Section: Neurology

Original Research Article

CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY: AETIOLOGY, CLINICAL AND ELECTROPHYSIOLOGICAL CHARACTERISTICS, AND PROGNOSTIC OUTCOMES

Nataraja Poli[1], Anil Kumar Kasukurthy[2], Bhuma Vengamma[3]

[1]*Assistant Professor, Department of Neurology, Sri Venkateswara institute of medical sciences (SVIMS), Tirupati, A.P, India.*
[2]*Assistant Professor, Department of Neurology, Sri Venkateswara institute of medical sciences (SVIMS), Tirupati, A.P, India.*
[3]*Senior Professor & HOD, Department of Neurology, Sri Venkateswara institute of medical sciences (SVIMS), Tirupati, A.P, India.*

Received *: 11/05/2025*
Received in revised form *: 05/07/2025*
Accepted *: 29/07/2025*

Corresponding Author:
Dr. Anupam Johri,
Professor, Department of Forensic Medicine, RUHS - College of Medical Science, Jaipur, Rajasthan, India.
Email: anupamjohry@gmail.com

DOI: 10.70034/ijmedph.2025.3.215

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 1160-1167

ABSTRACT

Background: The peripheral nervous system is made up of large myelinated motor and sensory axons responsible for transmitting proprioception, vibration, and light touch sensations; small myelinated axons that transmit light touch, pain, temperature, and preganglionic autonomic functions; and small unmyelinated axons that carry pain, temperature, and postganglionic autonomic functions. **Objectives:** • To Study the Aetiology, Clinico Electro Physiological profile of CIDP. • To study the outcome of CIDP.

Material and Methods: All patients who were admitted in inpatient services of Neurology department of Sri Venkateswara Institute of Medical Sciences (SVIMS), Tirupati, with the diagnosis of CIDP during the period of September 2015 to August 2016, were prospectively studied. In all of them demographic informations, past medical history, clinical presentation, routine hematological work-up, biochemical and Electrophysicological studies were recorded, cerebrospinal fluid analysis, Nerve Biopsy, Skin Biopsy were done in whom it was relevant and possible.

Results: Out of 42 patients, 18(42.8%) patients had symmetrical prox + distal sensory and motor symptoms, 13(30.9%) patients had symmetrical sensory symptoms, Asymmetrical sensory and motor symptoms (ASSM) in 8(19%) patients and 4(9.5%) patients had Distal symmetric sensory and motor symptoms (DSSM).

Conclusion: This study of 42 CIDP patients found sensorimotor symptoms (62%) and mixed axonal-demyelinating neuropathy (42.8%) as the most common features. While 57.1% were idiopathic, 19% had diabetes, and nerve biopsy revealed vasculitis in 50% of tested cases. Steroids were effective (61.9%), with 45.2% achieving partial remission.

Keywords: CIDP (Chronic Inflammatory Demyelinating Polyneuropathy), Demyelinating Neuropathy, Axonal Neuropathy, Steroid Treatment, Vasculitis.

INTRODUCTION

The peripheral nervous system is made up of large myelinated motor and sensory axons responsible for transmitting proprioception, vibration, and light touch sensations; small myelinated axons that transmit light touch, pain, temperature, and preganglionic autonomic functions; and small unmyelinated axons that carry pain, temperature, and postganglionic autonomic functions. Peripheral neuropathy has a variety of presentations and numerous underlying causes, necessitating a systematic and logical approach for an efficient diagnosis, particularly for neuropathies that can be treated. A comprehensive history of symptoms, family background, and occupational information, alongside general and systemic evaluations, assists in narrowing down the potential causes of neuropathy. Neurological assessments focusing on sensory, motor, and autonomic signs help clarify the location

and type of neuropathy. The absence of joint, position, and vibration sense along with sensory ataxia suggests large fiber neuropathy, whereas small fiber neuropathy is characterized by disruptions in pain, temperature, and autonomic functions.

Electrodiagnostic (EDx) assessments encompass sensory and motor nerve conduction tests, F response, H reflex, and needle electromyography (EMG). SEDx is useful for documenting the degree of sensory motor impairments and distinguishing between demyelinating (characterized by prolonged terminal latency, reduced nerve conduction velocity, temporal dispersion, and conduction block) and axonal (exhibited by slight slowing of nerve conduction and diminished compound muscle or sensory action potentials, along with denervation on EMG) types. Consistent demyelinating characteristics suggest a hereditary form of demyelination, while variations observed between different nerves and segments within the same nerve indicate acquired demyelination. Neuropathy is categorized into mononeuropathy, often resulting from entrapment or trauma; mononeuropathy multiplex, typically associated with leprosy and vasculitis; and polyneuropathy, which arises from systemic, metabolic, or toxic causes. Laboratory tests are performed as necessary, and specialized investigations such as biochemical, immunological, genetic analysis, cerebrospinal fluid (CSF) testing, and nerve biopsy are conducted for select patients. The main treatment methods include corticosteroids, plasma exchange, and IVIg. Alternative immunosuppressants are reserved for patients who do not respond well, experienced frequent relapses, or cannot tolerate the side effects of the primary treatment.

