ORIGINAL RESEARCH

Clinical profile of peripheral neuropathy patients in tertiary care hospital in northern India

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INTRODUCTION

The peripheral nerves consist of bundles of long neuronal axons as they exit the central nervous system (CNS). The peripheral nerves include the cranial nerves (with the exception of the second), the spinal nerve roots, the dorsal root ganglia, the peripheral nerve trunks and their terminal branches, and the peripheral autonomic nervous system. Peripheral nerves serve different motor, sensory, and autonomic functions.Peripheral neuropathies are typically characterised by distal sensory loss and diminished or lost tendon reflexes, with or without distal weakness and wasting, and affect the lower limbs before the upper limbs (**McLeod JG, 1995; Notermanset al, 1991**). Several disorders can damage peripheral nerves and cause peripheral neuropathy; it is important to differentiate actual neuropathy from other disorders that can have a similar clinical presentation. The prevalence in the general population may be as high as 2.4percent (**HughesRA,2002**). The prevalence of peripheral neuropathy in patients with type 2 diabetes mellitus is estimated to be around 30 percent **(Davies et al, 2006)** . In the developed world, diabetes mellitus is the most common cause of peripheral neuropathy. In India and other developing countries, the incidence of diabetes has increased; therefore, the incidence of diabetic neuropathy is also likely to increase. The clinician has to determine the underlying treatable cause, which can be achieved by adopting a systematic approach. Diagnostic algorithm for peripheral neuropathy has been published previously (**EnglandJD etal,2004**).

In the early stages of peripheral neuropathy, patients typically present with progressive symptoms, including sensory loss, numbness, and pain or burning sensations in distal limbs in a "stocking and glove" distribution. Over time, the numbness may extend proximally, and mild distal muscle weakness and atrophy may occur. In disorders that cause acute peripheral neuropathy, such as those produced by toxic exposures, patients may present with similar but more fulminant symptoms, and pain predominates; symptoms also typically have a faster progression. In other disorders, such as acute inflammatory demyelinating disorder(i.e.,Guillain-Barrésyndrome) and chronic inflammatory demyelinating polyneuropathy, weakness rather than sensory loss typically predominates and may be the earliest sign of thedisease.

The presence of neuropathic symptoms, decreased ankle reflexes, and decreased distal sensations, regardless of distal muscle weakness and atrophy, makes the diagnosis of peripheral neuropathy likely (**England JD et al, 2005)**.

Neuropathies can be categorized according to the fiber type that is primarily involved. Most toxic and metabolic neuropathies are initially sensory and later may involve the motor fibers. Pure sensory neuropathies or neuronopathies can result from drug toxicity (e.g., thalidomide, cisplatin), paraneoplastic syndromes or nutritional deficiencies (**Thomas PK et al, 1993; Donofrio PD et al, 1990**). Primarily motor neuropathies include Guillain-Barré syndrome. Alcoholism and diabetes can both cause small-fiber, painful neuropathies. Autonomic involvement occurs in many small-fiber neuropathies but can also occur in Guillain-Barré syndrome and is sometimes life-threatening. It is important to distinguish whether the neuropathy is axonal, demyelinating, or both. This differentiation is best achieved using nerve

conduction studies (NCS) and electromyography (EMG).

AIM

This observational study aimed at studying the clinical profile of peripheral neuropathy patients undergoing treatment in the department of Medicine, Government Medical College Jammu.

MATERIAL AND METHODS

Study design: This single center hospital basedObservational study was conducted prospectively on patients of peripheral neuropathy admitted under the dept of General Medicine, Government Medical College, Jammu. Time duration of this study was 2 years.

Inclusion criteria was: Patients presenting with one or more complete bilateralperipheral neuro deficits. Patients willing to give informed consent for participation in thisstudy and for laboratory investigation, lumbar puncture, nerve conduction study, electromyography as per the studyrequirement.

Exclusion criteria: included Neurodeficits arising from radiculopathy, injuries and stroke.

