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Current Treatment Progress in Intractable Neuralgia Subsequent to Ramsay Hunt Syndrome: A Mini Review

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Abstract

Ramsay Hunt Syndrome (RHS) is a neurological disorder triggered by the reactivation of the varicella-zoster virus (VZV) in the facial nerve, resulting in facial paralysis and neuralgia. Some atypical RHS, however, can lack the characteristic vesicular rash and present with additional cranial nerve involvement complicate diagnosis and management. In all complications, intractable neuralgia is the most challenging sequelae of RHS, which severely impact the quality of life. The complexity of neuralgia in RHS arises from the involvement of multiple cranial nerves pathways, making it resistant to conventional therapies. Current treatments include antivirals, corticosteroids, and pain management strategies, but they are often insufficient in providing long-term relief for neuralgia. Neuromodulation techniques, particularly peripheral nerve stimulation (FNS), have emerged as promising alternatives. These less invasive treatments target specific neural circuits responsible for pain transmission, offering significant benefits for patients with refractory neuralgia. As evidence supporting the efficacy of neuromodulation grows, it represents a critical advancement in the management of RHS, especially in atypical cases where traditional therapies fail to provide relief. Further research is needed to refine these techniques and understand their long-term impact.

Keywords: Ramsay-Hunt Syndrome; Neuralgia; Facial Paralysis; Pharmacotherapy; Neuromodulation

Abbreviations: RHS: Ramsay Hunt Syndrome; VZV: Varicella-Zoster Virus; FNS: Peripheral Nerve Stimulation; PHN: Postherpetic Neuralgia; CN: Cranial Nerve; TENS: Transcutaneous Electrical Nerve Stimulation; PRF: Pulsed Radiofrequency; MCS: Motor Cortex Stimulation

Introduction

Ramsay-Hunt Syndrome (RHS) is a rare neurological disorder caused by the reactivation of the varicella-zoster virus (VZV) in the geniculate ganglion of the facial nerve. It is estimated that the incidence is around 5 cases per 100,000 people annually, and accounts for approximately 7-10% and even up to 16.7% of all facial palsy cases, ranking as the second most common cause of acute facial paralysis after Bell's palsy [1-3]. RHS is more common in older adults, particularly those over 60 years, and immunocompromised individuals such as diabetes and HIV, due to increased susceptibility to varicella-zoster virus (VZV) reactivation [4]. In addition, other risk Factors also include stress, trauma, or other triggers of VZV reactivation. Epidemiological data estimate that approximately 1% herpes zoster cases are herpes zoster oticus, which can lead to RHS [5]. Recent study has suggested a potential link between viral infections and RHS occurrence [6].

The complex pathogenesis and clinical manifestations of RHS, particularly the severe neuralgia in some cases, which complicates both diagnosis and treatment strategies. The goal is to reduce the severity and duration of the condition, minimize pain, and improve the chances of recovery. The management of neuralgia, which is often one of the most distressing features of RHS, requires a multifaceted approach. In this targeted review, we briefly summarize and synthesize several areas of research. In the following sections, we review the pathophysiology and clinical presentation, summary the neuralgia characteristics, and introduce the current treatments and recent progress for the neuralgia. Finally, we discuss the limitations of this review.

Clinical Presentation in Typical and Atypical RHS

RHS can be classified into typical and atypical forms based on the clinical presentation and associated symptoms. The

differentiation is primarily based on the presence or absence of classic triad symptoms and other neurological or systemic manifestations. The typical RHS is marked by unilateral facial paralysis affecting the lower motor neurons of CN VII, vesicular eruptions around the external ear, ear canal, or on the eardrum, and otalgia [4,7]. The atypical RHS cases may lack the characteristic vesicular rash and the usual pattern of facial paralysis, and present with additional cranial nerve involvement complicate diagnosis and management.

