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Inaugural Descriptive Study of Chronic Inflammatory Demyelinating Polyradiculoneuropathy in West Africa, The Case of Burkina Faso

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Abstract

Introduction: *Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a rare acquired neuropathy, but one that is still poorly documented in sub-Saharan Africa. We are conducting an inaugural descriptive study on CIDP in Burkina Faso.*

Patients and methods: *This was a descriptive cross-sectional study with prospective data collection, conducted from January 2021 to December 2024 in three private health centres in Ouagadougou, Burkina Faso. The study consecutively included patients seen for consultation for CIDP diagnosed according to the EFNS/PNS (2010) criteria, updated by the EAN/PNS criteria of 2021. The sociodemographic, clinical, ENMG and CSF characteristics of the patients were analysed.*

Results: *A total of 37 cases of CPID were collected, with a median age of 53.3 years and a predominance of males (62.2%); hypertension and diabetes, each accounting for 45.9% of cases, were the most common comorbidities. Progressive motor weakness (73%) and paraesthesia (48.6%) were the main reasons for consultation. On initial clinical examination, motor deficit was present in 32 patients (86.5%), mainly in the form of paresis (68.7%) of the proximal-distal (64.7%) lower limbs (65.6%); Sensory disorders were dominated by paresthesia (91.9%) and hypoesthesia (83.8%), predominantly distal (51.3%) in all four limbs (37.8%) or the lower limbs (45.9%); ROTs were reduced in 51.4% of cases. Proprioceptive ataxia, cranial nerve involvement and dysautonomia were found in 81.1%, 13.5% and 16.2%* of cases, respectively. ENMG *and LP revealed segmental or multifocal demyelination (100%) and albumin-cytological dissociation (83.3%), respectively.*

Conclusion: *Improved accessibility and availability of neurologists trained in clinical neurophysiology, diagnostic tools such as ENMG, and immunological biological tests would help to improve and refine the diagnosis of CIDP.*

Keywords: *Motor Deficit; Paraesthesia; Hypoaesthesia; Lower Limbs; ENMG; Demyelination; Albumin-Cytological Dissociation.*

1. INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuritis (CIDP) is an autoimmune inflammatory peripheral neuropathy, usually acquired, characterised by progressive or fluctuating proximal and distal weakness over ≥8 weeks, signs of segmental or multifocal demyelination on electroneuromyography (ENMG) and a response to treatment [**1, 2,3].** The epidemiology of CDIMP in adults indicates an estimated prevalence of between 1 and 7 cases per 100,000 inhabitants, with a probable underestimation, while in France it varies between 2 and 8 per 100,000 [**1, 2,3].**

The diagnosis is based on a combination of clinical, biological, electrophysiological and sometimes radiological findings [**1, 2,3],** and early treatment with immunomodulators or immunoglobulins improves the prognosis [**4, 5].** Data for sub-Saharan Africa are scarce and

probably underestimated, likely due to the low prevalence of CIDP, but mainly because of its under-recognition due to limited access to specialised diagnosis and ENMG [**6].** Indeed, in resource-limited countries such as Burkina Faso, sources converge to show that Burkina Faso has a very low density of neurologists (≈0.04/100,000 in international comparisons) and a glaring shortage of EEG/EMG equipment and other specialised examinations, particularly in most public health facilities [**7, 8, 9].** Recent reviews on neuropathies in SSA show considerable methodological heterogeneity and a lack of studies focusing specifically on CPID; many studies report hospital case series rather than population surveys [**10],** while favourable response to treatment often depends on the early onset of treatment and the clinical phenotype [**4**].

In SSA in general and in Burkina Faso in particular, the lack of high-quality ENMG, the shortage of specialist neurologists and delays in diagnosis result in a risk of under-detection of CIDP **[10]**.

Conducting an inaugural descriptive study in private health centres in Ouagadougou, Burkina Faso, the only ones with ENMG equipment, would therefore provide new data that could be used to improve clinical recognition of the disease, guide diagnostic strategies, and advocate for better access to appropriate treatment options in our context.

