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Electroclinical aspects of diabetic neuropathy in the city of Ouagadougou

Alassane Zoungrana*[1], Anselme Alfred Dabilgou[2,3], Marie Raphael Kaboré[4], Athanase Millogo[1,2]

[1]*Department of Neurology, Souro Sanou University Hospital Center, Bobo-Dioulasso, Burkina Faso*
[2]*Health Sciences Training and Research Unit, Joseph KI-ZERBO University, Ouagadougou, Burkina Faso*
[3]*Department of Neurology, Yalgado Ouédraogo University Hospital, Ouagadougou, Burkina Faso*
[4]*Department of Neurology, Tengandogo University Hospital Center, Ouagadougou, Burkina Faso*

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***Corresponding Author**: Alassane Zoungrana, alassanezoungrana@yahoo.fr

ABSTRACT

Aims: Diabetic neuropathy (DN) is one of the most common microvascular complications of diabetes mellitus, often leading to severe sensorimotor disability. This study aimed to identify factors associated with the severity of electroneuromyographic (ENMG) involvement among diabetic patients followed in Ouagadougou, Burkina Faso.

Methods: We conducted a cross-sectional analytical study including 64 patients with clinically diagnosed DN who underwent electrodiagnostic (EDX) evaluation. Sociodemographic, clinical, and biological data were collected. Neuropathy severity was classified as mild/moderate or severe/very severe. Bivariate and multivariate logistic regression analyses were performed, with a significance level set at $p<0.05$.

Results: The mean age was 64.6±8 years, with a slight female predominance (53.1%). In bivariate analysis, severe EDX involvement was significantly associated with a diabetes duration ≥10 years [OR=6.55; 95% CI (1.95-21.9); p=0.002] and age ≥65 years [OR=3.33; 95% CI (1.15-9.61); p=0.024]. In multivariate analysis, only diabetes duration ≥10 years remained an independent predictor of severity [adjusted OR=5.32; 95% CI (1.52-18.5); p=0.009].

Conclusion: The severity of DN was mainly linked to long-standing diabetes, emphasizing the need for early screening and strict glycemic control.

Keywords: Diabetic neuropathies, nerve conduction studies, risk factors, Burkina Faso

INTRODUCTION

Diabetic neuropathy (DN) is the most common chronic complication of diabetes mellitus (DM) and a leading cause of peripheral neuropathy worldwide.[1] It is defined as the presence of symptoms and/or signs of peripheral nerve dysfunction in individuals with diabetes after exclusion of other potential causes.[2]

DN affects approximately 1% to 7% of the general population, with higher prevalence in individuals over 50 years of age and a slight female predominance.[3] It complicates both type 1 diabetes (T1D) and type 2 diabetes (T2D).[3,4] Reported prevalence varies widely, ranging from 7% to 80% depending on study design and diagnostic criteria.[5] DN encompasses several clinical syndromes, including distal symmetric polyneuropathy (the most common form, accounting for approximately 75% of cases), autonomic neuropathy, cranial neuropathies, mononeuropathies, radiculoplexopathies, diabetic neuropathic cachexia, and treatment-induced neuropathy.[6,7]

The diagnosis of DN is based on both clinical and electrophysiological evidence.[2] Electroneuromyography (ENMG) is a key diagnostic tool for functional assessment, allowing confirmation of diagnosis and characterization of the underlying pathophysiological mechanism, including axonal, demyelinating, or mixed neuropathies.[3] Although not always required for the diagnosis of typical clinical forms, ENMG is essential for detecting subclinical or mild neuropathy.[8] In Burkina Faso, ENMG remains a relatively recent diagnostic modality, with limited availability and a small number of trained specialists.

The prevalence of DN increases with disease duration, reaching approximately 34% after 25 years of diabetes, as reported in the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) study.[9] Similar findings have been reported in the EURODIAB prospective study.[10] In contrast, more than half of individuals with T2D develop signs and symptoms of

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diabetic peripheral neuropathy during their lifetime.[11,12] The prevalence of painful DN is also high, reaching 20% to 30% even in newly diagnosed or early-stage T2D, as demonstrated in large contemporary cohorts such as ADDITION, GRADE, and SEARCH.[13,14]

Data on DN in sub-Saharan Africa remain limited. In Burkina Faso, published studies addressing its prevalence, risk factors, and electrophysiological characteristics are scarce. A local study conducted in 2018 reported a prevalence of DN of 80.7%.[15] In that cohort, distal polyneuropathy accounted for 81.8% of cases and autonomic neuropathy for 72.7%, with poor glycemic control observed in 38.8% of patients. However, the electrophysiological characterization of neuropathy was not addressed.[15]

We conducted this study to characterize the electrodiagnostic (EDX) profile of DN in our setting and to identify its associated clinical and metabolic determinants.

METHODS

This study was conducted with the approval of the Institutional Ethics Committee of Saint Camille Hospital in Ouagadougou (Date: 09.11.2023, Decision No: 2023-11-09). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Written informed consent was obtained from all individual participants prior to their inclusion in the study.

This was a descriptive, cross-sectional, prospective study conducted over a nine-month period, from January 1, 2024, to September 30, 2024.