Objectives

- To Study the Aetiology, Clinico Electro Physiological profile of CIDP.
- To study the outcome of CIDP.

MATERIALS AND METHODS

All patients who were admitted in inpatient services of Neurology department of Sri Venkateswara Institute of Medical Sciences (SVIMS), Tirupati, with the diagnosis of CIDP during the period of September 2015 to August 2016, were prospectively studied. In all of them demographic informations, past medical history, clinical presentation, routine hematological work-up, biochemical and Electrophysicological studies were recorded, cerebrospinal fluid analysis, Nerve Biopsy, Skin Biopsy were done in whom it was relevant and possible.

Diagnosis of CIDP was made by the presence of symmetric / Asymmetrical, Progressing, relapsing positive and negative signs and symptoms of sensory, motor autonomic system more than 2 months duration.

Inclusion Criteria

- Patients with signs and symptoms of peripheral neuropathy with duration beyond 2 months.
- Patients with electro physiology suggestive of neuropathy.

Exclusion Criteria

Patient who are not willing to participate in the study.
Patient with clinical features "Neuropathy less than 2 months duration.

ETHICAL CLEARANCE

Institutional Ethical Committee clearance was obtained for conducting the study. (A & E /08/IEC/SVIMS/09.IEC NO329. TAC 206.)

INFORMED CONSENT

Written informed consent was obtained from all patients or from next responsible attendant in case the patient is unconscious for participation in study (Appendix 1).

Clinical and Electrophysiological evaluation of patients with CIDP

A detailed clinical history was taken from patient or by relative including demographic data regarding age, sex, etc.

The clinical parameters like symptoms onset, duration from onset to admission in neurology clinic, progressive of symptoms, weakness of upperlimbs, lower limbs, neck flexor weakness, trunk muscle weakness, bulbar symptoms, secondary symptoms were elicited. Informed consent was taken for the study. Disabilities were evaluated using Hughes functional grading at the peak of illness and during follow-up period.

Hughes Functional Grading[1]

Score	Functional Status
0	Normal
1	Able to run with minor symptoms & signs
2	Able to walk 5 metres independently
3	Able to walk 5metres with Aid
4	Chair bound/Bed ridden
5	Requiring assisted ventilation
6	Dead

On follow up, Partial remission with Hughes grade 1-2 (able to walk or run), Severe disability with Hughes grade 3 or more (unable to walk) was taken for treatment response.

Nerve conduction studies were performed with Neuropack ∑(Sigma) machine. Motor Nerve conductions were performed on bilateral median, ulnar, common peroneal Nerve and Posterior Tibial neurve in all the patients. Sensory Nerve conduction was one on bilateral medial, ulnar, and sural nerves. Compound muscle action potential, distal motor latency, motor nerve conduction velocity, conduction

block, temporal dispersion, sensory nerve action potential, distal sensory latency, sensory nerve conduction velocity, F wave persistence and F- wave latency were recorded all the patients. H-reflex and sympathetic skin response were recorded whenever needed.

Nerve conduction studies:

Electrophysiological studies play an important role in confirming the diagnosis of CIDP. Standard nerve conduction studies are done, using surface recording electrodes. Motor nerve conduction studies include recording from abductor polices brevis on median nerve stimulation, from extensor digitorum brevis on peroneal nerve stimulation, from abductor digiti minimi on ulnar nerve stimulation and from abductor hallucis on tibial nerve stimulation. Motor nerve conduction studies include assessment of compound muscle action potential (CMAP) amplitude, distal motor latency (DML) and muscle nerve conduction velocity (MNCV) along with assessment for temporal dispersion and conduction block. The electrophysiological study also evaluates F waves (absence, latency) in multiple motor nerves. Orthodromic (or) antidromic studies are done for sensory nerve conduction. Sensory nerve conduction evaluates sensory nerve latency Sensory nerve action potentiall (SNAP) and sensory nerve conduction velocity. Orthodromic studies on median, ulnar, and antidromic studies on sural nerve are done for sensory nerve conduction study.

