Traumatismes sonores aigus: approches thérapeutiques et moyens de prévention

Acute acoustic trauma: how to manage and how to prevent?

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RÉSUMÉ

Introduction: L'exposition à des sons d'intensité élevée résulte en une perte auditive temporaire voire définitive. En dépit d'une prise en charge thérapeutique rapide, des séquelles neurosensorielles de sévérité variable peuvent persister.

Méthodes : Les auteurs rapportent une série rétrospective à propos de 64 patients ayant été pris en charge pour un traumatisme sonore aigu sur une période de 8 ans (2006-2013). Un examen clinique et une audiométrie tonale ont été effectués dans tous les cas. Les seuils auditifs ont été mesurés aux fréquences 500, 1000, 2000 et 3000 Hertz. Des potentiels évoqués auditifs ont été demandés pour 17 patients. Les données thérapeutiques et évolutives ont été détaillées et discutées.

Résultats : Tous les patients étaient de sexe masculin avec un âge moyen de 34 ans. La cause du traumatisme sonore était un tir d'arme à feu dans 48 cas, une explosion ou une détonation dans 5 cas et un bruit intense près d'aéronefs militaires dans 11 cas. La plainte fonctionnelle était une perte auditive aigue associée à des acouphènes. L'audiométrie a permis de retrouver une surdité de perception avec une perte auditive de 38 décibels (dB) +/- 14 DS. Le traitement a comporté une corticothérapie parentérale associée dans tous les cas à des vasodilatateurs périphériques. Le relais par voie orale de vasodilatateurs a été prescrit pendant 1 à 3 mois. Une oxygénothérapie hyperbare a été administrée pour 25 patients. Le suivi s'est basé sur un interrogatoire à propos des symptômes, un examen clinique et des examens audiométriques. L'évolution a été jugée bonne chez 52 patients (81%) et le seuil auditif moyen à la fin du traitement était de 24 dB +/- 12 dB. Des acouphènes ont persisté dans 36 cas malgré un traitement prolongé par des médicaments vaso-actifs.

Conclusion : La réduction de la fréquence et des séquelles neurosensorielles liées aux traumatismes sonores aigus passe par un meilleur contrôle des sources sonores, l'éducation sanitaire des populations exposées et le développement des moyens de protection contre le bruit.

Mots-clés

Traumatisme sonore, hypoacousie, acouphènes, audiométrie, corticostéroïdes, oxygénothérapie hyperbare.

SUMMARY

Background The consequence of an exposure to intense sounds can be a temporary or permanent hearing loss and even with a rapid therapeutic management, severe sensorineural sequelae may persist.

Methods: the authors report a retrospective study about 64 patients followed for an acute acoustic trauma during a period of 8 years (2006 to 2013). For all the cases, a clinical examination associated to a pure-tone audiometry was conducted. Hearing levels were measured at the frequencies 500, 1000, 2000 and 3000 Hertz. Auditory evoked potentials were performed in 17 cases. The therapeutic and evolutive data were detailed and discussed.

Results: All our patients were male with a mean age of 34 years. The cause of acoustic trauma was a firing of a gun near the ear in 48 cases, an explosion near the ear in 5 cases and a sudden exposure to loud noises near military planes in 11 cases. Clinical complaints were acute hearing loss with tinnitus. Audiometric exams found a sensorineural hearing loss with a hearing level average of 38 decibels (dB) +/- 14 SD. The therapy consisted of systemic cortisteroids associated in all the cases to peripheral vasodilators. It was given intravenously during 10 days and then orally with vasodilators during one to 3 months. Hyperbaric oxygenotherapy have been administrated for 25 patients. The follow-up consisted of questioning about symptoms, clinical examination and pure-tone audiometry. A good evolution was noted in 52 cases (81%) and the mean of hearing level after therapy was: 24 dB +/- 12 dB. Despite a prolonged therapy with vaso-active drugs, tinnitus persisted in 36 cases.

Conclusion: Controlling noise and its harmful effects through technical devices and safety professionals programs are the best way to reduce the frequency and the sensorineural sequelae due to acute acoustic trauma.

Key-words

Acoustic trauma, hearing loss, tinnitus, audiometry, corticosteroids, hyperbaric oxygenotherapy.

