J MEDICINE 2014; 15: 3-8

Original Articles

Clinical Patterns of Polyneuropathy Attending in a Tertiary Level of Hospital

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Abstract:

A cross sectional study was carried out in the department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka to explore the different clinical pattern of polyneuropathy. A total of 60 subjects were included in this study and mean age of the entire patient was 42.25 year and male and female ratio was 3:1. Out of all patients 26.7% were service holder, 20.0% were student, 20.0% were housewife, 11.7% were unemployed and 33.3% were engaged in some other professions. Maximum 41.7% patients were educated up to graduate and above level followed by 20.0% secondary, 18.3% primary, 16.7% higher secondary and 3.3% were illiterate. Out of all patients 31.7% were smoker, 48.3% were non smoker and 20.0% past smoker. About 35.0% patients had history of betel leaf; nut chewing and no patient had history of alcohol intake. Out of all patients of polyneuropathy 33.3% had diabetic neuropathy, 11.7% had Guillain-Barré syndrome (GBS), 10.0% had chronic inflammatory demyelinating polyneuropathy, similar number had unknown etiology, 6.7% had renal failure, 5.0% had leprosy, Vitamin B 12 deficiency and chronic liver disease(CLD) of each, 3.3% had history of INH drug intake and similar had systemic lupus erythematosus(SLE). Regarding clinical presentation, out of all respondents 83.3% had muscle weakness, similar number had problems with object handling, 66.3% had muscle cramp, 63.3% had impaired standing or gait, 55.0% had distal paresthesia, 41.7% had burning feet, 8.3% had restless legs and 5.0% had stiffness. In the light of this study we conclude that polyneuropathy has wide variety of clinical pattern. The study will enrich our current knowledge and will improve the quality of management of polyneuropathy among the Bangladeshi population.

Key words: *Polyneuropathy, clinical patterns of polyneuropathy.*

Introduction

Polyneuropathy, in the broadest sense, refers to a scope of clinical syndromes affecting a variety of peripheral nerve cells and fibers, including motor, sensory, and autonomic fibers.[1] Polyneuropathy has an estimated incidence of 25–200/100 000 persons/year and a prevalence of up to about 5%.[2-4] Most of these patients have a slowly progressive course. They share common clinical features: a relatively mild, predominantly sensory polyneuropathy with axonal degeneration, male predominance, and a mean age of onset of around 60 years.[5] The peripheral nervous system can be involved in a wide range of medical disorders with various pathophysiologies.[6] It may be affected by numerous toxins, both drugs and industrial agents, and by a variety of chronic infections, including human immunodeficiency virus (HIV). Furthermore, a number of apparently immune-mediated disorders result in peripheral neuropathies, including Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), and multifocal motor neuropathy with conduction block syndrome (MMNCB).[7,8] Hereditary polyneuropathies may cause a wide range of peripheral neuropathy. Rare causes include certain cancers, such as lung cancer, and taking excessive amounts of vitamin B_6 (pyridoxine).[9]

Very recently, dramatic development has been made in different types of health services in Bangladesh. Several health care institutions for the management of neurological diseases have already been established in Dhaka and other part of the country both in private and public sectors. Though the exact statistics of incidence and prevalence of

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polyneuropathy in our country is not available but it is the common opinion that the number of such kind of problem is increasing day by day. Sophisticated technology and multidimensional services have been added a new dimension in this hospital for health care services. The study will enrich our current knowledge and better management of polyneuropathy among the Bangladeshi population. This knowledge will improve the quality of management of patients with polyneuropathy.

Methods and Materials:

A cross sectional study was carried out in the department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka. This study was conducted from January 2008 to February 2009 for duration of one year two months. A total of 60 subjects were included in this study and simple random sampling was the sampling technique. Prior to the commencement of this study, the research protocol was approved by the Local Ethical committee. The aims and objectives of the study were explained to the patients in easily understandable local language and then informed consent was taken from each patient. It was assured that all informed and records will be kept confidential and the procedure will be helpful for both the physician and the patients in making rational approach of the case management. Inclusion criteria for cases were patients > 12 years old, symptoms and signs of polyneuropathy with a nadir after three months, electrophysiological confirmation of polyneuropathy, work up in the outpatient, indoor and neuropathy clinic only and participants, who was given consent and willing to comply with the study procedure, were included. Exclusion criteria for cases were patients who refused to be included in the study.

