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Full Length Research Paper

Clinical and electrophysiological correlation of patients with chronic renal failure: The contributions of quantitative neurological scores

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Neuropathy is the most common neurological consequence of uremia and has the scarce symptoms and definition. Therefore the sensitivity/specificity of clinical scores the neuropathy symptom score (NSS) and the neuropathy disability score (NDS) were evaluated for uremic neuropathy in the present study. 38 hemodialysis patients (23 males, 15 females) and 15 age-sex matched healthy subjects were enrolled. Neurological interrogation and examination of the subjects has been performed before neurophysiological examinations. After the usual 2 days interval in dialysis, electrophysiological studies (EPS) have been performed. Before the EPS, blood samples has been taken before a midweek dialysis; hemoglobin, Hct, and albumin concentrations were measured. The Kt/V value was taken as the average of previous 12 sessions Kt/V values. The mean NSS was 1.66 ± 2.2, mean NDS was 5.02 ± 6.9 in the patient group. According to EPS, 25 patients (65.8%) were diagnosed as having neuropathy [Np (+)] and 13 (34.2%) were normal [Np (-)]. The mean values of median, sural nerve sensory, common peroneal and posterior tibial nerve motor conduction velocities were lower in the patient group compared to controls. Np (+) patients was older than that of Np (-) subjects (50.1 ± 13.8 versus 36 ± 13.6; p = 0.006). Age was the only significant predictor of neuropathy (OR = 1.08, 95% CI, 1.017 to 1.150; p = 0.013). Logistic regression analysis revealed that both NSS (OR = 2.651, 95% CI, 1.1 to 6.4; p = 0.03) and NDS (OR = 1.26, 95% CI, 1.001 to 1.6; p = 0.049) were significantly associated with increased risk of neuropathy. The current study showed that both NSS and NDS are sensitive and specific in the diagnosis of uremic neuropathy and could be used at least as a first step before turn towards the elecrophysiologic studies.

Key words: Uremic neuropathy, neuropathy symptom score, neuropathy disability score, electrophysiological studies, risk prediction.

INTRODUCTION

Neuropathy is the most common neurological consequence of uremia, occurring in at least 60% of patients who begin dialysis for chronic renal failure (Raskin, 2001). The prevalence of clinical signs of polyneuropathy in patients undergoing hemodialysis has ranged between 10 and 83%, with larger series in the range of 50 to 60% (Bolton and Young, 1990). Using electrophysiologic studies as a more sensitive index, the prevalence of polyneuropathy has been somewhat higher, ranging from 57 to 100% (Dyck et al., 1995). Symptoms of uremic polyneuropathy are restless legs, cramps, weakness, paresthesias, dysesthesia, pain, and burning feet (Nielsen,

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1971). The earliest signs of neuropathy are impaired vibratory sensation in the lower limb and loss of tendon reflexes, first the Achilles and then the patellar responses. Other signs are muscle atrophy, weakness and hypoesthesia. Sensory loss develops two-point discrimination, position sensation and light touch, pain and temperature sensation (Tyler, 1968; Bolton, 1976).

In routine clinical practice, the scarce symptoms definition and recording cause some problems in the defining and following process of the subjects. The quantitative scoring system for neuropathy symptoms and also examinations can be more beneficial in this aspect.

The electrophysiologic features of uremic neuropathy include prolonged distal motor latencies, conduction velocity slowing, and declines in the amplitudes of compound muscle action potentials (CMAPs) and sensory nerve action potential amplitude (SNAPs) (Bolton and Young, 1990; Nielsen, 1974). Although there is lot of electrophysiological study of uremic neuropathy, optimum methodological process and the real contributions of long latency reflexes (that is, F response, H reflexes) are also unclear (Ackil et al., 1981; Panayitopoulos et al., 1977). Slowed nerve conduction velocity (especially sural nerve in the early phase) is frequent in uremic patients without other symptoms or signs of neuropathy. Although there are some suggestive data about the contributions of late responses in the process, the correlation between motor nerve conduction studies and late responses are not clearly reported (Ackil et al., 1981).

