Review Article

The Association of Autoimmune Diseases with Tinnitus: A Narrative Review

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Short running title: The Association of Autoimmune Diseases with…

Highlights:

- Tinnitus is highly prevalent in autoimmune diseases
- Autoimmune inner ear disease has the highest prevalence, up to 95.7%
- Hearing assessments could be considered in autoimmune diseases

ABSTRACT

Background and Aim: Autoimmune Diseases (ADs), marked by immune system dysfunction and self-targeting immune responses, can damage the auditory system, leading to symptoms like tinnitus and sensorineural hearing loss. This study explores the prevalence, underlying mechanisms, diagnosis, and treatment of tinnitus within the context of ADs.

Recent Findings: This narrative review identified relevant articles through searches of Web of Science, Science Direct, PubMed, and Google Scholar, using keywords related to both tinnitus (e.g. "Tinnitus", "Ringing") and ADs (e.g. "Autoimmune Disease", "Multiple Sclerosis", "Lupus"). This review found a significant prevalence of tinnitus across various ADs. Autoimmune inner ear disease exhibited the highest tinnitus rates (50–95.7%), followed by sarcoidosis (61%) and Susac's syndrome (54–77%). Systemic lupus erythematosus (6.7–44.4%), Sjögren's syndrome (10.1–41.7%), and multiple sclerosis (up to 30%) also showed notable tinnitus prevalence. The main mechanisms involve vasculitis affecting inner ear vessels, immune complex accumulation, demyelination of auditory pathways, and hair cell damage from ototoxic immunosuppressants (e.g. cyclosporine). Regarding tinnitus treatment, corticosteroids (e.g. prednisone) offer potential benefit, though symptom relief is often temporary. Biologics (e.g. rituximab, adalimumab) have also been explored with varying degrees of success in specific cases.

Conclusion: Due to the significant prevalence of tinnitus observed in ADs, it is recommended that relevant specialists consider incorporating hearing assessments, particularly those specific to tinnitus, into their evaluation

protocols. This may facilitate earlier diagnosis and ultimately lead to more appropriate and timely management and rehabilitation strategies.

Keywords: Tinnitus; autoimmune diseases; hearing loss; vasculitis; hearing tests

Introduction

Autoimmune Diseases (ADs) represent a diverse group of conditions characterized by immune system dysfunction, leading to abnormal immune responses against the body's own tissues [1]. These diseases can affect nearly any organ, resulting in a wide range of complications from mild, easily overlooked issues to severe, life-threatening organ failure [1]. The occurrence of ADs can vary considerably, with estimates suggesting that they affect around 5% to 8% of individuals in developed countries [2]. ADs can be categorized into two main types: systemic and organ-specific. Systemic ADs, such as Systemic Lupus Erythematosus (SLE) and Multiple Sclerosis (MS), impact multiple organs throughout the body. In contrast, organ-specific ADs, such as autoimmune Ménière's disease and autoimmune inner ear disease, primarily target specific organs or systems [2, 3].

ADs can significantly affect the auditory system. These diseases damage inner ear structures (like the cochlea and vestibular system) via different immune responses, such as antibody –and complement –mediated cytotoxicity, cytotoxic T cell activity, and immune complex-mediated damage [4]. This damage leads to auditory symptoms like hearing loss and tinnitus [3]. Prior research has identified various ADs that can impact auditory system, including rheumatoid arthritis [5], lupus, Sjögren's syndrome, systemic sclerosis [6], granulomatosis with polyangiitis, Cogan’s syndrome, Behçet's disease [7], sarcoidosis, antiphospholipid syndrome, Susac’s syndrome, and MS [3]. Researchers have explored the link between ADs and Sensorineural Hearing Loss (SNHL). For instance, Di Stadio and Ralli found that 6–70% of lupus patients experienced SNHL [8], while MacMahon and El Refaie reported a hearing loss prevalence of 1–23% in MS patients [9].

