ROLE OF NERVE CONDUCTION STUDY IN POLYNEUROPATHY

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Abstract:

Background: *Polyneuropathy has many different causes. It is often very difficult to find out the cause. Nerve conduction study (NCS) can classify neuropathy as axonal and demyelinatig variety and direct the search for cause.*

Methodology: *Purposively selected 80 patients from the department of Neurology Dhaka Medical College during the period of January 2009 to June 2010 were taken for NCS whose were compatible with polyneuropathy by history and clinical examination. Clinical, electrophysiological feature and pattern of polyneuropathy were analyzed.*

Results: *Mean age of the patients was 34.5 ±6.8 and M: F was 1.8:1. Students, laborer and cultivators were the most affected people. 55% patients were acute cases and 35% patients were chronic Cases. 30% patient had no known risk factor for neuropathy 25% patient had antecedent infection, 15% had diabetes mellitus, 7.5% were exposed to drugs/toxins or solvents and 5% had family history of neuropathy. In clinical examination 37.5% patients were in motor type, 10% pure sensory type and 52.5% mixed sensorimotor type. In NCS47.5% were motor, 7.5% pure sensory 45% mixed sensorimotor type. Axonal were 47.5%, demyelinating 27.5% and 25% as mixed axonal and demyelinating type.*

Conclusion: *NCS in polyneuropathy play critical role by classifying it as axonal or demyelinating and shorten the cause.*

Key words: *polyneuropathy, nerve conduction study*

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Introduction:

Polyneuropathy is the disorder in which the functions of numerous peripheral nerves are affected at the same time and leads to distal and symmetrical deficit with loss of tendon reflexes[1]. It is a relatively common syndrome which is often distressing and sometime disabling or even fatal.[2] Polyneuropathy has an estimated incidence of 25-200/100,000 persons per year and a prevalence of about 5%.[3]

Peripheral nerves have motor, sensory and autonomic component. Nerve fibers (axons) can be classified as either small fibers or large fibers. Large nerve fibers neuropathy affect many functions including - motor function, vibration perception, position sense, perception of temperature. Symptoms associated with large fibers neuropathy includes -numbness, tingling, weakness, pain, loss of deep reflexes. Symptoms of small fiber neuropathy are many and includes—pain describes as burning, stabbing, prickling, jabbing or lancinating (piercing), sensation of broken glass, burning sands, or ice pick in the bone, tight band like pressure, insensitivity to heat or cold and autonomic dysfunctions related to the organs.

Sensory nerves damage produce symptoms such as pain, numbness, tingling, burning or loss of sensation or feeling. Lack of sensation can produce cuts or burns unnoticed and ulcer or poor wound healing. Motor nerves damage results in decreased movement and

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muscle wasting. Symptoms usually begin as weakness or heaviness of the hands and or feet and may deteriorate over time.[4]

Polyneuropathy (PN) as a syndrome has many different causes and worldwide diabetes mellitus is the commonest cause other being are hereditary neuropathy, deficiency of vitamins, B_1, B_{12}, uremia, autoimmune neuropathies, infections including leprosy and HIV, drugs and toxins, porphyria ,paraneoplastic state and 25% cases are idiopathic. [5, 6] About 80% of polyneuropathies are axonal and the remaining 20% are demyelinating. Most of the axonal polyneuropathies are either purely sensory or mixed sensory motor type.[2]

NCS play a critical rule firstly to confirm the diagnosis of PN and then to classify it as axonal or demyelinating variety and thereby directing the search for cause.[5,7] Furthermore electrophysiology is quite sensitive to detect sub clinical involvement of either motor or sensory component of apparently pure sensory or pure motor PN giving further clue to the aetiology.[8,9]

