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Review Article

Audiovestibular manifestations in patients with rheumatologic diseases: a review

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ABSTRACT

Rheumatologic disorders are chronic and systemic disorders with an estimated global prevalence of 3,200 per 100,000 inhabitants. Several clinical manifestations are known and may lead to physical disability and altered quality of life. The most common audio vestibular manifestations commonly reported are hearing loss, tinnitus, vertigo, and fullness, however, there are some more still unregistered. The aim of this review is to describe the most common audio vestibular features in three of the most common rheumatologic conditions such as rheumatoid arthritis (RA), systemic erythematous lupus and systemic sclerosis (SSc). A literature search was performed with articles published between 2000 to 2021. In this literature review an increased prevalence of hearing loss for RA of 40% and 70% for systemic erythematous lupus was observed. In SSc, due to its early onset and progression, there is no estimated prevalence, however, a notorious hearing impairment in early stages of the disease was described. Vertigo was also described in these disorders, affecting more than 60% of patients. Further studies are in need to describe the affection on early stages of all disorders as well as its progression.

Keywords: Sensorineural hearing loss, Rheumatology, Vertigo, Tinnitus, Vestibulocochlear nerve diseases, RA, Systemic scleroderma, Systemic lupus erythematosus

INTRODUCTION

Rheumatological disorders are frequent multisystemic and progressive affections that may lead to physical disability and deterioration in the quality of life.[1] Systemic lupus erythematosus (SLE), RA and SSc are the most widely known.[1] Nearly 3-5% of the world population is diagnosed by any autoimmune pathology, with an estimated global prevalence of 3,200 per 100,000 inhabitants. [1] Females are most affected population.[1]

Audio-vestibular manifestations in patients with rheumatological conditions currently represent a focus of research interest over the last 40 years.[2] In 1979, McCabe described the characteristics of autoimmune disease to the inner ear, in a case-series of 18 patients with rheumatological conditions, who complained of hearing loss, aural fullness, tinnitus and vertigo.[2] Subsequently, immunological involvement was described in other areas of the inner ear, such as stria vascularis, the spiral ligament, and the spiral ganglion cells.[3] Cellular and vasculitic changes associated to the action of T and B lymphocytes at cochlea, vestibule and semicircular canals, and the deposition of immune complexes in basal, middle and apical turns of cochlea have been described in the last 20 years.[3,4] However, to date, there are some controversies regarding the anatomical and functional involvement of the middle and inner ear in patients with rheumatological diseases.[5,6]

The aim of this review is to present a narrative review regarding the audiovestibular manifestations on patients with rheumatological conditions such as RA, SLE and SSc, describing their current prevalence, clinical manifestations and relevant histopathologic data correlated with functional and anatomic.

LITERATURE REVIEW

This narrative review was conducted between February and October 2022. We used the MeSH and terms were used: '"vertigo", "dizziness", "hearing loss", "autoimmune diseases", "tinnitus", "ear symptoms", "RA", "systemic erythematosus lupus", "SSc"; which were combined with the boolean operator "AND" to search PubMed, EBSCO, Scielo, Google Scholar and Bvsalud databases for retrospective chart reviews, prospective studies, cohort, cross-sectional, and case-control. This search aimed to describe the frequency of audiovestibular symptoms associated to some of the most common rheumatologic conditions such as RA, SSc and systemic erytemathous lupus and describe the audiometric and vestibular testing in patients who complained of audiovestibular symptoms during lifetime.

This search was limited to articles between 2001 and 2021. We included complete manuscripts published in English which reported patients with RA, SLE, and SSc who complained of hearing loss, vertigo, tinnitus, aural fullness, and hyperacusis and those who had available pure-tone audiometry, videonystagmography, videohead impulse test and other audiologic testing.