The study was conducted in two neurophysiology centers in Burkina Faso: the electro-neurophysiology unit of the Neurology Department at Saint Camille Hospital in Ouagadougou (HOSCO) and the neurophysiology unit at Agir Clinic.

The study population included all diabetic patients who underwent ENMG at both sites during the study period. Patients with a history of peripheral neuropathy due to causes other than diabetes (e.g., vitamin deficiency, alcoholism, or chemotherapy) were excluded. Patients who did not provide informed consent were also excluded.

Data collection was performed after approval from the Ethics and Research Committees of Saint Camille Hospital in Ouagadougou and Agir Clinic.

All included patients underwent a complete neurological examination and ENMG. Data were collected using a standardized case report form, including epidemiological, clinical (history and neurological examination findings), and electrophysiological data.

Clinical assessment tools included:

- A 128-Hz tuning fork, a brush, and a monofilament for assessment of sensory modalities (proprioceptive, tactile, nociceptive, and vibratory) and foot ulcer risk.
- A reflex hammer for assessment of deep tendon reflexes
- A blood pressure monitor and stethoscope for evaluation of cardiovascular autonomic neuropathy (CAN)
- Scales and a measuring tape for body-mass index (BMI) calculation and nutritional assessment
- The DN4 questionnaire for screening painful diabetic neuropathy (PDN)
- The Visual Analog Scale (VAS) for pain intensity assessment

ENMG was performed using Micromed and Natus devices.

ENMG procedures: Nerve conduction studies were performed using standard laboratory techniques with surface stimulation and recording.

Motor conduction studies focused bilaterally on four nerves: the deep peroneal, posterior tibial, median, and ulnar nerves.

Sensory conduction studies included the median and ulnar nerves in the upper limbs and the sural nerve in the lower limbs.

Needle electromyography was performed in selected cases.

Laboratory standards for ENMG interpretation were based on those described by Emmanuel Fournier.[16,17]

Abnormal nerve conduction parameters were defined according to standard neurophysiological guidelines (Fournier, 2018).[16] Lower-limb abnormalities included motor conduction velocity (MCV)<40 m/s, distal motor latency (DML)>4.5 ms, and sural sensory nerve action potential (SNAP) amplitude <5 µV.

The HOSCO reference values are presented in Table 1.

Table 1. HOSCO ENMG standards

Motor nerves	**Distal latency**	**Amplitude**	**Speed**	**F wavelatency**
Median	<3.7 ms	>6 mV	>48 m/s	≤32 ms
Ulnar	<3.2 ms	>6 mV	>50 m/s	≤32 ms
Radial	<3.4 ms	>6 mV	>50m/s	
Axillary (circumflex)	<4.5 ms	>4 to 6 mV		
Musculocutaneous UL			>50 m/s	
Fibular (CFN)	<5 ms	>3 mV	>42 m/s	≤56 ms
Tibial (TN)	<5.5 ms	>5 mV	>42 m/s	≤56 ms
Sensory nerves	**Amplitude**		**Speed**	
Median	15 Uv		>45 m/s	
Ulnar	>10 Uv		>45 m/s	
Radial	>15 Uv		>50 m/s	
Musculocutaneous UL	10 Uv		>50 m/s	
Sural	>10 uV		>40 m/s	
Musculocutaneous LL	>10 Uv		>40 m/s	

UL: Upper limb, CFN: Common fibular nerve, TN: Tibial nerve, LL: Lower limb

The severity of ENMG abnormalities was classified according to a system adapted from Baba et al.[18] as follows:

- **Mild (early):** Isolated conduction abnormalities or mild amplitude reduction limited to lower-limb nerves.
- **Moderate:** Significant amplitude reduction and slowing of conduction velocities in the lower limbs without upper-limb involvement.
- **Severe:** Absence of recordable sensory or motor responses in at least two lower-limb nerves.
- **Very severe:** Diffuse axonal loss with absent responses extending to upper-limb nerves (median and ulnar).

Operational definitions:

- **Diabetes mellitus:** Fasting plasma glucose >7 mmol/L (126 mg/dl) on two separate measurements after at least 8 hours of fasting, or random plasma glucose >11.1 mmol/L (200 mg/dl) in the presence of typical symptoms (weight loss, polyuria, polydipsia).
- **Diabetic neuropathy (DN):** Clinical diagnosis based on history and neurological examination, defined by subjective symptoms (paresthesia, neuropathic pain) and/or objective signs (sensory loss, weakness, muscle atrophy, reduced or absent deep tendon reflexes, cranial nerve involvement).
- **Polyneuropathy:** Simultaneous involvement of multiple peripheral nerves.
- **Mononeuropathy:** Involvement of a single peripheral nerve.
- **Polyradiculoneuropathy:** Diffuse involvement of peripheral nerves and nerve roots.
- **Painful diabetic neuropathy (PDN):** DN4 score ≥4.
- **Cardiovascular autonomic neuropathy (CAN):** Orthostatic hypotension defined as a drop in systolic blood pressure ≥20 mmHg or diastolic blood pressure ≥10 mmHg within 3 minutes of standing.
- **Digestive autonomic neuropathy (DAN):** Presence of gastroparesis and/or enteropathy (chronic constipation, diarrhea, or fecal incontinence).
- **Urogenital autonomic neuropathy (UGAN):** Bladder dysfunction, urinary retention, urgency, erectile dysfunction/retrograde ejaculation in male, or anorgasmia in female.
- **Sudomotor dysfunction:** Anhidrosis of the lower limbs and/or hyperhidrosis of the face and trunk.
- **Chronic renal failure (CRF):** Patients receiving treatment for renal failure or with estimated glomerular filtration rate (eGFR)<90 ml/min.
- **Dyslipidemia:** Defined by lipid-lowering therapy or LDL-c >1.8 mmol/L in high-risk patients, or >2.5 mmol/L in other diabetic patients.
- **Sedentary lifestyle:** Physical inactivity defined as <30 minutes of moderate-to-vigorous physical activity per day.
- **Diabetes control:** Assessed using glycated hemoglobin (HbA1c) and fasting plasma glucose levels.

