptions and published clinical studies of Ayurvedic interventions were interpreted alongside established research on epidermal barrier failure, type-2 inflammation, neuroimmune pruritus, JAK-STAT signalling, skin microbiota, and contemporary treatment. The review uses conceptual comparison rather than literal translation of Ayurvedic terms into cytokines or molecular pathways.

Synthesis: *Dosha* involvement, disturbance of *Twak* and *Rakta*, altered *Agni*, and continuing exposure to *Nidana* provide the classical framework. Modern research describes a self-reinforcing relationship among barrier injury, immune activation, scratching, and microbial imbalance. Ayurvedic treatment studies involving *Virechana*, *Raktamokshana*, topical therapy, and *Rasayana* report symptomatic benefit, but small samples and variable

methods limit certainty. Integrative investigation should retain standard dermatological care, define the Ayurvedic intervention precisely, and measure severity, itch, quality of life, barrier function, recurrence, and safety.

Conclusion: The most defensible relationship between *Vicharchika* and eczema is one of clinical overlap, not diagnostic identity. Ayurvedic treatment remains promising for research, but reliable conclusions require standardized products and procedures, controlled designs, validated outcome measures, and longer follow-up.

Keywords: atopic dermatitis; eczema; immunopathology; pruritus; *Rakta Dushti*; *Vicharchika*; skin barrier

INTRODUCTION

Chronic inflammatory skin disease affects far more than the visible lesion. Recurrent itching can interrupt sleep, scratching can damage the skin, and changes in appearance may interfere with school, work, relationships, and emotional well-being. Eczema is a broad clinical family rather than one uniform disease, and its members include atopic, contact, nummular, and other dermatitis patterns.

Ayurvedic texts place *Vicharchika* within *Kshudra Kushtha*. Its recognizable cluster includes *Kandu*, *Pidika*, *Srava*, and *Shyava Varna*.¹⁻³ Dryness, roughness, fissuring, and thickening may become more evident when disease is prolonged. These observations overlap with several eczematous presentations, but the breadth of modern eczema makes a simple statement that *Vicharchika equals eczema* scientifically inaccurate.

The biomedical model of atopic dermatitis has also moved beyond a simple view of superficial inflammation. Current evidence describes reciprocal effects among epidermal barrier weakness, type-2 immune activity, altered microbial communities, environmental exposure, and sensory-nerve signalling.^{4,5} This complexity creates a useful opportunity for dialogue with Ayurveda, provided that comparison remains critical and testable.

This review has three purposes: to restate the Ayurvedic pathogenesis of *Vicharchika* in clinically clear language, to summarize relevant mechanisms in eczema, and to propose an evidence-based way to study integrative management. It does not attempt to rename Ayurvedic concepts as modern biomarkers.

SCOPE AND INTERPRETIVE METHOD

The discussion draws on three classical sources, modern reviews and experimental literature on atopic dermatitis, four published Ayurvedic clinical reports, and contemporary therapeutic guidance. Sources were used to examine symptom description, disease mechanisms, treatment rationale, clinical outcomes, and safety. This is a critical narrative review rather than a systematic review or meta-analysis.

Two rules guided interpretation. First, similarities were described at the level at which evidence exists: a clinical symptom was compared with a clinical symptom, and a laboratory pathway with a measured laboratory pathway. Second, a traditional construct was not assigned a molecular equivalent unless it had been investigated directly. These rules prevent an attractive analogy from being mistaken for proof.

THE CLASSICAL CLINICAL MODEL

Recognizing the symptom pattern

The classical description can be read as a pattern rather than a single lesion. *Kandu* indicates prominent itch; *Pidika* denotes raised or eruptive lesions; *Srava* describes moisture or discharge; and *Shyava Varna* refers to a dusky or dark alteration in colour.¹⁻³ Chronic disease may also show *Rukshata*, scaling, fissuring, excoriation, or thickened skin from repeated rubbing. The balance of these features changes across patients and over time.

Dosha and lesion behaviour

The tridoshic description does not imply that every feature has equal intensity. A *Kapha*-predominant presentation may be interpreted through persistence, itch, and exudation; *Pitta* becomes more relevant when redness, heat, burning, or active inflammation dominates; and *Vata* helps explain dryness, roughness, cracking, and chronicity. This functional reading can guide individualized care without claiming that a *Dosha* is a cytokine or a cell type.