Data were collected by semi-structured questionnaire by the investigator as soon as possible after the admission of the patient. Data were collected by face to face interview. Information was collected by taking medical history and clinical examination and subsequent laboratory investigations. Proper permission was taken from the concerned departments. All the cases were informed about the nature of the study. After collection, data were checked for inadequacy, irrelevancy, and inconsistency. Irrelevant and inconsistent data were discarded. All data were recorded systematically in preformed data collection form and quantitative data were expressed as mean and standard deviation and qualitative data as frequency distribution and percentage. Statistical analysis was performed by using SPSS for windows version 15.0.

Results:

Mean age of the entire patient was 42.25 years and all patients were within 16 to 70 years age range. Table I shows the mean age of the patients of polyneuropathy having different disease. Table II shows the sex distribution of the patients. Out of all patients 75.0% were male and 25% were female. Male and female ratio was 3:1. Out of all patients 26.7% were service holder, 20.0% were student, 20.0% were housewife, 11.7% were unemployed and 33.3% were engaged in some other professions. Table III shows regarding socioeconomic condition of the patients that maximum 70.0% patients were from middle, 21.7% from lower and 8.3% from upper class family. Maximum 41.7% patients were educated up to graduate and above level followed by 20.0% secondary, 18.3% primary, 16.7% higher secondary and 3.3% were illiterate. Table IV shows the different types of personal habit of the patients. Out of all patients 31.7% were smoker, 48.3% were non smoker and 20.0% past smoker. 35.0% patients had history of betel leaf, nut chewing and no patient had history of alcohol intake. Table V shows the different etiological pattern of polyneuropathy. Out of all patients of polyneuropathy 33.3% had diabetic neuropathy, 11.7% had GBS, 10.0% had chronic inflammatory demyelinating polyneuropathy, similar number had unknown etiology, 6.7% had CMT disease, 6.7% had renal failure, 5.0% had leprosy, Vitamin B 12 deficiency and CLD of each, 3.3% had history of INH drug intake and similar had SLE. Table VI observed that out of all respondents 83.3% had muscle weakness, similar number had problems with object handling, 66.3% had muscle cramp, 63.3% had impaired standing or gait, 55.0% had distal paresthesia, 41.7% had burning feet, 8.3% had restless legs and 5.0% had stiffness.

Table-I

Distribution of the patients by age.

Age	Frequency	Percent
≤35	17	28.3
36-45	18	30.0
46-55	18	30.0
≥56	7	11.7
Total	60	100.0
Mean±SD (Range)	42.25±11.81	16-70

Table II

Distribution of the patients by sex and occupation.

Sex	Frequency	Percent
• Male	45	75.0
• Female	15	25.0
• Occupation		
Service	16	26.7
Student	5	8.3
Housewife	12	20.0
Unemployed	7	11.7
Others	20	33.3
Total	60	100.0

Table III

Distribution of the patients by socioeconomic condition and education.

Socioeconomic status	Frequency	Percent
Upper	5	8.3
Middle	42	70.0
Lower	13	21.7
Educational status		
Illiterate	2	3.3
Primary	11	18.3
Secondary	12	20.0
Higher secondary	10	16.7
Graduate and above	25	41.7
Total	60	100.0

Table IV

Distribution of the patients by personal habit

	Frequency	Percent
Smoking habit		
• Non smoker	29	48.3
• Ex-smoker	12	20.0
• Present smoker	19	31.7
Betel leaf, nut chewing	21	35.0
Alcohol intake	0	0.0

Table V

Distribution of the patients by etiological pattern of polyneuropathy

Causative diseases	Frequency	Percent
Diabetic neuropahty	20	33.3
GBS	7	11.7
Chronic inflammatory demyelinating polyneuropathy	6	10.0
Chr.renal failure	4	6.7
Charcot-Marry-Tooth disease	4	6.7
Leprosy	3	5.0
Vit B 12 deficiency	3	5.0
Chr. Liver disease	3	5.0
Drug (INH)	2	3.3
SLE	2	3.3
Unknown	6	10.0
Total	60	100.0

Table VI

Distribution of the patients by clinical presentations

Clinical presentations	Frequency	Percent
Muscle weakness	50	83.3
Problems with object handling	50	83.3
Muscle cramp	40	66.7
Impaired standing or gait	38	63.3
Distal paresthesia	33	55.0
Burning feet	25	41.7
Restless legs	5	8.3
Stiffness	3	5.0

• Multiple responses

Discussion:

