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BRIEF REPORT

Tinnitus in postherpetic neuralgia

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Abstract We describe a woman who developed postherpetic neuralgia (PHN) located on the skin areas of the left ophthalmic division of the fifth cranial nerve without ocular involvement. PHN was associated with tinnitus, which was located ipsilaterally to the painful side and increased in proportion to the intensity of pain. Tinnitus was responsive to treatment with duloxetine, 60 mg daily, and subsided when the PHN resolved. This is the first description of tinnitus in PHN.

Keywords Allodynia · Herpes zoster · Postherpetic neuralgia · Tinnitus

Introduction

Postherpetic neuralgia (PHN) is the most feared complication of herpes zoster. It presents as a neuropathic pain syndrome that according to the IHS classification criteria, persists or recurs $\geq$ 3 months after the onset of herpes zoster [1].

Subjective tinnitus is the perception of sound or noise without any external stimulation. It is commonly considered as a neural signal arising at some level within the auditory system [2].

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Neuro-otologic symptoms are common in migraine. Vertigo and dizziness are reported by migraineurs, as are phonophobia and low-frequency hearing [3–6]. By contrast, tinnitus is not a common symptom in primary headaches [7]. Moreover, it is a well recognized though uncommon component of the Ramsay-Hunt syndrome [8]. To our knowledge, there are no data in the literature on tinnitus in PHN.

Case report

We describe a 65-year-old woman who was referred to our outpatient unit because of severe head pain and atypical tinnitus. In November 2007, the patient suffered an attack of high fever (39°C) associated with cough, myalgia, anorexia, headache, and gastrointestinal symptoms. She lost 6 kg in 10 days. The nature of this fever and the cause of the symptoms associated with it were not investigated and remained unknown. She had no history of any other significant past medical conditions.

Some days after recovering, she had an episode of herpes zoster that affected the skin areas innervated by the left ophthalmic division of the fifth cranial nerve, but did not involve her eye.

The patient was promptly treated with antiviral therapy (acyclovir 200 mg every 4 h for 7 days) and the skin rash healed within 3 weeks. The pain, however, persisted after the rash had resolved. It manifested itself in a variety of ways, including a deep aching pain, sharp intermittent pain, lancinating pain and allodynia. It remained circumscribed to the skin areas innervated by the left ophthalmic division of the fifth cranial nerve.

Interestingly, this subject developed atypical tinnitus approximately 1 week after the rash resolved. The patient came to our outpatient pain unit in April 2008.

The carotid echo-color-doppler sonography was normal in this subject, as were the cerebral MR and MR angiography. A diagnosis of PHN without ocular involvement was made. The patient had no other auditory symptoms besides the tinnitus. The otolaryngological examination, audiometry and brainstem auditory potentials were all normal.

The patient was given duloxetine 60 mg daily for the PHN. A diary, given to the patient to record both the pain and tinnitus (scores from 0 to 10), confirmed, as the patient had referred, that the intensity of the tinnitus increased in proportion to the intensity of the pain.

Two months later, during a follow-up visit, the patient referred that both the pain and tinnitus had progressively improved. In particular, the tinnitus had totally resolved 16 days after the start of therapy, while the pain persisted as a mild, inconstant sensation over the skin area of the left ophthalmic division of the fifth cranial nerve.

One week before coming to us for the subsequent follow-up visit in September (i.e., 5 months after first coming to our attention), the patient decided to reduce the dosage of duloxetine to 30 mg daily. Shortly after, however, she once again started taking 60 mg daily after both the pain and tinnitus recurred. In November, when still on the same therapy, the patient referred a further improvement in the pain and the total remission of the tinnitus.

The treatment with duloxetine was discontinued in February 2009, after which neither the pain nor tinnitus recurred. In the last visit, in July, both the pain and tinnitus were still absent.

Discussion

The patient we describe had herpes zoster ophthalmicus on the left side, but did not fortunately develop any ocular involvement, possibly because of the prompt administration of antiviral therapy. The patient, however, developed PHN that persisted after the resolution of the rash and consisted of deep aching pain, sharp intermittent pain, lancinating pain, as well as allodynia (pain induced by non-painful stimuli) in the regions innervated by the left ophthalmic division of the fifth cranial nerve.

The pain in our patient was associated with atypical tinnitus. The diary kept by the patient revealed that the pain and tinnitus were indeed related, with the intensity of the tinnitus increasing in proportion to the intensity of pain.

Tinnitus is commonly considered as a neural sign arising at some level within the auditory system [2]. However, some observations point to connections between the somatosensory and auditory systems [9–11].

Since tinnitus developed together with PHN, increased in intensity in proportion to the intensity of the pain, was responsive to PHN treatment and disappeared with PHN recovery, it may be hypothesized that tinnitus was a symptom associated with the neuropathic pain in this patient.

Theoretically, tinnitus could be explained as an overexcitation of hearing structures in the same way as allodynia can be an overexcitation of neural structures in the somatosensory system. This interpretation may be supported by the observations of other authors, who described tinnitus in three migraine patients in whom tinnitus occurred during the course of a migraine attack and increased in intensity in proportion to the severity of pain [12].

Conflict of interest None.

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