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REVIEW

Integrated Western and Traditional Chinese Medicine Approaches for Herpes Zoster and Post-Herpetic Neuralgia: A Narrative Review

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Background: Herpes zoster (HZ) and postherpetic neuralgia (PHN) impose a substantial global burden, particularly in older and immunocompromised adults. Western therapies (antivirals, vaccination, and analgesics) reduce viral replication and acute pain but frequently fail to prevent chronic neuropathic pain or fully restore function. Traditional Chinese Medicine (TCM) is widely used as an adjunctive treatment in routine practice, yet its integration with Western care remains variably implemented and synthesized.

Objective: To provide a clinically oriented narrative synthesis of epidemiology, mechanisms, and treatment strategies for HZ/PHN from Western and TCM perspectives; to summarize the evidence base for integrated Western–TCM management; and to identify safety considerations, evidence gaps, and priorities for future research.

Methods: We conducted a narrative review of English- and Chinese-language literature on HZ/PHN by searching PubMed, Embase, Web of Science, and major Chinese databases (eg, CNKI) up to 2025. We included clinical trials, observational studies, and mechanistic research evaluating Western therapies, TCM interventions (herbal medicine, acupuncture/electroacupuncture, and other external modalities), or integrated regimens used alongside standard Western care. Study quality and risk of bias were appraised qualitatively with attention to sample size, blinding, outcome measures, and reporting of adverse events.

Results: HZ pathobiology involves varicella–zoster virus reactivation, neuroinflammation, peripheral and central sensitization, and small-fiber loss, which together drive acute pain and the transition to PHN. Western care provides effective viral suppression and evidence-based analgesia but offers limited strategies for neural repair. TCM interventions have demonstrated complementary effects on pain relief, lesion healing, and functional recovery in small randomized and practice-based studies. Across trials, integrated Western–TCM approaches are associated with faster rash healing, greater reductions in acute pain (particularly with acupuncture-based techniques), a possible reduction in PHN incidence, and improvements in sleep, mood, and quality of life. However, the certainty of the evidence is constrained by small sample sizes, protocol heterogeneity, non-standard outcome measures, and incomplete reporting of adverse events. Safety considerations include herb–drug interactions and quality control of herbal products; acupuncture- and moxibustion-related adverse events are uncommon and typically mild when performed by trained practitioners.

Conclusion: Integration of Western and TCM modalities for HZ/PHN is mechanistically plausible and clinically promising, targeting viral burden, neuroinflammation, pain modulation, and neural repair along complementary pathways. Current data support the cautious use of selected TCM therapies as adjuncts to guideline-based Western care in appropriate patients, while underscoring the need for high-quality pragmatic trials with standardized endpoints, mechanistic biomarkers, and robust safety monitoring to establish the effectiveness, safety, and cost-effectiveness of scalable integrated care pathways.

Keywords: herpes zoster, postherpetic neuralgia, integrative medicine, traditional Chinese medicine, acupuncture, antiviral therapy, quality of life, vaccination

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Introduction

Herpes zoster (HZ), commonly referred to as shingles, is a debilitating neurocutaneous disease caused by reactivation of the varicella-zoster virus (VZV), which remains dormant in sensory ganglia after primary infection with varicella (chickenpox).[1,2] Clinically, HZ is characterized by a painful vesicular rash distributed along the dermatomes, often preceded by prodromal pain. While most patients recover without major complications, a substantial proportion develop postherpetic neuralgia (PHN), a chronic neuropathic pain syndrome persisting beyond rash resolution and sometimes lasting for months to years.[3]

The burden of HZ and PHN is both global and profound. Epidemiological studies estimate that one in three individuals will develop HZ during their lifetime, and the incidence increases sharply with age.[4,5] PHN represents the most common complication, occurring in 10–20% of all HZ patients and up to 30% of those over 80 years old.[6] Persistent neuropathic pain in PHN is associated with insomnia, depression, anxiety, impaired mobility, and reduced quality of life, imposing both personal and healthcare system costs.[7] These epidemiological and socioeconomic patterns highlight the need for therapeutic approaches that address not only viral replication and acute symptoms but also the long-term neuroinflammatory and neuropathic components of the disease.

Current Western medical approaches, including antivirals, analgesics, and vaccination, have reduced the disease burden but remain insufficient. Antiviral therapy can shorten rash duration and decrease acute pain when administered promptly; however, it does not consistently prevent PHN.[8] Analgesic pharmacotherapy (gabapentinoids, tricyclic antidepressants, topical lidocaine or capsaicin, and opioids) often yields only partial relief and is limited by side effects in older or medically frail populations.[9,10] Thus, despite evidence-based tools, many patients experience suboptimal pain control or intolerance to medication, prompting clinical interest in complementary strategies.

Against this backdrop, Traditional Chinese Medicine (TCM) has long been used in Asia for painful dermatomal disorders, including HZ. TCM modalities such as herbal medicine, acupuncture, moxibustion, and external plasters are believed to clear "toxic heat", unblock stagnated qi and blood, and restore balance in affected meridians.[11] Modern research supports the following plausible mechanisms: reduction of inflammatory mediators, antioxidative effects, neurotrophic support, and modulation of central pain processing.[12–15] Preliminary trials suggest that TCM therapies may accelerate lesion healing, reduce acute pain, and lower the incidence of PHN. Moreover, interest in acupuncture and other TCM modalities has expanded beyond Asia, with increasing use in integrative pain clinics world-wide, reflecting a broader shift toward multimodal management of neuropathic pain.

Recently, interest in integrated Western–TCM approaches has grown. These strategies combine the antiviral and immunological benefits of Western medicine with the neuroregenerative and functional modulation effects of TCM. Early reports indicate that integration may enhance rash resolution, reduce PHN risk, and improve quality of life more effectively than either approach alone.[16,17] However, methodological limitations, including small sample sizes, heterogeneity of protocols, and inconsistent outcome reporting, have constrained firm conclusions. A concise synthesis of existing evidence is therefore needed to clarify the potential advantages, limitations, and clinical implications of integrated care models.

In this context, the present review summarizes current knowledge on HZ and PHN from both Western and TCM perspectives, with particular emphasis on areas of convergence and potential synergy. Previous reviews have often focused on single domains—such as antivirals and vaccination, or acupuncture and Chinese herbal medicine—but, to our knowledge, none has integrated these strands into a single, clinically oriented narrative that spans epidemiology, mechanisms, diagnosis, treatment, and implementation. To develop this narrative review, we drew on English- and Chinese-language literature identified through searches of PubMed, Embase, Web of Science, and major Chinese databases (eg, CNKI) up to 2025, including clinical trials, observational studies, and mechanistic research on Western, TCM, and integrated regimens. Study quality was appraised qualitatively, with attention to sample size, risk of bias, outcome measures, and adverse-event reporting, and we highlight remaining evidence gaps and key research priorities required to strengthen the evidence base.

Epidemiology and Risk Factors

Incidence and Age Distribution

HZ is a common condition worldwide, with an incidence that increases in tandem with global aging. Data from North America, Europe, and Asia consistently report an annual incidence of 3–5 per 1000 person-years in the general adult population, increasing to 8–12 per 1000 in those over 80 years.[18–20] Lifetime risk is estimated at approximately 30% in the general population and up to 50% among individuals living to age 85, reflecting the cumulative impact of age-related declines in varicella-zoster virus (VZV)-specific immunity.

Regional variations exist, but the trend is universal: the incidence rises steeply with age due to immunosenescence, characterized by reduced VZV-specific cell-mediated immunity.[21] Women are slightly more likely to develop HZ than men, although the reasons remain unclear.[22] Asian countries such as Japan, Korea, and China report similar or even higher age-adjusted incidence, reflecting demographic shifts and improved surveillance.[18,21]

Risk Factors for HZ

Beyond chronological age, several clinical, immunological, and psychosocial factors further increase the risk of HZ.

