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Clinical Features, Diagnosis, and Management of Herpes Zoster Oticus: A Narrative Review

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Abstract: Herpes Zoster Oticus (HZO) is a neurological disorder caused by reactivation of the varicella-zoster virus in the geniculate ganglion, resulting in facial paralysis and, in severe cases, auditory or vestibular impairment. This narrative review summarizes current evidence regarding the clinical features, diagnostic approaches, and management of HZO. A literature search was conducted in PubMed for articles published between 2015 and 2025 using the keywords "herpes zoster oticus," "Ramsay Hunt syndrome," "facial palsy," and "varicella-zoster virus." Only English-language, full-text case reports discussing the clinical presentation of HZO were included. Of the 38 articles initially identified, 18 met the inclusion criteria after screening titles, abstracts, and full manuscripts. The synthesized findings highlight recent advancements in understanding VZV pathogenesis, improved diagnostic accuracy, and refinement of treatment strategies. Evidence consistently indicates that early initiation of antiviral therapy, combined with corticosteroids, is associated with better facial nerve recovery and reduced long-term complications. Furthermore, the variability of clinical manifestations—including atypical presentations such as zoster sine herpete—underscores the importance of maintaining a high index of suspicion and adopting a multidisciplinary approach to care. Early diagnosis and timely treatment remain key determinants of patient outcomes. Future research should aim to identify reliable prognostic biomarkers, develop rapid diagnostic tools for atypical cases, and conduct randomized controlled trials to optimize therapeutic regimens, particularly regarding the role of corticosteroids and emerging antiviral agents.

Keywords: herpes zoster oticus, ramsay hunt syndrome, facial palsy, varicella-zoster virus, narrative review

1. Introduction

Herpes Zoster Oticus (HZO), referred to as Ramsay Hunt syndrome, is a neurological disorder resulting from the reactivation of the Varicella-Zoster virus (VZV) within the geniculate ganglion (Goswami & Gaurkar, 2023; Wu & Song, 2025). This reactivation primarily affects the facial and cochlear-vestibular nerves causing clinical presentations ranging from acute facial paralysis to long-term hearing

and balance dysfunction (Kim et al., 2016; Rim et al., 2023). Due to its complex nature, HZO continues to be a concern in both otology and neurology.

Ramsay Hunt syndrome is estimated to occur in almost 5 out of 100,000 people every year, which is fewer than Bell's Palsy which occurs in 15 to 30 out of 100,000 people every year (Rim et al., 2023). Ramsay Hunt syndrome occurs in people who are immunocompromised or immunocompetent and is included in about 7% of cases of acute facial paralysis. Zoster sine herpete, which is a variant of Ramsay Hunt syndrome, accounts for almost a third of these cases. Although it can occur at any age, it is most commonly seen in those who are 70 or 80 and are particularly vulnerable if they are immunocompromised, malnourished, have a chronic infection, or are under psychological stress, chemotherapy, or any other factors that can cause VZV reactivation or psychological stress. (Crouch et al., 2023)

There has been significant research on this disorder; however, there are still challenges with diagnosis and treatment, especially in patients with atypical presentations or cases without a rash, which are referred to as zoster sine herpete (Fader et al., 2022; K. M. Kim et al., 2021). The purpose of this narrative review is to consolidate the current evidence on HZO, including its definition, etiology, epidemiology, risk factors, pathogenesis, clinical features, diagnostic approach, differential diagnosis, management, complications, and prognosis (Malhotra et al., 2022; Rim et al., 2023). This review will consolidate the literature on HZO to better inform clinicians and researchers, to address the gaps in knowledge, and to promote a multidisciplinary approach to improving patient outcomes and reducing the overall burden of disease.

2. Materials and Methods

This narrative review began with a thorough sweep of the literature available on the PubMed database, which included the terms “herpes zoster oticus,” “Ramsay Hunt syndrome,” “varicella zoster virus” for literature review. The literature review was based on freely available, complete, English case reports. Literature published between the years 2015-2025 was the primary target. 38 articles were identified, and thorough exclusion criteria were applied. The criteria focused on the vision of primary literature pertaining to the clinical presentation of HZO, the articles were insufficiently clinical, and case reports were not the publication type. This screening process resulted in 18 case reports which were fully justifiable and relevant based on the quality inclusion criteria. Thematic analysis was performed by collecting literature based on inductive reasoning on the clinical patterns, with 18 case reports forming the corpus of literature. This review used a descriptive synthesis of findings from selected case reports. Themes were derived inductively to summarize clinical manifestations, diagnostic approaches, and treatment outcomes.