Tinnitus is the perception of a persistent sound in the ear or head without an external sound source to account for it [10]. Tinnitus is often linked to hearing loss or somatosensory system activation [11]. This disorder is associated with numerous conditions, including age-related hearing loss, ototoxicity, infections, autoimmune diseases, sleep and cognitive disorders, and psychiatric illnesses [12]. Tinnitus affects around 14% of people globally, with 2% experiencing severe symptoms [13]. Chronic tinnitus can significantly impair quality of life, causing anxiety, depression, sleep problems, and stress [14]. Several studies highlight tinnitus prevalence in ADs. For instance, Matsuoka and Harris found that nearly all Autoimmune Inner Ear Disease (AIED) patients experienced oscillatory hearing loss and 95.7% had tinnitus [15]. Kühn et al. reported hearing loss in 36% and tinnitus in 24% of granulomatosis with polyangiitis patients [16]. Hardy et al. noted that 93% of Susac’s syndrome patients experienced hearing loss and 54% had tinnitus at some point during their illness [17].

Tinnitus is evaluated using various methods. These include questionnaires like the Tinnitus Reaction Questionnaire (TRQ) (measuring tinnitus-related distress) and the Tinnitus Handicap Inventory (THI) (assessing the impact on daily life) [18], as well as audiological tests such as audiometry, tinnitus-specific measures like pitch matching and frequency matching, high-frequency audiometry and Transient Evoked Otoacoustic Emissions (TEOAEs) (evaluating hearing damage and cochlear health) [19, 20]. Tinnitus management involves various approaches. Some immunosuppressants (like adalimumab and rituximab) may reduce tinnitus [15]. Antianxiety drugs or steroids can also provide symptom relief. Additionally, auditory rehabilitation with hearing aids or sound maskers, along with behavioral interventions such as Cognitive Behavioral Therapy (CBT), can enhance quality of life and reduce symptom severity [13].

While tinnitus assessment has advanced, the evidence for its risk factors and mechanisms is relatively limited, which can hinder effective treatment [21]. Autoimmune diseases are among the factors contributing to tinnitus [3]. Given the significant heterogeneity in the existing literature —including varied study designs, diverse autoimmune conditions, and inconsistent outcome measures —a systematic review was not feasible. Therefore, a narrative review approach was chosen to allow for a broader synthesis of available evidence. The aim of our study was to review existing research to identify autoimmune conditions with a higher probability of tinnitus and to investigate the underlying mechanisms of tinnitus and its management strategies in the context of these autoimmune conditions. A better understanding of tinnitus mechanisms in ADs could lead to more effective treatments for these patients. By synthesizing this information, we aimed to highlight the importance of tinnitus evaluation in individuals with autoimmune diseases, potentially assisting professionals to provide more targeted intervention and treatment.

Methods

This narrative review aimed to investigate the relationship between ADs and tinnitus. A literature search was conducted using PubMed, Web of Science, Science Direct, and Google Scholar up to December 2025. The search

terms combined keywords related to tinnitus (Tinnitus, Ringing, and Buzzing) with keywords representing various ADs, including SLE, Multiple Sclerosis, Rheumatoid Arthritis, Sjögren's syndrome, Susac's syndrome, Cogan's syndrome, Granulomatosis with Polyangiitis, Behçet's Disease, Vogt-Koyanagi-Harada Disease, autoimmune inner ear disease, and Systemic Sclerosis, among others. We included studies that reported tinnitus prevalence in clinically diagnosed and confirmed patients with any type of ADs, without specifying the kind or subtype of tinnitus. Studies were excluded if they did not provide data on tinnitus prevalence or if the patients were not diagnosed with ADs. Based on the selection criteria, a total of 26 studies reporting tinnitus prevalence in ADs were ultimately included in this review.

Results

This review explored the association between ADs and tinnitus. We found that tinnitus is a prevalent, yet often overlooked, symptom across a range of autoimmune conditions. The occurrence of tinnitus varied based on the specific ADs, the extent of immune system involvement, and the underlying pathophysiological mechanisms. This section aims to present the current evidence regarding the prevalence, underlying mechanisms, diagnostic strategies, and therapeutic modalities for tinnitus in ADs. The findings are detailed as comprehensive descriptions for the six ADs demonstrating the most significant and substantive data on tinnitus, while information pertaining to the remaining ADs is summarized in tabular form.