Editorials, narrative reviews, mini-reviews, comments to the editor, and abstracts were excluded. Studies outside the field of otolaryngology and rheumatology were also excluded as those focused on pediatric populations. Results were cross-checked among the three authors. A total of 155 indexed articles were obtained in the initial search. From these 49 articles focused on SLE, 66 on RA, and 40 on SSc were selected. Most of studies were observational, including case-control studies, systematic reviews, and meta-analyses (Figure 1).

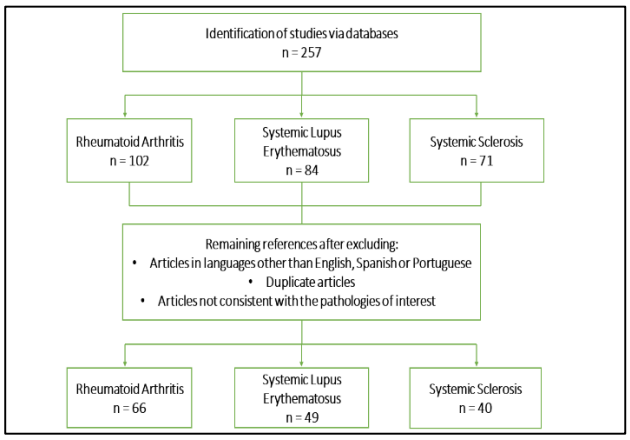

Figure 1: Study flowchart.

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic, systemic, and progressive autoimmune disease, mainly affecting synovium of synarthroses and diarthroses and also presenting extra-articular manifestations, such as pulmonary and cardiovascular involvement, rheumatoid nodules, systemic comorbidities and audiovestibular system alteration.[4,5] Up to 40% of patients diagnosed with RA present extra-articular manifestations, its presence is related to a significant increase in morbidity and mortality and deterioration in quality of life.[4] Other patients have reported audiovestibular symptoms, such as hearing loss and vertigo over the last 50 years.[4] Conductive and sensorineural hearing loss have been described, usually associated to laxity and stiffness at the incudomalleolar and incudostapedial joints and ototoxicity due to NSAIDs, prolonged use of aspirin and antibiotics.[3,4]

Hearing loss is the most common clinical otologic manifestation in patients with RA.[4] Nowadays, sensorineural hearing loss is usually described up to 40% of patients, while conductive hearing loss is described at least in 20% of patients diagnosed with RA.[5,6] In early 1970 and 1980, the prevalence found for sensorineural hearing loss was 24% to 60%, and for conductive hearing loss between 0% to 13%.[4] In a case-control study conducted at a tertiary care institution in Turkey, the prevalence of sensorineural and conductive hearing loss was reported to be 35.1% and 24.3%, respectively.[7] In a prospective study in the United States, a lower prevalence of hearing loss was reported; however, the highest figures were found for sensorineural hearing loss at 11.9%, and 7.1% corresponding to conductive hearing loss.[3] Other authors in Japan continued to report sensorineural hearing loss in patients with RA in 36.1%, but no findings for conductive hearing loss were detailed.[5] Only one study has specified the laterality of hearing loss, finding figures of 60% of sensorineural hearing loss in the patients studied with bilateral predominance, while for conductive hearing loss a prevalence of 11.42% was also found with a bilateral component.[8]

Development of RA involves a complex interaction between genetic and environmental factors, resulting in loss of immunologic tolerance and an increased activity of CD4+ T lymphocytes and proliferative factors associated with subsequent bone and cartilage erosion.[4,5] Sensorineural hearing loss is usually associated to immune mechanisms to the cochlea, deposition of autoantibodies at basal and middle turns of cochlea, cytotoxic events at outer and inner hair cells and vascular occlusion at stria vascularis, spiral ligament, labyrinthine artery, and cochlear nerve.[5,7] At cochlea, some otopathologic studies described an atrophy of the stria vascularis, decreased hair cells population, atrophy of the organ of Corti, and degeneration of spiral ganglion neurons.[4,7] Conductive hearing loss is associated with laxity at incudomallear and incudostapedial joints at the