PN are also classified into acute and chronic form. Acute forms PN are those that have relatively dramatic onset and usually recovers within six weeks. The classical and commonest example of that group is Guillaine Barre Syndrome (GBS). The other less common causes of acute PN are vasculitic, drugs and toxins, porphyria diphtheria, acute idiopathic sensory neuropathies. Chronic PN are those which usually develop over several months. Most of the classical chronic PN which presents with signs symptoms of distal symmetrical way falls into this category. The causes of chronic PN are diabetes mellitus, uremia, alcoholism and other toxins, drugs, underlying neoplasm, hereditary (Charcot Marie tooth disease) and idiopathic.[10, 11, 12] After exclusion of common causes of PN routine and specific investigations is the first stage of screening, neurophysiological studies in the form of nerve conduction study (NCS) and electromyogram (EMG) becomes the vital and important way of approach to the underlying cause in the second stage. The subsequent investigation in the third stage depends on the findings of NCS which distinguishes demyelinating from axonal polyneuropathies and divides axonal type into purely sensory, pure motor and mixed sensory motor groups. Chronic axonal PN has many causes. After NCS confirmation further approach in the third stage should includes investigations to identify cases of diabetes mellitus that were not detected by the screening 1st stage tests and to show the less common medical condition. Even after extensive investigations about 25% of cases remain idiopathic.[13, 14]

Methods:

This cross sectional observational study was carried out in the Department of Neurology, Dhaka Medical College and Hospital, Dhaka From January 2009 to June 2010 Sample Size: A total of 80(eighty) subjects were included purposively. Patients aged 18yrs to 60yrs of age with symptoms and signs of polyneuropathy were included and undergone electrophysiological investigation. Patients with mononeuropathy, traumatic or entrapment neuropathy, Conditions such as confusional state, pregnancy, skin diseases, oedema, prosthetic device that interfere with electrophysiological investigations, Neuropathy symptoms mimicking specific disease like motor neuron disease, myopathy, muscular dystrophy, spinal muscular atrophy or neuromuscular junction disorder were excluded. And patient who were not willing to participate in the study also excluded.

All the study cases were underwent meticulous history and asked for neuropathy symptoms and risk factors of polyneuropathy and physical examination were done by standard methods. Patients who presented with weakness, wasting and cramps were categorized clinically into motor type and those with tingling, numbness or paresthesias were categorized into sensory type and combination of sensory and motor features were classified into mixed type. Michigan neuropathy screening instrument questionnaire were used for quantitative assessment.

Nerve conduction study was carried out using standard techniques by Neuro pack II of 'Nihon Kohden' MEB 9200 machine (Japan). Skin temperature was maintained between 32^0C to 34^0C. The studies included motor and sensory nerve conduction in at least cross limbs or one arm and two legs. Electrophysiological study was set as a gold standard test for neuropathy assessment. General guidelines for performing nerve conduction study were followed. Other relevant investigations were done to find out the cause. Clinical features, electrophysiological features and pattern of poly neuropathy were analyzed.

Data Collection and Data Analysis

Data was collected by semi-structured questionnaire by the investigator. Face to face interview, medical history and clinical examination and subsequent laboratory investigations were done. Proper permission was taken from the concern departments. All the patients (cases) were informed about the about the nature of the study and their informed written consent were taken in a consent form before collecting data. Data was analyzed with the help of computer SPSS program version 16.0 software facility. A p-value of less then 0.05 was considered as statistically significant.

RESULTS:

A total of eighty patients of polyneuropathy (acute, subacute and chronic) were included in the study. Meticulous history and clinical examination was

undertaken before neurophysiological investigation. The findings of study obtained from data analysis are presented below.

Table-I

Patients Characteristics (n=80)

	No of patients	Percentage (%)
Men	52	65
Women	28	35
Age in years		
18 – 30	32	40
31 – 40	28	35
41 – 50	8	9
51 – 60	12	16
Mean age ±SD=34.4±6.8		
Range of age = 18- 60		
Occupation		
Service	12	15
Business	6	7.5
Student	10	12.5
Labourer	14	17.5
Cultivator	10	12.5
House wife	8	10
Unemployed	10	12.5
Retired	4	5
Others	6	7.5
Mode of onset/Duration		
Acute/upto 4 weeks	44	55
Subacute / 4 to 8 weeks	08	10
Chronic / > 8 weeks	28	35
Risk factor distribution		
Diabetes	12	15
Connective tissue disease	2	2.5
Hypothyroidism	4	5
Hereditary/ Family history	4	5
Preceding illness- diarrhea or RTI	20	25
Drugs/Toxins/Solvents exposure	6	7.5
Deficiency (vitamin)	4	5
Malignancy	2	2.5
Heavy metal(lead)	2	2.5
Not known (Idiopathic)	24	30

