ORIGINAL ARTICLE

Idiopathic Sudden Sensorineural Hearing Loss. University Hospital Experience

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Summary

Thirty patients with idiopathic sudden sensorineural hearing loss who presented to the University Hospital between January 1985 to January 1992 are presented. The combined regime of bed rest, intravenous dextran 40, vasodilator and steroid therapy produced good improvement in 63.4% of patients. Unfavourable prognostic factors were found to be, hearing loss of more than two weeks duration, vertigo and bilateral hearing loss.

Key words: Idiopathic sudden sensorineural hearing loss, Dextran 40, Steroids, Vasodilators.

Introduction

A useful definition of idiopathic sudden sensorineural hearing loss is a loss that is greater than 30db in three contiguous frequencies and occurs in less than three days with no apparent cause.

The first reported series of idiopathic sudden sensorineural hearing loss was in 1944 by De Kleyn[1]. At present there is no definite conclusion about the best treatment for this condition[2] due to its low incidence and high spontenous recovery rate[3] varying between 20 to 50 percent.

There have been two 'rival' theories as the causation of the idiopathic sudden sensorineural deafness-viral and vascular but none of these theories have been fully substantiated. It has been known for a long time that certain viruses, for example mumps, measles and rubella can cause sensorineural deafness and that the finding of a preceding 'viral' infection in many cases of sudden hearing loss, varies in incidence from 30 to 40 percent[4]. Conversely, the suddenness of onset made the analogy with similar events in the cardiovascular system equally attractive[5]. This opinion favours sudden vasospasm affecting the internal auditory artery with circulatory stasis in the cochlea and labyrinth. The theory of membrane rupture has also been put forward[6].

Methods And Materials

Over the last seven years, from January 1985 to January 1992, a total of 32 patients presented to University Hospital, Kuala Lumpur with sudden sensorineural hearing loss.

A full history of these patients was recorded and a thorough examination of the cardiovascular, central nervous and otorhinolaryngology systems was performed for all these patients.

All patients were immediately hospitalised and pure tone audiograms, tympanograms and acoustic reflexes responses were recorded. The patients were started on intravenous dextran 40 (one pint twice a day for 48 hours duration), Serc or Stugeron (one Tablet three times a day) and prednisolone 60mg daily in tapering doses over three weeks. These patients were also advised bed rest for 48 hours. Patients receiving merislon and methylcobalamine or cynocobalamine treatment were not included in the analysis.

Daily serial audiograms were done up to one week, then on follow up in the second week. Additional investigations included full blood count, erythrocyte sedementation rate, blood sugar, VDRL, immunological study (e.g C_3, C_4, ANF and Rh. factor) and brain stem evoked response. Vertigenous patients had an electronystamography study together with x-ray of the internal auditory meatus. Further evaluation with computerised tomography scan of internal audiotory meatus with or without air meatogram was done in cases where hearing did not improve within one month, or if the hearing loss progressed.

Results

Two out of 32 patients admitted for sudden sensorineural hearing loss were diagnosed as acoustic neuroma. The remaining 30 patients were cases of idiopathic sudden sensorineural hearing loss. Out of these cases 16 patients were male and 14 patients were female, the male to female ratio being 1:1. The age ranged from 21 to 51 years, with a mean of 32.8 years. The racial distribution revealed the ratio between Malays, Chinese and Indians was 1:1:1.

The hearing loss was right sided in 13 patients (43.3%) and left sided in 14 patients (46.7%). There were three cases (10%) of bilateral hearing loss. The hearing loss ranged from 40 db to 120 db in the affected ear within the range of 500 to 2,000 Hz. Only four cases (13.3%) had less then 60 db hearing loss. The duration of hearing loss prior to seeking medical treatment varied between one day and two months.

The pure tone audiogram findings revealed that patients who had low frequency loss formed 16.6%. Two patients (6.6%) had high frequency loss and about 76.7% of patients had a flat audiogram.

The outcome of the treatment revealed that about 63.4% of patients had improvement. Out of this, 46.7% had total improvement and 16.7% had more than 50% improvement. About 33.3% had no improvement and one patient was lost to follow up.

The cases which had no improvement included six cases who manifested mild vertigo and three patients with bilateral hearing loss.

Discussion

Idiopathic sudden sensorineural hearing loss presents as an otological emergency, occuring with an incidence[7] of 7.5 per 100,000 population per year. It occurs suddenly in a perfectly normal person with previously normal hearing. There is no specific aetiological factors causing it.

In view of its unknown aetiology, the treatment is still controversial, ranging from steroids, vasodilators, carbon dioxide inhalants, hyperbaric oxygen, anticoagulants, stellate ganglion block, low molecular weight dextran (rhemacrodex) and bed rest. An exploratory tympanotomy may be undertaken in the event of non-improvement of the hearing loss[8].

Experience for the last seven years showed that a combination of rest in bed, intravenous dextran 40, serc or stugeron and steroid, therapy produced good improvement in 63.4%. This is consistent with the larger retrospective series of sudden hearing loss (225 cases), reported by Byl[9].

The rationale of using the above combined regime is based on the possibility that the condition is vascular in origin. The dextran 40, steroid and vasodilators are used to increase blood perfusion to the cochlear. Low molecular weight dextran increases capillary blood flow in general by hypervolaemic haemodilution and by decreasing factor VIII, decreasing blood viscosity and resulting in an increased tissue blood flow. Hultcrantz *et al* [10] showed an increase in the cochlear blood flow of 75 percent in their experiment on guinea pigs when the animals were injected with dextran 40. Wilson *et al* [11] had showed that steroid therapy had a statistically significant effect on the recovery of hearing in patients with moderate sensorineural hearing loss.

The following are unfavourable prognostic factors noted in this series. The period of the hearing loss prior to initiation of treatment had a bearing on the prognosis. Our result with the above mentioned therapy showed about 63.4% of patients improved. Patients who received treatment within five days of the onset of hearing loss showed the best improvement. Partial response was noted when therapy was initiated about one to two weeks after onset of the loss. One case who came in two months after the onset of hearing loss showed no improvement.

Patients with bilateral hearing loss and vertigo were also noted to have no improvement regardless of the severity of the hearing loss. No doubt, severity of the hearing loss influences the rate of recovery. Most of the cases who had profound sensorineural hearing loss only improved partially as compared to patients with a moderate hearing loss. 63.4% of the patients responded within one week of starting treatment, some even after the third day.

Conclusion

The aetiology and management of idiopathic sudden sensorineural deafness still remains controversial. We believe that an approach combining bed rest, steroids, dextran 40 and vasodilators produced a significant improvement in these patients. There is no doubt that all cases of sudden sensorineural hearing loss need full investigation. Failure to investigate patients will inevitably lead to a missed diagnosis and a lost opportunity for treatment.

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