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Seasonal sensorineural hearing loss associated with allergic rhinitis: A case report

To the Editor:

The nasal cavity is anatomically connected to the middle ear by the eustachian tubes, and the middle ear mucosa can be considered an extension of the airways, at least in its inflammatory response.¹ Although mechanisms are not completely clarified, it is well known that inflammation occurring in the nose or paranasal sinuses can result in impairment of the ear function, and that respiratory allergy is a risk factor for otitis media with effusion.² In this sense, conductive hearing loss may complicate the most severe form of rhinosinusitis, mainly because of the eustachian tube involvement.^{3,4} Nevertheless, an effect of allergic rhinitis on the neural system of the ear, although possible in principle, has never been described. We report herein 1 case of documented sensorineural hearing loss provoked by allergic rhinitis in a young adult.

A 33-year-old man, hospitalized for sudden hearing loss, was referred in April 2004 for concomitant rhinoconjunctivitis. The main clinical problem reported on that occasion was the hearing loss with tinnitus. The first episode had occurred in April 1994, when the patient was hospitalized in an ear-nose-throat division because of the abrupt onset of hearing loss. At that time, the ear-nose-throat examination was normal except for a pale nasal mucosa with watery rhinorrhea. Audiometry documented a bilateral sensorineural hearing loss of about 30 dB, but tympanometry was completely normal. Systemic corticosteroids were given for about a week, and the symptoms rapidly remitted. A diagnosis of "sudden idiopathic hearing loss"⁵ was provisionally given. In September 1994, audiometry, tympanometry, and evoked acoustic potential were normal, and the brain computed tomography was negative as well. The same problem invariably relapsed in the subsequent years, always during the springtime, but with variable severity. In May 2000, the patient was hospitalized again because the symptoms (hearing loss and tinnitus) had become almost intolerable. Also on that occasion, no precise diagnosis could be established, and the allergic aspects of the patient were not further investigated. In April 2004, on the occasion of a new episode of troublesome hearing loss, the patient was referred to our unit for consultation. He was apparently well, and the general examination was

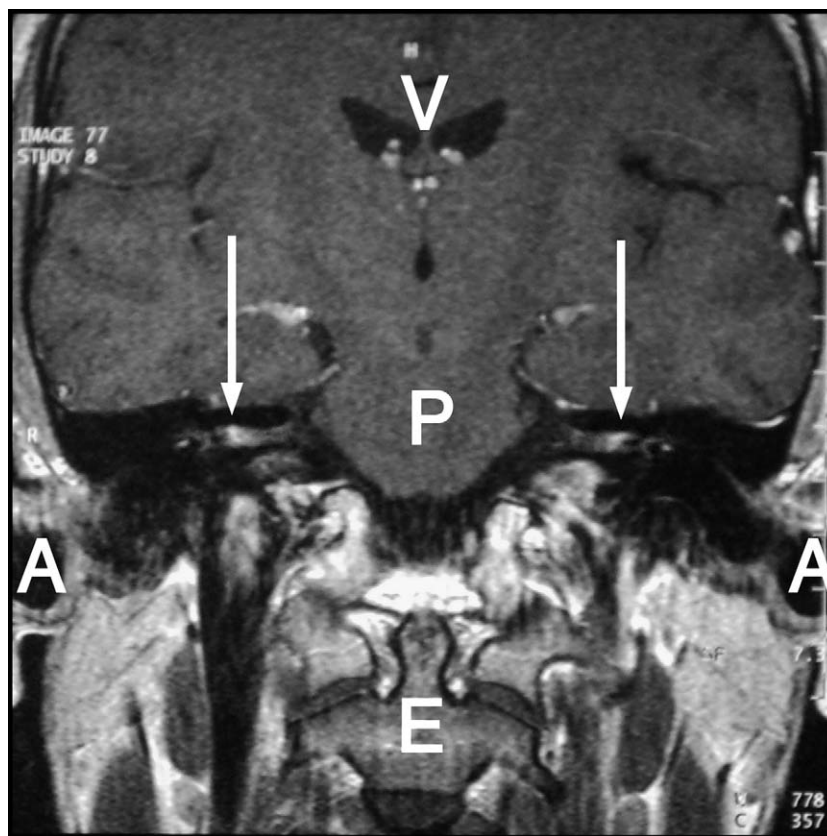


FIG 1. Magnetic resonance imaging of the brain (coronal view). The arrows indicate hyperintense signal of the acoustic nerves within the internal auditory canal. A, Acoustic meatus; E, epistropheus; P, pons; V, ventricula.

negative. The patient reported typical symptoms of allergic rhinitis, and a mild conjunctival redness was apparent. The clinical features were not consistent with common cold. Skin prick tests, performed with a standard panel of commercial extracts (Lofarma SpA, Milan, Italy), revealed positivity for olive (5.0 mm × 7.0 mm) and grass pollens (13 mm × 12 mm). The CAP-RAST assay (Pharmacia, Uppsala, Sweden) provided positive results for *Phleum pratense* (7.31 kU/L) and *Cynodon dactylon* (1.33 kU/L). The family history was positive in both parents for respiratory allergy. Assessments for nonspecific markers of inflammation were negative, as well as the search for autoantibodies. Again, the audiometric test showed a bilateral hearing loss (about 20 dB for low and very high frequencies), with a normal tympanometry and no sign of vestibular involvement. A nuclear magnetic resonance of the brain was then performed because the patient was symptomatic. The imaging after gadolinium administration revealed an inflammatory edema of the acoustic nerves (Fig 1). A short course of systemic corticosteroids (prednisone 25 mg/d for 5 days) led to prompt subjective and audiometric improvement. The patient was seen again in 2004 out of the pollen season. The audiometric test was normal, and the nuclear magnetic resonance revealed no abnormal finding. In March 2005, before the grass pollen season started, the patient was prescribed drug therapy with intranasal mometasone furoate (50 µg per nostril twice daily) and oral levocetirizine (5 mg, 1 tablet daily). During the subsequent season, rhinitis was optimally controlled, and no hearing loss appeared.

To our knowledge, there is no previous description of such a comorbidity of allergic rhinitis. Indeed, Toubi et al⁶ reported a slightly increased prevalence of atopy among subjects diagnosed with sensorineural hearing loss, but in their case series, there was no clear evidence of an association between rhinitis and hearing impairment. Also, there are few animal studies showing that a vestibulocochlear disease can be induced by repeated exposure to antigens,⁷ but the possible relationship between respiratory allergy and sensorineural hearing impairment is far from being elucidated.

The patient described herein was sensitized to grass pollens, and there was a very clear chronologic

relationship, reproducible along years, between the exposure to pollens and the onset of ear symptoms. Skin test was also positive for olive, but in the region around Brescia, Italy, olive trees are not a relevant allergenic source, and symptoms did not match with olive pollen season (end of May to June). The presence of a conductive hearing loss, as reasonably expected, was repeatedly excluded by both audiometric test and tympanometry. Moreover, the magnetic nuclear resonance clearly identified an inflammatory edema of the acoustic nerves. In addition to the seasonal behavior of the disease, there was no evidence of infectious or immunologic causes of hearing loss.⁵ Thus, we conclude for the causal relationship, and hypothesize a very localized allergic inflammation involving the cochlear apparatus.

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