SD= standard deviation, RTI= respiratory tract infection

Table-1 shows the age distribution of the patient in to four groups. Ages of the patient ranged from 18 – 60 years. Most of the patient fell into 18 – 30 and 31-40 years age group and are 32 (40%) and 28 (35%) respectively. Lowest 8 (9%) was in 41 – 50 years age group. The mean age was 34.4 years with a standard deviation of 6.8. Patients were divided into male and female gender. Out of them 52 (65%) were male and 28 (35%) were female patients. M: F=1.8: 1. In occupation distribution service category comprised 15%(12), in business category 7.5%(6), student 12.5%(10), laborer 17.5%(14) which was the highest category, cultivator 12.5%(10), house wife 10%(8), unemployed comprises 12.5%(10), and retired 5%(4) which was lowest category and other not specified were 7.5%(6). Distribution of the patients according to the duration of illness i.e. mode of onset most of patient presented acutely which were 44(55%), chronic onset were 28(35%) and rests 8(10%) were in sub-acute category. Risk factors distribution of disorder causes polyneuropathy, highest number of patient were in not known or idiopathic group which comprises 30%(24), next in preceding illness of diarrhea or respiratory tract infection were 25%(20), history of diabetes were present in 15%(12), drugs/toxins/solvents in 7.5%(6), hypothyroidism in 5%(4), hereditary or family history of neuropathy also in 5%(4).

Table-II

Clinical features of the study population (n=80)

Clinical feature	No of Patient affected	Percentage (%)
Symptoms		
Paresthesia	70	87.5
Tingling	72	90
Numbness	24	30
Lack of feeling	20	25
Weakness	60	75
Wasting	20	25
Cramps	24	30
Signs		
Cranial nerve palsy	26	32.5
Loss of muscle power	60	75
Loss of Pinprick	20	25
Loss of Vibration sense	10	12.5
Deep tendon reflex hypo/ areflexia	70	87.5
Autonomic dysfunction (any level)	10	12.5
Gait abnormality (any level)	70	87.5
Nerve thickening	2	2.5
Weakness Distribution		
Proximal and distal	40	50
Distal to proximal	20	25
No weakness	20	25
Clinical type of Polyneuropathy		
Motor	30	37.5
Sensory	8	10
Mixed	42	52.5

The multiple response table 2 shows that most of the patient had tingling, paresthesia and weakness which ranges from 75% to 90%, numbness in 30% patient, wasting in 25% patient, loss of muscle power were observed in 75%, deep tendon hypo or areflexia were in 87.5%, cranial nerve palsy in 32.5%, pinprick loss in 25%, loss of vibration in 12.5%, autonomic dysfunction at any level in only 12.5% and nerve thickening in only two patients. Distribution of weakness in study subject, 50% patient there were both simultaneous proximal and distal weakness, distal to proximal weakness were in 25% patient, and 25% patient were with no weakness at all. Clinical types of polyneuropathy among study population. Out of 80 patients, 42(52.5%) had mixed sensory motor neuropathy, followed by 30 (37.5%) had motor neuropathy and only 10% had sensory neuropathy.

Table-III

Electrophysiological classification of neuropathy in study population (n=80)

Type	No of patients	Percentage (%)
Axonal	38	47.5
Demyelinating	22	27.5
Mixed axonal & demyelinating	20	25
Motor	38	47.5
Sensory	6	7.5
Mixed sensorimotor	36	45

The above Table III shows the electrophysiological category of polyneuropathy in study population. Axonal varieties were highest and comprised 47.5 %(38), mixed variety were lowest 25 %(20) and demyelinating category were 27.5 %(22). Types determined by electrophysiological examination into motor, sensory and mixed sensorimotor polyneuropathy were 47.5 %(38), 7.5% (6) and 45% (36) respectively.