2. PATIENTS AND METHODS

This was a cross-sectional, descriptive, prospective data collection study conducted over four (4) years (01/01/2021 to 31/12/2024) in three (3) private health centres in the city of Ouagadougou, Burkina Faso (Polyclinique Notre Dame de la Paix, Clinique du Bois and Centre Médical Agir), each of which has a neurology unit run by a senior neurologist specialising in clinical neurophysiology and where ENMG examinations are routinely performed. The population of our study consisted of patients followed up in these private health centres. The study included patients treated at these three health centres for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) diagnosed according to the EFNS/PNS criteria (2010) [**11],** as updated by the EAN/PNS criteria of 2021 [**1].** We excluded patients who did not give their consent and those who had not undergone a usable ENMG. Similarly, patients who tested positive for HIV or hepatitis B were not included. We systematically recruited all patients relevant to the study who met the selection criteria.

Before starting the study, we listed and contacted public hospitals, private clinics and medical centres throughout the city of Ouagadougou. Among these healthcare facilities, only three health centres (Polyclinique Notre Dame de la Paix, Clinique du Bois and Centre Médical Agir) had a neurology unit that routinely performed ENMG interpreted by a neurologist specialising in clinical neurophysiology. In collaboration with the neurologists at these healthcare facilities, we compiled an exhaustive list of patients who met the electroclinical criteria for CIDP.

The data was collected by us using a questionnaire designed for this purpose. The data was recorded on a data collection form that included socio-demographic, clinical and paraclinical data (ENMG, lumbar puncture with cerebrospinal fluid analysis). We collected the following data: socio-demographic data : age, gender, level of education, socio-professional occupation, marital status, place of residence; clinical data: medical history, vascular risk factors, onset, duration, time to consultation and diagnosis, clinical examination (motor disorders, sensory-vegetative disorders, cranial nerve damage, osteotendinous reflexes, severity of functional disability (Hughes score) at diagnosis; paraclinical data: ENMG, lumbar puncture. Data analysis was performed using Epi-Info software, French version 7.2.2.2. Graphs and tables were created using Microsoft Excel 2019 software; the data obtained were analysed and commented on.

The descriptive analysis consisted of calculating percentages for qualitative variables and measures of central tendency (mean, median) and dispersion (standard deviation, minimum, maximum) for quantitative variables.

The study was conducted in accordance with medical ethics and professional standards. Patient confidentiality and anonymity were respected, following administrative authorisation from the directors of the health centres concerned.

3. OPERATIONAL DEFINITIONS

Diagnostic criteria for PIDC: We used the EFNS/PNS (2010) diagnostic criteria for PIDC **[11],** updated by the EAN/PNS criteria for 2021 **[1]**, which we adapted to our working context (Table I).

Table 1. *Clinical and electrophysiological criteria for CPMP used in our study.*

Category	Elements
Clinical	- Symmetrical, proximal and distal motor and/or sensory deficit, progressing for ≥ 8 weeks - Diffuse areflexia or hyporeflexia - Recognised variants: pure motor form, pure sensory form, acquired distal form (DADS), focal/multifocal
Electrophysiological (at least 1 criterion in ≥ 2 nerves, or 2 criteria in 1 nerve)	- Prolonged distal latency - Slowed motor conduction velocity - Conduction block - Temporal dispersion - Prolonged or absent F latencies
Supporting criteria	- **CSF**: hyperproteinorachia (> 0.45 g/L) without pleocytosis - **Clinical response to treatment** (corticosteroids)
Diagnostic categories	- **Definite**: confirmed clinical + electrophysiological criteria

Table 2. *Normal values and abnormalities/diagnostic thresholds for CIDP according to EFNS/PNS — EAN/PNS criteria.*

ENMG parameter	Usual normal values (general examples)	Abnormalities/diagnostic thresholds for CIDP (EFNS/PNS — EAN/PNS)
Distal motor latency (ms)	depends on the nerve; examples: median (APB) ≲ 3.7 ms, ulnar ≲ 3.2 ms, peroneal ≲ 5 ms, tibial ≲ 5.5 ms	Prolongation ≥ 50% above the upper limit of normal in ≥ 2 nerves (major criterion) EFNS) (median: >5.5 ms; ulnar: >4.8 ms; peroneal: >7.5 ms; tibial: >8 ms)
Motor conduction velocity (m/s)	typically ≥ ~50 m/s for upper limbs, ≥ ~40 m/s for lower limbs (depending on the nerve)	Reduction ≥ 30% below the lower limit of normal in ≥ 2 nerves (major criterion) (<35 m/s in upper limbs; < 28 m/s in lower limbs)
F latency (ms)	normal values: upper limbs ≤ 32 ms (median, ulnar); lower limbs ≤ 56 ms (peroneal, tibial)	Prolongation ≥ 30% above the upper limit of normal in ≥ 2 nerves (>42 ms in upper limbs; >72 ms in lower limbs) Absence of F waves in 2 nerves with distal CMAP ≥ 20% of the lower limit of normal
Distal compound motor action potential (CMAP) amplitude (mV)	depends on the nerve & muscle recorded	Conduction block: marked proximo-distal decrease > 50% between proximal and distal stimulation
Motor time dispersion (MAP duration, ms)	distal CMAP duration≈ 6–9 ms (depending on nerve)	Increase in CMAP duration >30% between proximal and distal stimulation = temporal dispersion (criterion for abnormality).

