Hearing status in Egyptian children with nephrotic syndrome

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Objective **Children with nephrotic syndrome (NS) have biochemical disturbances and many children with NS are treated with multiple courses of diuretics, which cause ototoxicity. This study aimed to assess the prevalence of hearing impairment in children with different clinical types of NS.**

Materials and methods **The study was a cross-sectional one conducted at the Nephrology Clinic, Children Hospital, Cairo University, from September 2013 until May 2014, and included 45 NS children and 15 age-matched and sex-matched healthy children as controls. The cases were divided into three groups, each consisting of 15 patients: group 1 steroid-sensitive NS), group 2 (frequently relapsing NS/steroid-dependent NS), and group 3 (steroid-resistant NS). Pure tone audiometry was done for all cases and controls, using the DANPLEX DA65 audiometer.**

Results **Hearing defect was detected in 10 of the 45 patients (22.22%). Six of them (60%) were from group 3. The number of relapses was significantly more in patients with hearing defect.**

Conclusion **Children with NS are more susceptible to sensorineural hearing defect compared with normal children. steroid-resistant NS children are the most affected ones. Hearing evaluation should be included in the routine management of NS. *Med Res J* 15:1–7 © 2016 Medical Research Journal.**

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Keywords: Egyptian children, hearing, nephrotic syndrome

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Introduction

Nephrotic syndrome (NS) is the most common childhood glomerular disease worldwide [1]. About 80% of children with NS show remission of proteinuria following treatment with corticosteroids and are classified as having steroid-sensitive nephrotic syndrome (SSNS). Some patients have multiple relapses [frequently relapsing nephrotic syndrome (FRNS)/steroid-dependent nephrotic syndrome (SDNS)], placing them at risk for steroid toxicity, systemic infection, and other complications such as hypocalcemia, hypertension, stunted growth, and thromboembolic complications. A small proportion of patients who do not respond to steroids are classified as having steroid-resistant nephrotic syndrome (SRNS) and are also at risk for similar complications and renal insufficiency [2].

An association exists between renal and inner ear disorders. There are a variety of hereditary and congenital nephropathies associated with sensorineural hearing loss (SNHL) [3]. The kidney and stria vascularis of the cochlea share physiologic, ultrastructural, and antigenic similarities that could explain the link between chronic kidney disease and hearing impairment [4]. The similarity between the stria vascularis and the renal tubules could account for the fact that most nephrotoxic medication is ototoxic in nature [5].

Children with NS have biochemical impairments that include hyponatremia, hypocalcemia, and hyperlipidemia [6]. These biochemical abnormalities are known to cause hearing impairment. Many children with NS are treated with multiple courses of diuretics, which also cause ototoxicity. These factors increase the risk for hearing impairment in children with NS. Children with FRNS/SDNS and SRNS are at risk for sensorineural hearing impairment [7].

This study aimed to assess the prevalence of hearing impairment in children with different clinical types of NS and compare this with a control group without any detectable physical illness.

Materials and methods

This was a cross-sectional study conducted at a Tertiary Care Hospital, with randomly chosen nephrotic patients from the Nephrology Clinic, New Children Hospital, Cairo University, Cairo, Egypt, from September 2013 till May 2014 and was approved by the Ethics Scientific Committee at the Cairo University Hospital. Patients included in the study fulfilled the following criteria: age between 5 and 16 years; having laboratory investigation results consistent with NS; and regularly attending periodic visits and regularly taking prescribed medications. Children with previously known hearing loss and those having chronic suppurative otitis media, patients with any other medical or surgical illness causing hearing impairment, and nephrotic patients with renal insufficiency were excluded from the study.

Written informed consent was obtained from the legal guardian of each child before enrollment.

Forty-five children were enrolled and 15 age-matched and sex-matched normal healthy children served as controls for the study.

The children were divided into four groups: group 1 included 15 patients with SSNS; group 2 included 15

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patients with FRNS/SDNS; group 3 included 15 patients with SRNS; and group 4 included 15 normal healthy controls.

SSNS was defined as remission in response to corticosteroid treatment alone. Frequent relapse was defined as two or more relapses within 6 months of the initial episode or more than three relapses within any 12-month period. Steroid dependence was defined as two consecutive relapses when on alternate-day prednisolone or within 14 days of discontinuation of prednisolone. Steroid resistance was defined as the absence of remission despite therapy with daily prednisolone at a dose of 2 mg/kg/day for 4 weeks [2].

