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Motor Nerve Conduction Studies in Patients with Chronic Renal failure

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Abstract

Background: **Detection of frequency of neuropathy in patients with chronic renal failure by utilizing nerve conduction studies (NCS)**

Methods: **In this cross sectional comparative study, thirty patients with chronic renal failure, of at least three months duration were included. Patients were further segregated into two groups. In group I twenty patients on regular hemodialysis and in group II ten patients with end stage renal failure waiting for hemodialysis were included. Motor nerve conduction studies were done by testing median nerve and peroneal nerve, in upper and lower limbs respectively. Parameters of motor conduction studies checked were proximal and distal motor latencies, amplitudes and conduction velocities.**

Results: **In group I, 93% of patients and in group II, 87% of patients showed altered motor nerve conduction parameters.**

Conclusions **There is predominantly distal nerve dysfunction and peripheral nerves of lower limb are affected more than upper limb. Sub clinical neuropathies can be detected by nerve conduction studies. .**

Key Words: **Nerve conduction studies, peripheral neuropathy, chronic renal failure**

Introduction

Chronic Renal Failure (CRF) is a functional diagnosis characterized by progressive and irreversible decline in glomerular filtration rate (GFR).[1] It is characterized by an increasing inability to maintain normal low levels of products of protein metabolism (such as urea), normal blood pressure, hematocrit, sodium, water, potassium and acid base balance.Reduction in renal mass causes structural and functional hypertrophy of surviving neurons.[2]

CRF patients usually pass through four overlapping phases. These phases are decreased renal reserve, mild renal insufficiency, overt renal failure and end stage renal failure. Polyneuropathy has been recognized as the most common complication of end stage renal failure. Uraemic neuropathy presents as painless, progressive, symmetrical, sensorimotor polyneuropathy.There is segmental demyelination and axonal degeneration in peripheral nerves.[3]

A clinician faces two problems while managing patients with peripheral neuropathy. These are establishing the existence of disease of peripheral nervous system and ascertaining its nature. It is necessary to perform a number of procedures such as biochemical tests, CSF examination, needle examination of muscle, nerve muscle biopsy and electrophysiological studies. Out of all the above mentioned investigations, nerve conduction studies have been found to be the most sensitive detector of neuropathy. This non-invasive procedure provides a definite evidence of sub clinical neuropathy and often precedes either signs or symptoms of uraemia.[4]

Usual parameters of electrophysiological studies include latency, amplitude, and duration of conduction. Onset latency shows the conduction time of fastest fibers.It is increased in uraemia. Conduction velocity has remained one of the best measurements of peripheral nerve function. Amplitude of compound muscle action potential depends upon number of active firing axons. When toxin enters at endoneural space and causes axonal damage, this results in considerable reduction of amplitude. [5]

Uraemic patients need close monitoring. Nerve conduction studies fulfill this purpose. This neuropathy is difficult to revert , but hemodialysis, peritoneal dialysis and successful transplantation result in improvement of different neurological measures. [6,7]

Patients and Methods

A total of consecutive 30 patients fulfilling the criteria of CRF were selected by purposive sampling with 10 normal control subjects. Written informed consent was obtained from all subjects and controls. Study was conducted in Medical unit II, Sir Ganga Ram Hospital, Lahore. Uraemic patients of at least 3 months duration with glomerular filtration rate (GFR) <30ml/min were included in the study. Patients with diabetes mellitus, paraplegia, systemic lupus erythematosis, polyarteritis nodosa, alcoholism and drug induced neuropathies were excluded.

Their ages ranged from 25-42 years. Patients were segregated in two groups. In Group I twenty patients receiving regular hemodialysis were included. In Group II ten patients with end stage renal failure not dialyzed yet were included.

Motor Nerve Conduction Study: Conduction characteristics of motor nerve fibers were assessed by studying compound evoked potentials recorded from the muscles. Use of standard methods allows precise lesion localization and accurate characterization of peripheral nerve functions. Belly tendon method was used in this study [8]. Motor nerve conduction parameters of median nerve in upper limb and common peroneal nerve in lower limb were assessed.

Duration of stimulation was 0.5 to 1.0 ms. Rate of stimulation was 30 to 50/sec. Intensity was gradually increased to get maximum response. Then 20 to 30 percent further increase was done. This supramaximal stimulation ensures activation of all the nerve fibers. Evoked motor response is called a compound muscle action potential (CMAP).The stimulation was given at two points along the course of motor nerve. Following parameters of CMAP were measured.

Proximal motor latency(ms)
Distal motor latency (ms)
Amplitude peak to peak(mv)
Conduction velocity (m/s) = Distance (mm) between proximal and distal stimulating sites
Proximal latency – distal latency

Statistical Analysis:

The significance of differences between means of measurements of two groups was determined by student t test. Correlation between two groups parameters were sought with linear regression analysis by the least square method. A probability value <0.05 was considered statistically significant. Results were expressed as mean ± standard error of mean.

