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ORIGINAL ARTICLE

Prevalence And Predictors of Peripheral Neuropathy in Children with End Stage Renal Disease on Regular Hemodialysis.

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ABSTRACT

Background: One of the heterogeneous chronic diseases affecting the kidneys is chronic kidney disease (CKD). Patients with chronic kidney disease, which is associated with high morbidity and mortality, are more likely to experience nervous system problems.

Objective: This work was performed to estimate the prevalence and predictors of peripheral neuropathy in children with End Stage Renal Disease (ESRD) on hemodialysis.

Patient and methods: A total of 80 children from Egypt were recruited and divided into 2 separate groups. As a first step, a case group of 40 children (5-18 years old) with ESRD who were receiving regular hemodialysis was established. Besides the control group of 40 healthy children matched by age and gender to the case group. We subjected all the participants to detailed history taking stressing on duration of dialysis, family history of renal diseases, consanguinity and history of neuromuscular symptoms such as distal paresthesia, or muscle weakness. General examination for weight, height, blood pressure, vital signs, systemic examination including: Heart examination, chest, abdominal and neurological examination. Routine laboratory investigations and nerve conduction studies were done.

Results: We found a statistically significant difference in weight and height between the two studied groups. 40% of patients had polyneuropathy. Multivariate analysis showed that longer disease duration (7 years), high parathyroid hormone level and high ferritin were independent predictors of polyneuropathy.

Conclusion:
Peripheral neuropathy is a common complication among children with end stage renal disease. Longer dialysis duration, high ferritin and parathormone levels were considered strong predictors of polyneuropathy among CKD patients.

Key words: Peripheral neuropathy, Chronic Kidney Disease (CKD), Regular Hemodialysis.

INTRODUCTION

With a frequency of fifteen percent in industrialized societies, chronic kidney disease (CKD) is a global health issue that is rapidly expanding. From minor kidney impairment, with no symptoms and only detectable by blood and urine tests, to end-stage disease, in which kidney function has been damaged so severely that the retention of metabolic waste products, salt, and water can be fatal [1].

An epidemic of chronic kidney disease (CKD) threatens to break out over the world due to its increasing incidence and prevalence [1]. It is defined as chronic kidney disease if there is structural or functional kidney impairment, or having less than 60 ml/min/1.73 m^2 glomerular filtration rate for more than three months [2]. Nephrotic syndrome, glomerular disease and other congenital kidney and urinary tract defects are the primary causes of ESRD in children[3].

Neurological deficits associated with kidney diseases could contribute to many comorbidities, one of the most common is uremic neuropathy. Some consequences are related to the renal disease itself, while others are subsequent to dialysis. Central and peripheral nervous systems are affected by these diseases [4]. Childhood CKD

has been more common in the past two decades, among the neurologic disorders that reported in children with CKD are sensorimotor polyneuropathy which is considered a common complication of CKD and called uremic neuropathy [5]. This type of neuropathy affects 60–90% of patients with CKD [5]. Glomerular filtration rate (GFR) below 12 mL/min were considered to cause uremic neuropathy. However, recent studies have shown that up to 70% of pre-dialysis patients suffer from neuropathy [6]. Pathophysiology of uremic neuropathy has yet to be identified, but there are several theories. In spite of this, there are two primary hypotheses. The first one is 'Middle Molecule Hypothesis'. Uremic neuropathy was thought to be the result of a build-up of neurotoxic substances (urea, creatinine, parathyroid hormone (PTH), "middle molecules," and others have been correlated with reduction of Nerve Conduction Velocity (NCV) and peripheral manifestations of neuropathy [5]. Hyperkalemia and hyperphosphatemia generate persistent uremic depolarization of neurons and contribute to the development of Uremic neuropathy, according to the second hypothesis [7]

Diagnosis of peripheral neuropathy is difficult and requires a variety of tests. A thorough clinical evaluation, including a history and neurologic examination, as well as laboratory tests and electro diagnostic investigations or nerve biopsy is essential for detection and proper management. When it comes to assessing peripheral nerve function, electro diagnostic testing such asymptomatic patients could be discovered by sensitive tests like nerve conduction and electromyography [8].

We aimed in this work to estimate prevalence of peripheral neuropathy in ESRD children on hemodialysis and its predictors.