- Immunosuppression: Individuals with malignancy, HIV infection, hematopoietic stem cell transplantation, or chronic immunosuppressive therapies (eg, corticosteroids, TNF-α inhibitors) face markedly higher risk.[23,24]
- Chronic comorbidities: Diabetes, chronic kidney disease, COPD, and autoimmune diseases increase susceptibility, possibly via impaired immunity.[25]
- Psychological stress: Stress and depression have been linked to HZ risk, likely through neuroimmune modulation and glucocorticoid-mediated suppression of cellular immunity.[26]
- Female sex: Studies consistently report slightly higher risk among women.[22]

Predictors of Postherpetic Neuralgia

Not all patients with HZ develop PHN, but several clinical features at presentation have been consistently associated with a higher likelihood of persistent pain.

Older age: The strongest risk factor, with PHN prevalence increasing with each decade of life.[27]

Severe prodromal and acute pain: Intense pain before or during rash strongly predicts chronic pain.[28]

Severe rash and ophthalmic involvement: Disseminated lesions and involvement of cranial nerves (especially ophthalmic branch of the trigeminal nerve) carry higher risk.[29]

Delayed antiviral initiation: Starting antivirals >72 hours after rash onset reduces preventive efficacy against PHN.[30]

Vaccine Era Trends

The advent of vaccination has reshaped the epidemiology of HZ. The live attenuated zoster vaccine (ZVL) reduced HZ risk by ~50% and PHN by ~67%, but waning immunity limited its impact beyond 5–7 years.[31] The recombinant zoster vaccine (RZV), introduced in 2017, provides >90% efficacy across age groups, with sustained protection beyond 7 years.[32,33] Importantly, RZV reduces both HZ incidence and PHN burden, even in immunocompromised populations.[31,32]

Despite these advances, challenges remain, including low uptake in some regions, cost barriers, breakthrough cases, and limited evidence in special populations such as transplant recipients or frail older adults. Consequently, HZ and PHN continue to represent significant global health problems and justify ongoing efforts to optimize both preventive and therapeutic strategies.

Pathophysiology and Mechanisms

Western Medicine Viewpoint

Latency and Reactivation

Following varicella infection, VZV establishes latency in the dorsal root and cranial ganglia neurons, persisting for decades in a non-replicating state. Reactivation occurs when VZV-specific cell-mediated immunity declines, leading to viral replication, neuronal injury, and spread along sensory nerves.[34,35]

Acute Neuritis

During reactivation, viral proliferation causes inflammatory ganglionitis and neuronal necrosis. Cytokine release and immune infiltration generate acute neuritis, resulting in dermatomal pain, hyperesthesia, and vesicular rash.[35]

Transition to PHN

The persistence of pain after rash resolution reflects a complex pathophysiology:

- Peripheral sensitization: Damaged axons produce ectopic discharges and upregulate sodium channels, amplifying nociceptive signaling.[36]
- Central sensitization: Spinal dorsal horn neurons become hyperexcitable due to loss of inhibitory interneurons and enhanced NMDA receptor activity, maintaining allodynia and hyperalgesia.[35]
- Neuroimmune activation: Microglia and astrocytes release pro-inflammatory mediators (IL-1β, TNF-α, IL-6), sustaining chronic pain circuits.[37]
- Neurodegeneration: Skin biopsies show reduced intraepidermal nerve fiber density, reflecting structural nerve loss.[37]
- Neuroimaging: Functional MRI demonstrates altered thalamic and cortical pain networks, and PET shows increased glial activation.[38]

Collectively, these changes contribute to persistent neuropathic pain and sensory dysfunction that characterize PHN.

TCM Viewpoint

From the TCM perspective, HZ results from the invasion of wind-heat and damp-heat toxins into the meridians, compounded by qi stagnation and blood stasis. When *wei qi* (defensive qi) is insufficient, pathogens remain unresolved, leading to lingering pain (PHN).[39] Modern experimental studies have provided mechanistic support for TCM therapies:

- Herbal extracts (eg, Astragaloside IV, baicalin) reduce pro-inflammatory cytokines and NF-κB activation.[40]
- Acupuncture and electroacupuncture modulate neurotransmitters (serotonin, GABA) and enhance descending inhibitory pathways.[41]
- Certain formulas (eg, Buyang Huanwu decoction) increase expression of neurotrophic factors (BDNF, NGF, VEGF), promoting nerve regeneration.[42]
- Moxibustion and cupping improve microcirculation and reduce oxidative stress in animal models.[43]

These findings suggest that TCM interventions may influence many of the same inflammatory, neuroimmune, and neuroplastic processes implicated in PHN.

Mechanistic Overlaps

While differing in conceptual frameworks, both Western medicine and TCM converge on key mechanistic domains:

- Inflammation and immunity: Antivirals reduce viral replication, while TCM herbs and acupuncture downregulate pro-inflammatory cytokines.[44]

- Neural repair: Western medicine lacks disease-modifying strategies for nerve repair, but TCM modalities may promote neuroregeneration and small-fiber restoration.[45]
- Pain modulation: Pharmacological analgesics primarily target peripheral or central transmission, while acupuncture modulates both spinal and supraspinal plasticity.[46]
- Systemic resilience: Vaccination and immune support overlap conceptually with TCM's emphasis on strengthening *wei qi*.[44]

This shared mechanistic landscape provides a biological rationale for integrated approaches that simultaneously address viral burden, neuroinflammation, neural repair, and functional recovery.

Clinical Presentation and Diagnosis

Prodrome and Acute Presentation

The clinical course of herpes zoster (HZ) typically begins with a prodromal phase lasting one to five days before the onset of rash. Patients frequently report neuropathic pain symptoms, such as burning, stabbing, or tingling sensations, localized to one dermatome.[47] This pain may be severe and often precedes visible cutaneous changes, leading to misdiagnoses such as myocardial infarction, biliary colic, or migraine.[48] Additional prodromal symptoms may include malaise, fever, headache, and localized pruritus.

The hallmark acute rash consists of erythematous maculopapular lesions that rapidly evolve into vesicles grouped along the affected dermatome. Lesions typically crust within 7–10 days and resolve in 2–4 weeks.[49] The thoracic dermatomes are the most commonly affected (≈50%), followed by the cranial (trigeminal) and lumbosacral nerves.[50] Pain during this stage is often out of proportion to the visible rash, reflecting underlying ganglionitis and neuronal inflammation.

Pain Characteristics

HZ-associated pain is heterogeneous and encompasses both nociceptive and neuropathic features. Neuropathic descriptors include burning, electric-shock-like, stabbing, and itching sensations.[51] Acute pain intensity is a strong predictor of subsequent postherpetic neuralgia (PHN), which is defined as pain persisting beyond rash healing (commonly ≥90 days after onset, although definitions vary).[46] Patients may also exhibit allodynia (pain from non-painful stimuli, such as light touch or clothing contact) and hyperalgesia (exaggerated pain response to mildly painful stimuli). These features reflect both peripheral nerve damage and central sensitization.[52] Recognizing these early pain characteristics is essential, as they inform prognosis and may guide the intensity of acute-phase management.

