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Comparative Effectiveness Of Ultrasound-Guided Erector Spinae Plane Block And Intercostal Nerve Block In Acute Thoracic Herpetic Neuralgia Management

Hebatallah Mohamed Saeed Hussein[1], Alaa El-Din Mohamed El-Kassaby[2], Salah Abdel-Fattah Mohamed Ismail[2], Islam Gamal Mohamed Mohamed El-Bayyady[3], Eman Mohamed Abdel-Haleem Teeima[3]
[1]M.B.B.CH., M.Sc., Assistant Lecturer of Anesthesiology, Intensive Care and Pain Management, Suez Canal University.
[2]Professor of Anesthesiology and Intensive Care, Faculty of Medicine, Suez Canal University.
[3]Lecturer of Anesthesiology and Intensive Care, Faculty of Medicine, Suez Canal University.

ABSTRACT
Herpes zoster (HZ) is frequently associated with acute herpetic neuralgia (AHN), a painful condition that can progress to post-herpetic neuralgia (PHN), one of the most challenging chronic pain syndromes. Effective early management of AHN is essential to improve patient outcomes and reduce the risk of PHN. Regional analgesic techniques such as ultrasound-guided erector spinae plane block (ESPB) and intercostal nerve block (ICNB) have emerged as promising interventions alongside conventional pharmacological therapy. This review summarizes the current evidence on the comparative effectiveness of ESPB and ICNB in AHN, highlighting their mechanisms, clinical applications, and reported outcomes in terms of pain reduction, functional recovery, and prevention of PHN. Emerging studies suggest that both blocks provide meaningful analgesia, with ESPB demonstrating potential advantages in sustained pain relief and reduced analgesic requirements. While evidence remains limited, these techniques represent valuable additions to multimodal pain strategies, and further research is warranted to establish optimal protocols and long-term benefits.
Keywords: *Herpes zoster, Acute herpetic neuralgia, post-herpetic neuralgia, Erector spinae plane block, Intercostal nerve block, regional anesthesia.*

1. INTRODUCTION

Herpes zoster, more commonly known as shingles, is the painful reawakening of the varicella-zoster virus that once caused chickenpox. After lying dormant in the sensory nerves for years, the virus can suddenly reactivate, producing burning skin eruptions and sharp, often unbearable pain. This acute pain, called acute herpetic neuralgia (AHN), doesn't just cause physical suffering it disrupts daily activities, lowers quality of life, and often drives patients to seek repeated medical care. What makes it even more concerning is that the longer the pain persists in the first episode, the higher the chance it will recur or evolve into a chronic problem [1].

Even after the rash fades, many patients continue to live with lingering pain that can stretch on for months. This condition, known as post-herpetic neuralgia (PHN), is one of the most feared complications of shingles, particularly in older adults. PHN follows the path of the original rash along the nerves, most often in the chest or face, and is marked by burning, stabbing sensations, touch sensitivity, and abnormal skin feelings. It is not only distressing but also difficult to treat once established. Because of this, modern pain management has shifted its focus: rather than waiting for PHN to appear, the goal is to stop it at the acute stage. Antiviral drugs combined with strong multimodal pain control are considered the best way to reduce nerve damage and long-term complications. Alongside oral medications, doctors are increasingly turning to interventional approaches such as nerve blocks [2].

Two techniques in particular have attracted attention: the intercostal nerve block (ICNB), a traditional method that provides quick pain relief but often requires multiple injections, and the more recently introduced erector spinae plane block (ESPB), which is easier to perform, safer, and capable of covering

larger areas of pain with a single injection. Both methods show promise in easing acute pain and possibly lowering the risk of PHN, but there is still debate about which works best [5].
This makes it important to explore their effectiveness side by side, to better understand how these blocks can improve the lives of patients suffering from the intense pain of shingles.