Autoimmune inner ear disease

AIED is a rare condition in which the body's immune system attacks the inner ear, leading to a progressive and typically bilateral SNHL that fluctuates and develops over weeks or months [22]. Tinnitus is highly prevalent in this condition, with reported rates ranging from 50% to as high as 95.7% – the highest observed prevalence among investigated ADs [15]. As an illustration, Bustos-Merlo et al. found that 56.25% (9 out of 16) of their 55 patients reported experiencing tinnitus. SNHL is another common finding, affecting 80% to 100% of patients, and it typically presents as a bilateral and progressive condition [23]. The underlying causes of tinnitus and hearing loss involve immune complex deposition and vasculitis affecting inner ear vessels, leading to reduced blood flow and subsequent damage to hair cells [4, 7]. Furthermore, anti-HSP70 antibodies contribute to inflammation, potentially worsening the damage [24]. Corticosteroid therapy, such as 60 mg of prednisone daily, demonstrates efficacy in 50-70% of cases, often leading to a reduction in tinnitus [25]. Biologic therapies, including adalimumab and rituximab, have shown even greater improvement in tinnitus, with success rates ranging from 70–100% [15]. Intratympanic injections have also demonstrated hearing improvement in 50% of the cases [25]. Fioretti et al. used the THI in their study evaluating the impact of tinnitus on patients' lives. In one case, a patient with severe bilateral tinnitus (8 kHz, 15 dB above threshold) experienced successful management of their symptoms with bilateral broadband sound generators (8–9 h/day) [26]. However, the temporary nature of treatment effects in this condition often requires ongoing monitoring [22].

Systemic lupus erythematosus

SLE is a chronic AD involving the production of autoantibodies against nucleic acids, proteins, and other cellular components, affecting multiple organs, including the auditory system [8, 27]. Tinnitus prevalence in SLE varies, with reported rates of 6.7% [28], 32% [29], 35.8% [30], 40% [31], and 44.4% [32]. SNHL is also frequently observed, with reported prevalences ranging from 6% to 70% [8]. The underlying causes of tinnitus and hearing loss in SLE are multifactorial. Immune complex deposition in inner ear vessels leads to vasculitis and reduced blood flow. Additionally, the release of Reactive Oxygen Species (ROS) and inflammatory cytokines contributes to direct damage to hair cells and ganglion cells [8]. Tinnitus may be worsened by anti-cardiolipin antibodies, which can lead to the formation of microthrombi in the cochlear vessels. Additionally, certain medications, such as hydroxychloroquine, can also contribute to the exacerbation of tinnitus [31, 33]. Corticosteroids (such as prednisone at 1–1.5 mg/kg/day) are the primary treatment for these patients, often supplemented with rituximab to further reduce the severity of tinnitus [3, 8].

Vogt-koyanagi-harada disease

Vogt-Koyanagi-Harada disease (VKH) is a rare systemic AD characterized by granulomatous inflammation targeting melanocytes of the inner ear and other tissues [34]. Tinnitus is a frequent auditory symptom in VKH, with a reported prevalence ranging from 24% to 54% [35]. For instance, Al Dousary found that 42% (10 out of 24) of patients experienced tinnitus [34]. Similarly, studies conducted in Brazil reported tinnitus in 24% of patients (16 out of 67), while studies in Japan found a higher prevalence of 54% (74 out of 137 patients) [35]. Ruiz-Allec et al. also reported that 42.85% (9 out of 21 patients) had tinnitus, which was bilateral in 8 patients