beginning of this disorder, and delayed stiffness and decreased synovial space in later stages of RA.[4,8] During lifetime, a persistent inflammation is usually seen and even at postmortem stages, bone erosion and diminished synovium space are seen.[4,5,8] However, biomechanical and vasculitic changes are not the only ones described.[4,5] The action of proinflammatory cytokines (IL-6), metalloproteinases (MMP-3, MPP-6) and tumor necrosis factor alpha (TNF-α) are associated with oxidative and enzymatic damage to the hair cells, leading to sensorineural hearing loss.[5,9] One of the most relevant studies that addressed the relationship between inflammatory mediators and sensorineural hearing loss was carried out in Japan in 2005.[5] The researchers performed audiometry and tympanometry, and also measured the amount of proinflammatory cytokines and metalloproteinases in the plasma of their patients with RA, concluding that chronic inflammation contributes to the incidence of sensorineural hearing loss because plasma concentration of IL-6 and MMP-3 is increased in patients with RA and is associated with hearing impairment. [5]

Some events associated to the onset of sensorineural hearing loss are environmental factors, such as smoking, alcoholism or prolonged exposition to noise.[4] Passive and active smoking are at increased risk of hearing impairment due to vasoconstriction, decreased oxygen concentrations, and elevated levels of monoxides produced by nicotine, leading to irreversible cell damage and decreased cochlear function.[4,9] Bellé described the relationship between alcohol consumption and sensorineural hearing loss, secondary to its harmful effects on cochlear function.[10] Prolonged exposition to noise leads to outer and inner hair cell damage and increased injuries to the basal turns of cochlea.[8] In addition to environmental factors, other elements specific to the patient with RA have been described in the literature, which could be related to audiovestibular impairment, including age, sex, presence of rheumatoid nodules, activity and duration of the disease, rheumatoid factor and elevation in acute phase reactants.[9]

Vestibular involvement in patients with RA has been reported in several studies, associated to vasculitic and cytotoxic events to the utricle, saccule, and semicircular canals.[11] Some prolonged inflammatory events at the superior vestibular nerve and ampullary nerves are related to unilateral vestibular deficits.[11] On vestibular testing, unilateral vestibular hypofunction and unilateral and bilateral areflexia have been described mainly at later stages of the disease.[12] Even though, vertigo is common in patients with RA, further studies are needed including videohead impulse testing and posturography to describe other involvements at semicircular canals, utricle and in proprioception.[12] Table 1 summarizes audiovestibular manifestations found in patients with RA in multiple studies performed from 2001 to 2021.

Currently there is no standard treatment for patients diagnosed with RA who present audio-vestibular involvement. Some options described include the use of antioxidants such as N-acetylcysteine, sodium salicylate and vitamin E to protect the inner ear, and vasodilators like betahistine to optimize cochlear circulation.[4] Patients with conductive hearing loss may require in some cases surgical intervention to improve sound conduction and air-bone gap.[7] In most of cases, hearing impairment in patients with RA is usually treated with hearing aids and implantable bone devices.[13,14] Interdisciplinary work with rheumatologists should be promoted to detect and follow-up these symptoms. Further studies are in need to explore these symptoms as possible future markers of progression of this disease.

Table 1: Audiovestibular manifestations in patients with RA.

