Original Article

Etiological patterns of polyneuropathy in a tertiary level hospital of Bangladesh

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Abstract

The causes of polyneuropathy are many and it is important to identify them, as some of them, especially the inflammatory types are treatable. To explore the different etiological factors of polyneuropathy, this cross sectional study was carried out in the department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka. Data were collected by taking medical history and clinical examination and subsequent laboratory investigations. A total of 60 subjects were included in this study and mean age of the entire patient was 42.25 year. 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 paraesthesia, 41.7% had burning feet, 8.3% had restless legs and 5.0% had stiffness. Out of all patients of polyneuropathy, 33.3% had diabetic neuropathy, 11.7% had Guillain-Barré syndrome, 10.0% had chronic inflammatory demyelinating polyneuropathy, similar number had unknown etiology, 6.7% had charcot-marry-tooth disease disease, 6.7% had renal failure, 5.0% had leprosy, Vitamin B 12 deficiency and chronic liver disease of each, 3.3% had history of isoniazide drug intake and similar had systemic lupus erythromatosus. We conclude that, polyneuropathy has wide variety of etiological factors. Among them, diabetes is the most common factor. Further large controlled study is needed to establish the etiological pattern in the context of Bangladesh.

Key words: polyneuropathy, etiological patterns of polyneuropathy

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Introduction

Polyneuropathy, in the broadest sense, refers to a clinical syndromes affecting a variety of peripheral nerve cells and fibers, including motor, sensory and autonomic fibers.[1] The peripheral nervous system can be involved in a wide range of medical disorders with various pathophysiologies.[2] 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).[3] Polyneuropathy has an estimated incidence of 25–200/100,000 persons per year and a prevalence of up to about 5%.[4] Although, by definition, patients with an unknown cause of polyneuropathy must form a heterogeneous group, these patients may share a common condition which causes or enhances the development of polyneuropathy.[5] The most common generalized polyneuropathy is diabetic sensorimotor polyneuropathy, which may be present in up to 66% of type 1 diabetic patients and in nearly 59% of type 2 diabetic patients.[6]

Despite comprehensive testing and assessments, an etiologic diagnosis may not be determined in nearly 25% of patients with polyneuropathy.[7] In most patients with a polyneuropathy related to a medical disorder or immune-mediated mechanism, specific therapies directed at the underlying mechanism are usually effective in controlling the polyneuropathy. When using a systematic approach with careful history, physical examination, electrophysiological, serological and sometimes cerebrospinal fluid (CSF) and histological studies, the cause can be identified in as many as 75% of polyneuropathy patients.[8] This study was designed to enrich our knowledge about the etiological pattern of polyneuropathy among the Bangladeshi population and this knowledge will improve the quality of management of patients with polyneuropathy.

Methods

This cross sectional study was carried out in the department of Neurology, Bangabandhu Sheikh Mujib

Medical University (BSMMU), Shahbag, Dhaka. The 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 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 out-patient, indoor and neuropathy clinic only and participants who gave consent to comply with the st uestionnaire supplied by the investigator as soon as possible after the admission of the patient. Data were collected by face to face interview. Information was obtained by taking medical history and clinical examination and subsequent laboratory investigations. 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. Mean age of the patients was 42.25 ± 11.81(Table-I).

Table-I: Age distribution of the patients by specific diseases

Specific diagnosis	Mean ± SD(years)
Diabetic neuropathy (n=20)	45.0±9.57
Leprosy (n=3)	40.0±.00
GBS (n=7)	39.57 ±13.2
Drug (INH) (n=2)	45.0 ±.00
Chronic renal failure (n=4)	33.0±3.46
Systemic lupus erythematosus (SLE) (n=2)	61.0±.00
CIDP (n=6)	44.83±20.13
Vitamin B 12 deficiency (n=3)	37.33±4.04
Chronic liver disease (CLD) (n=3)	46.33±9.81
Charcot-Marie-Tooth Disease (CMTD) (n=4)	49.0±2.00
Unknown (n=6)	29.67±10.75
Total (n=60)	42.25 ± 11.81

Out of all patients 75% were male and 25% were female. Male and female ratio was 3:1. Out of all patients 26.7% were service holder, 20% were student, 20.0% were housewife, 11.7% were unemployed and 33.3% were engaged in some other professions. According to socioeconomic condition, maximum (70.0%) patients were from middle income group, 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% 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% previous smoker. 35% patients had history of betel leaf and / or nut chewing and no patient had history of alcohol intake (Table II).

