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Sensorineural Hearing Loss in Hemodialysis Patients

Marko Jakić[1], Dubravka Mihaljević[1], Lada Zibar[1], Marijana Jakić[2], Željko Kotromanović[3] and Hrvoje Roguljić[4]

[1] Clinic for Internal Medicine, University Hospital Center Osijek, Osijek, Croatia
[2] Public Health Institute of the Osijek-Baranya County, Osijek, Croatia
[3] Clinic for Otorhinolaryngology, University Hospital Center Osijek, Osijek, Croatia
[4] Department of Mineral Metabolism, School of Medicine, University »J. J. Strossmayer«, Osijek, Croatia

A B S T R A C T

Chronic renal failure affects all organ systems. Senses are not exception and hearing impairment is common, particularly sensorineural hearing loss (SNHL). The term »SNOS of unknown origin« or »uremic deafness« is related to only a smaller part of the cases with unclear etiology of the impairment. The study searched for SNOS in 66 chronic hemodialysis (HD) patients, mean age 51.50±12.70 years. They were treated by HD for 69.70±53.80 months. The relation between the severity of the impairment and the patients' age, duration of HD treatment (months) and a set of laboratory parameters typical for chronic HD patients was examined. The aim of the study was to detect potential causes of the impairment. The increased hearing threshold (HT) of above 20 dB for all frequencies was found in 42 patients (mean HT 26±10.50 dB), for speaking area frequencies in 22 patients (mean HT 19.70±8.80 dB), and in 56 patients for high frequencies (mean HT 41.70±19.70 dB). The significant positive correlation of HT was found only with the patients' age ($r=0.49$, $p<0.01$). The patients older than 45 years had higher mean HT than those younger, and those older than 65 also had higher HT than the younger ones. Patients with pathological value of HT were significantly more common among the older subgroup of patients, when divided according to the age at both cutoff values of 45 and of 60 years. Mean HT did not differ significantly according to the duration of HD treatment (subgroups A- no longer than 60 months, B- from 61 to 120 months, and C- longer than 120 months). The patients with pathological HT did not differ significantly in frequency among those subgroups, and the subgroups were not different according to the mean age (A- 50.30±13.20 years; B- 51.40±12.75 years; C- 55.80±10.55 years). In conclusion, our results along with other authors' published data report on SNHL as very frequent finding among chronic HD patients and suggest multifactorial etiology. Accurate proportion of those with SNHL of unknown origin is not possible to determine. Those cases are probably not caused by uremic polyneuropathy and/or preterm vascular aging only, although those factors are likely to play crucial roles.

***Key words**: sensorineural hearing loss, uremic deafness, hemodialysis, chronic renal failure*

Introduction

Hearing disorders are the most frequent of all human sensorial impairments[1]. They make problems in social contacts in every tenth person at the age of 65 or more, and in every second at the age of 80 and older than that[2,3]. Hearing impairment is very common in end stage renal disease (ESRD) patients, sensorineural hearing loss more often than conductive impairments. They occur due to damaged sensory and neural inner ear structures, sensory cells of Cortie's organ, acoustic nerve and structures in central nervous system. Literature data report that 20–87% of ESRD patients have sensorineural hearing loss[4–9], depending on the accepted criteria and studied groups' features[10], but significantly more frequently than the age matched general population[11,12]. More than 80% of chronic renal failure patients with sensorineural hearing loss have a known cause of the hearing impairment (ototoxic antimicrobials, diuretics, exposure to noise, inborn or genetic otonephropathies or

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presbyacusis)[4,13–15]. Remaining less than 20% of the patients have impairment termed »sensorineural hearing loss of unknown origin« or »uremic deafness«[9]. Uremic toxins or unnamed endogenous[16,17] or exogenous toxins[18,19] that also cause uremic polyneuropathy are possible etiologic agents, or it occurs due to preterm vascular aging[10].

Sensorineural hearing loss is characterized by increased hearing threshold (HT) of 15–20 dB above HT for healthy or above HT in age matched otherwise healthy people with presbyacusis, commonly in high frequencies area of above 2000 Hz[10], equally for bone and air conductivity, without so called air bone gap[20].

