

Tympanic Membrane Perforation in IED Blasts

Patrick Depenbrock, MD, FS

ABSTRACT

Traumatic tympanic membrane (TM) perforation is a common finding in victims of IED blasts. Frequently it goes undiagnosed by medical providers on initial evaluation. Hearing loss, tinnitus, and vertigo are common complaints from Soldiers who have experienced acoustic trauma. Although symptoms are usually transient, their persistence is a cause for concern. Treatment of a ruptured TM is usually expectant. In certain instances specialty consultation is required. Since primary blast and neurologic injuries can accompany traumatic TM perforation, physicians should maintain a high index of suspicion for their presence. This article aims to address the pathophysiology, diagnosis, treatment, and associated complications of blast-induced tympanic membrane perforation.

Objectives

1. Understand the biophysics of primary blast injury and how to prevent blast-induced acoustic trauma.
2. Understand the common presenting signs and symptoms of tympanic membrane rupture.
3. Understand the treatment of tympanic membrane rupture and the indications for specialty referral.
4. Understand the long-term complications associated with traumatic tympanic membrane rupture.

Improvised explosive devices (IEDs) are responsible for most of the 3,200 American combat deaths and 29,000 wounded in action (WIA) that have occurred since the advent of Operation Iraqi Freedom (OIF).¹ IEDs, which were used sparsely at the outset of OIF in March 2003, now account for nearly 70% of all hostile U.S. casualties in Iraq. Since IED casualties reached a peak in May 2007, Coalition medical providers have been under mounting pressure to familiarize themselves with the immediate and long-term sequelae of blast injuries.² While victims of IED blasts often suffer an array of injuries, rupture of the tympanic membrane (TM) is the most common, yet frequently most overlooked, blast injury. This article aims to address the pathophysiology, diagnosis, treatment, and associated complications of blast-induced tympanic membrane perforation.

IED blasts create dynamic pressure changes at tissue-density interfaces, such as that found at the junction of the auditory canal and the tympanic membrane. When an explosion occurs, high frequency stress waves interact with low frequency shear waves at the eardrum, creating barotrauma that results in perforation of the tympanic membrane.³ This is an example of primary blast injury. Any organ damage occurring as a result of the direct effect of pressure from an explosion is termed primary blast injury. Tympanic membrane rupture, pulmonary tissue damage, and abdominal viscera perfora-

tion are the three most common examples of primary blast injury.³ While much has been made of the ability of body armor to protect military personnel from ballistic projectiles like bullets and blast fragmentation (i.e., secondary blast injury), it does not protect Soldiers from the barotrauma of primary blast injury. Only hearing protection has been shown to significantly reduce the incidence of blast-induced TM perforation, and thus should be stressed as the primary form of prevention.⁴

Following initial stabilization of blast injury victims, a portable otoscope is used to identify the presence of TM rupture. Any debris from the external auditory meatus, which is common in IED blasts, should be removed in order to visualize the TM. The external auditory canal (EAC); however, should not be irrigated, as this may provoke pain and vertigo in the patient. In the acute setting, providers should be aware of the association between traumatic TM rupture and more severe primary blast injuries (e.g., blast lung). Blast-induced TM ruptures have been reported to possess a 50% predictive finding of concomitant lung injury, although this finding has been debated.^{5,6} Due to the concern for concomitant lung injury, Soldiers with ruptured TMs should undergo screening chest radiography and observation for at least eight hours as clinically indicated to monitor for the development of pulmonary complications.³ (**Editor's Note:** This is one approach, but the Israeli data presented as a corre-