In spite of some studies suggested, a negative correlation existed between serum creatinine elevation and decreased velocity of motor nerve conduction (Jebsen et al., 1967; Nielsen, 1973; Ackil et al., 1981). There is also conflicting evidence in the relationship between the severity of neuropathy and age, gender, biochemical variables and type of renal disease (Dyck et al., 1995). The neuropathy generally evolves over several months but on occasion follows a fulminate course (Ropper, 1973). The factors that determine these differences in the clinical course of the neuropathy and the optimum method for the follow-up process are unclear.

Peripheral neuropathy has a high prevalence in diabetic patients also (Dyck et al., 1993). Objective and quantified measures of diabetic neuropathy are recommended in the follow up of disease and for epidemiological studies or therapeutic trials (Diabetes Care, 1992). Clinical scores have been developed and validated to quantify the severity of neuropathy such as the neuropathy symptom score (NSS) and the neuropathy disability score (NDS) in patients with diabetic neuropathy (Dyck, 1988, 1985). Significant associations between both NSS and NDS and some individual variables in nerve conduction studies were reported in diabetic neuropathy (Dyck, 1985). A combined index of polyneuropathy derived from nerve conduction studies was found to be well correlated with both NSS and NDS in diabetic sub- jects also (Feki and Lefaucheur, 2001). However clinical applicability and reliability of those clinical scoring systems in uraemic patients is not extensively studied.

In the present work, our aim was to investigate peripheral nervous system abnormalities in the light of clinical scoring systems and broad electrodiagnostic surveys, to correlate demographic, laboratory and clinical data with the objective and subjective determinants of neuropathy in hemodialysis patients. Furthermore, we investigated the contributions of quantitative neurological scoring systems (NSS and NDS) when neuro-physiological diagnosis is accepted as the gold standard.

PATIENTS AND METHODS

Patients and clinical evaluation

We studied 38 patients undergoing chronic maintenance hemodialysis treatment two or three times weekly, for 3 to 5 h per session. The mean length of hemodialysis treatment before the study protocol was 4.5 ± 2.3 years (range: 1 to 11 years). The etiology of uremia was as follows; atherosclerosis in 12 patients (31.6%), polycystic renal disease, interstitial nephritis or glomerulonephritis in 9 patients (23.7%), obstructive nephropathies in 4 patients (10.5%) and other etiologies in 5 patients (13.2%). The real etiology of renal failure in 8 patients (21%) was not known. All the patients were taking medications including calcium containing phosphate binders, a standard B+C vitamin complex pill after dialysis session, oral or intravenous iron supplementation and erythropoietin alpha/beta 4000 to 5000 IU subcutaneous 1 to 3 times in a week, to stabilize hemoglobin levels > 10.5 gr/dl. Patients with other possible causes of neuropathy such as diabetes mellitus, alcoholism, amyloidosis, or other systemic causes were excluded from the study. All patients gave written informed consent for the study protocol and detailed explanation has been made according to Helsinki declaration.

Neurological interrogation and examination of the subjects has been performed before neurophysiological examinations. After the usual 2 days interval in dialysis, electrophysiological measurements have been performed. Before the electrophysiological investigations, blood samples have been taken before (fasting) a midweek dialysis session; hemoglobin, hematocrit, and albumin concentrations were measured using appropriate biochemical methods. The Kt/V is defined as dialyzer urea clearance (K), multiplied by the dialysis session length (t), divided by the urea distribution volume (V) (Basile et al., 1990). Kt/V value was taken as the average of previous 4 weeks (12 sessions) Kt/V values per session.

The major symptoms encountered in peripheral neuropathy are listed in the neurological symptom score (NSS) developed by Dyck et al. (1999) specifically for diabetic neuropathy; where a total of 18 points. (1) Symptoms of muscle weakness (bulbar/limbs), (2) sensory disturbance [(negative/positive symptoms), autonomic symptoms] are questioned and the presence of each symptom is considered as 1 point to reach a maximum total 18 points. Neurological impairments of the subjects has been calculated using neuropathy impairment score (NIS) developed by Dyck et al. (1995) after the neurological disability score for the specific follow-up of neuropathy patients. Where neuropathic deficits for cranial nerves (3rd nerve, 6th nerve, facial weakness, palate weakness, and tongue weakness) were examined and scored as 1 point for each of them. Muscle weakness is scored: normal = 0; 25% weak = 1; 50% weak = 2; 75% weak = 3; and paralyzed = 4 for 18 separate joints handled by the different muscle groups. Respiratory muscle weakness is scored as present = 1, absent = 0. Reflexes were graded as normal = 0, decreased = 1, or absent = 2 (Nielsen et al, 1972).

