

An Analysis Of Ear Trauma Occurring Secondary To Improvised Explosive Devices

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Abstract

Objective: This study aims to examine what ear pathologies may occur in law-enforcement officials exposed to the effects of improvised explosive devices in regions where high levels of terrorist activity are expected. The issues faced by ENT and head and neck surgeons in treating such injuries are also examined.

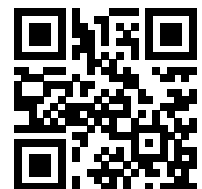
Methods: A retrospective review of examination findings and initial treatment offered was performed on clinical records written by specialists in Emergency Medicine, General Surgery, ENT and Head and Neck Surgery during a particular period (14/3/2016 to 25/7/2016) when a curfew was in operation. The records were taken from Nusaybin State Hospital. Some 260 individuals were included in the review, all of whom had ear problems related to exposure to an explosion. The particulars of the trauma incurred, the presenting complaint, examination findings, and initial surgical and medical treatment offered were determined for each case.

Results: In 224 out of 260 cases, a primary explosion injury was recorded. The most frequently documented symptoms were tinnitus and loss of hearing. On physical

examination, 25 individuals had traumatic perforation of the tympanic membrane. 12 cases featured nystagmus and dizziness and fistula testing was positive in 2 cases, possibly as a result of a perilymph fistula. In case where abrupt hearing loss was apparent on audiological testing, intravenous methylprednisolone was administered at a dosage of 1mg/kg. Cases of multiple trauma were referred onto more specialised clinical units. For cases where hearing loss was mild and the only symptom, the treatment was a reducing dose of oral methylprednisolone over twelve days. Thirty-six individuals were diagnosed with secondary trauma from the explosion. Such trauma included soft tissue and bony injury in the auricular, pre- and post-auricular regions, and was treated by surgery.

Conclusion: Ear trauma secondary to improvised explosive devices is a common injury in law-enforcement officials. In individuals exposed to such hazards, symptoms of hearing problems should be sought, otoscopic and vestibular examination and audiological testing should be carried out at an early stage, and the patient should be referred to an ENT or Head and Neck Surgeon.

Keywords: Hearing impairment, explosives.



Introduction

Blast injury occurs as a direct result of excessive pressure produced by a shock wave. At the moment such a shock wave strikes the body, surface pressure waves and deformation waves are set up. Figure 1 illustrates the Friedlander curves associated with an explosion, with the pressure waves forming a triphasic pattern. The first phase consists of the shock wave, which attains maximal pressure within an extremely brief interval lasting only a few milliseconds. Positive pressure from this phase of the wave can produce organ trauma of a severe type.^[1] The abrupt alteration in pressure associated with the shock wave may perforate the tympanic membrane, cause lung injury, embolise air or rupture the viscera.^[2] The second phase, where negative pressure exists, returns the air to the normal atmospheric pressure. The third phase consists of blast flow, where air is rapidly replaced by an identical volume of gas produced by the explosion. This inrush of explosive gases destroys or dislocates any object that has been damaged by the preceding shockwave.^[1]

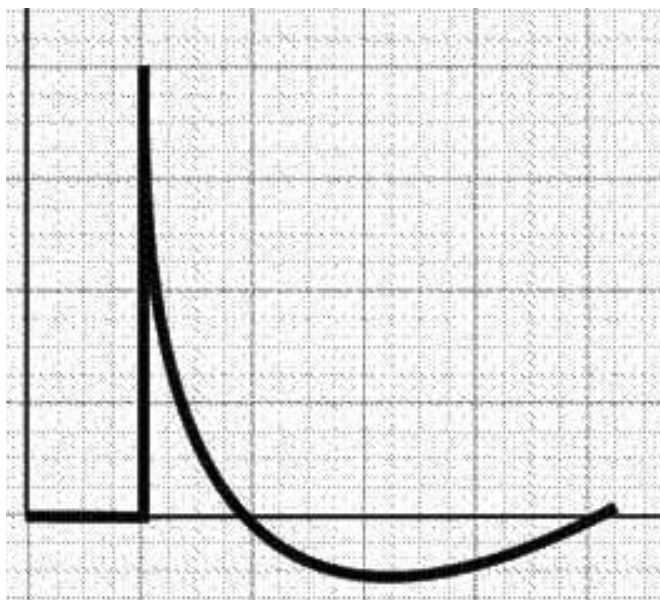


Figure 1. The Friedlander curves representing the pressure waves occurring during explosion