Method of data collection: After approval from institutional ethics commitee and consent from patientsfollowing data was collected: sociodemographic profile of patients , detailed history and examination, Lab investigations including routine blood work and csf analysis (wherever required) was done. Additional tests in selected patients included ANA, RA factor, serum electrophoresis, HIV, anti HCV, cryoglobulins, Vit B-12 / folate levels were done. Electrodiagnosticstudiesweredoneifthediagnosisremaineduncleararfter initial diagnostic testing and a careful history and physicalexamination

DATA ANALYSIS

The collected data was first entered on Microsoft Excel spreadsheet. For descriptive statistics, data was grouped, tabulated and represented as percentages.P value <0.05 was considered as statistically significant.

RESULTS AND ANALYSIS

This study included 80 patients of peripheral neuropathy out of which 23%(19)were female and 77% (61) were male. The mean age of the patients was 46 years. Age group of 60-70 years had the maximum number of patients.

Fig 1: Distribution of the causes of peripheral neuropathy

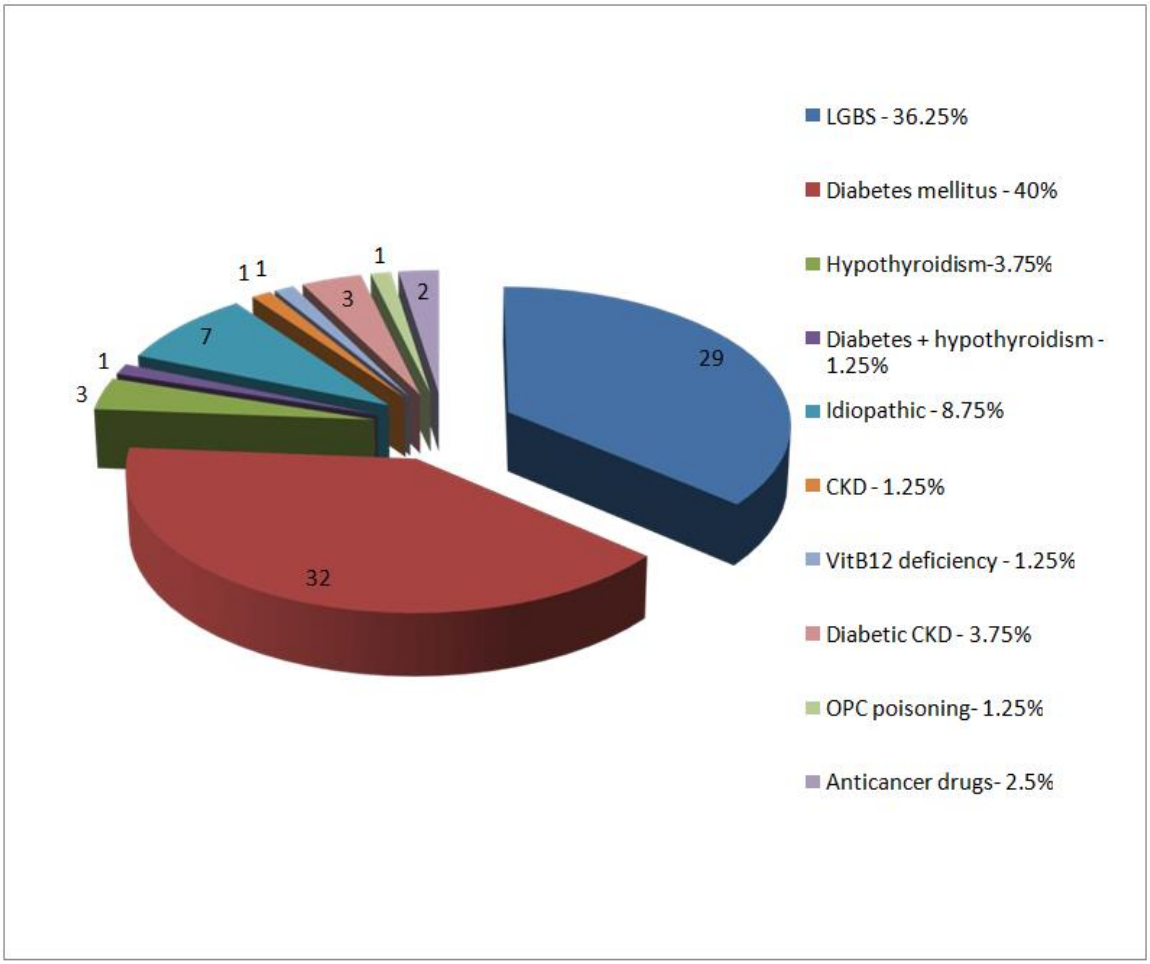
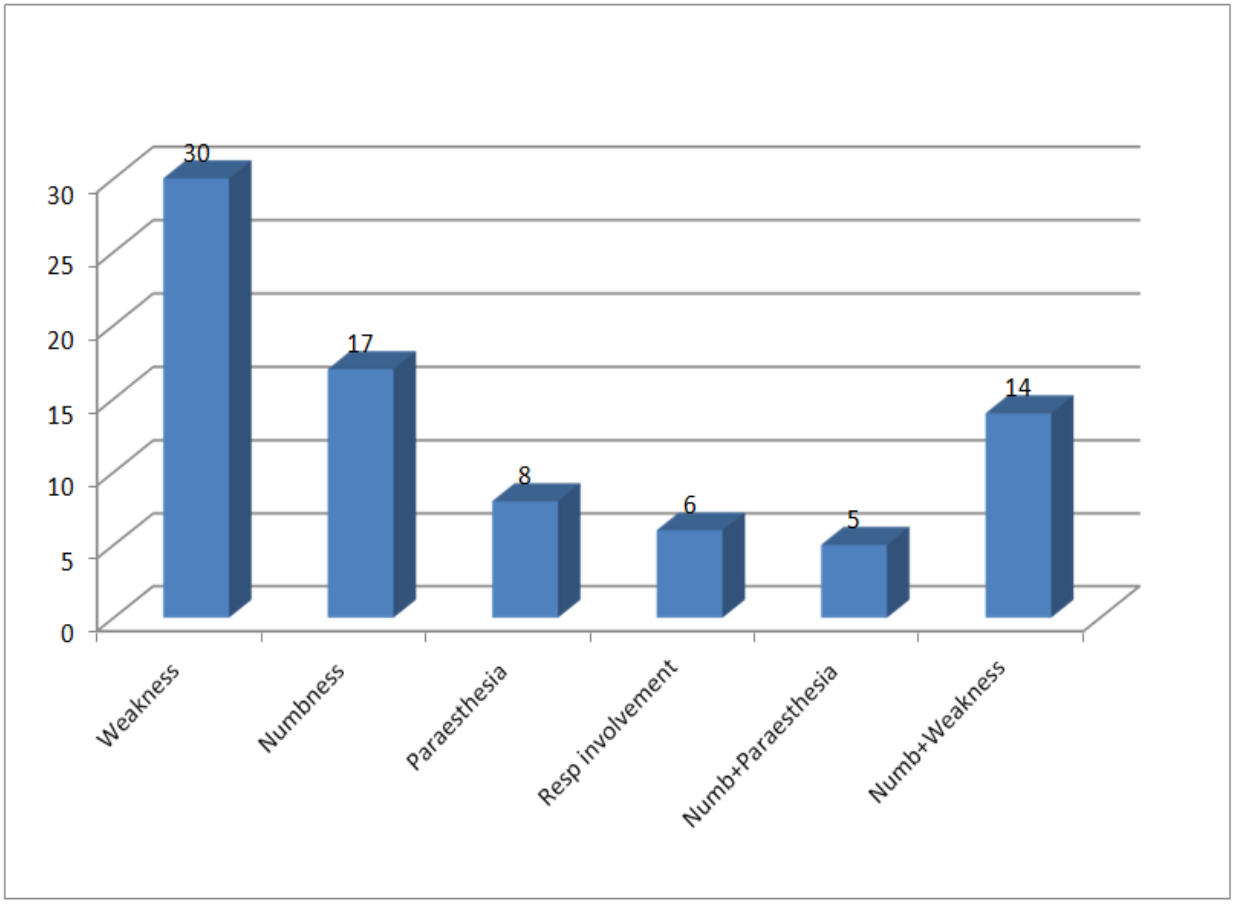