Touch-pressure, pinprick and vibration are assessed on the dorsal surface of the terminal phalanx of index finger and great toe

Table 1. Demographic characteristics of patient and control groups.

Parameter	Patient (n = 38)	Control (n = 15)	p
Age	45.7±15.1	41±12	NS
Sex	23M/15F	11M/4F	0.04
Time on dialysis	4.5±2.3	-	-
BUN	96.4±29.1	-	-
Creatinine	4.1±1.7	-	-
Albumine	3.8±0.96	-	-
NSS	1.7±2.2	-	-
NDS- total	20±5.2	-	-
Neuropathy (+/-)	26(68.4%)/12(31.6%)	- /15	0.000

and were graded as normal = 0, decreased = 1, or absent = 2. Right and left extremities are scored separately and total score were calculated as the sum of the right and left sides, the highest possible score being 208. This score has been recommended to use extensively in medical practice simply to worsening or improvement of neuropathies. Total score has been calculated for each subject and used in the statistical analysis.

Nerve conduction studies

The whole neurophysiologic measurements were done using Medelec Synergy EMG Equipment (Medelec-Oxford, England) and appropriate analysis programs. The temperature of the extremities kept at least 29°C. If they were cooler, the limbs were warmed. The motor and sensory nerve conduction studies have been performed from each extremity of the subjects except if the extremity has arteriovenous (AV) fistula for hemodialysis. The motor nerve conduction velocity, distal motor latency, and CMAP amplitude of the median, ulnar (one side), peroneal and posterior tibial nerves (two side) were measured. The F-wave minimal, mean and maximal latencies and chronodispersion of F-waves were studied after 10 supramaximal stimuli. The sensory nerve conduction velocity and SNAP of median, ulnar (one side) and sural (two side) nerves were measured. The exact details of the methods for each nerve have been described elsewhere (Flack B-1991, EMG yöntem kitabı). Electrophysiological values of patients were compared with those of control group. Values exceeding the mean ± 3 standard deviation (SD) of the control group were considered as abnormal.

Statistical analysis

The results of descriptive analyses were tested and found to show normal distribution, thus data were given as the means and standard deviations. Parametric data were compared using unpaired t test and nonparametric data were compared using the χ^2 or Fisher exact tests. The correlations between the neuropathy, quantitative neurological scoring and the biochemical or demographic variables were determined using appropriate statistics. Binary logistic regression analysis was used to determine the predictive factors including NSS and NDS for neuropathy development.

The sensitivity, specificity, and positive predictive value (PPV) were calculated for NSS and NDS using ROC-curve analysis and the electrophysiological neuropathy diagnosis as the gold standard. Sensitivity here refers to the proportion of patients with these NSS or NDS abnormality who meet the neuropathy diagnosis. Specificity refers to the proportion of patients who do not have these NSS or NDS abnormality and who did not meet the neuropathy diagnosis. PPV refers to the proportion of patients with the NSS or NDS abnormalities who actually have the disease. Significant differences (two-tailed p) less than 0.05 were regarded as significant. Neural network analysis was performed to determine the sensitivity, specificity and the cut off values of NDS and NSS for differentiation of polyneuropathy types including motor or sensory or mixed polyneuropathy. Radial basis function was used for the classification of neuropathy types according to NDS score due to having better performance for this data set than the other models.

RESULTS

The mean age of our patients was 44.4 ± 14.3 years (from 21 to 75 years) and males were predominating (61%, 23 patients). Fifteen volunteers have been interviewed as control subjects. Similar to the patient group, males were predominating (73%, 11 subjects) in the control group. The mean age of the control subjects was 41.1 ± 12.02 years (from 26 to 67 years). According to selection criteria, groups were comparable for age and gender. Demographic features of the study and control groups have been shown in Table 1.

