Introduction

Sudden sensorineural hearing loss (SSNHL) is a frequently observed condition by the otologist. On the contrary, hearing loss after non-otological surgical procedures under general anesthesia is not a frequent entity. There have been described only a few cases, mainly following cardiopulmonary bypass surgery with extracorporeal circulation or after spinal anesthesia.

We report a case with bilateral sensorineural hearing loss after varicose vein surgery under general anesthesia.

Case report

A 60-year-old male patient with a history of chronic vein insufficiency, mainly in the left lower limb (C2EpAsPr, according to CEAP classification), underwent stripping of the left greater saphenous vein with concomitant stab avulsions of the tributaries under general anesthesia. Past medical history was significant for diabetes mellitus and hypertension. He had also a history of preoperative mild, bilateral hearing loss.

During the operation, anesthesia was maintained with a mixture of nitrous oxide and sevoflurane in oxygen. On awakening the patient developed tinnitus, without vertigo, fullness sensation and here was worsening of deafness in both ears. Given his previous history of hearing loss, this condition was underestimated by the patient who did not refer his disturbs. The day after surgery was discharged from hospital.

On the fifth postoperative day the patient returned to the hospital for the postoperative control, but complained of persistent tin-
ninnitus and impairment of his hearing acuity. He was immediately referred to the otorhinolaryngological department to be examined. The outer ear presented no abnormalities and the tympanic membrane was intact. Weber and Rennie tests were performed, confirming sensorineural hearing loss. An audiogram revealed a profound hearing loss (Figs. 1 and 2). The laboratory exams were within the normal range. The neurological and cardiovascular examinations revealed no pathological findings. His admission to the hospital was mandatory in order to achieve a short but intensive course of oral prednisolone, carbogen and vasodilators. He was treated with 60 mg of prednisolone daily, for 10 days, followed by progressive tapering of dose. Additionally, the therapeutic scheme contained carbogen (5% carbon dioxide and 95% oxygen) and pentoxyfilline. All the audiograms performed during his hospital stay revealed no significant improvement. Over the next 2 years his profound deafness persisted without any improvement.

Discussion

SSNHL is defined as hearing loss of 30 dB or more in at least 3 sequential frequencies, occurring in less than 72 hours. SSNHL reflects an acute dysfunction of the inner ear. In most cases it is unilateral and is accompanied by tinnitus and sometimes by vertigo. The severity is variable and can be affected low, high or even all frequencies. In general population, as etiologic factors of SSNHL there can be recognized infections, autoimmune diseases, traumatic damages, vascular causes, acoustic neuroma, multiple sclerosis or it may be idiopathic.

For patients who have undergone non-otologic surgery carried out under general anesthesia, the recognition of a specific cause may be of extreme difficulty. There are many hypotheses in regard. It was thought to be the result of microembolic phenomena, taking into consideration that the blood supply to the cochlea takes place through an end artery. Consequently, the occlusion of this vessel may cause permanent damage. Another interesting theory is the effect of nitrous oxide during general anesthesia. Nitrous oxide can cause an increment in the middle ear pressure, rupturing the round window. In this way a perilymph fistula is formed and, through this abnormal communication, fluid drains between inner and middle ear. In addition, some anesthetic agents, such as propofol or fentanyl, can cause hemodynamic instability with perioperative hypotension and, therefore, are indirectly implicated in the pathogenesis of hearing loss after surgery. Other etiologic factors include ototoxic drugs (aminoglycosides, diuretics), hypercoagulable states and vasospasm.

In our case there are several factors that should be considered. First of all, the patient has been taking diuretics in order to control his blood pressure. Some diuretics present ototoxic effects and are susceptible to hearing impairment. Furthermore, it should be taken into account the fact that he was diabetic and it could be established a diabetic microangiopathy at the artery of the cochlea. This possible microangiopathy in association with an episode of vasospasm which occurred during the operation could interrupt blood supply to the cochlea, causing a permanent damage. Another hypothesis that can be made is related to the nitrous oxide administration. In our patient nitrous oxide was used for the maintenance of anesthesia and even a small pressure increment in the middle ear could be enough to worsen the preexisting hearing deficit. In the current case there was a delay in
diagnosis and treatment onset. This condition was under-estimated due to the pre-existing history of mild deafness.

In the literature there are conflicting data in regard with the prognosis of SSNHL. Although, some authors have postulated several prognostic factors such as the age of the patient, timing of diagnosis and treatment, initial severity of hearing loss, hearing in the opposite ear, tinnitus, affected range of audiographic frequencies (1), no definitive conclusions can be reached. Byl et al. supported that there could be little or no recovery and in less than 50% of the cases there will be a partial recovery (2). Thus, in our case, the prompt diagnosis and treatment onset would not guarantee the recovery of the auditory function.

In idiopathic cases of SSNHL the treatment consists of oral corticosteroids, which are believed to reduce endolymphatic pressure. Association of other therapeutic agents, include aspirin, warfarin or heparin, which improve emorheological conditions. Furthermore, plasma expanders and vasodilators, can improve microcirculation, while carbogen, can increase the oxygen in the perilymph. All the above, have variable benefit and are still in debate. If a specific cause could be identified, treatment could possibly be effective.

Conclusion

In conclusion, our case of persistent SSNHL following varicose vein surgery is an infrequently event and its etiology remains obscure. Clinical suspicion is required to prompt diagnosis of this rare complication. The treatment is empirical and the prognosis is variable.

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References