Results

In group I, abnormalities of motor conduction parameters were demonstrated in 93% of the patients. Median nerve proximal and distal motor latencies were prolonged in 79% of the patients. Amplitude of latencies was decreased in 86% of the patients. Conduction velocity was decreased in 80% of the patients (Table-1). Peroneal nerve proximal & distal motor latencies were prolonged in 78% of patients. Amplitudes were decreased in 87% of the patients. Conduction velocity was decreased in 82% of the patients (Table 2.)

In group II median nerve proximal and distal motor latencies were prolonged in 71% of patients. Amplitudes were decreased in 80% of the patients. Conduction velocity was decreased in 82% of the patients (Table 3).

Peroneal nerve proximal and distal motor latencies were prolonged in 78% of the patients in group II. Amplitude of latencies was decreased in 80% of the patients. Conduction velocity was decreased in 80% of the patients (Table 4).

Discussion

Uraemic neuropathy is a common complication of chronic renal failure. Usually it is distal, symmetrical mixed polyneuropathy or central to peripheral axonopathy.[9] The present study investigated excitability properties of upper & lower limb motor axons in CRF patients .Previous studies have demonstrated prevalence rates of neuropathy from 60 to 100% depending on the choice of nerve segment, the indices measured, and number of nerves studied. [10,11] The slowing of MNCV in upper & lower limb confirmed the previous observations of distal polyneuropathy. It is seen in many CRF patients, with end stage renal failure or those who undergo long term hemodialysis. [12]

The electrophysiological findings confirmed the result of previous study that peripheral nerves are more severely affected in lower limb than in upper limb.[13] While there was no clinical evidence of peripheral neuropathy in some patients motor nerve conduction studies disclosed definite evidence of sub clinical neuropathy as previous studies have demonstrated. [14] The rate of neuropathy in the present study was 93% in group 1 & 91% in group 2 in keeping with the previous studies of uraemic neuropathy which have demonstrated similarly high rates of neuropathy. There was significant decrease in amplitude of peroneal nerve CMAP. These electrophysiological findings are in partial agreement with those obtained by Arum.[2] The observations of predominantly distal nerve dysfunction in some CRF patients agrees with the findings of Makkar.[4]

There was no significant difference of conduction parameters in these two groups which provide a contrast to Arum[2] who found a clear improvement in excitability parameters following dialysis. These studies serve the purpose of detection of not only the presence and severity of peripheral nerve dysfunction but also its precise localization and pathophysiology.[15]

Table 1: Median Nerve Motor studies in upper limb in Group I

GROUPS	Proximal motor latency (ms)	Amplitude (mv)	Distal motor latency (ms)	Amplitude (mv)	Velocity (m/s)
Control					
Mean	3.2	13.7	8.3	13.2	53.17
SEM	0.1	0.2	0.2	0.4	2.2
p value	<0.01	<0.01	<0.01	<0.01	<0.01
Experimental					
Mean	4.9	8.9	11.6	8.4	39.14
SEM	0.009	0.23	0.18	0.13	0.9

Table 2: Peroneal Nerve Motor studies in lower limb in Group I

GROUPS	Proximal motor latency (ms)	Amplitude (mv)	Distal motor latency (ms)	Amplitude (mv)	Velocity (m/s)
Control					
Mean	3.3	13.3	9.4	13.0	56.3
SEM	0.1	0.2	0.1	0.3	1.7
P value	<0.01	<0.01	<0.01	<0.01	0.007
Experimental					
Mean	8.1	3.9	17.4	3.9	37.9
SEM	0.2	0.3	0.3	0.3	1.3

Table 3: Median Nerve Motor studies in upper limb Group II

GROUPS	Proximal motor latency (ms)	Amplitude (mv)	Distal motor latency (ms)	Amplitude (mv)	Velocity (m/s)
Control					
Mean	3.2	13.7	8.3	13.2	53.17
SEM	0.1	0.2	0.2	0.4	2.2
p value	<0.01	<0.01	<0.01	<0.01	0.007
Experimental					
Mean	4.8	9.3	11.2	8.1	40.9

SEM	0.17	0.4	0.2	0.1	1.4

Table 4: Peroneal Nerve Motor studies in lower limb Group II

GROUPS	Proximal motor latency (ms)	Amplitude (mv)	Distal motor latency (ms)	Amplitude (mv)	Velocity (m/s)
Control					
Mean	3.3	13.3	9.4	13.0	56.3
SEM	0.1	0.2	0.1	0.3	1.7
p value	<0.01	<0.01	<0.01	<0.01	0.007
Experimental					
Mean	8.8	3.4	17.9	4.5	38.5
SEM	0.4	0.1	0.8	1.1	2.8

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