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Herbal Approaches to Herpes Zoster Management: A Review of Eucalyptus Oil and Ashwagandha as Topical Therapeutic Agents

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Abstract

Herpes zoster (HZ), caused by reactivation of latent varicella-zoster virus (VZV) in dorsal root or cranial nerve ganglia, remains a major cause of dermatomal pain and post-herpetic neuralgia (PHN), particularly among older and immunocompromised individuals. Standard management relies on systemic nucleoside analogues (acyclovir, valacyclovir, famciclovir) initiated within 72 hours of rash onset, together with analgesics and, in selected cases, corticosteroids; however, drug resistance, side effects, and incomplete prevention of PHN continue to drive interest in adjunctive and complementary approaches. This narrative review synthesizes the preclinical and mechanistic evidence supporting two botanicals with topical therapeutic potential in HZ: eucalyptus oil (Eucalyptus spp., principally standardized to its major monoterpene 1,8-cineole) and ashwagandha (Withaniasomnifera). Eucalyptus oil and its monoterpene constituents (1,8-cineole, α-pinene, γ-terpinene, α-terpineol) have demonstrated direct virucidal activity against herpes simplex virus (HSV-1 and HSV-2) in cell culture, acting principally at the stage of viral adsorption and entry, alongside anti-inflammatory and analgesic actions relevant to zoster-associated pain. Ashwagandha, rich in withanolides such as withaferin A and withanolide A, exhibits immunomodulatory, antiviral, anti-inflammatory, and wound-healing properties in preclinical models, including suppression of NF-κB/MAPK signaling and pro-inflammatory cytokines in human keratinocytes. Taken together, the mechanistic profiles of both agents are pharmacologically compatible with the goals of HZ management—viral suppression, inflammation control, analgesia, and lesion healing—supporting a biological rationale for their adjunctive topical use. However, no dedicated clinical trials of eucalyptus oil, ashwagandha, or their combination specifically in herpes zoster were identified in the literature reviewed; the evidence base rests on HSV surrogate models, in vitro/animal anti-inflammatory studies, and dermatological safety data. This review therefore also identifies critical

gaps and outlines a research agenda—including VZV-specific antiviral testing, standardized topical formulation development, and controlled clinical trials—needed before these agents can be recommended as evidence-based adjuncts to standard HZ care.

Keywords: Herpes zoster; varicella-zoster virus; eucalyptus oil; 1,8-cineole; Withaniasomnifera; ashwagandha; withanolides; topical phytotherapy; complementary medicine

1. Introduction

Herpes zoster (HZ), commonly known as shingles, results from the reactivation of latent varicella-zoster virus (VZV) that persists in sensory ganglia following primary varicella infection [1,2]. Reactivation is followed by viral replication within ganglionic and neural tissue, retrograde axonal transport to the skin, and the development of a painful, dermatomally distributed vesicular rash [2]. The lifetime risk of HZ in the general population is estimated at roughly one in three individuals, with incidence rising sharply after age 50 due to immunosenescence, and further increased risk among immunocompromised patients [1]. Beyond the acute eruption, HZ is clinically significant for its most feared complication, post-herpetic neuralgia (PHN)—persistent neuropathic pain that can substantially impair quality of life for months or years after the rash resolves [3].

Current standard management is centered on systemic antiviral therapy—acyclovir, valacyclovir, or famciclovir—ideally initiated within 72 hours of rash onset, which reduces viral shedding, accelerates lesion healing, and lowers (though does not eliminate) the risk of PHN [1,3,4]. Analgesics and, in select cases, corticosteroids are used adjunctively for pain and inflammation, alongside supportive local skin care [1]. Despite these options, unmet needs persist: antiviral therapy does not fully prevent PHN, treatment must be started early to be effective, some patients cannot tolerate or access standard antivirals, and there is ongoing concern about the emergence of antiviral-resistant herpesvirus strains, particularly in immunocompromised hosts [5]. These gaps have sustained clinical and research interest in complementary and adjunctive approaches, including topical botanical agents that might contribute antiviral, anti-inflammatory, analgesic, or wound-healing activity alongside conventional care.