Table 1: Baseline characteristics of the patients

	Minimum	Maximum	Mean	Std deviation
Age (yrs)	16	89	**46.2**	18.3
Weight(kg)	45	78	**61.1**	7.5
Height(cm)	151	182	**163.9**	7.4
BSA(m^2)	1.41	1.98	**1.66**	0.13
BMI(kg/m^2)	16.1	28.3	**22.6**	2.36
Duration (days) LGBS	1	10	**4.03**	2.7
Duration(days) Others	20	200	**83.02**	50.9
SBP(mmHg)	100	160	**125.4**	13.1
DBP(mmHg)	60	100	**82.1**	9.6

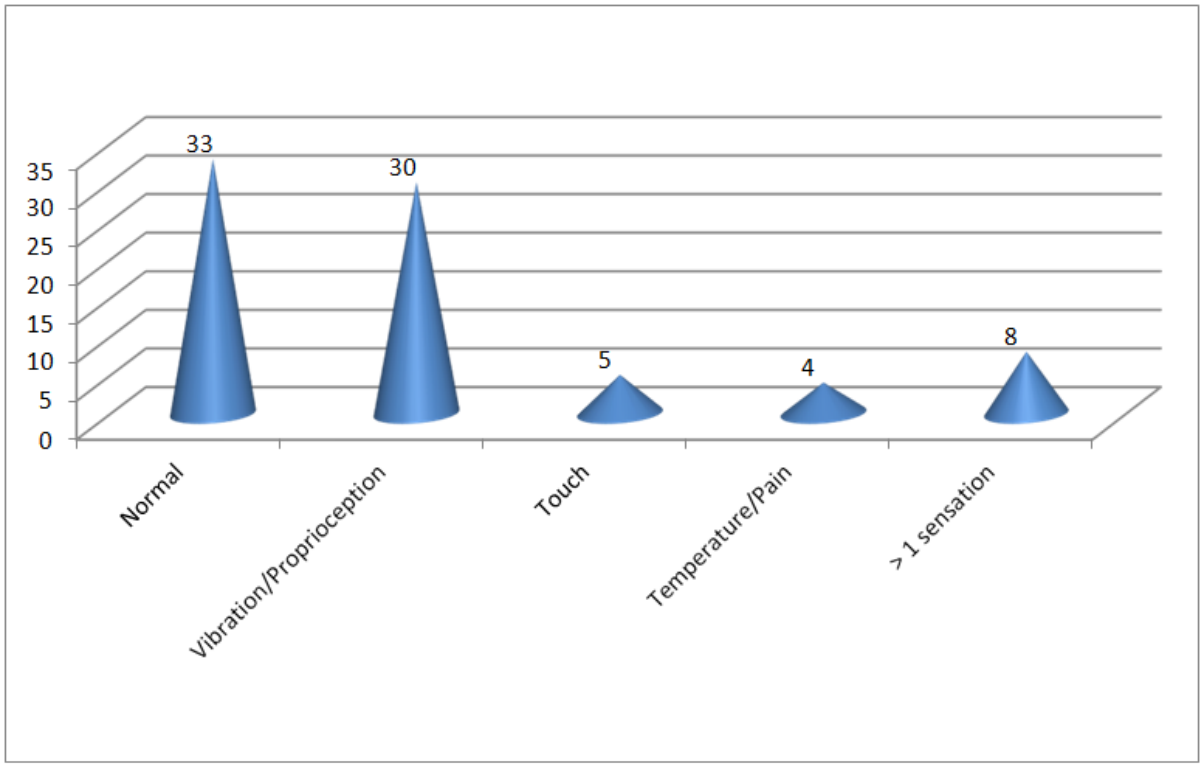
BSA- body surface area, BMI- body mass index, LGBS- Landry Guillain–Barré syndrome, BP- systolic blood pressure, DBP – diastolic bloodpressure