spondence in *NEJM* 352:2651-2653 June 2005 based on 30 mass casualty incidents shows that all those who developed pulmonary complications had initial hemoptysis and or tachypnea, there was no “silent pulmonary injury” and thus a chest film is not required if no pulmonary symptoms exist and these patients may be discharged after four to six hours if vital signs remain stable. This is in fact the approach being used at many locations in OIF now. This should be presented as an alternative clinical pathway that can be followed in place of the mandatory chest x-ray and eight hours observation. Perforation of the tympanic membrane is considered a sentinel finding of exposure to blast overpressure. Due to the anatomy of the intervening external auditory canal, the only way to damage the TM is from the blast component of the explosion.) If TMs are found to be intact, serious primary blast injury can be conditionally excluded in the absence of other symptoms such as dyspnea, respiratory distress, and acute abdominal pain.³ This is attributable to the fact that TM rupture occurs at significantly lower blast pressures (as low as five pounds per square inch above atmospheric pressure) compared with the pressure gradient required to induce pulmonary and hollow viscera barotrauma. The estimated threshold for lung injury in man exposed to a single short-duration airblast is 14.9 to 20 psi over ambient pressure. As a result, severe primary blast injury to internal organs rarely occurs in the absence of TM rupture.

Treatment of a ruptured TM is usually expected.³ Ninety percent of perforated TMs resolve spontaneously. Most small perforations will heal within a few weeks. (**Editor’s Note:** There is an inverse relationship between the extent of initial perforation and the probability of its spontaneous closure. Perforations larger than 30% of the total tympanic membrane surface area have a significantly lower rate of spontaneous healing.) While the perforation is still patent, Soldiers should avoid probing or introducing water into the auditory canal, as this can provoke pain and precipitate vertigo. For similar reasons, swimming or immersing the head underwater is strictly prohibited until the TM is fully healed. If contaminated debris is spotted in the auditory canal or behind the TM, antibiotic eardrops should be started as soon as possible to facilitate clearance of the ear canal and prevent infection.³ A seven-day regimen of a topical fluoroquinolone antibiotic is a reasonable choice. Blast survivors with ruptured TMs often go undiagnosed upon initial evaluation. These Soldiers frequently return to duty complaining of hearing loss and tinnitus, which are manifestations of temporary neuropraxia in the receptor organs of the ear.³ Vertigo, the sensation of spinning, should be differentiated from dizziness. Hearing loss due to acoustic trauma is usually transient and is termed temporary threshold shift (TTS). TTS is often accompanied by tinnitus, aural fullness, recruitment (ear pain with loud noises), diffi-

culty localizing sounds, and difficulty hearing in a noisy background. Hearing loss that does not resolve is termed permanent threshold shift (PTS). Hearing loss that persists 72 hours after acoustic trauma warrants audiometric testing, which can be accomplished in theater. Unit surgeons should consider evacuation from theater for Soldiers with a hearing threshold greater than 60 decibels (dB) at three consecutive frequencies. Until hearing loss resolves, Soldiers should be restricted from noise hazardous environments such as firing ranges, airfields, etc.

Sometimes blast-induced tympanic membrane perforation requires specialty evaluation. Absolute indications for referral to an ENT surgeon include vertigo lasting more than three days, presence of clear otorrhea, and presence of discolored otorrhea that persists despite seven days topical antibiotic therapy. Eardrum perforations greater than 50% of the eardrum, debris in the EAC that does not resolve with topical antibiotics, and inability to visualize the TM despite removal of debris from the EAC are relative indications for ENT referral.⁷ An average hearing threshold of greater than 30 dB at frequencies 500, 1000, and 2000 Hertz (Hz) is an absolute indication for audiology referral. Hearing thresholds greater than 35 dB at any of the aforementioned frequencies, hearing thresholds greater than 55 dB at 3000 or 4000 Hz, or new onset asymmetrical hearing loss are also absolute indications for audiology referral. Significant communication problems (regardless of audiometric testing results) and tinnitus significantly affecting quality of life are relative indications for audiology referral.⁷