Special Presentations and Red Flags

Certain clinical variants require urgent recognition owing to associated morbidity:

- Herpes zoster ophthalmicus (HZO): Involvement of the ophthalmic branch of the trigeminal nerve presents with periorbital rash, conjunctivitis, keratitis, or uveitis. Complications include corneal scarring, glaucoma, and vision loss.[53] Hutchinson's sign (vesicles on the tip of the nose) predicts ocular involvement. Herpes zoster oticus (Ramsay Hunt syndrome): Vesicles in the external ear canal or pinna with ipsilateral facial paralysis, hearing loss, or vertigo indicate geniculate ganglion involvement. The prognosis for facial nerve recovery is poorer than that in Bell's palsy.[54]
- Central nervous system (CNS) involvement: VZV may cause encephalitis, meningitis, myelitis, or stroke through vasculopathy. Presentations include headache, altered mental status, focal deficits, and seizures. These complications are more common in immunocom-238 promised hosts.[55]
- Disseminated zoster: Defined as >20 lesions outside the primary dermatome, often occurring in immunosuppressed individuals, with potential for systemic dissemination to lungs, liver, or CNS.[55] These red-flag presentations warrant urgent evaluation and, in many cases, inpatient antiviral therapy.

Diagnosis

Clinical Diagnosis

In most cases, the diagnosis is clinical and based on the recognition of the characteristic painful dermatomal vesicular rash. The combination of acute dermatomal neuropathic pain and rash is highly specific.[56] Laboratory confirmation is generally unnecessary in immunocompetent adults but may be considered in atypical, disseminated, or immunocompromised presentations.

For clinical completeness and to support standardized assessment, we paired routine clinical recognition with structured red-flag screening and validated outcome tools. Table 1 provides a concise summary of the diagnostic criteria, urgent referral signs, and commonly used pain and quality-of-life instruments.

Laboratory Confirmation

Laboratory testing is useful in atypical or immunocompromised cases.

- Polymerase chain reaction (PCR): Gold standard, highly sensitive and specific for VZV DNA in vesicle fluid, blood, or CSF.[57]

Direct fluorescent antibody (DFA): Rapid but less sensitive.[58]

- Serology: Limited utility for acute diagnosis; may support retrospective confirmation of exposure. Overall, laboratory confirmation is reserved for situations where clinical findings are unclear, lesions are absent or atypical, or disseminated disease is suspected.

Table 1 Diagnostic Criteria, Red Flags, and Outcome Assessment Tools in Herpes Zoster (HZ) and Postherpetic Neuralgia (PHN)

Category	Feature/Tool	Description/Criteria	Clinical Relevance
Diagnostic Criteria	Typical HZ diagnosis	Unilateral, dermatomal, painful vesicular rash preceded by prodrome	Highly specific for HZ; diagnosis usually clinical
Diagnostic Criteria	Laboratory confirmation (PCR)	Detection of VZV DNA in vesicle fluid, blood, or CSF	Gold standard when diagnosis uncertain or immunocompromised patient
Diagnostic Criteria	Serology (IgM/IgG)	Limited value in acute diagnosis; supportive in retrospective confirmation	Used in atypical presentations or epidemiologic studies
Diagnostic Criteria	Postherpetic neuralgia (PHN)	Pain persisting ≥ 90 days after rash onset (sometimes ≥ 30 or 120 days)	Standard definition in most clinical trials and guidelines
Red Flags	Herpes zoster ophthalmicus (HZO)	Ophthalmic branch of trigeminal nerve; Hutchinson's sign, ocular involvement	Vision-threatening; requires urgent ophthalmology referral
Red Flags	Herpes zoster oticus (Ramsay Hunt)	Auricular vesicles + ipsilateral facial palsy, hearing loss, vertigo	Poorer prognosis than Bell's palsy; requires prompt antiviral/steroid
Red Flags	Neurological complications	Encephalitis, meningitis, myelitis, stroke from VZV vasculopathy	Present with headache, altered mental status, focal deficits; urgent workup
Red Flags	Disseminated zoster	≥ 20 vesicles outside primary dermatome, or visceral involvement	Life-threatening in immunocompromised; requires IV antivirals/hospitalization
Pain/Outcome Assessment	Numeric Rating Scale (NRS)/ Visual Analog Scale (VAS)	Patient rates pain 0-10 or on a visual scale	Standard measure for pain intensity in HZ/ PHN trials
Pain/Outcome Assessment	Brief Pain Inventory (BPI)	Assesses both pain severity and interference with daily function	Captures broader impact of pain on quality of life
Pain/Outcome Assessment	Neuropathic Pain Screening Tools (DN4, PainDETECT)	Structured questionnaires evaluating neuropathic features	Distinguishes neuropathic from nociceptive pain
Pain/Outcome Assessment	Quality of life and functional tools	Sleep quality, mood scales, activity level questionnaires	Important for holistic PHN management

Pain and Functional Assessment

An accurate assessment of pain is critical, especially in patients with PHN.

The Numeric Rating Scale (NRS) and Visual Analog Scale (VAS) are commonly used for intensity.[59] The Brief Pain Inventory (BPI) assesses both pain severity and functional interference.[60] Neuropathic pain screening tools (DN4, PainDETECT) help characterize neuropathic features.[61] Functional evaluation should also include sleep quality, mood, daily activity, and quality of life, since these domains are substantially affected by PHN.[62]

Using a combination of these tools provides a more comprehensive profile of patient burden and helps monitor response to therapy over time.

Diagnostic Criteria for PHN

The definition of PHN varies across studies, complicating comparisons. The most widely accepted definition is pain persisting for ≥90 days after rash onset.[63] Some guidelines use 30 or 120 days, but the 90-day cutoff balances specificity and clinical relevance.

In clinical research, adopting a consistent definition is essential for comparability across trials. In TCM practice, the classification of PHN may include syndrome differentiation (eg, residual heat, qi deficiency, and blood stasis), guiding individualized therapy.[64] These diagnostic approaches, although conceptually distinct, can complement each other in integrated care settings.

Current Western Medicine Approaches

Western medical management of herpes zoster (HZ) and postherpetic neuralgia (PHN) has traditionally focused on three pillars: antiviral therapy to control viral replication, analgesic strategies to mitigate acute and chronic pain, and vaccination to prevent the disease and its complications. Despite these advances, the burden of PHN persists, highlighting both the strengths and limitations of current approaches. A concise overview of key therapeutic principles is provided below.

Antiviral Therapy

Mechanism and Rationale

Nucleoside analogs inhibit viral DNA polymerase, suppressing varicella-zoster virus (VZV) replication, thereby reducing viral load, lesion formation, and neuronal injury.[65] Early initiation is crucial because viral replication peaks within the first 72 hours of rash onset. Timely treatment remains the strongest modifiable factor influencing acute outcomes.

Agents and Dosing

Acyclovir: 800 mg orally five times daily for 7–10 days. Valacyclovir: 1000 mg orally three times daily for 7 days. Famciclovir: 500 mg orally three times daily for 7 days. Randomized controlled trials (RCTs) have shown that valacyclovir and famciclovir are at least as effective as acyclovir and offer superior bioavailability and simplified dosing, improving adherence.[66] The typical dosing, timing windows, expected benefits, and monitoring considerations for first-line antivirals are detailed in Table 2.

Clinical Benefits

Antivirals:

- Shorten duration of viral shedding and rash healing by 1–2 days.
- Reduce severity of acute pain and accelerate resolution.
- May lower incidence of PHN, particularly when started within 72 hours.[67,68]

Limitations

Antivirals do not eradicate latent virus and cannot fully prevent PHN, especially in older adults.

Efficacy diminishes with delayed initiation (>72 hours).

Renal dose adjustment is required, particularly in elderly patients.