Statistical Analysis

Data will be recorded on a predesigned proforma and managed using Excel 2007 (Microsoft Corporation, Redmond, WA, USA). All the entries will be double-checked for any possible error. Descriptive statistics for the categorical variables will perform by computing the frequencies (percentage) in each category. For the quantitative variables, approximate normality of the distribution will be assessed. Variables following normal distribution will be summarized by mean and standard deviation. The outcome will analyze in terms of functional recovary. The association between categorical variables and the outcome will be evaluated by c2 test or Fisher's exact test as appropriate. For quantitative variables Student's 't'-test (for normally distributed variables), Mann-Whitney U test (for variables that are not normally distributed) will be used to compare the difference in mean and median values respectively in the two groups (recovered vs. not). Statistical software package IBM SPSS Statistics 20 (I.B.M. Corporation, Chicago, IL, USA) will be used for statistical analysis.

RESULTS

Total of 42 patients with CIDP were studied prospectively during the study period, who were admitted in Department of Neurology of Sri Venkateswara Institute of Medical Sciences, Tirupati.

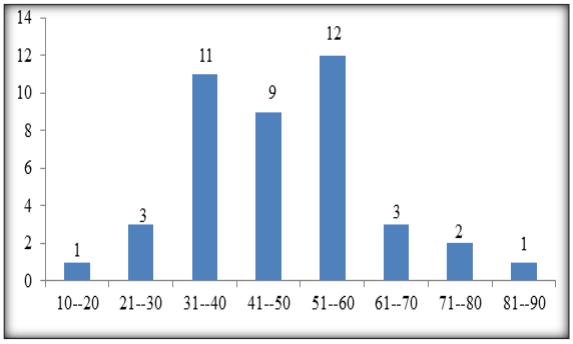

Figure 1: Age distribution in the study population

Mean age of patients with CIDP was 46.95+ 14.62, with a range of 15-82 years. Most of the patients were in middle age group. In the present study, majority of patients were male with male: female ratio of 1.82:1. Mean duration of illness of patients with CIDP was 22.48+ 28.82 months with a range of 2 months – 120 months. Clinical Symptomatology: Out of 42 patients, 26 patients (62%) had sensory + Motor symptoms, 13 patients (31%) had only sensory symptoms and 3 patients (7%) had only pure motor symptoms.

Table 1: Symptoms of 42 patients

Symptoms	No of pts(%)
Motor Symptoms	
Prox+ Distal	25(59.5%)
Prox	1(2.6%)
Distal	3(7.1%)
Sensory Symptoms	
Paresthesias of both feet	25(59.5%)
Paresthesias of both feet & Hands	11(26%)
Diminished pain	2(4.7%)
Burning sensation	1(2.3%)
Cranial Nerves	
Dysphagia	3(7.1%)
Facial deviation	1(2.3%)
Autonomic manifestations	
Orthostatic Hypotension	1(2.3%)

In motor symptoms: Out of 42 patients. 25 patients had both proximal and distal weakness. 3 patients had distal weakness and 1 patient had proximal weakness, 13 patients had no motor symptoms.

Sensory Symptoms: Out of 42 patients, 25 patients had paresthesias of both feet, 11 patients had paresthesia of both feet and hands, 2 patients had diminished pain sensation and 1 patient had burning pains and 3 patients have no sensory symptoms.

Table 2: Clinical Signs of 42 pts of CIDP

Variable	Sensory + Motor (26)	Sensory (13)	Motor (3)
Weakness			
Prox + Distal	23	-	2
Prox	1		
Distal	2		1
Pain diminished			-
Distal to ankle	16	10	
Distal to wrist	8	3	
Definitive sensory level	2		
Light touch diminished			-
Distal to ankle	16		
Distal to wrist	8	10	
Definitive sensory level	2	3	
Vibration diminished			
Below knee	16	10	-
Below Ulnar Styloid process	8	3	
Vertebral level			
	2		
Joint,position sense			-
Great toe	6	10	
Middle finger	20	3	
Romberg sign	16	13	
DTR			
Total areflexia	17	10	2
Ankle absent only	9	3	-
Abnormal finger flexion reflex	1		1
Wasting	7	-	1
CNS involvement	1		-
Extensor plantor response	2	-	
Definitive sensory level	1		
Stroke			
Autonomic involvement			
Orthostatic Hypotension	1	-	-
Cranial nerves		-	-
7th nerve	1		
9th & 10th nerve	3		

Cranial Nerves :4 patients had cranial nerve involvement, in which 3 patients had 9th and 10th Nerve involvement and 1 patient had 7th nerve involvement. One Patient had orthostatic hypotension. One Patient had respiratory failure, who was treated with mechanical ventilation.

Central Nervous System: CNS involvement seen in 4 patients in which 1 patient had extensor plantar response, 2 patients had definitive sensory level and 1 patent had stroke.