Concussion is often found to occur in Soldiers with acoustic trauma. While traumatic TM perforation has been found to be a marker of more serious primary blast injury, it may also be a marker for mild traumatic brain injury (mTBI). A study of 541 blast-injury victims at Balad Air Base in Iraq discovered a significant association between TM perforation and loss of consciousness.⁴ In a separate study, researchers found that Soldiers experiencing combat-related loss of consciousness were more likely to suffer from depression and post-traumatic stress disorder (PTSD) on redeployment.⁸ As a result, physicians should maintain a high index of suspicion for concomitant neurologic injury in blast survivors with ruptured eardrums. This can be accomplished with the aid of mTBI and PTSD screening tools such as the Military Acute Concussion Evaluation (MACE) card and the four-question Primary Care PTSD (PC-PTSD) screening instrument, both of which are largely incorporated into the Army’s Post Deployment Health Assessment.^{9,10}

Tympanic membrane perforation is a frequent finding in victims of IED blasts. As enemy forces continue to utilize IEDs as the weapon of choice against

coalition forces in Iraq, medical providers will continue to treat Soldiers with tympanic membrane perforations. While up-armored HMMWVs, body armor, ballistic helmets, and ballistic eyewear offer limited protection against ballistic projectiles, they do not confer protection against the barotrauma of primary blast injury. Hearing protection has been shown to significantly reduce the incidence of traumatic eardrum rupture and should be stressed by unit surgeons and commanders alike. Treatment of traumatic tympanic membrane rupture itself is generally expectant, but providers should be alert to the short-term and long-term complications of acoustic trauma and its associated injuries.

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CPT(P) Patrick Depenbrock, MD, FS is a battalion surgeon that is currently deployed.

Editorials

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Michael S. Xydakis MD, MSc, FS, Lt Col, USAF MC

I congratulate Dr. Depenbrock on his manuscript entitled, “Tympanic Membrane Perforation in IED Blasts” and would offer the following observations.

Exposure to powerful blast waves from the explosion of ordinance is a common and unique feature of combat operations and is characteristic of military casualties. The incidence of blast loading trauma to the body in significant numbers is relatively rare in the civilian trauma world and is primarily associated with industrial accidents and isolated terrorist bombings. The resultant blast front produces a pressure load distribution on the surface of the body in marked excess of normal ambient atmospheric pressure. Barotraumatic (i.e., blast) pathophysiology differs significantly from other forms of trauma and typically results in distinctly patterned injuries.

Direct blast injuries are characterized as a form of mechanical damage to biological targets. Each biological target and organ system has its own threshold of injury. Because of the effects of compressibility, of particular susceptibility to the effects of blast overpressures are air-containing organs (e.g., ear, lung) and organs surrounded by fluid-filled cavities, such as the central nervous system (CNS).

The most common symptoms following direct blast exposure are audio-vestibular in nature. As the level of overpressure increases, pulmonary injury becomes the dominant effect. The lungs are the most critical major biologic target in terms of immediate pathophysiological effects. The levels at which direct

blast overpressure become a hazard to the human central nervous system are still unknown.

The vulnerability to personnel to direct blast overpressure is highly dependent on proximity to the blast. Blast waves lose overpressure extremely rapidly with increased distance. The severity of these injuries grows with the intensity of the blast. However, range-yield-effects data for detonations contain considerable variability.

In conclusion, my experience as a surgeon at Landstuhl, Germany and at Balad, Iraq has revealed that these injuries may be easily overlooked in a combat environment as more dramatic and life-threatening traumatic impairments are treated. Moreover, few guidelines (e.g., “return-to-battlefield” recommendations) are available to assess levels of functional incapacitation in Soldiers after blast exposure and individual disposition is usually performed. A better predictive scheme is urgently required.

Michael S. Xydakis MD, MSc, FS, Lt Col, USAF MC
Assistant Professor of Surgery
Director - ENT/ Head & Neck Surgery
Department of Surgery - A3017
Uniformed Services University of the Health Sciences
Tel: (301) 295-3155