## **Original Research Article**

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# Blast injury to the ear: management and long term follow up at a tertiary care hospital in a terrorism affected area of North India

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#### **ABSTRACT**

**Background:** A large proportion of blast victims suffers ear injuries; however, these injuries are often overlooked. **Methods:** We assessed 411 blast victims to detect ear injury employing detailed history, otological examination, pure tone audiometry, auditory steady state response and distortion product otoacoustic emissions. TM perforations were managed by thorough cleaning of ear by suctioning, edges approximation and gelfoam splinting in group A and only suctioning in group B. Prednisolone in tapering doses was prescribed for managing hearing loss in all except those in whom it was contraindicated.

**Results:** Out of 411 blast victims, 228 (55.47%) had aural trauma. All cases were males (being serving soldiers) between ages of 21 and 57 years. (Mean 37.77 years, SD 10.38). 285 ears had a perforated TM. Of these, 145 were assigned to group A while 140 were in group B. There was no significant difference in spontaneous healing of perforation between the two groups but at 5 years' follow up group A had significantly higher number of unscarred, healthy TMs than group B (Z score=2.2111, p=0.0271). Mean pure tone average was 51.16 dB (SD 8.79 dB) at presentation and 38.91 dB (SD 7.86 dB) at 5 years. Recovery of SNHL component was significantly better in steroid treated patients.

**Conclusions:** Edges approximation and gelfoam splinting helped in reducing scarring of TM on healing, on long follow up. Steroid treated group showed better recovery of hearing loss.

Keywords: Blast trauma, Tympanic membrane, Hearing loss, Tympanoplasty, Steroids

## INTRODUCTION

Blast is a sudden explosive force generated by bursting of an explosive device like shells, bombs or improvised devices. All the three parts of the ear i.e. external ear, middle ear and inner ear can be damaged by the blast. The blast injuries can be primary, secondary, tertiary or quaternary. Primary injuries result from pressure changes occurring due to the initial blast wave which mainly affects air filled cavities like lungs, middle ear and intestines. In the ear, primary injuries comprise tympanic membrane (TM) perforation mainly. Blast waves cause significant changes on the mechanical properties of TM

due to the damage induced in the circumferential fibers and the stiffening in the radial fibers.<sup>1</sup>

Secondary injuries result due to flying debris, while tertiary injuries can result due to throwing off of the body against some object. Quaternary injuries include burns, crush injuries, and lung damage from inhalation of dust, smoke, or chemicals.

Effects of blast exposure on the ear also include hearing loss (mainly sensori neural), tinnitus, vestibular dysfunction, implantation of epithelium in the middle ear leading to cholesteatoma formation, perilymph fistula,

ossicular chain damage etc. When the pressure is directly delivered through inner ear, it can damage the oval and round window. As a result, traumatic disruption of the oval or round window can cause permanent hearing loss.<sup>2</sup>

Blast injuries are on the rise in all parts of the world due to global unrest and terrorism. So far blast injuries were faced by military personnel in war or anti-terror operations, but, now more and more civil population is getting exposed to these injuries. Hence, all ENT specialists in military as well as civil need to be familiar with causation and management of blast injury to the ear.

Ear trauma in blast victims may go unnoticed initially due to more obvious injuries to other body parts. In a study on military personnel exposed to blast, an estimated 49% of risk for hearing loss could be attributed to the blast-related injury event.<sup>3</sup> Also, some short-term recovery of temporary threshold shifts, over a time period after the blast may occur and the victims may not seek medical advice for remaining hearing loss or may delay it.<sup>4</sup>

To prevent permanent disability, early recognition and management of blast trauma to the ear is important. In the present study, we provide an account of various aspects of causation, management, outcomes and follow up of blast injuries to the ear treated by us at a tertiary care hospital situated in a terrorism affected area of North India.

#### **METHODS**

411 victims of blasts occurring due to militant act in our area during the period from April 2006 through August 2012 presenting consecutively to us were assessed to detect ear injury employing detailed history, ENT examination, otomicroscopy (or otoendoscopy), pure tone audiometry (PTA), distortion product otoacoustic emissions (DPOAE), and auditory steady state response (ASSR).

## Inclusion criteria

All patients admitted to our hospital with history of blast exposure.

## Exclusion criteria

Individuals with history of previous ear disease or surgery were excluded. The pre- blast exposure pure tone hearing thresholds of individuals were obtained from their records of annual/periodic medical examination immediately preceding the blast exposure. If these thresholds were raised, the individual was excluded from the study. Critically injured patients needing ICU admission and non-consenting individuals were also excluded.

#### **PTA**

PTA was performed using Amplaid audiometer (model-A321 Twin channel). Masked pure-tone hearing thresholds were recorded for both ears separately at 500 Hz,1,000 Hz, 2,000 Hz, 3,000 Hz, 4,000 Hz, 6,000 Hz and 8000 Hz. Air Conduction thresholds exceeding 25 dB at any frequency were considered as hearing loss. Average of thresholds at 500, 1000 and 2000 Hz was called Low frequency pure tone average while high frequency pure tone average was obtained by averaging the thresholds at 4000, 6000 and 8000 Hz. 4 tone pure tone averages were calculated by averaging thresholds at 500 Hz, 1 KHz, 2 KHz and 3 KHz. These were used for gauzing predominant type of hearing loss at a quick glance and for the purpose of initial record as well as for follow up of the recovery of hearing.