Patients were subjected to detailed demographic data and clinical history evaluation, careful clinical evaluation and anthropometric measurements, and laboratory evaluation (including urine analysis and quantification of proteinuria, serum electrolytes, blood urea, and serum creatinine). Corrected calcium was calculated using the following formula: corrected serum calcium = serum calcium + 0.8 (4 – serum albumin) [8].

If done, histological diagnosis of NS (renal biopsy) and treatment regimen in each patient were reviewed and analyzed. Special attention was given to diuretic use.

Pure tone audiometry was performed using the DANPLEX DA65 audiometer. Otoscopic examination was performed in all children. The ears were first examined for any wax and discharge, and those with ear wax were tested for hearing loss after wax removal. Biological calibration of the audiometer was done before each hearing assessment. The test was carried out in an empty quiet room in the Nephrology Clinic and was thoroughly explained to each child before the hearing assessment was started. Both air conduction and bone conduction (BC) were tested using the conventional 5-up–10-down method (the Hughson–Westlake approach as modified by Carhart–Jerger).

The hearing thresholds were obtained at frequencies of 250, 500, 1000, 2000, 4000, and 84 000 Hz, and intensity was measured in decibels. The results of each test were plotted graphically as the pure tone audiogram and interpreted both qualitatively and quantitatively.

Qualitative information provided information on the type of hearing loss – that is, whether it was conductive, mixed, or SNHL. Conductive hearing loss was diagnosed when BC was normal (< 20 dB) and the difference in the air-to-bone gap was greater than or equal to 20 dB. SNHL was diagnosed when the BC level was more than 20 dB and the difference in the air-to-bone gap was less than or equal to 15 dB. Mixed hearing loss was diagnosed when the BC level was more than 20 dB and the difference in the air-to-bone gap was more than 15 dB.

Quantitative information on the air conduction threshold allowed deafness to be graded into mild (26–40 dB), moderate (41–55 dB), moderately severe (56–70 dB), severe (71–91 dB), and profound (> 91 dB). The average hearing threshold level at frequencies of 250, 500, 1000, 2000, 4000, and 8000 Hz, respectively, was calculated as the pure tone average.

Statistical analysis

Data were collected, checked, revised, and entered into the computer. Data were analyzed with SPSS statistical package, version 17 (SPSS Inc., Chicago, Illinois, USA). The Excel computer program was used to tabulate the results and represent it graphically. Quantitative variables were expressed as mean and SE. Qualitative variables were expressed as count and percentage.

One-way analysis of variance was used to declare the significant difference between groups at P less than 0.05. Duncan multiple comparison test at P less than 0.05 was used to declare the significance between two groups. The χ^2-test used to declare the significant difference in the distribution between groups at P less than 0.05 [9].

Results

Out of the 45 patients, 32 were male and 13 were female, with a male to female ratio of 2.4 : 1. In the control group, nine patients were male and six were female. Figure 1 shows the sex distribution in the three patient groups and in the control group.

Group 1: SSNS group.
Group 2: SDNS group.
Group 3: SRNS group.
Group 4: control group.

Only five patients had hypertension (11.11%) and all of them were controlled. Mild to moderate edema was detected in eight (17.78%) patients and underweight was noted in four patients whose weight was below the third percentile for age and sex according to Egyptian Growth Charts [10].

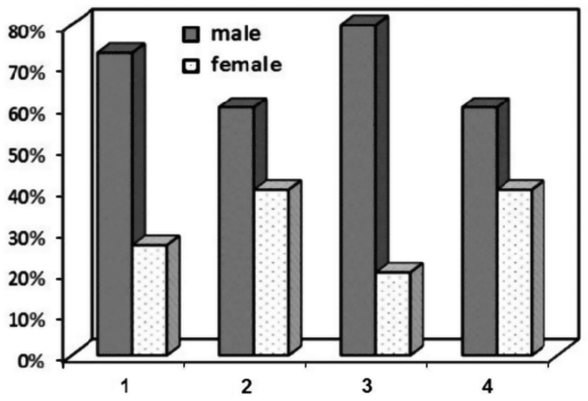
There were no statistically significant differences between the three groups as regards complications during periods of relapses in the form of infections: peritonitis and urinary tract infection were seen in three patients (6.67%), two of them belonging to the SRNS group and the third to the SDNS group; neuropsychiatric complications (depression, psychosis, hypertensive encephalopathy, and hemiplegia) were detected in four patients (8.89%), two of them belonging to the SRNS group, one to the SDNS group, and the fourth to the SSNS group.

