Effect of Renal Transplantation on Uraemic Deafness

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Summary

In order to investigate the influence of a functioning transplant on uraemic deafness audiometrical studies were performed in 13 patients before and after renal transplantation.

During RDT there was a marked hearing loss for the higher frequencies between 2048 and 8192 Hz. Hearing capacity improved to normal after transplantation, the optimal audiometrical results being obtained 21.4 (8–42) months after surgery. There was a significant increase in hearing capacity especially for the middle and high-tone frequencies.

It is assumed that uraemic toxins, possibly responsible for inner-ear deafness in chronically uraemic patients, may be adequately removed by a functioning transplant.

Introduction

Hearing loss is a common finding in patients with chronic renal failure (Kopsa et al, 1972; Bergstrom et al, 1973). Previous studies showed that uraemic deafness is probably due to a lesion of the hair cells in the cochlea. Furthermore, the data revealed that the uraemic hearing defect is reversible after successful renal transplantation (Mitschke et al, 1973, 1974, 1975).

The present paper is concerned with the long term effect of a functioning graft on the hearing capacity after transplantation.
MATERIAL AND METHODS

The material under investigation consists of 13 renal transplant patients (10 males and three females) aged 17 to 54 (mean age: 38.2 years). Patients with Alport’s syndrome were excluded from the study. The underlying renal disease was chronic nephritis in all patients. None had a previous history of primary otological disease. While on RDT no ototoxic drugs, except gentamicin in three cases, were administered. Mean observation time after surgery was 28.5 (8–46) months. During RDT mean predialysis values for BUN and serum creatinine were 106.0 ± 30.8 mg/100 ml and 14.6 ± 4.4 mg/100 ml respectively. Clinically mild hearing loss existed in 10 out of 13 patients on RDT.

Ninety-two audiometrical studies were performed before and after renal transplantation. The sound threshold test was carried out by means of a Peters audiometer; the hearing loss in decibels was determined for the frequency ranges between 256 Hz and 8192 Hz. The short increment sensitivity index (SISI) was measured with a MAICO-MA-24 research audiometer at the frequency range of 2896 Hz.

RESULTS

In accord with the clinical impression, the audiometrical studies during RDT revealed a marked hearing loss for the higher frequencies between 2048 Hz and 8192 Hz (Figure 1). The uraemic hearing defect before transplantation was due to inner ear deafness as shown by a positive SISI-test in all patients.

Hearing capacity improved to normal after successful renal transplantation, the optimal audiometrical results being obtained 21.4 (8–42) months after surgery. There was a significant increase in hearing capacity especially for the middle- and high-tone frequencies (Table I) between 2048 Hz and 8192 Hz. The SISI-test normalised after transplantation indicating that a lesion of the cochlea was no longer evident. Mean BUN and serum creatinine values at this time were 24.3 ± 9.5 mg/100 ml and 1.5 ± 0.6 mg/100 ml respectively. The final audiometrical studies, 28.8 (8–46) months after surgery, however, revealed a mild but insignificant deterioration in hearing capacity for the higher frequencies despite unchanged transplant function (Table I). Mean BUN and serum creatinine values at the final otological controls were 24.5 ± 10.5 mg/100 ml and 1.7 ± 0.6 mg/100 ml respectively.

DISCUSSION

Since no exogenous causes such as ototoxic drugs, skull trauma, injuries due to noise or poisoning could be found to explain inner ear deafness in our material on RDT, an endogenous cause must be considered. Previous studies (Kopsa et al, 1972) showed that hypertension and disturbed electrolyte metabolism had no
Figure 1. Audiometrical findings in 13 patients before and after renal transplantation
TP = renal transplantation.

pathogenetic significance for uraemic deafness. However, it was assumed that
cumulating uraemic toxins, incompletely removed by chronic dialysis treatment,
may lead to impaired cochlear function. This assumption is supported by the
fact that with normalisation of the blood chemical values uraemic deafness is
reversible after successful renal transplantation.

In this context similar observations have been made in uraemic polyneuropa-thy. Progressive neurological disorders despite vigorous haemodialysis treat-
ment may only be improved by a functioning graft (Bolton et al, 1971).