PATIENTS AND METHODS

Cross-sectional research was conducted in this study that contained total of 80 Egyptian children were recruited and divided into 2 separate groups, 1st one was case group which included 40 children with ESRD on regular hemodialysis (21 male and 19 female), their ages range from 5 to 18 years, after exclusion of children with diabetes mellitus, children with family history of neuro muscular diseases, children with history of ICU admission for more than 2 weeks in the 6 months prior to the study, clinical evidence of spinal dysraphism, and those on medications known to cause peripheral neuropathy. Besides the control group of 40 healthy children matched by age and gender to the case group. They were apparently healthy with no history of chronic illness and did not receive any medications. This study was conducted at the Zagazig university hospitals nephrology Unit of pediatric department and neurology department from March 2019 until November 2020.

Ethical approval: Institutional Review Board (IRB) approval was obtained from Zagazig University. child's parents provided written consent before study initiation. According to (Declaration of Heliniski), the ethical code of the world medical association, we conducted the study of human subjects.

In all of the groups the following was done: History taking including Age, sex, cause of ESRD, duration of dialysis, family history of renal diseases, consanguinity and history of neuro muscular symptoms. Clinical examination including General examination for; Weight, height, blood pressure, vital signs, in addition to systemic examination of heart, chest, abdominal and finally neurological examination was done.

Routine laboratory investigations were done including**,** CBC (2 ml blood on EDETA for CBC using sysmex –XS 500- Germany)**,** Kidney function for BUN and serum creatinine by automated auto-analyzer COBAS 8000**,** Liver function test**,** C-Reactive protein (CRP) by automated –analyzer COBAS c 501**,** Iron indices (ferritin, iron)**,** PT, PTT, INR**,** Serum parathormone level and finally Serum electrolytes including sodium, potassium, calcium, and magnesium.

Nerve conduction studies were done in the neurology outpatient clinic using the Micro med machine (Italy). For motor nerve conduction study: Median, ulnar, common peroneal and tibial nerves were selected , For sensory nerve conduction study : median and sural nerves were selected . Motor conduction studies were carried out with surface disk electrodes, with the active electrode inserted on the muscles and supramaximal stimulation of the corresponding nerves. Sensory NCS were performed. The parameters were compared between patients and controls. Individual values of conduction velocities, latencies, or amplitude were considered abnormal when outside the mean±2 SD of controls. If two or more nerves had at least one abnormal parameter compared with the age-matched controls, it is electro physiologically considered peripheral neuropathy [9].

Statistical analysis

The data entered into the computer was analyzed. We used the 20th version of IBM SPSS software. IBM Corporation is based in Armonk, NY. When describing qualitative data, numbers and percentages were used. In order to determine if the distribution was normal, the Klmogoro-

Smirno test was utilized. In order to characterize quantitative data, we used the range (minimum and maximum), mean (average), stander deviation, median, and interquartile range (IQR). In order to determine the significance of the acquired results, a 5-percent threshold was used. It was a Chi-square test for categorical variables. Mann Whitney test was used when data is not normally distributed. P≤0.05 was considered statistically significant. P≤0.001 was considered statistically highly significant.

Binary logistic regression analysis is used to predict the odds of having PN based on the values of the independent variables (predictors) which were significant on univariate analysis..

RESULTS

Weight and height were both significantly higher in the control group. A non- significant difference was found between them in terms of gender or age as shown in (Table 1).

Latency, velocity and amplitude of peroneal nerve, posterior tibial (latency and velocity), median nerve (latency, velocity and amplitude) and ulnar nerve (latency, velocity and amplitude) showed a statistically significant difference between the two studied groups. Distal latency was higher among case group while velocity and amplitude were lower among case group. On the other hand, posterior tibial amplitude did not show any statistically significant difference between the groups (Table 2).

Latency, velocity and amplitude of sural nerve, posterior tibial (latency and velocity), median nerve (latency, velocity and amplitude) and ulnar nerve (latency, velocity and amplitude) showed a statistically significant difference between the two studied groups. Sensory latency was higher among case group while velocity and amplitude were lower among case group (Table 3).

The prevalence of Polyneuropathy among our patients was 40% (Table 4). Polyneuropathy was significantly related to long (>7 years) duration of dialysis (table 5). Risk factors for Polyneuropathy in our patients were high serum ferritin and parathormone levels (Table 6).