## **DPOAE**

DPOAEs at 2f1-f2 were recorded with the GSI Audioscreener<sup>+</sup> supplied by Grason-Stadler Inc, Denmark, in a sound proof room. The f1 was given at 65 dB SPL and f2 at 55 dB SPL. Recorded DPOAEs were plotted on Y axis in dB SPL while corresponding f2 frequencies 2, 4, and 8 KHz were plotted on X axis. DPOAEs of 5-15 dB SPL were considered 'normal', 0-5 dB SPL 'present but abnormal' and 0 dB SPL or below were taken as 'absent'.

## **ASSR**

ASSR recordings were performed using the machine EPIC 16.29 (EPIC PLUS S-N-EPC 130-29 from Labat, CE 0051). ASSR module 2.0.0.307 was used. Modulated stimulus was given at 500 Hz, 1, 2, 4 and 8 KHz at varying intensities to determine the threshold of response at different frequencies. The thresholds in dB HL were plotted on Y axis, while frequencies were shown on X axis. These were converted to pure tone thresholds by using appropriate correction factor/regression formulae.

## Management of TM perforations

Patients with TM perforations were randomly assigned to either group A or group B using table of random numbers.

## Intervention in group A

Ear was examined under microscope/otoendoscope using strict aseptic precautions. All blood clots, debris and skin fragments were sucked out from external ear canal and middle ear. Edges of the perforation were brought into neutral position if inverted or everted and gently approximated over a piece of gelfoam kept in middle ear. Another compressed and flattened gelfoam piece was placed for support from lateral aspect of TM.

#### Intervention in group B

Only cleaning and suctioning of debris and skin fragments from external ear canal and middle ear was done. No approximation of edges or gelfoam splinting was attempted.

In both groups the ear was kept absolutely dry. No ear drops were used. Oral antibiotics – amoxicillin 500 mg plus clavulinic acid 125 mg twice daily were prescribed for a week. In case of hypersensitivity to penicillins, oral Ciprofloxacin 500 mg twice daily was prescribed.

If the perforation did not heal by 12 weeks post injury, tympanoplasty was undertaken.

## Management of sensori-neural hearing loss (SNHL) and mixed hearing loss

All patients who had SNHL or mixed hearing loss were given oral steroid (prednisolone)—beginning with 40mg/day and tapered over 15 days. Steroid therapy was not given if there was any contraindication to its use like peptic ulcer, diabetes mellitus, severe infection etc.

## Follow up

All patients were followed up at weekly intervals for one month and thereafter at 6 month, 01 year and 05 years. The follow up included eliciting history for symptoms, clinical examination and otoscopy. The PTA, DPOAE and ASSR were recorded only on day 1, day 30, 01 year and 05 year post injury.

## Ethical aspect

Ethical clearance was provided by our hospital's Institutional Ethics Committee and the study was approved by Scientific Review Committee of the hospital. Informed consent was obtained from all participants.

## Statistical analysis

Statistical analyses were performed using IBM© SPSS© Statistics for Windows, Version 22.

Z test was performed to compare the proportions between groups. Statistically significant difference was defined as p<0.05. Pearson's correlation coefficient was calculated for finding correlation between two variables. X² test was used for analyzing categorical variables. The Wilcoxon Rank Sum test was used to evaluate for differences in hearing thresholds (4 tone pure tone averages) between ears with perforation and those without perforation and also for correlation of initial hearing loss with degree of recovery of hearing. One way ANOVA was used to compare means of independent samples.

#### **RESULTS**

Out of 411 blast victims, 228 (55.47%) had aural trauma in one form or the other (TM congestion/ perforation/ hearing loss etc). 12 were excluded due to the presence of exclusion criteria.

Finally 216 patients were included in the study. Of these 165 (76.4%) had bilateral ear injury while 51 (23.6%) had unilateral trauma. Thus, total number of affected ears was 381.

All cases were males (being serving soldiers) between ages of 21 and 57 years (mean 37.77 years, SD 10.38).

#### Locations of blast

All victims were travelling inside military motorised transport while the blasts occurred on roadside in hilly terrain.

## Types of explosive devices

Out of 411 victims, 271 (65.93%) were exposed to blast of an improvised explosive device (IED) while in 140 (34.06%) the blast was due to factory made devices.

## Distance of victim from blast site

Based on detailed history given by the victims it was found that 84 cases (38.9%) were at an approximate distance of less than 10m from the explosive device, 106 cases (49.1%) were at a distance between 10-15 m and 26 cases (12%) were more than 15 m away from the explosive device. The severity of aural trauma was inversely related to the distance from the blast site – victims closer to the site of blast showing larger perforations of TM and/or greater degree of hearing loss (Pearson's correlation, R=-0.9197 and -0.8796 respectively).

The affected ear in unilateral cases and also the worse affected ear in cases of bilateral injury were facing the blast in 144 (66.7%) cases while it was away from the blast in 55 (25.5%). In 17 (7.9%) both ears were equally affected.