Vestibular dysfunction like dizziness or sensorineural hearing loss may occur if the virus extends to the vestibulocochlear nerve (CN VIII) [8,9]. Involvement of other cranial nerves, such as the trigeminal nerve (CN V) [8-11] glossopharyngeal nerve (CN IX) [8,9,12-14] and vagus nerve (CN X) [8,9,12] can lead to symptoms like dysphagia (difficulty swallowing), hoarseness, pain in the throat, or trigeminal neuralgia. Of these, intractable neuralgia is the most challenging sequelae of RHS, which severely impact the quality of life. Correlation of Nerves Affected, Clinical Presentation, and Pathophysiology in Typical and Atypical RHS exhibited in (Table 1).

Table 1: Correlation of Nerves Affected, Clinical Presentation, and Pathophysiology in Typical and Atypical RHS.

Cranial Nerve Affected	Clinical Presentation	Pathophysiology	Typical RHS / References	Atypical RHS / References
CN VII (Facial Nerve)	1. Facial 2. Paralysis 3. Hyperacusis loss of taste (anterior 2/3 tongue) 4. Vesicles in external ear 5. Otalgia	VZV reactivation in the geniculate ganglion leads to inflammation, edema, and compression of the facial nerve, causing facial motor deficits and sensory dysfunction	Yes Goswami Y, et al. 2023 [4] Yu C, 2025 [7]	Yes Ananthapadmanabhan et al., 2021 [8] Ani RMA 2022 [12]
CN VIII (Vestibulocochlear Nerve)	1. Vertigo 2. Tinnitus 3. Sensorineural hearing loss 4. Dizziness 5. Imbalance	Spread of VZV from geniculate ganglion to vestibular/cochlear nerve, leading to viral labyrinthitis and inflammatory damage to the cochlea	Yes Goswami Y, et al. 2023 [4]	Yes Ananthapadmanabhan et al., 2021 [8] Shinha T, et al. 2015 [9]
CN V (Trigeminal Nerve)	1. Facial pain (electric shock-like) 2. Jaw pain 3. Trigeminal neuropathy	Viral inflammation spreads to trigeminal ganglion, causing sensory disturbances and neuropathic pain via afferent fibers of CN V	No	Yes Ananthapadmanabhan et al., 2021 [8] Shinha T, et al. 2015 [9] Jin HS, et al. 2012 [10] Anam N, et al. 2021 [11]
CN IX (Glossopharyngeal Nerve)	1. Oropharyngeal pain 2. Dysphagia 3. Taste loss (posterior 1/3 tongue)	Involvement of inferior ganglion of CN IX leads to dysfunction of sensory pathways from the pharynx and tongue, affecting swallowing	No	Yes Ananthapadmanabhan et al., 2021 [8] Shinha T, et al. 2015 [9] Ani RMA. 2022 [12] Lee KM, et al. 2017 [13] Zhang J, et al. 2020 [14]
CN X (Vagus Nerve)	1. Hoarseness 2. Dysphagia, 3. Autonomic dysfunction (bradycardia)	Viral spread affects nucleus ambiguus and autonomic fibers, leading to motor dysfunction (larynx/pharynx) and autonomic dysregulation	No	Yes Ananthapadmanabhan et al., 2021 [8] Shinha T, et al. 2015 [9] Ani RMA. 2022 [12]

Pathophysiology

RHS results from aricella-zoster virus (VZV) reactivation, which remains latent in the geniculate ganglion of the facial nerve (cranial nerve VII) after primary chickenpox (varicella) infection. Once reactivated, VZV rapidly proliferates in geniculate ganglion and spreads along the facial nerve. Which induces neuroinflammation and edema that increase pressure within the facial canal, leading to nerve compression and ischemia and worsening nerve dysfunction. VZV also triggers demyelination and axonal degeneration, impairing nerve conduction and leading to prolonged facial dysfunction and pain. In addition, the immune

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response against VZV may worsen nerve damage, leading to prolonged or incomplete recovery [15] Some atypical cases without a vesicular rash may result from a less localized or subclinical viral reactivation, and other atypical cases with additional cranial nerve involvement, including cranial nerves V, VIII, IX and X, cause a range of neurological symptoms [4]. Although the mechanism of atypical RHS with cranial polyneuropathy is unclear, some theories are proposed to explain viral dissemination, such as virus spreads of proximity by contiguous nerves [1,16]. penetration of cerebrospinal fluid and inner ear fluid in the inner ear [17], Haematogenous dissemination [18] and deficits of cranial nerves following the microinfarctions of brainstem [19] (Table 1).