Acute acoustic trauma (AAT) is defined as an acute impairment of hearing occurring a short time after an exposure to an intense sound in both military and civilian work environments (1,2). The most frequent causes in military environment are explosions, blasts, weapons and noise from vehicles ranging from 100 to 140 dB (3,4). In spite of the development of hearing protection devices and the establishment of hearing conservation programs in all military branches, hearing loss rates are still rising (5). The goal of this work is to study the clinical, therapeutical and evolutive profiles of AAT.

METHODS

Our work is a retrospective study about 64 patients treated and followed for an acute acoustic trauma. The period of the study was 8 years: from January 2006 to December 2013. All the patients were armed forces personnel and the acoustic trauma occurred during their professional activities. A clinical examination was conducted for all the cases. Audiometric records were systematically performed and regularly repeated. All patients underwent baseline-hearing thresholds using pure-tone audiometry (PTA) measuring the hearing levels at the frequencies 500, 1000, 2000 and 3000 hertz (Hz) as recommended by the American academy of otolaryngology head and neck surgery (AAO-HNS). Auditory brainstem responses (ABR) were performed in 17 cases to confirm the thresholds. The clinical, audiometric, therapeutic and evolutive data were detailed and discussed.

RESULTS

All our patients were male with a mean age of 34 years. The cause of acoustic trauma was a firing of a gun near the ear in 48 cases, an explosion near the ear in 5 cases and a sudden exposure to loud noises near military planes in 11 cases. All the patients complained of acute hearing loss with tinnitus. Audiometric exams found a sensorineural hearing loss with a hearing level average of 38 decibels (dB) +/- 14 SD. All these armed forces personnel had anterior audiometric data that didn't show any hearing impairment. The therapy consisted of systemic cortisteroids (Hydrocortisone at the dosage of 5mg/kg/d) associated in all the cases to peripheral vasodilators (Pentoxifylline at the dosage of 1200mg/d).

The interval from the onset of first hearing loss symptoms to time of therapy varied from 12 hours to 7 days (mean: 36 hours).

The treatment was given intravenously during 10 days and then orally with vasodilators during one to 3 months.

Hyperbaric oxygenotherapy (HOT) have been administrated for 25 patients, especially those who didn't respond to the treatment or who had severe initial sensorineural hearing loss. They were given one to two sessions per day for 5 to 10 days.

The follow-up began during the recovery by daily questioning about symptoms, clinical examination and pure-tone audiometry every 2 days. Auditory brainstem responses (ABR) were performed in 17 cases to confirm the thresholds during the period of recovery or during the regular controls. Unfortunately, we couldn't perform systematically this objective test for all the patients at the onset of therapy and at the first control.

The evolution was good in 52 patients (81%) with a progressive improvement of the initial hearing levels. The mean of hearing level after therapy was: 24 dB +/- 12 dB tested by pure-tone audiometry at an average period of 45 days after the end of treatment. Tinnitus persisted in 36 cases and the prescription of peripheral vasodilators and vaso-active drugs didn't give notable results for those patients.

DISCUSSION

Acute acoustic trauma (AAT) consists on hearing loss or impairment caused by short exposure to intense impulse sounds of 100-150 dB (4,6,7). The prevalence of AAT is higher in certain groups such as military (7,8).

Auditory injuries caused by an AAT accounted for up to 47% of all wounded in action evacuations from the war in Iraq and Afghanistan and is ranked as the fourth reason leading to medical referral for military personnel returning from deployment (5,9).

In a military environment, AAT is caused by noise from weapons and equipment systems, turbine engines, and high-performance aircraft and helicopters (10).

Actually, it is well known that two different pathophysiological mechanisms are involved: mechanical and metabolic. Mechanical damage occurs through a detachment of the tectorial membrane, disconnection of the interciliary bridges, or even ruptures of the basilar membrane. Metabolic disorders have multiple origins: ionic, ischemic, excito-toxic or finally, through the production of cochlear free radicals and lipid peroxidation products (2,5,10).

Acute hearing loss represents an alarming symptom leading to an urgent medical visit (11). Speech discrimination can be affected and cases of total unilateral deafness have been reported (12,13). The noise, as reported by the patient, is usually unexpected, intense, and of short duration (12).

The auditory impairment is often asymmetrical and unilateral, especially controlateral to the dominant hand due to the head shadow effect (7,14). Hearing loss may be associated with other symptoms such as tinnitus and vertigo (11,14).

