The effect of a gluten-free diet on a patient complaining of severe tinnitus

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Abstract

We report a case of a patient complaining of severe high pitched tinnitus, dizziness, joint/muscle pain and gastrointestinal symptoms. A mild high-frequency sensorineural hearing loss of cochlear origin was diagnosed in the ear where tinnitus was present. The patient reported a considerable decrease in tinnitus and other symptoms with a gluten-free diet. Gluten sensitivity may have contributed to the pathogenesis of tinnitus in our patient; further research is needed to determine the exact role of gluten in this condition.

Keywords: hearing loss, quality of life, sensorineural, tinnitus.

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INTRODUCTION

Tinnitus, the perception of a phantom sound in the absence of a corresponding external acoustic stimulus, is a frequent phenomenon that can be bothersome and may have a severe impact on a patient's quality of life. No curative treatments are available. Among the several therapeutic options that have been proposed to alleviate tinnitus symptoms there are dietary supplements and restrictions. Nutritional intervention has been demonstrated to reduce symptoms in patients with metabolic disorders¹⁻³. Vitamin and mineral dietary supplements have been suggested as a possible treatment for tinnitus, but the evidence regarding their efficacy is scarce^{4,5}.

New interest has developed concerning diets without gluten-containing cereals: besides wheat allergy and celiac disease, there are also cases of gluten reactions, defined as gluten sensitivity (GS), in which neither allergic nor autoimmune mechanisms are involved⁶⁻⁸. Recently, GS has been reported in patients affected by Ménière's disease and a gluten free diet (GFD) proved to be effective in eliminating symptoms in one such patient^{9,10}.

No paper has demonstrated the effect of a gluten free diet on tinnitus alone. We illustrate the case of a woman complaining of severe tinnitus in whom a gluten free diet caused a substantial reduction in loudness and distress.

CASE PRESENTATION

A 62-year-old woman presented with a 6-month history of continuous tinnitus in the right ear. The tinnitus was likened to the sound made by a pressure cooker with associated whistling. She had undergone several audiometric tests that demonstrated normal middle ear function and normal hearing in low-middle frequencies but a mild high frequency hearing loss in the right ear. Drug therapy had not been successful. She also suffered from dizziness, drunken sensation, joint/muscle pain and gastrointestinal manifestations such as nausea, constipation, epigastric pain, bloating. She had been diagnosed with gastroesophageal reflux and irritable bowel syndrome.

During the first examination, pure-tone audiometry confirmed normal hearing in the left ear (PTA: 0.5-1-2 kHz: 10 dB HL; 4-8 kHz: < 20 dB HL) and a mild hearing loss in the high frequency range (4-8 kHz) in the right ear (PTA: 11.6 dB HL; 4kHz: 30 dB HL; 6 kHz 30 dB HL; 8kHz: 40 dB HL). Tympanometry showed type A tympanograms; contralateral acoustic reflexes were bilaterally present. Distortion product otoacoustic emissions were bilaterally present but reduced in amplitude in the low-frequency range (< 1 kHz) and above 6 kHz. Auditory brainstem

responses and MRI of the brain with gadolinium excluded retrocochlear impairment and internal acoustic canal pathologies.

Tinnitus related distress was assessed using the Italian version of the Tinnitus Handicap Inventory (THI)¹¹ in which the resulting tinnitus-distress scores range from 0 to 100 (with 0 indicating no tinnitus handicap and 100 the worst level of annovance). According to the THI score. tinnitus is considered: slight (THI 0 to 16), mild (THI 18 to 36), moderate (THI 38 to 56), severe (THI 58 to 76) and catastrophic (THI 78 to 100)12. In this case the THI score was 66. Subjective tinnitus loudness was measured using a visual analog scale with scores from 0 to 10, with 0 corresponding to the "inaudible" end of the scale and 10 to a "very loud" sound, just like a jet taking off. The visual analog scale score for this patient ranged from 6 to 8. In order to determine the tinnitus pitch, a set of narrow-band noises was used as comparison stimuli to assess noise-like tinnitus. The patient was asked which of the comparison sounds was most similar in pitch to the dominant pitch of her sensation that proved to be 6 kHz.

