Review Article

Pathophysiology and Inflammatory Pathway in Vestibular Neuritis

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Article info:
Received: 23 Nov 2025
Accepted: 14 Feb 2026

Citation: Fayaz MA, Alsaad SSA, Rasouli A. Pathophysiology and Inflammatory Pathway in Vestibular Neuritis. Journal of Modern Rehabilitation. 2026;20(4):?-?

Running title: Vestibular neuritis and inflammation

Abstract

Introduction: Vestibular neuritis (VN) causes acute vertigo from sudden unilateral vestibular dysfunction, mainly in adults aged 30–60. Previous reviews have focused on clinical and therapeutic aspects, but the inflammatory and immune mechanisms are less well understood. This review summarizes recent evidence on viral, immune, and vascular pathways in VN.

Method: A narrative review was conducted using PubMed, Scopus, and Web of Science databases. Forty articles published between 2001 and 2025 were included, focusing on pathophysiology, immune pathways, and therapeutic approaches implications.

Results: VN is primarily caused by HSV-1 reactivation, leading to vestibular nerve inflammation. Other viruses, such as SARS-CoV-2 and Epstein–Barr, are also involved. Immune dysregulation, characterized by alterations in leukocytes and cytokines, drives neuroinflammation. Vascular issues, especially blood-labyrinth barrier disruption, worsen swelling. Corticosteroids reduce inflammation; vestibular rehab aids recovery. Combining treatments improves early outcomes.

Conclusion: VN is a complex disorder caused by viral reactivation, immune inflammation, and vascular issues. Recovery primarily depends on central compensation, rather than peripheral nerve regeneration. Combining anti-inflammatory treatment with early rehab yields the best results.

Future research should investigate the molecular connections between viral infection, immune response, and vestibular damage to develop targeted therapies.
Keywords: Vestibular neuritis; Vertigo; Inflammation; Pathophysiology

Introduction

Vestibular neuritis (VN) is a common cause of severe and prolonged vertigo, resulting from a sudden loss of function in one of the vestibular systems. It ranks as the third most prevalent peripheral vestibular disorder, with an annual incidence between 3.5 and 15.5 cases per 100,000 people (1,2). This condition most commonly affects adults aged 30 to 60, with no significant differences noted between genders (3,4). The acute phase lasts for several days, characterized by vertigo and imbalance, followed by a gradual recovery over weeks. Recurrence is rare, occurring in only 1.9–10.7% of cases (5). Long-term outcomes are more heavily influenced by central compensation and psychological factors than by the extent of peripheral damage (6).

Patients with vestibular neuritis commonly present with acute, severe, and persistent vertigo that lasts longer than 24 hours. This condition is often accompanied by nausea, vomiting, postural imbalance, and spontaneous horizontal–torsional nystagmus, which beats away from the affected ear. Hearing remains intact, and no other neurological deficits are observed. Vestibular testing shows an abnormal head impulse response and caloric weakness on the affected side. Subtypes of vestibular neuritis include superior, inferior, and total. Inferior involvement generally presents milder symptoms and may mimic central disorders (4,7–9).

The diagnosis of vestibular neuritis is primarily clinical and must be distinguished from other vestibular disorders. Unlike Meniere's disease, it does not involve hearing loss or tinnitus, and symptoms remain stable. Vestibular migraine differs from vestibular neuritis, as it includes recurrent episodes of vertigo often linked to a history of migraines, with vestibular tests typically showing expected results. In contrast, vestibular neuritis presents as a single, prolonged episode of vertigo accompanied by unilateral vestibular hypofunction, as confirmed by an abnormal head impulse test or a caloric deficit. Central vestibular disorders can mimic peripheral vertigo but exhibit additional neurological signs, such as diplopia or limb ataxia, and may also display abnormal neuroimaging findings. Inferior vestibular neuritis, a rare variant, can present with down-beating nystagmus and milder vertigo, necessitating careful testing and imaging for accurate diagnosis (10).

Numerous reviews have thoroughly discussed the clinical features, diagnosis, and treatment strategies for vestibular neuritis (2, 3), A significant gap still exists in understanding the molecular and cellular inflammatory pathways underlying it. Recent progress has started to outline the complex roles of viral reactivation, immune dysregulation, and vascular integrity in this context pathogenesis (5, 11). This review aims to deliver an updated, focused overview of the pathophysiology and inflammatory pathways in VN. It integrates evidence from viral triggers, innate and adaptive immune responses, and blood-labyrinth barrier disruption to provide a comprehensive understanding of the mechanisms involved.