The Neuralgia Characteristics Between Typical and Atypical RHS

Neuralgia in Ramsay Hunt Syndrome (RHS) presents differently in typical and atypical cases, with varying degrees of intensity, localization, and persistence. Thus, RHS-related neuralgia ranges from localized to widespread, acute to chronic, depending on the presentation. In typical RHS, neuralgia primarily affect the facial nerve (CN VII) and sometimes the vestibulocochlear nerve (CN VIII). The pain is often sharp, burning, and localized to the ear, external auditory canal, and facial region, especially before or during the appearance of the vesicular rash. The facial nerve's involvement can lead to facial weakness and altered sensation, contributing to localized neuralgia.

The pain is generally short-term, but can linger in some cases, especially if postherpetic neuralgia (PHN) develops. Atypical RHS features more complex neuralgia due to widespread cranial nerve involvement. In cases such as zoster sine herpete (no rash), trigeminal neuralgia may occur, with intense pain in the face, jaw, and oral cavity. Multiple cranial nerves may be affected, leading to more diffuse and debilitating neuralgia in areas like the throat, tongue, and neck [8,10,20]. The pain often becomes chronic, particularly with postherpetic neuralgia, which can last for months or years, leading to persistent neuropathic pain. This makes diagnosis and management more challenging (Table 2).

Table 2: Correlation of Cranial Nerve Involvement, Pain Location, Characteristics, and Intensity in Typical and Atypical RHS.

Cranial Nerve Involved	Pain Location	Pain Characteristics	Pain Intensity	Typical RHS / References	Atypical RHS / References
CN VII (Facial Nerve)	Auricle External ear canal Mastoid	Sharp Burning Stabbing and Itching	Severe	Yu C, 2025 [7]	Liao YM, et al. 20213 [5]
CN V (Trigeminal Nerve)	Face Jaw	Electric shock-like Sharp Itching	Moderate to Severe	No	Jin HS, et al. 2012 [10] Liao YM, et al. 20213 [5]
CN IX (Glossopharyngeal)	Soft palate Pharynx	Burning Tingling	Moderate	No	Liao YM, et al. 2021 [35]
CN X (Vagus Nerve)	Throat Deep neck region	Deep Tight pressure	Moderate to Severe	No	Zhang J, et al. 2020 [14]

Current Treatment Progress in Intractable Neuralgia Typical and Atypical RHS

The typical RHS often presents with a triad of symptoms: auricular vesicles, facial paralysis, and otalgia. In some cases, atypical RHS may include polycranial neuropathies, throat pain, and deep facial pain, making treatment more challenging. Intractable neuralgia in RHS, particularly postherpetic neuralgia (PHN), requires a multimodal treatment approach combining antiviral therapy, pain management, and interventional procedures.

Medication is the primary treatment. Studies suggest that early administration of antivirals (e.g., Acyclovir, Valacyclovir) and corticosteroids can inhibit virus replication as much as possible, which can reduce neuroinflammation and nerve damage and significantly decrease pain and improves facial nerve function recovery [21-23].

Patients developing neuralgia should be treated with analgesic drugs. Gabapentinoids (Gabapentin, Pregabalin) reduce neuronal excitability and pain perception [21]. Tricyclic Antidepressants (Amitriptyline, Nortriptyline) modulate serotonin and norepinephrine pathways to alleviate neuropathic pain [24,25]. SNRI antidepressants (Duloxetine, Venlafaxine) are effective in cases of atypical RHS with widespread pain by modulating serotonin and norepinephrine pathways to reduce pain perception [26]. Opioids and tramadol are reserved for severe, intractable neuralgia when first-line treatments fail [22,27]. In addition, Lidocaine patches (5%) provide local anesthetic effect on affected nerve areas [21].