Multivariate analysis of factors associated with polyneuropathy among patients showed that dialysis duration for 7 years or more, high parathormone hormone level and high ferritin levels increased risk of polyneuropathy by 14.45, 18.427 and 15.047 folds respectively (Table 7).

Table 1. Comparison between the studied groups regarding demographic data:

Parameter	Group		Test	
	Case group N=40 (%)	Control group N=40 (%)	χ^2/t	*P*
Gender: Male Female	 21 (52.5) 19 (47.5)	 13 (32.5) 27 (67.5)	3.274	0.07
Age: Mean ± SD Range	 13.775 ± 3.655 5 – 18	 14.325 ± 3.979 4 – 18	-0.644	0.521
Weight: Mean ± SD Range	 34.64 ± 9.47 15.5 – 55	 52.28 ± 15.56 14 – 63	-6.122	<0.001**
Height: Mean ± SD Range	 143.93 ± 17.46 95 – 162	 153.23 ± 18.76 110 – 165	-2.295	0.024*

χ2: Chi square test, *significant (*P*≤0.05), **highly significant (*P*≤0.01)

Table 2. Motor nerve conduction studies in the studied groups:

Motor nerves	Group		Test	
	Case group Median (range)	Control group Median (range)	t/Z	*P*
Peroneal nerve latency	4.266 ± 1.25	3.276 ± 0.335	4.9	<0.001**
Peroneal nerve amplitude¥	4.5 (1.5 – 21.5)	9 (7 – 9)	-4.193	<0.001**
Peroneal nerve velocity	42.922 ± 4.068	56 ± 1.34	-19.555	<0.001**
Posterior tibial nerve latency	4.068 ± 1.219	3.483 ± 0.328	2.97	0.005*
Posterior tibial nerve amplitude¥	9.5 (1.8 – 27)	12 (9 – 12.5)	-0.75	0.453
Posterior tibial nerve velocity	43.207 ± 8.227	52.195 ± 3.219	-6.515	<0.001**
Median nerve latency	3.393 ± 0.652	2.759 ± 0.278	5.727	<0.001**

Median nerve amplitude¥	7.8 (2.7 – 16)	14 (12 – 16)	-3.891	<0.001**
Median nerve velocity	51.434 ± 2.847	57.171 ± 2.756	-9.27	<0.001**
Ulnar nerve latency	2.881 ± 0.751	2.566 ± 0.106	2.657	0.011*
Ulnar nerve amplitude	7.544 ± 1.802	14.354 ± 2.367	-14.565	<0.001**
Ulnar nerve velocity	51.163 ± 4.026	61.171 ± 2.407	-13.661	<0.001**

*significant *(P*≤0.05), **highly significant (*P*≤0.01)

Table 3. Sensory nerve conduction studies in the studied groups

Sensory nerves.	Group		Test	
	Case group Median (range)	Control group Median (range)	t/Z	*P*
Median nerve latency¥	3.3 (2.1 – 14.3)	2.3 (2 – 2.5)	-5.655	<0.001**
Median nerve amplitude¥	28.2 (11.46 – 75.4)	34 (30.1 – 40)	-3.097	0.002*
Median nerve velocity	45.85 ± 5.01	56.63 ± 3.02	-11.367	<0.001**
Sural nerve amplitude	7.23 ± 1.10	13.16± 0.53	-30.251	<0.001**
Sural nerve latency	3.571 ± 0.969	2.453 ± 0.134	7.295	<0.001**
Sural nerve velocity	39.459 ± 3.14	52 ± 1.32	-23.095	<0.001**

*significant (*P*≤0.05), **highly significant (*P*≤0.01)

Table 4. Comparison between the studied groups regarding presence of polyneuropathy by nerve conduction studies:

	Group		Test	
	Case group N=40 (%)	**Control group N=40 (%)**	χ^2	***P***
Polyneuropathy: Absent Present	 24 (60) 16 (40)	 40 (100) 0 (0)	Fisher	<0.001**

**highly significant (*P*≤0.01)

Table 5. Relation between presence of polyneuropathy and duration of dialysis:

Parameter	Polyneuropathy		Test	
	Present N=16 (%)	Absent N=24 (%)	χ^2	*P*
Duration of dialysis: 7 years ≤7 years	 10 (62.5) 6(37.5)	 6 (25) 18 (75)	5.625	0.018*

χ2: Chi square test, *significant ((*P*≤0.05)