Since a possible hypersensitivity to gluten had been suggested, immuno-allergy tests and serology tests were performed in order to rule out wheat allergy and celiac disease: serum anti-tissue transglutaminase (tTG) resulted absent and IgA deficiency was ruled out (Ig A: 4 RU/mI); a genetic test showed the HLA-DQ2 haplotype (homozygous) while duodenal histopathology revealed intraepithelial lymphocytes (Marsh stage 1). On the basis of these results we suspected GS and proposed a GFD, without wheat, rye, barley, farro, kamut and their derivatives.

At the follow up visit, three months later, the patient reported a considerable reduction of symptoms: the pressure cooker sensation had disappeared while the high pitched sound had decreased and occasionally disappeared. Nausea, dizziness and joint/muscle pain had also disappeared and constipation was less frequent. The THI score was 34, a reduction of about 50%; the visual analog scale showed a loudness of the tinnitus ranging from 2 to 3. The audiometric and immittance tests remained unchanged. The gastroenterologist suggested repeating the endoscopy after reintroducing gluten, but the patient refused since she was afraid that symptoms would worsen after gluten re-challenge. The improvement of the clinical conditions continued to persist, even one year after the beginning of the diet.

DISCUSSION

In the case we have presented, perceived tinnitus loudness and tinnitus-related distress decreased with a GFD. The patient also recovered from other disorders including nausea, constipation, dizziness and joint/muscle pain. The presence of gastrointestinal and systemic disorders and their improvement with the new diet are typical of the clinical picture of GS.

GS is defined as a variety of immunological, morphological and symptomatic manifestations that are precipitated by the ingestion of gluten by individuals in whom celiac disease has been excluded¹³. The symptoms of GS may resemble those associated with celiac disease but with a prevalence of extraintestinal symptoms, such as behavioral changes, bone or joint pain, muscle cramps, leg numbness, 'foggy mind', dermatitis or skin rash, depression, anxiety, headache, weight loss and chronic fatigue. Currently, the diagnosis is made by exclusion since objective biomarkers are not available¹⁴. GS has recently been reported in patients affected by Ménière's disease. Di Berardino et al. found a positive gliadin skin test response in 57% of 58 Ménière's patients9 and a GFD eliminated Ménière's symptoms in a subject with GS¹⁰. GS could be raised as a possible contributor to the genesis of tinnitus in our patient who had the HLA-DQ2 haplotype, negative autoantibody serology (tTG-lgA), increased intraepithelial lymphocytes and whose symptoms improved shortly after beginning the new diet.

Which are the mechanisms that could link GS to tinnitus in this case? One of the most reliable hypotheses concerning the origin of tinnitus regards changes in the central nervous system induced by sensory deprivation linked to cochlear damage, even when it is subtle and hearing loss is not visible in the audiogram¹⁵. Our patient presented a subtle cochlear dysfunction demonstrated by the mild high-frequency sensorineural hearing loss in the right ear, the reduction in amplitude of otoacoustic emissions at the low and high frequencies, and the absence of retrocochlear involvement in auditory evoked potentials. Among various factors which could contribute to a cochlear dysfunction in our patient, an immune reaction cannot be excluded. There is strong evidence that the inner ear may function as an immune organ¹⁶.

GS is associated with prevalent gluten-induced activation of innate immune responses¹⁴. We can hypothesize that the inner ear, like other extraintestinal sites involved in GS, could become the target of an immune mediated response. Likewise, a subclinical sensorineural hearing loss, affecting mainly the high frequencies, has been associated with irritable bowel syndrome¹⁷, a syndrome in which GS has been suggested as a possible pathogenic factor¹⁸.

Tinnitus is a symptom of multifactorial origin. The aim of this report is to suggest that GS could be involved in tinnitus generation in a subgroup of patients with associated gastrointestinal disorders. Identification of serological biomarkers indicating GS and proof as to whether they predict responsiveness to gluten restriction in tinnitus patients would enhance clinical management of this common symptom.

CONCLUSIONS

GS may have contributed to the pathogenesis of tinnitus in our patient, however it is still unclear exactly how gluten could affect this condition. We hope that this report will serve to spread an interest in researching possible correlations between immune hypersensitivity reactions to specific food components and tinnitus, with a view to devising new management strategies.

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