Varicella Zoster Virus and Post-Herpetic Neuralgia

Varicella zoster virus represents one of medicine's most intriguing examples of viral persistence and reactivation. This remarkable pathogen demonstrates the complex relationship between infectious agents and the human nervous system, creating a lifelong connection that can resurface decades after the initial encounter. Understanding this virus and its long-term consequences has become increasingly important as our population ages and more individuals face the risk of painful complications.
The story of varicella zoster virus is essentially a tale of two diseases affecting the same person at different stages of life. What begins as the familiar childhood illness of chickenpox can later return as the painful condition known as shingles, potentially leaving behind chronic pain that significantly impacts quality of life [1]. This duality makes the virus unique among common human pathogens and presents ongoing challenges for healthcare providers worldwide.
Varicella zoster virus belongs to the herpes virus family and serves as the causative agent for both chickenpox and shingles [2]. This dual role makes it particularly fascinating from a medical perspective, as few pathogens can cause such distinctly different clinical presentations in the same individual. When someone first encounters this virus, typically during childhood, it begins its journey in the respiratory tract before systematically spreading throughout the body, ultimately manifesting as the characteristic chickenpox rash after an incubation period of 10-21 days [3].
The initial infection process reveals the virus's sophisticated survival strategy. Rather than simply causing acute illness and disappearing, varicella zoster virus demonstrates remarkable evolutionary adaptation by establishing a permanent residence within the human nervous system [4]. During the primary infection, viral particles travel along nerve pathways to reach nerve cell clusters called dorsal root ganglia, where they settle into a dormant state that can persist for decades. This dormancy represents a delicate balance between viral survival and host immune surveillance.
What makes this virus particularly noteworthy is its ability to remain completely silent for years while maintaining the potential for reactivation. Unlike other infections that either resolve completely or cause ongoing symptoms, varicella zoster virus exists in a state of controlled hibernation [5]. This latent phase can last from childhood through old age, with the virus patiently waiting for the right conditions to become active again. The trigger for reactivation often involves weakening of the immune system, whether through aging, stress, illness, or immunosuppressive treatments.

The Molecular Architecture of Viral Survival

The structural complexity of varicella zoster virus reflects its sophisticated approach to survival and replication [6]. The virus presents itself as a multi-layered entity, with each component serving a specific purpose in its lifecycle. At its outermost layer, a lipid-rich envelope derived from host cellular membranes houses essential viral proteins that facilitate cellular entry and immune evasion. This envelope isn't merely a protective covering but represents a sophisticated interface that allows the virus to communicate with and manipulate host cells.
Beneath this envelope lies the tegument layer, a protein-rich region that serves as the virus's toolkit for immediate infection processes [7]. These proteins are released immediately upon cellular entry, allowing the virus to quickly establish control over host cellular machinery before the cell's natural defenses can respond effectively. This rapid takeover represents millions of years of evolutionary refinement, resulting in a pathogen exquisitely adapted to human cellular biology.
The viral core contains the genetic blueprint that orchestrates this entire process - a linear double-stranded DNA genome protected within an icosahedral nucleocapsid structure [8]. This geometric

arrangement isn't accidental but represents an optimal solution for packaging genetic material while maintaining structural integrity during the harsh journey between hosts.

The Cellular Infection Process

When varicella zoster virus encounters a susceptible cell, it employs one of two sophisticated entry mechanisms [9]. The first involves direct fusion with the cellular membrane, a process that requires precise molecular recognition between viral surface proteins and cellular receptors. This fusion process represents a remarkable feat of biological engineering, as the virus must successfully breach the cell's primary defense barrier while avoiding detection by cellular surveillance mechanisms.

Alternatively, the virus can gain entry through endocytosis, essentially tricking the cell into voluntarily engulfing viral particles. Once inside, the virus faces the challenge of navigating the cellular environment to reach its ultimate destination - the nucleus, where cellular DNA replication machinery resides. The viral tegument proteins play a crucial role during this phase, immediately beginning their work to reprogram cellular functions in favor of viral reproduction [10].

The journey from initial cellular entry to the production of new viral offspring takes approximately 9-12 hours, a timeline that reflects the virus's need to balance rapid reproduction with stealth [11]. Too fast, and cellular alarm systems might successfully mount a defense; too slow, and competing cellular processes might interfere with viral replication. The virus achieves this balance through precise coordination of viral gene expression and hijacking of cellular translation mechanisms.

The final stages of viral reproduction involve a complex process of assembly and packaging within specialized cellular compartments, particularly the trans-Golgi network [12]. This cellular hijacking represents one of virology's most elegant examples of pathogen adaptation, transforming normal cellular processes into viral production facilities while maintaining enough cellular function to sustain the infection process.