## Types of aural trauma

Out of the 381 affected ears 293 (76.9%) had injury to TM with or without SNHL.

Out of the 293 ears which had TM injury, 8 had only congestion of the TM while remaining 285 had a perforated TM. All the perforations were in the pars tensa, their size varied from pinhole to subtotal. Further, there was variation in the number and shape of the perforation also (Table 1). 88 ears (23.1%) had only SNHL without TM injury. Three patients had ossicular chain disruption, in two at incudostapedial joint and in

one at incudomalleolar joint. There were no cases of perilymph fistula or facial nerve injury. Out of 285 ears

with perforation of TM, 195 had everted edges; while in 53 edges were inverted and neutral in 37 ears.

Table 1: Various types of TM perforations and their rates of spontaneous healing.

Type of TM perforation	Total No (%)	Group A No./healed (%)	Group B No./healed (%)	Z, P value*
Pin hole	2 (0.7)	1/1 (100)	1/1 (100)	NaN, 0
Small ragged	27 (9.2)	11/11 (100)	16/15 (93.75)	0.845, 0.4009
Small clean punched	7 (2.4)	4/4 (100)	3/2 (66.67)	1.2472, 0.2113
Moderate ragged	176 (60.1)	86/82 (95.35)	90/81 (90.00)	1.3562, 0.1738
Moderate punched	6 (2.0)	3/2 (66.67)	3/1 (33.33)	0.8165, 0.4122
Large ragged	17 (5.8)	9/7 (77.78)	8/6 (75.00)	0.1368, 0.8966
Large bean shaped	11 (3.8)	8/4 (50.0)	3/1 (33.33)	0.4944, 0.6241
Subtotal perforation	19 (6.5)	10/0 (0.0)	9/0 (0.0)	NaN, 0
Linear perforation	4 (1.4)	2/2 (100)	2/2 (100)	NaN, 0
Double perforation	8 (2.7)	5/4 (80.00)	3/2 (66.66)	0.4216, 0.6745
Multiple perforations	5 (1.7)	4/4 (100)	1/1 (100)	NaN, 0
Perforation traversed by a thin strand of TM	3 (1.0)	2/1 (50)	1/1(100)	NaN, 0
Total	285 (100)	145/122 (84.13)	140/113 (80.71)	0.7597, 0.4472

TM, Tympanic membrane; No., Number; NaN, Not a number i.e. value is infinity; \* Two tailed Z test comparing proportion of healed perforations in Group A with Group B at 0.05 significance level.

Table 2: Time taken for healing of tympanic membrane perforations (N=285).

Time of observation	Group A (N=145) Number healed (%)	Group B (N=140) Number healed (%)	(Z score, P value)*
Healed in two weeks	2 (1.37)	2 (1.43)	0.0353, 0.9681
Healed in 3 <sup>rd</sup> to 4 <sup>th</sup> week	3 (2.07)	1 (0.71)	0.9719, 0.3320
Healed in 5 <sup>th</sup> to 6 <sup>th</sup> week	36 (24.82)	19 (13.57)	2.4072, 0.0159
Healed in 7 <sup>th</sup> to 8 <sup>th</sup> week	62 (42.76)	68 (48.57)	-0.985, 0.3270
Healed in 8 <sup>th</sup> to 12 <sup>th</sup> week	19 (3.1)	23 (16.43)	-0.7917, 0.4295
Total healed spontaneously	122 (84.13)	113 (80.71)	0.7597, 0.4472
Total did not heal (underwent tympanoplasty)	23 (15.86)	27 (19.28)	0.7597, 0.4472
Healed after tympanoplasty	21 (91.3)**	22 (81.48)**	0.9977, 0.3173

\*Two tailed Z test comparing proportion of healed perforations in Group A with Group B at 0.05 significance level; \*\*As a% of ears which underwent tympanoplasty; Note: The tympanic membrane perforation in the 3 cases of ossicular chain disruption did not heal spontaneously; hence these underwent tympanoplasty with ossiculoplasty, which was successful in all 3 cases.

Table 3: Status of TM perforations at 5 year's follow up.

Otoscopic findings	Group A (N=141) No. (%)	Group B (N=128) No. (%)	Total (N=269)** No. (%)	Z, P value*
Cholesteatoma formation	0 (0)	3 (2.34)	3 (1.11)	-1.8281, 0.06724
Epithelial pearls	3 (2.13)	7 (5.47)	10 (3.72)	-1.4465, 0.1470
Tympanosclerosis involving less than 25% of TM area	11 (7.8)	9 (7.03)	20 (7.43)	-0.2405, 0.8103
Tympanosclerosis involving 25-50% of TM area	4 (2.84)	7 (5.47)	11 (4.08)	-1.0886, 0.2757
Tympanosclerosis involving more Than 50% of TM area	2 (1.42)	3 (2.34)	5 (1.86)	-0.5612, 0.5754
Persisting perforation	2 (1.42)	5 (3.90)	7 (2.60)	-1.28, 0.2005
Intact, healthy TM	119 (84.4)	94 (73.44)	213 (79.18)	2.2111, 0.0271
Total	141 (100)	128 (100)	269 (100)	

\*Two tailed Z test comparing proportion of healed perforations in Group A with Group B at 0.05 significance level. \*\* 04 ears (03 patients ie 01 bilateral & 02 unilateral) in Group A; and 12 ears (07 patients i.e. 05 bilateral & 02 unilateral); making a total of 16 ears (10 patients) were lost to follow up after one year.