Table-II: Distribution of the patients by personal habit

	Frequency	Percent
Smoking habit		
• Non-smoker	29	48.3
• Ex-smoker	12	20
• Present smoker	19	31.7
Betel leaf, nut chewing	21	35
Alcohol intake	0	0.0

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% had distal paraesthesia, 41.7% had burning feet, 8.3% had restless legs and 5% had stiffness (Figure 1).

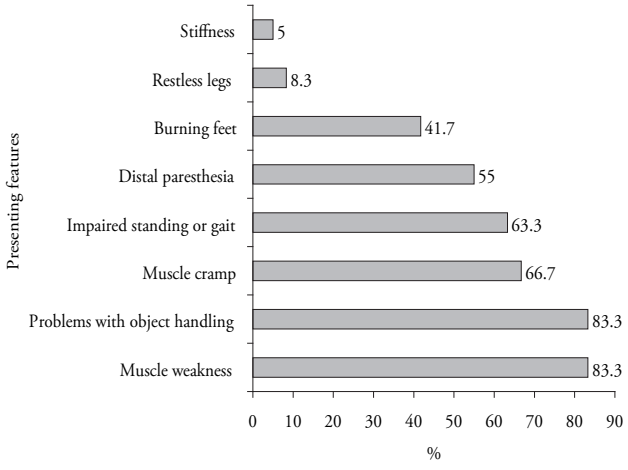

Figure-1: Distribution of the patients by clinical presentations

Sixty polyneuropathy patients of different etiology have been studied in series. Out of all patients of polyneuropathy, 33.3% had diabetic neuropathy, 11.7% had Guillain-Barré syndrome, 10% had chronic inflammatory demyelinating polyneuropathy, similar number had unknown etiology, 6.7% had Charcot-Marie-Tooth disease, 6.7% had renal failure. Five percent had leprosy, vitamin B 12 deficiency and Chronic liver disease of each. Three and half % had history of INH drug intake and similar percentage of patients had Systemic lupus erythematosus (Table III).

Table-III: Distribution of the patients by etiological pattern of polyneuropathy

Causative diseases	Frequency	Percent
Diabetic neuropathy	20	33.3
GBS	7	11.7
Chronicin flammatory demyelinating polyneuropathy	6	10.0
Chronic renal failure	4	6.7
Charcot-Marry-Tooth disease	4	6.7
Leprosy	3	5.0
Vit B 12 deficiency	3	5.0
Chronic Liver disease	3	5.0
Drug (INH)	2	3.3
SLE	2	3.3
Unknown	6	10
Total	60	100

Discussion

This cross sectional study was carried out from January 2008 to February 2009 to explore the different etiological factors of polyneuropathy. In a 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 same of our finding (3:1).[10] Tondel et al carried out a study including 232 cases of cryptogenic polyneuropathy. Male sex and increasing age were significant determinants for cryptogenic polyneuropathy.[11] In the current study, out of all patients of polyneuropathy 33.3% had diabetic neuropathy,[11] 7% had Guillain-Barré syndrome, 10% had chronic inflammatory demyelinating polyneuropathy, similar number had unknown etiology, 6.7% had Charcot-Marry-Tooth disease, 6.7% had renal failure, 5.0% had leprosy, Vitamin B 12 deficiency and chronic liver disease of each, 3.3% had history of INH drug intake and similar had systemic lupus erythromatosus. In a study by George and Twomey on seventy-four patients over 65 years with electrophysiologically confirmed polyneuropathy, common causes were diabetes (27%), neoplasms (13%), GBS (11%) and 28% with no identifiable cause.[12] In a prospective study by Ghosh et al, common varieties of neuropathy were Guillain-Barré syndrome, diabetes mellitus, hereditary motor sensory neuropathy, chronic inflammatory demyelinating neuropathy, drugs and toxin.[13] Lin et al. evaluated the etiology over 520 patients with generalized neuropathy in 5 neurological centers. They observed that the neuropathy was diabetic in 49.23%, alcoholic in 8.65%, inflammatory in 6.53%; malignancy in 2.31%, dysproteinemia in 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%.[14]