RESULTS

Descriptive Study

A total of 64 patients with DN were recruited, representing 20% of all ENMG examinations performed during the study period. The mean age was 64.6±8 years (range: 45-81 years), with a slight female predominance (53.1%; female-to-male ratio: 1.13). Most patients resided in urban areas (84.4%) and were married (75.0%). Retired individuals represented the largest socio-professional group (40.6%). The sociodemographic characteristics of the study population are summarized in Table 2.

Table 2. Sociodemographic characteristics (n=64)

Characteristic	Number (n)	Percentage
Age (years) mean±SD: 64.6 years ±8 years (extremes: 45-81 years)		
(45-55)	2	3.1
(55-65)	32	50.0
(65-75)	22	34.4
(75-85)	8	12.5
Gender (gender ratio F/M=1.13)		
Female	34	53.1
Male	30	46.9
Place of residence		
Urban	54	84.4
Rural	10	15.6
Education		
Educated	44	68.7
Not in school	20	31.3
Marital status		
Married	48	75.0
Widowed	16	25
Professional activity		
Retired	26	40.6
Homemaker	18	28.1
Merchant	6	9.4
Civil servant	6	9.4
Private individual	6	9.4
Farmer	2	3.1

SD: Standard deviation, F/M: Female/male

Type 2 DM (T2D) accounted for 96.9% (n=62) of cases. The mean duration of diabetes was 11.3±7.8 years, and 56.2% of patients had a disease duration between 5 and 15 years. All patients were on antidiabetic treatment, with 65.6% managed without insulin. Glycemic control was poor, with 87.5% showing elevated fasting blood glucose levels and 93.8% having HbA1c >7%. Diabetes-related characteristics and glycemic control parameters are presented in Table 3.

Table 3. Diabetes characteristics and glycemic control (n=64)

Characteristic	Number (n)	Percentage
Type of DM		
Type 2 (T2D)	62	96.9
Type 1 (T1D)	2	3.1
Duration of diabetes mellitus (years) average SD: 11.3 years ±7.8 years (extremes: 0.08-39)		
<5	10	15.6
(5-10)	18	28.1
(10-15)	18	28.1
(15-20)	6	9.4
≥20	12	18.8
Relationship to treatment (insulin)		
Non-insulin dependent	42	65.6
Insulin-dependent	12	18.8
Insulin-requiring	10	15.6
Glycemic control (short term)		
Fasting blood glucose >6.1 mmol/L	56	87.5
Fasting blood glucose <6.1 mmol/L	8	12.5
Glycemic control (long term)		
HbA1c >7%	60	93.8
HbA1c <7%	4	6.2

DM: Diyabetes mellitus, SD: Standard deviation, HbA1c: Hemoglobin A1c

Hypertension was the most frequent cardiovascular risk factor (81.3%), followed by sedentary lifestyle (56.3%). The most common chronic complications were heart disease (28.1%) and diabetic nephropathy (12.5%).

Cardiovascular risk factors, diabetes-related complications, and associated comorbidities are detailed in Table 4.

Table 4. Cardiovascular risk factors, diabetes complications, and associated comorbidities (n=64)

Characteristic	Number (n)	Percentage
Associated cardiovascular risk factors		
HBP	52	81.3
Sedentary lifestyle	36	56.3
Dyslipidemia	24	37.5
Obesity	20	31.3
Alcohol	18	28.1
Tobacco	4	6.2
Chronic complications of diabetes		
Heart disease	18	28.1
Diabetic nephropathy	8	12.5
Diabetic retinopathy	6	9.4
Diabetic arteriopathy	4	6.3
Ischemic stroke	2	3.1
Comorbidities associated with neuropathy		
Lumbosciatica	6	9.4
Asthma	2	3.1
Severe weight loss	2	3.1
Polyarthralgia	2	3.1

HBP: High blood pressure

All patients presented at least one sensory symptom. Pain was the most frequent complaint (87.5%), leading to a diagnosis of painful DN (DN4 ≥4) in 84.4% of cases. Autonomic dysfunction was observed in 87.5% of patients, predominantly erectile dysfunction (32.1%). Motor impairment was present in 71.9% of patients. Overall, clinical evaluation showed painful diabetic polyneuropathy in 84.4%, autonomic neuropathy in 78.1%, and generalized peripheral neuropathy in 87.5%.