Fig 2: Presenting complaints of the patients



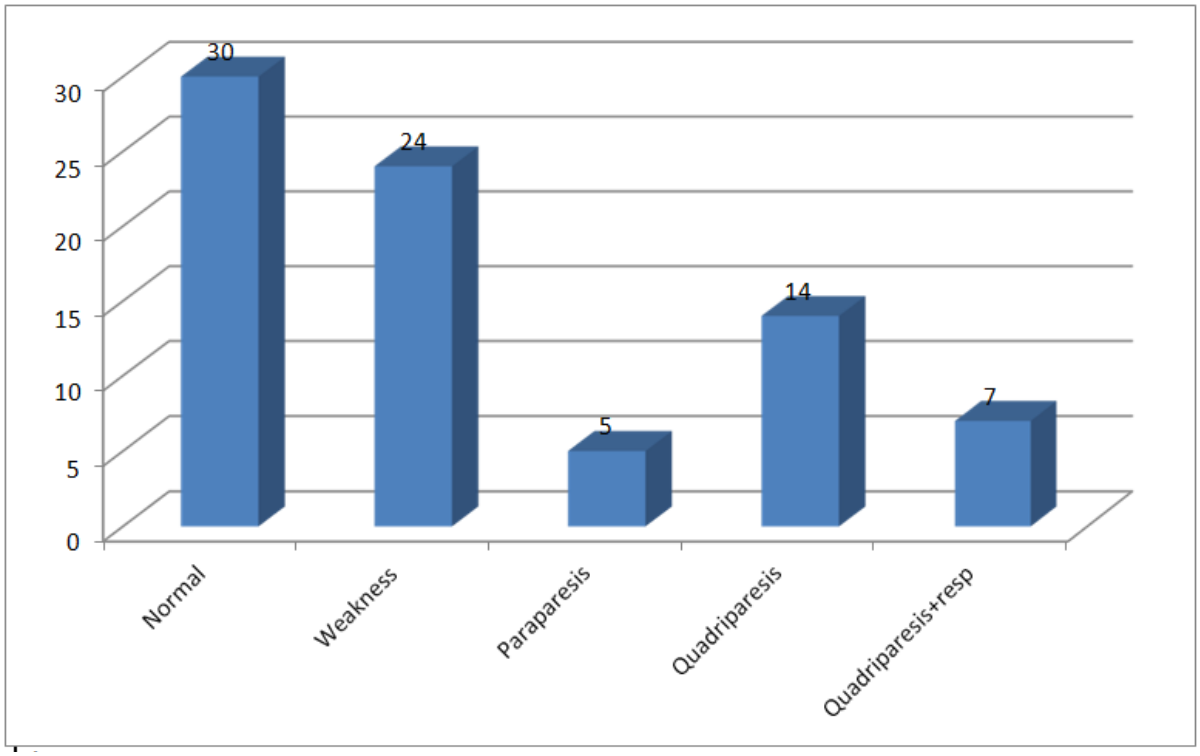
6 out of the 29 patients with LGBS had respiratory discomfort ranging from mild dyspnoea to respiratory arrest requiring ventilator support. Paraesthesia was most frequent in the diabetic patients.