In study by Wolfe et al patients presented with a mean age of 63.2 years.[9] In the case histories of 519 patients with peripheral neuropathy by McLeod et al., the mean age of onset of symptoms was 50.6 years. Males were affected more commonly than females in a ratio of 3:1 which is the same of our finding (3:1). [10] Tondel et al. in Sweden including 232 cases of cryptogenic polyneuropathy. Male sex and increasing age were significant determinants for cryptogenic polyneuropathy. [11] In current studies out of all patients of polyneuropathy 33.3% had diabetic neuropathy, 11.7% had GBS, 10.0% had chronic inflammatory demyelinating

polyneuropathy, similar number had unknown etiology, 6.7% had CMT disease, 6.7% had renal failure, 5.0% had leprosy, Vitamin B 12 deficiency and CLD of each, 3.3% had history of INH drug intake and similar had SLE. In study conducted by George and Twomey, Common causes were diabetes (27%), neoplasms (13%), the Guillain-Barré syndrome (11%) and 28% with no identifieable cause in their reviewe on seventy-four patients over 65 years, with electro-physiologically confirmed polyneuropathies. [12]

In a prospective study on two hundred and twenty-five patients of polyneuropathy by Ghosh et al, conducted in two large hospitals from Kolkata in order to find out the spectrum of peripheral neuropathy Common varieties of neuropathy were Guillain-Barré syndrome, diabetes mellitus, hereditary motor sensory neuropathy, chronic inflammatory demyelinating neuropathy, drugs and toxin related Ghosh et al.[13] In Taiwan, Lin et al. evaluate the etiology over 520 patients with generalized neuropathy in 5 neurological centers. The neuropathy was diabetic in 49.23%, alcoholic in 8.65%, inflammatory in 6.53%; malignancy 2.31%, dysproteinemia 1.73%, uremic in 4.23%, hereditary motor and sensory in 4.23%, toxic in 2.69%, ischemic in 2.31%, hypothyroidism in 1.92%, nutritional deficiency and malabsorption in 1.15%, chronic liver disease in 0.77%, other diseases in 2.12% and unclassified in 12.12%. This survey provided a crude etiological picture of generalized neuropathy on this island. [14]

In a study by Barreira et al, alcohol was considered as culprit for polyneuropthy in 25% cases. [15] In our study alcoholism did not found as a cause, which may be due to low consumption alcohol in Bangladesh and its the sociocultural background. The incidence of leprosy induced neuropathy in our series was 5%, which is the same incidence found in large study by Ghosh et al.[13] In current study all leprotic neuropathics were demyelinating, 66% had sensory and 34% had motor defect. Bharucha et al carried out a door-to-door survey to screen for neurologic diseases, including peripheral neuropathy, in a community of 14,010 Parsis living in housing colonies in Bombay, India. The most common neurologic disorder was peripheral neuropathy with 334 cases (2,384 cases/100,000 population). The most common neuropathy was compressive, with diabetes the most common noncompressive etiology. There was no leprosy, and nutritional neuropathies were rare. In our study second common etiology was inflammatory neuropathy, including 11.7% had GBS and 10.0% had chronic inflammatory demyelinating polyneuropathy (CIDP). All cases of GBS and CIDP were demyelinating, 85% of GBS were mixed and 15% were motor type. Among CIDP patients 33% had sensory defect, 17% motor and 50% had mixed defect. [16]

In Lin et al. series, out of 520 patients inflammatory cause was found in 34 (6.53%) including 21 (4.04%) with acute inflammatory demyelinating polyneuropathy and 12 (2.31%) with chronic inflammatory demyelinating polyneuropathy, and 1 with chronic relapsing polyneuropathy. Renal failure was found in 6.7% cases of our series, which was described in (4.23%) cases of large series of Lin et al.[14] Charcot-Marie-Tooth disease is the most common hereditary neuropathy, affecting 1 of 2,500 people,[4] in our series 6.7% of our cases was diagnosed as Charcot-Marry-Tooth disease (CMTD), 75% and 25% of all CMTD patients were demyelinating and axonal type respectively, all of them had motor defect. In our series 3.3% patients were diagnosed case of SLE, all were sensory type and axonal defect. Honczarenko et al. in 137 patients with systemic lupus erythematosus (SLE), severe neurological syndromes indicating central, peripheral or autonomic nervous system involvement were frequently found. [17] Polyneuropathy was present in 17 (12.41%) of cases of SLE. 5% of cases were deficient of vitamine-B12 in our study and all had mixed with sensory defect. Nardin et al. in a retrospective cohort study of 581 patients presenting with polyneuropathy over a 2-year period; 4% had definite vitamin B12 deficiency the sole or contributing cause for their polyneuropathy. [18] This finding is very much consisted with our findings.