Method

This study was conducted as a narrative review intended to offer an integrated overview of current understanding of the pathophysiology, immune mechanisms, and clinical features of vestibular neuritis. Relevant literature was gathered through extensive searches of PubMed, Scopus, and Web of Science. The search used key terms and Boolean operators to ensure comprehensive coverage: ("vestibular neuritis" OR "vestibular neuronitis") AND ("viral infection" OR "immune-mediated"

OR "inflammation" OR "pathophysiology" OR "Herpes simplex virus type 1" OR "HSV-1" OR "blood-labyrinth barrier"). Inclusion criteria included original research articles and reviews published in English from 2001 to 2025, focusing on clinical features, pathophysiology, immune/inflammatory pathways, and treatments for vestibular neuritis. The exclusion criteria comprised studies that did not focus mainly on VN's pathophysiology and articles without accessible full texts. This process yielded 40 relevant articles for detailed review and synthesis. Since this review is narrative in nature, a formal risk-of-bias assessment or quality appraisal tool was not used.

Etiology and Pathogenesis

Viral hypothesis

The viral hypothesis suggests that VN is mainly caused by a viral infection or reactivation, particularly of HSV-1. This theory is the most widely accepted explanation for the development of VN, although other viruses and mechanisms have also been considered. HSV-1 is significantly implicated in the cause of VN due to its capacity to establish lifelong latency in sensory neurons, including those found in the vestibular ganglion (11).

Reactivated latent HSV-1 can trigger inflammatory responses that damage the vestibular nerve, resulting in symptoms of VN, including vertigo, spontaneous nystagmus, imbalance, and nausea. Studies have detected HSV-1 DNA in vestibular ganglia and labyrinth tissues, supporting this theory (12,13).

The text provides direct molecular evidence of the presence of a virus in the affected neural structures. Additionally, the histopathological characteristics of nerve damage observed in viral neuropathy closely resemble those seen in other viral neuropathies, such as herpes zoster oticus. This resemblance reinforces the idea that a viral-mediated mechanism is involved (14). Experimental studies using animal models have further supported this association, as inoculating HSV-1 into the middle ear or pinna has been shown to replicate both the vestibular dysfunction and neuronal degeneration that are characteristic of human VN (11,15).

Serological and epidemiological data indicate that a substantial proportion of patients with VN exhibit evidence of recent or past viral infections, particularly following upper respiratory tract infections. These observations suggest a link between viral reactivation, particularly of HSV-1, and the onset of VN (16). Other viral agents, in addition to HSV-1, have been associated with the onset of vestibular neuritis. Recent observations during the COVID-19 pandemic have highlighted SARS-CoV-2 as a possible trigger for vestibular dysfunction. Patients with confirmed COVID-19 infections may experience acute vertigo, spontaneous nystagmus, or symptoms resembling vestibular neuritis, often accompanied by hearing loss (17–19).

Although comprehensive vestibular testing has not been consistently conducted in the reports, the timing suggests that SARS-CoV-2 may cause vestibular nerve injury either through direct viral invasion or by immune-mediated inflammation. There have also been rare cases of VN documented following COVID-19 vaccination, which include both mRNA and adenoviral vector vaccines. However, these occurrences are isolated and do not have established causal relationships (20,21).

Together, these findings suggest that while HSV-1 is the most established viral agent responsible for vestibular neuritis, other viruses, including SARS-CoV-2, Epstein-Barr virus, and possibly influenza, may be associated with either direct infection or immune-mediated mechanisms (22).

Immune-mediated mechanisms

Recent evidence suggests that immune-mediated processes play a crucial role in the development of VN, beyond the direct reactivation of the virus. VN may not be caused solely by the cytopathic effects of the virus but rather by the host's immune response triggered by viral infection or reactivation, particularly of HSV-1. In some patients, damage to the vestibular nerve appears to result from an inappropriate immune reaction following HSV-1 infection, rather than from direct viral invasion. The presence of HSV-1 DNA in the vestibular ganglia, along with the association of genetic factors that regulate HSV-1 replication and susceptibility to the disease, strongly supports this hypothesis (2).

Recent studies using Mendelian randomization have offered further insights into the immunological mechanisms involved. Specific inflammatory mediators, such as eotaxin and monocyte chemotactic protein-2, have been linked to a decreased risk of vascular neovascularization. Conversely, higher levels of the T-cell surface glycoprotein CD5 are associated with an increased risk of VN. This suggests that these proteins may act as potential biomarkers or therapeutic targets (23).