Two botanicals frequently discussed in this context are eucalyptus oil, an essential oil obtained from Eucalyptus spp. and dominated by the monoterpene 1,8-cineole (eucalyptol), and ashwagandha (Withaniasomnifera), a foundational herb of Ayurvedic medicine whose principal bioactive constituents are steroidal lactones known as withanolides. Both have documented antiviral activity against herpes simplex virus (HSV) in vitro, together with anti-inflammatory, analgesic, and skin-healing properties reported across a range of preclinical and dermatological studies [6-15]. Because HSV and VZV are related alphaherpesviruses that share broad features of viral entry and replication, evidence generated against HSV is frequently cited as indirect support for potential activity against VZV, although this inference has not been directly tested. This review aims to (i) summarize the pathophysiology and current management of HZ, (ii) critically synthesize the available phytochemical, antiviral, anti-inflammatory, and dermatological evidence for eucalyptus oil and ashwagandha as topical agents, (iii) evaluate the biological plausibility of their adjunctive use in HZ, and (iv) identify the research gaps that must be addressed before evidence-based clinical recommendations can be made.

2. Review Methodology

This is a narrative literature review. Relevant publications were identified through structured searches of PubMed/MEDLINE, Scopus-indexed journals, and Google Scholar using combinations of the search terms: "herpes zoster," "varicella-zoster virus," "post-herpetic neuralgia," "eucalyptus oil," "1,8-cineole," "eucalyptol," "Withaniasomnifera," "ashwagandha," "withanolides," "topical," "antiviral," "anti-inflammatory," and "herpes simplex virus." Peer-reviewed original research articles, systematic and narrative reviews, and pharmacological/toxicological reports published in English were prioritized, with emphasis on articles from indexed biomedical journals. Given the absence of clinical trials on eucalyptus oil or ashwagandha specifically in HZ, surrogate evidence from HSV models and general dermatological/immunological studies was included and is explicitly flagged as indirect throughout the review.

3. Herpes Zoster: Epidemiology, Pathophysiology, and Current Management

3.1 Epidemiology and clinical course

HZ arises from reactivation of VZV that has remained latent in the dorsal root, geniculate, or trigeminal ganglia after primary varicella (chickenpox) infection [2]. Waning cell-mediated immunity to VZV—whether due to advancing age, HIV infection, malignancy, immunosuppressive therapy, or other causes—permits viral reactivation, retrograde transport of virus along sensory nerves, and replication in the skin, producing the characteristic dermatomal, grouped vesicular eruption on an erythematous base, typically preceded by neuropathic pain, paresthesia, or itching [1,2].

3.2 Complications: post-herpetic neuralgia and beyond

The most significant morbidity associated with HZ is PHN, a persistent neuropathic pain syndrome that can continue long after cutaneous lesions have healed and substantially affects patients' physical, psychological, and social functioning [3]. Other complications include herpes zoster ophthalmicus, secondary bacterial infection of lesions, and, rarely, disseminated disease in immunocompromised patients [1].

3.3 Current standard of care

First-line management is systemic antiviral therapy with acyclovir, valacyclovir, or famciclovir, ideally started within 72 hours of rash onset to reduce pain severity, accelerate cutaneous healing, and reduce (though not eliminate) the incidence of PHN [1,3,4]. Analgesics are used for acute pain control, and corticosteroids may be added in selected patients to reduce acute inflammation, although they do not reliably prevent PHN [1]. Supportive measures include local wound care and, where appropriate, isolation precautions to prevent transmission to susceptible individuals [1]. Vaccination with recombinant zoster vaccine is the principal preventive strategy in eligible populations [19]. Despite these interventions, disease burden remains substantial, particularly in Southeast Asian and other regions where awareness, vaccine uptake, and treatment pathways are reported to be suboptimal [19], underscoring the rationale for exploring adjunctive, low-cost topical therapies.