There are four classes of injury produced by explosions: primary, secondary, tertiary and quaternary. Primary injuries are usually the result of the shockwave. Secondary in-

juries are produced by shrapnel or fragments of rock or soil scattered by the blast. Tertiary injuries arise from the blast wave. Quaternary injuries are burns caused by the heat of the blast or chemicals released in the explosion.^[3]

Trauma to the ear is the most common injury seen following an explosion. The pressure waves set up by an explosion may also cause trauma to the inner ear. Such injuries may present as temporary or chronic auditory dysfunction, marked vertigo or ringing in the ears. Where similar problems arise directly from the effect of the pressure wave, the term used is “primary explosion injury”. This type of injury includes perforated tympanic membranes and damage to the ossicles. Secondary explosion injuries, by contrast, involve the whole ear and are the result of flying fragments of shrapnel, rock or gunpowder, etc. Typical secondary explosion injuries are trauma to the pinna, perforation of the ear drum, injury to the ossicles or trauma more generally to the inner ear.^[4,5]

In this research, ear trauma secondary to improvised explosive devices was evaluated. The existing literature on this topic was reviewed and the objective was to add to the knowledge base of doctors who are called on to diagnose such injuries speedily and accurately and to institute appropriate treatment. This will then aid in mitigating the effects of trauma sustained as a result of terrorist actions.

Methods

Ethical approval was obtained from the Research Ethics Committee of Gulhane Military Medical Academy (2016/124). A curfew was operative between 14/3/2016 and 25/7/2016. A retrospective, case-record based analysis was performed on cases of hearing symptoms incurred secondary to high energy explosions during this period of increased activity by the security services. Any individual with pre-existing hearing problems was not included in the retrospective analysis. The cases reviewed had been attended by specialists in Accident and Emergency (A + E), General Surgery, ENT or Head and Neck surgery. The details of trauma, symptoms experienced, and results of physical examination were noted. Auditory testing had been performed with thresholds of 0.25, 0.5, 1, 2, 4, and 8 kHz. The details of any treatments given were carefully noted.

The study methodology was retrospective and covered one centre only. Guidelines for the preservation of patient confidentiality were carefully adhered to. All the individuals

Results

The research involved review of 260 case records of male subjects, who suffered exposure to the effects of improvised explosive devices, having no known previous ear trauma. The age range was 23-50 years, with a mean age of 31.8 years. 36 cases suffered damage to the auricle, in 30 of which the ear was the only site of trauma. In 18 cases, soft tissue was deficient in the superior part of the helix, in 10 cases the fold was lacerated completely between the superior part of the helix and the crus helix, and in 2 cases, the lobule of the pinna was traumatised. Four individuals had injuries extending from the zygoma to the cavum conchae, with laceration of the cartilaginous tragus. There were also two patients whose mastoid cortex had deficient bony mass secondary to injury.

In each of the 260 cases, hearing loss and tinnitus was present. Twelve individuals suffered vertigo. Twenty-five out of 34 cases where otoscopy was performed were found to have a perforated eardrum. Sixteen such cases were perforated on one side only, with the perforation being anteroinferior in 9 individuals and posteroinferior in 7. Nine individuals had kidney-shaped perforations that covered both the posterior and anteroinferior areas and were bilateral. The margins of the perforation were bleeding and the remaining eardrum was congested with blood. There was evidence of perforation edge necrosis in 18 of the eardrums examined otoscopically. Where vertigo was present, secondary nystagmus was noted ipsilateral to auditory loss. Two cases where a perilymph fistula was diagnosed were Bing and fistula positive. Figure 2 illustrates the otoendoscopic appearances of the tympanic membrane when perforated through trauma.