Uremic polyneuropathy is rare in patients with glomerular filtration rate above 10% of normal values. Not only the stage but also the duration of chronic renal failure plays an important role in the development of uremic polyneuropathy. Less important is the role of age, since it is well known that the prevalence of the disorder increases with age. At the start of chronic HD treatment two thirds of the patients have uremic polyneuropathy. Dialysis usually gets the control over the condition or moderately improves it. Complete cure is very seldom. It is not quite clear whether peritoneal dialysis has better effect on uremic polyneuropathy due to better middle molecules clearance or because of longer preservation of residual renal function. Only kidney transplantation enables complete cure[21].

Vascular or even more accurately cardiovascular diseases are very common in ESRD patients[22]. They are caused by traditional and nontraditional risk factors[23]. ESRD patients are at increased risk of cardiovascular mortality for 20 times in comparison with age and comorbidities (including diabetes mellitus) matched non ESRD patients[24]. Forty to 50% of ESRD die of cardiovascular diseases[25], as well as more than a half of chronic dialysis patients[24]. Based upon the data the mortality of those patients is greater than among the most patients that suffer from malignant diseases[26].

After excluding rare causes of sensorineural hearing loss in chronic renal failure patients[27,28], both in those in predialysis stage and those undergoing dialysis, we come to two main possible causes: uremic polyneuropathy and preterm vascular aging. Related to that we should have in mind that both causes were more frequent and more manifested with increased age and duration of chronic renal failure, and that conservative as well as dialysis treatment could have an impact only on uremic polyneuropathy but not on preterm vascular aging. However, considering the two potential causes there is no way to explain why sensorineural hearing loss does not always occur bilaterally.

The study reports on the prevalence of sensorineural hearing loss in a selected group of chronic HD patients and on the correlation of severity of the impairment with patients' age, HD treatment duration and with a set of laboratory parameters characteristic for the group of patients. The aim was to detect possible causative factors for its development.

Patients and Methods

The study included 66 ESRD patients treated with bicarbonate HD, three times a week, for 4–4.5 hours, using capillary dialyzers made of cellulose diacetate or polysulphone, of the surface area of 1.5–2.2 m^2, of predominantly low permeability, sterilized by γ-irradiation or ethylene oxide, with common blood (250–300 mL/min) and dialysate flow (500 mL/min).Water for dialysis was prepared by reverse osmosis, and conductivity of below 10 $\mu S/cm^3$ was ensured. Exclusion criteria were history of exposure to noise, Alport's syndrome and those with conductive and/or mixed hearing loss confirmed by audiometry and tympanography. Thanks to the long duration of chronic renal failure we presumed certain ototoxic antimicrobials' and diuretics' contribution for all chronic HD patients, considering the group homogenous regarding this issue.

The patients underwent examination by the otorhynolaryngologist which was familiar with the study. Tympanography using tympanometer AMPLAID 720 and audiometry using audiometer AMPLAID 455 (Italy) were performed. HT was measured for air and bone conductivity, for both ears, for frequencies of 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz. Only audiograms of the type A were analyzed. Mean air conductivity HT for each ear for all frequencies together was determined, and also separately for the frequencies in the speaking area (250–4000 Hz) and in the area of high frequencies (6000 and 8000 Hz). Common mean HT was calculated for each patient from values obtained for both ears separately, for all frequencies together and separately for speaking area frequencies and for high frequencies.

According to the Bureau International de Audiophonologie normal HT comprises values from 0 to 20 dB. Mild hearing loss have those with HT from 21 to 40 dB, moderate from 41 to 70 dB, serious from 71 to 90 dB, while patients with severe hearing loss have HT above 90 dB.[29]

Mean values for a set of laboratory parameters were calculated from the results of regular checkups obtained during the last year. Normal serum values were considered as follows: hematocrit (htc) 0.36–0.46 l/L for women, 0.41–0.55 l/L for men, determined using Coulter Counter, automatic cell counter; calcium (Ca) 2.25–2.75 mmol/L, determined using colorimetry with ortocresolphtalein; phosphates (P) 0.60–1.40 mmol/L, determined using molibdat UV method; alkaline phosphatase (AP) from 40 to 110 U/L for women and 43–88 U/L for men, determined by the method recommended by IFCC, using AMP buffer; total proteins 65–85 g/L, determined using Biuret spectrophotometry, modified by Henry with blind serum test; albumin 35–52 g/L, determined using BCG method with brilliant cresil green; glucose 4.2–6.4 mmol/L, determined using glucose oxidase PAP method; sodium (Na) from 135 to 150 mmol/L, using method with ion selective electrode; potassium (K) 3.6–5.6 mmol/L, determined by method with ion selective electrode; urea from 3.4 to 8.9 mmol/L, determined by enzymatic UV method