SANRA guidelines were applied, which primarily focused on the narrative review and was published along with the justification for the methodological approach taken. The review followed all necessary requisite guidelines pertaining to the transparency of the research, the synthesis of the collected information, and the chronological presentation of the manuscript.

3. Results and Discussion

Table 1. Extracted Data from Case Reports

Author (Year)	Gender & Age (Year)	Comorbidity	History of Varicella	Clinical Features	Treatment	Outcome
Kim, C. H., Choi, H., & Shin, J. E. (2016)	Male, 14 & Female, 13	Not specified	Not specified	Sensorineural hearing loss (high-frequency)	Antiviral + steroids	Variable hearing recovery; incomplete in severe cases
Kim, K. M., Yu, K., Jeon, E. J., & Lee, H. J. (2021)	Male, 71	Hypertension	Not specified	Delayed facial palsy, CN IX, X, XI involvement	Acyclovir IV, steroids	Gradual improvement of neurological symptoms
Al-Ani, R. M. (2022)	Female, 35	None	Not specified	Cranial polyneuropathy (CN V, VII, VIII, IX, X), delayed facial palsy	Acyclovir, prednisolone	Significant recovery at 3-month follow-up
Qavi, A., & Tiwari, A., et al. (2020)	Male, 24	None	Not specified	Unilateral cranial polyneuropathy (CN V, VII, VIII, IX, X, XI, XII)	Acyclovir, steroids	Partial recovery with mild residual deficits
Dai, S., & Huang, X., et al. (2020)	Male, 33	Immunocompetent	Not specified	Bilateral asymmetrical facial palsy, vesicles	Acyclovir IV, methylprednisolone	Good recovery of facial and auditory function
Dhatrak, V. M., & Mohod, S., et al. (2024)	Male, 60	Submandibular hemangioma (incidental)	Not specified	Facial palsy, vesicles, hypoacusis, vertigo	Acyclovir, steroids, analgesics	Symptom improvement within 2 weeks
Cavalcante, M. S. M., & Rodríguez, K. L., et al. (2020)	Female, 7	Leishmaniasis (on antimonials)	Not specified	Facial palsy, vesicles on ear and mouth	Stop antimonials, acyclovir, steroids	Complete recovery within 3 months
Sheik-Ali, S., & Jiang, Y., et al. (2024)	Male, 70	Multiple Myeloma (immunocompromised)	Not specified	Bilateral sequential facial palsy, vesicles	Acyclovir IV, steroids	Partial recovery with residual facial weakness

Pitton Rissardo, J., & Fornari Caprara, A. L. (2018)	Female, 80	DM, Hypertension	Yes (childhood)	HZ Oticus, HZ Ophthalmicus, cutaneous dissemination	Acyclovir IV	Deceased (systemic complications)
Cunniffe, H. A., & Cunniffe, N. G. (2019)	Male, 75	DM, IHD	Not specified	Meningitis, otalgia, no initial vesicles (atypical)	Acyclovir IV, antibiotics, steroids	Complete recovery after correct diagnosis
Kakeeto, J., Atukunda, R. A., Asio, P., & Pitua, I. (2025)	Female, 68	Not specified	Not specified	Facial palsy, hearing loss, vertigo	Acyclovir, prednisolone	Significant neurological and audiological improvement
Gillette, B. T., & Heilbronn, C. M. (2023)	Male, 40	None	Not specified	Vocal cord paralysis (CN X), otalgia, facial palsy	Valacyclovir, prednisone, vocal cord injection	Voice improvement, near-complete facial recovery
Yu, C., Lee, H. Y., & Chen, Y. C. (2025)	Male, 30	None	Not specified	Facial palsy, vesicles, hearing loss	Early antiviral + steroids	Complete recovery of facial and auditory function
Gaudencio, M., & Bertão, M. I., et al. (2021)	Female, 44	Ulcerative Colitis (on Infliximab)	Not specified	Facial palsy, vesicles, tinnitus	Stop Infliximab, acyclovir, steroids	Skin lesions resolved, partial facial improvement
Kang, D.H.; Kwak, B.O.; Park, A.Y.; Kim, H.W. (2021)	Children (Cohort)	Various	Some had VZV	Neurological complications (incl. HZO) more common in children >10 yrs	Antiviral	Generally good recovery; complications rare
Khan, Y. M. T., & Fatema, N. (2019)	Female, 25	None	Not specified	Facial palsy, vesicles, severe pain	Acyclovir, steroids, analgesics	Complete recovery without sequelae
Montague, S. J., & Morton, A. R. (2017)	Male, 45	None	Not specified	Facial palsy, vertigo, vesicles	Valacyclovir, prednisone	Full facial function recovery at 6 weeks