From the total of 260 cases reviewed, 256 (98%) underwent hearing tests, using thresholds of 0.25, 0.5, 1, 2, 4, and 8 kHz. The centre where the research was undertaken was not equipped to perform tympanometry or to test the acoustic reflex. No hearing test was performed in four cases where surgery was performed using a general anaesthetic. Audiometry was performed within two hours of the patients' admission to the clinic. Some 21 of the 25 cases (84%) featuring perforation of the tympanic membrane experienced auditory loss of mixed type and decreasing towards higher frequencies. The mean value for the air-bone gap was 13.2dB.

In 235 individuals, otoscopic examination revealed no abnormality, the eardrum appearing intact. Audiometry showed that, in 48 cases (22%), hearing loss was within

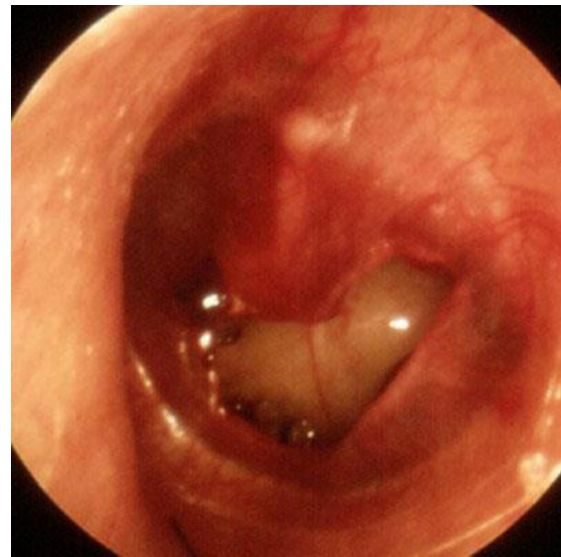


Figure 2. Traumatic tympanic membrane perforation

the normal range. In 187 patients (78%), hearing loss was sensorineural. 62 cases had bilateral auditory deficits affecting high frequencies (descending type, 33%, see figure 3). 111 cases had bilateral auditory deficits affecting low frequencies (ascending type, 59%, see figure 4). 14 cases had bilateral auditory deficits affecting every frequency (flat type, 33%, see figure 5). Where an auditory deficit of sensorineural type with a magnitude of at least 30 dB at three consecutive intervals was demonstrable, a diagnosis of acute hearing loss was given.

1mg/kg methylprednisolone was administered as first line therapy to all the individuals with acute hearing loss. Piracetam was co-administered with dimenhydrinate if vertigo symptoms were present. Those individuals in whom a perilymph fistula was found were given ceftriaxone. The cases were referred on to more specialised units for ongoing assessment and therapy. Oral steroids (methylprednisolone 1mg/kg) were given at a reducing dose over a twelve day period to patients with acute hearing loss as their sole problem. Lesions affecting the pinna and surrounding tissues received prophylactic tetanus injections and were treated with intravenous antibiotics. For 22 individuals, areas of necrosis on the pinna underwent surgical debridement, for which local anaesthesia was given. However, for 14 individuals, general anaesthesia was necessary to allow foreign bodies to be extracted and primary reconstruction begun. These cases all had lesions to the maxillofacial area as a result of polytrauma.