with urease and glutamate dehydrogenase; creatinine from 42 to 115 μmol/L, using enzymatic colorimetry PAP method; albumin/globulins ratio; intact parathormon (i-PTH) from 8 to 62 pg/mL, determined by ELSA PTH immunoradiometric assay, GIF-SUR-YVETE CEDEX, France). Single hemodialysis dose, Kt/V, was calculated from predialytic and postdialytic urea, using equation according to Jindal et al.[30], (Kt/V = {4 × (U1 – U2/U1)} – 1.2).

TABLE 1
DEMOGRAPHICS, COMMON LABORATORY FINDINGS AND HEARING THRESHOLD IN CHRONIC HEMODIALYSIS (HD) PATIENTS

	$\overline{X}$±SD, n=66
Age (years)	51.50±12.70
Duration of chronic HD treatment (months)	69.70±53.80
Hematocrit (L/L)	0.28±0.04
Total serum proteins (g/L)	71.70±5.10
Serum albumin (g/L)	40.60±3.70
Intact-parathormon (pg/mL)	263±247
Serum calcium (mmol/L)	2.30±0.20
Serum phosphates (mmol/L)	2.30±0.65
Serum alkaline phosphatase (U/L)	104±137
Serum creatinine (±mol/L)	798±193
Serum urea (mmol/L)	19.50±4.50
Kt/V	1.35±0.10
Hearing threshold (all frequencies) (dB)	26.00±10.50
Hearing threshold (speech area) (dB)	20.00±9.00
Hearing threshold (high frequencies) (dB)	41.70±19.50

The obtained results were analyzed for the whole group and for the subgroups according to age and to the hemodialysis treatment duration. Mean values were expressed as means and standard deviation. Differences were determined using Student's t-test for independent variables. Proportions were compared using χ^2-test. Single correlations were examined. Differences at 5% and 1% levels were considered significant ($p<0.05$ and $p<0.01$)[31].

Results

There were 66 participants, 34 women and 32 men, 51.50±12.70 years of age (ranging from 21.45 to 70.90 years), treated by chronic HD for 69.70±53.80 months on average (ranging from 3 to 263 months). Their mean HT was 26±10.50 dB for all frequencies, 19.70±8.80 for frequencies in the speaking area and 41.70±19.70 dB for high frequencies (Table 1). Increased HT above 20 dB (according to the Bureau International de Audiophonologie[29]) was found in 42 patients (63.64%) for all frequencies, in 22 patients (33.33%) for frequencies in the speaking area and in 56 patients (84.85%) for high frequencies. According to the severity of the hearing loss,

TABLE 2
DIFFERENCE IN HEARING THRESHOLD (DB) BETWEEN CHRONIC HEMODIALYSIS PATIENTS YOUNGER THAN 45 YEARS (A) AND OLDER THAN 45 YEARS OF AGE (B)

	A n=19	B n=47	t-test	p
Hearing threshold (dB)				
All frequencies	19.30±5.90	28.60±10.70	4.49	**s
Speech area	14.85±4.20	21.60±9.40	4.02	**s
High frequencies area	30.30±16.15	46.35±19.25	3.45	**s
Age (years)	35.25±7.80	58.05±7.10	11.06	**s

* $p<0.05$ ** $p<0.01$

TABLE 3
INCREASED HEARING THRESHOLD (ABOVE 20 DB) FREQUENCIES AMONG CHRONIC HEMODIALYSIS PATIENTS AND THE RELATED DIFFERENCE BETWEEN YOUNGER THAN 45 YEARS (A) AND OLDER THAN 45 YEARS OF AGE (B)

	All frequencies	Speech area	High frequencies area
	n–%	n–%	n–%
A (n=19)	6–31.58	2–10.53	13–68.42
B (n=47)	36–63.16	20–42.55	43–91.49
Total (n=66)	42–63.64	22–33.33	56–84.85
χ^2-test	**11.85	*6.25	*5.60
p	$p<0.01$	$p<0.05$	$p<0.05$

*$p<0.05$ ** $p<0.01$

there were 35 patients with mild (83.33%) and 7 patients (16.67%) with moderate impairment. HT significantly correlated positively only with the patients' age (r=0.49, p<0.01). There was not significant correlation between HT and duration of HD treatment (r=0.03, p>0.05), neither between HT and laboratory parameters typical for that group of patients.