4. Eucalyptus Oil as a Topical Therapeutic Agent

4.1 Phytochemistry

Eucalyptus essential oil is obtained by steam distillation of the leaves of Eucalyptus spp. (family Myrtaceae) and is typically dominated by the monoterpene oxide 1,8-cineole (eucalyptol), which can constitute up to 80% or more of the oil, alongside smaller amounts of α-pinene, γ-terpinene, p-cymene, α-terpineol, terpinen-4-ol, and limonene [6,7]. 1,8-Cineole itself is a bicyclic monoterpene increasingly studied as a standalone phytopharmaceutical, and is the active ingredient of at least one clinically approved respiratory medication, underscoring its established pharmacological profile [13,14].

4.2 Antiviral activity against herpesviruses

The most direct evidence relevant to this review comes from in vitro studies of eucalyptus oil against HSV. In a foundational plaque-reduction assay using RC-37 cells, eucalyptus oil demonstrated a 50% inhibitory concentration in the range of 0.008–0.009% against HSV-1 and HSV-2, and non-cytotoxic concentrations reduced viral titers by approximately 58% (HSV-1) and 75% (HSV-2) [6]. Time-of-addition experiments indicated that the oil acts principally before or during viral adsorption to host cells rather than after penetration, consistent with a mechanism of direct virion inactivation rather than intracellular replication inhibition [6].

A more recent systematic review of eucalyptus essential oils as antiviral agents against human viruses similarly concluded that the principal antiviral mechanism is direct binding of monoterpene constituents to viral envelope proteins involved in adsorption and cell entry, thereby blocking infection before it is established [7]. In dose-response comparisons, individual monoterpenes—particularly α-pinene, γ-terpinene, and p-cymene—showed greater potency against HSV-1 than 1,8-cineole alone, suggesting that the antiviral effect of whole eucalyptus oil likely reflects a synergistic contribution of multiple constituents rather than a single active compound [7]. Complementary work isolating individual compounds from Eucalyptus globulus has also reported anti-HSV activity together with anti-inflammatory effects (suppression of NF-κB/AP-1 signaling and reduction of IL-1β and TNF-α), suggesting a dual antiviral–anti-inflammatory action relevant to the mixed viral/inflammatory pathology of herpetic lesions [8].

No published study was identified that directly tests eucalyptus oil or its constituents against VZV. Because HSV and VZV are both members of the Alphaherpesvirinae subfamily and share general mechanisms of envelope-mediated cell entry, the HSV data are commonly cited as indirect, biologically plausible support for potential anti-VZV activity; however, this remains an extrapolation rather than a demonstrated effect, and VZV-specific antiviral testing is an important research gap.

4.3 Anti-inflammatory and analgesic properties

Beyond direct antiviral effects, eucalyptus oil and 1,8-cineole possess anti-inflammatory and analgesic activity that may be relevant to the pain and cutaneous inflammation of acute HZ. 1,8-Cineole inhibits pro-inflammatory cytokine production (including IL-1 and IL-6) in macrophage models and downregulates pattern-recognition and inflammasome signaling pathways (TREM-1, NLRP3, NF-κB) [9]. In skin-relevant models, eucalyptus oil suppressed mast-cell degranulation and IgE-mediated allergic skin responses via downregulation of IgE-FcεRIsignaling [10], and 1,8-cineole has been characterized as a natural antagonist of the TRPA1 nociceptive ion channel, conferring an analgesic

effect against chemically induced sensory irritation in vivo [11]. A broader pharmacological review of 1,8-cineole further documents anti-inflammatory, antioxidant, analgesic, and antimicrobial activity across respiratory, dermatological, and neuropathic pain contexts [12,13].

