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# Prevalence and Electroclinical Profile of Peripheral Neuropathies in PLHIV Followed Up in Brazzaville

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## Abstract

**Background:** The HIV pandemic leads to neurological impairments, which can manifest at any stage of the disease. The peripheral nervous system is often the most affected. **Aim:** To determine the prevalence and electroclinical profile of peripheral neuropathies (PNs) among PLHIV treated at the Brazzaville Outpatient Treatment Centre (OTC). **Patients and Methods:** A descriptive cross-sectional study was conducted from June to September 2021. Participants were PLHIV aged 18 years or older, on antiretroviral therapy (ART), with or without PN symptoms, in whom PN was confirmed by electroneuromyography (ENMG). Sociodemographic, anthropometric, clinical, and electrophysiological data were collected and analyzed using SPSS 28.0. **Results**: Among 62 patients who underwent ENMG, 50% presenting PN. Their mean age was 57.1 ± 8.9 years, and there was a female predominance (sex ratio = 0.7). Most were at HIV clinical stages 3 or 4, and all had undetectable viral load. Subjective neuropathic symptoms were reported in 19 (29.7%) patients; decreased osteotendinous reflexes were noted in 14 (87.5%) patients. ENMG findings showed that PN was symptomatic in 14 (43.7%) patients and subclinical in 18 (56.2%). The most frequently observed neuropathies were mononeuropathy (46.9%), particularly carpal tunnel syndrome (84.1%), and axonal sensory polyneuropathy (40.6%). **Conclusions:** Peripheral neuropathy is common among PLHIV, frequently presenting in a subclinical form. Early detection through detailed clinical history, thorough neurological examination, and systematic ENMG is crucial to optimizing overall HIV care.

**Keywords**

Peripheral Neuropathies, ENMG, PLHIV, Brazzaville

## 1. Introduction

Human immunodeficiency virus (HIV) infection continues to be a major global health challenge, affecting diverse populations across all world regions [1]. According to recent estimates by UNAIDS, approximately 37.7 and 40.4 million people were living with HIV (PLHIV), respectively in 2020 and 2024, with sub-Saharan Africa bearing the highest burden. In East and Southern Africa, around 20.6 million people are estimated to be living with HIV, and an additional 4.7 million reside in West and Central Africa, in 2020 [2] [3]. In the Republic of Congo, the HIV epidemic has led to the identification of nearly 110,000 PLHIV [4].

One of the hallmarks of HIV infection is its capacity to affect multiple organ systems, including the nervous system, owing to the virus's notable neurotropism [1] [5]. Neurological manifestations of HIV are common and rank third after dermatological and gastrointestinal disorders in terms of frequency [1]. These manifestations can appear at any point during infection, from the early stages to advanced stages (AIDS), where immunosuppression makes patients susceptible to opportunistic infections and other complications [5] [6]. Moreover, some antiretroviral drugs (ARVs) themselves may contribute to neurological side effects [6] [7].

Among the various neurological complications, peripheral neuropathies (PN) are the most frequently observed in PLHIV, with reported prevalence rates ranging widely from 17.7% to 68.2% [5] [8]-[12]. This broad range reflects differences in study populations, diagnostic criteria, disease stage, and access to comprehensive HIV care. PNs in HIV can be symptomatic—manifesting with pain, paresthesia, or motor weakness in up to 52% of cases—or subclinical, detected only by neurophysiological testing in as many as 71% [6] [13] [14].

Before the initiation of antiretroviral therapy (ART), the prevalence of PNs in PLHIV was approximately 22.6% of PLHIV, with only 4.3% presenting symptoms [15]. Following ART initiation, however, these rates rose progressively to 32.1% or 52.8% at three years, despite successful virological suppression and immunological reconstitution [15] [16]. While symptomatic NPs remained relatively lower, their prevalence also increased to 8.6% or 24% at three years [15] [16].

Several factors are associated with the development of PNs in PLHIV, affecting both adults and children. Some factors are time-related, such as older age and longer disease duration. Others are therapy-related, including previous or current use of protease inhibitors, isoniazid, neurotoxic ARVs, or newer ARVs [12] [15]-[19]. In addition, Black race, female sex, low initial or current CD4 count, and comorbidity with diabetes have also been reported [13] [15] [16].

The spectrum of HIV-associated peripheral neuropathies is extensive and in-

cludes mainly distal symmetric polyneuropathies (DSPN) and antiretroviral toxic neuropathies (ATN), the most common forms and predominantly sensory or sensorimotor that follow a subacute or chronic course [20]-[22]. Other presentations included: polyradiculoneuropathies, affecting nerve roots and peripheral nerves; mononeuropathies and multiple mononeuropathies, affecting single or multiple isolated nerves; meningoradiculoneuropathies, involving both meninges and nerve roots; and autonomic neuropathies, leading to dysautonomia with cardiovascular, gastrointestinal, or genitourinary symptoms [15] [21]-[23].

Clinical assessment is critical for the initial recognition of PNs, typically based on patient-reported symptoms, physical examination findings (e.g., reduced distal reflexes, sensory deficits), and risk-factors evaluation [14]-[17] [21]. However, electroneuromyography (ENMG) plays a key confirmation role, characterizing the pathophysiological mechanism (demyelinating or axonal) and helping to differentiate HIV-related neuropathies from those caused by comorbidities such as diabetes or vitamin deficiencies [14]. Despite their clinical significance, the ENMG features of HIV-associated neuropathies remain underexplored in many regions, including sub-Saharan Africa.

PNs affect the quality of life of PLHIV by compromising their functional status and increasing the incidence of depression, anxiety, and insomnia when sensory disturbances are present [12] [16] [21].