and unilateral in 1 patient [36]. In VKH, SNHL is also a common finding, with reported prevalence rates ranging from 30% to 75% [35]. The underlying mechanism of tinnitus involves an autoimmune T-cell response targeting melanocytes in the stria vascularis, disrupting endocochlear potential generation [37]. Furthermore, the deposition of immune complexes and ROS intensifies cochlear inflammation, leading to tinnitus [35]. Increased interferon-gamma secretion also contributes to tinnitus by increasing the activity of auditory neurons [38]. Tinnitus assessment typically involves pure-tone audiometry, often revealing high-frequency involvement. Absent Otoacoustic Emissions (OAEs) in severe cases can further confirm cochlear damage [34, 39]. Systemic corticosteroids (e.g. prednisolone, 1–2 mg/kg/day) are the primary treatment approach [33], while immunosuppressive drugs (e.g. methotrexate) and intraocular steroids can also be effective in managing tinnitus [40].

Behçet's disease

Behçet's syndrome, characterized as chronic small vessel vasculitis, is a systemic autoimmune disease with relapsing episodes [41]. In Behçet's syndrome, tinnitus prevalence varies, with studies by Kulahli et al. and Karadağ et al. reporting rates of 11% and 27.3%, respectively. Karadağ et al. found that 27.3% (n=12) of 44 Behçet's syndrome patients experienced tinnitus, compared to none in the control group [20]. Kulahli et al. found that 11% (n=7) of 65 Behçet's patients reported tinnitus [42]. Tinnitus frequently co-occurs with hearing loss, and SNHL prevalence has been reported between 12% and 80% [6, 40, 41]. The underlying mechanism of tinnitus involves vasculitis affecting the small vessels of the inner ear, leading to impaired blood supply to the cochlea [41]. Immune complex deposition and the production of ROS contribute to exacerbated inflammation and abnormal auditory neuron activity [7]. Furthermore, cytokines such as TNF-α and IL-1 have been implicated in the pathogenesis of tinnitus [43]. The Visual Analogue Scale (VAS) and TRQ are frequently used to measure how tinnitus affects daily life, with studies showing a strong correlation between the two assessment tools [20]. The VAS is a simple, reliable tool used to measure tinnitus severity by having patients rate their distress or annoyance on a 0 to 10 scale [18]. Cochlear damage, indicated by impaired TEOAE in 20.5% of patients, has been observed [20]. Treatments offer varying success: corticosteroids (like prednisolone) improve tinnitus in 44–70% of cases, while adalimumab completely eliminated it in one instance [43]. Amantadine has also demonstrated temporary reduction of tinnitus symptoms [44].

Multiple sclerosis

MS, an autoimmune disease that causes inflammation and demyelination in the central nervous system, can also affect auditory system [45]. Tinnitus is frequently reported in MS patients, with a prevalence ranging from 19% to 30%, and reaching 100% in cases of Sudden Sensorineural Hearing Loss (SSNHL) [9, 46, 47]. SNHL is another auditory issue seen in MS patients, with a prevalence of 0.4% to 23.3%, and it can develop suddenly or gradually [9, 47]. Tinnitus in MS is linked to demyelination within auditory pathways (including the eighth cranial nerve and brainstem), leading to irregular auditory neuron activity [3, 47]. Tinnitus can also be caused by autoimmune inflammation in the cochlea, which leads to the production of ROS and inflammatory substances like IL-1β and TNF-α [45]. In these patients, the assessment of tinnitus involved pure tone audiometry and OAE evaluation, indicating cochlear damage in 34% of the cases [45]. Corticosteroid treatment (methylprednisolone 1 g/day) has shown to improve tinnitus and SSNHL in 77% of cases [47].