The HD patients older than 45 years (n=47) had significantly higher HT values in comparison with the younger patients (n=19) (Table 2). There was a significantly higher proportion of patients with pathological value of HT above 20 dB for all frequencies, for speaking area frequencies and for high frequencies among the patients older than 45 years than in the subgroup of youn- ger patients (Table 3).

The patients older than 60 years (n=23) had higher HT for all frequencies and for higher frequencies in comparison with the subgroup of younger patients (n=43), whereas HT for frequencies in the speaking area did not differ between those subgroups (Table 4). There was a significantly higher proportion of patients with pathological value of HT above 20 dB for all frequencies and for high frequencies among the patients older than 60 years than in the subgroup of younger patients, whereas the difference was not significant for speaking area frequencies (Table 5).

TABLE 4
DIFFERENCE IN HEARING THRESHOLD (DB) BETWEEN CHRONIC HEMODIALYSIS PATIENTS YOUNGER THAN 60 YEARS (A) AND OLDER THAN 60 YEARS OF AGE (B)

	A n=43	B n=23	t-test	p
Hearing threshold (dB)				
All frequencies	23.60±10.95	30.30±7.95	2.82	**s
Speech area	18.40±9.30	22.00±7.35	1.73	ns
High frequencies area	36.85±10.50	50.85±14.30	3.23	**s
Age (years)	44.75±10.50	64.10±2.80	11.31	**s

* p<0.05 ** p<0.01

TABLE 5
INCREASED HEARING THRESHOLD (ABOVE 20 DB) FREQUENCIES AMONG CHRONIC HEMODIALYSIS PATIENTS AND THE RELATED DIFFERENCE BETWEEN YOUNGER THAN 60 YEARS (A) AND OLDER THAN 60 YEARS OF AGE (B)

	All frequencies	Speech area	High frequencies area
	n–%	n–%	n–%
A (n=43)	21–48.84	11–25.58	33–76.74
B (n=23)	21–91.30	11–47.83	23–100.00
Total (n=66)	42–63.64	22–33.33	56–84.85
χ^2-test	**11.68	3.34	*6.30
p	p<0.01	p>0.05	p<0.05

* p<0.05 ** p<0.01

TABLE 6
DIFFERENCE IN HEARING THRESHOLD (DB) BETWEEN SUBGROUPS DIVIDED ACCORDING TO CHRONIC HEMODIALYSIS (HD) TREATMENT DURATION (MONTHS): A – SHORTER THAN 60, B –LONGER THAN 60 AND SHORTER THAN 120, AND C – LONGER THAN 120

	A n=31	B n=26	C n=9	t-test		
				A:B	A:C	B:C
Hearing threshold (dB)						
All frequencies	25.60±11.10	26.10±10.60	27.80±8.40	0.28	0.75	0.50
Speech area	20.00±9.40	19.90±8.70	17.80±7.20	0.05	0.75	0.71
High frequencies area	38.30±19.10	42.00±19.60	52.80±20.00	0.73	1.92	1.39
HD treatment duration (months)	27.60±14.90	83.85±15.42	174.10±47.00	**13.93	**9.21	**5.65
Age (years)	50.30±13.20	51.40±12.75	55.80±10.55	0.31	1.29	1.02

*p<0.05 ** p<0.01

TABLE 7
INCREASED HEARING THRESHOLD (ABOVE 20 DB) FREQUENCIES AND THE RELATED DIFFERENCE BETWEEN SUBGROUPS DIVIDED ACCORDING TO CHRONIC HEMODIALYSIS (HD) TREATMENT DURATION (MONTHS): A – SHORTER THAN 60, B –LONGER THAN 60 AND SHORTER THAN 120, AND C – LONGER THAN 120