According to Teter et al, four different audiometric curves may be found in victims of auditory damage by explosions: Type A with a drop at 1 kHz; Type B with a slope in 1 and 4 kHz; Type C with a drop at 4 kHz and

Type D with a drop at 2 kHz (15). Some authors reported a normal hearing threshold in conversational range with an increase observed with higher frequencies after an AAT (14,16). Performing hearing tests only in the range of the causative sound may lead to overlooking an acoustic trauma. Therefore, a high frequency hearing test must be performed regardless the frequency level of the noise (14).

In Abaamrane's study, the evolution of the auditory brainstem response (ABR) on the one hand and the measuring of distortion product otoacoustic emissions (DPOAE) on the other was different. DPOAEs continued to decrease until the 7th day, whereas the ABR recovered, suggesting a better sensitivity of the DPOAE (9,17). In our series, we performed only pure-tone audiometry and ABR for 17 cases. DPOAE were not available in our hospital.

Whatever the therapeutic option chosen, emergency treatment of the AAT is indicated, within 1 hour of onset according to some authors since therapy can make difference if initiated in this transitional phase (1,11). Many therapeutic options have been studied and evaluated for AAT treatment (11). Research studies focused on reduction of the cellular hyper-reaction, repair of the impaired microcirculation, and removal of the metabolites that occur during the high functions of the outer hair cells (1,18).

Oral steroids, hyperbaric oxygen, or rheologics therapy are recommended as treatment options (11). In France, for example, the recommended treatment consists in placing the patient in silent environment, injection of glucocorticoid (1.5 mg/kg methylprednisolone (MP), IV during 6 days), associated or not with vasodilator substances, hyperbaric oxygenotherapy or normovolemic hemodilution (9). In our series, almost the same protocol has been used. In the Belgian Armed Forces, it is based on high-dose corticosteroids combined with piracetam (19). The efficiency of Glucocorticoids in AAT has been noted in many studies. It may increase the expression of cochlear anti-oxidant enzymes leading to neutralize the reactive oxygen species and the suppression of inflammatory mediator production (9).

Intratympanic administration of Glucocorticoids can avoid from adverse systemic effects and allow a higher drug concentration in the inner ear (20).

The effect of anti-oxidants on AAT has been reported by many authors (5,6,10). N-acetyl-L-cysteine (NAC) and acetyl-L-carnitine (ALCAR) have shown their efficiency in AAT treatment by countering rapidly the oxidative stress reactions (5).

Hyperbaric oxygen therapy (HBOT) is a procedure with a proven efficiency in AAT treatment in many studies (7,19). Noise trauma causes a decrease in the oxygen supply to the organ of Corti and induces hypoxia in the auditory cortex, the hippocampus and the inferior colliculus (10). HBOT increases the arterial partial pressure of oxygen enhancing this way tissue oxygenation especially in hypoxic areas (21). In our study, we noted a significant benefit with the combination of HBOT and medical therapy (19).

It's difficult to predict the functional prognosis after an AAT. The evolution can range from total recovery to no change (16). Some reports have associated prognosis to hearing level at presentation, therapeutic procedure, and delay between the trauma and the start of treatment (22,23). A longer interval between the time of acoustic injury and the start of treatment, and poor high tone average levels at presentation are often associated with a poor outcome (16). Psillas reported that the greater number of patients who reached complete recovery after AAT was when the treatment started within the first hour and the averaged hearing threshold was statistically better ($P < 0.001$) (1). The auditory sequelae of an AAT are most commonly between 3 and 4 kHz and usually involve higher frequencies (12,16).

In spite of the development of hearing protection devices and the establishment of hearing conservation programs in all military and para-military branches, hearing loss rates due to AAT are still rising (3). Many preventive measures have been proposed: identification of noise hazards, engineering controls where applicable, hearing protectors, educational programs, audiometric surveillance, enforcement of safety procedures and the use of personal protective equipment (24). Some authors reported preventive effects of some drugs such as glucocorticoid and magnesium (9,19). Hearing protection by earplugs or earmuffs could reduce highly intense sound to the hearing organs even if earmuffs, less comfortable than earplugs, are considered as the best hearing protection equipment (24).

CONCLUSION

In spite of extensive researches in its pathophysiology, acute acoustic trauma remains a severe and a frequent disease especially in armed forces personnel. Many treatments are of great interest and a combination of therapeutic means could be of stronger efficiency. Hearing loss prevention is mandatory through the generalization of prevention educational programs and the systemically use of hearing protection measures.

Declaration of interest: The authors report no conflict of interest.

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