The clinical manifestations and different forms of DN observed in the study population are presented in Table 5.

Table 5. Clinical manifestations and types of neuropathy diabetic (n=64)

Characteristic	Number (n)	Percentage
Subjective sensory signs (n=64)		
Total pain	56	87.5
Burning pain	44	68.8
Numbness	38	59.4
Tingling	34	53.1
Itching	14	21.9
Neuropathic pain (DN4 ≥4)		
Diagnosed	54	84.4
Objective sensory signs (n=64)		
Hypoesthesia to touch	12	18.8
Hyperesthesia to touch	8	12.5
Motor signs (n=46: 71.9% of N=64)		
Motor deficit LL	16	(34.8%)
Motor deficit of one LL	14	(30.4%)
Motor deficit of one UL	7	15.2
Motor deficit of both UL	9	19.6
Neurovegetative signs (n=56: 87.5% of N=64)		
Erectile dysfunction	18	(32.1%)
Urinary disorders	14	(25.0%)
Sweating disorder	12	(21.4%)
Gastrointestinal disorders	8	(14.3%)
Clinical types of neuropathy (n=64)		
Painful diabetic polyneuropathy	54	84.4
Peripheral neuropathy (total)	56	87.5
Autonomic neuropathy (total)	50	78.1
Urogenital neuropathy	24	37.5
Cardiac autonomic neuropathy	14	21.9
Radiculopathy	4	6.2
Multiple mononeuropathy	2	3.1

DN4: Douleur neuropathique en 4 questions, LL: Lower limb, UL: Upper limb

ENMG was abnormal in 87.5% (n=56) of patients. Quantitative analysis revealed prolonged distal motor latencies and reduced motor and sensory conduction velocities, predominantly in the lower limbs (mean tibial motor conduction velocity: 37.5±10.6 m/s; mean sural sensory amplitude: 4.0±4.7 µV).

Quantitative ENMG findings are summarized in Table 6.

Qualitative analysis showed that 92.8% of abnormal ENMGs were consistent with axonal damage. Chronic length-dependent sensory-motor axonal polyneuropathy was the most frequent electrophysiological pattern (75.0%). Nearly 50% of patients presented with severe or very severe involvement (50.0% of pathological ENMGs).

Table 6. Average quantitative characteristics of ENMG in patients

Nerve		n	Mean/SD	Min	Max	Unit
Median nerve (upper limb)	MCV	56	49.8±7.7	34	67.6	m/s
	DML		8.1±5.3	2.3	15.7	ms
	F wave latency		46.9±9.3	27.5	68	ms
	Motor amplitude		6.1±2.5	1.0	16	mV
	SCV		49.9±11.5	10.8	75.0	m/s
	Sensory amplitude		9.1±13.1	2.0	24	µV
Ulnar nerve (upper limb)	MCV	50	51.8±9.5	27.0	70.0	m/s
	DML		9.8±4.2	3.3	20.2	ms
	F wave latency		48.4±9.3	26.2	68.0	ms
	Motor amplitude		5.5±2.7	0.6	11.7	mV
	SCV		52.3±14.7	22.0	76.7	m/s
	Sensory amplitude		10.1±13.9	1.0	24.0	µV
Fibular nerve (lower limb)	MCV	60	39.8±8.7	17.0	61.7	m/s
	DML		8.7±5.2	1.2	20.4	ms
	F wave latency		48.7±9.9	32.0	83.0	ms
	Motor amplitude		2.7±2.6	0.1	13.3	mV
Tibial nerve (lower limb)	MCV	64	37.5±10.6	15.5	64.0	m/s
	DML		9.1± 5.3	1.0	20.8	ms
	F wave latency		48.7±9.0	28.9	66.0	ms
	Motor amplitude		2.8±2.4	0.1	11.7	mV
Sural nerve (lower limb)	SCV	64	45.8±12.8	20.5	68.8	m/s
	Sensory amplitude		4.0±4.7	0.1	19.0	µV

ENMG: Electroneuromyography, SD: Standard deviation, Min: Minimum, Max: Maximum, MCV: Motor conduction velocity, DML: Distal motor latency, SCV: Sensory conduction velocity

The qualitative ENMG findings and severity classification are presented in Table 7.

Table 7. Qualitative characteristics and classification of ENMG findings (n=56 pathological ENMG)

Characteristic	Number (n)	Percentage
ENMG assessment (n=64)		
Pathological ENMG	56	87.5
Normal ENMG	8	12.5
Dominant pathophysiological type (n=56)		
Axonal damage (axonopathy)	52	92.8
Demyelinating disorder (myelinopathy)	4	7.2
Electrophysiological profile (n=56)		
Sensory-motor axonal polyneuropathy (L-D)	42	75.0
Sensory axonal polyneuropathy (L-D)	6	10.7
Chronic demyelinating polyneuropathy	4	7.1
Other (mono/multifocal, radiculoplexopathy)	4	7.2
Severity of ENMG findings (n=56)		
Early	2	(3.6%)
Moderate	26	(46.4%)
Severe	18	(32.1%)
Very severe	10	(17.9%)

ENMG: Electroneuromyography, L-D: Length-dependent

Analytical Study

In bivariate analysis, severe ENMG involvement was significantly associated with diabetes duration ≥10 years (OR=6.55; 95% CI: 1.95-21.9; p=0.002), age ≥65 years (OR=3.33; 95% CI: 1.15-9.61; p=0.024), motor impairment (OR=2.52; 95% CI: 0.89-7.09; p=0.078), and diabetic nephropathy (OR=3.50; 95% CI: 0.67-18.2; p=0.131). Factors associated with severe ENMG involvement in bivariate analysis are presented in Table 8.

