CASE REPORT

Tinnitus and Coxsackie B infections: A case series

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Abstract

Tinnitus is a frequent and often debilitating condition. There is consensus in the scientific community that there exist various forms of tinnitus, which differ in their pathogenesis.

Here we report a series of five cases where the onset of tinnitus was associated with viral infections. In all five patients elevated antibodies against Coxsackie B have been detected. This observation suggests that Coxsackie B Virus infections might be involved in the development of some cases of tinnitus and indicate that further systematic investigations are warranted.

INTRODUCTION

Tinnitus, the perception of a sound in the absence of external acoustic stimulation is a common phenomenon disturbing millions of individuals worldwide [1]. Even if the pathophysiology of tinnitus is not entirely known, there is considerable evidence that altered input from the peripheral auditory system can result in reorganization of central nervous system structures (auditory, but also limbic areas), which may lead to neuronal signals that cause phantom sensations [2]. Very often tinnitus is related to hearing disorders such as presbyacusis, noise trauma or sudden hearing loss. In addition, alterations of peripheral auditory function have also been shown to occur as a symptom of a wide range of viral infections [3–5] with best evidence for varizella zoster [6,7]. Support for a potential relationship between viral infections and tinnitus comes from clinical experience with a frequently reported temporal connection between onset of tinnitus and influenzal infections. Here we report a series of five cases which suggest the involvement of a viral infection with Coxsackie B in the pathogenesis of subjective tinnitus.

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One of the authors had suffered a viral meningoencephalitis with pleocytosis in the cerebrosopinal fluid (CSF) more than 50 years ago. At that time neither serum nor CSF were investigated for specific antibodies. During the course of this meningoencephalitis a unilateral, narrow band high frequency

tinnitus developed, which worsened over the following decades. There was no subjective hearing impairement and audiological exams were normal. Several decades later an elevated titter of IgG antibodies against Coxsackie B was detected.

During his work as a occupational health practitioner one of the authors (H.S.) witnessed the occurrence of tinnitus in four employees over a short period of time. These four employees worked closely together in the same building and all of them reported that tinnitus occurred after they had suffered an influenza with headache, muscle aches and fever. None of the patients complained about hearing loss. Serological screening revealed typical findings of an acute Coxsackie B infection with elevated antibody titters in all four patients. In none of them tinnitus improved during the following weeks, in spite of inpatient treatment in the local ENT department.

DISCUSSION

Here we report the occurrence of tinnitus associated with influenzal infections in four patients which worked closely together and one case of tinnitus occurring after viral meningoencephalitis. In all five patients elevated antibodies against Coxsackie B have been detected. We are aware that a causal relationship between the infection with Coxsackie B and tinnitus cannot be proofed by this observation. Furthermore, the polymerase chain reaction (PCR) technique for the detection of enterovirus gene sequences was not available at that time.

Nevertheless it is highly probable, that all five patients experienced tinnitus as a consequence of a Coxsackie B infection. Recently, sudden deafness has been reported as a symptom of enterovirus meningitis [8]. A systematic study which used PCR for virus detection in 55 consecutive patients with sudden hearing loss, revealed positive diagnosis of enterovirus infection in 22 patients, among them 13 with Coxsackie B. In the majority of these patients sudden hearing loss was associated with tinnitus [9]. In all five cases which we describe here, tinnitus occurred without detectable hearing loss. However, it is conceivable that a viral infection, which can cause hearing loss by infection of the cochlea or the auditory nerve, might also cause an alteration of neuronal functioning of the peripheral auditory system, resulting in tinnitus without hearing loss. In addition, a possible hearing deficit in the high frequency range, not detected by routine audiological methods, cannot be precluded. A potential relationship between Coxsackie B and tinnitus is further supported by the expression of Coxsackie adenovirus receptors in the cochlea [10]. These could represent the structural substrate underlying the effects of Coxsackie B on the auditory system.

Even if at this stage the assumption of a relationship between Coxsackie B Virus infections and the development of tinnitus is largely hypothetical, our findings suggest that further systematic investigations are warranted. The possibility that enterovirus infections may be associated not only with sudden hearing loss, but also with some hitherto "idiopathic" cases of tinnitus, should be recognized. The availability of highly sensitive PCR techniques, together with the recent development of pleconaril, which may be used as a specific therapy for enterovirus infections [11,12], highlights the importance of early diagnosis of enteroviruses as the cause of otherwise idiopathic tinnitus.

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