Table 4: State of Sensori-neural hearing loss at 5 years since blast.\*

Status	Steroids group No. (%)	No Steroids group No. (%)	Total No. (%)
Complete recovery of SNHL	212 (68.63)	15 (26.31)	227 (61.98)
Partial recovery of SNHL	82 (26.8)	20 (35.09)	102 (28.09)
No appreciable recovery of SNHL	14 (4.58)	22 (38.6)	36 (9.91)
Total	308	57	365

 $X^2$  test. The chi-square statistic is 71.75. The p-value is < 0.001. The result is significant at p <0.05; \*04 ears (03 patients i.e. 01 bilateral & 02 unilateral) in Group A; and 12 ears (07 patients ie 05 bilateral & 02 unilateral); making a total of 16 ears (10 patients) were lost to follow up after one year; No., Number; SNHL, Sensori-neural hearing loss; Complete recovery= 4 tone average 20dB or less; Partial recovery= Improvement in 4 tone average by 10 dB or more but not reaching 20 dB; No recovery= Improvement in 4 tone average by less than 10 dB.

Of these, 145 were assigned to group A while 140 were in group B. In group A, 122 (84.13%) and in group B, 113 (80.71%) perforations healed spontaneously, however, the difference was not significant (Z score 0.7597, p=0.4472). Further, there was no significant difference in the rate of spontaneous healing between the two groups for various size and shape of perforation (Table 1, Figures 1 and 2). However, irrespective of the group, small and moderate perforations and multiple small perforations showed highest healing rate, while none of the subtotal perforations healed spontaneously. Similarly, no significant difference was observed between the two groups in their rate of spontaneous healing except in the 5<sup>th</sup> and 6<sup>th</sup> week where the rate in group A was significantly higher than group B (Z score=2.4072, p=0.0159) (Table 2).



Figure 1: Serial photographs of a spontaneously healing perforation from group B.

(A): At 02 weeks; thin advancing epithelium from the lower part of the perforation can be seen. Posteriorly a large TS patch is visible – probably result of previous ear disease, Hence, excluded from final analysis; (B): At 04 weeks; Advancing epithelium now can be seen from the edges of the perforation from all directions; (C): At 06 weeks; perforation has healed completely.

TM perforation closure after tympanoplasty (with or without ossiculoplasty) also did not show significant difference between the two groups (21 ie 91.3% in group A; 22 i.e. 81.48% in group B; Z score=0.9977, p=0.3173).

Thus, overall spontaneous closure of TM perforations in our study was seen in 235 (82.46%) cases and after successful tympanoplasty the number of healed perforation became 278 (97.54%).



Figure 2: Photograph of a non healing perforation at 08 weeks from group B.

Epithelium from anterior edges has grown towards the promontory instead of growing towards other edges. The epithelium from remaining edges has got duplicated on itself causing thickening of the edges. This perforation is not likely to heal spontaneously unless the edges are freshened again and some scaffolding is provided to prevent growth of epithelium towards promontory.

At 5 years' follow up group A had significantly higher number of unscarred, healthy

TMs than group B (Z score=2.2111, p=0.0271); while higher incidence of cholesteatoma formation, epithelial pearl formation and tympanosclerosis was seen in group B. However, the difference was not statistically significant (Table 3). Thus, edge approximation and gelfoam splinting in group A helped only in better spontaneous healing in  $5^{th}$  and  $6^{th}$  week and achieving higher rate of unscarred healthy TM in this group on long term follow up.

## Hearing loss

In all, 381ears (46.35% of all blast victims and 88.2% of all cases with ear injury) in our study showed hearing loss - mixed hearing loss in 291 ears (76.4%), pure SNHL in

88ears (23.1%) and pure conductive loss in only 2 ears (0.5%).

The PTA curve was right sloping in 262 (68.77%) ears, predominantly high frequency loss type with preserved lower frequencies in 69 (18.11%) and flat in 50 (13.12%). Audiograms were asymmetrical in 311(81.63) ears and symmetric in 70 (18.37) ears. Dip at 4-6 KHz (Acoustic notch) was not seen in any audiogram.

Mean low frequency pure tone average was 47.06 dB HL (SD 9.33 dB) while mean high frequency pure tone average was 83.77 dB (SD 9.15 dB). 4 tone pure tone average was 51.16 dB (SD 8.79 dB). The difference was statistically highly significant (One way ANOVA; f-ratio value=55.382; p<0.001).

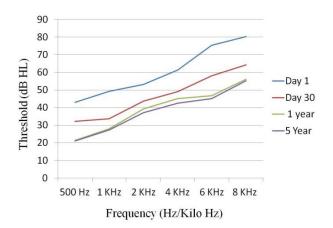


Figure 3: Averaged PTA thresholds of all patients at various points of time during the study.