In multivariate analysis, only diabetes duration ≥10 years remained an independent predictor of severe ENMG involvement (adjusted OR=5.32; 95% CI: 1.52-18.5; p=0.009).

After adjustment, age and motor impairment were no longer significant, suggesting that they act as proxies of diabetes duration rather than independent risk factors (Table 9).

The results of the multivariate logistic regression analysis are presented in Table 9.

DISCUSSION

Our cohort of 64 patients, representing 20% of all ENMG examinations performed at the two study sites during the study period, consisted predominantly of older adults (mean age: 64.6 years) and patients with T2D (96.9%). This distribution reflects the ongoing epidemiological transition and the rising burden of T2D in aging populations in sub-Saharan Africa.[19,20] The slight female predominance (53.1%) and urban residence (84.4%) are consistent with reports from West Africa and likely reflect referral patterns to specialized neurophysiology services concentrated in urban centers.[21,22] The predominance of retired individuals (40.6%) suggests that DN primarily affects patients with long disease duration and reduced occupational activity.

A major finding of this study is the very poor glycemic control observed in our cohort, with 93.8% of patients presenting HbA1c levels >7% and 87.5% exhibiting elevated fasting blood glucose. These proportions are considerably higher than those reported in high-income countries, where glycemic targets are achieved in approximately 25-50% of patients.[24,25] Our findings are consistent with data from sub-Saharan Africa, where chronic hyperglycemia remains a major determinant of diabetic peripheral neuropathy.[26] This sustained metabolic imbalance likely represents a key contributor to microvascular complications, including diabetic neuropathy, and may reflect limited access to optimal diabetes care and structured follow-up.[20]

Hypertension (81.3%), sedentary lifestyle (56.3%), and dyslipidemia (37.5%) indicate a high burden of cardiometabolic comorbidities. These factors are known to synergistically contribute to small fiber dysfunction and axonal degeneration.[27] Their combined impact in African populations warrants further investigation.

Pain was the most frequent clinical manifestation, reported in 87.5% of patients, and painful DN (DN4 ≥4) was diagnosed in 84.4%. This high prevalence likely reflects referral bias. In Burkina Faso, access to ENMG remains limited due to resource constraints and shortage of trained personnel. Consequently, patients referred for EDX evaluation are often those with severe symptoms or refractory neuropathic pain. This selection bias may explain the high proportion of severe and very severe electrophysiological abnormalities (~50%) observed in our cohort and limits extrapolation to the general

Table 8. Factors associated with severe ENMG involvement

Factors	Severe/very severe damage (n=28)	Mild/moderate damage (n=28)	OR (95% CI)	p-value
Duration of diabetes				
≥10 years (vs. <10 years)	24 (85.7%)	13 (46.4%)	6.55 (1.95-21.9)	0.002
Age				
≥65 years (vs. <65 years)	18 (64.3%)	10 (35.7%)	3.33 (1.15-9.61)	0.024
Diabetic nephropathy				
Yes (vs no)	6 (21.4%)	2 (7.1%)	3.50 (0.67-18.2)	0.131
Clinical motor signs				
Yes (vs. no)	20 (71.4%)	14 (50.0%)	2.52 (0.89-7.09)	0.078
Dyslipidemia				
Yes (vs. no)	14 (50.0%)	10 (35.7%)	1.81 (0.64-5.10)	0.266
Hypertension				
Yes (vs no)	24 (85.7%)	21 (75.0%)	1.97 (0.52-7.49)	0.317
Gender				
Male (vs female)	16 (57.1%)	14 (50.0%)	1.34 (0.46-3.91)	0.587
Heart disease				
Yes (vs no)	8 (28.6%)	10 (35.7%)	0.74 (0.24-2.25)	0.601
HbA1c				
≥7% (vs. <7%)	27 (96.4%)	27 (96.4%)	N/A	1.000

ENMG: Electroneuromyography, OR: Odds ratio, CI: Confidence interval, HbA1c: Hemoglobin A1c

Table 9. Multivariate analysis: independent factors for severe ENMG involvement

Independent variables	Reference category	OR	95% CI	p
Duration of diabetes (≥10 years)	<10 years	5.32	1.52-18.5	0.009
Age (≥65 years)	<65 years	1.28	0.33-5.00	0.713
Clinical motor signs	No	1.90	0.51-7.05	0.336
Diabetic nephropathy	No	2.15	0.38-12.1	0.387

ENMG: Electroneuromyography, OR: Odds ratio, CI: Confidence interval

diabetic population. As a result, early and asymptomatic neuropathy is likely underdiagnosed.

