Cases of Probable Aero Otitis Interna

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Introduction

MIDDLE ear damage *i.e.* Aero Otitis Media as a result of Otitic Barotrauma is an established clinical entity. However, reports on inner ear damage due to the Barotrauma are scanty. Goodhill (1971) had first shown its occurrance in a diver, where exploration of the middle ear showed a ruptured round window membrane. Subsequently Freeman (1972) reported five such cases in experienced divers, but in each of these cases there had been difficulty in clearing the ears during descent and recourse to a forceful valsalva had led to giddiness and deafness. He termed this condition as "Inner Ear Barotrauma." In 1974 Freeman further reported two similar cases in skilled divers and this time he showed definite tears in the round window membrane, which he sealed with fat taken from the ear lobule and thus improved hearing and relieved giddiness.

In experimental studies, vertigo was produced in susceptible individuals by middle ear over pressure by INGELSTDTET (1974) which was relieved by pressure normalisation.

Seven case reports are presented here to substantiate that damage can occur to the inner ear due to barotrauma. Four cases are from the Indian Air Force, who underwent rapid descent but did not give any history of forced valsalva. One of them also had associated middle ear barotrauma. One case is of a Naval Officer and one case is of a young girl who developed inner ear symptoms on a commercial Indian Airline Flight.

Details of Cases

Case I : G.V.K., a 30 year old transport pilot reported sick after a routine sortie in a Dakota aircraft during October 1970 with complaints of pain, tinnitus, blockage and deafness in the right ear. He was treated conservatively. His pain was relieved as also the blockage of ear but deafness and tinnitus persisted. He continued to fly with these symptoms, even though they were aggravated during flight and pain was experienced in the right ear whenever R/T. communication was loud. Nasal blockage on right side was more persistent.

Scrutiny of documents showed that he was initially examined in 1964 at AF CME, ENT Examination including a pure tone audiogram was normal.

He again reported sick in 1971 and was found to have perceptive deafness in the right ear with a hearing loss of 40 to 70 dB in all the frequencies, progressively increasing in the middle and higher ranges. Hearing recorded was 10 ft. for conversational voice and 8 ft. for whispered voice.

He was referred to AF CME where he was seen by an Otologist. His complaints were deafness and tinnitus in the right ear. He was found to have slight deflected nasal septum to the left. Tuning fork tests showed perceptive deafness – right ear. Hearing was 5 ft. for conversational voice. Pure tone audiogram showed 30 to 65 dB loss in right ear, more in middle and higher ranges. Left ear showed 25 dB loss in 125, 250 and 500 HZ. Caloric tests were normal. Loudness balance test and difference limen tests showed recruitment on the right side but there was no tone decay. Speech audiometry showed P.B. max. 64 percent in the right ear. X-Ray skull Towne's and Perorbital views were normal. WR, Kahn and VDRL were non reactor. Blood sugar was 104 mgm %.

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He was considered to have sustained end organ type of sensorineural hearing loss in the right ear as a result of otitic barotrauma and was temporarily grounded.

He had later on undergone tonsillectomy and subsequent reviews showed no change except that there was definite decrease in the tinnitus with passage of time. He was permitted limited flying with another qualified pilot.

In the review after six months and subsequent reviews audiological findings showed no change except that tinnitus was absent. He has flown 102 hours without any appreciable change in the findings.

Case 2 : G.M.P., a 28 year old airman who was recruited in 1966 with normal hearing, had to fly occasionally in transport aircraft in the course of his service duties from 1968. Each time he flew he had pain in left ear accompanied by giddiness during descent of the aircraft. In 1970, in one such sortie he complained of acute pain in the same ear with severe giddiness during descent. Suddenly he observed a blood stained discharge from the ear with complete relief of pain. There was hearing loss which persisted even after the discharge had ceased with treatment.