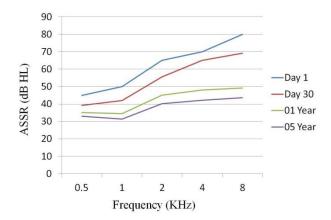


Figure 4: Averaged thresholds on ASSR (converted for pure tones) of all patients at various points of time during the study.

Averaged hearing thresholds of all patients at various frequencies on day 1, day 30, 01 year and 05 years post injury are depicted in Figure 3 for quick understanding of status of hearing loss at various time intervals of the study period. It brought out the predominant damage at high frequencies. ASSR thresholds reflected similar patterns as PTA (Figure 4). DPOAEs were absent across

all frequencies on day 1. On day 30 DPOAEs appeared only at 2 KHz but were 'abnormal'. At 01 year and 05 year DPOAEs were recorded in all frequencies but were still 'abnormal' at 8 KHz (Figure 5).

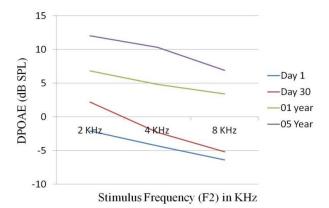


Figure 5: Averaged DP grams of all patients at various points of time during the study.

Earliest symptomatic onset of recovery of hearing was seen within 8 hours of the blast. Majority of the cases showed some recovery within 24-48 hrs. Maximum recovery occurred during the first 06 months which continued up to 01 year. Thereafter, very less recovery of hearing loss was seen.

The degree of recovery correlated with the degree of initial sensorineural hearing loss (Wilcoxon Rank-Sum Test; Zc=1.96, Z=2, R=z:|z|>1.96; p=0.0455), but not with presence or absence of TM perforation (Wilcoxon Rank-Sum test; Zc=1.96; |z| = 0.541 < Zc; p=0.05885).

Recovery of SNHL component was better in patients in whom steroid therapy was initiated within 24 hrs of occurrence of blast as compared to those in whom the steroids were not given. This difference was maintained at 5 years follow up (Table 4). Overall, at 5 years Mean low frequency pure tone average was 27.01 dB HL(SD 7.69 dB) while mean high frequency pure tone average was 45.22 dB (SD 8.12 dB). 4 tone pure tone average was 38.91 dB (SD 7.86 dB).

Use of steroids did not delay the healing of TM perforation. (Wilcoxon Rank-Sum test; |z| = 0.9; Zc=1.96, p=0.3682).

## Associated symptoms

There were associated symptoms of tinnitus in 304 ears, otalgia in 302, bleeding from the ear in 169 and hyperacusis in 26 ears. All these symptoms were self-limiting and did not require active intervention.

## **DISCUSSION**

Due to variation in quantity and quality of explosive material used for explosion and the situation of blast (i.e. open or confined quarters, open air or under water blast), the incidence, pattern and severity of ear injuries is likely to vary greatly. Ianov et al found ear injury in 70-75% of all mine blast injuries.<sup>5</sup> On the other hand, Persaud et al found TM perforation in 62% and hearing loss in all of the 17 Nail Bomb Blast victims.6 Incidence of TM perforations in earlier studies has been reported as 16 -90%.<sup>7-11</sup> In our study, 55.47% of all surviving injured victims had evidence of ear trauma in the form of SNHL alone or with TM perforation. This is quite high and may be explained by higher quantity and high explosive nature of the material used in IEDs used in our area. As mentioned above, majority of blasts occurred due to IED in our cases, rather than standard factory made bombs. Due to variable composition of IEDs, the incidence and severity of ear injuries will also vary greatly.

Greater damage to the ear facing the blast has been reported in some studies and refuted by others. In our study in 66.7% cases the worse affected/only affected ear was facing the blast. There cannot be universal pattern of injury in blast as many factors are involved in its causation like situation of the blast–open air, semi closed/closed space or under water blast. A reflecting surface can lead to more damage to the ear which is actually not facing the blast.

In our cases, the severity of aural trauma was inversely related to the distance from the blast site – victims closer to the site of blast showing larger perforations of TM and /or greater degree of hearing loss, but, Klamkam et al found that the correlation among the characteristic of the incident area, the weight of the explosive materials and the distance from the explosion is not statistically significant for SNHL, but age is- every 1 year increase in age resulted in a greater risk of SNHL of approximately 1.103 times, with statistical significance (p<0.01). 12

Status of torn edges of TM after the blast, whether everted, inverted, or neutral, has also been debated in the past. In our cases, mostly the edges were found to be everted. This is explainable by the fact that the perforation of TM occurs during the positive phase of blast. During the negative phase, which follows the positive phase, the torn edges of TM get sucked outwards and get everted. However, in some cases the edges may get stuck in inverted position or may stop at neutral position. Neutral edges favour spontaneous healing compared to inverted or everted edges.