In a study by Barreira et al, alcohol was considered as culprit for polyneuropthy in 25% cases.[15] In our study alcoholism was not found as a cause, which may be due to low consumption alcohol in Bangladesh according to its the socio-cultural 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] Bharucha et al carried out a survey to screen for peripheral neuropathy. They observed that the most common etiology of neuropathy was compressive, with diabetes. The second common etiology was inflammatory neuropathy, including 11.7% had GBS and 10.0% had chronic inflammatory demyelinating polyneuropathy (CIDP).[16] Polyneuropathy was present in 5% of cases with deficiency of vitamine-B12 in our study and all had mixed with sensory defect. Nardin et al. found that, 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.[17] These findings are very much consisted with our findings.

In the light of this study we conclude that polyneuropathy has wide variety of etiological factors, including diabetes, GBS, chronic inflammatory demyelinating polyneuropathy, renal failure, charcot-marry-tooth disease, leprosy, vit B12 deficiency, CLD, drugs (INH) and SLE. Among them, diabetes is the most common etio-pathological factor. Further large controlled study is needed to establish the etiological pattern in the context of Bangladesh.

References

1. Lacomis D. Small-fiber neuropathy. Muscle Nerve. 2002; 26 : 173-188
2. Hughes RAC. Peripheral neuropathy. BMJ. 2002; 324: 466-469
3. Saperstein DS, Katz JS, Amato AA, Barohn RJ. Clinical spectrum of chronic acquired demyelinating polyneuropathies. Muscle Nerve. 2001; 24: 311-324.
4. Vrancken AFJE, Kalmijn, Buskens SE, Franssen H, Vermeulen M, Wokke JHJ et al. Feasibility and cost efficiency of a diagnostic guideline for chronic polyneuropathy: a prospective implementation study. J Neurol Neurosurg Psychiatry. 2006; 77: 397–401
5. Teunissen L, Gabriel JE, Rinkel, Ale-Algra, Van Gijn J, Luijten JAFM, et al. Approch to polyneropathy. J Neurol Neurosurg Psychiatry. 2002; 73(2):112 - 118
6. Dyck PJ, Kratz KM, Karnes JL. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology. 1993; 43: 817-824.
7. Wolfe GI, Barohn RJ. Cryptogenic sensory and sensorimotor polyneuropathies. Semin Neurol. 1998; 18:105-111
8. Bosh M, Bradley WG, Daroff RB, Fenichel GM, Marsden CD. Disorders of peripheral nerves. Neurol Clin Pract. 1996; 5: 456-474
9. Wolfe GI, Baker NS, Amato AA, Jackson CE, Nations SP, Saperstein DS et al. Chronic cryptogenic sensory polyneuropathy: clinical and laboratory characteristics. Arch Neurol. 1999; 56(5):540-547.
10. McLeod JG, Tuck RR, Pollard JD, Cameron J, Walsh JC. Chronic polyneuropathy of undetermined cause. J Neurol Neurosurg Psychiatry. 1984; 47(5):530-535
11. Tondel M, Lindh J, Jönsson P, Vrethem M, Persson B. Occupational Determinants of Cryptogenic Polyneuropathy. Neuroepidemiology.2006; 26: 187-194
12. George J, Twomey JA. Causes of polyneuropathy in the elderly. Age Ageing. 1986;15(4):247-249
13. Ghosh B, Sengupta S, Bhattacharjee R, Pal S, Saha SP, Ganguly G et al. Spectrum of peripheral neuropathy in eastern India. J Indian Med Assoc. 2006; 104(4): 170-173
14. Lin KP, Kwan SY, Chen SY, Chen SS, Yeung KB, Chia LG et al. Generalized neuropathy in Taiwan: an etiologic survey. Neuroepidemiology. 1993; 12(5): 257-261
15. Barreira AA, Marques Júnior W. Diagnosis of peripheral neuropathies : age, sex and occupation in relation to etiology. Arq Neuropsiquiatr. 1992; 50(4); 458-462
16. Bharucha NE, Bharucha AE, Bharucha EP. Prevalence of peripheral neuropathy in the Parsi community of Bombay. Neurology.1991; 41(8): 1315-1317
17. Nardin RA, Amick AN, Raynor EM. Vitamin B12 and methylmalonic acid levels in patients presenting with polyneuropathy. Muscle Nerve. 2007; 36(4): 532-535