	All frequencies	Speech area	High frequencies area
	n–%	n–%	n–%
A (n=31)	21–67.74	10–32.26	23–74.19
B (n=26)	15–57.69	11–42.31	24–92.31
C (n=9)	6–66.67	1–11.11	9–100.00
Total (n=66)	42–63.64	22–33.33	56–84.85
χ^2-test	2.76	2.96	5.47
p	p>0.05	p>0.05	p>0.05

* p<0.05 ** p<0.01

The subgroups formed according to the duration of HD treatment (A – no longer than 60 months, B – from 61 to 120 months, C – longer than 120 months) neither differed significantly in HT, nor in the proportions of patients with pathological HT above 20 dB (Tables 6 and 7, resp.). Despite the difference in the duration of HD treatment, those three subgroups did not differ in age (A – 50.30±13.20 years; B – 51.40±12.75 years; C – 55.80±10.55 years; $t_{A:B}$=0.31, $t_{A:C}$=1.29, $t_{B:C}$=1.02, p>0.05 for each).

Discussion

The results of the study on hearing loss in the group of 66 chronic HD patients, mean age 51.50±12.70 years and treated with HD for 69.70±53.80 months on average, showed that 42 of them (63.64%) had increased HT above 20 dB. That proportion included pathological findings for all categories of frequencies that were analyzed by audiometry. According to the severity of the hearing loss, mild impairments prevailed and were related to more than 4/5 of the affected patients. The proportion of the patients with hearing loss determined for speaking area frequencies was significantly lower (22–33.33%) than for higher frequencies (56–84.85%) patients. Audiometry and tympanometry showed that the found impairments can be classified as sensorineural hearing loss. Even more important is the finding that HT correlated significantly positively with age (r=0.49, p<0.01) and did not correlate either with the duration of HD treatment (r=0.03, p>0.05) or with characteristic laboratory parameters. Older patients, both older than 45 and older than 60, had higher HT, accordingly higher proportions of pathological HT related to the older subgroups of patients. The results are in concordance with the data published by most other authors[7–8,32–33]. Klingerman et al.[32] found increased HT in 64% of their HD patients, with the objection that they took a distinct cutoff value (15 dB) for pathological HT for area of frequencies between 250–4000 Hz whereas 20 dB for the frequencies between 6000 and 8000 Hz. Sensorineural hearing loss prevailed, as it was recorded in 53.57% of the patients and related to 83.33% of all cases of hearing impairments. In the study of Santos et al.[33] increased HT above 25 dB was detected in 37.78% of their HD patients. Sensorineural or other nature of the impairments was not specified. Sensorineural hearing loss was found in 56% of HD patients in the publication of Shaheen et al.[8] and in 61.54% of those studied by Bazzi et al.[7].

Bazzi et al.[7] found hearing impairment in 56% of their patients which were younger than 45 years and in even 94% of the older. Gatland et al.[20] published that ESRD patients older than 50 years of age had significantly greater hearing loss for higher frequencies area than the younger.

As it was mentioned in the introduction chapter, sensorineural hearing loss is characterized with equal affection of both bone and air conductivity[20], without so called air-bone gap, with increased HT of 15 to 20 dB above HT for healthy people, or above HT value for normal presbyacusis for certain age which is commonly above 2000 Hz[10]. An important proportion of our patients, 63.64% of them (42 of 66) had that particular type of sensorineural hearing loss. That finding is in part the consequence of inability to exclude the cases of ototoxic drugs side effects or concomitant presbyacusis, although the study group was a selected one. According to Seidman et al.[34] there was namely 23% of general population between 65 and 75 years of age with presbyacusis, which was very similar impairment as sensorineural hearing loss in HD patients.

Many authors considered presbyacusis the hearing loss caused by aging[34–36], but they also stress the influence of genetic and environmental factors, socioeconomic conditions and diet[34], particularly folic acid and vitamin B_{12} defficiency[36]. They also point out that hearing deteriorates with increasing age, but that there are some older people with completely normal hearing ability[37]. Therefore physiologic presbyacusis does not exist[37].

If sensorineural hearing loss in ESRD is really caused by preterm vascular aging, it remains difficult to explain that even children with ESRD and with excluded ototoxic drugs influence also have that type of hearing loss (30% of them)[4–9,11–12].