Table 3. *Values of Parameters Measured By Enmg in Our Study.*

	Motor conduction velocities (MCV) and proximal conduction velocities (PCV) of F waves (in m/s)	**Sensory conduction velocities (SCV) (in m/s)**
Upper limbs		
Medullary-elbow	> 52 (median 62)	> 55 (median 66)
Elbow-wrist	> 48 (median 57)	> 50 (median 60)
Wrist-palm	> 36 (median 46)	> 45 (median 55)
Lower limbs		
Knee marrow	>45 (median 55)	> 50 (median 57)
Knee-ankle	> 42 (median 50)	> 45 (median 52)
Ankle-foot		> 40 (median 48)

	Distal motor response		**Sensory potential Amplitude**
	Distal latency	**Amplitude**	
Upper limbs			
Median nerve	< 3.7 ms	> 6 mV	> 15 µV
Ulnar nerve	< 3.2 ms	> 6 mV	> 8 µV
Other nerves	< 3 to 5 ms	> 3 to 6 mV	> 10 to 15 µV
Lower limbs			
Peroneal nerve	< 5 ms	> 3 mV	> 5 µV
Tibial nerve	< 5.5 ms	> 6 mV	> 5 to 10 µV
Other nerves	< 3 to 5 ms	> 3 to 6 mV	> 5 to 10 µV

The objective assessment of motor deficit was performed by rating motor strength according to the Medical Research Council (MRC) scale for muscle strength [Medical Research Council. *Aids to the Examination of the Peripheral Nervous System.* Memorandum no. 45. London: Her Majesty's Stationery Office; 1976]: 0/5: No visible or palpable contraction; 1/5: Visible or palpable contraction without movement; 2/5: Movement possible, but only in the horizontal plane, without gravity; 3/5: Movement possible against gravity, but without resistance; 4/5: Movement possible against gravity and partial resistance; 5/5: Normal muscle strength (complete movement against gravity and maximum resistance). Plegia was defined as an MRC score between 0 and 2/5, paresis as an MRC score between 3 and 4/5, and normal motor strength as an MRC score of 5/5.

4. HUGHES SCORE

The Hughes disability score is a rating system developed to assess the functional status of patients. It was originally described by Hughes et al. (1978) and since then, various iterations have appeared in the literature. It ranges from 0 to 6 and is detailed as follows :

0	:	Asymptomatic
1	:	Minor symptom, patient able to run
2	:	Unable to run but able to walk more than 10 metres without assistance
3	:	Able to walk but for less than 10 metres and/or with assistance
4	:	Unable to walk, confined to bed or wheelchair
5	:	Requires ventilatory assistance
6	:	Death

For this study, we classified these scores as follows:

1. Mild disability: Scores 1 and 2
2. Moderate disability: Scores 3 and 4
3. Severe disability: Score

5. RESULTS

5.1. Socio-Demographic Data

A total of 37 patients monitored or diagnosed for PIDC were consecutively enrolled in private health centres in Ouagadougou. The median age of patients was 53.3 years IQ ± 14.5 years (range 24 to 79 years).

Patients aged > 50 years accounted for 62.1% and in 62.2% of cases, they were male. Patients resided in urban areas in 73% of cases and were married in 75.7% of cases. Traders accounted for 18.9% of patients in our series and 35.1% had a university education. Table IV shows the distribution of patients according to socio-demographic characteristics.