Fig 3: Sensory deficits in the patients



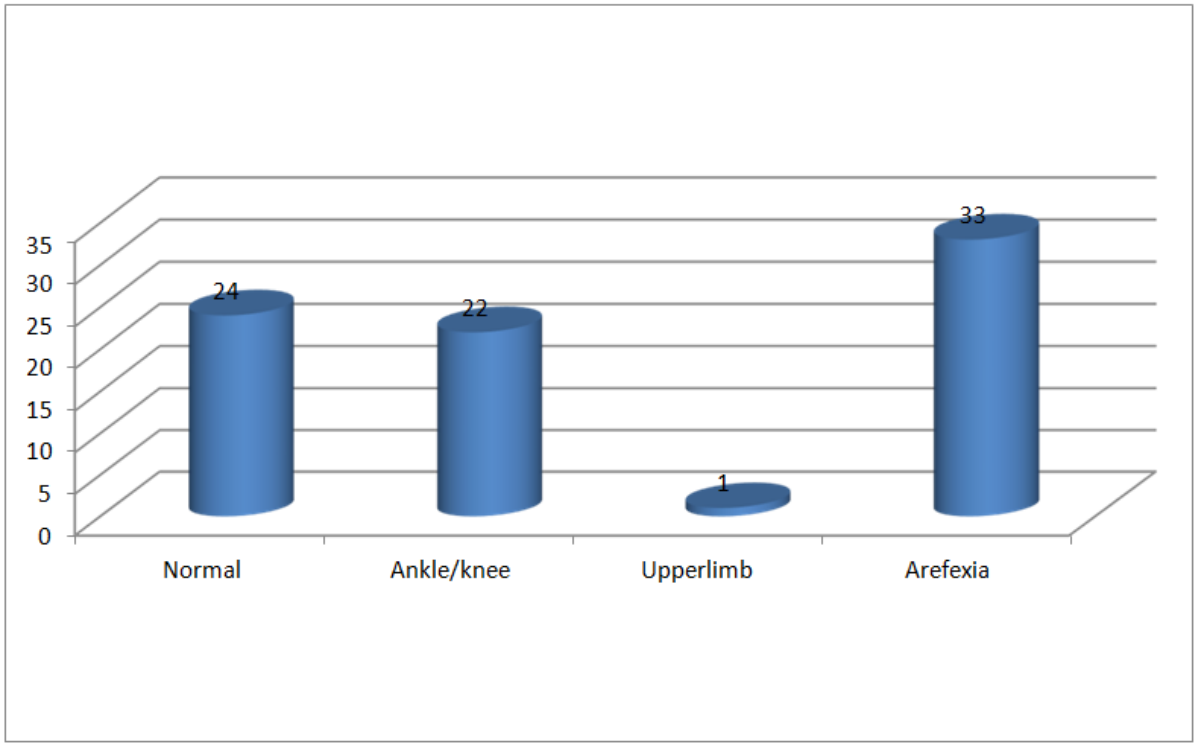
Painful sensory neuropathy was most evident in patients who had received cisplatin and vincristine as treatment for underlying malignancy. The patient who had deficiency of Vit B12 presented with striking loss of the posterior column sensations including fine touch, proprioception and vibration. This patient had marked gait abnormality.

Fig 4: Motor deficits in the patients



Weakness of both lower limbs was seen in 6.25% of the patients and most of these were elderly diabetics.the most common abnormality in diabetic patients was absent or weak lower limb reflexes with intact upper limb reflexes.