Figure 2 shows the number of relapses in the three patient groups. It shows that the number of relapses is more in group 2 (SDNS) and group 3 (SRNS) than in group 1 (SSNS).

Group 1: SSNS group.
Group 2: SDNS group.
Group 3: SRNS group.

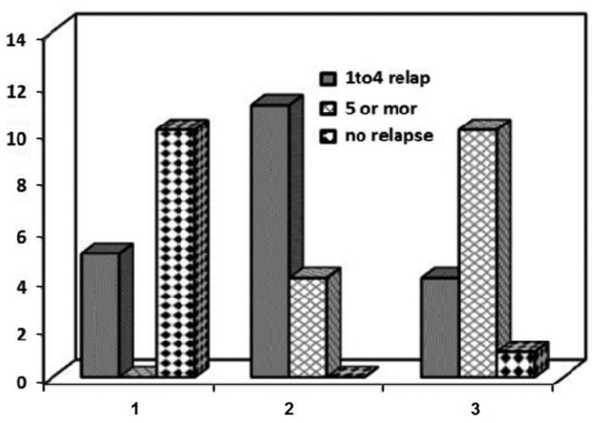
Table 1 shows the age, duration, and laboratory investigation results of the three patient groups. Patients with SRNS were significantly older than patients of the other two groups ($P = 0.007$). Duration of disease was significantly longer in the SRNS group than in the other two groups ($P = 0.001$). Total proteins, serum albumin, and serum calcium levels were significantly lower in the SRNS group than in the other two groups ($P = 0.037$, 0.023, and 0.046, respectively). Serum cholesterol level

Fig. 1



Sex distribution in the three patient groups and in the control group.

Fig. 2



Number of relapses in the three patient groups.

Table 1 Age, duration and laboratory investigations of the studied groups

	SSNS	SDNS	SRNS	*P* value
Age (years)	9.37 ± 3.12	8.57 ± 3.05	12.21 ± 3.22	0.007*
Duration of illness (years)	3.20 ± 3.46	4.55 ± 2.72	7.59 ± 2.69	0.001*
Laboratory investigations				
24 h proteins (g/day)	0.62 ± 1.28	0.41 ± 1.03	2.48 ± 5.1	0.143
Serum creatinine (mg/dl)	0.47 ± 0.14	0.42 ± 0.19	0.58 ± 0.28	0.125
Total proteins (mg/dl)	6.68 ± 1.03	6.62 ± 0.38	5.99 ± 0.80	0.037*
Serum albumin (mg/dl)	3.81 ± 0.65	3.83 ± 0.53	3.11 ± 1.06	0.023*
Serum cholesterol (mg/dl)	193.8 ± 66.28	186.73 ± 39.99	276.87 ± 157.86	0.034*
Serum calcium (mg/dl)	9.09 ± 0.54	9.43 ± 0.63	8.81 ± 0.78	0.046*
Corrected calcium (mg/dl)	9.25 ± 0.70	9.57 ± 0.71	9.53 ± 1.22	0.583

*Significant and highly significant *P* values.
SDNS, steroid-dependent nephrotic syndrome; SRNS, steroid-resistant nephrotic syndrome; SSNS, steroid-sensitive nephrotic syndrome.