Hearing ability did not differ among three subgroups of our patients distributed according to the duration of HD treatment (A – no longer than 60 months, B – from 61 to 120 months, and C – longer than 120 months). As it was already mentioned, HT did not correlate with the duration of HD treatment, very similar to the data published by Antonelli et al.[10] Furthermore, it is important to point out that the three subgroups were not different according to age, either. Bazzi et al.[7] made the same grouping by their patients (N=91) and found sensorineural hearing loss in 61.70% patients in the subgroup A, 59.30% patients in the subgroup B and 64% in subgroup C. They also found that mean HT and age were comparable between the subgroups, and finally concluded that HD did not negatively affect hearing ability.

Neither we, not Bazzi et al.[7] have audiograms from the period before starting HD treatment. As some of those patients are 10 years older now and HD itself did not retard vascular aging, they must have had lower HT by that preceded time. Their HT must have increased with time due to vascular aging, but must not have reached pathological values. Another possibility is that the observed HD period is too short for significant vascular aging, and the third and the most probable explanation is that hearing loss in chronic renal failure patients is caused not only by preterm vascular aging, but also by some other factors which might have benefited from HD.

In conclusion, our results along with other authors' published data report on SNHL as very frequent finding among chronic HD patients and suggest multifactorial etiology. Accurate proportion of those with SNHL of unknown origin is not possible to determine. Those cases are probably not caused by uremic polyneuropathy and/or preterm vascular aging only, although those factors are likely to play crucial roles.

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M. Jakić

Ukrinska 21, 31 000 Osijek, Croatia
e-mail: mjakic1@gmail.com

SENZONEURALNA OŠTEĆENJA SLUHA HEMODIJALIZOM LIJEČENIH BOLESNIKA

S A Ž E T A K

U kroničnom bubrežnom zatajenju (KBZ) patološkim zbivanjem zahvaćeni su svi organski sustavi. Nisu pošteđena ni osjetila, pa su tako česta i oštećenja sluha, pogotovo senzoneuralna (SNOS). Etiologija nije jasna samo za dio njih, a nazvana su »SNOS nepoznate etiologije« ili »uremijskom nagluhosti«. U ovom radu ispitivali smo prevalenciju SNOS 66 bolesnika prosječne dobi 51,50±12,70 godina, prosječno hemodijalizom (HD) liječenih 69,70±53,80 mjeseci. Intenzitet smo korelirali s dobi bolesnika, duljinom trajanja liječenja i s nizom laboratorijskih parametara karakterističnih za ovu skupinu bolesnika. Cilj nam je bio naći moguće uzročne čimbenike odgovorne za njegov nastanak. Povišen prag sluha (PS), iznad 20 dB, za sve frekvencije (prosjek 26±10,50 dB) imalo je 42, za frekvencije u govornom području (prosjek 19,70±8,80 dB) 22, a u području visokih frekvencija (41,70±19,70 dB) 56 bolesnika. Nađena je značajna pozitivna korelacija PS samo s dobi bolesnika ($r=0,49$, $p<0,01$). HD bolesnici, stariji od 45, odnosno od 60 imali su od bolesnika mlađih od 45, odnosno od 60 godina značajno više prosječne vrijednosti PS. Prema χ^2-testu među HD bolesnicima starijim od 45, odnosno od 60 godina značajno češće su bolesnici s patološkim PS. Prosječne vrijednosti PS u podskupinama bolesnika određenim prema duljini trajanja HD liječenja (A: do 60 mjeseci, B: 60–120 mjeseci, C: dulje od 120 mjeseci) nisu se značajno razlikovale. U 3 navedene podskupine bolesnika nije bilo značajno češće onih s patološkim PS, a podskupine se nisu razlikovale ni po prosječnim vrijednostima njihove dobi (A: 50,30±13,20 godina, B: 51,40± 12,75 godina, C: 55,80±10,55 godina). U zaključku možemo, na osnovi vlastitih i rezultata drugih ispitivača reći da su SNOS HD bolesnika vrlo česta i da su posljedica niza čimbenika. Točan udio bolesnika sa SNOS nepoznate etiologije nemoguće je odrediti. Vjerojatno ne nastaju samo zbog uremijske polineuropatije i/ili prerane vaskularne starosti iako je vjerojatno da njima pripadaju vodeće uloge.