The mean NSS score was 1.66 ± 2.2 (0 to 8) and the mean NDS score was 5.02 ± 6.9 (0 to 20). There was a strong positive correlation between NSS and NDS results ($r = 0.844$; $p = 0.001$). NSS and NDS examinations revealed superficial sensory loss in 4 patients (10.5%), deep sensory loss in 7 patients (18.4%), abnormality in deep tendon reflexes in 9 patients (23.7%), trophic defect in 3 patients (7.8%) and autonomic defect in 1 patient (2.6%). Electropyhsiologic study (EPS) results showed that the mean values of median and sural nerve sensory, common peroneal and posterior tibial nerve motor conduction velocities were lower in the patient group compared to controls. However, ulnar sensory and motor conduction velocities were similar between patient and control groups.

Median motor distal latency was longer in patients compared to control subjects. Except for posterior tibial nerve, all motor nerve amplitudes studied were shown to be decreased in patient group compared to controls. Minimum F wave latencies and F wave chronodispersion values were not different between patient and control groups (Table 2) while the EPS results of each of the

Table 2. Comparison of the values in nerve conduction studies between patient and control groups.

Parameter	Patient (n = 38)	Control (n = 15)	p
Median sensory conduction velocity	52.1±5.5	56.2±3.8	0.011
Ulnar sensory conduction velocity	52.6±4.8	54.3±2.7	0.113
Sensory distal latency difference of median and ulnar nerves recorded IVth digit	0.3±0.02	0.2±1.12	0.02
Median motor distal latency	3.9±0.6	3.4±0.3	0.000
Median motor conduction velocity	54.2±4.5	54.2±4.5	0.985
Median motor amplitude	7.0±3.2	9.6±3.5	0.014
Ulnar motor conduction velocity	57.0±4.9	57.8±6.1	0.619
Ulnar motor amplitude	8.3±2.2	10.2±1.7	0.005
Common peroneal motor conduction velocity	40.8±7.2	46.7±3.4	0.004
Common peroneal motor amplitude	3.2±2.0	4.6±1.7	0.02
Posterior tibial motor conduction velocity	37.3±4.2	43.6±2.8	0.000
Posterior tibial motor amplitude	5.7±2.5	7.3±2.6	0.051
Sural sensory conduction velocity	32.5±18.7	44.4±4.3	0.001
Median F min	27.5±2.5	26.3±1.6	0.08
Median F chronodispersion	2.3±1.0	2.7±1.4	0.323
Ulnar F min	28.7±3.3	27.9±2.2	0.411
Ulnar F chronodispersion	1.7±0.9	1.9±0.8	0.47
Tibial F min	52.2±9.0	48.2±4.7	0.11
Tibial F chronodispersion	3.4±1.9	3.1±1.3	0.663

patients compared with the mean values of normal controls separately; 3 patients (7.8%) showed a decrease in median nerve conduction velocity, 7 patients (18.4%) showed a decrease in sural nerve conduction velocity, 3 patients (7.8%) had decreased peroneal BKAP amplitudes, 3 patients (7.8%) had decreased common peroneal conduction velocity, 9 patients (23.7%) had decreased tibial nerve conduction velocity, 11 patients (28.9%) showed an extended median nerve F wave latency. Ulnar F wave latency was extended in 11 (28.9%) and tibial F wave latency was extended in 12 (31.6%) patients.