Table 6. Relation between presence of polyneuropathy and laboratory indices:

	Polyneuropathy Present N=16 (%)	Absent N=24 (%)	Test χ^2	p
TLC: Leucopenia Normal	 5 (31.2) 11 (68.8)	 9 (37.5) 15 (62.5)	0.165	0.685
Hemoglobin: Normal Anemic	 13 (81.2) 3 (18.8)	 18 (75) 6 (25)	Fisher	0.717
Platelet count: Decreased Normal	 1 (6.2) 15 (93.8)	 1 (4.2) 23 (95.8)	Fisher	>0.999
Ferritin: Normal Increased	 11 (68.8) 5 (31.2)	 3 (12.5) 21 (87.5)	13.352	<0.001**

	Polyneuropathy Present N=16 (%)	Absent N=24 (%)	Test χ^2	p
Serum iron:				
Decreased	5 (31.2)	11 (45.8)	0.851	0.356
Normal	11 (68.8)	13 (54.2)		
Parathyroid hormone:				
Normal	1 (6.2)	9 (37.5)	Fisher	0.032*
Increased	15 (93.8)	16 (62.5)		
Calcium:				
Decreased	5 (31.2)	6 (25)		
Normal	10 (62.5)	15 (62.5)	0.081	0.775
Increased	1 (6.2)	3 (12.5)		
Phosphorus:				
Normal	7 (43.8)	8 (33.3)	0.444	0.505
Increased	9 (56.2)	16 (66.7)		
Sodium:				
Decreased	3 (18.8)	3 (12.5)	0.02	0.889
Normal	13 (81.2)	20 (83.3)		
Increased	0 (0)	1 (4.2)		
Potassium:				
Normal	7 (43.8)	7 (29.2)	0.897	0.343
Increased	9 (56.2)	17 (70.8)		

$\chi 2$: Chi square test, *significant ($P \leq 0.05$), **highly significant ($P \leq 0.01$)

Table 7. Multivariate analysis of factors significantly associated with polyneuropathy among patients:

	β	*P*	AOR	95% C.I.	
				Lower	Upper
Dialysis duration (≥ 7 years))	2.671	.015*	14.450	1.665	125.437
High parathyroid hormone	-2.914	.057	18.427	0.916	370.715
High ferritin	2.713	.006*	15.074	2.153	105.529

CI: confidence interval, AOR: adjusted odds ratio, *Significant ($P \leq 0.05$)

DISCUSSION

Uremic neuropathy is a common peripheral neuropathic disorder that affect chronic kidney disease patients. Diabetic Adults are mostly affected by Diabetic CKD as one of the major complications [10].This type of sensorimotor polyneuropathy which is more frequent in the lower limbs than the upper limbs, has an unexplained male predominance [11]. Accumulation of uremic toxins lead to uremic neuropathy because of harmful effect of oxidative stress which cause autonomic, motor and sensory nervous impairment. Hyperkalemia and hyperphosphatemia could contribute to uremic neuropathy (UN). When the usual ionic gradient is disturbed by potassium, axonal death follows [12]. Several researches reported that, excess serum potassium can be removed from the body in order to normalize nerve function when hyperkalemia is present [13].

Including nerve conduction investigations, which measure conduction velocity and amplitude, clinical neurological examination, is the gold standard for diagnosing neuropathy [8].

In the current study, 52.5 % of cases were males, 47.5 % were females, and their mean age was 13.775 ± 3.655, with non-significant difference between them regarding gender and age. There was statistically significant difference between the studied groups regarding weight and height. Control group had significantly higher weight and height, and this was in agreement with **Abd El Naby et al**. [14] who studied the neurological manifestation and complications of chronic kidney disease (CKD) in fifty people, 23 male and 27 female, with a mean age of 12.08+ 3.46 years, regardless of gender. In accordance with our conclusions, **Jumana et al.** [15]study included total of 68 children with ESRD were reviewed, mean age was 9 ± 5.6 years, 37 were females.