Interventional pain management may be considered in patients whose intractable neuralgia do not respond well to medication. Lidocaine or corticosteroid injections provide short-term relief [26]. Stellate ganglion block and trigeminal nerve

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block can interrupt nerve signaling for pain relief [28] Botulinum toxin injection reduce nerve hyperactivity and muscle spasm-related pain [29]. Neuromodulation techniques offer promising options for managing intractable pain in RHS when conventional treatments fail. Intrathecal drug delivery (Morphine, Baclofen) directly administers pain-relieving drugs into the cerebrospinal fluid [30]. Transcutaneous Electrical Nerve Stimulation (TENS) is applied to the skin to stimulate sensory nerves, helping to modulate pain perception and alleviate discomfort associated with the condition [26].

Pulsed radiofrequency (PRF) applying to the greater auricular nerve has provided significant pain relief in refractory otalgia following RHS [31]. Johnson and Covington reported a case whose post-herpetic neuralgia associated RHS was successfully treated by peripheral nerve stimulation (FNS) targeting the lesser occipital and greater auricular nerves, demonstrating the potential of PNS as an effective alternative to traditional pain management in cases of RHS with PHN [32]. Bowen and Zheng explore the use of motor cortex stimulation (MCS) for a 68-year-old patient with RHS-related persistent facial pain and paresthesia, and the patient reported significant pain and sensory relief following the procedure [33]. We also apply the PNS targeting facial nerve at the exit of stylomastoid foramen for typical RHS and PNS targeting facial nerve and supraorbital nerve for atypical RHS for their intractable neuralgia that showed excellent pain relief, especially for breakthrough pain (Figure 1 and 2).

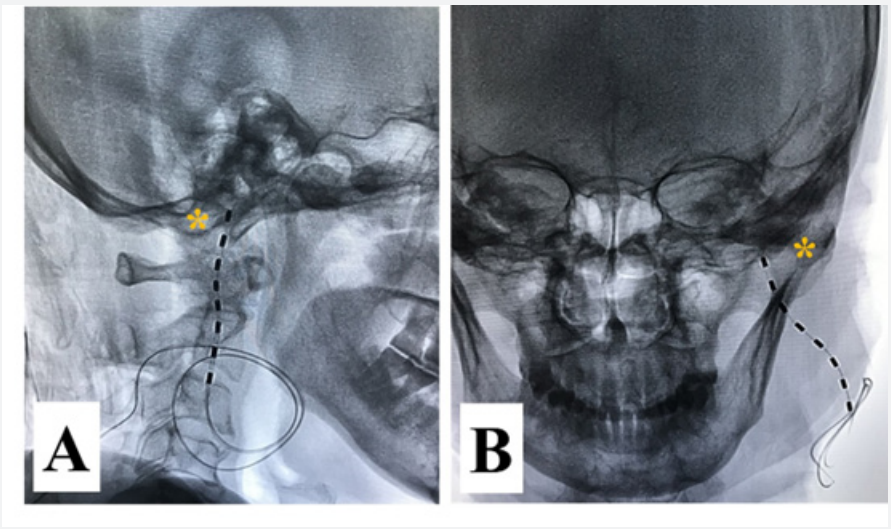

Figure 1: Transcutaneous facial nerve stimulation. A: The lead was again placed anterior to the mastoid process in the lateral view of the DSA. B: The anteroposterior view of the DSA confirmed that the lead was placed medial to the mastoid process. The yellow asterisk indicates the mastoid process.