Autonomic dysfunction was observed in 87.5% of patients, predominantly erectile dysfunction (32.1%), while motor impairment was present in 71.9%. The coexistence of sensory, motor, and autonomic involvement suggests advanced and diffuse peripheral nerve damage. Similar patterns have been reported in West African cohorts, particularly in Senegal and Mali among patients undergoing ENMG evaluation.[28,29]

EDX studies revealed abnormal findings in 87.5% of patients, with axonal neuropathy accounting for 92.8% of cases. The predominant pattern was chronic length-dependent sensory-motor axonal polyneuropathy (75.0%). This predominance of axonal damage is consistent with experimental and clinical evidence showing that chronic hyperglycemia induces oxidative stress, mitochondrial dysfunction, and accumulation of advanced glycation end products, ultimately leading to axonal degeneration.[30,31]

Approximately half of the patients (50.0%) presented with severe or very severe electrophysiological impairment, a proportion higher than that reported in screening-based cohorts, where milder forms predominate. This finding further supports the hypothesis of delayed diagnosis and late referral in our setting.

Diabetes duration ≥10 years was the only independent predictor of severe ENMG abnormalities (adjusted OR=5.32; 95% CI: 1.52-18.5; p=0.009). This is consistent with large international studies, including EURODIAB and DCCT/EDIC, which identify disease duration as the most consistent risk factor for diabetic neuropathy, independent of glycemic control and other covariates.[32,33] In the study by Tesfaye et al.,[33] the risk of neuropathy increased progressively after 10 years of diabetes, reaching more than a fourfold increase in long-term disease.

Although age ≥65 years was associated with severity in univariate analysis, this association lost significance after adjustment for diabetes duration, suggesting that age mainly reflects cumulative metabolic exposure rather than an independent causal factor. Similar observations have been reported in several African and Asian cohorts.[34-36]

Motor impairment showed a non-significant trend toward association with severe neuropathy (p=0.078), consistent with electrophysiological evidence indicating that motor involvement may become more prominent with increasing neuropathy severity.[37,38] This underscores the importance of early screening before irreversible nerve damage occurs.

Although diabetic nephropathy and dyslipidemia are recognized comorbidities, no significant association was observed in this study. However, previous studies from Kenya and Egypt have reported a moderate association between renal dysfunction and severe neuropathy, possibly reflecting shared microvascular mechanisms.[39,40] This discrepancy may be explained by sample size limitations, cross-sectional design, and heterogeneity in diagnostic criteria.

Overall, our findings are consistent with regional data from sub-Saharan Africa, where studies conducted between 2021 and 2024 report high rates of severe neuropathy and consistently identify disease duration as the main determinant.[41,42] These results suggest that, despite regional

variability, the underlying pathophysiological mechanisms-oxidative stress, microangiopathy, and chronic metabolic injury-are universal but amplified in low-resource settings due to delayed diagnosis and suboptimal glycemic control.[40]

Limitations

This study provides combined clinical and electrophysiological data in a resource-limited African setting. Strengths include prospective data collection and standardized clinical and neurophysiological assessment. However, limitations include the relatively small sample size, two-center design, and absence of longitudinal follow-up, which limit generalizability. Additionally, the lack of quality-of-life assessment and quantitative pain evaluation represents another limitation.

CONCLUSION

This study identified the main determinants of DN severity in our setting. Diabetes duration emerged as the strongest independent predictor of severe EDX impairment, confirming its central role in the progression of diabetic peripheral nerve damage.

Although advanced age and the presence of clinical motor signs were associated with severity in bivariate analysis, they did not retain statistical significance after multivariate adjustment, suggesting that they mainly reflect disease chronicity rather than independent pathogenic factors.

These findings are consistent with recent international and African literature and reinforce the importance of early detection, regular neurophysiological assessment, and strict glycemic control to prevent progression to severe forms and reduce functional disability.

Further longitudinal studies with larger sample sizes are required to confirm these associations and to explore additional metabolic and therapeutic determinants of DN severity.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was conducted with the approval of the Institutional Ethics Committee of Saint Camille Hospital in Ouagadougou (Date: 09.11.2023, Decision No: 2023-11-09).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Conceptualization: AZ, AAD, AM; Study Design: AZ, AAD; Methodology: AZ, AAD, MRK, AM; Investigation: AZ; Data Collection: AZ, AAD, MRK; Data Analysis and Interpretation: AZ; Literature Review: AZ; Manuscript Drafting: AZ; Clinical: AAD; Electrophysiological Investigation: AAD; Interpretation of Findings: AAD, MRK, AM; Critical Review of the Manuscript: AAD, MRK, AM; Clinical Investigation: MRK; Study Supervision: AM.