Sjögren's syndrome

Sjögren's Syndrome, a chronic autoimmune disease, causes dry eyes and mouth due to immune cell infiltration, and can also impact the auditory-vestibular system [48, 49]. Tinnitus is frequently observed in individuals with Sjögren's syndrome, with studies reporting its occurrence in 10.1% to 41.7% of patients [49]. In Sjögren's patients, tinnitus may be bilateral, be constant or episodic, and commonly occurs alongside hearing loss, vertigo, temporomandibular joint issues, or nerve damage [49]. SNHL is another frequent finding in Sjögren's syndrome, affecting 22.5% to 46% of patients [3]. The development of tinnitus is thought to involve the accumulation of immune complexes in the stria vascularis, leading to microvasculitis and reduced blood flow (ischemia) [49]. Furthermore, anticardiolipin antibodies and M3 muscarinic receptors (mAchRs) are believed to contribute to the progression of hearing loss, neurological issues, and tinnitus in these individuals [3]. The creation of ROS and inflammatory cytokines, including type I interferon, contributes to the damage of hair cells and the development of tinnitus [49, 50]. In addition, hydroxychloroquine has the potential to worsen neurotoxicity, which can result in the sudden onset of tinnitus accompanied by headache and ataxia [51]. For managing tinnitus related to this condition, corticosteroids like prednisolone show effectiveness in 44–70% of cases [33]. In instances of severe

SNHL, hearing aids or cochlear implants are advised. CBT is also a recommended approach for managing the tinnitus itself [49].

Prevalence of tinnitus in other autoimmune disorders

Tinnitus is a frequently reported symptom across various uncommon ADs that affect hearing, often occurring alongside SNHL. Studies show that tinnitus prevalence reaches 80% in Cogan's syndrome and 13% in children with the condition [52, 53]. Systemic vasculitides like Granulomatosis with Polyangiitis (GPA) exhibit a tinnitus prevalence of 24–40% [16, 54]. Studies indicate tinnitus prevalence ranges from 54–77% in individuals with Susac's syndrome [17], is 38% in those with rheumatoid arthritis [6], and reaches 50% in systemic sclerosis [55]. Lower rates of tinnitus are observed in dermatomyositis/polymyositis (16.1%) [56], while higher rates are seen in neurosarcoidosis (61%) [57], and Takayasu arteritis (49%) [30]. The underlying causes of tinnitus in these conditions are varied, involving processes like vasculitis, the build-up of immune complexes, and inner ear ischemia [3]. To enhance reader comprehension, Table 1 summarizes tinnitus and SNHL prevalence data reported in previous studies.

Discussion

This narrative review examined the prevalence of tinnitus in various ADs and also explored the underlying mechanisms of tinnitus, as well as tinnitus evaluation and treatment methods. The results indicated a high occurrence of tinnitus in conditions like AIED, ranging from 50–95.7% [15, 26], as well as in sarcoidosis (61%) [57] and Susac's syndrome (54–77%) [17]. However, tinnitus is less common in MS, with a prevalence of less than 30% [46, 47]. The variability in tinnitus prevalence across ADs likely reflects differences in the underlying pathophysiological mechanisms and the degree to which each condition directly affects the auditory system [58]. Disorders such as AIED and Susac's syndrome typically involve direct cochlear or vestibulocochlear nerve damage, immune-mediated inner ear injury, or vasculitis affecting the cochlear microvasculature, leading to high tinnitus prevalence (50–95.7%) [4, 59]. These are primarily organ-specific autoimmune diseases, where the immune response directly targets inner ear structures or adjacent vessels [4]. In contrast, systemic autoimmune diseases (e.g. multiple sclerosis, Behçet's disease) affect the audiovestibular system through less direct or less frequent mechanisms, such as central nervous system demyelination, systemic vasculitis, or indirect inflammatory effects, rather than primary cochlear damage [60]. This difference in underlying pathophysiology likely explains the lower and more variable tinnitus rates observed in systemic diseases, such as <30% in MS [9, 45]. Conditions like Flammer syndrome and Sweet syndrome had insufficient data for inclusion in this review [61, 62].