Immunological profiling of VN patients has shown an imbalance in circulating leukocytes. This is characterized by an increased ratio of neutrophils to lymphocytes, indicating acute inflammation. Additionally, there is a reduction in the total number of T lymphocytes and $CD8^+$ T cells. As a result, there is an elevated $CD4^+/CD8^+$ ratio, which reflects immune dysregulation similar to what is observed in autoimmune conditions (24), suggesting that activated $CD40^+$ monocytes and macrophages can contribute to microvascular occlusion within vestibular structures, leading to decreased perfusion, ischemic injury, and subsequent neuronal dysfunction (25,26). HSV-1 may worsen this process due to its ability to evade immune surveillance. By inhibiting antigen presentation and altering T-cell responses, HSV-1 can remain in a latent state, allowing it to cause prolonged inflammation upon reactivation. This persistence can lead to ongoing tissue damage within the vestibular system (11). The findings suggest that VN is not a uniform disorder caused by a single pathogen; rather, it is a multifactorial condition where viral infections and immune activation work together to cause injury to the vestibular nerve. This combination of factors indicates that neuroinflammation may be the common pathway leading to neural dysfunction and the clinical symptoms associated with the condition.

Clinical studies consistently demonstrate that patients with viral infections often have increased neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios. These results suggest enhanced systemic inflammation and a rapid immune response. Additionally, peripheral blood mononuclear cells exhibit higher levels of tumor necrosis factor-alpha and CD40 subsets, reflecting a coordinated effort from both innate and adaptive immune systems. These cells play a crucial role in supporting leukocyte adhesion, migration, and local cytokine production. Such processes can lead to microvascular damage and endothelial dysfunction, which may cause thrombotic changes in the vestibular organ (5,27,28).

Inflammatory and vascular changes can weaken the blood-labyrinth barrier (BLB), leading to local swelling and worsening vestibular problems. The BLB plays a vital role in maintaining ionic and fluid balance in the inner ear and shielding vestibular sensory structures from harmful circulating substances. Infections or autoimmune responses can induce inflammation that damages endothelial tight junctions, impairs pericyte function, and increases vascular permeability. This can cause fluid buildup, or edema, in the vestibular system. Research indicates that pericyte detachment, basement membrane irregularities, and endothelial cell injury are linked to barrier failure and fluid accumulation. Although the precise link between BLB disruption and vestibular neuritis remains under investigation, pericyte dysfunction and vascular alterations are increasingly

recognized as important factors. Nerve edema within narrow bony canals may compress nearby vessels, worsening ischemia and barrier integrity. The loss or dysfunction of pericytes appears to control this process, while inflammatory mediators and neutrophil extracellular traps may further impair endothelial function and promote vascular leakage (29–33).