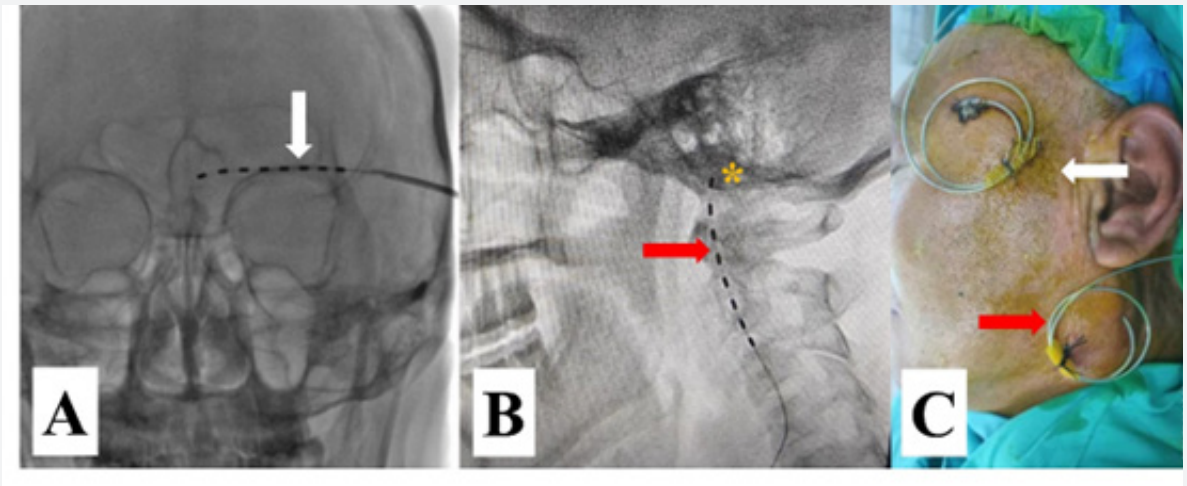

Figure 2: Transcutaneous facial nerve stimulation and supraorbital nerve stimulation. A and B: The lead tip was located anterior and medial to the mastoid process. C: The lead for transcutaneous supraorbital nerve stimulation (white arrow) and the lead for transcutaneous facial nerve stimulation (Red arrow) were attached separately to the skin. The yellow asterisk indicates the mastoid process.

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Surgical option and alternative & supportive therapies have also been proposed in severe studies. Tympanic nerve neurectomy resulted in significant pain relief and improvement in quality of life in a 45-year-old patient with chronic right-sided facial, ear and jaw pain that persisted for 9 years after RHS [34,35]. Liao et al implemented a multimodal approach, combining oral gabapentin, pulsed radiofrequency (PRF) application to the Gasserian ganglion for pain in the trigeminal nerve region, linear-polarized near-infrared light irradiation for pain in the facial nerve region, and 2% lidocaine spray for pain in the glossopharyngeal nerve region. This method improved pain management and quality of life in a 78-year-old patient with 3-month history of PHN secondary to RHS with polycranial nerve (V, VII, VIII, and IX) involvement [35].

limitations of this Review

The limitations of this review are largely attributed to the nature of the existing research, which predominantly relies on case reports. These reports, while informative, often involve small sample sizes and limited follow-up, making it difficult to draw broad conclusions or generalize findings to larger populations. Due to the rarity of Ramsay Hunt Syndrome (RHS), many studies are based on isolated cases, which restricts the overall quality and robustness of the data available. Another significant limitation is the complex etiology and pathophysiology of RHS. The mechanisms behind the syndrome involve the reactivation of the varicella-zoster virus in the facial nerve, yet the full spectrum of factors contributing to the disease's development, including immune responses and neural damage, remains poorly understood.

This complexity complicates both diagnosis and treatment strategies, especially in atypical cases that do not present with the classic symptoms of RHS. The review also highlights the need for multicenter studies, given the low prevalence of RHS. Large-scale, multicenter research would allow for a more comprehensive understanding of the disease and the effectiveness of various treatment modalities, such as neuromodulation. The current body of research is insufficient to establish definitive treatment protocols, and further studies are needed to confirm the long-term efficacy and safety of emerging therapeutic approaches for RHS.

Conclusion

Intractable neuralgia in both typical and atypical Ramsay-Hunt Syndrome poses a significant therapeutic challenge. Early diagnosis and aggressive management are essential to minimize the risk of chronic pain. A multidisciplinary approach, combining pharmacological, interventional, and psychological therapies, is often necessary to achieve optimal outcomes. Neuromodulation, especially FNS of facial nerve and trigeminal nerve, is a very promising treatment for RHS because it is less invasive and more effective in current cases reports. Further research is needed to better understand the underlying mechanisms of intractable neuralgia in RHS and to develop more effective treatment strategies.

Author Contribution(s)

Hao-dong Guo: Conceptualization; Validation; Visualization; Writing, review & editing.

Yu-xian Lin: Conceptualization; Supervision; Writing review & editing.

Wei-yi Gong: Conceptualization; Investigation; Methodology; Visualization; Writing original draft; Writing review & editing.

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