Author/year	Country	#Individuals	Conductive HL	Sensorineural HL	Mixed HL	Tinnitus	Vertigo
Raut, 2001[8]	England	74	+	+	-	-	NE
Özcan, 2002[7]	Turkey	72	+	+	+	-	NE
King, 2002[13]	Netherlands	40	NE	NE	NE	-	-
Öztürk, 2004[31]	Turkey	119	-	+	-	-	NE
Takatsu, 2005[5]	Japan	72	-	+	-	-	NE
Halligan, 2006[32]	United States	59	+	+	+	-	+
Murdin, 2007[33]	England	54	+	+	-	-	NE
García-Callejo, 2007[34]	Spain	301	-	+	-	-	NE
Dikici, 2009[4]	Turkey	40	-	+	-	+	NE
Pascual-Ramos, 2012[35]	Mexico	101	-	+	-	-	NE
Özkırıs, 2013[11]	Turkey	162	NE	NE	NE	-	+
Treviño-González, 2015[36]	Mexico	117	NE	+	NE	+	+
Ahmadzadeh, 2017[3]	Iran	82	+	+	-	-	NE
Rahne, 2017[37]	Germany	56	-	+	-	-	NE
Dessouky, 2017[38]	Egypt	60	-	+	-	-	+

SYSTEMIC LUPUS ERYTHEMATOSUS

SLE is a chronic multiorganic disorder including a wide spectrum of clinical manifestations mainly at skin, kidneys, and eyes. Currently, the incidence is estimated in 12 to 39 per 100,000 inhabitants in the United States.[15] This disorder mainly affects females, African and Asian descendants.[16] Its etiology includes genetic and environmental factors, and the interaction of innate and adaptative cellular responses, leading to increased tissue damage, inflammation and multiorgan compromise.[16,17] An increased action of autoantibodies called anti-DNA in SLE leads to increased production of oxidative processes, apoptosis of cochlear hair cells, in addition to increased deposition of the immune complexes at the microvessels and reduction of the vessel's caliber and decreased oxygenation to the inner ear.[16,17] The use of ototoxic drugs in sustained remission episodes, including antimalarials, may affect the cochlea and the cochlear nerve, inducing sensorineural hearing loss.[17] A cross-sectional study conducted in Brazil that included 43 patients with SLE, an attempt was made to evaluate the auditory function of patients with this pathology who were using antimalarials. In this sample, 37/43 patients (86%) used chloroquine or hydroxychloroquine for more than seven years, these 7/37 (18.9%) presented sensorineural hearing loss, while in the group that did not use antimalarials, half presented sensorineural hearing loss (3/6=50%).[18] However, it is difficult to investigate the effect of antimalarials on hearing loss, since autoimmune pathology itself induces alterations in the inner ear, making it difficult to know which factor is responsible for hearing loss.

To the ear, these events may occur leading to hearing impairment. The most common audio vestibular finding in SLE is sensorineural hearing loss, with an estimated prevalence of 6-70%.[16,17] The onset of hearing loss in SLE may be progressive or sudden in some specific cases, being in most of the patients with bilateral predominance.[16] Asymptomatic hearing loss is widely reported in patients with SLE. [16,19] One prospective study, indicated a prevalence of asymptomatic sensorineural hearing loss in nearly 66% of patients with SLE, presenting at acoustic immittance, curves type A and as without alterations in stapedial reflexes.[19] Other audio-vestibular symptoms known in SLE are tinnitus and vertigo, reporting for tinnitus a prevalence of 44.4% and 31.1% for vertigo.[20] There is few information reporting how vertigo and tinnitus occurs. Further studies are in need for better understanding in this disorder.

A meta-analysis carried out in 2017 described the findings of 52 temporal bones of patients with SLE.[16] The symptoms that the patients presented before death were studied, the most common was hearing loss. Hearing loss was sensorineural in 96% of the cases and mixed in 4%, in addition, 30% of the patients reported vertigo and 8% tinnitus.[16] Regarding histopathological findings, polymorphonuclear infiltration and vasculitis were the most common, followed by fibro-osseous reaction, new bone formation and granulation. Besides, cochlear findings confirmed moderate to severe inner and outer hair cell damage, stria vascularis atrophy, and spiral ganglion degeneration. With respect to vestibular system, type I vestibular hair cell damage was the most frequent finding, followed by vestibular fibrosis and hydrops.[16]

