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RESEARCH ARTICLE

CLINICAL PROFILE OF PERIPHERAL NEUROPATHIES IN HOSPITAL SETTING OF KASHMIR, INDIA

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Abstract

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Objective: To study the clinical profile of peripheral neuropathies in a tertiary care setting.

Methods: 200 patients in the age group of 16-85 years were enrolled for the study. 50.5% of these belonged to middle age group. Besides usual demographic profile, detailed examination and relevant haematological and biochemical investigations, electro-diagnostic studies were conducted in all.

Results: Apart from abnormalities on general physical and systemic examination, sensorimotor dysfunction was seen in 87% subjects, motor system abnormalities in 73.5%, hyporeflexia in 90%, thickened nerves in 3.5% and sensorimotor neuropathy was the commonest presentation found in 60.5% of the study subjects. Diabetic neuropathy was the commonest form (55% subjects) followed by Guillain-Barre syndrome (13% cases).

Conclusion: Peripheral neuropathies are fairly common in population of Jammu and Kashmir. Diabetes mellitus is observed as the commonest cause. Future large sample studies are needed to demonstrate the scenario in more elaborate way.

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INTRODUCTION

Peripheral neuropathy is a common neurological problem in clinical practice, and, because of the variable presentation and disparate causes, a logical and sequential approach is necessary for the evaluation and proper management. Through a combination of clinical findings, electrodiagnostic tests and other relevant laboratory investigations tailored to individual patients, most neuropathies can be categorized by subtypes and on the basis of etiology. Such classifications allow rational assessment of prognosis and treatment options. Treatment modalities are divided into those, that are specific for the subtype of neuropathy and those that are useful for neuropathies in general.¹

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The overall prevalence of peripheral neuropathies is around 2400 per 100000 population, but in people older than 55 years, the prevalence rises to about 8000 per 100000.² In the developed world, diabetes mellitus is the commonest cause of peripheral neuropathy (PN). Since the prevalence of diagnosed diabetes mellitus has increased in general population in the USA, the prevalence of diabetic peripheral neuropathy is also expected to rise³. Although common in the USA and Europe, leprous neuritis is still highly prevalent in south-east Asia, India, Africa, Central and South America. Other common causes of peripheral neuropathy include a range of metabolic disorders, infectious agents, vasculitis, toxins, drugs and inherited polyneuropathies.^{2,4} In India, diabetic neuropathy (DN) has been found to be the commonest cause.⁵ However, no such published study is yet available from the Jammu and Kashmir State of India. To study the pattern and etiology of various types of peripheral neuropathies, formed the basis of the present study. To our knowledge, this is the first study of its kind, from this state.

MATERIAL AND METHODS

This was a prospective study conducted in the Postgraduate Department of Medicine of the Government Medical College, Srinagar - a tertiary care center of Jammu and Kashmir State. This included 200 patients studied from May 2011 to November 2012. The patients recruited for the study were subjected to detailed history, clinical examination and necessary investigations. The age of patients ranged from 16 to 85 years, and maximum (50.5%) were in the middle age group. Majority (73%) of patients were males, and the overall male to female ratio was 2.7:1. Apart from general demographic parameters, a detailed neurological examination was conducted in every study subject. This included study of higher mental functions, cranial nerve examination, and examination of motor and sensory systems, and gait. Baseline investigations included complete hemogram, erythrocyte sedimentation rate (ESR), blood sugar estimation, and study of serum electrolytes, functions of kidneys and liver, and, all other routine biochemical investigations. Chest radiography and electro-cardiography were performed in all the study subjects. Selected subjects were subjected to special investigations like toxin screening of lead and arsenic, retroviral serology using ELISA method, serum and urine electrophoresis, rheumatoid factor, antinuclear antibodies (ANA) and antinuclear cytoplasmic antibodies (cANCA and pANCA), cerebrospinal fluid examination for cell count, protein, glucose and electrophoresis was conducted in selected subjects. Electrodiagnostic study was conducted in all the subjects to observe the pattern and type of various peripheral neuropathies. For motor nerve conduction studies, surface electrodes were used. For antidromic (proximal stimulation with distal recording opposite to physiological direction of impulse flow) and orthodromic (distal stimulation with proximal recording, in the physiological direction of flow for normal sensory impulses) sensory nerve conduction studies, surface ring electrodes and unipolar needle electrodes were used, respectively. Evaluated variables included distal motor latency, motor and sensory conduction velocities and amplitude of compound muscle and nerve action potentials. The variables were considered abnormal when they exceeded mean+SD. A demyelinating neuropathy was assumed if the conduction velocity was < 28 m/sec and compound muscle action potential (cMAP) > 1 m. Axonal neuropathy was assumed if CMAP was $< 1 \square \square$ m with nerve conduction velocity of >28 m/s. Nerve biopsy was done in patients with no etiological diagnosis. These biopsies were conducted on sural nerves, and subjected to histopathological examination. Patients with features of myeloneuropathy, and myelopathy were excluded from the study. Clearance was obtained from the ethical clearance committee of the institution.