Fader, F., Gendeh, H. S., & Goh, B. S. (2022)	Femal e, 33	None	Not specified	Facial palsy, otalgia, no vesicles (zoster sine herpete)	Acyclovir, steroids	Near-complete recovery from facial palsy

Based on a synthesis of data from 18 case reports, Herpes Zoster Oticus (HZO), or Ramsay Hunt Syndrome, presents across a wide demographic spectrum, affecting patients from early childhood to advanced age (Cavalcante et al., 2020; Pitton Rissardo & Fornari Caprara, 2019). A notable finding is the occurrence of severe or atypical manifestations in immunocompromised individuals, such as those with multiple myeloma or on immunosuppressive biologics like infliximab, suggesting that host immune status is a critical determinant of disease severity (Gaudêncio et al., 2021; Sheik-Ali et al., 2022).

The clinical presentation of HZO is highly variable. While the classic triad of otalgia, vesicular rash, and peripheral facial nerve palsy is well-documented, the synthesized data reveal a significant number of atypical cases. These include presentations without a vesicular eruption, known as zoster sine herpete (Fader et al., 2022), delayed onset of facial palsy (Al-Ani, 2022; K. M. Kim et al., 2021), and extensive unilateral or even bilateral cranial polyneuropathy affecting nerves V, IX, X, XI, and XII (Dai et al., 2020; Qavi et al., 2020). Less common but serious complications such as meningitis and vocal cord paralysis were also reported, underscoring the potential for VZV to cause significant neurological morbidity (Cunniffe & Cunniffe, 2019; Gillette & Heilbronn, 2023).

Management uniformly involves a combination of antiviral agents and corticosteroids across the cases. A pivotal observation from this synthesis is that early initiation of this therapeutic regimen appears to be strongly correlated with superior outcomes, particularly the complete recovery of facial nerve function (Montague & Morton, 2017; Yu et al., 2025). In contrast, delayed treatment, extensive neurological involvement at presentation, or an immunocompromised state were often associated with residual deficits, such as incomplete facial palsy or permanent hearing loss (C.-H. Kim et al., 2016; Sheik-Ali et al., 2022).

The clinical presentation of Herpes Zoster Oticus (HZO) shows wide variability because treatment timing and patient immune system function determine both diagnosis and treatment results. Medical professionals need to monitor patients carefully for unusual or hard-to-detect symptoms because this enables them to make timely and correct diagnoses. The initiation of treatment at an early stage helps patients avoid permanent neurological damage while producing superior results in their overall treatment.

3.1. Definition

The medical condition known as Herpes Zoster Oticus (HZO) appears under the name Ramsay Hunt Syndrome (RHS) when the Varicella-Zoster virus which causes chickenpox reactivates after years of dormancy. The geniculate ganglion near the ear contains the dormant Varicella-Zoster virus which becomes active to produce HZO (Goswami & Gaurkar, 2023; Wu & Song, 2025). The three main symptoms of HZO include severe ear pain (otalgia) and vesicular eruptions that appear on or near the ear and in the mouth and paralysis of one facial side (ipsilateral facial nerve paralysis) (Altıner, 2023; Goswami & Gaurkar, 2023). The complex neurological condition known as cranial neuropathy requires medical specialists to work together for proper diagnosis and complete treatment of HZO. The treatment strategy focuses on managing various symptoms to stop the development of potential lasting complications (Qavi et al., 2020; Rim et al., 2023).