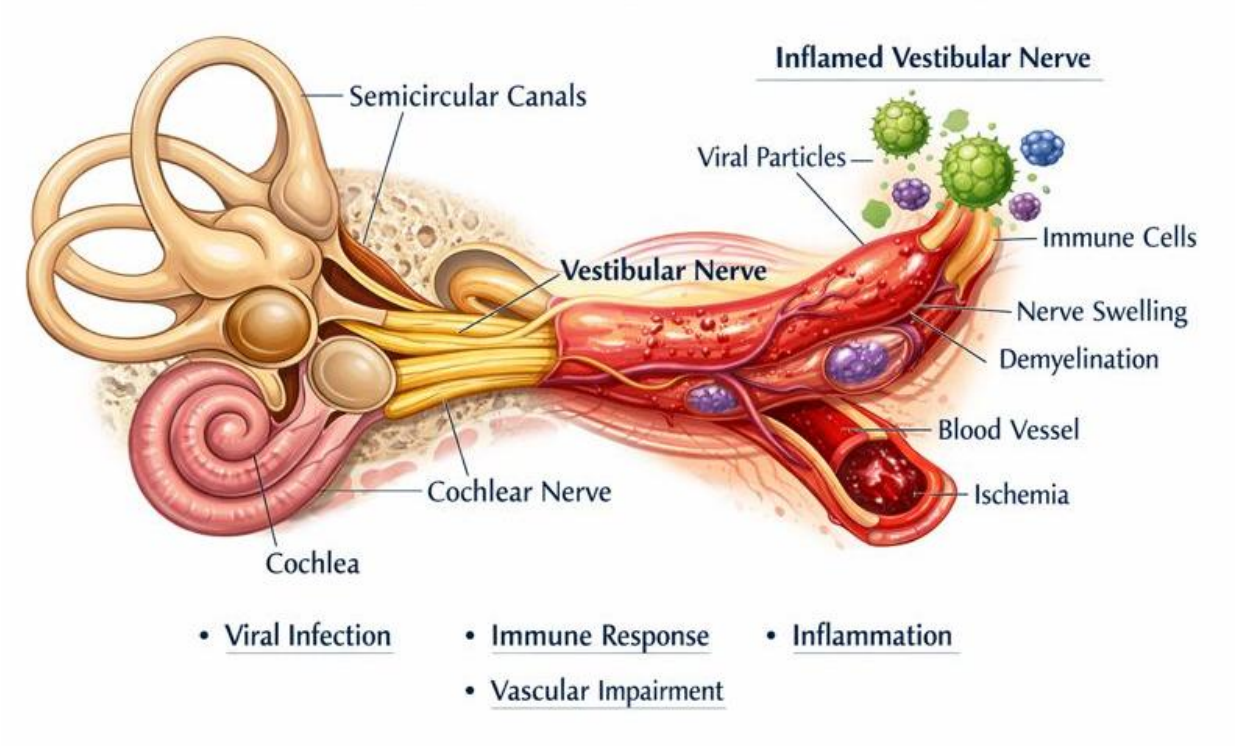

Figure 1: Vestibular neuritis pathophysiology

Therapeutic Implications

Corticosteroids are commonly used to manage VN due to their potent anti-inflammatory and immunosuppressive properties, which help reduce swelling and promote recovery of the vestibular nerve. A review of clinical trials and systematic reviews suggests that steroid therapy may provide modest short-term benefits, particularly in accelerating the recovery of vestibular function in the early stages of recovery.

Meta-analyses of both randomized and non-randomized studies have shown that patients receiving corticosteroids have higher rates of caloric test normalization. The pooled odds ratios for these studies range from approximately 2.4 to 3.1 in favor of treatment, indicating that about six to seven patients need to be treated to achieve early functional improvement. However, evidence regarding long-term outcomes—such as relief from symptoms, restoration of balance, and quality-of-life measures like dizziness handicap scores—remains inconsistent. Systematic reviews and umbrella analyses suggest that while corticosteroids may enhance caloric recovery within the first month, this improvement does not lead to sustained reductions in vertigo severity or functional disability (34,35). A prospective randomized trial comparing corticosteroid therapy, vestibular rehabilitation, and their combination found no significant differences in outcomes after 12 months. This suggests that while corticosteroids may accelerate recovery, they do not influence long-term prognosis (36). Additionally, the overall certainty of the evidence is limited due to small sample sizes, varying

study quality, and methodological differences across trials. Although adverse effects are typically mild, they occur more frequently in patients treated with steroids (37), highlighting the need for careful patient selection. Overall, current data indicate that corticosteroids may help facilitate early vestibular compensation but do not provide a lasting benefit for long-term recovery. Early initiation of vestibular rehabilitation is crucial, and future well-designed randomized controlled trials are necessary to establish the optimal role, timing, and dosage of corticosteroid therapy in cases of vestibular neuritis.

Vestibular rehabilitation therapy is crucial for recovery after vestibular neuritis, a common peripheral vestibular disorder that leads to sudden and prolonged vertigo, typically without accompanying hearing or neurological symptoms. While the exact cause of VN remains unclear, several potential factors have been suggested, including viral infections, ischemic injury to the anterior inferior cerebellar artery, and immune-mediated mechanisms (3). Traditional management often involves symptomatic treatment using vestibular suppressants and antiemetics, as well as specific pharmacologic interventions such as corticosteroids or antivirals, and structured vestibular rehabilitation exercises aimed at enhancing central vestibular compensation (3).

Clinical evidence consistently shows that introducing vestibular rehabilitation early in the disease process accelerates symptom resolution and promotes functional recovery. Patients who begin targeted rehabilitation exercises soon after diagnosis typically experience a quicker reduction in dizziness, greater improvement in daily activities, and better adaptation to head and body movements. These improvements are often accompanied by reduced anxiety and dizziness-related disabilities, indicating both physical and psychological benefits of rehabilitation (38).