Table 4. *Distribution of patients with PIDC according to socio-demographic characteristics (N=37).*

Variable	Number (N=37)	Percentage (%)
Age		
<50 years old	14	37.8
[50-70 years old]	18	48.6
>70 years old	5	13.5
Gender		
Male	23	62.2
Female	14	37.8
Area of residence		
Urban	27	73
Rural	10	27
Marital status		
In a relationship	29	78.4
Single	5	13.5
Widowed	3	8.1
Socio-professional occupation		
Shopkeeper	7	18.9
Retired civil servant	6	16.2
Housewife	6	16.2
Civil servant	6	16.2
Private sector employee	4	10.8

Farmer	2	5.4
Pupil/Student	2	5.4
Unemployed	2	5.4
Other (Imam, Artist)	2	5.4
Level of education		
None	12	32.4
Primary	4	10.8
Secondary	8	21.6
University	13	35.1

Hypertension and type 2 diabetes, with 17 cases each (45.9%), were the most common comorbidities, affecting 45.9 %.

Table V shows the distribution of patients with CIDP according to comorbidities.

Table 5. *Distribution of patients with CIDP according to comorbidities (N=37)*

History	Number (N=37)	Percentage (%)
HTA	17	45.9
Type 2 diabetes	17	45.9
Rheumatological conditions (osteoarthritis, rheumatoid arthritis, herniated disc, gout)	8	21.6
Chronic viral hepatitis B	2	5.4
Gastro-duodenal ulcer	2	5.4
Asthma	2	5.4
Pulmonary embolism	1	2.7
Stroke	2	5.4
Chronic subdural haematoma	1	2.7
History of COVID-19	1	2.7
Hallucinatory psychosis	1	2.7

The onset was gradual in all our patients (100%). The median time to diagnosis was 9.2 months (IQR 12.6 months) (range 6 months to 36 months); in 40.6% of patients, the time to diagnosis was 16 months. The main reasons for consultation were progressive motor weakness in the limbs and sensory disturbances (paresthesia, pain, hypoesthesia), in 73% and 48.3% of patients, respectively.

On initial clinical examination, motor deficit was present in 32 patients (86.5%), with a proximal-distal distribution in 24 patients (64.7%), exclusively localised to the lower limbs in 21 patients (56.8%) and to all four limbs in 11 patients (34.4%); the motor deficit was plegic in 10 patients (27.0%). At the initial clinical examination, subjective sensory disturbances (paresthesia/dysesthesia/neuropathic pain) were reported in 34 patients (91.9%); Hypoesthesia was found in 83.8%, localised in all four limbs (37.8%) or in the lower limbs (45.9%), with distal (51.3%) or proximal-distal (37.8%) distribution. Deep tendon reflexes (DTRs) were reduced in 51.4% of cases. Proprioceptive ataxia, cranial nerve damage and dysautonomia were found in 35 patients (81.1%), 5 patients (13.5%) and 6 patients (16.2%) respectively. Table VI shows the distribution of patients with CIDP according to the time and reasons for consultation and the clinical signs found during the initial clinical examination.

Table 6. *Distribution of patients with CIDP according to the timing and reasons for consultation and clinical signs at the initial clinical examination.*

Signs	Number (N=37)	Percentage
Time and reasons for consultation	Number (N=37)	Percentage (%)
Diagnosis time		
[6-12 months]	12	32.4
]12-18 months]	10	27
]18–36 months]	15	40.6
Reasons for consultation		
Progressive motor weakness	27	73
Sensory disturbances (paresthesia/pain/hypoesthesia)	18	48.6

Gait disturbances/postural instability	12	32.4
Neurological signs on admission		
Motor deficit	32	86.5
Severity		
Plegia	10	31.3
Paresis	22	68.7
Topography		
Both lower limbs	21	65.6
All four limbs	11	34.4
Respiratory muscles (associated with the above)	3	9.4
Distribution		
Proximal-distal	24	75
Distal	8	25
Subjective sensory disturbances	37	100
Type		
Paresthesia/dysesthesia	34	91.9
Pain	28	75.7
Numbness	21	56.7
Topography		
Lower limbs	19	51.3
Lower and upper limbs	18	48.6
Trunk (associated)	6	16.2
Objective sensory disorders	32	86.5
Type		
Hypoaesthesia	31	83.8
Anaesthesia	1	2.7
Topography		
Lower limbs	17	45.9
Upper and lower limbs	14	37.8
Diffuse throughout the body	1	2.7
Distribution		
Distal	19	51.3
Proximal-distal	13	35.1
Cranial nerve damage (isolated or associated)	5	13.5
Unilateral or bilateral facial paresis (VII)	3	8.1
Unilateral or bilateral oculomotor paresis (III/VI)	1	2.7
Swallowing disorders (IX/X)	1	2.7
Dysautonomia	6	16.2
Sudomotor disorders (heat intolerance, hyperhidrosis or anhidrosis)	2	5.4
Orthostatic hypotension	1	2.7
Erectile dysfunction	1	2.7
Gastrointestinal disorders (fluctuation between diarrhoea and chronic constipation)	2	5.4
Proprioceptive ataxia	3	81.1
Decreased ROT	19	51.4