Diverse therapeutic alternatives have been proposed for patients with SLE and audiovestibular disorders, most of them focused on prevention, and on hearing restoration for those patients with sudden hearing loss.[16] In these cases, steroids are the first line of treatment because of the high availability and low cost compared to other therapies, and due to their anti-inflammatory action, that may restore the caliber of the vessels, and immunosuppressants, which should reduce the formation of immune complexes.[16] Other options include plasmapheresis, which improves oxygen concentrations to the inner ear, leading to better hearing function, but is an invasive and expensive procedure.[21] Anticoagulant therapy improves blood flow by optimizing the vascularization of the vessels compromised by SLE. And immunosuppressive drugs such as cyclophosphamide or azathioprine decrease the formation of immune complexes. [16,22] Another group of drugs that have been implemented recently to achieve the same objective are monoclonal antibodies such as rituximab or alemtuzumab, these are an alternative in patients with aggressive forms of SLE or proven resistance to other therapies. [16,22] However, despite large number of patients with SLE who present hearing disorders, there is no standard treatment focused. [16,17] Table 2 shows summary of results of some studies that address this topic.

Table 2: Audiovestibular manifestations in patients with LES.

Author/year	Country	#Individuals	Conductive HL	Sensorineural HL	Mixed HL	Tinnitus	Vertigo
Kastanioudakis, 2002[27]	Greece	93	+	+	-	NE	NE
Roverano, 2006[19]	Argentina	56	+	+	+	NE	NE
Gomides, 2007[20]	Brazil	90	-	+	-	+	+
Karatas, 2007[39]	Turkey	56	-	+	-	+	+
Maciaszczyk, 2011[28]	Poland	67	-	+	-	+	+
Abbasi, 2013[15]	Iran	90	-	+	-	+	NE
Batuecas, 2013[40]	Spain	89	-	+	-	-	+
Rahne, 2017[37]	Germany	62	+	+	+	NE	NE

SYSTEMIC SCLEROSIS

Systemic sclerosis (SSc) is a multisystemic and progressive disorder of unknown etiology, characterized by skin fibrosis, increased thickness of blood vessels, and clinical manifestations at visceral organs such as the gastrointestinal tract, lungs, kidneys, and heart.[23] The current prevalence in the United Stated ranges from 4-12 people per 1.000.000 inhabitants per year.[23] The average age of its onset usually happens between 30 and 50 years. Females are mostly affected, with an increased presentation in those at childbearing ages.[23] Viral infections, some chemical agents such as benzene chloride, and drugs like bleomycin have been described as main triggers for SSc.[23] Patients with SSc are classified into 2 groups based on the extension of skin involvement: diffuse SSc, which affects the skin of trunk, extremities, face, and neck; and limited SSc, where there are no lesions on trunk and its involvement has a distal pattern, up to knees and elbows, in addition to face and neck.[24] CREST syndrome is limited presentation of SSc, including calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly and telangiectasia.[23,24]

The first otopathologic and pathophysiologic description of this disorder was made by Abou-Taleb in 1987, where he stated that blood vessels and endothelial cells are mainly affected by humoral and cellular autoimmune hyper-reactivity of the host.[25] Although it was described and considered a systemic pathology since the 19th century, its etiology and pathogenesis remain unknown, but there are several hypotheses about it.[23] Vasculopathy is a determining factor in the clinical presentation of SSc because it causes inflammation, fibrosis, and collagen deposition in organs and tissues.[24] A proliferation of the intimal layers of the arteries lead to vascular occlusion and causes isolated pulmonary hypertension, myocardial perfusion disorders, Raynaud's phenomenon, and arterial hypertension secondary to the reduction of the renal artery lumen.[24,26] Hearing impairment would potentially explain by this same hypothesis since vascular occlusion leads to hypoxia and apoptosis of hair cells in the cochlea consequently lead to sensorineural hearing loss.[26] In temporal bones, cochleovestibular vascular changes have been described including atrophy and loss of the organ of Corti and atrophy of the stria vascularis. [25]