Statistical Analysis: The data was compiled and studied by experienced statistician. Student 't' test and chi-square tests were performed wherever necessary, p value of < 0.05 were considered significant.

RESULTS

Among the studied subjects, the clinical presentation was variable (depicted in Table 1). Paresthesias were found in 53%, weakness of both lower limbs was observed in 15.5%, followed by weakness of all the four limbs in 15% of cases. Bilateral distribution was observed in 97.5% cases, insidious onset in 82%, and progressive course was seen in 69.5% of the cases. Duration was from 2 days to 4 years with mean of 135 ± 14 days. Significant family history was present in 20% of subjects, paresthesias prior to weakness in 82%, preceding illness prior to weakness in 11% and diabetes mellitus was the presentation among 67.5% of the subjects. On clinical examination, hypopigmentation was observed in 1% and hyperpigmentation in 9.5% of cases. Chest examination abnormality was found in 6% and cardiovascular abnormalities in 9.5% of the subjects. On neurological examination cranial nerve palsy (facial) was seen in 1.5% cases, (2 had bilateral and one had unilateral involvement) sensory abnormalities were found in 87% subjects, motor system abnormalities were in 73.5%, hyporeflexia in 90%, thickened nerves were observed in 3.5% cases and gait was abnormal in 33% patients. Mixed (sensory and motor) neuropathy was the commonest presentation in 60.5% cases. Pure sensory abnormality was seen in 26.5% and pure motor abnormality

in 13% of cases. Rheumatoid factor and ANA were positive in 3.1% subjects. Serum and urine electrophoresis was positive in 3.1% and nerve biopsy was suggestive of neuropathy in 22.7% cases. Diabetic neuropathy was the commonest (55% cases) type of neuropathy, followed by Guillian-Barre syndrome in 13% subjects (Table 2). Nerve conduction studies revealed senorimotor demyelinating neuropathy in majority of cases (Table 3), and this finding was present in all the cases of uremic neuropathy, and rheumatoid arthritis. Common peroneal nerve was the most commonly involved in majority of the study subjects (Table 4).

| | Table – 1 Clinical Features at Presentation | n | | | | |
|-----------------|--|-----------------|------------|--|--|--|
| Characteristics | | No. of Patients | Percentage | | | |
| Weakness | Both lower | 31 | 15.5 | | | |
| | All 4 limbs | 30 | 15.0 | | | |
| | Parasthesias both lower limbs | 106 | 53.0 | | | |
| | Parasthesia and numbness in both upper limbs | 4 | 2.0 | | | |
| | Parasthesia and numbness in right upper limb | 2 | 1.0 | | | |
| | Parasthesias and weakness in left lower limb | 1 | 0.5 | | | |
| | Parasthesias and weakness in right upper limb | 2 | 1.0 | | | |
| | Parasthesia and weakness in both lower limbs | 24 | 12.0 | | | |
| Distribution | Unilateral | 5 | 2.5 | | | |
| | Bilateral | 195 | 97.5 | | | |
| Onset | Acute | 13 | 6.5 | | | |
| | Subacute | 23 | 11.5 | | | |
| | Incidious | 164 | 82.0 | | | |
| Progression | Progressive | 139 | 69.5 | | | |
| | Static | 22 | 11.0 | | | |
| Duration (day) | Mean \pm SE 135.2 \pm 14.1 (2,14 | | | | | |