At the initial disability assessment using the Hughes score, 43.2% of patients presented with moderate disability. Figure 1 shows the distribution of patients according to the Hughes score at the initial clinical examination.

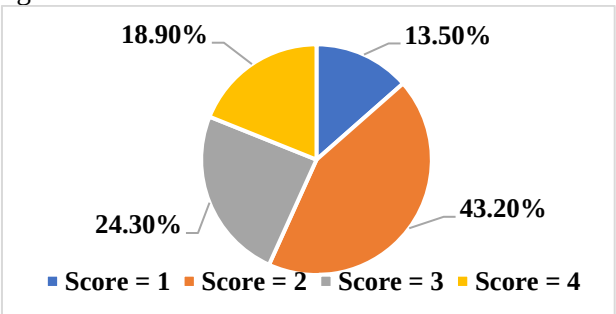

Figure 1. *Distribution of patients according to Hughes score*

ENMG was performed in all patients (100%). Segmental or multifocal demyelination parameters were found in all our patients (100%) : Motor conduction velocity (MCV) and Sensory conduction velocity (SCV) were decreased in 97.3% and 43.2% of patients, respectively; distal motor latency and F-wave latency were increased in 94.6% and 83.8% of patients, respectively; motor conduction block was found in 37.8% of patients. CIDP was of the sensorimotor type in 94.6% of patients. A lumbar puncture (LP) with cerebrospinal fluid (CSF) examination was performed in 30 patients (81.1%), revealing hyperproteinorachia in 60% of cases and normal cytorachia in all our patients (100%), defining an albumin-cytological dissociation in 60%. The median proteinorachia was 0.9 g/L (IQ 0.4) (extremes 0.35-1.6 g/L). Cytorachia (< 10 g/L) and germ detection (absence of germs in cyto-bacteriological, parasitological and mycological examination and after culture) returned to normal in the 30 patients (100%) who underwent LP. The median cytorrhea was 5 cells/mm³ (IQR 2) (range 1 to 7 cells/mm³). Table VII shows the distribution of patients with PIDC according to the results of ENMG and LP with CSF analysis.

Table 7. *Distribution of patients with PIDC according to ENMG results (N=37) and lumbar puncture results (N=30)*

Characteristic	Number (N=37)	Percentage
Motor conduction velocity (MCV)		
Decreased	36	97.3
Normal	1	2.7
Sensory conduction velocity (SCV)		
Decreased	16	43.2
Normal	21	56.7
Increased latencies		
F wave latency	31	83.8
Distal motor latency	35	94.6
Motor time dispersion	18	48.6
Motor conduction block	14	37.8
Electroclinical summary		
Classic sensorimotor form	35	94.6
Pure motor form	2	5.4
Pure sensory form	0	-
Other forms	0	-
Associated axonal damage	2	5.4
Lumbar puncture results	Number (N=30)	Percentage (%)
Biochemistry		
Hyperproteinorachia	18	60
Normal proteinuria	12	40
Cytorachia		
Normal	30	**100**
Microbiology (bacteriology, parasitology, mycology)		
Normal	30	**100**

6. Discussion

In sub-Saharan Africa in general, and in Burkina Faso in particular, the diagnosis, management and study of CIDP and other peripheral nervous system disorders pose a real challenge due to the limited availability and accessibility of neurologists and diagnostic and therapeutic resources. In Burkina Faso and other SSA countries, recent local and/or hospital studies note that paraclinical neurological investigations such as ENMG are gradually being rolled out, particularly in urban areas and in the private health sector, but remain virtually non-existent in public hospitals and rural areas in most SSA countries [**13, 14].** Furthermore, the number of neurologists and technicians trained in clinical neurophysiology remains low and local training s limited [**15].** Similarly, inadequate infrastructure and maintenance limit the continuous operation of MRI and ENMG machines [**7].**

The consequences are diagnostic inaccuracy and delays [**13, 16]**; unequal access favouring urban and wealthy patients in private healthcare facilities, to the detriment of rural and/or poor patients; this bias affects the quality of local clinical series and research [**7]**; reliance on basic clinical examinations, PL and therapeutic tests [**13]**. These obstacles guided our decision to limit the

present study to private healthcare facilities in urban areas in Ouagadougou. Our study was discussed with other data from the literature.