Neuropathic pattern: Out of 42 patients, 18(42.8%) patients had symmetrical prox + distal sensory and motor symptoms, 13(30.9%) patients had symmetrical sensory symptoms, Asymmetrical sensory and motor symptoms (ASSM) in 8(19%) patients and 4(9.5%) patients had Distal symmetric sensory and motor symptoms (DSSM).

Table 3: Electrophysiological study of 42 pts

NERVE	↓CMAP/SNAP	ABSENT CMAP/SNAP	↓CV	↑DL	↑ F WAVE	ABSENT F WAVE
MOTOR		-				
MEDIAN	15(36%)	2(5%)	11(26%)	8(19%)	5(12%)	2(5%)
ULNAR	15(36%)	1(2.3%)	11(26%)	4(9.5%)	8(19%)	5(12%)
CP	22(52%)	11(26%)	17(40%)	9(21%)	5(12%)	14(33%)
PT	21(50%)	14(17%)	17(40%)	10(24%)	7(17%)	10(24%)
SENSORY						
MEDIAN	16(38%)	12(28%)	5(12%)	5(12%)		
ULNAR	16(38%)	16(38%)	6(14%)	5(12%)		
SURAL	14(33%)	25(60%)	11(26%)	10(24%)		

In present study of 42 patients decreased CMAP in15(36%) median, 15(36%)ulnar motor ,22(52%) common peroneal nerves, 21 (50%) posterior tibial nerves, decreased SNAP in 16(38%)median,16(38%)ulnar nerves and 14 (33.3%) sural nerves, inexcitable 2(5%)median

nerves, 1 (2.3%) ulnar motor,11(26%) common peroneal nerves, 14(33.3%)posterior tibial nerves,12(28%) median sensory nerves,16(38%) ulnar sensory nerves,25(60%)sural nerves, decreased conduction velocity seen in 11(26%)median, ulnar nerves each 17 (40.4%) common peroneal posterior tibial nerves each, ,5(12%)median, 6 (14.2%) ulnar sensory, and 11(26%) sural nerves, prolonged distal latency in 8 (19%)median,4(9.5%) ulnar,9(21%) common peroneal and 10(24%) posterior tibial nerves,5(12%) median, ulnar sensory nerves each and 10(24%) sural nerves, prolonged F wave in 5(12%)median,8(19%) ulnar,5(12%)common peroneal and 7(17%) posterior tibial nerves, absent F waves in 2(5%)median, 5(12%)ulnar, 14(33%)common peroneal nerves and 10 (24%) posterior tibial nerves.

CSF Findings: Only 2 patients had elevated CSF protein.

Imaging:

• MRI Spine : 1 patient had enhanced lumbar nerve roots and 1 patient had C4-C6 hyper intensities.

• CT Chest: Mediastinal lymphadenopathy seen in one patient of Gastric carcinoma.

Nerve Biopsy: 8 patients had nerve biopsy, out of these 4 patients had vasculitis, 2 patients had axonal neuropathy with regeneration and 2 patients found to be leprosy.

Treatment: 26(61.9%) patients was treated with pulse dose of methyle prednisone followed by maintenance steroids,9 (21.4%) symptomatically 3(11.53%) patients with steroids + Cyclophosphamide, 2(4.7%) patients with plasmapheresis and 2 (4.7%) patients with anti-leprosy drugs.

Out of 42 patients, no cause was identified in 24 (57.1%) patients, 8(19%) patients associated with diabetes mellitus, 2(4.7%) patients with hypothyroidism, 2(4.7%) patients with leprosy, 4(9.5%) patients with vasculitis, and malignancy, rheumatoid arthritis seen one (2.3%) patient each.

Outcome: In present study of 42 patients, 5 (11.9%) patients lost to follow up,4 (9.5%) followed for 3 months, 16 (38%) patients followed for 6 months, 8 (19%) patient followed for 12 months, 9 21.4%) patients followed for 18 months.