A recent case study confirms the effectiveness of vestibular rehabilitation in cases of post-infectious vertigo. During the COVID-19 pandemic, numerous patients with vestibular neuritis–like symptoms after SARS-CoV-2 infection were successfully treated using standard vestibular rehabilitation techniques. Although symptom severity and recovery times varied, a structured home-based approach significantly reduced dizziness-related disability and resolved vertigo, even without medication. These results indicate that vestibular rehabilitation remains beneficial for COVID-19–related vestibular neuritis and can be safely conducted remotely or at home when in-person therapy is not feasible (39).

While objective measures, such as postural stability and gait speed, may not always reflect significant changes, patients frequently report increased confidence and balance in their daily lives. Additionally, combining vestibular rehabilitation with pharmacological therapy tends to provide extra benefits compared to medication alone. Evidence suggests that while corticosteroids may help control the acute inflammatory response and restore peripheral vestibular function, rehabilitation promotes central reorganization and long-term adaptation. This integrative approach enhances vestibular compensation, reduces residual dizziness, and leads to improved overall recovery outcomes (40).

Vestibular rehabilitation should be considered an essential part of managing vestibular neuritis. Customized exercise programs that focus on gaze stabilization, postural control, and dynamic balance—when started early and progressively advanced—can help restore functional stability and enhance the quality of life. When paired with appropriate medical treatment, VRT maximizes the potential for neural plasticity, resulting in a more comprehensive and lasting recovery.

In summary, effectively managing VN requires addressing both the acute peripheral recovery and the long-term central compensation. Corticosteroids can assist with the early restoration of vestibular function by reducing inflammation and neural edema; however, their benefits tend to be temporary. In contrast, vestibular rehabilitation offers lasting improvements through neuroplastic

adaptation, restoring balance, gaze stability, and daily functioning. The early initiation of personalized rehabilitation is a crucial component of therapy, with corticosteroids primarily serving as a supportive measure during the acute recovery phase.

Conclusion

VN is a complex disorder resulting from the interaction of viral, immune, and vascular mechanisms, which together lead to acute vestibular dysfunction and neuroinflammation. The herpes simplex virus type 1 is the most commonly implicated cause; however, there is growing evidence that immune dysregulation, cytokine-mediated damage, and disruptions to the blood-labyrinth barrier also play significant roles in the development of this condition.

Clinically, VN is generally self-limiting, with recovery largely depending on the brain's ability to compensate for central vestibular loss, rather than the extent of damage to the peripheral nerves. Among the available treatments, corticosteroids may help restore vestibular function in the early stages by reducing inflammation, though their long-term benefits are limited. On the other hand, early and tailored vestibular rehabilitation is crucial for effective treatment, as it promotes neural plasticity, enhances postural stability, and facilitates psychological adaptation.

A comprehensive treatment strategy that combines medical interventions with rehabilitation appears to be the most effective way to maximize recovery.

Future research should focus on elucidating the molecular mechanisms that link viral and immune factors to vestibular injury, as well as on developing evidence-based treatment protocols that support both peripheral and central recovery.

Conflict of interest:

There is no conflict of interest to report.

Author contribution:

All authors contributed to the conception and design of the review. **M.A., F.** performed the literature search, screened relevant articles, and drafted the initial manuscript. **S.S., A.A.** contributed to the critical revision of the manuscript and refinement of the scientific content. **A.R.** critically revised the manuscript for important intellectual content. All authors read and approved the final version of the manuscript.

Funding:

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. Adamec I, Krbot Skorić M, Handžić J, Habek M. Incidence, seasonality and comorbidity in vestibular neuritis. Neurological Sciences. 2015 Jan 2;36(1):91–5.
2. Strupp M, Brandt T. Vestibular Neuritis. Semin Neurol. 2009 Nov 15;29(05):509–19.
3. Bae CH, Na HG, Choi YS. Current diagnosis and treatment of vestibular neuritis: a narrative review. Journal of Yeungnam Medical Science. 2022 Apr 30;39(2):81–8.
4. Strupp M, Brandt T. Vestibular Neuritis. Semin Neurol. 2009 Nov 15;29(05):509–19.
5. Greco A, Macri GF, Gallo A, Fusconi M, De Virgilio A, Pagliuca G, et al. Is Vestibular Neuritis an Immune Related Vestibular Neuropathy Inducing Vertigo? J Immunol Res. 2014;2014:1–8.