With regard to the frequency of polyneuropathy, Polyneuropathy was prevailed in 40% of our

patients which was approximate to the results of **Yoganathan et al.**[16] who demonstrated a prevalence of 52% in their study upon pediatric CKD . **Abd El Naby et al**. [14]study found that 22% of people had peripheral neuropathy. a scanty previous case series had shown prevalence of polyneuropathy extending from 0% to 59% according to **Ackil et al.** [17] and **Elzouki and collegues** [18]. On comparison to the results of previous studies conducted on adult population with CKD, **Chao and collegues** [10] demonstrated that 37.5 % of patients has peripheral neuropathy , and **Ezzeldin et al.** [19] in their study observed that 55% of the studied patients has clinically manifested polyneuropathy. Moreover, **Krishnan et al.** [5] reported that up to 90% of CKD patients develop polyneuropathy.

When it came to peroneal nerve, there was a statistically significant difference between both study groups regarding (latency, velocity and amplitude), posterior tibial (latency and velocity), median nerve (latency, velocity and amplitude) and ulnar nerve (latency, velocity and amplitude). Nerve latency was higher among case group while velocity and amplitude were lower among case group. On the other hand, the posterior tibial amplitude did not differ significantly between the two groups, while **Abd El Naby et al**. [14]study that included fifty CKD children, Eighty-nine percent of them showed normal electrophysiological studies. 18% of their patients had abnormal electro encephalopathy findings, with the majority having generalized and focal epileptogenic activity (temporal, occipital, and frontal). Both the ulnar and median nerves showed normal motor and sensory nerve conduction in their study, which was considered with **Yoganathan et al** [16]**.**

There were 81.8% axonal motor and sensory neuropathies, and 18.2% demyelinating motor neuropathy. Patients with tibial and common peroneal nerve damage had a reduced conduction velocity and amplifying effect on these nerves. While 69% of children on maintained dialysis had sural nerve conduction abnormalities according to **Ackil et al**. [17].

The association between polyneuropathy and ferritin and parathyroid hormone was statistically significant in this study, but there was statistically non-significant relation between polyneuropathy and either total leucocytic count, hemoglobin, platelet count, iron, serum, serum phosphorus, calcium, potassium or sodium. Albumin, eGFR and serum creatinine were significantly associated with peripheral neuropathy in a study by **Abd El Naby et al**. [14] with 100% sensitively and specificity (100, 97, and 85% respectively). Among patients with neuropathy, ferritin levels were high on average. Symptoms of systemic inflammation and renal failure can lead to elevated ferritin levels. It was found that oxidative stress increased intracellular ferritin, but the relationship between serum ferritin and oxidative stress is poorly understood [20]**.** There was a significant correlation between nerve excitability parameters and serum potassium in patients with chronic renal failure according to previous research] 10[. According to a study conducted by **Yoganathan and colleagues**. [16], age, serum albumin levels, GFR, serum copper levels and ferritin levels were all associated with peripheral neuropathy prediction with a sensitivity of 84.6 % and specificity of 69.5% ,respectively.

In the current study, we demonstrated that dialysis duration for 7 years or more, high parathyroid hormone level and high ferritin levels were significant determinants for neuropathy in multivariable logistic regression. . They increased risk of polyneuropathy by 14.45, 18.427 and 15.047 folds respectively.

In agreement with this study, **Ezzeldin et al**[19] showed that disease duration is considered a strong predictor of developing peripheral neuropathy, in contrary, **Laaksonen et al**[21] found no association between the duration of dialysis and the presence of polyneuropathy.

A study done in children with β-Thalassemia detected that patients with high ferritin are exposed to higher risk of peripheral neuropathy [22].

Yoganathan et al. [16] study showed that gender, when it came to significant risk factor, the presence of hypertension and CKD stages did not come into play. They found that duration of dialysis therapy did not appear to be related to the development of dialysis peripheral neuropathy, while found strong association with low serum copper levels and dialysis therapy. Serum copper was not measured in our study. Dialysis might help patients with CKD neurological complications, according to one theory. It has been suggested that peritoneal dialysis patients have a lower incidence of peripheral neuropathy than their hemodialysis counterparts, which could support "the middle molecule hypotheses" [16].

CONCLUSION

Peripheral neuropathy is a common complication among children with end stage renal disease. Longer dialysis duration, high ferritin and parathormone levels were considered strong predictors of polyneuropathy among CKD patients. Nerve conduction studies are simple, cost effective, and noninvasive techniques that enable the early diagnosis of polyneuropathy in

clinically asymptomatic patients. Early diagnosis and detection of polyneuropathy using the electrodiagnostic studies is very important to early management and prevention of long standing neurologic complication such as polyneuropathy.

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