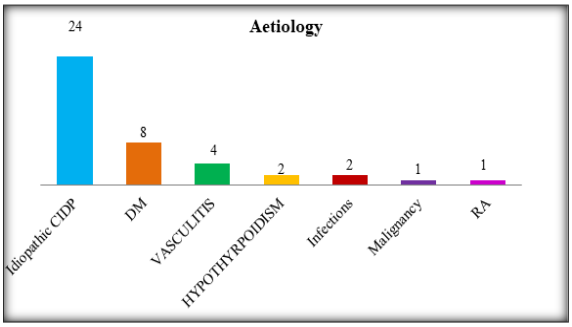

Figure 2: Aetiology of 42 patients

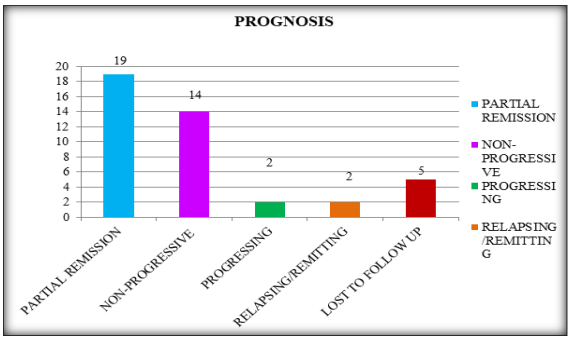

Figure 3: Prognosis of 42 patients

Prognosis of 42 patients: 5 patients lost to follow-up, 19 patients had partial remission, 14 patients had static, no further progression, relapsing/remitting and progressive course in 2 patients each.

TOTAL = 42
LOST TO FOLLOW UP - 5
NON-PROGRESSIVE - 14
PARTIAL REMISSION - 19
RELAPSING /REMITTING - 2
PROGRESSING - 2

Table 4: Outcome of 37 patients

VARIABLE	STATIC /NON PROGRESSIVE	PARTIAL REMISSION	PROGRESSING/ RELAPSING	PROGRESSING	P value
AGE					
≤40 (n=14)	5	8(57.14%)	1	0	
≥40(n=23)	9	11(47.8%)	1	2	o.o3
SEX					
MALE (n=23)	9	11(47.8%)	1	2	
FEMALE(n=14)	5	8(57.14%)	1	0	0.50
HUGHES GRADING AT ADMISSION					
1-2 (n=8)	6	1(12.5%)	0	1	0.043
3-5(n=29)	8	18(62%)	2	1	
MODE OF ONSET					
SUBACUTE(n=3)	1	1(33.35%)	1	0	0.000
CHRONIC(n=34)	13	18(52.9%)	1	2	4
ASYMMETRY(n=4)	2	2(50%)	0	0	0.000
SYMMETRY(n=33)	12	17(51.5%)	2	2	7
WASTING(n=5)	2	3(60%)	0	0	
NO WASTING(n=32)	12	16(50%)	2	2	

					0.0013
ELECTROPHYSIOLOGY					
AXONAL SENSORY AND MOTOR(n=9)	1	6(66.6%)	1	1	
AXONAL SENSORY(n=5)	1	4(80%)	0	0	
MIXED(n=14)	7	5(35.7%)	1	1	
DEMYELINATION(n=9)	5	4(44.4%)	0	0	
TREATMENT					
STEROIDS(n=24)	9	12(50%)	2	1	
STEROIDS+CYCLOPHOSPHAMIDE(n=3)	1	1(33.3%)	0	1	
PLASMAPHERESIS(n=2)	0	2(100%)	0	0	
SYMPTOMATIC(n=8)	4	4(50%)	0	0	
PRIMARY CIDP(n=22)	9	12(54.5%)	1	0	0.6291
SECONDARY CIDP(n=15)	5	7(46.6%)	1	2	

- Age < 40 yrs group (14) showed (8)57.14%remission, where as in age > 40 yrs age group (23) showed (11)47.18% remission. I t was statistically significant (p = 0.03).
- Out of 23 male patients, 11(47.8%) patients had partial remission, 1 relapsing, remititng, 2 progressive course and 9 had non-progressive course. Out of 14 female patients 8(57.14%) had partial remission, 1 patient relapsing/ remitting course, 5 patients with non-progressive course. Sex is statistically significant (p value-0.05)
- Hughes functional grading at admission (1-2) seen in 8 patients, in which 6 patients had static, partial remission and relapsing/remitting course was seen 1(12.5%) patient each. Hughes functional grading at admission 3-5 seen in 29 patients, in which 8 patients had static, partial remission in 18(62%), progressive course in 2 and relapsing/remitting course was seen 1 patient. Statistically significant (p value –0.043).
- Subacute onset seen in 3 patients,in which static course, remission and progressive course seen 1(33.3%) patient each. Chronic course seen in 34 patients, in which 13 had static course, 18(52.9%) had remission ,1 progressive course and relapsing/remitting in 2 patients. statistically significant (p value-0.0004)
- Asymmetry was seen in 4 patients, in which static and remission seen 2(50%) patients each. Symmetry was seen in 33 patients, in which static in 12, remission in 17(51.5%), progressive course and relapsing/remitting course in 2 patients each. Statistically significant (p value-0.0007).
- Wasting was seen in 5 patients, in which static in 2 and remission seen 3(60%) patients. 32 patients had shown no wasting, in which static in 12, remission in 16(50%), progressive course and relapsing/remitting course in 2 patients each. Statistically significant (p value-0.0013).
- Electrophysiological pattern of Mixed Axonal and Demyelinating type was seen in 14 patients, in which remission in 5(35.7%), static course in 7, progressive course and relapsing/remitting course in 1 patient each.
- Electrophysiological pattern of Demyelinating type was seen in 9 patients, in which remission in 4(44.4%), static course in 5 patients.
- Electrophysiological pattern of Axonal sensory and motor type was seen in 9 patients, in which remission in 6(66.6%), static course, progressive course and relapsing/remitting course in 1 patient each.
- Electrophysiological pattern of Axonal sensory type was seen in 5 patients, in which remission in 4(80%), static course in 1 patient.
- 24 patients were treated with steroids, in which remission in 12(50%), static course in 9, progressive course in 2 and relapsing/remitting course in 1 patient.
- 3 patients were treated with steroids+ cyclophosphamide, in which remission in 1(33.3%), static course and relapsing/remitting course in 1 patient each.
- 2 patients were treated with plasmapheresis, both (100%) showed remission.
- 8 patients were treated symptomatically, in which remission, static course 4(50%) patients each.
- In 22 patients with primary CIDP, in which remission in 12(54.5%), static course in 9, progressive course in 1 patient.
- In 15 patients with secondary CIDP, in which remission in 7(46.6%), static course in 5, progressive course in 1 patient and relapsing/remitting course in 2 patients. Statistically not significant (p value – 0.6291).