Dushya and the place of Rakta

Twak, *Rakta*, *Mamsa*, and *Lasika* are described among the affected *Dushya* in *Kushtha*. In *Vicharchika*, visible alteration of colour and texture, recurrent inflammation, itch, and discharge make *Twak* and *Rakta* especially important.^{1,2} *Rakta Dushti* is a broad pathological idea within Ayurveda. It should not be reported as though it were a laboratory value or a synonym for one inflammatory mediator.

Agni Nidana and recurrence

Classical reasoning gives importance to unsuitable diet, incompatible combinations, irregular habits, and repeated exposure to causative factors. Through disturbance of *Agni*, these influences are understood to promote *Dosha Prakopa*, affect susceptible tissues, disturb channels, and permit localization in the skin. Clinically, continuing exposure helps explain why a temporary improvement may be followed by another flare.

A concise sequence is: continued *Nidana* → disturbed *Agni* and aggravated *Dosha* → involvement of *Twak* and *Rakta* → local expression as itch, eruptions, oozing, dryness, or discolouration. This is an Ayurvedic model of pathogenesis. *Ama* should not be presented as endotoxin, oxidative stress, or a cytokine without direct evidence.

CONTEMPORARY DISEASE BIOLOGY

Barrier failure as an initiating and sustaining factor

Healthy epidermis limits water loss while restricting the entry of irritants, allergens, and microorganisms. Changes in epidermal lipids and proteins, including filaggrin, reduce that protective function.⁶⁻⁸ The result is greater transepidermal water loss, dryness, and easier penetration of environmental substances. Once inflammation begins, cytokines can further suppress barrier-protein expression. Barrier damage and immune activation therefore amplify one another rather than occurring as independent events.

This reciprocal process provides the biological basis for routine moisturization. Barrier care is not merely cosmetic: it reduces dryness and may lower exposure of deeper tissue to external triggers. It also explains why anti-inflammatory treatment alone may be followed by relapse if the barrier remains poorly supported.

Type 2 immunity and targeted pathways

Type-2 inflammation is prominent in many patients with atopic dermatitis. IL-4 and IL-13 contribute to allergic inflammation and interfere with normal epidermal differentiation.^{5,9} The efficacy of therapies directed at these pathways supports their clinical relevance.¹⁰ This is stronger evidence than an association alone because modifying the pathway can modify disease activity. Several cytokine signals relevant to atopic dermatitis are transmitted through JAK-STAT pathways. This includes signalling linked with IL-4, IL-13, and IL-31.¹³ JAK inhibition has consequently become an option for selected moderate-to-severe disease. These advances illustrate the value of connecting a defined mechanism with a measurable response, an approach that future Ayurvedic trials can also adopt.

Neuroimmune itch and the scratch response

Pruritus is not explained adequately by histamine alone. IL-31 receptors on sensory neurons and direct communication between immune pathways and nerve fibres contribute to persistent itch.^{11,12} Scratching then disrupts the epidermis, increases inflammatory signalling, and provokes further itch. This feedback loop helps account for excoriation, lichenification, pigmentary change, and secondary infection. The prominence of *Kandu* in the classical description is therefore clinically important. The comparison must stop at that level unless IL-31 or another pathway is measured: *Kandu* is a symptom construct and should not itself be labelled an IL-31 state.

Microbial imbalance

Active atopic dermatitis is often accompanied by lower microbial diversity and greater colonization by *Staphylococcus aureus*.¹⁴ Barrier disruption makes colonization easier, while microbial products can intensify inflammation. The microbiome, epidermis, and immune response may therefore form another reinforcing cycle. Standardized topical Ayurvedic products could be studied in this context, but antimicrobial claims require direct microbiological testing.

CLINICAL PARALLELS AND THEIR LIMITS

Table 1 maps observable features to domains that can be measured in contemporary research. It deliberately avoids declaring the paired concepts biologically identical.