Active Infection Versus Latent Survival

The transition from active viral replication to dormant persistence represents one of the most fascinating aspects of varicella zoster virus biology [13]. During active infection within dorsal root ganglia, the virus operates at full capacity, expressing genes necessary for protein synthesis, genome replication, and viral particle assembly. This active state affects both neurons and their supporting satellite cells, creating a localized environment of intensive viral activity that paradoxically leads to its own suppression.

The cellular environment during active infection becomes a battleground between viral replication machinery and host cellular defense mechanisms [14]. Specialized cellular structures called promyelocytic leukemia nuclear bodies emerge as crucial players in this conflict, effectively creating molecular cages that trap newly formed viral particles. These structures represent the cellular equivalent of quarantine facilities, preventing viral spread while allowing the host cell to survive.

The establishment of latency requires a fundamental shift in viral behavior. Rather than continuing aggressive replication, the virus adopts a minimalist approach, maintaining only essential genetic elements while shutting down production machinery [15]. This transition involves the preservation of viral genomes and specific RNA transcripts while halting the synthesis of proteins required for active infection. The virus essentially enters a state of suspended animation, maintaining just enough activity to ensure survival without triggering host immune responses.

This latent state represents an evolutionary masterpiece of pathogen persistence. The virus must maintain genetic integrity over potentially decades while remaining responsive to reactivation signals [16]. It achieves this through a carefully orchestrated reduction in metabolic activity that allows it to persist in post-mitotic neurons that rarely divide. The balance is so delicate that disruption of specific viral proteins can prevent proper latency establishment, leading to continued low-level viral production and tissue destruction.

The molecular mechanisms governing this transition involve complex interactions between viral

proteins, particularly glycoproteins E and I [17]. When these proteins fail to interact properly, the virus cannot successfully enter latency, resulting in continued tissue damage through persistent viral replication. This observation has provided valuable insights into potential therapeutic targets for preventing both acute infection complications and chronic pain development.

The Clinical Spectrum: From Acute Pain to Chronic Suffering

Pain represents one of medicine's most complex and poorly understood phenomena, encompassing not merely physical sensations but emotional, psychological, and social dimensions that profoundly impact human experience [18]. The International Association for the Study of Pain recognizes this complexity in defining pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. This definition acknowledges that pain transcends simple nerve signal transmission, involving sophisticated processing mechanisms that can amplify, diminish, or completely alter the perception of painful stimuli.

Neuropathic pain, the category encompassing post-herpetic neuralgia, represents a particularly challenging subset of pain disorders [19]. Unlike normal pain that serves a protective function by alerting us to tissue damage, neuropathic pain results from dysfunction within the nervous system itself. This creates a situation where the pain signaling system becomes the source of suffering rather than serving its intended protective role. The nervous system, designed to accurately detect and transmit information about harmful stimuli, instead generates false signals that create the perception of ongoing tissue damage even when none exists.

The Emergence of Acute Herpes Zoster

Acute herpes zoster, commonly known as shingles, represents the clinical manifestation of varicella zoster virus reactivation after years or decades of dormancy [20]. This reactivation typically occurs when immune surveillance mechanisms weaken, allowing the latent virus to resume active replication and spread from nerve clusters to the skin areas they serve. The process creates a perfect storm of viral activity, immune response, and tissue inflammation that generates intense pain and lasting neurological changes.

Most individuals remain unaware that shingles can evolve into a chronic pain condition that persists long after the visible rash disappears [21]. This lack of awareness often leads to inadequate early treatment and missed opportunities for preventing long-term complications. The transformation from acute viral illness to chronic pain syndrome represents one of medicine's most frustrating examples of how temporary infections can create permanent suffering.

The clinical presentation of acute herpes zoster follows a predictable pattern that reflects the underlying viral biology [22]. Initial symptoms often include general malaise and fever, followed by the development of characteristic pain in a specific nerve distribution pattern called a dermatome. This pain typically precedes the appearance of any visible skin changes, creating a diagnostic challenge for healthcare providers who must recognize shingles based on pain patterns alone.

Epidemiological Patterns and Population Impact

The global burden of herpes zoster reflects both the universal nature of varicella zoster virus infection and the aging of world populations [23]. Annual incidence rates vary geographically but consistently demonstrate the increasing risk associated with advancing age. Countries with comprehensive surveillance systems report incidence rates ranging from approximately 3 to 5 cases per 1,000 individuals annually, with dramatic increases in older age groups [24,25,26,27].