Ear injury was predominantly bilateral in our cases (76.4%). There is lot of variation in this observation among various studies. For example, in cases of Shah et al, only 8% had bilateral perforations while Ballivet et al reported bilateral perforation to be  $2/3^{rd}$  of their cases. <sup>10,11</sup> As discussed above, the location of the blast may have shielding effect on one of the ear protecting it from injury while reflection of blast wave from a nearby surface may damage opposite ear also.

Similar to our cases, variations in shape and size of the perforations have been observed by previous workers. In the series reported by Wani et al, 92.3% had single while 7.7% had multiple TM perforations while in the cases of Sridhara et al, twenty-two (65%) were total or near-total perforations. <sup>13,14</sup>

Three patients (0.78%) had ossicular chain disruption in our series; while it was seen in 6 (18%) of patients in the study by Sridhara et al. <sup>14</sup> The difference can be attributed to variable intensity of the blast in different series.

Overall spontaneous closure of TM perforations in our study was seen in 235 (82.46%) cases and after successful tympanoplasty the number of healed perforation became 278 (97.54%). High spontaneous healing was also reported by Orji et al and Wani et al (91.1%), and 78% by Persaud et al. <sup>6,13,15</sup> However, it was only 38% in cases of Remenschneider et al, and 55.6% in cases of Cohen et al. <sup>8,16</sup>

At 5 years' follow up group A had significantly higher number of unscarred, healthy TMs than group B. Thus, edge approximation and gelfoam splinting in group A helped in better spontaneous healing in 5<sup>th</sup> and 6<sup>th</sup> week and achieving higher rate of unscarred healthy TM on long term follow up. We are of the opinion that due to high spontaneous healing of TM perforation in these cases initially no difference may be observed in the two groups. However, on long term follow up the healing is likely to be better and with less scarring in the patients undergoing gelfoam splinting and edges approximation at the time of presentation.

Cholesteatoma formation has been regarded as one of the long term complications of blast injuries and the incidence has been reported as 1-5% of all blast cases. In our study, cholesteatoma occurred in 3 patients of group B (1.12%) but in none in group A. However, the difference was not significant. Cholesteatoma was discovered in 3 (9%) patients by Sridhara et al. <sup>14</sup> Lower incidence of cholesteatoma in our cases may be attributed to thorough cleaning of the ear under magnification to remove all skin tags and debris at the time of presentation.

Time of tympanoplasty has been recommended at 12 weeks by most researchers. In our opinion, tympanoplasty should be undertaken without further delay when edges become epithelized because there are poor chances of spontaneous healing if the edges get reepithelialized. This occurred in our cases in 8 to12 th week. The results of tympanoplasty were as good as normal population in our series ie 81 – 91%. In the study by Ballivet et al, tympanoplasty was successful only in 44% cases. <sup>11</sup> The incomplete closures were with large perforations, those with foreign bodies (shrapnel), and in 1 with postoperative water exposure. <sup>14</sup>

#### Hearing loss

In all, 381 ears (46.35% of all blast victims and 88.2% of cases with ear injury) in our study showed hearing loss, while it was 77% in the study by Ritenour et al. The type of hearing loss in our cases was - mixed hearing loss in 291 ears (76.4%), pure SNHL in 88 ears (23.1%) and pure conductive loss in only 2 ears (0.5%). This pattern is at variance with the series of Jalilvand where in sensorineural, mixed and conductive hearing losses were seen in 80%, 18% and 2% cases respectively. More TM perforations in our case increased the proportion of mixed hearing loss in our cases. In the study of Pusz et al, 49% of all hearing loss loss was sensorineural.

The PTA patterns and findings in our cases were in line with other studies. Most cases showed a right sloping curve. Jalilvand reported that the asymmetrical audiogram pattern (in terms of degree, type and pattern of hearing loss) is more common than the symmetrical pattern (70% versus 30% of cases for asymmetrical and symmetrical, respectively). This was the case with our study also. The asymmetry in an audiogram is caused by the effect of head shadow – the near ear is more susceptible than the far ear. However, as mentioned above, the reflecting surfaces may change the outcome.

In our study, Mean low frequency pure tone average was 47.06 dB HL (SD 9.33 dB) while mean high frequency pure tone average was 83.77 dB (SD 9.15 dB). 4 tone pure tone average was 51.16 dB (8.79 dB). In the immediate period (less than 1 month) 10 of the 13 cases (76.9%) had pure tone threshold of more than 70 db in the worse ear in cases of Raju, which is on same lines as in our cases.<sup>9</sup>

We used ASSR and DPOAEs in addition to PTA, for diagnosing and monitoring hearing loss as these are more objective tests. ASSR thresholds reflected similar patterns as PTA in our study. DPOAEs were absent across all frequencies on day 1 indicating hearing loss across all frequencies. On day 30 DPOAEs appeared only at 2 KHz but were 'abnormal'. At 01 year and 05 year DPOAEs were recorded in all frequencies but were still 'abnormal' at 8 KHz, signifying incomplete recovery of high frequencies. Cho et al also used DPOAEs and auditory brainstem response to assess cochlear damage caused by blast in their experiment on mice and found that DPOAE threshold shifts were correlated with blast intensity. <sup>19</sup>

It has been suggested in the past reports that TM perforation may not provide protection against SNHL. For example, there was no statistically significant difference in hearing outcomes between blast-injured patients with tympanic membrane perforations and those without in a study by Shah et al. We also did not find any significant difference between the pure tone averages of victims with TM perforation and without it. (t-value - 0.85404; p=0.402)

Recovery of hearing loss to varying degree does occur after the blast. In our cases it was seen up to 01 year post injury. Thereafter, very less recovery of hearing loss was seen. Incomplete recovery of SNHL has been reported in other studies also. Pusz reported an average SNHL improvement of 51% at follow-up. Chait et al showed 30 dB hearing loss at 4000 and 8000 Hz that was still present 1 year after exposure to explosion. Audiometry 2 years after trauma showed pure tone threshold more than 60 db in 12 of the 13 cases (92.3%) studied by Raju.