Discussion:

Polyneuropathy is relatively common and often a distressing chronic condition. It has many diverse underlying causes and in different diseases the incidence of PN varies considerably.[15]This cross sectional study was designed to see the clinical and electrophysiological features of polyneuropathy patients. This study also addressed the clinical and electrophysiological pattern of polyneuropathy patient.

In this study patients of all age group ranging from 18-60 years were included. Majority of the patients 32 (40%) were in 18 to 30 years of age with mean ±SD = 34.4 ±6.8. In this study 65 % were male and 35 % were female with M: F = 1.8: 1. In one local study[15] the M: F was 1.88: 1 and in another local study [16] the M: F ratio was 1.9:1 which resembles with the present study and it is observed that polyneuropathy is about two times more common in male. McLeod et al.[17] also found an overall predilection for men (3:1). In this study polyneuropathy were widely distributed in different occupations, labourers, cultivator and students were affected more. In this regard there are a few studies elsewhere. In the study of Chistee, [18] more or less similar findings were observed but in his series cultivators were less affected but housewives were more affected as well as labourer and students.

It was observed in this study that 55% patients presented acutely and 35% had chronic onset and 10% patients had sub-acute onset. Study on polyneuropathy patients comprising acute, sub-acute and chronic cases are few. Local study Chistee[18] of 50 polyneuropathy cases GBS cases were 50% and the findings were similar with the present study.

In this study majority (30%) patients had no known history of risk factors i.e. idiopathic, antecedent infections (preceding illness either diarrhoea or RTI) was the next common risk factors (25%), next was diabetes mellitus (15%), followed by combined drugs & toxins (7.5%). In a study of chronic polyneuropathy by Vrancken et al.[3] idiopathic were 43%, diabetes mellitus 32%, alcohol abuse 14%, paraproeinaemia 9%, deficiency of vitamin 6% and autoimmune or systemic disease 4% were observed. In a Dutch study on chronic polyneuropathy, Rosenberg NR et al.[6] observed 60(57.1%) patients of diabetes mellitus, followed by HIV infection in 21(20%) patients, alcoholism in 11(10.5%) patients; drug induced in 7(6.7%) patients and renal failure in 6(5.8%) patients in a study of 105 chronic polyneuropathy cases. In Lubec et al.[19] frequency of causal factors in 124 cases were : - diabetes mellitus in 26(21%) cases, alcohol in 20(16.1%) cases, vitamin deficiency in 13(10.5%) cases, GBS in 9(7.3%) cases, paraproteinamias in 6(4.8%) cases, hypothyroidism in 5(4.03%) cases, borreliosis in 6(4.8%) cases, paraneoplasia in 4(3.2%) cases, CIDP in 5(4.03%) cases, hereditary in 3(2.4%) cases, hyperthyroidism in 3(2.4%) cases, critical illness in 2(1.6%) cases, vasculitis in 3(2.4%) cases, and each one(0.8%) case of sarcoidosis, vincristine, azathioprine, Refsum's disease, Sneddon's syndrome, Ehlers-Danlos syndrome, crohn's disease inflammatory polyarthritis and solvent. In an Asian study of 124 cases of chronic polyneuropathy Habib and Ferdousi[15] observed diabetes were 45.2%, idiopathic 45.2%, hereditary 5.7% and CIDP in 3% cases. So the distribution of polyneuropathy in different diseases varies worldwide.