Table 2 Biopsy and audiometry results and treatment modalities of the three patient groups

	SSNS ($n=15$)	SDNS ($n=15$)	SRNS ($n=15$)
Biopsy			
Biopsy not done	15	14	6
MCNS	0	1	4
Focal proliferative	0	0	1
Membranous GN	0	0	1
Membanoproliferative GN	0	0	3
ACE-II use (captopril)	6	11	12
Steroids (mg)			
No steroids	8	5	3
Daily<30	0	1	2
Daily ≥ 30	0	1	1
EOD<30	2	4	4
EOD ≥ 30	5	4	5
Immunosuppressants			
No immunosuppressant	15	2	0
1 immunosuppresant	0	10	6
2 immunosuppresants	0	2	5
3 immunosuppresants	0	1	2
4 immunosuppresants	0	0	2
Diuretic use			
No diuretic use	13	12	7
1 diuretic	2	3	6
2 diuretics	0	0	2
Hearing defect by audiometry	2	2	6

ACE-II, angiotensin-converting enzyme inhibitor; EOD, every other day; GN, glomerulonephritis; MCNS, minimal change nephrotic syndrome; SDNS, steroid-dependent nephrotic syndrome; SRNS, steroid-resistant nephrotic syndrome; SSNS, steroid-sensitive nephrotic syndrome.

was significantly higher in the SRNS group than in the other two groups ($P = 0.034$).

Table 2 shows the results of renal biopsy and audiometry results and also treatment modalities in the three patient groups. Some patients used one, two, three, or four immunosuppressants. The immunosuppressants used in our patients were cyclophosphamide, levamizole, cyclosporine A, mycophenolate mofitil, and chlorambucil. The diuretics used were either spironolactone or furosemide.

There was a positive correlation between the number of relapses and duration of illness in the total patients.

Sensorineural hearing defect was found in 10 out of 45 nephrotic cases (22%) (two from the SSNS group, two from the SDNS group, and six from the SRNS group] and in two out of 15 children in the control group (13%).

Table 3 shows the comparison between nephrotic patients with normal hearing and patients with hearing loss.

Table 4 shows the comparison between nephrotic patients with normal hearing and patients with hearing loss as regards the number of relapses, duration of illness, serum albumin, and corrected calcium. Number of relapses was significantly more in patients with hearing defect ($P = 0.006$).

All hearing affection was bilateral. SNHL in NS predominantly occurred at lower frequencies. Isolated low-frequency affection (250–500 Hz) was found in three patients out of 45 (6.67%) (one from the SSNS group, one from the SDNS group, and one from the SRNS group) compared with none of the 15 control children (0%). All frequencies affection (250–500–1000–2000–4000 Hz) was found in seven cases out of 45 (15.56%)

Table 3 Comparison between nephrotic patients with normal hearing and patients with hearing loss

	Normal hearing	Hearing defect
Sex		
Male	26	6
Female	9	4
Weight percentiles		
Third or less weight percentile	3	1
3rd–97th	30	9
More than 97th	2	0
Hypertension	4	1
ACE-II (captopril) use	21	8
Steroids (mg)		
No steroids	14	2
Daily <30	2	1
Daily ≥ 30	2	0
EOD<30	10	6
EOD ≥ 30	7	1
Immunosuppresants		
No immunosuppressant	15	2
1 immunosuppresant	14	2
2 immunosuppresants	4	3
3 immunosuppresants	1	2
4 immunosuppresants	1	1
Diuretic use	8	5
Complications		
No complications	30	7
Infections	2	1
Superficial gangrene	1	0
Neuropsychiatric	2	2

Infections include UTI, peritonitis, and HAV.
Neuropsychiatric complications include hypertension encephalopathy, hemiplegia, depression, and psychosis.
ACE-II, angiotensin-converting enzyme inhibitor; EOD, every other day; UTI, urinary tract infection.

Table 4 Clinical and laboratory comparison between nephrotic patients with normal hearing and patients with hearing loss

	Normal hearing	Hearing defect	*P* value
Relapse number	2.49 ± 2.58	5.5 ± 3.95	0.006
Duration (years)	4.78 ± 3.15	6.82 ± 4.36	0.232
Serum albumin (mg/dl)	3.52 ± 0.85	3.78 ± 0.78	0.396
Corrected calcium (mg/dl)	9.47 ± 0.79	9.36 ± 1.26	0.721

(one from the SSNS group, one from the SDNS, and five from the SRNS group), compared with two out of 15 control children (13.3%).

Discussion

Children with NS are at risk of hearing impairment due to nephrotoxic drugs and biochemical impairments [7].

Hearing organ in children with NS in the course of chronic glomerulopathy is significantly worse than in healthy children [11].

In our study, the male to female ratio was 2.4 : 1. Ghobrial *et al.* [12] reported a male to female ratio of 5 : 1.