It is not clear from our study whether steroids added to the recovery of SNHL over and above spontaneous recovery which occurs after single episode of acoustic trauma. This aspect was not the focus of our study as such; and based on some earlier reports indicating a role of steroids in recovery of SNHL, we prescribed steroids to all patients except to those in whom it was contraindicated. We could not plan a control group for such comparison considering the emergency nature of such cases requiring quick management decision. Hence, despite significantly better recovery of SNHL noted in the steroid treated group, we cannot comment whether or not this difference was due to steroid therapy; and what was the extent of contribution of steroids in the recovery of SNHL.

It is also worth noting that the patients in whom steroids were contraindicated, were likely to show slow healing due to their medical conditions like DM, sepsis, immunecompromised status, other critical injuries etc. Not with standing above, studies do point to the positive role of steroids in recovery of SNHL. In the study of Remenschneider et al, after oral steroid therapy in eight patients, improvement in hearing at 2 and 4 kHz was seen, although changes did not reach statistical significance.<sup>8</sup> In the study by Noam et al, in the 21 participants who received steroid treatment for earlydiagnosed Acute Acoustic Trauma, bone-conduction hearing thresholds significantly improved in the post treatment audiograms, when compared with untreated participants; if initiated within 7 days after noise exposure (p<0.01, for 1-4 kHz).<sup>21</sup>

Patterson and Hamernik have pointed out that there are damage processes which continue after the traumatic event and intervention can reduce some of the damage and hearing loss.<sup>22</sup> Hence, we recommend use of steroid therapy within 24 hours of blast for management of SNHL. Inner ear changes following blast include microfractures in reticular lamina, mixing up of perilymph and endolymph through these fractures and dislocation of organ of Corti. Structural damage to hair cells initially is less, hearing loss being due to functional derangement. However, structural damage and death of hair cells increases tremendously over next 24 hours due to changed composition of inner ear fluids. Injury to hair cells may continue for several weeks after the event if reticular lamina is not repaired, leading to severe

permanent SNHL. However, if repair of reticular lamina is achieved within 24 hours, restoration of function of hair cells occurs and permanent structural damage is prevented. Anti-inflammatory effect and reduction of intracochlear oedema brought about by prednisolone may protect the hair cells from ongoing damage till the reticular lamina gets completely repaired.

Contrary to above, the histological studies by Cho et al one week and three months after the blast demonstrated no disruption or damage to the intra-cochlear membranes. However, there was loss of outer hair cells (OHCs) within the basal turn of the cochlea and decreased spiral ganglion neurons (SGNs) and afferent nerve synapses which produced auditory dysfunction. Niwa et al, based on study on mice using Laser Induced Shock Wave indicated that threshold elevation of the auditory brainstem response after blast exposure was primarily caused by outer hair cell dysfunction induced by stereociliary bundle disruption. 23

Use of steroids did not delay the healing of TM perforation in our study. It appears that short term use of prednisolone does not adversely affect wound healing.

#### Associated symptoms

Associated symptoms similar to our cases have been reported by other authors in variable proportions. Ritenour reported otalgia in 15%, aural fullness in 19%, otorrhea in 25%, tinnitus in 50% and vertigo in 8% of their cases. Jalilvand suggested that tinnitus is very common among war veterans (92% of cases) and the relationship between pattern of hearing loss and tinnitus pitch in acoustic or blast trauma is very high. <sup>17</sup>

#### Limitations

This study is based only on the patients who needed hospitalization after exposure to the blast. The patients who were exposed to blast but did not require hospitalization, were not available to us for assessment. Hence, this population may not be true representative of the actual population exposed to blast injury. Our results should be seen in the light of this fact.

The groups for analyzing the effect of steroid therapy on recovery of SNHL were not well matched and the group not receiving steroids was not a true control group.

## **CONCLUSION**

We have provided an account of various aspects of causation and management of blast injuries at a tertiary care hospital in terrorism affected area of North India.

The practice of edges approximation and gelfoam splinting after thorough suctioning of the external and middle ear to remove all debris, foreign material, skin and epithelial tags helped in the following ways:-

- Better rate of spontaneous healing of tympanic membrane in 5<sup>th</sup> & 6<sup>th</sup> week post injury.
- Higher number of healthy, scar free TM on long follow up.
- Lesser incidence of epithelial pearl formation.
- No cholesteatoma formation till 5 yrs follow up.