According to those results, 25 patients (65.8%) were diagnosed as having neuropathy [Np (+)] and 13 (34.2%) were normal [Np (-)]. Among the Np (+) patients, 5 patients (20%) had sensory neuropathy, 1 patient (4%) had motor neuropathy and 19 patients (76%) had mixed neuropathy. Entrapment of the median nerve in carpal tunnel (Carpal Tunnel Syndrome) was observed in 11 patients (28.9%). Np (+) patients was older than that of Np (-) subjects (50.1 ± 13.8 versus 36 ± 13.6; p = 0.006). Haemoglobin, hematocrit (%), serum albumin, kt/V and time on dialysis values were similar between Np (+) and Np (-) patients. Np (+) patients had significantly higher NSS and NDS scores than that of Np (-) subjects (Table 3). Logistic regression analysis revealed that both NSS (OR = 2.651, 95% CI, 1.1 to 6.4: p = 0.03) and NDS (OR = 1.26, 95% CI, 1.001 to 1.6; p = 0.049) were significantly associated with risk of neuropathy. The determinative values for the occurrence neuropathy in NSS and NDS were as follows: for NSS = 0.5 [Se = 0.654, Sp = 0.667; p = 0.014, positive predictive value (ppv): 81%, negative predictive value (npv): 47.1%], for NDS = 1.5 (Se = 0.538, Sp = 0.833; p = 0.033, ppv = 87.5%, npv = 45.5%), (Figure 1). A logistic regression analysis performed on the possible predictive factors (age, time on dialysis, Kt/V, hemoglobin, albumin for neuropathy showed that

Table 3. Comparison of demographic features, laboratory analyses, NSS and NDS scores between Np (+) and Np (-) patients.

Parameter	Np (+)	Np (-)	p
Age	50.1±13.8	36 ±13.6	0.006
Sex (male/female)	16/9	8/5	NS
Time on dialysis	4.6±2.4	4.3±2.1	NS
Hb	8.3±1.4	7.8±1.3	NS
Hct	28.2±1.8	27.3±3.7	NS
Albumin	3.6±0.9	4.1±0.9	NS
Kt/v	1.27±0.04	1.28± 0.05	NS
NSS	2.3±2.2	0.3±0.5	0.008
NDS	6.9±7.6	0.8±1.9	0.009

NSS: neural symptom score, NDS: neural disability score, Np: neuropathy.

Table 4. Classification of patients with different neuropathy types using NDS (neural network analysis).

Parameter	Classification			
	Mixed PNP	Absence of PNP	Motor PNP	Sensory PNP
Total	19.0	13.0	1.0	5.0
Correct	11.0	13.0	0.0	0.0
Wrong	8.0	0.0	1.0	5.0
Unknown	0.0	0.0	0.0	0.0
Correct (%)	57.9	100.0	0.0	0.0
Wrong (%)	42.1	0.0	100.0	100.0
Unknown (%)	0.0	0.0	0.0	0.0

the age was only a significant predictor of neuropathy (OR: 1.08, 95% CI, 1.017 to 1.150; p = 0.013).

Neural network analysis revealed that the NDS was the most sensitive test for discrimination of neuropathy types sensory, motor or mixed. However, NSS was neither sensitive nor specific for the classification of neuropathy types. Radial basis function was used for the score due to having better performance for this data set classification of neuropathy types according to NDS than the other models. 58% of the patients with mixed type polyneuropathy and 100% of Np (-) patients have been accurately diagnosed using NDS. However, patients with motor or sensory polyneuropathy were not correctly diagnosed due to low patient numbers in those groups (Table 4). Increase in NDS score led to increase in the probability of having mixed type polyneuropathy. Patients with a NDS > 12 had 0.75 probability of having mixed type polyneuropathy. Patients with NDS < 7 had a probability between 0.5 to 0.7 to be np (-). The probability of being np (-) progressively decreased in patients with NDS > 7 (Figure 2).

DISCUSSION

The present study showed a high prevalence of uremic neuropathy (65.8%) according to EPS results that was in line with the reports in literature (Zochodne, 2005). Male predominance of our patient group may contribute to this high prevalence also (Galassi et al., 1998). Neuropathic patients were older than non-neuropathic subjects. Likewise, Bazzi et al. (1991) reported a more severe electrophysiologic impairment of ulnar and sural nerves in older hemodialysis patients compared to younger ones. They reported a more severe neurologic impairment in patients with longest duration of dialysis (more than 10 years).