The age-related increase in shingles incidence represents more than simple statistical correlation - it reflects fundamental changes in immune system function that occur with aging [28]. At age 60, approximately 6-8 individuals per 1,000 will develop shingles annually. By age 80, this rate increases to over 800 per 1,000, representing more than a 100-fold increase in risk. These statistics translate to a

lifetime risk of approximately 30%, meaning that nearly one in three individuals will experience shingles at some point in their lives [29].

These epidemiological trends have profound implications for healthcare systems worldwide [30]. As populations age and medical advances allow more individuals to live with conditions that compromise immune function, the absolute number of shingles cases continues to increase. Cancer patients undergoing chemotherapy, organ transplant recipients, and individuals with autoimmune diseases all face elevated risks that compound the natural increase associated with aging.

The economic implications extend far beyond direct medical costs [31,32]. Lost productivity, caregiver burden, and long-term disability associated with chronic pain create substantial indirect costs that affect families, employers, and society as a whole. Conservative estimates suggest that annual costs associated with shingles and its complications reach billions of dollars in developed countries, with expectations for continued increases as demographic trends continue.

The Clinical Journey: From Warning Signs to Resolution

The clinical manifestation of herpes zoster follows a characteristic timeline that provides important clues for early diagnosis and intervention [33]. The journey typically begins with a prodromal phase characterized by dermatomal discomfort that can precede visible skin changes by several days to more than three months in some documented cases [34]. This preliminary phase often puzzles both patients and healthcare providers, as the pain lacks obvious external signs that might explain its presence.

During this prodromal period, patients frequently describe sensations that seem disproportionate to any visible pathology [35]. The discomfort may manifest as burning, tingling, itching, or deep aching sensations that follow specific nerve distribution patterns. These sensations represent the early stages of viral reactivation and neural inflammation, occurring as the virus begins its journey from dormant nerve clusters toward the skin surface.

The anatomical distribution of herpes zoster reflects the segmental organization of the human nervous system [36]. The thoracic region, encompassing the chest and upper back, accounts for 50-70% of all cases, likely due to the large number of thoracic nerve segments and their extensive skin coverage. Cranial involvement, particularly affecting the ophthalmic division of the trigeminal nerve, represents 10-20% of cases and carries special significance due to potential vision-threatening complications. Cervical and lumbar involvement each account for similar percentages, while sacral involvement remains relatively uncommon at 2-8% of cases.

The evolution from prodromal symptoms to active rash typically occurs over several days, with the characteristic vesicular eruption appearing in a unilateral dermatomal distribution [37]. This unilateral pattern represents one of shingles' most distinctive features, reflecting the virus's residence in individual nerve clusters that serve only one side of the body. The rash progression follows a predictable sequence: initial erythematous patches evolve into fluid-filled vesicles, which subsequently rupture and crust over before eventually healing completely within 2-4 weeks.

Pain Characteristics and Their Clinical Significance

The pain associated with herpes zoster encompasses a broad spectrum of sensory abnormalities that reflect the complex pathophysiology underlying nerve dysfunction [38]. Patients commonly report burning sensations, deep aching, sharp stabbing pains, tingling, and paradoxically, intense itching in areas that simultaneously experience pain. This diverse symptomatology reflects damage to different types of nerve fibers, each contributing its own characteristic sensation to the overall pain experience.

The timing of pain onset provides important insights into the underlying disease process [39]. Individuals who experience painful prodromal symptoms typically continue to have pain throughout the acute phase and face higher risks for developing chronic complications. Conversely, those who develop pain concurrent with rash appearance may have different underlying mechanisms that could influence both treatment responses and long-term outcomes.

The intensity and quality of acute pain often correlate with the extent of underlying nerve damage and inflammation [40]. Severe burning pain suggests significant involvement of small unmyelinated nerve fibers, while deep aching may indicate involvement of larger myelinated fibers. Sharp, electric shock-like sensations often reflect irritation of nerve roots near the spinal cord; while tingling and numbness suggest partial nerve dysfunction rather than complete destruction.

Some patients develop a particularly puzzling condition known as zoster sine herpete, where characteristic dermatomal pain occurs without any visible rash [41]. This presentation challenges traditional diagnostic approaches and requires careful evaluation to differentiate from other causes of dermatomal pain. The presence of varicella zoster virus DNA in cerebrospinal fluid from some of these patients supports the concept that viral reactivation can cause pain without necessarily producing visible skin lesions.