THERAPEUTIC REASONING

Classical observation	Measurable contemporary domain	Required caution
<i>Kandu</i>	Patient-rated pruritus and scratching burden	A symptom score does not identify one cytokine pathway
<i>Pidika</i>	Morphology and distribution of papular or vesicular lesions	Several dermatitis subtypes can produce similar lesions
<i>Srava</i>	Exudation, crusting, and evidence of secondary infection	Oozing is a clinical sign, not a diagnosis by itself
<i>Rukshata</i>	Xerosis, hydration, and transepidermal water loss	Dryness may reflect barrier failure from different causes
<i>Shyava Varna</i>	Post-inflammatory pigmentary alteration	Colour change varies with skin tone and chronicity
<i>Daha</i>	Burning, erythema, and active inflammatory symptoms	The feature cannot be equated directly with <i>Pitta</i> or one mediator
Repeated scratching	Excoriation, lichenification, and itch-scratch behaviour	Clinical correspondence does not establish molecular equivalence

Remove avoidable aggravating influences

Nidana Parivarjana begins with identifying exposures that repeatedly precede or worsen disease. The practical enquiry may include soaps, occupational irritants, fabrics, climate, sweating, scratching behaviour, and a carefully assessed dietary history. Modern care similarly removes confirmed irritants or allergens. Broad food restriction is unsafe, particularly in children, when allergy has not been established clinically.

Match treatment to the lesion and the patient

Shamana treatment is individualized according to dominant features, involved tissues, chronicity, constitution, and tolerance. Ayurvedic dermatology commonly discusses *Nimba*, *Khadira*, *Guduchi*, *Manjistha*, *Sariva*, *Haridra*, *Patola*, and *Aragvadha*. Mention in traditional practice is not proof that every product containing these drugs is effective. Botanical identity, plant part, preparation, dose, and quality must be specified. Topical treatment should also follow lesion morphology. A wet and exudative surface may require a different vehicle and strategy from a dry, fissured, or lichenified plaque. Irritation, allergy, contamination, and worsening infection must be monitored; the label herbal does not guarantee local tolerance.

Evidence concerning Shodhana

Classical management of *Kushtha* may include *Shodhana* after considering strength, disease stage, and *Dosha* predominance. *Virechana* is often selected when *Pitta* and *Rakta* involvement is judged prominent. Kaur and Chandola reported clinical improvement and a possible reduction in recurrence after *Virechana Karma*.¹⁵ Talekar et al. also reported improvement with *Trivrita* powder and *Aragvadha Patra Lepa*.¹⁶ These studies justify further investigation, but their size and methods do not establish definitive efficacy.

Raktamokshana and procedural safety

The classical emphasis on *Rakta* provides the rationale for considering *Raktamokshana* in selected patients. Raval and Thakar reported symptomatic improvement after *Jalaukavacharana* and *Siravedhana*.¹⁷ Both procedures are invasive. Anaemia, coagulopathy, anticoagulant use, local infection, impaired healing, and other contraindications must be assessed, and sterile technique with defined adverse-event reporting is essential.

Long-term control and Rasayana

A flare-based approach is incomplete when disease repeatedly returns. A study of *Rasayana*-based care following initial management reported improvement with less recurrence.¹⁸ The result is clinically relevant because maintenance is also central to contemporary eczema care. Confirmation requires a controlled design with an explicit

definition of recurrence and observation long enough to distinguish sustained control from temporary remission.

CONTEMPORARY STANDARD CARE

Regular emollient use, gentle cleansing, and reduction of avoidable irritant exposure form the base of eczema management.¹⁹ Active inflammation is commonly treated with topical corticosteroids, while topical calcineurin inhibitors or other non-steroidal agents may be chosen according to age, anatomical site, and severity. Phototherapy or systemic treatment may be required when disease is more extensive or resistant.

Biologics directed at IL-4 and IL-13 pathways and oral or topical JAK inhibitors have expanded options for moderate-to-severe atopic dermatitis.^{10,13,20} These treatments have defined indications and safety monitoring. Ayurvedic care should not delay appropriate assessment, infection management, or indicated anti-inflammatory treatment. An integrative plan must state whether the Ayurvedic intervention is an alternative, an adjunct, or a maintenance strategy and test that role explicitly.

QUALITY OF THE AYURVEDIC EVIDENCE

Published clinical reports involving *Virechana*, *Raktamokshana*, topical applications, and *Rasayana* suggest possible symptom improvement.¹⁵⁻¹⁸ Confidence remains limited because samples are small, blinding is uncommon, treatment packages vary, and outcomes often depend on unvalidated symptom grades. These limitations do not prove that the therapies are ineffective; they prevent a reliable estimate of the size and durability of benefit.

Future trials should define both the Ayurvedic diagnosis and the corresponding dermatological diagnosis before enrolment. Atopic dermatitis, allergic contact dermatitis, irritant dermatitis, and other eczematous disorders should not be combined without justification. Investigators should also document concomitant moisturizers, topical medicines, diet changes, and rescue treatment because each can alter the result.