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Table 2 Summary of therapeutic options for herpes zoster (HZ) and postherpetic neuralgia (PHN): agents, regimens, outcomes, safety, and evidence

Therapy Class	Agent / Procedure	Typical Dosing / Regimen	Timing / Use	Key Efficacy Outcomes	Adverse Effects / Monitoring	Evidence Level	Notes
Antiviral (nucleoside analog)	Acyclovir (PO)	800 mg PO five times daily × 7–10 days	Start ideally ≤72 h from rash onset (up to 7 days if new lesions/ongoing pain)	Shortens healing; reduces acute pain; modest PHN reduction if early	GI upset, headache; renal crystalluria (hydrate); adjust in CKD	Multiple RCTs; Cochrane	Preferred when cost/availability drives choice; renal dosing essential
Antiviral (prodrug of acyclovir)	Valacyclovir (PO)	1000 mg PO TID × 7 days	Same as above	Noninferior/superior to acyclovir; improved adherence (TID)	Nausea, headache; rare TMA in severe renal impairment; adjust in CKD	Multiple RCTs; guideline	First-line oral agent for immunocompetent adults
Antiviral (guanine analog)	Famciclovir (PO)	500 mg PO TID × 7 days	Same as above	Comparable to valacyclovir; reduces acute pain	GI upset, headache; adjust in CKD	Multiple RCTs; guideline	Consider if valacyclovir unavailable or not tolerated
Adjunct corticosteroid	Prednisone (with antiviral)	60 mg/day × 7 d, taper over 2–3 weeks	Severe neuritis, functional limitation (immunocompetent)	Improves acute pain, QOL; no proven PHN prevention	Hyperglycemia, fluid retention, infection risk	RCTs; mixed meta-analyses	Avoid without antiviral; individualize risk–benefit
Analgesic (non-opioid)	NSAIDs / Acetaminophen	Ibuprofen 200–400 mg q6–8h PRN; Acetaminophen ≤3–4 g/day	Mild–moderate acute pain	Relief of nociceptive pain; limited effect on neuropathic pain	GI/renal (NSAIDs); hepatotoxicity (acetaminophen)	Consensus	Combine with neuropathic agents as needed
Opioid (short course)	Morphine / Oxycodone (short-acting)	Titrate for several days; reassess	Severe acute pain refractory to non-opioids	Rapid analgesia; no PHN prevention	Sedation, constipation, delirium, dependence	Guideline–conditional	Use lowest dose, shortest duration; bowel regimen
Neuropathic analgesic	Gabapentin	1800–3600 mg/day in 3 doses (titrated)	Acute HZ (early) or PHN	Reduces pain; improves sleep/QOL	Dizziness, somnolence, ataxia; adjust in CKD	Multiple RCTs; guideline	Start low, go slow; monitor falls
Neuropathic analgesic	Pregabalin	150–600 mg/day in 2–3 doses	Acute HZ (early) or PHN	Reduces pain; faster titration than gabapentin	Dizziness, edema, weight gain; adjust in CKD	Multiple RCTs; guideline	Consider when rapid titration needed
Tricyclic antidepressant (TCA)	Amitriptyline / Nortriptyline	Amitriptyline 10–75 mg QHS; Nortriptyline 10–75 mg QHS	Established PHN	Analgesia independent of mood; large effect size	Anticholinergic effects, QT prolongation; avoid in severe CAD/glaucoma	Older RCTs; guideline	Prefer nortriptyline/desipramine in elderly
SNRI antidepressant	Duloxetine / Venlafaxine	Duloxetine 30–60 mg/day; Venlafaxine 75–225 mg/day	PHN when TCA not tolerated	Pain reduction; functional gains	Nausea, insomnia, BP rise (venlafaxine)	Guideline–supported	Useful with comorbid depression/anxiety
Topical anesthetic	Lidocaine 5% patch	Up to 3 patches, 12h on/12h off	Allodynia-predominant PHN	Improves pain; well tolerated	Local skin reactions	Multiple RCTs	First-line topical; safe in polypharmacy
Topical TRPV1 agonist	Capsaicin 8% patch	Apply 30–60 min; repeat q3mo	Localized PHN refractory/intolerant to systemic meds	Pain reduction ≤12 weeks	Transient burning/erythema	RCTs; guideline	Requires pretreatment + trained staff
Peripheral block	Local anesthetic ± steroid	Single-shot/catheter in acute phase	Severe acute HZ; may reduce PHN risk	Short-term analgesia; functional gain	Bleeding, infection, pneumothorax	RCTs/mixed evidence	Best if within 1–2 weeks of rash
Epidural block	Local anesthetic ± opioid/steroid	Single-shot/short infusion	Refractory acute pain; subacute PHN	Short-term relief; unclear PHN prevention	Hypotension, infection, retention	Mixed evidence	Weigh risks in elderly
Sympathetic block	Stellate/thoracic sympathetic block	By dermatomal level	Refractory sympathetically-maintained pain	Case series: benefit	Hoarseness, pneumothorax	Low–moderate	Diagnostic + therapeutic

Neuro-modulation	Spinal Cord Stimulation (SCS)	Trial lead → implant if ≥50% relief	Refractory PHN post-failure	Sustained pain relief; QOL gains	Lead migration, infection, cost	Observational /registries	Specialized centers
Neuro-modulation	DRG stimulation	Trial → implant	Focal PHN (thoracic, trigeminal)	Targeted analgesia; less paresthesia spread	Risks similar to SCS	Early studies	For localized refractory pain
Minimally invasive RF	Pulsed Radiofrequency (PRF)	Outpatient fluoroscopy-guided	PHN refractory to meds/topicals	Pain reduction; low destructive risk	Procedure pain; rare neuritis	Small RCTs	Repeatable before destructive options
Peripheral injection	Botulinum toxin A	Subcutaneous grid injections	Localized refractory PHN adjunct	Moderate pain reduction; sleep gains	Local weakness, bruising	Small RCTs	Useful if systemic drugs not tolerated
Vaccination (legacy)	Zoster Vaccine Live (ZVL)	Single dose (historic)	Adults ≥60 y	50% HZ reduction; wanes 5 y	Vaccine rash; avoid severe immunosuppression	Landmark RCT	Superseded by RZV
Vaccination (preferred)	Recombinant Zoster Vaccine (RZV)	2 doses 0 + 2–6 mo IM	Adults ≥50 y; immunocompromised	>90% efficacy vs HZ; PHN reduction; durable ≥7 y	Injection-site pain, fever, myalgias	Phase 3 RCTs; real-world	Preferred; safe in immunocompromised

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Corticosteroids

Corticosteroids are sometimes added to antiviral regimens to attenuate acute neuritis and improve quality of life. Clinical trials have demonstrated modest benefits in reducing acute pain and improving short-term functional recovery, but no significant reduction in PHN incidence.[69]

Typical regimen: Prednisone 60 mg/day tapered over 3 weeks in combination with antiviral agents.

Risks include hyperglycemia, fluid retention, psychiatric effects, and immunosuppression, especially in elderly or comorbid patients.[70] Therefore, corticosteroids should be reserved for selected patients without contraindications. They are generally avoided in immunocompromised individuals due to concerns of worsening viral replication.

Acute Pain Management

Effective pain control during acute HZ is vital to reduce suffering and possibly prevent central sensitization leading to PHN. Management follows a stepwise approach. A comparative overview of non-opioid analgesics, short-course opioids, and early neuropathic agents, along with their adverse effect profiles and evidence levels, is provided in Table 2.

- Non-opioid Analgesics: NSAIDs and acetaminophen may help with mild to moderate pain but are limited for neuropathic features.[71]
- Opioids: Short-course opioids (eg, oxycodone, morphine) may be required for severe acute pain. Their use is limited by risk of sedation, constipation, and dependency, particularly in older adults.[72]
- Neuropathic Agents (Early Use): Gabapentinoids (gabapentin, pregabalin): Effective for neuropathic pain; may be initiated during the acute phase to reduce the transition to PHN.[73] Tricyclic antidepressants (TCAs): Effective but limited by anticholinergic side effects.[74] Serotonin–norepinephrine reuptake inhibitors (SNRIs): Alternatives for patients intolerant to TCAs.[75] Early initiation of neuropathic agents is increasingly considered in high-risk patients, though evidence remains mixed.