Beyond Pain: The Broader Impact of Herpes Zoster

The consequences of herpes zoster extend far beyond the immediate discomfort of acute infection [42]. Neurological complications can include motor weakness when the virus affects nerve fibers controlling muscle function, typically occurring in 1-5% of patients. This weakness usually resolves gradually over weeks to months but can leave some individuals with permanent functional impairment.

Ophthalmological complications represent some of the most serious acute consequences of herpes zoster, particularly when the infection involves the ophthalmic division of the trigeminal nerve [43]. These complications can include corneal involvement leading to scarring and vision loss, increased intraocular pressure, and secondary glaucoma. Early recognition and aggressive treatment of ophthalmic zoster are essential for preserving vision and preventing permanent eye damage.

Cutaneous complications beyond the typical rash can include secondary bacterial infections, particularly in immunocompromised patients or those with diabetes [44]. These secondary infections can lead to cellulitis, abscess formation, or in severe cases, necrotizing fasciitis requiring emergency surgical intervention. Proper wound care and monitoring for signs of bacterial superinfection represent important aspects of comprehensive herpes zoster management.

Visceral complications, while uncommon, can occur when the virus affects nerve fibers supplying internal organs [45]. These complications might include gastrointestinal dysfunction, bladder dysfunction, or in rare cases, involvement of the central nervous system leading to meningitis or encephalitis. Such complications are more common in immunocompromised patients and require specialized medical management.

Impact on Patients and Healthcare Systems

The impact of post-herpetic neuralgia on patients' quality of life is significant [46]. As a result of their constant pain, many individuals suffer severe physical, occupational, social, and psychosocial problems. Drug dependence, hopelessness, depression, and even suicide might result from ongoing suffering and the medication's poor effectiveness [47].

Regarding immediate financial costs and missed productivity, family and community are also impacted [48]. As a result, failing to avoid post-herpetic neuralgia incurs enormous costs for both the patient and the health care system. Annual national spending on post-herpetic neuralgia in the United Kingdom was estimated to be between £4.8 and £17.9 million in 1994 [49]. The cost of controlling post-herpetic neuralgia over a lifetime was projected to be £770 per patient. Another study indicated that the entire cost of treating herpes zoster in England and Wales in 1998 was £47.6 million [50].

Understanding the Pain Mechanisms

Acute Pain Development

When the virus reactivates, it causes inflammation in both the nerve and surrounding skin [51]. This inflammation produces chemicals that make pain receptors more sensitive and likely to fire. The

continuous stimulation leads to changes in how the nervous system processes pain signals, making the area hypersensitive to touch and causing spontaneous pain [52,53].
At the cellular level, changes occur in sodium and potassium channels in nerve cells, making them more likely to send pain signals [54]. Additionally, the body's natural pain-blocking systems become less effective. Evidence at the cellular level revealed an increase in the number of subtypes of voltage-gated sodium channels, changes in voltage-gated potassium channels, and activation of pain receptors [55].

Chronic Pain Development

The sympathetic nervous system, which normally operates independently from pain nerves, can become coupled with pain pathways after nerve injury [56]. This coupling can amplify pain signals. In acute shingles, viral reactivation causes severe inflammation that triggers sympathetic nervous system activation, leading to blood vessel constriction and reduced blood flow to nerves. This creates a cycle of nerve damage and increased pain sensitivity [57].
Two main mechanisms explain chronic post-shingles pain [58]:
Sensitization: The nervous system becomes hypersensitive, causing normal touch to be perceived as painful and increasing sensitivity to all stimuli [59]. Patients report mechanical allodynia and normal or hyperalgesic thermal sensation due to irritable nociceptors, which are functionally abnormal but anatomically intact primary afferent nociceptors.
Deafferentation: Large nerve fibers that normally help block pain signals are damaged more than smaller pain fibers [60]. This disrupts the natural balance and allows more pain signals to reach the brain. Additionally, touch fibers may inappropriately connect to pain pathways, causing light touch to trigger severe pain. Dynamic and tactile allodynia is caused by nerve fiber rewiring in the dorsal root ganglia, which connects to the pain-transmitting spinothalamic tracts.
The pathophysiology of post-herpetic neuralgia pain may be influenced by gate-control disturbance [61]. When large myelinated afferents are lost most, their ability to stop small diameter C-fiber nociceptive afferents from sending pain signals to the brain is lost.