In present study, duration of dialysis, weekly Kt/v, hemoglobin, hematocrit and albumin values were not different between neuropathic and non-neuropathic subjects. However, in our patient group, mean duration of dialysis was shorter (4.5 ± 2.3 years) compared to their results. Laaksonen et al. (2002) could not find an association between occurrence of neuropathy and duration or efficiency of dialysis also. Uremic neuropathy can affect motor, sensory, autonomic and cranial nerves (Galassi et al., 1998); however it clinically presents a symmetrical distal sensory loss for all modalities which is more pronounced in lower extremities (Raskin, 2001).

In the current study, the most prevalent neuropathy type was mixed (sensory + motor) polyneuropathy (76%), followed by isolated sensory (20%) and isolated motor

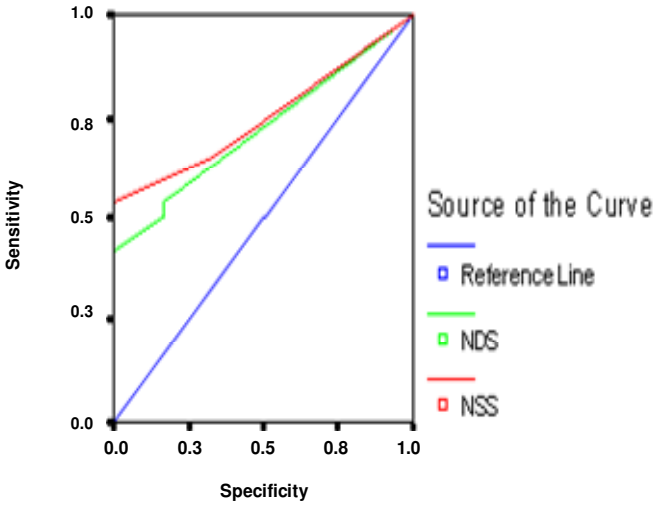

Figure 1. ROC-Curve analysis showing sensitivity and specificity of NSS and NDS in electrophysiologic diagnosis of neuropathy in uremic patients.

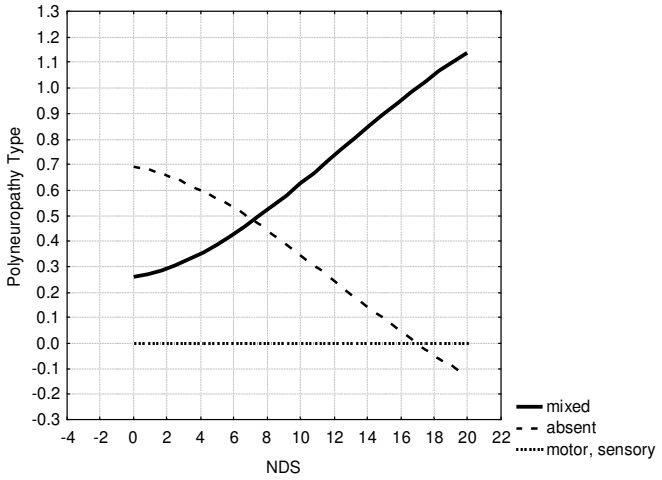

Figure 2. Confidence response graph showing the determinative effect of NDS on mixed type polyneuropathy and the absence of neuropathy.

neuropathies (4%). Although we did not perform an objective test for autonomic neuropathy, NSS results determined autonomic defect in 1 patient (2.6%). There is no strict definition for a predominant type of neuropathy in uremic patients. However, the pathological state uremic neuropathy is a multiple neuropathy due to axonal degeneration of the sensory and motor nerves, starting from the lower extremities with secondary development of demyelination (Thomas et al., 1971). Furthermore, as a support to our findings, previous studies based on either EPS results or quantitative neurologic scoring systems revealed the synchronous presentation of sensory and motor neuropathies in uremic subjects (Laaksonen et al., 2002; Ogura et al., 2001).

Original description of uremic neuropathy were made by Marin and Tyler (1961) as subacute weakness, distal sensory loss to pinprick, cold, light touch, vibration and position and loss of deep tendon reflexes. The electrophysiologic features of uremic neuropathy include prolonged distal motor latencies, conduction velocity slowing and declines in the amplitudes of CMAPs and SNAPs (Bolton and Young, 1990; Nielsen, 1974). Sural nerve conduction slowing is suggested to be most sensitive electrophysiologic parameter of early polyneuropathy (Ackil et al., 1981). In agreement with those previous findings, our results revealed significant slowing of sural sensorial conduction velocities in patient group compared to healthy controls.