The socio-demographic data found in our series are consistent with those usually reported in the literature. Indeed, the average age of 53 years (range 44-94 years) among our patients falls within the age range of middle-aged adults and the elderly. Most series on PIDC report a mean age of patients at diagnosis between 40 and 60 years (median often around 50–58 years) and emphasise the rarity of paediatric forms [**17, 18]**. As with most literature series, a male predominance (62%) was found in our study. The exact reason for this is not yet fully understood. Studies suggest that it may be related to hormonal differences or genetic factors that affect the immune system [**6, 19].**

In our series, hypertension and type 2 diabetes were the most common comorbidities, each accounting for nearly 50% of cases. On the one hand, these two comorbidities constitute public health problems in developing countries due to the epidemiological transition marked by an increase in cardiometabolic diseases **[20]**. On the other hand, the association between CIDP and diabetes mellitus is particularly common [**20].**

Clinically, available African studies confirm that CIDP presents with classic phenotypes (proximal and distal weakness, sensory signs, decreased reflexes) but highlight certain regional specificities: a significant proportion of patients co-infected with HIV in South African series, earlier presentations in HIV-positive subjects and frequent CSF pleocytosis in HIV-positive patients, suggesting that the endemic infectious context modifies the clinical and investigative profile of CIDP in SSA [**21].** This particularity was not found in our series, as we excluded all HIV-positive patients.

In our study, 57% of patients had mild disability without any real loss of independence (Hughes score between 1 and 2), while 43% had moderate to severe disability (Hughes score between 3 and 4) with loss of independence, particularly in terms of walking and grasping. This finding is corroborated by several recent large multicentre cohorts, which confirm that at the time of diagnosis, the majority of patients already have a measurable disability at inclusion. The initial clinical severity of CIDP is most often characterised by moderate motor weakness affecting walking and grasping; frequent sensory disturbances, sometimes associated with ataxia; moderate overall functional disability, with an impact on mobility in daily life. Severe impairment from the outset (inability to walk, significant dependence) is rarer, found in a minority (< 15% of patients) [**22, 23, 24, 25]**.

Cranial nerve involvement is estimated to occur in 10% to 20% of cases overall **[26]**, which includes the 10% rate found in our series. Regarding the specific types of cranial nerves affected, the most common are facial and bulbar paralysis (9% each) and oculomotor involvement (III, IV, VI) (5%) [**27].** According to Rare Disease Advisor, cranial nerve involvement occurs in approximately 15% of cases, with facial and oculomotor involvement being the most common (ophthalmoplegia: 3–8%) [**28]**. In our series, facial involvement was the most common, at 8%.

According to recent data in the literature, dysautonomia in CIDP is more common than previously estimated, especially in specialised settings with extensive testing. An epidemiological study based on the Piedmont registry in Italy found clinical dysautonomia in 12.9% of cases, with a higher rate (64%) in patients with severe forms of the disease [**29].** According to a recent review covering 12 studies (346 patients), the prevalence of dysautonomia, whether clinical or subclinical, varies between 25% and 89%, depending on the tests used and the populations studied [**30].** In our series, the rate of clinical dysautonomia observed was 16%, dominated by sudomotor and gastrointestinal disorders (5% each), whereas in the literature, the most frequently reported clinical dysautonomic disorders are dominated by orthostatic hypotension (23%), which reflects sympathetic adrenergic impairment [**2**]; postural orthostatic tachycardia syndrome (13%) in patients in the same study [**2]** linked to cardiovascular/adrenergic imbalance; sweating disorders (anhidrosis, hyperhidrosis, thermal dysregulation) (34% to 63%) [**2, 31];** gastrointestinal disorders (constipation, diarrhoea, gastroparesis) (20–30%) [**2]** and genitourinary disorders (pollakiuria, incontinence, erectile dysfunction) [**2].**