3.2. Etiology

The Varicella-Zoster virus (VZV) causes Herpes Zoster Oticus (HZO) through its reactivation from dormant states in the geniculate ganglion following chickenpox infections (Goswami & Gaurkar, 2023; Wu & Song, 2025). The Varicella-Zoster virus (VZV) will hide itself in sensory ganglia after primary infection, including the geniculate ganglion, which controls the facial nerve (Guan et al., 2024; Wu & Song, 2025). The virus will start to reinvade and replicate when the host's immune cells weaken, therefore causing nerve inflammation (Sinha et al., 2023). The clinical manifestation of HZO includes three main symptoms, which are ear pain (otalgia), vesicular eruptions in the ear or oropharynx, and paralysis of the facial nerve on the affected side (Altıner, 2023; Goswami & Gaurkar, 2023). The medical condition known as Ramsay Hunt Syndrome Type 2 exists as a separate entity from Type 1, which presents with facial paralysis without known causes (Altıner, 2023; Wu & Song, 2025). The understanding of HZO origins enables healthcare professionals to create optimal prevention strategies and treatment plans.

3.3. Epidemiology

The worldwide occurrence of Herpes Zoster Oticus (HZO) remains difficult to determine because HZO cases frequently appear in reports that combine data from Herpes Zoster and Ramsay Hunt Syndrome (Bardach et al., 2021; Sinha et al., 2023). The age distribution of HZO follows the pattern of Herpes Zoster in general. People who are older (above 50 years old) are more likely to get herpes zoster oticus because of the immunosenescence effect (Bardach et al., 2021; Sinha et al., 2023). The incidence of HZO remains rare in children even though it can affect people of all ages (Kang et al., 2021; Swain et al., 2020).

There is no correlation between the incidence of HZO and gender patterns. In fact, the risk of VZV reactivation leading to HZO is more common in people with underlying health conditions, such as patients who have immunosuppression, diabetes mellitus, and autoimmune disease (Gaudêncio et al., 2021; Sinha et al., 2023). Until now, HZO has still had a major impact on long-term public health (Malhotra et al., 2022; Rim et al., 2023). It remains significant because the patient's symptoms can develop into long-term complications such as postherpetic neuralgia and facial weakness. Therefore, proper prevention and effective treatment strategies were required for this condition (Malhotra et al., 2022; Rim et al., 2023). Research needs to investigate how genetic and environmental elements affect geographic HZO incidence patterns (Bardach et al., 2021).

3.4. Risk Factors

Reactivation of the dormant varicella-zoster virus (VZV) starts when the immune system, especially the cell-mediated immune system, weakens (Sinha et al., 2023). This causes older people with a decaying immune system due to the immunosenescence phenomenon to have a higher chance of developing HZO (Bardach et al., 2021; Sinha et al., 2023). Besides that, the risk of getting HZO is also higher in those who have a comorbid condition that weakens the immune system, like HIV/AIDS patients, cancer patients, and those who are taking immunosuppressive drugs such as corticosteroids or post-transplantation medication (Gaudêncio et al., 2021; Sinha et al., 2023).

It happens because the immune system can't keep the latent virus dormant. The reactivated VZV will begin to replicate and cause clinical manifestations through inflammation and nerve damage (Guan et al., 2024; Wu & Song, 2025).

3.5. Pathogenesis

The pathogenesis of Herpes Zoster Oticus (HZO) is characterized by the reactivation of the varicella-zoster virus (VZV) within the geniculate ganglion. A synthesis of recent studies confirms this ganglion as the primary site of viral latency and reactivation. (Guan et al., 2024; Wu & Song, 2025).

However, an analysis of the triggering factors reveals a broader spectrum of risks. It is widely known that the virus is able to become active again because of natural aging of the immune system. However, recent studies show it is not solely dependent on immunosenescence, but is also significantly promoted by iatrogenic immunosuppression and major psychological stress. This convergence of evidence indicates that any compromise in VZV-specific T-cell immunity can create a permissive environment for viral reactivation. (Gaudêncio et al., 2021; Sinha et al., 2023).