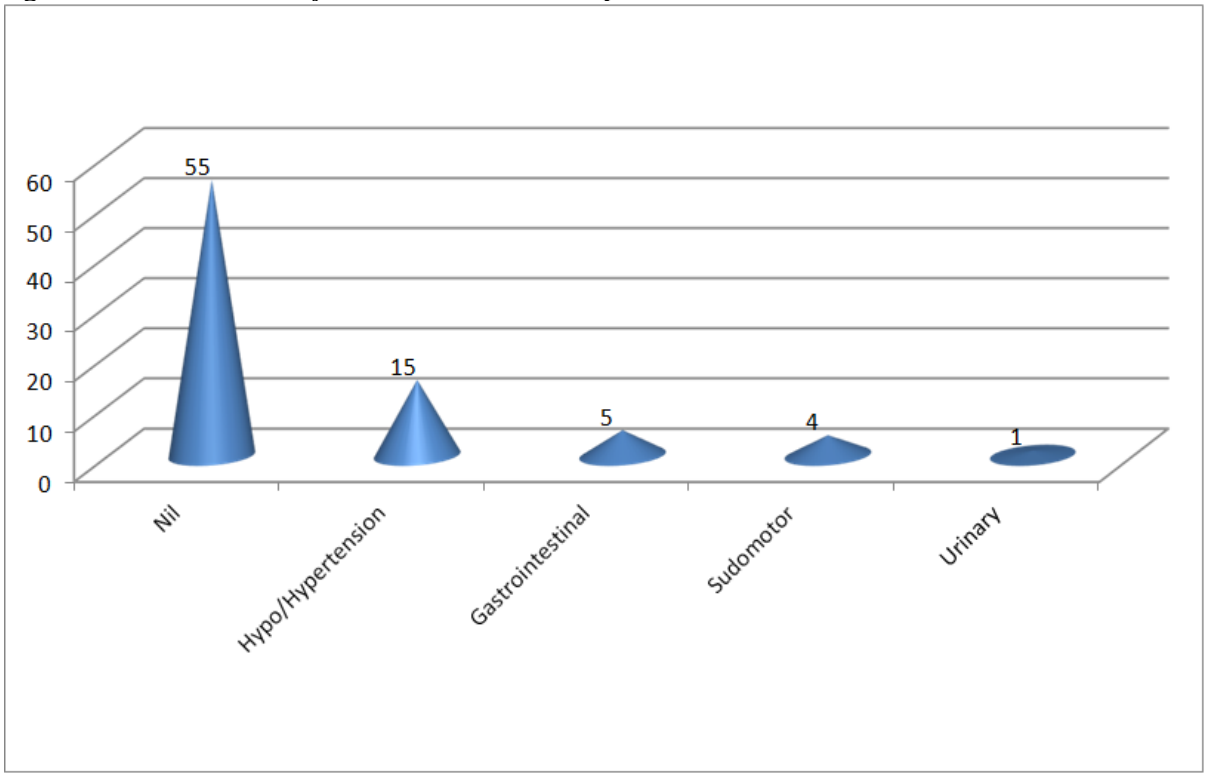
Fig 5: Deep tendon reflexes impairment in the patients



Complete areflexia patients mostly included LGBS as the underlying diagnosis. The patient with organophosphorus compound poisoning had complete areflexia. Two patients with idiopathic peripheral neuropathy also had a complete loss of reflexes. One of the diabetic patients had complete areflexia as well.

Autonomic nervous involvement was largely restricted to the patients with LGBS as cause of peripheral neuropathy. Facial nerve (41%) was the most commonly nerve involved in the LGBS patients and most of these patients had bilateral facial palsy. We didnt encounter any cranial nerve palsies in the diabetic patients.

Fig 6: Autonomic nervous system involvement in the patients



31% of the patients had autonomic symptoms. Autonomic involvement was most frequent in diabetic patients.

DISCUSSION

Peripheral neuropathy accounts for a frequent problem reffered to neurologists and Neuromusculararclinics. Peripheral neuropathy occurs as a component of several commonand many rare disease. It is heterogenous in aetiology, diverse in pathology and varied in severity. Except in the areas of diabetesand Guillain barre syndrome,there have been few epidemiological investigation of peripheral neuropathy. A major obstacle to epidemiological studies of symmetric peripheral sensory neuropathy has been the lack of agreement on suitable operational definition. Definitions that require electrophysiological studies, expensive equipment (eg, computer-assisted sensory examination), or invasive testing(eg,skin or nerve biopsy) makes larger study difficult.

In our study clinical profile of 80 patients presenting with signs and symptoms of Peripheral Neuropathy showed Guillain-Barré syndrome and diabetic patients constituting almost ¾ of total study population supported by study done by **MoldJWetal(2004)**which showed 17%prevalence of peripheral neuropathy in diabetics.