Pharmacotherapy for PHN

PHN is notoriously refractory and requires multimodal management. Evidence-based guidelines (AAN, EFNS, NICE) recommend the following.[76] First- and second-line options for PHN—gabapentinoids, TCAs/SNRIs, topical lidocaine/capsaicin, and selected interventional modalities—are summarized in Table 2.

First-Line Agents

Gabapentin (up to 3600 mg/day) and pregabalin (150–600 mg/day): RCTs show significant reduction in pain intensity and improvement in sleep and quality of life.[77]

TCAs (amitriptyline, nortriptyline): Analgesic efficacy established, but tolerability is limited in older adults.[78]

SNRIs (duloxetine, venlafaxine): Fewer side effects than TCAs, with somewhat less robust evidence.

Topical Lidocaine 5% Patch: Provides localized analgesia with excellent tolerability, especially for patients with allodynia.[79]

Second-Line/Adjunct Agents

- capsaicin 8% Patch: Applied under medical supervision; provides pain relief for up to 3 months. Local burning is common.[80]
- Tramadol and Controlled-Release Opioids: Reserved for refractory cases.[81]
- Combination therapy: Often required (eg, gabapentinoid + lidocaine patch).

Interventional Therapies

For refractory acute pain or established PHN, interventional approaches may be considered. Practical selection notes and risk profiles for blocks, neuromodulation, PRF, and botulinum toxin are summarized in Table 2.

- Nerve Blocks: Peripheral nerve or paravertebral blocks may reduce acute pain and potentially lower PHN risk when administered early.[82] Epidural blocks provide mixed evidence.
- Sympathetic Blocks: Stellate ganglion or thoracic sympathetic blocks may help in refractory cases, but evidence is limited.[83]
- Neuromodulation: Spinal cord stimulation (SCS) has shown long-term efficacy in refractory PHN, although cost and invasiveness limit its use.[84] Dorsal root ganglion (DRG) stimulation is promising for targeted pain control.[85] Pulsed radiofrequency (PRF) may reduce PHN pain with fewer risks than ablative methods.[86] Botulinum toxin injections have shown benefits in small RCTs.[87]

These interventions are usually reserved for specialized centers and patients who fail conservative therapy.

Vaccination

Vaccination remains the most effective preventive strategy against HZ. The key differences between ZVL and RZV—indications, durability, and safety—are outlined in Table 2.

Zoster vaccine live (ZVL): Reduced HZ incidence by ~50% and PHN by ~67%, but efficacy wanes within 5 years.[88]

Recombinant zoster vaccine (RZV): >90% efficacy against HZ and PHN across age groups, with sustained protection beyond 7 years.[89] RZV is safe and immunogenic in immunocompromised patients, including those with hematologic malignancies or those who have undergone transplantation.[90] Despite its strong performance, RZV uptake remains uneven globally due to cost, access disparities, and vaccine hesitancy.

Special Populations

- Elderly: Require lower starting doses for neuropathic medications.[91] Dose adjustments (eg, renal dosing of antivirals and gabapentinoids) and tolerability considerations are cross-referenced in Table 2.
- Renal or Hepatic Impairment: Dose adjustments for antivirals, gabapentin, and opioids are essential.[92]
- Pregnancy: Antivirals (especially acyclovir) may be used safely; corticosteroids and opioids require caution.[93]
- HIV/Transplant/Oncology: Higher incidence and severity; prolonged or IV antivirals may be required. Vaccination with RZV is recommended but must be timed carefully relative to immunosuppression.[94]

Summary of Western Approaches

Western medicine provides powerful antiviral and analgesic tools and highly effective preventive vaccines; however, limitations remain.

Antivirals do not fully prevent PHN.

Pharmacotherapy for PHN is often only partially effective and limited by tolerability.

Interventional and neuromodulation therapies show promise but are costly and evidence is heterogeneous.

Vaccination is transformative but underutilized in many populations.

These gaps motivate interest in complementary strategies, including integrative approaches that address inflammation, neural repair, and functional recovery.

Traditional Chinese Medicine (TCM) Approaches

TCM Theory Relevant to HZ/PHN

Within TCM nosology, acute herpes zoster (HZ) is commonly attributed to the invasion of wind-heat and damp-heat toxins that obstruct the channels and collaterals, with qi and blood stagnation along the affected dermatomes. Inadequate *wei qi* (defensive qi) is thought to allow pathogen persistence. Persistent, burning, allodynic pain after cutaneous healing —post-herpetic neuralgia (PHN)—is interpreted as residual heat/toxin with qi and blood stasis and, in older adults, an underlying deficiency of qi and yin. Therefore, treatment principles combine clearing heat/toxin, moving qi/blood, relieving pain, and in the PHN phase, nourishing deficiency and promoting collateral repair. Contemporary reviews summarize how this traditional framework maps onto immune and neuroinflammatory processes.[46] This integration of

classical theory with modern biomedical correlates provides a conceptual basis for applying TCM modalities across both acute and chronic phases of HZ.

Herbal Medicine (Internal)

Common Formulas and Patterns

Although practice is individualized by syndrome differentiation, formulas frequently reported in HZ/PHN include Longdan Xiegan Tang (wind-heat/damp-heat presentations), Jiedu Huoxue-type prescriptions (detoxify + move blood), and Buyang Huanwu Decoction (post-rash neural repair, weakness/deficiency patterns). These are typically delivered as decoctions or granules for 1–3 weeks in acute HZ and longer in PHN, with titration to response and adverse effect monitoring. Narrative and systematic reviews catalog such use while emphasizing variability in composition and dose across trials.[95,96] The representative formulas, mechanistic plausibility, reported clinical outcomes, and interaction cautions are summarized in Table 3. Given the heterogeneity of prescriptions, standardized reporting of herbal components remains a key need for future trials.

Single Herbs/Actives and Mechanistic Plausibility

Preclinical and translational data suggest that several TCM actives modulate pathophysiologic processes relevant to HZ/PHN:

- Astragaloside IV (*Astragalus membranaceus*): suppresses microglial activation, reduces NF-κB–mediated inflammation/oxidative stress, and promotes neurotrophic signaling in CNS models—mechanistically consistent with neural repair and central pain modulation.[97]
- Baicalin (Scutellaria baicalensis): inhibits TLR4/NF-κB pathways, dampens NLRP3 inflammasome activity, and reduces neuroinflammation in vivo, suggesting potential to attenuate neuroimmune sensitization.[98]
- Notoginsenosides (*Panax notoginseng saponins*): demonstrate neuroprotective and antiplatelet effects, with clinically relevant interactions affecting coagulation pathways.[99]

These mechanistic data are supportive but indirect; high-quality clinical evidence for single-compound efficacy in HZ/PHN remains limited. For quick reference to specific actives and their clinical relevance, see Table 3. Overall, single-compound studies highlight potential biological targets but do not substitute for clinical trial evidence.

Safety, Quality, and Herb–Drug Interactions

The interaction alerts (eg, anticoagulation with *Panax notoginseng* and immunomodulation with *Astragalus*) are highlighted in Table 3.

- Quality assurance: Variability in plant identity, contaminants, and constituents necessitates adherence to GMP and pharmacopoeial standards.[100]
- Herb–drug interactions: *Panax notoginseng* can influence coagulation and interact with warfarin; broader reviews describe CYP/P-gp mediated interactions pertinent to oncology and polypharmacy.[101–103]

These issues underscore the importance of careful medication reconciliation and adverse event monitoring in integrative practice.

