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# Tuberculosis-related middle ear otitis: a rare occurrence

Sir Editor:

On account of the discussions raised during the analysis of the work, Y send to you the letter, whose publication remains at your discretion.

Tuberculosis was a serious public health problem for several decades[8]. After a period in which the disease had apparently been under control by the use of various therapeutic procedures, it is once again a cause for concern, as much because of the increase in resistance to the drugs used as by the increase in the number of immunodeficient patients, especially among those infected with the human immunodeficiency virus (HIV)[7]. Between the mid-1980s and the early 1990s, the combination of a deteriorating public health infrastructure, inadequate instutional control of infection, urban crowding and epidemic of HIV infection resulted in a resurgence of tuberculosis[3 7].

The disease is of an infectious and contagious character, caused by *Mycobacterium tuberculosis*[8],and may affect various organs and tissues. The most common form is the pulmonary one[4], although the pleura, lymph nodes, central nervous system, genitourinary tract, osteoarticular system, skin, serous membranes, intestines, adrenal glands, eyes, larynx and ears may also be affected[7 8]. Diagnosis depends on which part of the body is affected, and the identification of the bacteria in a culture growth (Löwestein-Jensen medium) should be undertaken, especially in HIV-infected patients, who are more resistant to drugs used against *M. tuberculosis* and may also be carriers of other microbacteria[4 8]. Recommended treatment follows Course I (rifampin + isoniazid + pyrazinamide for six months, except for tuberculous meningitis, in which case the treatment should last nine months)[4 7 8]. If rifampin is not used, 12-18 months is the minimal duration of therapy[4 7 8].

In relation to otorhinolaryngocological manifestations of the disease, the middle ear is one of the rarer parts of the body to be affected[5 8]. It is largely disseminated by hematogenical means and may occur by way of the ear canal, by coughing or by regurgitation[1 2 5].

It may be observed in two forms: acute and chronic. In the former situation, the tympanic membrane has multiple perforations which rapidly develop to form only one perforation. Other occurrences may include the inner ear being affected and the destruction of the small-bone chain accompanied by edema and granulation in the middle ear mucous membrane[5]. In the chronic form, one may observe painless otorrhea and otoscopic findings that are disproportionate to early loss of hearing of the conductive type. Otalgia may also be present[6].

Some criteria to be considered in making clinical diagnosis are: unsuccessful treatment using antibiotics that are not meant specifically for tuberculosis, the presence of tissue with abundant granulation, a medical history of pulmonary tuberculosis, either active or cured, significant impairment of conductive hearing, and localized lymphadenitis[6 7]. One should also consider a probable cause as being related to tuberculosis when it involves non-cholesteatomatic cases of the middle ear which evolve with facial paralysis, especially in children[1].

Definite diagnosis is obtained if the histopathological examination of the ear granulomes give positive results, especially if *M. tuberculosis* is cultivated using this material. The presence of the bacillus may also be investigated by examining the auricular secretion using the Ziehl-Neelsen method, which usually gives negative results[2].

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Treatment is based on drugs used for treating tuberculosis, as has been previously presented[4 8]. After-effects are treated after the disease is cured in the same manner as the after-effects of chronic middle ear otitis of any origin. Needless to say, the earlier diagnosis and treatment are done, the better the prognosis of the disease[5].

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