Current Treatment Approaches

Managing shingles is challenging because there are no universally accepted treatment guidelines. The goals are to treat the viral infection, control acute pain, and prevent chronic pain development [62].

Antiviral Medications

All immunocompetent patients who meet specific criteria should receive systemic antiviral treatment for acute herpes zoster during the first 72 hours of rash onset [63]. Antiviral medications (acyclovir, valacyclovir, and famciclovir) lower the severity and duration of acute pain, speed up rash healing, and minimize the viral shedding period [64]. At 6 months, proper antiviral medication reduced post-herpetic neuralgia by 50% [65].

Corticosteroids

Corticosteroids have anti-inflammatory properties that may reduce nerve injury and post-herpetic neuralgia risk [66]. Combined antivirals and corticosteroids therapy enhanced acute herpes zoster pain control and patient quality of life. It facilitated a faster return to normal daily activities and sleeping patterns. Combined therapy should be considered only in individuals with severe symptoms at presentation due to their side effects [67].

Pain Management Options

Topical treatments: During healing, when temperature sensitivity is the primary concern, topical treatments can help manage symptoms [68].
Basic pain relievers: Acetaminophen and nonsteroidal anti-inflammatory drugs can help manage acute

herpes zoster pain [69].

Antidepressants: When treating acute herpes zoster pain, antidepressants may be helpful [70]. They lessen the incidence of post-herpetic neuralgia and reduce the major sleep disturbance. If amitriptyline is taken within 48 hours of the onset of the rash, there has been a 50% reduction in pain prevalence at six months.

Anticonvulsants: Anticonvulsants may be useful in addition to sympathetic neural block in the treatment of acute herpes zoster pain [71,72]. The Food and Drug Administration approved gastro-retentive gabapentin as an extended-release gabapentin formulation for the treatment of post-herpetic neuralgia in 2011. Over 90% of patients attain the recommended dose of 1,800 mg/day gastroretentive gabapentin within 2 weeks [73].

Opioids: Opioids are commonly used to alleviate severe pain in the short term [74]. Tramadol and oxycodone help relieve acute herpes zoster pain.

Nerve Block Procedures

Erector Spinae Plane Block (ESPB)

This innovative technique involves using ultrasound guidance to inject local anesthetic between the erector spinae muscle and the underlying spine bone [75]. The procedure is performed with the patient sitting comfortably while a ultrasound probe is placed about 3 cm to the side of the spine. The doctor can see three distinct muscle layers on the ultrasound screen, and carefully guides a needle between specific muscle layers to deliver the medication [76].

The technique involves identifying the trapezius, rhomboid major, and erector spinae muscles that are superficial to the hyperechoic transverse process shadow. The needle is then placed until the tip is in the interfascial plane between the rhomboid major and erector spinae muscles [77]. The procedure has been successfully performed with patients in sitting, prone, and lateral positions [78,79,80].

While the exact mechanism isn't fully understood, this "fascial plane block" appears to work by spreading anesthetic along tissue planes to block multiple nerve branches [81]. Studies using cadavers and imaging have shown that the medication reliably covers the back branches of spinal nerves, and often affects the front branches as well, though the spread is somewhat unpredictable [82].

Medical societies classify this as a "superficial low-risk block," meaning it's generally safe even for patients taking blood-thinning medications [83]. Major complications like collapsed lung, motor weakness, or drug toxicity are extremely rare [84]. The main advantage is its simplicity - the ultrasound anatomy is easier to learn compared to other nerve blocks, making it more forgiving for practitioners [85].

However, complications can occur. Pneumothorax was described in two case reports [86,87]. The failure of the procedure and an involuntary motor block brought on by a low thoracic erector spinae plane block must also be highlighted as potential block-related problems [88,89].

Intercostal Nerve Block

This established technique targets the nerves that run along the ribs, providing pain relief for various chest wall conditions including shingles, post-surgical pain, and nerve injuries [90,91].