In Lin et al. series it was 12.02% and in George and Twomey series it was 28%. [14,12] In current studies 10% cases were of unknown etiology. Among the idiopathic cases 33% had demyelating, 17% axonal and 50% had mixed type defect. Sensory defect were found in 33% cases, motor 17% and mixed defect found in 50% cases. Chia et al. reviewed the clinical and nerve biopsy findings of 100 patients of over 65 years age group thus 35% of the patients included in this series had one form or another of vasculitic neuropathy. [19] Our study lacks such biopsy evidence of vasculopathy or other cause of neuropathy. In the most common form of chronic polyneuropathy, only sensation is affected. Usually, the feet are affected first, but sometimes the hands are. A pins-and-needles sensation, numbness, burning pain, and loss of vibration sense and position sense (knowing where the arms and legs are) are prominent symptoms. Because position sense is lost, walking and even standing become unsteady. Consequently, muscles may not be used. Eventually, they may weaken and waste away. Out of all respondents of current studies 83.3% had muscle weakness, similar number had problems with object handling, 66.3% had muscle cramp, 63.3% had impaired standing or gait, 55.0% had distal paresthesia, 41.7% had burning feet, 8.3% had restless legs and 5.0% had stiffness.

Grant et al. retrospectively studied 54 patients with sicca complex and peripheral neuropathy to determine mode of presentation, neuropathic patterns, frequency and pattern of serologic abnormalities, and frequency of systemic disease, including necrotizing vasculitis. [20] Peripheral neuropathy was the presenting problem in 87%. Although sicca symptoms occurred in 93%, they were a presenting complaint in only 11% and were usually mild, reported only after specific inquiry. Sensory neuropathies strongly predominated; 61% of patients manifested either sensory polyneuropathy or polyganglionopathy. Less common patterns included sensorimotor polyneuropathy (17%) and polyradiculoneuropathy (11%). Vasculitic neuropathy was demonstrated in only two patients, but nonspecific epineurial inflammation was present in 70% of nerve biopsies.

Lewis and Sumner in one study compare the electro-diagnostic studies of 40 patients with chronic acquired demyelinative neuropathy and 18 patients with familial demyelinative neuropathy. Patients with acquired neuropathy had differential slowing of conduction velocity when distal latencies were compared with more proximal conduction velocities in the same nerve, when equivalent segments of different nerves were compared, and when dispersion of compound motor action potentials was examined. Conduction block was noted in some patients. In their study the patients with familial disease had uniform conduction slowly of all nerve segments, and conduction block was not seen. They found that the chronic acquired demyelinative neuropathy is characterized by multifocal slowing of nerve conduction, whereas familial demyelinative neuropathy is characterized by uniform conduction slowing. [21]

Notermans prospectively studied the clinical and electrophysiological features of 75 patients (46 men and 29 women) with chronic polyneuropathy presenting in middle or old age in whom a diagnosis could not be made even after extensive evaluation and a follow up of six months. The mean age at the onset of symptoms was 56.5 years. The clinical features of chronic idiopathic polyneuropathy are heterogeneous. On clinical grounds 44 patients had a sensorimotor, 29 patients a sensory, and two patients a motor polyneuropathy. The overall clinical course in chronic idiopathic polyneuropathy was slowly progressive. None of the patients became severely disabled. They found that the electrophysiological and nerve biopsy studies were compatible with an axonal polyneuropathy. Antibodies against myelin associated glycoprotein, gangliosides, and sulphatides were assessed in 70 patients and found to be negative. [22]

Seventy five patients with chronic idiopathic axonal polyneuropathy (CIAP) were studied for five years by **Notermans** et al. The standardized and quantified neurological examination shows that progression of CIAP is slow, and handicap, if present, is not severe. During the follow up period a definite cause of the neuropathy was found in only four patients (two hereditary motor and sensory neuropathy type 2, one sensory chronic inflammatory demyelinating polyneuropathy, one alcoholic neuropathy). At the end of the follow up CIAP was not related to malignancy or gammopathy. Routine repetition of laboratory tests was not informative and these tests should be performed on clinical grounds only. [23]

Conclusion:

In the light of this cross-sectional study we conclude that polyneuropathy has wide variety of clinical presentation associated with diabetes, GBS, chronic inflammatory demyelinating polyneuropathy, renal failure, Charcot-Marry-Tooth disease, leprosy, vit B 12 deficiency, CLD, drugs(INH) and SLE. Further large controlled study is needed to establish the clinico-etiological pattern in context of Bangladesh.

Conflict of Interest : None

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