Moreover, the data suggest that in the later course of renal transplantation
hearing capacity will deteriorate again. Since mean BUN and serum creatinine
values remained unchanged throughout the observation time, uraemia following
progressive transplant failure was not the cause of the high frequency depression.
As a possible explanation for these findings vascular changes of the inner ear due
to steroid induced hyperlipidaemia (Zazgornik & Schmidt, 1974) should be
considered.
TABLE I  Hearing loss in dB in 13 patients before and after renal transplantation.

<table>
<thead>
<tr>
<th>Frequency ranges Hz</th>
<th>before transplantation</th>
<th>after transplantation I</th>
<th>II</th>
</tr>
</thead>
<tbody>
<tr>
<td>256</td>
<td>14.2 ± 9.6</td>
<td>6.2 ± 2.6</td>
<td>6.9 ± 3.2</td>
</tr>
<tr>
<td>512</td>
<td>13.1 ± 9.0</td>
<td>6.5 ± 3.2</td>
<td>6.9 ± 3.2</td>
</tr>
<tr>
<td>1024</td>
<td>11.9 ± 8.6</td>
<td>5.4 ± 3.2</td>
<td>6.5 ± 3.1</td>
</tr>
<tr>
<td>2048</td>
<td>17.4 ± 11.8*</td>
<td>5.8 ± 4.5*</td>
<td>6.2 ± 3.6</td>
</tr>
<tr>
<td>2896</td>
<td>16.5 ± 13.1*</td>
<td>5.0 ± 4.1*</td>
<td>5.8 ± 4.5</td>
</tr>
<tr>
<td>4096</td>
<td>21.2 ± 16.1*</td>
<td>7.7 ± 7.0*</td>
<td>9.3 ± 10.3</td>
</tr>
<tr>
<td>5792</td>
<td>30.8 ± 15.8**</td>
<td>9.6 ± 6.7**</td>
<td>12.3 ± 9.0</td>
</tr>
<tr>
<td>8192</td>
<td>29.3 ± 21.6*</td>
<td>7.7 ± 8.1*</td>
<td>12.3 ± 10.7</td>
</tr>
</tbody>
</table>

I  =  Optimal hearing capacity after surgery (21.4 (8–42) months)
II =  Hearing capacity at the end of observation period (28.8 (8–46) months)
*  =  Paired t-test : p < 0.005
** =  Paired t-test : p < 0.001

References

Bergstrom, L, Jenkins, P, Sando, I and English, G M (1973) Annals of Otolaryngology, Rhinology and Laryngology, 82, 555
Mitschke, H, Schmidt, P, Kopsa, H and Zazgornik, J (1973) Deutsche medizinische Wochenschrift 98, 2445
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Open Discussion

VAN YPERSELE (Louvain)  Do you have any correlation between the changes in audiometry and possible changes in peripheral nerve function in your patients?

SCHMIDT  No, we did not correlate polyneuropathy and deafness. I think there was no correlation because all the patients have the same behaviour concerning audiometrical controls; that means that they all had impaired hearing capacity before surgery and all normalised after transplantation.
PARSONS (London)  Did any of your patients receive, prior to transplantation, ototoxic drugs, and if so did their hearing improve? We have several patients who have had Gentamicin, Kanamycin and similar drugs whose hearing has not really improved following transplantation.

SCHMIDT  As I mentioned we had three patients who had had Gentamicin and these patients also improved. On the other side we had two patients, not mentioned in this paper, with Alport’s syndrome and these two patients unfortunately received Gentamicin. These two patients showed no improvement.

McGEOWN (Belfast)  Have you considered the noise level during regular haemodialysis as a factor in the production of uraemic deafness, as it is well known that industrial processes associated with a high noise level lead to high frequency deafness? The cessation of exposure to noise after transplantation may contribute to the improvement in hearing which you report.

SCHMIDT  Did I understand right? You mean that the noise of the machine has an influence on the hearing capacity?

McGEOWN  Yes!

SCHMIDT  I think that the noise level of our machines was not high enough to explain uraemic deafness.