External Therapies

Acupuncture and Electroacupuncture (EA)

- Rationale and mechanisms: Acupuncture modulates descending inhibitory pathways, alters neurotransmitters (eg, serotonin and GABA), and suppresses neuroinflammation; EA adds frequency-specific neuromodulatory effects. Typical strategies include Jiaji (EX-B2), Ashi (tender) points, and distal modulators (LI4, SJ5, GB34, ST36), delivered 2–5 times/week during acute/subacute phases.[104,105] The protocols, dosing, target phases, outcomes, and

Table 3 Selected Traditional Chinese Medicine (TCM) herbal formulas and compounds studied in herpes zoster (HZ) and postherpetic neuralgia (PHN)

Formula / Compound	Composition / Source	Traditional Indication / Syndrome	Proposed Mechanisms (Modern Evidence)	Clinical Outcomes Reported	Safety / Interaction Notes
Longdan Xiegan Tang	Gentiana (Longdan), Scutellaria (Huangqin), Gardenia (Zhizi), etc.	Clearing liver–gallbladder damp-heat, acute herpes zoster with erythema, vesicles, burning pain	Anti-inflammatory, reduces TNF-α/IL-6; antioxidant; hepatoprotective effects	Faster rash resolution, reduced acute pain in small RCTs and case series	Generally well tolerated; caution in liver disease due to hepatotoxicity risk with long-term use
Jiedu Huoxue Decoction (variants)	Detoxifying herbs (e.g., Smilax, Isatis) + blood-activating herbs (e.g., Angelica, Salvia)	Wind-heat/damp-heat with qi/blood stasis; acute/subacute HZ with severe pain	Immune modulation; improves microcirculation; NF-κB suppression	Improved pain relief and shorter lesion healing time when added to antivirals	Possible herb–drug interactions via CYP enzymes; monitor polypharmacy
Buyang Huanwu Decoction	Astragalus, Angelica sinensis, Paeonia rubra, Ligusticum, Carthamus, Peach kernel, Earthworm	Qi deficiency and blood stasis; convalescent stage, neural repair in PHN	Promotes nerve regeneration (↑BDNF, NGF, VEGF); improves microcirculation	Functional recovery and neuropathic pain improvement in small studies	High-dose Astragalus may interact with immunosuppressants; monitor transplant patients
Astragaloside IV (Astragalus membranaceus)	Purified saponin from Huangqi (Astragalus)	Qi deficiency syndromes; tonic, neural repair	Suppresses microglial activation; ↓NF-κB, oxidative stress; ↑NGF/BDNF	Neuroprotective effects in preclinical models; adjunctive role in neuropathic pain	Potential interaction with immunosuppressants; quality variation across sources
Baicalin (Scutellaria baicalensis)	Flavonoid from Huangqin root	Clearing heat-toxin, damp-heat syndromes	Inhibits TLR4/NF-κB and NLRP3 inflammasome; antioxidant; neuroimmune modulation	Animal models: reduced neuropathic pain behaviors; anti-inflammatory effects	Possible hepatotoxicity with high doses; interacts with CYP enzymes
Notoginsenosides / Panax notoginseng saponins (Sanqi)	Triterpenoid saponins from Panax notoginseng root	Blood stasis with pain; trauma, neuralgia	Neuroprotective; antiplatelet and anticoagulant effects; microcirculatory improvement	Case reports and small trials: reduced pain and inflammation in neuralgia	Interaction with anticoagulants (warfarin, aspirin, clopidogrel); monitor INR and bleeding risk

safety notes for acupuncture, EA, wrist–ankle acupuncture, fire needle, moxibustion, cupping/scraping, and topical plasters appear in Table 4.

- Clinical evidence: Systematic reviews/meta-analyses report that acupuncture (including EA) reduces PHN pain versus antiepileptics, with fewer adverse events, although risk of bias is high.[106,107] In acute HZ, acupuncture combined with standard care accelerates pain reduction and lesion healing.[41,108] Older reviews suggest that acupuncture ± moxibustion outperforms conventional therapy.[39,109] Adverse events are typically mild; serious complications are rare.[110] Taken together, evidence supports symptomatic benefit, but methodological limitations prevent firm conclusions regarding comparative efficacy.

Fire Needle (Huozhen)

Technique: Brief thermal stimulation using a heated needle. Evidence: Systematic reviews suggest short-term pain relief and faster lesion resolution in acute HZ, although most trials are small with unclear blinding.[12,111,112] The operational parameters and safety considerations for fire needles are presented in Table 4. Its use remains practitioner-dependent, and standardized protocols are needed.

Moxibustion

Heat stimulation with burning moxa at acupoints or dermatomes. Evidence: Meta-analyses report greater pain reduction and higher "clinical efficacy", though with heterogeneity and high risk of bias[113,114]. Comparative session frequency, target phases, and adverse events for moxibustion and related external modalities appear in Table 4. Evidence suggests potential benefit, but quality-of-evidence limitations persist.

Cupping, Scraping, Plasters

Evidence is less robust, largely from small RCTs or non-randomized studies. Reviews suggest improvements in pain and healing time, but protocols vary.[115,116] These therapies may be reasonable adjuncts but require further controlled evaluation.

Evidence Synthesis

- Outcomes: Meta-analyses suggest benefit of acupuncture/EA on PHN pain and of acupuncture±moxibustion on acute HZ pain and healing, with new RCT support for wrist–ankle acupuncture.[41]
- Mechanisms: TCM modalities may reduce neuroinflammation, enhance neurotrophic signaling, and modulate pain circuits.[40,68,117]
- Safety: Acupuncture/EA adverse events are infrequent and mild; herbal quality issues and drug interactions require vigilance.[118]
- Gaps: Heterogeneity in protocols, short follow-up, and limited blinding. Priorities include pragmatic RCTs with standardized outcomes, factorial antiviral+TCM designs, and mechanistic endpoints.[119–122] Overall, current evidence supports the potential for TCM modalities to complement Western care, while emphasizing the need for more rigorous and standardized research.

Integrated Western–TCM Approaches

Conceptual Rationale

Western medicine and Traditional Chinese Medicine (TCM) offer complementary therapeutic approaches. Western therapy focuses on viral suppression (antivirals), vaccination (prevention), and symptomatic pain control (analgesics and interventional procedures). In contrast, TCM emphasizes pathogen clearance, restoration of qi and blood flow, and neural repair using herbal medicines, acupuncture, and external therapies.

Integration seeks to address multiple dimensions of HZ/PHN pathophysiology simultaneously:

- Antiviral drugs reduce viral load and lesion burden, while herbal formulas modulate host immunity and inflammation.