Steroid treated group showed better recovery of SNHL but exact contribution of steroid cannot be ascertained from this study. Randomized controlled trials are suggested to further elucidate this aspect.

ASSR and DPOAE were seen to reflect the PTA thresholds correctly and can be used to assess the hearing levels more objectively especially in difficult to test cases. To prevent permanent disability, early otological examination of all blast victims is recommended

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#### **REFERENCES**

- 1. Luo H, Jiang S, Nakmali DU, Gan RZ, Lu H. Mechanical Properties of a Human Eardrum at High Strain Rates After Exposure to Blast Waves. J Dynamic Behavior Mater. 2016;2:59–73.
- 2. Wolf SJ, Bebarta VS, Bonnett CJ. Blast injuries. Lancet. 2009;374:405–15.
- 3. Joseph AR, Shaw JL, Clouser MC, MacGregor AJ, Galarneau MR. Impact of blast injury on hearing in a screened male military population. Am J Epidemiol. 2018;187(1):7–15.
- 4. Fausti SA, Wilmington DJ, Gallun FJ, Myers PJ, Henry JA. Auditory and vestibular dysfunction associated with blast-related traumatic brain injury. J Rehab Res Develop. 2009;46(6):797-810.
- 5. Ianov IK, Gofman VR, Glaznikov LA, Maksimova TG. Clinical and statistical patterns of the hearing system lesions caused by explosive mine trauma. Voen Med Zh. 2001;322(9):32-7.
- 6. Persaud R, Hajioff D, Wareing M, Chevretton E. Otological trauma resulting from the Soho Nail Bomb in London, April 1999. Clin Otolaryngol 2003;28(3):203-6.
- 7. Ritenour AE, Wickley A, Ritenour JS. Tympanic Membrane Perforation and Hearing Loss From Blast Overpressure in Operation Enduring Freedom and

- Operation Iraqi Freedom Wounded. J Trauma Injury, Infection, Critical Care. 2008;64:2:174–7.
- 8. Remenschneider AK, Lookabaugh S, Aliphas A, Brodsky JR, Devaiah AK, Dagher W, et al. Otologic Outcomes After Blast Injury: The Boston Marathon Experience. Otol Neurotol. 2014;35(10):1825-34.
- 9. Raju G. Disability evaluation in acoustic blast trauma. Indian J Occup Environ Med. 2015;19(3):138–40.
- 10. Shah A, Ayala M, Capra G, Fox D, Hoffer M. Otologic assessment of blast and nonblast injury in returning Middle East-deployed service members. Laryngoscope. 2014;124(1):272–7.
- 11. Ballivet de Régloix S, Crambert A, Maurin O, Lisan Q, Marty S, Pons Y. Blast injury of the ear by massive explosion: a review of 41 cases. J Royal Army Med Corps. 2017;163:333-8.
- Klamkam P, Jaruchinda P, Nivatwongs S, Muninnobpamasa T, Harnchumpol P, Nirattisai S, Moungthong G. Otologic manifestations from blast injuries among military personnel in Thailand. American J Otolaryngol Head Neck Med Surg. 2013;34:287–91.
- 13. Wani A, Rehman A, Lateef S,Malik R, Ahmed A, Ahmad W, et al. Traumatic tympanic membrane perforation: An overview. Indian J Otol. 2016;22:100-4.
- 14. Sridhara SK, Rivera A, Littlefield P. Tympanoplasty for Blast-Induced Perforations :The Walter Reed Experience. Otolaryngol Head Neck Surg. 2013;148(1):103-7.
- 15. Orji FT, Agu CC. Determinants of spontaneous healing in traumatic perforations of the tympanic membrane. Clin Otolaryngol. 2008;33:420-6.

- 16. Cohen JT, Ziv G, Bloom J, Zikk D, Rapoport Y, Himmelfarb MZ. Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. Isr Med Assoc J. 2002;4(7):559-62.
- 17. Jalilvand H. Effects of blast and acoustic trauma: assessment of hearing status on war veterans. ENT Audiol News. 2014;23:3:61-4.
- 18. Pusz, M D, Robitschek, J. Traumatic Hearing Loss in the Context of Blast-Related Tympanic Membrane Perforation. Military Med. 2017;182(1):1645-8.
- 19. Cho S-I, Gao SS, Xia A, Wang R, Salles FT, et al. Mechanisms of Hearing Loss after Blast Injury to the Ear. PLoS ONE 2013:8(7):e67618.
- 20. Chait, RH, Casler J, Zajtchuk JT. Blast injury to the ear: historical perspective. Ann Otol Rhinol Laryngol Suppl. 1989;140:9 –11.
- 21. Noam Y, Nir F, Manor S, Tal M. Acute Acoustic Trauma among Soldiers during an Intense Combat. J Am Acad Audiol. 2017;28(5):436-43.
- 22. Patterson JH Jr, Hamernik RP. Blast overpressure induced structural and functional changes in the auditory system. Toxicology. 1997;121(1):29-40.
- 23. Niwa K, Mizutari K, Matsui T et al. Pathophysiology of the inner ear after blast injury caused by laser induced shock wave. Sci Rep. 2016;6:31754.

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