In 1973 he was admitted to a service hospital when otological examination showed no abnormality. Blood for WR, Kahn and VDRL were non-reactor. X-Ray skull Towne's and perorbital views were normal. Blood sugar was normal. Caloric tests showed hypoactivity on left side. Pure tone audiogram showed total deafness in the left ear and 20 dB loss in right ear in the speech frequencies. Malingering was excluded. He has since then been under periodic reviews but there has been no change in the otological findings.

The mode of onset was more than suggestive of otitic barotrauma as being the cause of deafness by damage to the inner ear.

Case 3 : H.S.R., a 34 year old pilot was initially examined in December 1962 when ENT examination revealed gross retraction of tympanic membranes. Audiogram revealed 15 to 20 dB loss in each ear in 1500 HZ only.

He was declared unfit temporarily due to DNS (Rt) with Eustachian tube insufficiency (Rt). Submucous resection of Nasal septum was done and he was declared fit.

In January 1967 while flying a Hunter aircraft he felt pain in right ear during descent which was relieved by valsalva but he noticed that he could not hear by the right ear. He had no giddiness. Otoscopic examination on landing revealed intense redness of the drum head but there was no perforation. He was treated with parenteral antibiotics and repeated eustachian catheterization for 5 days without any improvement. Exploration of middle ear was carried out on 21.2.67 and no pathology to account for his deafness was detected. His hearing was 3 ft. for conversational voice right ear ; left ear being normal. Audiogram showed 30 to 85 dB loss in 500 to 8000 HZ in right ear only. Caloric tests were normal, periodic reviews since then have shown no change.

Mode of onset of deafness and absence of any middle ear pathology on exploration indicated sensorineural deafness due to inner ear Otitic Barotrauma.

Case 4 : G.S.R., a 44 year old civil pilot developed symptoms of heaviness in the ears in Feb 75 during descent in a Caravalle aircraft (cabin altitude 3000—4000 ft). He landed safely without autoinflation. He was treated by an Otologist by bilateral myringotomy and insertion of grommets in April 1975, when conservative treatment failed to give him any relief. Grommets were removed after three months.

After one year, in July 1976, he complained of repeated episodes of eustachian insufficiency during the descent of the aircraft. ENT examination revealed evidence of pansinusitis. Review after treatment in Oct 1976 revealed no abnormality. Ascent to 10,000 ft. in a decompression chamber with rapid descent to ground level at a rate of 3000 ft. per minute did not produce any sign or symptoms of tubal insufficiency. He was, however, considered temporarily unfit due to hepatomegaly.

He later on subjected himself to detailed otological investigations at AIIMS, New Delhi in Nov 76 when his hearing tests, impedence and pressure studies and ENG were normal.

In Jan 77 he complained of tubal insufficiency and described giddiness on autoinflation. ENT examination was normal with mild DNS (Lt). Audiogram showed average 15 to 20 dB loss (Bil) in all the frequencies. He was subjected to ascent in a decompression chamber to an altitude of 12,000 ft. and rapid descent to ground level at a rate of 3,000 ft. per minute all the time monitoring with an ENG. He complained of pain in right ear followed by left ear during descent at an altitude of 5 to 6 thousand feet. Pain increased markedly at 4,000 ft. and tubes failed to open by autoinflation. ENG showed 5° nystagmus to right from 7,000 ft. to 4,000 ft. Otoscopic examination revealed indrawn and intensely congested drum heads. He was immediately taken to 8,000 ft. when pressure in the middle ears was equalised by autoinflation. ENG showed disappearance of nystagmus and pain in the ears subsided. He was subsequently brought to ground level very gradually with repeated auto-inflation.

ENT examination after 3 days revealed no abnormality and tubes functioned normally at ground level.

Case 5 : R.P. is a 14 year old girl who reported in Mar 77 with complaints of giddiness, tinnitus and deafness in left ear 3 days after flying in a Caravelle aircraft on a routine Indian Airline flight from place A to B. She initially had pain in the ear which subsided later on, but other symptoms persisted. Examination revealed a small central perforation in the left tympanic membrane. Audiogram revealed 20 to 40 dB perceptive hearing loss in left ear and ENG revealed nystagmus with a velocity of over 5° in left lateral position.