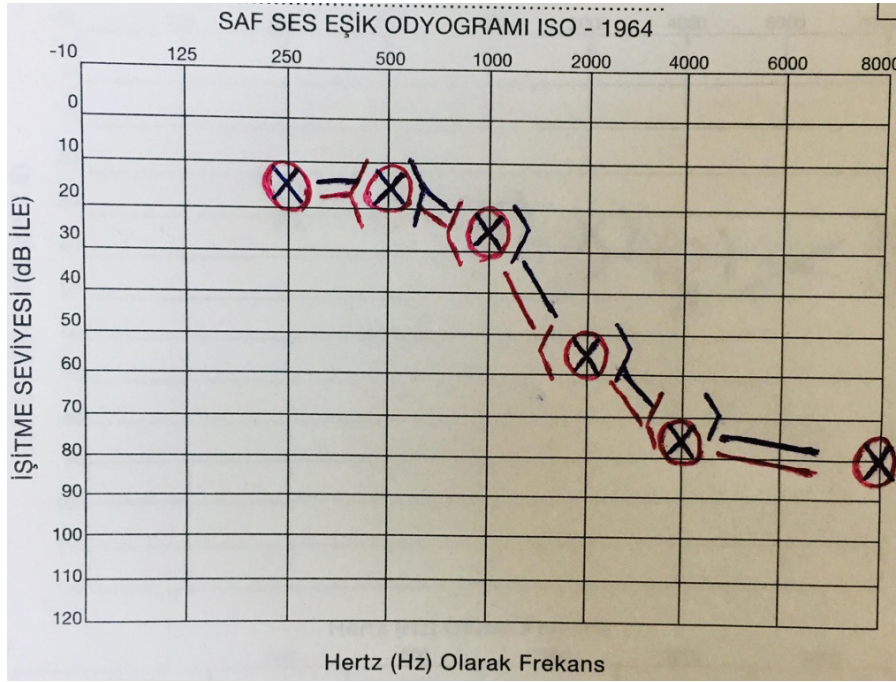


Figure 3. Descending type audiogram

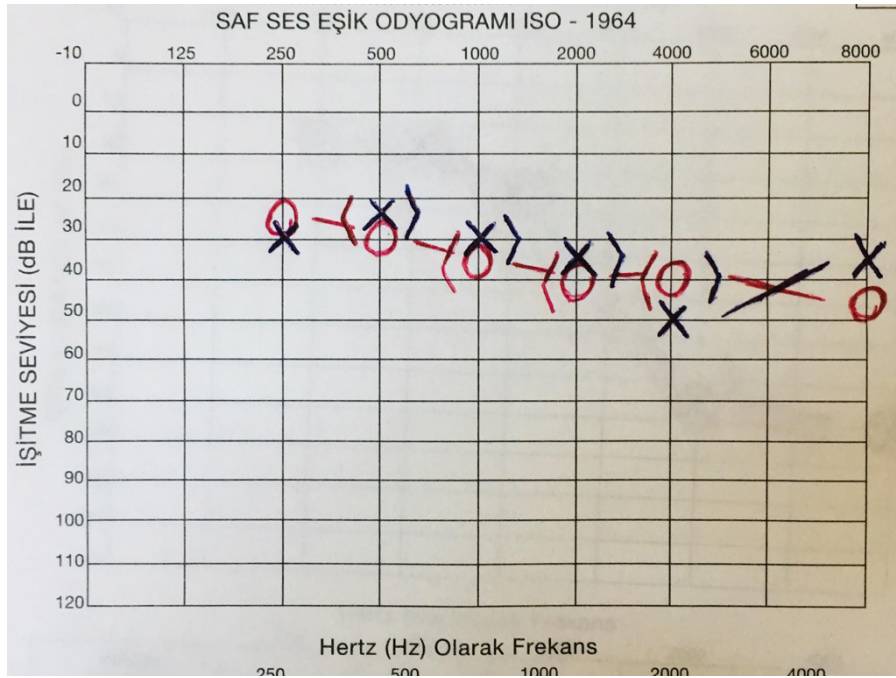


Figure 4. Ascending type audiogram

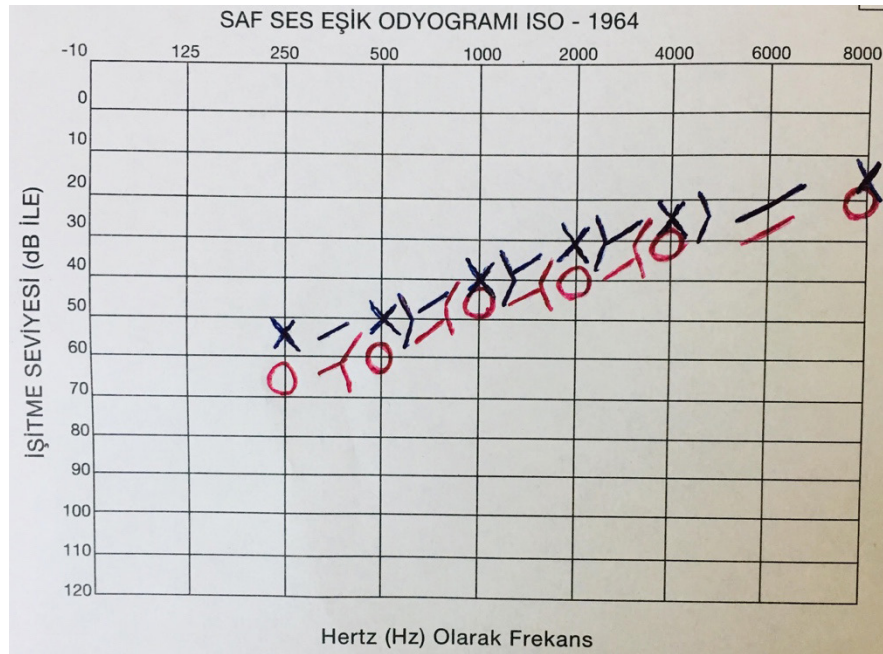


Figure 5. Flat type audiogram

Discussion

Primary blast injuries, alternatively termed barotrauma, arise from the direct action of the very high pressures produced by the shock wave from an explosion. The most common trauma seen in such situations is perforation of the eardrum. In addition to rupture of the tympanic membrane, other types of trauma that are frequently seen include pulmonary injuries, solid viscus injury and intracranial injury. Secondary injuries in explosions occur through flying shrapnel, bomb casing or other debris from the vicinity of the explosion (shards of glass, stone or vegetation). Tertiary blast injuries occur when victims are thrown around by the blast, buried under large items of debris, or struck by heavy items causing blunt trauma. Either the explosion proper or the subsequent blast wind may produce crush injuries, asphyxiation, limb fracture or paralysis, as well as entombment under destroyed structures. Quaternary blast injuries are the result of the intense heat (up to 3000°C) generated by combustion (flare). Flash burns may result. Surrounding structures may also catch fire. Flash burns are most common on the hands and face, as they are less well-protected by clothing, usually. Clothing, however, can also ignite. If the area burnt exceeds 30% of the total surface area, death

typically occurs. Toxic fumes, including carbon monoxide, are generated by explosions, and these may asphyxiate the victim. Breathing in dust, smoke and noxious fumes will, in any case, impair respiratory function.^[6] The degree of trauma is closely associated with the degree of proximity to the blast and the energy release that has occurred. The symptoms of trauma depend on the anatomical location of the trauma. For the tympanic membrane to rupture, it is likely a high-energy explosion was involved.^[7]

Virtually anyone who comes into contact with a major explosion will describe loss of auditory function and vertigo in the immediate aftermath of the event. It is possible for trauma to the inner ear to occur even at a pressure that is insufficient to perforate the eardrum. Such trauma will often resolve within minutes or hours of the explosion. However, perforation of the eardrum is very common even in low energy explosions and differs in nature from the temporary hearing loss of sensorineural type and vertigo described above.^[8] Ritenour et al^[9] studied 436 individuals who had been in contact with an explosion, of whom 16% had a perforated eardrum. Sridhara *et al*^[10] noted that the tympanic membrane was perforated in its