There are few studies regarding SSc and hearing loss. One prospective study of patients with SSc, 20% of the cases presented sensorineural hearing loss, and 3.3% mixed hearing loss; however, no correlation was found between hearing loss and age, systemic manifestations of the disease, positivity of autoantibodies or pharmacological treatment received by these patients. [27] In 2010, a prospective study evaluated hearing function in patients with SSc in relation to the duration, type and severity of disease, and presence of Raynaud's phenomenon in 20 cases (which were separated according to the type of sclerosis).[28] The results reported sensorineural hearing loss in eight patients (40%), mainly bilateral and symmetric. In this study, no association was found between type of hearing loss and the duration of the disease.[28] Other symptoms associated with hearing loss were vertigo in 12 patients (60%), 11 patients with headaches (55%), 10 patients with tinnitus (50%), 8 with hyperacusis (40%) and 6 patients with aural fullness (30%).[28]

Benign paroxysmal positional vertigo (BPPV) is the most frequently described vestibular manifestation in this group of patients, its prevalence ranges between 11-63%.[23] This could be associated with reduced or interrupted blood flow to the vestibular system secondary to vasculopathy, which is one of the main clinical manifestations of SSc, however, there is a lack of information about the possible hypothesis associated to the pathogenesis of these symptoms.[26,29] One of the few studies was made in Latin America reporting a prevalence of BPPV of 17% that compromised mainly the posterior semicircular canal.[29]

There is no standardized treatment for audiovestibular disorders in patients with SSc, however, medication with vasodilator effect could provide a net benefit in the prevention of hearing loss by improving the vasculopathy that precedes the fibrosis and subsequent damage of the skin and internal organs.[26] The literature reports the case of a 65-year-old male patient with a diagnosis of SSc and a history of recent-onset bilateral sensorineural hearing loss who received immunosuppressive treatment with intravenous immunoglobulin (IVIG) and methotrexate for his rheumatological condition, and after one month, in addition to improving the clinical manifestations of his disease, the hearing loss also improved.[30] This case demonstrated the effectiveness of IVIG and methotrexate in the management of hearing loss associated with SSc; however, more studies are required to characterize the audiovestibular symptoms and the most optimal management when the diagnosis is confirmed. A brief review of the results of some studies conducted in patients with SSc and audiovestibular manifestations is found in Table 3.

Table 3: Audiovestibular manifestations in patients with scleroderma (SSc).

Author/year	Country	#Individuals	Conductive HL	Sensorineural HL	Mixed HL	Tinnitus	Vertigo
Kastanioudakis, 2001[27]	Greece	79	+	+	+	NE	NE
Amor-Dorado, 2008[41]	Spain	94	+	+	+	+	+
Amor-Dorado, 2008[29]	Spain	116	NE	NE	NE	NE	+
Maciaszczyk, 2010[28]	Poland	46	+	+	+	+	+

Continued.

Author/year	Country	#Individuals	Conductive HL	Sensorineural HL	Mixed HL	Tinnitus	Vertigo
Monteiro, 2011[24]	Brazil	72	-	+	-	+	+
Mendes-Silva, 2018[26]	Brazil	50	+	+	+	+	+
Shenavandeh, 2018[42]	Iran	114	+	+	+	-	NE

CONCLUSION

Hearing loss is one of the most common audiovestibular findings in RA, SS and SLE, most of them presenting loss and/or deterioration of the organ of Corti, decreased number of hair cells, atrophy of the stria vascularis, and degeneration of the neurons of the spiral ganglion. Sensorineural hearing loss is the most frequent type described and may be present in up to 70% of patients. However, there is no standard treatment strategy for this symptom. Further randomized controlled studies and prospective studies are in need to have a better understanding and follow-up of this symptom.

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