Baghiet al (1995) also showed higher prevalence (18.3%) of polyneuropathy in diabetic patientsit also demonstrated diabetes as the commonest independent risk factor for polyneuropathy.

Rangananthanet al(2009) also showed diabetes as the most common cause of peripheral neuropathy followed byGuillain barre syndrome.In 9% cause was unknownin this study, despite all investigations. In other studies also proportion of idiopathic/cryptogenic peripheral neuropathy has been variable ranging from 13-22% (**DYEK et al,1981)**comparable to our study.

Johannsen et al(2001) showed 25% prevalence of unknown causes and age being significantly associated with polyneuropathy.

In a study done in parsi community by **Bharucha et al,(1991)** showed compressive neuropathy as most common cause and diabetes mellitus as the most common cause among non- compressive neuropathy patients.

Fewer studies has been done in Indian population.**Saseet al,(2009)** showed diabetes mellitus (35%) followed byGuillain barre syndrome (21%)as the common causes of peripheral neuropathy, comparable to our study. Average age in this study was 50 which was comparable to ours.Combined demyelinating and axonal neuropathy was most common finding in this study. In contrast, our study subjects more often had isolated axonal or demyelinating neuropathy. Isolated axonal neuropathy being most common in diabetic and diabetic chronic kidney disease patients and demyelinating neuropathy being common in vit b 12deficiency patients ,in our study.

Ranganathan et al, (2009)alsoshowed weakness of limbs (54%)as the most common presenting symptom majority of the patients had axonal neuropathy .

LIMITATIONS

In addition to small sample size, Patients included in our study were not true representation of true burden of peripheral neuropathy in present population. Nerve Conduction Study was conducted in few patients owing to technical constraints.

CONCLUSION

Early identification of the causative factor of peripheral neuropathy is necessary for timely intervention and for the management of the patients for better outcome and to prevent long term morbidity.Diabetes is among the commonest cause of peripheral neuropathy worldwide specifically in developing countries like India its rampant so its imprudent to timely diagnose and manage complications of diabetes. Peripheral neuropathy being one of its most common and major complication needs to be assessed on time for better life quality of patients. India being resource constraint country, efficient and cost effective diagnostic techniques are required for timely diagnosis of peripheral neuropathy.

BIBLIOGRAPHY

1. Dyek PJ, Oviatt KF, Lambert EH, intensive evaluation of referred unclassified neuropathies yields improved diagnosis. Ann Neurol1981;10:222-6
2. England JD, Asbury AK. Peripheral neuropathy. Lancet.2004;363:2151-61
3. MoldJW,VeselySK,Keyl BA, Schenk JB, Roberts M: The prevalence, predictors, and consequences of peripheral sensory neuropathy in older patients. J Am Board Fam Pract2004;17:309-18.
4. Renganathan R, Atkinson A, McNamara B: How to work up a patient with polyneuropathy. Ir Med J.2009 Jan;102(1):21-3.
5. Sase NS, Correia PWM : Clinical Profile of peripheral neuropathy – A study of 100 patients. J Assoc Physicians India.2009:57.
6. Beghi E, MonticelliML,AmorusoL,et al. chronic symmetrical polyneuropathy in the elderly -a field screening investigation in 2 italian regions1. Prevalence and general characteristics of the sample.Neurology1995;45:1832-6.
7. Bharucha NE, Bharucha AE, Bharucha EP: prevalence of peripheral neuropathy in the parsi community of Bombay. Neurology1991;41:1315
8. England JD, Asbury AK. Peripheral neuropathy. Lancet.2004:363:2151-61
9. Johannsen L,Smith T, Havsager AM, Madsen C, Kjeldsen MJ, Dalsgaard NJ, Gaist D, Schroder HD, Sindrup SH: Evaluation of patients with symptoms suggestive of chronic polyneuropathy. J Clin Neuromuscul Dis.2001 Dec;3(2);47-52