6. Bronstein AM, Dieterich M. Long-term clinical outcome in vestibular neuritis. Curr Opin Neurol. 2019 Feb;32(1):174–80.
7. Kim JS, Kim HJ. Inferior vestibular neuritis. J Neurol. 2012 Aug 4;259(8):1553–60.
8. Lee JY, Park JS, Kim MB. Clinical Characteristics of Acute Vestibular Neuritis According to Involvement Site. Otology & Neurotology. 2020 Jan;41(1):143.
9. Vlastarakos P V, Sideris G, Vasileiou E, Michailidou E, Papadimitriou N, Palantzas D, et al. Developing a Diagnostic Algorithm for Identifying Vestibular Neuronitis in Acute Dizziness: An Overview of Epidemiology, Pathogenesis, and Evidence-Based Guidelines for Diagnostic Approaches. Cureus. 2025 Jan 28;
10. Ahmadi SA, Vivar G, Navab N, Möhwald K, Maier A, Hadzhikolev H, et al. Modern machine-learning can support diagnostic differentiation of central and peripheral acute vestibular disorders. J Neurol. 2020 Dec 11;267(S1):143–52.
11. Zhao Z, Liu X, Zong Y, Shi X, Sun Y. Cellular Processes Induced by HSV-1 Infections in Vestibular Neuritis. Viruses. 2023 Dec 20;16(1):12.
12. Arbusow V, Theil D, Strupp M, Mascolo A, Brandt T. HSV-1 Not Only in Human Vestibular Ganglia but Also in the Vestibular Labyrinth. Audiology and Neurotology. 2001;6(5):259–62.
13. Himmelein S, Lindemann A, Sinicina I, Horn AKE, Brandt T, Strupp M, et al. Differential Involvement during Latent Herpes Simplex Virus 1 Infection of the Superior and Inferior Divisions of the Vestibular Ganglia: Implications for Vestibular Neuritis. J Virol. 2017 Jul 15;91(14).
14. Richard C, Linthicum FH. Vestibular Neuritis. Otology & Neurotology. 2012 Sep;33(7):e59–60.
15. Rujescu D, Hartmann AM, Giegling I, Konte B, Herrling M, Himmelein S, et al. Genome-Wide Association Study in Vestibular Neuritis: Involvement of the Host Factor for HSV-1 Replication. Front Neurol. 2018 Jul 20;9.
16. Haeussler SM, Zabaneh SI, Stegemann M, Olze H, Böttcher A, Stölzel K. Is Vestibular Neuropathy Rather a Neuritis? Cureus. 2022 Oct 5;
17. Fancello V, Hatzopoulos S, Corazzi V, Bianchini C, Skarżyńska MB, Pelucchi S, et al. SARS-CoV-2 (COVID-19) and audio-vestibular disorders. Int J Immunopathol Pharmacol. 2021 Jan 18;35.
18. Malayala S V, Raza A. A Case of COVID-19-Induced Vestibular Neuritis. Cureus. 2020 Jun 30;
19. Mat Q, Noël A, Loiselet L, Tainmont S, Chiesa-Estomba CM, Lechien JR, et al. Vestibular Neuritis as Clinical Presentation of COVID-19. Ear Nose Throat J. 2023 Mar 11;102(3):NP129–32.
20. Jeong J. Vestibular neuritis after COVID-19 vaccination. Hum Vaccin Immunother. 2021 Dec 2;17(12):5126–8.
21. Shahali H, Hamidi Farahani R, Hazrati P, Hazrati E. Acute vestibular neuritis: A rare complication after the adenoviral vector-based COVID-19 vaccine. J Neurovirol. 2022 Dec 25;28(4–6):609–15.
22. Ergul Y, Ekici B, Tastan Y, Sezer T, Uysal S. Vestibular Neuritis Caused by Enteroviral Infection. Pediatr Neurol. 2006 Jan;34(1):45–6.
23. He Y, Guo T, Dai T, Zhou B, Xie H. Inflammatory proteins and vestibular neuronitis: A Mendelian randomization study. Medicine. 2024 Dec 20;103(51):e41081.