entirety or almost so in 65% of cases, and injury to the ossicles occurred in 18% of cases. Cholesteatoma was seen in 9% of cases. It is thought that cholesteatoma occurred as a result of ectopic epithelium being seeded by the blast.^[11] It is therefore important to follow-up blast victims to see whether the injuries have resolved or cholesteatoma has supervened. The eardrum may recover from perforation without intervention in 38-74% of cases, but this depends on the size of the tear.^[12] In our study cohort, there were 25 cases in which the eardrum was perforated, 16 on one side only, the remaining 9 affecting both sides. The appearance of haemorrhage and blackened necrosis at the margins of the tear may be thought pathognomonic. Since all the cases in our cohort were referred on to more specialised centres afterwards, follow-up that would indicate how long perforation took to heal was not possible.

Traumatic injury to the inner ear may be the sole trauma seen or may occur in conjunction with other types of trauma. Absolute auditory loss, profound dizziness, high pitched tinnitus or paralysis of the muscles of the face may occur. A common sequel to exposure to a blast is a transient alteration in the ability to hear particular sound frequencies, and ringing in the ears. If the individual so affected is isolated in a soundproof room, it is usual for hearing to be restored over the course of a few hours. For a subset of these patients, hearing loss becomes chronic, but vertigo seldom progresses to a chronic sensation.^[13] Research using an animal model for primary explosion injury noted that the brainstem auditory centres and otoacoustic reflexes were impaired by the blast itself.^[14] Jagade *et al* report on the risk of confounding auditory loss secondary to explosive trauma with loss produced by acoustic trauma.^[15] Auditory loss caused by acoustic trauma tends to result in a pattern of sensorineural auditory impairment with the perception of higher pitched sounds being most impaired. For certain individuals, a characteristic notch is discernible at a frequency of 4 kHz.^[16] Shah *et al* examined 110 individuals exposed to an explosion, noting that 24% of those who had an audiogram performed had demonstrable auditory impairment.^[17] According to research conducted by Ritenour *et al*, high pitched auditory loss of sensorineural type was observed in audiometric testing of blast victims.^[9] It is more prognostically favourable, both in terms of treatment and outlook, if acute hearing loss affects only high or low frequencies, whilst auditory deficits at every

level were associated with no treatment response.^[18] Our study had 25 cases (out of 256) where auditory loss was of mixed type. 183 cases were sensorineural type. The cutoff we employed for diagnosing acute hearing loss and hence initiating treatment was a loss of at least 30dB at 3 consecutive pitches. Unfortunately, since all cases involving polytrauma were referred to more specialised units, it was not possible to adequately assess the impact of treatment in such individuals.

Dizziness arising from the primary blast effect is typically seen as due to injury to the cranium. In addition, however, other potential aetiologies require exclusion, such as peripheral vertigo, perilymph fistula, benign positional vertigo, and superior semicircular channel dehiscence occurring in conjunction with acute auditory loss.^[19] Shah *et al* report that tinnitus, auditory loss and vertigo all rose at a significant level in those who had been in contact with a blast.^[17] Vertigo predicts a worsened prognosis in cases of acute auditory loss.^[20] Twelve patients in our cohort reported vertigo in conjunction with acute auditory loss. The physical examination of two individuals led to a diagnosis of perilymph fistula, and they were treated accordingly.

Our research was subject to certain limitations. The findings need to be interpreted in the light of the following methodological handicaps: follow-up was limited; tympanometry and acoustic reflex testing were unavailable; polytrauma cases were only partially assessed; the majority of cases moved to more specialised units; few cases were available to follow-up due to a change in the patients' physical location.

Given the increasing use of improvised explosive devices occurring in the current circumstances in Turkey, we can expect to see increasing numbers of blast victims coming to medical attention. Our hope is that this article will benefit those ENT or Head and Neck specialists whose caseload is likely to include such cases.

Conclusion

Ear trauma secondary to terrorist atrocities has been increasing over the last few years as improvised explosive devices have achieved prominence. Such trauma cases need careful assessment at an early stage and should be referred to an appropriate ENT or Head and Neck surgeon without delay.

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