Furthermore, comparing the anatomical progression across studies clarifies the link between viral spread and clinical symptoms. The virus starts to multiply inside the ganglion before it moves through the intermediate nerve and cranial nerve VII (the facial nerve) pathways (Guan et al., 2024; Wu & Song, 2025). This trajectory may explain why the infection manifests in specific regions like the outer ear canal, earlobe, and tympanic membrane, which are innervated by these nerves. The symptoms of HZO emerge because of nerve damage and inflammation which result from the infection (Goswami & Gaurkar, 2023; Rim et al., 2023). These findings suggest that the hallmark symptoms of HZO such as facial palsy, neuropathic pain, and vesicular eruptions are a direct result of intense neuritis and ganglioneuritis. Critically, this synthesized pathogenesis underscores why early antiviral therapy is essential, as it directly targets viral replication to limit neurological damage and thereby improves clinical outcomes.

3.6. Pathophysiology

The widely known pathophysiology of Herpes Zoster Oticus (HZO) originates from the reactivation of the Varicella-Zoster Virus (VZV) within the geniculate ganglion. While the majority of studies consistently identify this site as the epicenter of reactivation, a deeper analysis of the subsequent inflammatory cascade provides a clearer link to the disease's clinical signature. (Guan et al., 2024; Wu & Song, 2025). The viral replication triggers a severe inflammatory response that affects the facial nerve and adjacent cranial nerves, leading to significant neurological impairment (Goswami & Gaurkar, 2023; Rim et al., 2023). This pathophysiological process directly explains the classic triad of symptoms, which are painful vesicular eruptions, facial nerve inflammation, and unilateral paralysis as extensively documented in clinical reports (Altıner, 2023; Goswami & Gaurkar, 2023).

Crucially, the link between VZV reactivation and these symptoms is not merely theoretical but is robustly supported by molecular evidence. Studies have proven this process by identifying VZV DNA through PCR tests in the fluid of vesicles and ear discharge from patients with HZO (Lee et al., 2022). Therefore, these findings do not just describe the process, they empirically validate the model that VZV reactivation in the geniculate ganglion is the direct cause of the nerve damage and inflammation responsible for the full spectrum of HZO symptoms. This synthesis of clinical observation and molecular data strengthens the pathological model and underscores the importance of targeting viral replication in therapeutic strategies. (Lee et al., 2022).

3.7. Clinical Manifestations

Individual presentations of Herpes Zoster Oticus can be particularly complex. The clinical picture can be considered most problematic when assessing the preemptive characteristics of the rest of the clinical picture (Altıner, 2023; Goswami & Gaurkar, 2023). The genesis of these complications may be indicative of the diagnostic picture as a result of considering the symptoms of the disease in isolation (Goswami & Gaurkar, 2023). In a sense, these first vague symptoms form the bulk outline of the complication and thus may be descriptive of high intensity head pain and other features, which in turn can be conceivably errant. What is particularly complex though is the whole perception and the symptoms tied autonomously to facial paralysis, considering how people clinically accept such a multi-teared skull structure. Radiologists and neurosurgeons, in particular,

depend on intricate relationships they assume characterize complex patterns. (Fader et al., 2022; K. M. Kim et al., 2021)

Part of the whole thesis may only surface when appreciating how systemic the other symptoms are. These are likely due to the systemic nature of the connective tissue and result from the virus showcasing how broad in scope its infection may be (C.-H. Kim et al., 2016; Lee et al., 2022; Rim et al., 2023). Its reach reaches plural forms of the heads where loss and interstitial tissue may form, ruins a narrow corridor from the skull's foundation, to the vast space above where the head is having lost connective tissue. The excessive strain from the muscle may lose its grip and result in a deflection of the bony structure, leaving the head suspended between loss and balance (C.-H. Kim et al., 2016; Lee et al., 2022; Rim et al., 2023).

Even though infrequent, the risk of serious neurologic complications, as described in the case studies, is on the most critical end of the clinical spectrum. The underlying pathological process of some disorders is capable of invading the central nervous system, presenting as meningitis, encephalitis, or vasculitis of the brain (Cunniffe & Cunniffe, 2019; Wu & Song, 2025). It is necessary to understand the clinical presentation in its entirety, which includes isolated anodyne pain, and the very delayed, or asynchronous, palsies of the cranial nerves. (Al-Ani, 2022; K. M. Kim et al., 2021; Qavi et al., 2020).