| Table – 2 Etiological Classification of Neuropathy | | | | | | | | | |
|---|-----------------|------------|--|--|--|--|--|--|--|
| | No. of Patients | Percentage | | | | | | | |
| Diabetic | 110 | 55.0 | | | | | | | |
| Gullian-Barre syndrome | 26 | 13.0 | | | | | | | |
| Chronic inflammatory demyelinating polyradiculoneuropathy | 18 | 9.0 | | | | | | | |
| Idiopathic | 17 | 8.5 | | | | | | | |
| Uremic neuropathy | 11 | 5.5 | | | | | | | |
| Entrapment neuropathy | 9 | 4.5 | | | | | | | |
| Hypothyroid neuropathy | 4 | 2.0 | | | | | | | |
| Critical illness neuropathy | 3 | 1.5 | | | | | | | |
| Rheumatoid arthritis | 2 | 1.0 | | | | | | | |

| Table – 3 Pattern of Neuropathy According to Nerve Conduction Study in Various Groups of Neuropathies | | | | | | | | | | |
|---|----------------------|------|-----------------------------|------|---|-------|---|------|-------|------|
| Neuropathy | Pure Motor Axonal | | Pure Motor Demyelinating | | Sensorimotor Demyelinating Neuropathy | | Sensorimotor Predominant Axonal Neuropathy | | Total | |
| | n | % | n | % | n | % | n | % | n | % |
| Diabetic | 0 | 0.0 | 0 | 0.0 | 98 | 89.1 | 12 | 10.9 | 110 | 55.0 |
| Gullian-Barre Syndrome | 10 | 38.5 | 5 | 19.2 | 4 | 15.4 | 7 | 26.9 | 26 | 13.0 |
| Chronic Inflammatory Demyelinating Polyradiculo- neuropathy | 0 | 0.0 | 1 | 5.6 | 16 | 88.9 | 1 | 5.6 | 18 | 9.0 |
| Critical illness neuropathy | 0 | 0.0 | 0 | 0.0 | 1 | 33.3 | 2 | 66.7 | 3 | 1.5 |
| Uremic Neuropathy | 0.0 | 0.0 | 0 | 0.0 | 11 | 100.0 | 0 | 0.0 | 11 | 5.5 |
| Entrapment neuropathy | 0 | 0.0 | 0 | 0.0 | 7 | 77.8 | 2 | 22.2 | 9 | 4.5 |
| Hypothyroid neuropathy | 0 | 0.0 | 0 | 0.0 | 3 | 75.0 | 1 | 25.0 | 4 | 2.0 |
| Rheumatoid arthritis | 0 | 0.0 | 0 | 0.0 | 2 | 100.0 | 0 | 0.0 | 2 | 1.0 |
| Idiopathic | 0 | 0.0 | 1 | 5.9 | 13 | 76.5 | 3 | 17.6 | 17 | 8.5 |