4.4 Formulation and safety considerations

For topical use, eucalyptus oil requires dilution in a carrier oil or emulsion base, as undiluted application can cause skin irritation or sensitization; patch testing prior to use on broken or inflamed skin (as in active HZ lesions) is prudent [15]. Systemic absorption through inflamed or non-intact skin, and the well-documented toxicity of eucalyptus oil if ingested, particularly in children, warrant caution in any proposed topical formulation and clear labeling. No dedicated safety data in the specific context of open or crusting HZ lesions were identified, representing a further gap to be addressed before clinical use is recommended.

5. Ashwagandha (Withaniasomnifera) as a Topical Therapeutic Agent

5.1 Phytochemistry

Withaniasomnifera (ashwagandha, Indian ginseng) is a cornerstone herb of Ayurvedic medicine, valued traditionally for its adaptogenic, rejuvenating (Rasayana), and wound-healing properties [16,17]. Its principal bioactive constituents are withanolides—steroidal lactones including withaferin A, withanolide A, and related withanosides and sitoindosides—concentrated in the roots and, at even higher levels, in the leaves, alongside alkaloids and other secondary metabolites [16-18]. Analytical characterization by UHPLC-PDA and mass spectrometry has confirmed the presence of at least 11 distinct withanoside/withanolide compounds in standardized root extracts [16], and phytochemical profiling of root extracts using FTIR, UV-Vis, and NMR has confirmed characteristic lactone and enone functional groups consistent with withaferin A and withanolide A [21].

5.2 Immunomodulatory and antiviral properties

Ashwagandha is well documented in the pharmacological literature for immunomodulatory activity, including regulation of natural killer cell activity, modulation of T- and B-cell responses, and alteration of cytokine profiles [17,18]. Withanolides as a class have been reported to exhibit antibacterial, antiviral, antitumor, and immunomodulatory activities in various experimental systems [18]. As with eucalyptus oil, no dedicated study specifically evaluating ashwagandha or its withanolides against VZV was identified in the literature reviewed; the antiviral designation in the pharmacological literature derives largely from broader screening studies and reviews rather than HZ- or VZV-specific trials, and should be regarded as a plausible but unconfirmed extension.

5.3 Anti-inflammatory, analgesic, and dermatological/wound-healing evidence

The strongest direct evidence supporting a topical dermatological application of ashwagandha comes from studies in human keratinocytes and skin models. A hot-water extract of ashwagandha root significantly suppressed lipopolysaccharide-induced expression of pro-inflammatory cytokines (IL-6, IL-8, TNF-α, IL-1β, IL-12) in HaCaT human keratinocytes, while upregulating the anti-inflammatory cytokine TGF-β1; mechanistically, this was associated with inhibition of p38/JNK phosphorylation and nuclear translocation of NF-κB p65, with corresponding downregulation of TNF-α and upregulation of TGF-β1 confirmed in vivo in mouse skin [20]. These findings provide direct evidence that ashwagandha

root extract can modulate cutaneous inflammatory signaling relevant to the erythema and inflammation seen in acute HZ lesions.

Analgesic and anti-inflammatory activity has also been demonstrated systemically: ethanolic root extracts significantly prolonged latency in the hot-plate pain test and reduced carrageenan-induced paw edema by over 60%, an effect comparable in magnitude to hydrocortisone in the same model, with withanolide content (confirmed by TLC/NMR) implicated as the active principle [21]. Traditional Ayurvedic use of ashwagandha root and leaf paste for ulcers, leukoderma, scabies, and skin swelling is supported by more recent experimental wound-healing studies: topical application of Withania leaf paste and gel accelerated excision and incision wound closure in a rat model [22], and an ashwagandha ghrita (medicated ghee) formulation has been described in a clinical case report as promoting cleansing and healing of a non-healing wound [23]. A 2025 review focused specifically on non-malignant dermatological applications of Withaniasomnifera concluded, based on 18 studies meeting inclusion criteria from PubMed, Google Scholar, and Scopus, that the plant shows a favorable safety profile alongside antioxidant, anti-inflammatory, and adaptogenic activity relevant to pigmentary disorders, skin aging, inflammatory skin conditions, and wound healing [24]. Novel topical delivery formats are also being explored: an emulgel incorporating essential oils isolated from Withaniasomnifera demonstrated wound-healing activity in preclinical testing, illustrating feasible approaches to topical formulation [25].