Diagnosis of aero-otitis interna was made and she was treated with bed rest, Calmpose, tetracycline and antihistaminics. Deafness recovered completely in 10 days time with disappearance of giddiness and return of ENG to normal.

This case is considered to be educative in that probably rupture of tympanic membrane had probably prevented severe and permanent damage to inner ear, confirming that middle ear pressure changes if not corrected can lead to severe inner ear damage.

Case 6 : S.G. is a young Naval Officer who reported to us with complaint of severe deafness in the left ear.

Ear examination revealed large dry central perforation in left tympanic membrane. No other ENT abnormality was detected. Audiometry revealed total deafness by left ear. Vestibulometry revealed left labyrinthine paralysis.

The Officer gave past history of three episodes of giddiness accompanied by deafness and ear ache during descent on routine diving during his training period.

Discussion

Middle ear damage in otitic barotrauma is due to a negative pressure created during descent when eustachian tubes fail to equalise pressure of air in the middle ear with that of outer atmosphere. This causes retraction of the tympanic membrane and vasodilatation leading to transudation. This is now well established. Since most of these cases were not seen initially by us no microsurgical procedure could be undertaken except in case No. 3 where exploration on 26th day revealed no middle ear pathology to account for deafness. Exploration in case No. 5 was not considered necessary since the deafness and giddiness were mild and response to treatment was good.

Inner ear damage due to sudden changes in middle ear pressure prior to the onset of sensori-neural deafness, has been reported. It was hypothesised that the sudden increase of middle ear pressure caused an outward pull of the stapes leading to labyrinthine hydrodynamic changes and the consequent inner ear damage. Subsequently, microsurgical interference showed a rupture of the Round Window Membrane. A further study of deafness after violent sneezing and coughing, (where CSF pressure is suddenly increased), showed similar pathology. It was thus postulated that a sudden change of pressure in relation to inner ear by any cause could lead to inner ear damage by a rupture of the Round Window Membrane or Stapes Subluxation resulting in deafness.

The mechanism of giddiness due to an increase in middle ear pressure is not clearly understood but is probably related to the hydrodynamics associated with the membranous labyrinth.

In the cases reported by us it is interesting to observe that it was negative inner ear pressure, which caused sensorineural damage in the inner ear. The mechanism of this damage, however, has not been established and further studies are required.

It is obvious that due to lack of literature on the subject, the treating clinicians have not been aware of this aspect of ear damage due to barotrauma. In order to substantiate this view, there is a case on record where an airman complaining of deafness was considered to be malingering and on reference was seen by us, where inner ear damage was established with permanent deafness resulting probably from recurring otitic barotrauma in each flight when finally in one such sortie he sustained permanent ear damage. This could have been avoided if the case had been seen by otologist earlier.

Otitic barotrauma must not be treated as a common entity clearing up with decongestive nose drops, which in themselves further aggravate the condition by causing a rebound phenomenon unknown to the individual. It is necessary to give immediate relief with one of the nasal decongestants, which have less of a rebound nasal congestion but internal medication with one of the many antihistamine decongestive combinations available should be preferred for future use. The individual should be kept under observation, away from flying or diving when under medication.

Persistent cases of otitic barotrauma should receive the attention of an Otologist aware of this entity since operative procedures with microsurgery can repair the damage to the round window membrane and restore full hearing.

Conclusions

Barotrauma has been known to cause damage to the inner ear and all such cases should be referred to the nearest otology centre where facilities for microsurgery are available so that its mechanism could be studied further with the intention of its avoidance and treatment when necessary. Early action can avoid permanent deafness, as also relieve giddiness.

Personnel connected with flying and diving must receive appropriate education in autoinflation, while avoiding practice of violent valsalva manoeuvre to ventilate the middle ear.

Inner ear barotrauma being a definite clinical entity should be treated as a surgical emergency, if hearing is not to be impaired permanently.

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