| Paper – 4 Percentage of Neuropathy Involvement According to Nerve Conduction Study in Various Groups of Neuropathies | | | | | | | | | | | | | |
|--|-------|------|----|------|---|------|----|-------|----|--------------------|-----|-------|--|
| Neuropathy | Ulnar | | Ra | | | | | Sural | | Common Peroneal | | Total | |
| | n | % | n | % | n | % | n | % | n | % | n | % | |
| Diabetic | 3 | 2.7 | 3 | 2.7 | 2 | 1.8 | 42 | 38.2 | 60 | 54.5 | 110 | 55.0 | |
| Gullian-Barre Syndrome | 1 | 3.8 | 4 | 15.4 | 5 | 19.2 | 7 | 26.9 | 9 | 34.6 | 26 | 13.0 | |
| Chronic Inflammatory Demyelinating Polyradiculo- neuropathy | 2 | 11.1 | 3 | 16.7 | 4 | 22.2 | 4 | 22.2 | 5 | 27.8 | 18 | 9.0 | |
| Critical illness neuropathy | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 1 | 33.3 | 2 | 66.7 | 3 | 1.5 | |
| Uremic Neuropathy | 1 | 9.1 | 1 | 9.1 | 2 | 18.2 | 3 | 27.3 | 4 | 36.4 | 11 | 5.5 | |
| Entrapment neuropathy | 7 | 77.8 | 1 | 11.1 | 1 | 11.1 | 0 | 0.0 | 0 | 0.0 | 9 | 4.5 | |
| Hypothyroid neuropathy | 3 | 75.0 | 0 | 0.0 | 1 | 25.0 | 0 | 0.0 | 0 | 0.0 | 4 | 2.0 | |
| Rheumatoid arthritis | 1 | 50.0 | 0 | 0.0 | 1 | 50.0 | 0 | 0.0 | 0 | 0.0 | 2 | 1.0 | |
| Idiopathic | 3 | 17.6 | 1 | 5.9 | 2 | 11.8 | 3 | 17.6 | 8 | 47.1 | 17 | 8.5 | |

Table – 4

DISCUSSION

Peripheral neuropathy is a common neurological problem with variable presentations caused by various infective,^{6,7,8} non-infective causes,^{9,10,11} and variety of other etiologies.^{12,13,14,15} In our study, majority of the patients were males. In India, similar was the finding in the study of Goel and co-workers⁵. Regarding insidious mode of onset in 82% of our subjects, the findings are similar to the study of Sase and co-workers¹⁶ from India. They reported insidious onset of the illness in 60% and bilateral presentation amongst 72% of the study subjects. Again Chuttani and Chawla¹⁷ have observed lower values of clinical symptomatology compared to our study. They found cranial nerve palsy in 1.5% cases compared to 5.8% in our study. Similar observations have been in the study of Lee HS and co-authors.¹⁸ Diabetes was the commonest cause of neuropathy among our case series. Similarly, diabetic neuropathy has been found the most common in previously published studies as well.^{16,19,20} The next common cause of neuropathy observed in our series was the Guillian-Barre syndrome, found in 13% of the cases. Again in this regard similar have been the observations of the previous studies.^{5,17} Baheti²¹ and co-workers reported that there have been several descriptions of reflex preservation and hyper-reflexia in axonal variant of GBS in Chinese, Japanese and European populations, but it is not common in the Indian subcontinent. Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) was observed in 11% of our case study. Previous studies^{22,23} have also demonstrated it in the same range, however, Lubee and co-workers²⁴ have observed CIDP in only 4% of patients.

Sase and co-workers¹⁶ reported entrapment neuropathy in 9% of patients, whereas only 4.5% of our study subjects had this type of neuropathy. The ulner nerve entrapment found in 77.8% of our patients, is the second most common cause of peripheral nerve involvement after carpel tunnel syndrome as reported previously.²⁵ Critical illness neuropathy representing an acute axonal neuropathy developing during acute illness, was observed in 3 (1.5%) of our patients. This entity has been increasingly recognized over last two decades.^{26,27,28} Hypothyroidism. as cause of sensorimotor neuropathy, was found in 4 (2%) of our study subjects. However, Duff²⁹ and Yeasmin³⁰ reported it in 19% and 67.5% of hypothyroid patients, respectively.

To conclude in future, more studies using large population samples are needed to clarify the profile of neuropathies in variety of medical disorders.

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