24. Zhang DG, Yu WQ, Liu JH, Kong LG, Zhang N, Song YD, et al. Serum/glucocorticoid-inducible kinase 1 deficiency induces NLRP3 inflammasome activation and autoinflammation of macrophages in a murine endolymphatic hydrops model. Nat Commun. 2023 Mar 6;14(1):1249.
25. Hegemann SCA, Wenzel A. Diagnosis and Treatment of Vestibular Neuritis/Neuronitis or Peripheral Vestibulopathy (PVP)? Open Questions and Possible Answers. Otology & Neurotology. 2017 Jun;38(5):626–31.
26. Udit S, Blake K, Chiu IM. Somatosensory and autonomic neuronal regulation of the immune response. Nat Rev Neurosci. 2022 Mar 7;23(3):157–71.
27. Şahin Mİ, Kökoğlu K, Gülmez E. Mean platelet volume, neutrophil- and platelet to lymphocyte ratios are elevated in vestibular neuritis. Journal of Clinical Neuroscience. 2019 Sep;67:134–8.
28. Quaranta NAA, Salzo AE, Pontillo V, Scarano E, Sergi B, Picciotti PM. Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) in Ménière's disease and vestibular neuritis. Hearing Balance Commun. 2021 Sep;19(4):235–9.
29. Ishiyama G, Lopez IA, Ishiyama P, Vinters H V., Ishiyama A. The blood labyrinthine barrier in the human normal and Meniere's disease macula utricle. Sci Rep. 2017 Mar 21;7(1):253.
30. Maniaci A, Briglia M, Allia F, Montalbano G, Romano GL, Zaouali MA, et al. The Role of Pericytes in Inner Ear Disorders: A Comprehensive Review. Biology (Basel). 2024 Oct 8;13(10):802.
31. Eliezer M, Hautefort C, Van Nechel C, Duquesne U, Guichard JP, Herman P, et al. Electrophysiological and inner ear MRI findings in patients with bilateral vestibulopathy. European Archives of Oto-Rhino-Laryngology. 2020 May 8;277(5):1305–14.
32. Büki B, Mair A, Pogson JM, Andresen NS, Ward BK. Three-Dimensional High-Resolution Temporal Bone Histopathology Identifies Areas of Vascular Vulnerability in the Inner Ear. Audiology and Neurotology. 2022;27(3):249–59.
33. Yi Z, Wang X, Yin G, Sun Y. The Blood-Labyrinth Barrier: Non-Invasive Delivery Strategies for Inner Ear Drug Delivery. Pharmaceutics. 2025 Apr 7;17(4):482.
34. Leong K, Lau T, Stewart V, Canetti EFD. Systematic Review and Meta-analysis: Effectiveness of Corticosteroids in Treating Adults With Acute Vestibular Neuritis. Otolaryngology–Head and Neck Surgery. 2021 Aug 2;165(2):255–66.
35. Oliveira J. e Silva L, Khoujah D, Naples JG, Edlow JA, Gerberi DJ, Carpenter CR, et al. Corticosteroids for patients with vestibular neuritis: An evidence synthesis for guidelines for reasonable and appropriate care in the emergency department. Academic Emergency Medicine. 2023 May 4;30(5):531–40.
36. Ismail EI, Morgan AE, Abdel Rahman AM. Corticosteroids versus vestibular rehabilitation in long-term outcomes in vestibular neuritis. Journal of Vestibular Research. 2019 Apr 16;28(5–6):417–24.
37. Bogdanova A, Dlugaiczyk J, Heckmann JG, Schwab S. Corticosteroids in patients with vestibular neuritis: An updated meta-analysis. Acta Neurol Scand. 2022 Nov 27;146(5):429–39.
38. Tokle G, Mørkved S, Bråthen G, Goplen FK, Salvesen Ø, Arnesen H, et al. Efficacy of Vestibular Rehabilitation Following Acute Vestibular Neuritis: A Randomized Controlled Trial. Otology & Neurotology. 2020 Jan;41(1):78–85.

39. Jafarzadeh S. Vestibular Assessment and Rehabilitation in Three Patients with Covid-19 Induced Vestibular Neuritis. Journal of Rehabilitation Sciences and Research [Internet]. 2022 Jun 1 [cited 2025 Dec 22];9(2):89–92. Available from: https://www.researchgate.net/publication/362057533_Vestibular_Assessment_and_Rehabilitation_in_Three_Patients_with_Covid-19_Induced_Vestibular_Neuritis_Original_Article
40. Huang HH, Chen CC, Lee HH, Chen HC, Lee TY, Tam KW, et al. Efficacy of Vestibular Rehabilitation in Vestibular Neuritis. Am J Phys Med Rehabil. 2024 Jan;103(1):38–46.