In this study less diabetic and infectious cases were observed as because major bulk of diabetes mellitus are cared by internationally reputed separate diabetic hospital and infectious disease hospitals.[16]

The features of polyneuropathy may be exclusively motor, sensory, autonomic or combined. Most PN present with mixed sensory motor symptoms. Sensory symptoms were usually the presenting features. These were tingling, pins and needles, burning sensation, pain and numbness in the extremities. Motor symptoms were usually those of weakness and wasting. [20] This is reflected in the present study where paresthesias were present in 87.5 %, tingling in 90%, numbness in 30% cases. Weakness in this study was in 75% cases, deep tendon reflex hypo/areflexia in 87.5% and abnormality of gate at any level were also 87.5%. Similarity was observed in the study of Habib and Ferdousi [15] also. A relative lack of muscle wasting in relation to the degree of weakness, weakness of proximal muscle as well as distal muscle, disproportionate loss of joint position and vibration sensation compared to relative preservation of pain and temperature are suggestive of demyelinating neuropathy.[21] In this present study proximal and distal weakness was in 50% cases and distal to proximal weakness was observed in 45% cases.

One of the most important aims of the study was to detect the clinical and neurophysiological type of polyneuropathy. In Rosenberg et al.[6] 77.5% were mixed sensorimotor type, 13.75% were pure sensory type and 8.75% were pure motor type. In Konig et al.[22] 42% were mixed sensory motor, 30% sensory, no case of motor type. In the study of Konig et al.[22] cases of mononeuropathy and mononeuritis multiplex were included. In Macleod et al.[17] 64% were mixed sensory motor type and 27% pure sensory type and 9% were pure motor type. In our present study mixed sensory motor types were 52.5%, motor types were 37.5% and pure sensory types were only 10%. Though in this present series mixed sensory motor type was the most common, the high motor type reflects the inclusion of significant acute polyneuropathy cases.

In this study of 80 polyneuropathy cases either cross limbs or both the lower limbs and an upper limb nerves were examined electrophysiologically. 80 median nerves, 80 ulnar nerves, 120 tibial nerves, 130 common peroneal nerves and 140 sural nerves were studied. In this study electrophysiological types of polyneuropathy were axonal type 47.5%, demyelinating type 27.5% and mixed type 25% which were near similar with Vrancken[3] et al. Where axonal type was 57%, demyelinating type 13% and not specified were 31%. In another European study[6] (Rosenberg NR et al.) of 56 chronic polyneuropathy cases, axonal types were 87.5% and demyelinating type were 12.5% and the findings resembles the present study. In a Bangladehi study by Habib and Ferdousi[15] 26.6% were axonal, 16.1% demyelinating and 31.5% were mixed axonal and demyelinating. The above mentioned local study does not match with our study due to the fact that 25.8% patient were not labeled in any particular pathological type.

It is important to know neuropathy as axonal or demyelinating as it helps proper management. Highly significant association was seen in motor and mixed sonsorimotor type of clinical and electrophysiological classification. In sensory polyneuropathy distribution in clinical and electrophysiological types varies. Comparison of the severity of polyneuropathy in clinical and electrophysiological grade there were poor relation among them. To determine the relation between neurophysiological data and clinical examination Lefaucheur[23] et al. observed that clinical and neurophysiological classifications and severity scores were correlated whatever the type of neuropathy. These differences with the present study might be due to that Lefaucheur[23] et al studied the sensory neuropathy according to fiber type involvement. Latov[24] et al observed that the number and type of demyelinating abnormalities in patients with polyneuropathy vary with the clinical phenotype. Rajabally et al.[25] in their studied patients with CIDP demonstrated the predominance of demyelination in upper limbs nerves, of axonal loss in lower limbs nerves and presence of about 50% of demyelinating – range abnormalities in clinically unaffected territories. Vittadini[26] et al. found significant correlation between alcoholic polyneuropathy, the duration of alcoholism and the type of alcoholic beverage consumed.

In this present study there are some relation and there are some variation among the clinical and electrophysiological spectrum of polyneuropathy .

Conclusion:

Nerve conduction study is a very important investigation in the evaluation of polyneuropathy. It confirm the diagnosis and classify neuropathy as axonal and demyelinatig category and thus direct the cause and help in management

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