DISCUSSION

Total of 42 patients with CIDP were studied prospectively who were admitted in department of Neurology of Sri Venkateswara Institute of Medical Sciences.

Table 5: Demographic profile

Variable	Col.S.P Gorthi.et al[2]	I E C Ormerod et al[3]	Nicolette et al[4]	Vaibhav Wadwekar[5] et al	Y-C Chan et al[6]	Kuwabara et al[1]	Present study
No of Pts	35	30	75	65	50	38	42
Mean age	47.6	37.6	56.5	45yrs			46.9
M:F	4:1	1:1.14	1.58:1	2.25:1	1.63:1	1.92:1	1.8:1
Mean duration of illness		5.8 yrs		6 months			22.48 months

The present study showed mean age of presentation was 46.95±14.62 with range of 15-82years majority of patients were male, with male to female ratio was 1.8:1 and mean duration of illness at presentation was 22.48 ±28.82 months with a range of 2 months to 120 months.

Various studies showed that mean age of onset varied from 4th to 6th decade (47.6, 37.6, 56.5, 45) (17,44,47,43) with males preponderance (4:1, 1.58:1 ,2.25:1, 1.63:1, 1.92;1) (17,44,46,47,43) except in slight female preponderance (1:1.14) in Nicolette et al (4).Mean duration of illness presentation was 5. 8 years with a range of 1 to22 yrs (3) and 6 months, with a range of 2 months to 108 months (5).

Present study showed predominant presentation was sensory+ motor symptoms (62%), followed by pure sensory (31%) and pure motor symptoms (7%). Most of the earlier studies also showed predominant presentation was sensory + motor symptoms (71%, 58.6%), followed by pure sensory (26%, 38.6%) and pure motor symptoms (3%,2.6%) (2,4).

In present study, 25(59.5%) patients had both proximal and distal weakness, 3 (7.1%) patients had distal weakness and 1 (2.3%) patient with proximal weakness. 36 (85.7%) were presented with paresthesias in which 25 (59.5%) patients with paresthesias of both feet, 11(26%) patients with parasthesias in both feet and hands and 2(4.7%) patients with diminished pain sensation and 1 (2.3%) patient with burning sensation of feet.

In a study of 65 patients (5), 38 (58.4%) patients presented initially with distal weakness, 29 (44.6%) patients with proximal weakness, 7(10.7%) patients with both proximal + distal weakness, 65(97%) patients with sensory symptoms, 9(13.6%) patients with cranial nerve involvement, autonomic involvement with orthostatic hypotension in 7(10.7%) patients, sweating abnormalities in 2(3%) patients and skin abnormalities in 3(4.6%) patients. In another study of 30 patients (3) motor symptoms in 14(46.6%), sensory+motor symptoms in 4(13.3%), sensory symptoms in 9 (30%), cranial nerves in 1(3%) and CNS involvement in 5(16%) patients.