The literature describes a classic form ("typical CIDP") and variants. The classic form accounts for 50 to 60% of cases and is characterised by symmetrical, proximal and distal polyneuropathy, developing over > 8 weeks, with predominant motor weakness, frequent sensory impairment and osteotendinous areflexia. ENMG finds the EFNS/PNS criteria for demyelination

[**1].** The recognised electroclinical variants of CIDP are: 1) Lewis-Sumner syndrome (10-15%), presenting as asymmetric multifocal sensory-motor neuropathy, with focal conduction blocks in several nerve trunks on ENMG **[1, 32]**; 2) acquired symmetrical distal demyelinating neuropathy (7-10%) associated with distal slowing [**1, 33]**; 3) pure motor CIDP (5-10%) characterised by motor weakness without marked sensory disturbances, associated with ENMG signs of motor demyelination, with preserved sensory conduction [**1]**; 4) pure sensory CIDP (4-7%) characterised by isolated sensory deficit, ataxia, osteotendinous areflexia, without motor deficit, associated with demyelinating abnormalities in the sensory nerves, while motor potentials are preserved [**1, 34].** In our series, classic CIDP accounted for up to 95% of cases, while the only variant identified was pure motor CIDP (5%); the other variants were probably underdiagnosed due to the difficulties in recognising them diagnostically in our context.

Lumbar puncture (LP) with CSF analysis provides supporting evidence for the diagnosis of CIDP. CSF analysis reveals elevated protein levels ranging from 0.6 to 1.5 g/L, rarely exceeding 2 g/L, contrasting with normal cell counts (≤ 10 cells/mm3), resulting in the classic albumin-cytological dissociation found in 42 to 77% of patients, depending on the series. In our series, the albumin-cytological dissociation was found in 60% of cases, which is consistent with the data in the literature. This essential characteristic of CIDP reflects radicular inflammation with increased permeability of the blood-nerve barrier [**1, 35, 36].** Conversely, a normal LP does not rule out CIDP, particularly in atypical forms; hyperproteinorrachia in the presence of peripheral neuropathy is not specific to CIDP and may be found in Guillain-Barré syndrome, advanced diabetic neuropathy or other polyradiculopathies. Due to its moderate diagnostic yield, LP is a useful supporting argument for the diagnosis of CIDP but is insufficient on its own. It is particularly indicated when the diagnosis is uncertain or in atypical forms [**1]**.

In our series, type 2 diabetes was found in almost 50% of cases, confirming the observations made in several series and reviews suggesting that CIDP occurs more frequently in diabetic patients than in the general population, although the exact strength of the association varies depending on the studies and methods used. Some population studies and reviews report a higher proportion of diabetics among CIDP patients, but other studies emphasise selection bias and the impossibility of concluding a direct causal link [**37, 38].** Thus, the high frequency of the CIDP-diabetes association found in our series, confirmed in the literature, constitutes a persistent diagnostic challenge. This challenge is to make a differential diagnosis between severe diabetic neuropathy and authentic CIDP, as their management and prognoses differ. Current recommendations propose the combined use of strict ENMG criteria, CSF, nerve ultrasound and documented therapeutic trials, which improve diagnostic accuracy and guide therapy [**39, 40].** In SSA in general, and in Burkina Faso in particular, diagnostic obstacles contribute to the underestimation of the disease: the diagnosis of CIDP is demanding, relying on a combination of clinical criteria and ENMG evidence of segmental or multifocal demyelination, sometimes supplemented by CSF analysis and imaging, particularly nerve ultrasound. However, access to ENMG, the availability of neurologists trained in clinical neurophysiology and referral times are often insufficient, leading to diagnostic delays, inappropriate empirical management and mislabelling (compressive, deficiency or infectious neuropathies) [**10].**

7. Conclusion

This preliminary study conducted in Ouagadougou, Burkina Faso, shows that CIDP is a relatively rare neurological condition, characterised by an age of onset around 50, a male predominance and a frequent association with diabetes. The predominant profile was chronic demyelinating sensory-motor form, predominantly affecting the lower limbs in a proximal-distal distribution, with albumin-cytological dissociation of the CSF, causing mild to moderate disability on admission. Better accessibility and effective availability of neurologists trained in clinical neurophysiology, diagnostic tools such as ENMG, and immunological biological tests would help to improve and refine the diagnosis of CIDP. In Burkina Faso and SSA. In order to better characterise this disease in SSA, robust prevalence and incidence studies conducted on population bases are needed to estimate the true burden of CIDP.

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