Tinnitus mechanisms in ADs often involve cochlear damage stemming from vasculitis (e.g. Cogan syndrome [63]), immune complex deposition (e.g. rheumatoid arthritis [64]), or demyelination (e.g. MS [47]). Furthermore, immunosuppressive drugs (like cyclosporine and anti-TNFα) can worsen tinnitus and SNHL via ototoxicity, disrupting the blood-inner ear barrier and damaging hair cells [65]. Taken together, autoimmune-mediated auditory dysfunction can be conceptualized as a multi-step process in which systemic autoimmunity initiates vascular, neural, or barrier injury in the inner ear [63]. These pathways converge in cochlear hair cell and auditory nerve damage, and secondary factors such as ototoxic immunosuppressants amplify injury [65]. The resulting disruption of cochlear homeostasis and abnormal neural signaling provides a mechanistic basis for tinnitus and SNHL in ADs. Clinically, tinnitus and SNHL can serve as early indicators of autoimmune diseases, aiding in early diagnosis [7]. Recognizing these symptoms, particularly in conditions like sarcoidosis and Cogan's syndrome, can lead to faster identification of the underlying disease and potentially prevent further damage to the auditory system [57, 63].

Strengths, limitations, and directions for future research

To our knowledge, this is the first narrative review to synthesize evidence on the association between ADs and tinnitus, offering an overview of tinnitus prevalence across a wide range of ADs. Regarding the evidence base, key limitations include incomplete data for some diseases, methodological inconsistencies across studies, and a lack of detailed information on tinnitus severity, treatment response, and relevant environmental or genetic factors. Additionally, some reported tinnitus cases may be confounded by medication-induced side effects rather than directly attributable to the primary autoimmune process itself. Many prevalence estimates come from small case series or case reports, contributing to heterogeneity in the available evidence. As a narrative review, our study did not apply a formal quality assessment or risk of bias tool, which is a recognized methodological constraint of this review type. Furthermore, our search was limited by a lack of access to certain databases. For future research, given the rarity of many ADs, large multicenter registries and collaborative research networks

represent the most feasible approach to accumulate adequate sample sizes. Well-designed cross-sectional and prospective cohort studies are also needed to track tinnitus progression and identify variations across disease subtypes. Additionally, future studies should explore potential mechanisms linking specific ADs to tinnitus, including inflammatory, autoimmune, and vascular pathways. Finally, this review highlights the importance of clinician awareness regarding the association between tinnitus and SNHL with ADs, emphasizing the need for more comprehensive diagnostic and therapeutic approaches.

Conclusion

This review shows that tinnitus is reported with notable frequency in several autoimmune diseases, with particularly high rates observed in autoimmune inner ear disease (50–95.7%) and sarcoidosis (61%). Vasculitis, immune complex deposition, and demyelination, which often cause cochlear damage, are key mechanisms in these disorders. Our findings emphasize the importance of audiologic evaluation and consideration of tinnitus management options in this patient population.

Ethical Considerations

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Authors' contributions

AD: Study design, interpretation of the results, and critical revision of the manuscript; MAS: Search and screening of papers, preparing the first draft; ZJ: Search and screening of papers, revision of the manuscript; AH: Study design and supervision, interpretation of the results, revision of the manuscript.

Conflict of interest

The authors declare that they have no competing interests.

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Table 1. Summary of reported prevalence (%) of tinnitus and sensorineural hearing loss in autoimmune diseases based on literature

Autoimmune disease	Tinnitus prevalence	SNHL prevalence
Systemic lupus erythematosus	6.7 [28]–44.4 [32]	6–70 [8]
Multiple sclerosis	<30 [9]	0.4–23.3 [45]
Sjogren's syndrome	10.1–41.7 [49]	5.6–54.17 [3]
Granulomatosis with polyangiitis	24–40 [16]	8–65 [16]
Systemic sclerosis	50 [55]	20–77 [55]
Behcet's disease	11 [42]–27.3 [20]	12 [41]–80 [42])
Susac's syndrome	54–77 [17]	37–97 [17]
Vogt-koyanagi-harada disease	24–54 [35]	30–75 [35]
Cogan's syndrome	13–80 [53]	31–65.2 [53]
Dermatomyositis/polymyositis	16.1 [56]	9.2 [56]
Rheumatoid arthritis	38 [6]	25–75 [64]
Sarcoidosis	61 [57]	90 [57]
Autoimmune inner ear disease	50 [23]–95.7 [15]	80–100 [22]

SNHL; sensorineural hearing loss