5.4 Safety considerations

Ashwagandha has a generally favorable safety profile in oral pharmacokinetic and bioequivalence studies of standardized extracts [19], and topical formulations described in the dermatological literature have not raised major safety signals [24]. As with eucalyptus oil, however, no data specific to application on open, vesicular, or crusted HZ lesions were identified, and formulation stability, allergenicity, and appropriate concentration for inflamed skin remain to be established.

6. Rationale for Adjunctive Topical Use in Herpes Zoster

Considered together, eucalyptus oil and ashwagandha offer overlapping but mechanistically complementary properties that map onto the core objectives of HZ management: limiting viral spread, controlling cutaneous inflammation, relieving pain, and promoting lesion healing. Eucalyptus oil's principal strength, based on the available evidence, is direct virucidal activity against the adsorption/entry stage of a closely related alphaherpesvirus (HSV), together with anti-inflammatory and TRPA1-mediated analgesic effects relevant to neuropathic and inflammatory pain [6,7,9,11]. Ashwagandha's principal strength is potent, mechanistically well-characterized suppression of keratinocyte inflammatory signaling (NF-κB/MAPK pathway) together with demonstrated topical wound-healing activity in experimental models, complementing eucalyptus oil's more virus-focused actions [20,22]. A combination formulation—for example, a diluted eucalyptus oil preparation for early-stage antiviral/analgesic effect alongside an ashwagandha-based topical for inflammation control and healing during lesion resolution—is biologically plausible as an adjunct to, not a replacement for, standard systemic antiviral therapy, given that neither agent has demonstrated the potency or pharmacokinetic profile of acyclovir-class drugs. It is important to emphasize that this rationale is derived from mechanistic and surrogate (largely HSV) evidence; it has not been validated in VZV models or in HZ patients, and should be interpreted as hypothesis-generating rather than practice-changing.

7. Research Gaps and Future Directions

Several gaps must be addressed before eucalyptus oil and ashwagandha can be considered evidence-based adjuncts for HZ:

1. Direct testing against VZV. No identified study has evaluated eucalyptus oil, 1,8-cineole, or ashwagandha withanolides in VZV-specific plaque-reduction or cell-culture assays; current antiviral claims rely on HSV surrogate data.
2. Formulation development. Standardized, stable, dermatologically validated topical formulations (concentration, vehicle, penetration enhancers) suitable for application on vesicular/crusted skin have not been developed or tested for either agent in the HZ context.
3. Safety on compromised skin barrier. Irritancy, sensitization, and systemic absorption through open HZ lesions require dedicated dermatological safety evaluation.
4. Controlled clinical trials. Randomized, placebo- or active-controlled trials evaluating these agents as adjuncts to standard antiviral therapy—assessing time to lesion healing, pain scores, and incidence of PHN—are needed to move from mechanistic plausibility to clinical recommendation.
5. Drug-herb interaction and combination studies. Potential interactions with systemic antivirals, corticosteroids, or analgesics used concurrently in HZ management have not been characterized.

8. Conclusion

Herpes zoster remains a common and potentially debilitating condition for which current antiviral therapy, though effective, does not fully prevent complications such as post-herpetic neuralgia. Eucalyptus oil and ashwagandha each possess pharmacologically relevant properties—direct antiviral activity against a related alphaherpesvirus, anti-inflammatory signaling suppression, analgesic action, and wound-healing effects—that provide a coherent biological rationale for their investigation as topical adjuncts in HZ management. At present, however, this rationale rests on preclinical, mechanistic, and HSV-surrogate evidence rather than direct data in VZV or in HZ patients. Rigorous formulation development, VZV-specific antiviral testing, dermatological safety evaluation, and controlled clinical trials are required before eucalyptus oil, ashwagandha, or their combination can be recommended as evidence-based components of herpes zoster care.

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