In present study, diminished pain distal to ankle observed in 27 (64.2%) patient, distal to wrist in 11 (64.2%) patient, and definitive sensory level in 2 (4.7%) patients, diminished light touch distal to ankle observed in 29 (69%) patients, distal to wrist 6 (14%) patients and definitive sensory level in 2 (4.7%) patients, diminished vibration sense distal to knee observed in 24 (57%), distal to ulnar styloid process in 12 (28%) and definitive sensory level in 2 (4.7%) patients , impaired joint, position sense at great toe observed in 30 (71.4%) patients, at middle finger in 9 (21.4%) patients, positive Romberg sign seen in 13(30.9%) patients, total areflexia was observed in 29 (69%) patients, followed by only absent ankle reflex in 12 (28.5%) patients and abnormal finger flexion reflex in 2 (4.7%) patients, wasting was seen 8 (19%) patients.

In a study of 75 patients (4), diminished pain sensation below ankle observed in 64 (85%) patients, below wrist in 12 (16%) patients, diminished touch sensation distal to ankle was seen in 69 (92%) patients, distal to wrist in 12 (16%) patients, diminished vibration sense below knee in 68 (90%) patients, below ulnar styloid process in 28 (37%) patients, impaired joint, position sense at great toe observed in 14 (18.6%) patients, at middle finger in 3 (4%) patients, position Romberg sign observed in 31 (41%) patients, all 75(100%) showed absent ankle reflexes, total areflexia in 16 (21.3%) and wasting was seen in 9(12%).

In present study, Symmetrical proximal and distal sensory motor neuropathy in 18 (42.8%) patients, followed by symmetrical sensory neuropathy 13 (30.9%), ASSM in6 (14.2%) DSSM in 4 (9.5%) patients and mononeuritis multiplex in 1 (2.3%) patient.

In a study of 50 patients (6), clinical pattern of symmetrical sensory motor neuropathy was seen in 30 patients (60%) followed by distal symmetrical sensory motor (DSSM) in 9 (18%) patients, asymmetrical sensory motor (ASSM) in 8 (16%) patients, symmetrical sensory in 2 (4%), and symmetrical motor in 1 patient (2%), in another study of 35 patients(17) DSSM was seen in 26(74%),symmetrical sensory in 4(11%),asymmetrical sensory in 3(9%) and asymmetrical motor and mononeuritis multiplex in 1(3%) patient each.

In present study, 8 patients (19%) with elevated HbA1c > 6, elevated ESR in 3(7.1%), elevated TSH in 2 (4.7%) and dimorphic anaemia, hepatic dysfunction, anti CCL positive, dsDNA positive, position RA factor in 1 (2.3%) patient each.

In a study of 65 patients (5) elevated ESR in 21(31.3%), 16 (24.6%) patients with Diabetes, elevated IgG, anemia in 9 (13.8%) patients, hepatic dysfunction, increased TSH, M band electrophoresis in 7 (10.7%) patients each, renal dysfunction in 4 (6.1%), increased IgM in 3 (4.6%) and increased IgA in 2 (3%) patients. No patients had shown positive Vasculitis profile.

In present study, 8 patients had nerve biopsy in which 4 (50%) patients with vasculitis, 2 (25%) with axonal

neuropathy with regeneration and 2 (25%) patients with leprosy was found. In a study of 46 patients with chronic peripheral neuropathy (7), nerve biopsy showing reduction in myelinated fibre density was most frequent (93.3%) followed by demyelination (82.8%), Inflammation (58.7%) and onion bulb formation in 28.3%.

In present study, 42 patients, 24 (57.1%) was idiopathic CIDP, 8 (19%) with Diabetes, 4 (9.5%) with vasculitis, hypothyroidism, infection was seen 2 (4.7%) patients each and malignancy, Rheumatoid arthritis was seen 1 (2.3%) patient each. In a study of 65 patients (5), 40 (61.5%) patients were idiopathic CIDP, 16 (24.6%) had Diabetes, 4 (6.1%) had POEMS, 2 (3%) monoclonal gammapathy of undetermined significance (MGUS), myeloma, lymphoma and malignancy in 1 (1.5%) patient each.

In present study, 42 patients 26 (61.9%) patients were treated with steroids, 9 patients (42.3%) symptomatically, 3 (11.53%) with cortico steroid + cyclophosphamide, 2 patients (4.7%) with plasma pheresis and 2 (4.7%) with anti-leprosy drugs. In a study of 38 patients (1), (89%) were treated with cortico steroids, Immunoglobulin's infusion in (45%), plasma pheresis in (34%) and 58% received combination therapy.

In present study of 42 patients, 5 (11.9%) patients lost to follow up,4 (9.5%) patients followed for 3 months, 16 (38%) patients followed for 6 months, 8 (19%) patients followed for 12 months, 9 (21/4%) patients followed for 18 months. In present study 19 (45.2%) had partial remission with Hughes function grading 1- 2 (able to walk), 14 (33.3%) has static course with no further progression and progressive course, relapsing/ remitting course was seen in 1 (4.7%) patient each. 5 (11.9%) had lost to follow up. In a study of 38 patients (1) who were followed for 5 years after treatment begun, 10 (26%) had complete remission, 23 (61%) had partial remission with (26%) or without (34%) Immune treatment.