Table 4 Acupuncture and external modalities used for herpes zoster (HZ) and postherpetic neuralgia (PHN): protocols, outcomes, safety, and evidence

Modality	Protocol / Key Parameters	Typical Points / Sites	Session Frequency & Course	Clinical Phase	Primary Outcomes Reported	Safety / Monitoring	Evidence Notes
Manual acupuncture	Segmental + distal approach; deqi sought; 20–30 min retention	Jiaji (EX-B2), Ashi, LI4, SJ5, GB34, ST36, LV3	2–5 sessions/week; 2–3 weeks acute, 4–8+ weeks PHN	Acute HZ & PHN	↓Pain, ↓allodynia, faster healing (acute); improved sleep/QoL (PHN)	Local soreness, minor bleeding/ bruising; rare syncope	Multiple RCTs & meta-analyses show benefit vs meds; heterogeneity in protocols
Electro-acupuncture (EA)	Biphasic stimulation, common 2/100 Hz alternating; 20–30 min	Jiaji + Ashi; add distal points (LI4, SJ5, GB34, ST36)	2–3 sessions/week; 4–6 weeks	Acute HZ & PHN	Greater pain reduction vs manual alone; improved responder rates	Same as acupuncture; avoid across chest in pacemaker patients	PHN RCTs suggest added benefit; frequency/dose–response not standardized
Wrist–Ankle Acupuncture (WAA)	Subcutaneous needling at wrist/ ankle zones; no deqi required	Standard WAA lines per affected dermatomes	Daily or QOD, taper after 1–2 weeks	Acute HZ (adjunct)	Higher pain-free rates at day 7; faster daily pain reduction	Minimal AEs; monitor for vasovagal reactions	Recent RCT (2025) positive; needs replication and longer follow-up
Fire needle (Huozhen)	Heated needle insertion at vesicle margins/Ashi points	Perilesional, segmental distribution	Every 2–3 days × 3–5 sessions during vesicular/early crusting	Acute HZ	↓Acute pain; faster crusting in small RCTs	Risk of burns/infection; avoid in immunocompromised or confluent lesions	Evidence mainly small trials; blinding/ quality often limited
Moxibustion	Indirect moxa over points /dermatomes or on needle handles	Jiaji, Ashi, ST36, GB34, dermatomal lines	2–3 sessions/week; 2–4 weeks acute, 4–8 weeks PHN	Acute HZ & PHN	↓Pain scores; improved composite efficacy in meta-analyses	Monitor for burns; ventilation for smoke; avoid sensory-impaired skin	Positive pooled effects; high heterogeneity and risk of bias
Cupping	Negative pressure cups 5–15 min along dermatomes	Paraspinal at affected levels, pain band	1–2 sessions/week; 2–4 weeks	Acute HZ (selected) & PHN (adjunct)	Symptom relief; improved ROM; limited RCTs	Skin ecchymosis; avoid anticoagulation/coagulopathy, fragile skin	Evidence low–moderate; best as adjunct
Gua sha (scraping)	Light–moderate scraping with smooth tool	Paraspinal and affected dermatomes (avoid vesicles)	Weekly × 2–4 sessions post-crusting	PHN (post-acute)	Subjective pain relief	Transient petechiae/ecchymosis; avoid anticoagulated/frail skin	Mainly observational/small trials
Topical herbal plasters/ liniments	Analgesic/anti-inflammatory herbal formulas	Over maximal tenderness (avoid broken skin)	Daily application; 1–3 weeks acute, PRN in PHN	Acute HZ (post-vesicular) & PHN	↓Local pain; improved clothing tolerance (allodynia)	Contact dermatitis risk; avoid open lesions	Formulations vary; need standardization + RCTs

- Analgesics and nerve blocks relieve nociception, while acupuncture and moxibustion regulate central sensitization and neuroplasticity.
- Western care improves acute outcomes, while TCM contributes to functional recovery, sleep, and quality of life.

This multimodal synergy is particularly relevant for older or medically complex patients, in whom single-modality therapy may provide incomplete relief.

Clinical Combinations in Practice and Research

Antiviral + Herbal Formula

Several RCTs and cohort studies report that adding heat-clearing and blood-activating formulas (eg, Jiedu Huoxue Decoction) to acyclovir or valacyclovir can accelerate rash crusting, reduce acute pain intensity, and shorten healing time compared with antivirals alone. Some studies also suggest lower PHN incidence at 90 days; however, results remain inconsistent due to heterogeneity in formula composition and trial quality.[39,123] Overall, findings are promising but require validation in standardized, higher-quality trials.

Antiviral + Acupuncture/Electroacupuncture (EA)

Evidence suggests that combining antivirals with manual acupuncture or EA yields superior acute pain control, fewer new vesicles, and improved functional recovery compared with antivirals alone. A 2025 RCT demonstrated that wrist–ankle acupuncture (WAA) combined with standard pharmacological therapy significantly increased complete pain relief on day 7 (87% vs 65%) without increasing adverse events.[124] These data support acupuncture as a clinically meaningful adjunct during the acute phase.

PHN Management: Gabapentinoids + Acupuncture ± Topicals

For established PHN, integrated regimens often combine gabapentinoids or TCAs with acupuncture/EA, sometimes supplemented with topical therapies (lidocaine, capsaicin, or TCM plasters). RCTs show that these combinations improve pain scores, responder rates (≥30% reduction), and sleep quality compared with pharmacotherapy alone. For example, lidocaine patches combined with acupuncture produce greater reductions in tactile allodynia.[77,79] Combination therapy appears beneficial, particularly for refractory neuropathic pain.

Complex Multimodal Packages

In integrated pain clinics, pragmatic protocols frequently use triple therapy (antiviral + acupuncture + herbal decoction) for acute HZ or gabapentin + EA + moxibustion/topical herbal plasters for PHN. Observational data suggest reduced opioid use and improved quality of life, although formal RCTs remain sparse.[41,125] These real-world models illustrate common clinical practice, but controlled evaluation is still needed.

Effectiveness Signals

Across trials and practice-based studies, integrated approaches consistently demonstrate:

- Faster rash resolution and shorter time to crusting when TCM is added to antivirals.
- Reduced acute pain intensity, particularly with acupuncture or fire needle.
- Lower incidence or earlier resolution of PHN, though data vary due to definitional heterogeneity.
- Improved sleep, mood, and quality of life—outcomes less frequently assessed in Western-only studies.

Systematic reviews and meta-analyses support these signals, although the certainty of evidence varies.[39,122,126,127] Taken together, results support potential benefit while highlighting the need for rigorous, methodologically consistent studies.

Mechanistic Synergy

Integration is biologically plausible because the interventions target distinct but convergent mechanisms:

- Antivirals suppress viral replication, while herbal medicines enhance immune clearance, suppress NF-κB and NLRP3 signaling, and reduce microglial activation.
- Analgesics/nerve blocks reduce peripheral excitability, while acupuncture/EA enhance descending inhibitory pathways and normalize central sensitization.
- Herbal neurotrophic effects (eg, astragaloside IV upregulating NGF/BDNF) may complement Western symptom control and support neural repair.

Systemic resilience from vaccination overlaps conceptually with TCM's strengthening of *wei qi*.

These mechanistic intersections provide a coherent biologic framework for integrative treatment strategies.

Safety and Interactions

Integration requires careful attention to herb–drug interactions and patient monitoring:

- *Panax notoginseng* (Sanqi) has antiplatelet/anticoagulant effects, relevant for patients on warfarin, clopidogrel, or DOACs.[128]
- Astragalus derivatives may interact with immunosuppressants and require caution in transplant recipients.[129]
- Acupuncture/moxibustion are generally safe but contraindicated in severe thrombocytopenia, uncontrolled diabetes, or infected lesions.[110,130]
- Quality assurance of herbal products (identity, contamination, adulteration) must follow WHO GMP standards.[131]

Effective integrated care requires structured monitoring frameworks, including medication reconciliation, interaction screening, and adverse event reporting.

Limitations and Research Needs

While integration shows promising signals, limitations remain.

Trial heterogeneity (different herbal formulas, acupuncture protocols, PHN definitions).

Small sample sizes and short follow-up (most $\leq$3 months).

Outcome variability, with reliance on composite "effective rate" instead of standardized pain/QoL measures.

Blinding challenges, particularly in acupuncture trials.

These issues limit comparability across studies and reduce the certainty of pooled estimates in meta-analyses.

Future directions include:

Pragmatic RCTs with standardized integrated protocols.

Factorial trial designs testing antiviral ± TCM and acupuncture dose–response.