Slowing in common peroneal and tibial motor conduction velocities, prolongation of median motor distal latencies and decrease in motor amplitudes except posterior tibial nerve were noticed in patient group compared to controls. Although slowing in median sensorial conduction velocity is observed, median motor, ulnar motor and sensorial conduction velocities were not seemed to be affected. That may be due to entrapment of the median nerve in carpal tunnel (Carpal Tunnel Syndrome/CTS) which was observed in 11 patients (28.9%). Significant difference in sensory distal latencies of median and ulnar nerves recorded IVth digit was a further evidence for important entrapment of median nerve in carpal tunnel. It is well known that hemodialysis patients are prone to compression of median nerve in carpal tunnel secondary to dialysis associated amyloidosis or uremic tumoral calcinosis (Gejyo et al., 1997; Cofan et al., 2002). So, the abnormalities seen in the median nerve recordings were probably not caused by the polyneuropathy alone. The latencies of ‘’ long loop’’ F waves and H reflexes are prolonged in uremic neuropathy (Panayitopoulos, 1980, 1977).

Laaksonen et al. (2002) showed that the F wave latencies of the lower extremity nerves were by far the single most sensitive neurophysiologic parameter in detection of uremic neuropathy. Although comparison of mean F wave recordings were not different between our patient and control groups, comparison of the patients individually with the mean values of control group revealed prolongation of F wave latencies in median (11 patients; 28.9%), ulnar (11 patients; 28.9%), and tibial (12 patients; 31.6%) nerve recordings. Similar F wave latency values between patient and control groups may be caused by the low patient number in our study.

The current study is mainly conducted to evaluate the predictive roles of subjective neurologic symptom (NSS) and neurologic disability scores (NDS) which are mainly validated for diabetic neuropathy (Dyck, 1988) on EPS results as a gold standard in the diagnosis of uremic neuropathy. Both NSS and NDS in our patient group were higher than that of controls. Furthermore, both NSS and NDS were found to be sensitive and specific tests in de-

termination of uremic neuropathy. Previously, vibration perception threshold (vibrameter testing) was shown to be a valid and valuable, simple, quick and non-invasive method for evaluation of severity in peripheral uremic neuropathy by different authors (Klima et al., 1991). Krishnan et al. (2006) reported a significant correlation between total NSS and both pre-dialysis subexcitability and depolarizing threshold electrotonus.

Significant correlations between different neurophysiologic records (sural conduction velocity and amplitude, tibial conduction velocity and amplitude, tibial F wave latency) and related local symptoms were reported also (Laaksonen et al., 2002). However to our knowledge till date, no study evaluated the sensitivity and specificity of NSS and NDS in diagnosis of occurrence or typing of uremic neuropathy. The present study showed that both NSS and NDS were sensitive and specific tests in diagnosis of uremic neuropathy. Our results demonstrated that NDS may also predict the occurrence of mixed type polyneuropathy in hemodialysis patients. Because of the low patient numbers in motor and sensory neuropathy groups, we could not make a comment about the predictive role of NDS on these neuropathy types.

Conclusion

Uremic polyneuropathy is probably the most common complication of chronic renal failure and affect the significant proportion of the hemodialysis patients. To date, there is no universally accepted approach to the diagnosis and the management of neuropathy in hemodialysis patients. Although EPS is the gold standard for the diagnosis of neuropathy, it is time consuming and not feasible for all the patients. Both NSS and NDS are simple, quick and practicable tests and could be applicable to hemodialysis patients with regular intervals. The current study demonstrated that both NSS and NDS are sensitive and specific in the diagnosis of uremic neuropathy and could be used at least as a first step before turning towards the elecrophysiologic studies.

ABBREVIATIONS

OR, Odds ratio; **EPS,** electropyhsiologic study; **Se**, sensitivity; **Sp,** specificity; **ROC**, receiver operating characteristic; **BUN**, blood urea nitrogen; **NSS**, neuropathy symptom scale**; NDS**, neurological disability scale.

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