Idiopathic Sudden Sensorineural Deafness in a Pilot : A Case Report

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A case of sudden sensorineural deafness in a pilot is presented. Idiopathic sudden sensorineural hearing loss is now a well established clinical entity though of controversial aetiology. A rational therapeutic approach to idiopathic sudden sensorineural hearing loss requires an understanding of the pathophysiology. An update on its aetiopathogenesis and modes of treatment is given.

Keywords : Deafness, virus induced, vascular

Case report

A 41 yrs old healthy pilot noticed sudden deafness and tinnitus right ear on getting up from bed. This progressed rapidly and by the same afternoon he was not able to hear even telephone calls with his right ear. He reported to the Medical Officer and was given antibiotics, 'Cold' tablets and nasal decongestant drops for two days. Since there was no appreciable improvement in his hearing or tinnitus, he was referred to the hospital.

History revealed that the Officer had flown six to eight hours each for 2 days prior to onset of symptoms in a pressurised transport aircraft maintaining a normal flight profile. He had no inflight or immediate post flight ENT complaints. There was no history of otorrhoea, otalgia, vertigo, headache or eye symptoms. There was no history of any preceding upper respiratory infection, exanthemata, ingestion of ototoxic drugs or sudden stress. There was no history suggestive of diabetes mellitus, hypertension or thyroid disorders. Personal history revealed that the officer was a moderately heavy smoker. Past, personal and family history were otherwise non-contributory.

Examination revealed a slightly obese, normotensive individual. General and other systemic examination was within normal limits. ENT examination revealed mild congestion of nose and throat with normal, intact and mobile tympanic membranes. Postnasal space was clear. Tuning fork tests showed severe sensorineural deafness right ear with normal hearing left ear. Free field hearing tests revealed hearing of conversation voice (CV) and forced whisper (FW) in the left ear at 600 cm, while in the right ear only CV was audible at 60 cm. There was no vestibular deficit.

Investigations revealed a normal complete haemogram, coagulogram, blood sugar levels, lipid profile, serum T3/T4 levels and serological tests for syphilis (STS) was non-reactor. Plain radiography of the internal auditory meati, mastoids and paranasal sinuses was within normal limits. Audiogram revealed a 55 dB to 80 dB air conduction (AC) loss in the right ear with air-bone gap of less than 30 dB, the hearing in the left ear being normal. Specialised audiometric tests like short interval sensitivity index (SISI), LDL and tone decay were negative for retrocochlear pathology. Caloric test showed bilateral normal responses.

The patient was treated with oral steroids, vasodilators (Tab Complamina and Tab Vertin), antihistamines and supportive treatment for 10 days. The tinnitus and deafness persisted for 5 days after admission but then the patient showed a dramatic recovery of his symptoms. The patient was discharged after 11 days of hospitalisation, symptom free and with normal hearing.

Discussion

The aetiology and treatment of idiopathic sudden sensorineural hearing loss (Incidence 7.5/100,000 per year[1]) remains an enigma. The maze of clinical data available reveal resigned therapeutic nihilists offering no treatment at one end of the spectrum to over zealous, blunderbuss therapists at the other end.

The increased incidence of idiopathic sudden sensorineural hearing loss in patients of diabetes mellitus, arteriosclerosis, hypertension, hyperlipoproteinaemia, anxiety and allergic diathesis - suggests that there is probably a vascular cause involving blood vessel narrowing, hypercoagubility, sludging of blood and/or thrombosis with subsequent ischaemia of the end

organ[2]. Microvascular studies of the cochlea have identified a high haematocrit value and a slow rate of blood flow within the vessels of the stria vascularis which make the cochlea particularly vulnerable to intravascular coagulation[3].

However, the concept that a viral infection induced endolymphatic labyrinthitis and not vascular embarrassment is the cause of idiopathic sudden sensorineural hearing loss has gained ground over the years. This concept is supported by the following observations :-

(a) Megighian et al[4] found a seasonal influence on sudden deafness and as such draw a link between recurrent infections of upper respiratory tract (subclinical/clinical) and sudden deafness.

(b) Schuknecht et al[5] suggested from the study of their post-mortem material that viral infections are the most likely aetiological factor. Again, Schuknecht et al[6] report that isolated cochlear neuronal atrophy indicates a viral neuropathy. Also, results of serological studies are often positive for viral infections.

(c) The disease occurs commonly in young individuals with no known vascular disease. In this group of patients, a vascular hypothesis would require sudden hearing loss to occur as an isolated vascular event without other systemic manifestations, which seems highly unlikely.

(d) Many, if not most, patients who suffer sudden deafness have no vestibular symptoms or findings[7]. It is noteworthy that in the experimental animal, cochlear involvement is always accompanied by coincident vestibular involvement[8]. This is because the auditory and vestibular labyrinth have a shared blood supply. Thus a vascular hypothesis cannot adequately explain the clinical findings in sudden deafness.

(e) Many clinically observed audiometric patterns (down-sloping, upsloping and mid frequency loss) and associated vestibular deficits in sudden deafness cannot be explained by assigning a site of presumed vascular occlusion e.g., a mid frequency loss without involvement of low frequencies, if explained on a vascular basis would require selective ischaemia of the middle turn but no ischaemia of the distal turn of the cochlea! Also, when recovery occurs, it is always greater at the apex of the cochlea than at the base[7] which is contrary to what a recovery from vascular embarrassment should be normally.

(f) The irreversible results of experimental vascular embarrassment are not consistent with the reversibility of hearing loss in a fair number of clinically observed cases[8].

(g) It is possible that some of these cases of idiopathic sudden sensorineural hearing loss are due to herpes zoster oticus without obvious vesiculation. Hall and Kerr[9], based on this concept, used Acyclovir, a recently introduced antiviral agent, in cases of facial paralysis and sensorineural deafness secondary to herpes zoster oticus and were impressed by the results of administration of this drug.

(h) The nature of the hearing loss and its susceptibility to improvement with steriod therapy lend support to the hypothesis that viral cochleitis is the primary cause of idiopathic sudden sensorineural hearing loss.

The treatment of this entity is far from scientific[10] considering the various causative theories. Various regimes include vasodilators like Nicotinic acid or inhalation of a mixture of 5% carbon dioxide and 95% oxygen, steroids, diuretics, plasma expanders and vitamins. Suffice it to say that steroids have been shown by Wilson et al[11] to have a statistically significant effect on recovery of hearing in patients with moderate hearing loss.

Prognostic indicators in this emergency are:

(a) The younger the patient and the earlier the treatment started, the better it is.

(b) Vertigo indicates a less favourable outcome, especially if associated with a hypoactive caloric response.

(c) The severer the hearing loss, lesser are the chances of a complete recovery.

(d) Ascending and flat audiometric curves carry a much better prognosis than descending curves[10].

(e) A falling curve between 4000 Hz and 8000 Hz is a bad prognostic indicator[10].

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