OUTCOMES FOR FUTURE STUDIES

A credible outcome set should combine patient experience, clinician-rated severity, objective barrier assessment, recurrence, and safety. Biomarkers can be included in mechanistic substudies, but they should not replace outcomes meaningful to patients.

Outcome domain	Suitable measure	Why it matters
Extent and severity	EASI or SCORAD	Provides a validated clinician-rated assessment
Patient-reported disease	POEM	Captures symptoms experienced

Outcome domain	Suitable measure	Why it matters
		across the preceding week
Itch	Numerical rating scale or visual analogue scale	Measures a core symptom and permits responder analysis
Quality of life	Dermatology Life Quality Index or an age-appropriate equivalent	Shows whether change is meaningful in daily life
Barrier function	Transepidermal water loss and skin hydration	Tests whether the intervention alters measurable barrier performance
Disease control	Time to flare and prespecified recurrence rate	Distinguishes maintenance from short-lived symptom relief
Safety	Adverse events, infection, irritation, laboratory monitoring when indicated	Allows benefit to be interpreted against treatment burden
Mechanistic exploration	Selected IL-4, IL-13, IL-31, microbiome, or other prespecified measures	Useful only when linked to a clear hypothesis and clinical outcome

RESEARCH DESIGN PRIORITIES

An informative trial would use a standardized diagnosis, concealed randomization, a prespecified primary endpoint, adequate sample size, and analysis according to the assigned groups. Usual barrier care should be comparable in all arms. The Ayurvedic intervention must be reproducible, whether it is a medicine, topical product, *Shodhana* procedure, or combined protocol. Product authentication, manufacturing details, procedural dose, adherence, rescue medication, and treatment fidelity should be reported.

Follow-up should extend beyond the treatment period because recurrence is one of the central clinical problems.

Reporting should include absolute change, between-group effect, confidence intervals, responder proportions, missing data, withdrawals, and adverse events. A mechanistic claim should be made only when the relevant mechanism was measured and changed in a way that corresponds with clinical improvement.

DISCUSSION

Vicharchika and eczematous dermatitis share an observable language of itch, eruptions, oozing, dryness, pigmentary alteration, and recurrence. The overlap is useful for generating clinical questions, but it does not establish identical disease boundaries. Modern eczema comprises several diagnoses, while the Ayurvedic classification is organized through a different account of causation, tissue involvement, and individual variation.

Both perspectives nevertheless discourage an excessively narrow view. Modern atopic dermatitis involves the epidermis, immune response, sensory nerves, microorganisms, and environment. Ayurveda also describes interacting *Dosha*, *Dushya*, *Agni*, exposure, and host factors. The similarity lies in recognizing a network of influences; it does not license equations such as *Rakta Dushti equals IL-13*, *Ama equals cytokines*, or *Pitta equals inflammation*.

The strongest bridge between the systems is empirical. Researchers can define an Ayurvedic pattern before treatment and then ask whether that pattern predicts itch severity, barrier impairment, recurrence, or response. They can also test a standardized intervention against an appropriate comparison while maintaining established eczema care. This produces knowledge that is meaningful in both clinical languages without changing the identity of either.

This review is limited by its narrative design and by the quality of the available Ayurvedic clinical literature. It does not provide a pooled estimate, formal risk-of-bias rating, or recommendation for a specific formulation. The proposed correlations and trial outcomes are a research framework, not evidence that the associated mechanisms have already been demonstrated.

CONCLUSION

Vicharchika is a clinically recognizable Ayurvedic pattern whose itching, eruptions, exudation, dryness, and pigmentary change overlap with features of eczematous dermatitis. Classical pathogenesis emphasizes *Dosha*, *Twak*, *Rakta*, altered *Agni*, and continued exposure to causative factors. Contemporary research describes impaired epidermal barrier function, type-2 inflammation, neuroimmune itch, JAK-STAT signalling, and microbial imbalance.

Ayurvedic strategies such as *Nidana Parivarjana*, individualized *Shamana*, external treatment, selected *Shodhana*, *Raktamokshana*, and *Rasayana* warrant careful investigation, but present clinical evidence is preliminary. Their place in integrative dermatology should be determined through reproducible interventions, validated eczema outcomes, objective barrier measures, explicit

safety monitoring, and long-term follow-up. Clinical overlap can guide research; it should not be used to claim untested molecular equivalence.

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