In our study, patients with SRNS were significantly older than those in the other groups. This is in contrast to the results of Ghobrial *et al.* [12], who found that there was no statistically significant difference between the three groups as regards age.

In our study, five patients (11.11%) had hypertension, which were under control. This is in agreement with the results of Ghobrial *et al.* [12], who found that 15% of nephrotic patients were hypertensive.

In our study, the number of relapses was more in the SDNS and SRNS groups than in the SSNS group and there was a positive correlation between the number of relapses and duration of disease in the total 45 patients.

Our study revealed a sensorineural hearing defect in 10 out of 45 nephrotic cases, representing 22%, and in two out of 15 children in the control group, representing 13%. This matches the results obtained by Orendorz-Fraczkowska *et al.* [11], who evaluated the hearing status in 28 children with NS and in 28 healthy children and concluded that hearing was significantly worse in children with NS than in healthy children.

Nephrotic patients with hearing defects were distributed as follows in the different categories of NS: two cases in the SSNS group (13%), two cases in the SDNS group (13%), and six cases in the SRNS group (40%). Thus it is clear that the SRNS group was the most affected. This is similar to the study done by Saha *et al.* [7], who diagnosed SNHL in 10 out of 20 children with SRNS (50%) and in three out of 20 children with SDNS (15%). The small percentage of SNHL in SSNS patients matched the results obtained by Bayazit *et al.* [13], who found that minimal change nephrotic syndrome patients did not have an altered hearing status, both in remission and in relapsed periods of the disease.

All hearing affection was bilateral. SNHL in nephrotic syndrome predominantly occurred at lower frequencies. Isolated low-frequency affection was found in three cases out of 45 (6.67%) compared with none in the 15 control children (0%). All frequencies affection was found in seven cases out of 45 (15.56%) compared with two out of 15 control children (13.3%)

We found many parameters significantly affected in the SRNS group in comparison with the two other groups with NS. A relation was found between these parameters and the higher incidence of SNHL in the SRNS cases. Total calcium was significantly lower in the SRNS group than in SSNS and SDNS groups ($P = 0.046$). The association of hypocalcemia and SNHL was explained by Brookes [14], who reported that calcium is involved in the basic organization of biological systems and physiochemical reactions of cellular function. It also plays a role in cellular adhesion, regulation of membrane permeability, and control of neuromuscular excitability. ATPase activity, which maintains the differential biochemical integrity of the inner ear fluid, is calcium dependent.

Fahlke and Fischer [15] stated that ClC-K channels form a subgroup of anion channels within the ClC family of anion transport proteins. They are expressed predominantly in the kidney and in the inner ear and are necessary for NaCl resorption in the loop of Henle and for $K+$ secretion by the stria vascularis.

Also, total plasma protein and serum albumin were significantly lower in the SRNS than in both SSNS and

SDNS ($P = 0.037$ and 0.023, respectively). Similarly, Ghobrial *et al.* [12] reported that there were statistically significant differences between the three groups as regards total protein, serum albumin, and calcium (being lowest in SRNS group).

In this study, mean serum cholesterol level was significantly higher in the SRNS group than in patients of the other two groups. Ghobrial *et al.* [12] reported that there were statistically significant differences between the three groups as regards 24 h urinary proteins and cholesterol (being highest in the SRNS group).

We noticed in our study a significant P value of 0.01 concerning the higher incidence of SNHL in cases taking furosemide compared with its incidence in those not taking it. Thus, an association was deduced between furosemide use and SNHL. This finding may explain the exaggerated deleterious effect of furosemide in the SRNS cases that exposes them to a higher risk for SNHL.

Rybak *et al.* [16] compared the ototoxicity of furosemide in normal and analbuminemic rats and found that analbuminemic rats were much more susceptible to furosemide toxicity than were normal rats. These authors also found that the access of furosemide to its site of ototoxic action in the cochlea depends on the quantity of unbound furosemide in the serum. Mudd *et al.* [17] stated that ototoxic medication, including furosemide, can affect ionic gradients between the endolymph and perilymph, resulting in edema of the epithelium of the stria vascularis. It is further indicated in the literature that furosemide affects cochlear function by altering the endocochlear potential [18].