3.8. Diagnosing Herpes Zoster Oticus

Because its symptoms usually resemble other disease processes and zoster sine herpete patients do not exhibit the characteristic rash, the diagnosis of Herpes Zoster Oticus (HZO) can be problematic (Fader et al., 2022; K. M. Kim et al., 2021). The resolution of a case typically starts with lab testing for VZV after a clinical assessment featuring the Ramsay Hunt triad. The triad is clinically assessed and then VZV is lab-tested (Goswami & Gaurkar, 2023; Lee et al., 2022). Blood tests, or serology, are also conducted, but they cannot confirm if the infection is being experienced acutely; they are used to aid the physician in evaluating the patient's immune response (Sinha et al., 2023).

Such limitations in diagnostics in HZO are a call for other, multi-faceted methods to improve diagnostics and clinical analysis of HZO. Nerve function tests such as electromyography (EMG) and electroneuronography (ENoG) have been shown to provide extremely valuable prognostic information with respect to predicting the chances of nerve recovery (Malhotra et al., 2022). In a similar fashion, contrast-enhanced MRI of the temporal bone can visually confirm the inflammation of the tissues and soft tissue constituents, thus augmenting the clinical diagnosis of HZO (Lee et al., 2022). It appears that a blend of functional and imaging approaches results in a clinical diagnosis of HZO that is beyond the constellation of presenting symptoms.

This may explain why the timely and precise identification of HZO allows for the initiation of antiviral treatment. It has been shown that such treatment is associated with better clinical outcomes and fewer neurologic complications (Rim et al., 2023; Yu et al., 2025). Therefore, the current literature suggests the need for developing rapid point-of-care tests, prognostic biomarkers, and standardized diagnostic protocols to improve the timing of the initiation of treatment and the accuracy of the diagnosis (Rim et al., 2023).

3.9. Differential Diagnosis

The identification of Herpes Zoster Oticus (HZO) must be approached with caution because its initial manifestations may be confused with many other causative factors of temporary facial paralysis (Altıner, 2023; Goswami & Gaurkar, 2023). Of other possible conditions, early clinical presentation is often inadequate to distinguish HZO from Bell's palsy, which is described by sudden

onset unilateral facial weakness of unknown aetiology. Here, though, a key distinguishing feature is the presence of a vesicular rash on the ear, or in the canal of the affected ear. The vesicular rash that appears on the affected ear is a defining clinical feature that directly distinguishes HZO from Bell's palsy (Fader et al., 2022; K. M. Kim et al., 2021).

The diagnostic challenge is intensified in the case that patients develop Zoster Sine Herpete, or HZO without a rash (Fader et al., 2022; K. M. Kim et al., 2021). The absence of a rash certainly eliminates the most important clinical clue, thus rendering the symptoms indistinguishable from Bell's palsy. In these atypical presentations, more extensive clinical reasoning is required to minimize the risk of misdiagnosis.

Considering each cause of facial paralysis demonstrates the varied triggers for the condition. The range of possible conditions like otitis media, mastoiditis, Lyme disease, parotid and cerebellopontine angle tumors, head trauma, sarcoidosis, and Guillain-Barr syndrome, indicates the need to broaden the differential diagnoses to include more than HZO (Goswami & Gaurkar, 2023). The absence of these conditions from the differential diagnoses indicates an oversight.

Within such situations, the need for structured and systematic manners of approach seems obvious. This begins with the patient's history, particularly any relevant varicella and herpes zoster infections, and ends with the comprehensive clinical assessment and screening for possible neurologic examination constituents. The patient history and clinical presentation should outline the framework of the diagnosis, and the assessment should include neurological examination and otoscopy to evaluate for the presence of vesicles and inflammation and any anomalies within the external auditory canal (Goswami & Gaurkar, 2023; Rim et al., 2023). This type of analysis suggests that even slight clinical signs may aid in early differentiation.

Healthcare providers must conduct more tests if the first evaluation does not provide a clear diagnosis. The most definitive test for diagnosing HZO is the PCR test which detects Varicella-Zoster Virus (VZV) DNA in vesicular fluid and varying amounts of blood and other fluids (Lee et al., 2022).

3.10. Complications

According to the post herpetic neuralgia setbacks experienced by patients (Malhotra et al., 2022; Rim et al., 2023). The persistent neural inflammation along with viral sensitization may extend well beyond the rash subsiding. The subsequent neuralgia then experienced by other patients can often mask the underlying HZO condition. Hearing loss and extreme discomfort from uncorrectable dizziness have been postulated to be the result of the same uncorrectable condition (Malhotra et al., 2022; Rim et al., 2023).