Core outcome sets (pain intensity, responder rates, PHN incidence, QoL).

Mechanistic studies linking cytokine changes, small-fiber density, or neuroimaging with clinical outcomes.

Implementation research in real-world integrated care models, including cost-effectiveness analysis.

Addressing these gaps is essential for establishing reproducible and evidence-based integrated pathways.

Summary

Integrated Western–TCM approaches offer multidimensional benefits for HZ and PHN by combining viral suppression, immunomodulation, analgesia, and neural repair. Clinical evidence indicates improvements in rash healing, pain intensity, PHN risk, and QoL, although certainty remains limited by trial quality. Future studies with standardized protocols and long-term follow-up are needed to determine the true clinical value and scalability of integrated care.

Special Clinical Scenarios

Although most cases of herpes zoster (HZ) and postherpetic neuralgia (PHN) follow a relatively typical course, several clinical scenarios require nuanced management. These involve higher morbidity, unique therapeutic considerations, and the potential role of integrated Western–TCM approaches.

Herpes Zoster Ophthalmicus (HZO)

- Epidemiology and risk. HZO results from reactivation of VZV in the ophthalmic branch of the trigeminal nerve and accounts for 10–20% of all HZ cases.[132] The risk increases with age and immunosuppression.
- Complications. Ocular involvement may include conjunctivitis, keratitis, uveitis, retinitis, glaucoma, and optic neuritis, potentially leading to permanent vision loss.[53] Hutchinson's sign (vesicles on the nose tip) strongly predicts ocular involvement.
- Western management. Systemic antivirals (valacyclovir or acyclovir) should be initiated promptly, ideally within 72 h. Topical corticosteroids may be required for stromal keratitis or uveitis under ophthalmological supervision. Analgesia follows neuropathic pain guidelines.[133]
- Role of TCM. Acupuncture near the ocular structures is contraindicated during the acute infectious phase. Distal points (LI4, ST36, GB20) may be used for adjunctive analgesia and systemic regulation.[134] Herbal formulas such as Qingre Jiedu decoctions are traditionally used, although clinical evidence is limited.[39]
- Integration. Multidisciplinary care is essential. Early antiviral therapy remains central, while TCM may support systemic recovery and post-acute pain relief.

Cranial Neuropathies (Eg, Ramsay Hunt Syndrome)

- Clinical presentation. Ramsay Hunt syndrome results from reactivation in the geniculate ganglion, producing auricular vesicles, ipsilateral facial paralysis, hearing loss, and vertigo.[54] Prognosis is poorer than in idiopathic Bell's palsy.
- Western management. Early antiviral therapy plus corticosteroids improves facial nerve recovery. Neuropathic agents address neuralgia. ENT/neurology referral is indicated for audiovestibular involvement.[54,135]
- Role of TCM. Acupuncture points supporting facial motor recovery (ST4, ST6, LI4, SJ17) may aid rehabilitation. Herbs that "nourish qi and blood" (eg, Astragalus, Angelica sinensis) are used during recovery.[136]
- Integration. Combining Western antiviral/steroid therapy with acupuncture-based rehabilitation may enhance functional recovery, although most evidence is extrapolated from Bell's palsy studies.[137]

Immunocompromised Hosts

Burden. Immunocompromised patients (HIV, post-transplant, chemotherapy) have higher incidence, prolonged viral shedding, and more severe complications.[138]

Western management. High-dose IV acyclovir (10 mg/kg q8h) is standard for severe/disseminated disease. Oral valacyclovir/famciclovir may be used for stable patients. RZV vaccination is recommended in selected immunosuppressed populations.[23]

TCM considerations. Immunostimulatory herbs (Astragalus, Ginseng) require caution due to interactions with immunosuppressive drugs.[139] Acupuncture/moxibustion may be used only after lesions heal and systemic stability is achieved.[43]

Integration. Western antivirals and vaccination are foundational. TCM may aid symptom management and convalescence with careful interaction monitoring.

Recurrent or Disseminated HZ; Atypical Dermatomes

Clinical features. Recurrent zoster affects up to 6% of patients, especially the elderly and immunocompromised.[140] Disseminated HZ (>20 lesions outside the primary dermatome) risks visceral involvement. Atypical dermatomes may mimic other dermatologic conditions.[141]

Management. Requires systemic antiviral therapy, often IV. Evaluation for underlying immunodeficiency is recommended. Integrated care may include adjunctive herbal or acupuncture support only after acute viral control.

Frailty and Polypharmacy

Challenge. Frail older adults often have multiple comorbidities and high vulnerability to drug side effects.[142]

Integrated approach. Topical-forward strategies (lidocaine, capsaicin, herbal plasters) minimize systemic toxicity. Gentle acupuncture protocols may reduce pain without excessive stimulation. Herbal prescriptions should be simplified, with vigilant interaction checks. Integrated care is particularly valuable in this population, where low-toxicity multimodal regimens can improve comfort and function.

Summary. Special clinical scenarios—including HZO, Ramsay Hunt syndrome, immunocompromised hosts, recurrent/disseminated disease, and frailty—pose distinct challenges. Western care remains foundational, but appropriately selected TCM modalities may support pain relief, functional recovery, and systemic resilience when used within a multidisciplinary safety framework.

Digital Health and Remote Care

Digital tools are increasingly being leveraged in pain management and can be adapted for HZ/PHN.

- Smartphone apps and wearables can track daily pain scores, sleep, activity, and medication adherence.[143]
- Tele-acupuncture models (remote-guided self-acupressure, moxibustion devices) may extend access to TCM in rural areas.[144]
- Digital phenotyping may identify patients at highest risk for PHN, enabling earlier integrated interventions.
- Artificial intelligence (AI) tools such as thermal imaging may improve diagnosis and predict PHN development, supporting hybrid care models that combine Western prescriptions with digital-supported TCM self-care.[145]

Conclusion

Herpes zoster (HZ) and postherpetic neuralgia (PHN) remain conditions of substantial global burden, especially in aging and immunocompromised populations. Western medicine offers effective antivirals, preventive vaccination, and evidence-based pharmacologic and interventional pain management; however, its capacity to prevent or fully resolve PHN remains limited. Traditional Chinese Medicine (TCM) provides complementary modalities—including herbal formulations, acupuncture, and external therapies—that target inflammation, immune regulation, neural repair, and quality of life, although the evidence is heterogeneous and often methodologically constrained.

Emerging literature suggests that integrated Western–TCM approaches may offer multidimensional benefits, including accelerated rash healing, reduced acute pain, lower PHN incidence, and improved functional outcomes. Mechanistic data lend biological plausibility, with TCM interventions modulating pathways (eg, NF-κB, microglial activation, and neurotrophic signaling) that are not directly addressed by conventional therapies. These complementary actions highlight the potential for integrated care models to address gaps in both acute and chronic phases of the disease.

Nonetheless, the quality of evidence remains uneven, with challenges including small sample sizes, protocol heterogeneity, limited blinding, and underreporting of adverse events. Standardized diagnostic criteria, transparent reporting, and consistent outcome measures are needed to strengthen the evidence base. Future research should prioritize pragmatic multicenter randomized trials with standardized protocols, validated outcome measures, mechanistic endpoints, and long-term follow-up. Implementation science will also be critical for embedding integrated models into routine care, particularly in low-resource settings where cost and access barriers remain high.

In conclusion, the management of HZ and PHN is entering a new era. The integration of Western and TCM modalities represents a promising strategy to address unmet needs in viral suppression, pain control, neural repair, and quality of life. With rigorous evaluation and structured implementation, integrated approaches may evolve into a safe, scalable, and comprehensive standard of care for HZ and its complications.

Disclosure

The authors report no conflicts of interest in this work.

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