Regarding the duration of the disease, SRNS patients had significantly longer disease duration compared with other groups ($P = 0.001$). This is in contrast to the results of Ghobrial *et al.* [12], who found that there were no statistically significant differences between SSNS, SDNS, and SRNS groups. The longer duration increases the risk for ototoxicity from higher cumulative doses of furosemide. This was confirmed by Saha *et al.* [7], who stated that higher cumulative doses of furosemide were a risk factor for SNHL.

It is postulated by Rarey and Ross [19] that furosemide inhibits the activity of the sodium–potassium ATPase of the stria, thereby preventing the active transport of sodium into the capillaries. The accumulated sodium would retain water and produce the characteristic water spaces and lead to an osmotic gradient that would eventually result in an increase in the fluid content of the endolymph. This in turn would create a hydrops-like scenario, the maximum impact of which would be felt at the apex of the cochlea. As the apical region is responsible for hearing at low frequencies, hydrops would lead to low-frequency hearing loss. Furosemide also causes strial edema, degeneration of the intermediate cell, and alterations in the endoperilymphatic barrier in the inner ear.

When comparing patients with hearing loss with patients with normal hearing, only one patient with hearing defect had controlled hypertension compared with four patients with controlled hypertension among patients with normal hearing. Therefore, hypertension was not a risk factor for hearing loss in this study.

The number of relapses was statistically higher in nephrotic patients with hearing defect. This is in contrast to the results of Saha *et al.* [7], who found no statistically significant differences between patients with normal hearing and those with hearing defect as regards the number of relapses ($P = 0.19$).

A major limitation of this study is the small sample size and lack of follow-up.

Conclusion and recommendation

Children with NS are more susceptible to SNHL than are normal children. SRNS children are the most affected ones among the NS categories. The longer the duration of the glomerulopathy, the more the number of relapses and thus greater the exposure to the risk for SNHL. The risks deduced from our study were low serum calcium level, low serum protein, and low serum albumin levels. A restricted use of diuretics in selected cases and simultaneous use of albumin should be encouraged in case of diuretic use to decrease its deleterious effect. Finally, a hearing evaluation should be included in the routine management of NS cases.

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Conflicts of interest

There are no conflicts of interest.

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الملخص العربي

الحالة السمعية في الأطفال المصريين الذين يعانون من المتلازمة النفروزية

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إن الأطفال الذين يعانون من المتلازمة النفروزية لديهم اضطراب كيميائي كما أنهم يتلقون علاجا بمدرات البول الذي قد يسبب ضعف بالسمع. إن الهدف من هذا البحث هو دراسة انتشار ضعف السمع بين الأطفال الذين يعانون من أنواع مختلفة من المتلازمة النفروزية. هذه الدراسة كانت دراسة مقطعية أجريت في عيادة الكلي بمستشفى الأطفال الجامعي بجامعة القاهرة في الفترة من سبتمبر ٢٠١٣ إلى مايو ٢٠١٤ وقد شملت ٤٥ طفلا مصابون بالمتلازمة النفروزية وكذلك ١٥ طفلا صحيحا من نفس المرحلة السنية ومن الجنسين كمجموعة ضابطة. وقد تم تقسيم المرضى إلى ثلاثة مجموعات كل منها يتكون من ١٥ مريضا: المجموعة الأولى شملت المرضي المستجيبين للكورتيزون، المجموعة الثانية شملت المرضي المعتمدين على الكورتيزون وكثيري الانتكاسة والمجموعة الثالثة شملت المرضى غير المستجيبين للكورتيزون. وقد تم عمل فحص مقياس سمع لكل من المرضى والمجموعة الضابطة. وقد وجد ضعف السمع في ١٠ مرضى من ٤٥ مريضا (٢٢٪,٢٢) وكان ٦ منهم من المجموعة الثالثة وقد وجد أيضا أن عدد مرات الانتكاسة كانت أكثر في المرضى الذين يعانون من ضعف بالسمع. نستخلص من هذه الدراسة أن الأطفال الذين يعانون من المتلازمة النفروزية معرضون لضعف السمع أكثر من الأطفال الأصحاء وأن المرضى غير المستجيبين للكورتيزون هم الأكثر إصابة بضعف السمع. لذلك نوصى بوضع تقييم السمع كجزء من العلاج الروتيني لمرضى المتلازمة النفروزية.