REFERENCES

1. Said G. Diabetic neuropathy. *Handb Clin Neurol*. 2013;115:579-589. doi:10.1016/B978-0-444-52902-2.00033-3
2. Boulton AJ, Gries FA, Jervell JA. Guidelines for the diagnosis and outpatient management of diabetic peripheral neuropathy. *Diabet Med.* 1998;15(6):508-514. doi:10.1002/(SICI)1096-9136(199806)15:6<508::AID-DIA613>3.0.CO;2-L
3. Boulton AJ, Vinik AI, Arezzo JC, et al. Diabetic neuropathies: a statement by the American Diabetes Association. *Diabetes Care*. 2005; 28(4):956-962. doi:10.2337/diacare.28.4.956
4. Tesfaye S, Boulton AJ, Dyck PJ, et al. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. *Diabetes Care*. 2010;33(10):2285-2293. doi:10.2337/dc10-1303
5. The DCCT Research Group. Factors in development of diabetic neuropathy: baseline analysis of neuropathy in feasibility phase of Diabetes Control and Complications Trial (DCCT). *Diabetes*. 1988;37(4):476-481. doi:10.2337/diab.37.4.476
6. Boulton AJM, Cavanagh PR, Rayman G, eds. The Foot in Diabetes. 4th ed. John Wiley & Sons; 2006. doi:10.1002/0470029374
7. Tracy JA, Dyck PJ. The spectrum of diabetic neuropathies. *Phys Med Rehabil Clin N Am*. 2008;19(1):1-v. doi:10.1016/j.pmr.2007.10.010
8. Hartemann A, Lozeron P. Peripheral neuropathies in diabetics. *MCED*. 2015;74:70-73.
9. Braffett BH, Gubitosi-Klug RA, Albers JW, et al. Risk factors for diabetic peripheral neuropathy and cardiovascular autonomic neuropathy in the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) Study. *Diabetes*. 2020; 69(5):1000-1010. doi:10.2337/db19-1046
10. Tesfaye S, Stevens LK, Stephenson JM, et al. Prevalence of diabetic peripheral neuropathy and its relation to glycaemic control and potential risk factors: the EURODIAB IDDM Complications Study. *Diabetologia*. 1996;39(11):1377-1384. doi:10.1007/s001250050586
11. Pop-Busui R, Boulton AJ, Feldman EL, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. *Diabetes Care*. 2017;40(1):136-154. doi:10.2337/dc16-2042
12. Ziegler D, Strom A, Lobmann R, Reiners K, Rett K, Schnell O. High prevalence of diagnosed and undiagnosed polyneuropathy in subjects with and without diabetes participating in a nationwide educational initiative (PROTECT study). *J Diabetes Complications*. 2015;29(8):998-1002. doi:10.1016/j.jdiacomp.2015.09.008
13. Mather KJ, Bebu I, Baker C, et al. Prevalence of microvascular and macrovascular disease in the Glycemia Reduction Approaches in Diabetes-A Comparative Effectiveness (GRADE) Study cohort. *Diabetes Res Clin Pract*. 2020;165:108235. doi:10.1016/j.diabres.2020.108235
14. Andersen ST, Witte DR, Dalsgaard EM, et al. Risk factors for incident diabetic polyneuropathy in a cohort with screen-detected type 2 diabetes followed for 13 years: ADDITION-Denmark. *Diabetes Care*. 2018;41(5):1068-1075. doi:10.2337/dc17-2062
15. Dabilgou AA, Tieno H, Tiendrebeogo JZ, Yameogo VN, Zett CM, Millogo A. Frequency, risk factors and clinical forms of neuropathies in diabetics in hospitals in Burkina Faso. *J Diabetes Mellitus*. 2018;8(3):70-83. doi:10.4236/jdm.2018.83008
16. Fournier E. Practical Guide to Electromyography and Evoked Potentials. 3rd ed. Masson; 2018.
17. Fournier E. Manuel d'électromyographie. Lavoisier Médecine Sciences; 2021.
18. Baba M, Suzuki C, Ogawa Y. Severity grading system of diabetic neuropathy in type-2 diabetes by nerve conduction study: five-year prospective study on occurrence of diabetic foot, macroangiopathic events, and eventual death. *Jpn J Clin Neurophysiol*. 2018;46(2):71-77. doi:10.11422/jscn.46.71

19. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. International Diabetes Federation; 2021.
20. Motala AA, Mbanya JC, Ramaiya K, Pirie FJ, Ekoru K. Type 2 diabetes mellitus in sub-Saharan Africa: challenges and opportunities. *Nat Rev Endocrinol*. 2022;18(4):219-229. doi:10.1038/s41574-021-00613-y
21. Kuate-Tegueu C, Temfack E, Ngankou S, Doumbe J, Djientcheu VP, Kengne AP. Prevalence and determinants of diabetic polyneuropathy in a sub-Saharan African referral hospital. *J Neurol Sci*. 2015;355(1-2):108-112. doi:10.1016/j.jns.2015.05.035
22. Maiga Y, Diallo S, Konipo FD, et al. Diabetic polyneuropathy with/out neuropathic pain in Mali: a cross-sectional study in two reference diabetes treatment centers in Bamako (Mali), Western Africa. *PLoS One*. 2020;15(11):e0241387. doi:10.1371/journal.pone.0241387
23. Liu X, Xu Y, An M, Zeng Q. The risk factors for diabetic peripheral neuropathy: a meta-analysis. *PLoS One*. 2019;14(2):e0212574. doi:10.1371/journal.pone.0212574
24. Carls G, Huynh J, Tuttle E, Yee J, Edelman SV. Achievement of glycated hemoglobin goals in the US remains unchanged through 2014. *Diabetes Ther.* 2017;8(4):863-873. doi:10.1007/s13300-017-0280-5
25. Fina Lubaki JP, Omole OB, Francis JM. Glycaemic control among type 2 diabetes patients in sub-Saharan Africa from 2012 to 2022: a systematic review and meta-analysis. *Diabetol Metab Syndr.* 2022;14:134. doi:10.1186/s13098-022-00902-0
26. Shiferaw WS, Akalu TY, Work Y, Aynalem YA. Prevalence of diabetic peripheral neuropathy in Africa: a systematic review and meta-analysis. *BMC Endocr Disor*d. 2020;20(1):49. doi:10.1186/s12902-020-0534-5
27. Callaghan BC, Price RS, Feldman EL. Distal symmetric polyneuropathy: a review. *JAMA*. 2015;314(20):2172-2181. doi:10.1001/jama.2015.13611
28. Fentie D, Solomon Y, Abebe F, Derese T, Tigstu F, Hailemariam T. Diabetic peripheral neuropathy and their associations with overweight/obesity and impaired blood glucose among diabetic patients in eastern Ethiopia: institutional-based study. *SAGE Open Med*. 2023;11:20503121231220784. doi:10.1177/20503121231220784
29. Chokote SE, Lemdjo G, Idiaquez Rios JF, et al. Clinical and electrophysiological characterization of diabetic neuropathy in a sub-Saharan African cohort. *J Peripher Nerv Syst.* 2025;30(2):e70021. doi:10.1111/jns.70021
30. Feldman EL, Callaghan BC, Pop-Busui R, et al. Diabetic neuropathy. *Nat Rev Dis Primers*. 2019;5(1):41. doi:10.1038/s41572-019-0092-1
31. Feldman EL, Nave KA, Jensen TS, Bennett DLH. New horizons in diabetic neuropathy: mechanisms, bioenergetics, and pain. *Neuron*. 2017;93(6):1296-1313. doi:10.1016/j.neuron.2017.02.005
32. Elliott J, Sloan G, Stevens L, et al. Female sex is a risk factor for painful diabetic peripheral neuropathy: the EURODIAB prospective diabetes complications study. *Diabetologia*. 2024;67(1):190-198. doi: 10.1007/s00125-023-06025-z
33. Tesfaye S, Kempler P. Conventional management and current guidelines for painful diabetic neuropathy. *Diabetes Res Clin Pract*. 2023;206(suppl 1):110765. doi:10.1016/j.diabres.2023.110765
34. Valensi P, Giroux C, Seeboth-Ghalayini B, Attali JR. Diabetic peripheral neuropathy: effects of age, duration of diabetes, glycemic control, and vascular factors. *J Diabetes Complications*. 1997;11(1):27-34. doi:10.1016/S1056-8727(95)00086-0
35. Amour AA, Chamba N, Kayandabila J, et al. Prevalence, patterns, and factors associated with peripheral neuropathies among diabetic patients at tertiary hospital in the Kilimanjaro Region: descriptive cross-sectional study from North-Eastern Tanzania. *Int J Endocrinol*. 2019;2019:5404781. doi:10.1155/2019/5404781
36. Bansal D, Gudala K, Muthyala H, Esam HP, Nayakallu R, Bhansali A. Prevalence and risk factors of development of peripheral diabetic neuropathy in type 2 diabetes mellitus in a tertiary care setting. *J Diabetes Investig*. 2014;5(6):714-721. doi:10.1111/jdi.12223
37. Lamontagne A, Buchthal F. Electrophysiological studies in diabetic neuropathy. *J Neurol Neurosurg Psychiatry*. 1970;33(4):442-452. doi:10.1136/jnnp.33.4.442
38. Sloan G, Selvarajah D, Tesfaye S. Pathogenesis, diagnosis and clinical management of diabetic sensorimotor peripheral neuropathy. *Nat Rev Endocrinol*. 2021;17(7):400-420. doi:10.1038/s41574-021-00496-z
39. Issar T, Arnold R, Kwai NCG, et al. Relative contributions of diabetes and chronic kidney disease to neuropathy development in diabetic nephropathy patients. *Clin Neurophysiol*. 2019;130(11):2088-2095. doi:10.1016/j.clinph.2019.08.005
40. Jasti DB, Mallipeddi S, Apparao A, Vengamma B, Sivakumar V, Kolli S. A clinical and electrophysiological study of peripheral neuropathies in predialysis chronic kidney disease patients and relation of severity of peripheral neuropathy with degree of renal failure. *J Neurosci Rural Pract*. 2017;8(4):516-524. doi:10.4103/jnrp.jnrp_186_17
41. Kibirige D, Chamba N, Andia-Biraro I, et al. Indicators of optimal diabetes care and burden of diabetes complications in Africa: a systematic review and meta-analysis. *BMJ Ope*n. 2022;12(11):e060786. doi:10.1136/bmjopen-2022-060786
42. Negussie YM, Bekele NT. Diabetic peripheral neuropathy among adult type 2 diabetes patients in Adama, Ethiopia: health facility-based study. *Sci Rep*. 2024;14:3844. doi:10.1038/s41598-024-53951-y