CONCLUSION

This study of 42 CIDP patients found sensorimotor symptoms (62%) and mixed axonal-demyelinating neuropathy (42.8%) as the most common features. While 57.1% were idiopathic, 19% had diabetes, and nerve biopsy revealed vasculitis in 50% of tested cases. Steroids were effective (61.9%), with 45.2% achieving partial remission. However, 11.9% were lost to follow-up, highlighting the need for better long-term monitoring. Early electrophysiology and metabolic screening are crucial for diagnosis and management.

REFERENCES

1. S Kuwabara, S Misawa, M Mori, N Tamura, M Kubota, and T Hattori Long term prognosis of chronic inflammatory demyelinating polyneuropathy: a five year follow up of 38 cases J Neurol Neurosurg Psychiatry. 2006 January; 77(1): 66–70.
2. Chronic Inflammatory Demyelinating Neuropathy (CIDP): CURRENT PERSPECTIVES.Col.SP.Gorthi . MEDICINE UPDATE 2012.VOL 22
3. IEC Ormerod, HM Waddy, AG Kermode, NMF Murray, PK Thomas.Involvement of CNS in CIDP: a clinical, electrophysiological and magnetic resonance imaging study: JNNP 1990;53; 789-793.
4. Nicolette C Notermans, John H J Wokke, Hessel Franssen, Yolanda van der Graaf, Marinus Vermeulen, Leonard H van den Berg, Peter R Bar, Frans G I Jennekens. Chronic idiopathic polyneuropathy presenting in middle or old age: a clinical and electrophysiological study of 75 patients. Journal of Neurology, Neurosurgery, and Psychiatry 1993;56:1066-1071.
5. Vaibhav Wadwekar,Jayantee Kalita,Usha Kanth Misra.Department of Neurology,Sanjay Gandhi PGIMS,Lucknow,India. Does the chronic Inflammatory demyelinating polyradiculoneuropathy due to secondary cause differ from primary. Neurology India, vol.59, No 5. september-october,2011. pp.664-668.
6. Y-C Chan, D C Allen, D Fialho, K R Mills, R A C Hughes. Predicting response to treatment in chronic inflammatory demyelinating polyradiculoneuropathy.
7. Alport, Adina R. MD; Sander, Howard W. MD. Clinical Approach to Peripheral Neuropathy: Anatomic Localization and Diagnostic Testing.CONTINUUM: Lifelong Learning in NeurologyFebruary 2012; Volume 18(1); p 13–38.Address correspondence to Dr Howard W. Sander, New York University School of Medicine, 400 E. 34th St, RR-311, New York, NY 10016, howard.sander@nyumc.org.
8. Data from Cornblath DR, Asbury AK.AIbers JW, et.al. Report from an Ad Hoc Subcommittee of the American Academy of Neurology AIDS. Task Force. Research criteria for the diagnosis of chronic inflammatory demyelinating polyneuropathy (CIDP)."Neurology1991;41:617-8.
9. Richard J Barohn,Mazen M. Dimachkie,Randolph W.Evans. Neurology clinics,peripheral neuropthies;MAY 2013, VOL 31. Number 2. Page 515-17
10. Eichhorst, H. Beitrage zur pathologie der nerven und muscle. Cor Kl Schweiz Aertze. 1890; 20:59–71.
11. Austin, J.H. Recurrent polyneuropathies and their corticosteroids treatment. Brain. 1958; 8:157–192.
12. Dyck, P.J.Lais, A.C.Otha, M. et al, Chronic inflammatory polyradiculoneuropathy. Mayo Clin Proc. 1975; 50:621–637.
13. Dyck, P.J.Arnason, B.G. Chronic inflammatory demyelinatingpolyradiculoneuropathy. in: P.J. DyckP.K. ThomasE.H. Lambert et al, (Eds.) Peripheral neuropathy. 2nd edition. WB Saunders, Philadelphia; 1984:2101–2114.
14. Dyck, P.J.Oviatt, K.F.Lambert, E.H. Intensive evaluation of referred unclassified neuropathies yields improved diagnosis. Ann Neurol. 1981; 10:222–226.
15. Barohn, R.J. Approach to peripheral neuropathy and neuronopathy. Semin Neurol. 1998; 18:7–18.
16. Khan, S.Wolfe, G.Nascimento, O. et al, North America and South America (NA-SA). Neurology. 2006;66.