This is only a hypothesis and needs further research, though it explains the sharp improvement among patients with anti-viral therapy attributing neural-viral damage and control along with inflammation suppression. (C.-H. Kim et al., 2016; Lee et al., 2022; Rim et al., 2023).

3.11. Treatments

The initial treatment of Herpes Zoster Oticus (HZO) requires prompt intervention to protect nerve tissue and control pain symptoms and stop potential long-term damage (Rim et al., 2023; Yu et al., 2025). There are several pharmacological treatments that are used in the management of herpes zoster oticus. The first one is antiviral medications, which must be given immediately within 72 hours after symptoms appear to stop Varicella-Zoster virus (VZV) growth (Rim et al., 2023; Yu et al., 2025). The selection of antiviral medication depends on three main factors, which include treatment effectiveness and drug availability, and cost efficiency. The most effective treatment for herpes

zoster oticus is valacyclovir 1000 mg three times daily (Goswami & Gaurkar, 2023). But, for a cost-effective choice, acyclovir 500 mg taken five times a day was the best choice (Kang et al., 2021). Famciclovir 500 mg three times a day provides better results than acyclovir with a price lower than valacyclovir (Goswami & Gaurkar, 2023; Kang et al., 2021). The antiviral treatment needs to be given for 7 to 10 days to achieve the best possible treatment outcomes (Goswami & Gaurkar, 2023). The use of prednisone corticosteroids alongside antiviral medications helps decrease nerve swelling but their standalone effectiveness remains uncertain according to medical studies (Rim et al., 2023; Yu et al., 2025).

The treatment of herpes zoster oticus in cludes supportive care which includes pain management through analgesics and tricyclic antidepressants and gabapentin for postherpetic neuralgia and eye protection against keratitis from eyelid paralysis (Rim et al., 2023; Yu et al., 2025). Patients with HZO receive two types of non-drug treatments which include facial physical therapy to restore facial nerve function and hearing, and balance rehabilitation therapy to treat audiovestibular symptoms (Malhotra et al., 2022; Rim et al., 2023). The complete management of HZO requires medical teams to perform facial nerve decompression procedures and surgical interventions for severe hearing issues (Rim et al., 2023). The timing of starting antiviral treatment along with other therapies directly affects the speed of HZO patient functional recovery (Rim et al., 2023; Yu et al., 2025).

3.12. Prognosis

The recovery period for patients suffering from Herpes Zoster Oticus is determined by the patient's age, the degree of facial paralysis, and the timing of the initiation of antiviral therapy. From a clinical standpoint, much better if antiviral medication is started within the first three days of having the symptoms (Malhotra et al., 2022; Rim et al., 2023; Yu et al., 2025). Without treatment and if the disease is allowed to progress further, there can be very serious complications such as meningitis and encephalitis, or even vascular complications that can lead to a stroke (Cunniffe & Cunniffe, 2019; Wu & Song, 2025). Treatment tailored to the individual patient is very important, rehabilitation programs are developed and implemented based on the individual's symptoms, clinical pictures and medical requirements (Malhotra et al., 2022; Rim et al., 2023).

4. Conclusions

The purpose of this narrative review is to document the latest developments surrounding Herpes Zoster Oticus (HZO) its treatment advancements, the means of overcoming its elusive diagnosis, and the variety of its clinical manifestations. Notable findings attest to the varying degrees of HZO presentation. While the classical Ramsay Hunt triad is pertinently diagnostic, there are many other cases that present with atypical features such as zoster sine herpete or herpetic lesions that engage multiple cranial nerves accompanied with peculiar lagomorphic or pan-symptomatic manifestations. A key point is that early aggressiveness is paramount; combination antiviral and corticosteroid treatment starting as soon as possible, ideally within the first 72 hours, is predictive of enhanced facial nerve recovery and reduced complications of delayed presentation.

This review adds to the significance of recent case reports and argues for a high index of clinical suspicion for atypical cases, and its accompanying evidence-based, multidisciplinary management. Several priorities for research arise: identifying and validating reliable, prognostic treatment response biomarkers, creating zoster sine herpete rapid diagnostic tests, and well-structured, randomized controlled trials that focus on optimizing treatment regimens inclusive of corticosteroids and emerging antiviral agents.

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