The intercostal nerves are branches of spinal nerves that travel along the underside of each rib in a groove [92]. These nerves run alongside blood vessels in what's called a neurovascular bundle. The close relationship with blood vessels explains why this type of block can lead to higher absorption of local anesthetic into the bloodstream.

Using ultrasound guidance, the doctor places the probe about 4 cm to the side of the spine and identifies the ribs and the space between them [93]. The needle is carefully advanced until it reaches just below the rib, where the local anesthetic is injected. The ultrasound allows real-time visualization of important structures like the lung lining, ensuring safer needle placement.

Real-time visualization of blood vessels and lung tissue, ability to use smaller volumes of medication, more precise needle placement closer to the spine, and better chance of blocking the entire nerve before

it branches [94,95].
The technique is highly dependent on operator experience [96]. Ultrasound image quality can vary based on the practitioner's skill level, and deeper tissues may be harder to visualize clearly. Rib shadows can sometimes interfere with seeing the nerve bundle clearly [97].
The main risks stem from the nerve's close proximity to lungs and blood vessels [98]. Potential complications include lung puncture (pneumothorax) with risk varying from less than 1% to 19%, bleeding especially in patients on blood thinners, accidental injection into blood vessels causing systemic toxicity, rare instances of medication reaching the spinal canal, and infection at the injection site [99,100,101,102].

Clinical Evidence for Nerve Blocks in Shingles Treatment

Research Findings on ESPB

Several studies have demonstrated the effectiveness of erector spinae plane blocks for shingles pain. El-Sayed et al. investigated the role of ultrasound-guided erector spinae plane block in acute herpetic neuralgia and post-herpetic neuralgia prevention [103]. Ultrasound-guided erector spinae plane block reduced pain intensity more than medical treatment alone, improved patient satisfaction, and reduced drug use earlier in treatment.
Lin et al. tested erector spinae plane block for its effect on post-herpetic neuralgia in elderly patients with acute or subacute herpes zoster [104]. Sequential erector spinae plane block for 3 days reduced post-herpetic neuralgia at 12 weeks, enhanced analgesia at one week and 12 weeks, and reduced neuropathic pain, poor sleep, and anxiety/depression.
Abdelwahab et al. examined the efficacy and safety of one bolus injection thoracic paravertebral block and erector spinae plane block in avoiding post-herpetic neuralgia in acute thoracic herpes zoster [105]. After 6 months, both techniques controlled acute and persistent herpetic pain, but erector spinae plane block was safer with no pneumothorax or hypotension.

Research on Intercostal and Paravertebral Blocks

Zhao & Mei examined the effects of ultrasound-guided paraspinal nerve block on herpes zoster [106]. The observation group received thoracic paravertebral block with ultrasound guidance. Visual analogue scale scores, skin healing time, and post-herpetic neuralgia incidence were all lower in the observation group than in the control group.
Ma et al. compared ultrasound-guided thoracic paravertebral block to routine antiviral therapy for acute herpes zoster [107]. Three and six months after inclusion, the thoracic paravertebral nerve block group had significantly lower post-herpetic neuralgia rates than control group. The thoracic paravertebral nerve block group had better quality of life throughout.
The growing body of evidence suggests that nerve blocks, particularly erector spinae plane block and paravertebral blocks, offer several advantages: enhanced pain control superior to medication alone for acute pain management, prevention focus that may reduce the risk of developing chronic pain, improved recovery with faster return to normal activities and better sleep patterns, safety profile that is generally well-tolerated with low complication rates when performed by experienced practitioners, and patient satisfaction with higher satisfaction scores compared to medication-only approaches [108,109,110].

CONCLUSION

Varicella zoster virus causes a spectrum of conditions from initial chickenpox to reactivated shingles and potentially chronic post-herpetic neuralgia. Understanding the virus's behavior, the mechanisms of pain development, and available treatment options is crucial for effective management. Early recognition and treatment of acute shingles may help prevent the development of chronic pain, which can have devastating effects on patients' quality of life. A multimodal approach combining antiviral therapy,

appropriate pain management, and potentially nerve block procedures offer the best chance for optimal outcomes. Both erector spinae plane blocks and intercostal nerve blocks represent practical, minimally invasive approaches that can be tailored to individual patient needs and clinical circumstances. Their effectiveness in managing acute herpetic pain, combined with their favorable safety profiles, positions them as important tools in our ongoing efforts to optimize care for patients suffering from this challenging condition.

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