In Congo, where HIV prevalence remains a significant public health concern, a deeper understanding of the patterns and electrophysiological profiles of PNs in PLHIV is crucial for early detection, targeted management, and improved quality of life. Previous investigations have primarily focused on clinical manifestations of neuropathies, with limited attention given to their electroneuromyographic correlates [24] [25].

The present study aims to determine the prevalence of PN and define their clinical and ENMG profiles, among PLHIV attending the Brazzaville Outpatient Treatment Centre (OTC), thereby contributing evidence to guide better clinical and therapeutic decision-making in this high-burden setting.

## 2. Patients and Methods

This descriptive cross-sectional study was conducted from June 1 to September 30, 2021, at two sites in Brazzaville (Republic of Congo). The Outpatient Therapy Center (OTC), the main center dedicated to the care and monitoring of PLHIV, and the clinical neurophysiology unit of the neurology department of the Brazzaville University Hospital.

### 2.1. Patient Selection and Data Collection Procedure

- **Screening and recruitment at the OCT**

All PLHIV attending their routine medical appointments at the Brazzaville OCT during the study period were systematically evaluated for potential inclusion. The OTC maintains an active detailed follow-up register of approximately 2900 pa-

tients and provides comprehensive care for PLHIV. Its services include screening, medical follow-up, medication dispensation, laboratory monitoring, days hospitalizations, nursing care, preventive interventions, psychological support, nutritional assistance and home visits. For this study, the research team collaborated with OTC healthcare professionals to identify eligible patients using the selection criteria and to conduct a standardized screening interview, which included a review of medical, laboratory (viral load and CD4 count) and treatment history, assessment of comorbidities (diabetes, tuberculosis, hepatitis C) and exclusion of conditions known to cause PNs.

- **Inclusion and exclusion criteria**

PLHIV aged 18 years or older, at any clinical stage of disease, with or without signs suggestive of neuropathy, and receiving ART were eligible for inclusion in the study. Patients were not considered for inclusion if they were taking medications other than ARVs known to cause neuropathy, had comorbidities such as chronic alcohol use, diabetes, hepatitis C, leprosy, tuberculosis or an autoimmune disease, or if they had a previously diagnosed and documented neuropathy at the time their HIV status was discovered. Those who refused to undergo electroneuromyography or to continue participation in the study were excluded.

All participants included in the study provided informed consent to participate.

## 2.2. Data Collection Process

Data were collected in two phases. First, clinical screening and interviews were conducted to gather sociodemographic and anthropometric data, and HIV-related information. A peripheral neuropathy assessment was performed to document subjective symptoms (such as pain, paresthesia, and numbness) and objective findings (neurological examination), with the DN4 questionnaire used to diagnose neuropathic pain. Second, an ENMG evaluation was conducted in the clinical neurophysiology unit for all participants. This included motor and sensory nerve conduction studies of the median, ulnar, fibular, tibial, and sural nerves, measuring of F-wave latencies for each tested nerve, and needle electromyography as needed. All recordings were obtained at a standardized skin temperature of 37˚C, and measurement accuracy was verified in accordance with established reference values by our clinical neurophysiology unit [26].

## 2.3. Diagnosis of Peripheral Neuropathies

The diagnosis of PN required both clinical and electrophysiological criteria to be met. Clinically, participants had to present with neuropathic symptoms (pain, paresthesia, numbness, cramps) and/or findings examination (decreased sensory perception, reduced or absent reflexes). Electrophysiological confirmation was based on abnormal nerve conduction parameters indicative of axonal, demyelinating, or mixed neuropathies. These criteria followed the French recommendations for diagnosing PNs [27].

### 2.4. Study Variables

The study variables were divided into primary and secondary categories.

Primary variables included previous and current subjective neuropathic complaints (neuropathic pain was assessed by DN4 questionnaire and score ≥ 4/10 indicated its presence) and objective neuropathic signs documented during clinical examination and ENMG evaluation (type of PN, fiber involvement, laterality, and underlying mechanism).

Secondary variables encompassed HIV-related factors (type of HIV, ART regime, duration of HIV infection and ART, viral load category < 50, 50 - 100, or >1000 copies/ml, clinical stage, presence of opportunistic infections, and Morisky adherence score high ≥ 8, moderate between 6 - 7, or low < 6), as well as sociodemographic and anthropometric parameters (age, sex, occupation and body mass index in $kg/m^2$).

### 2.5. Statistical Analysis

Data were entered and analyzed using SPSS version 28.0. Categorical variables were expressed as percentages. Continuous variables were reported as mean ± standard deviation (SD) if normally distributed or as median with interquartile range (IQR) otherwise.

As this study was primarily descriptive in nature, no comparative tests were conducted.

### 2.6. Ethical Considerations

All participants provided written informed consent. The study protocol was reviewed and approved by Ethics and Research Committee of the Faculty of Health Sciences, and all procedures were conducted in accordance with the Declaration of Helsinki.

In this descriptive cross-sectional study, no formal longitudinal follow-up was specifically designed to assess the progression or therapeutic efficacy of neuropathy. However, patients diagnosed with symptomatic neuropathy received standard clinical follow-up at the OTC. Specifically, these patients were referred for appropriate management—such as adjusting ART if needed, managing pain, and providing prevention and rehabilitation advice—and were re-evaluated during subsequent visits as part of their routine HIV care. Therefore, although the study did not include a prospective assessment focusing on neuropathy treatment efficacy, these patients were not lost to follow-up and continued to receive regular monitoring within the OTC's standard care pathway.

## 3. Results

Of the 2503 PLHIV registered at the OTC, 79 consented to participate. ENMG was not performed in 15 of these participants due to reluctance, and in 32 others, ENMG results did not confirm PN. Consequently, 32 (50%) participants with ENMG-confirmed PN were included in the study.

### 3.1. Sociodemographic and Anthropometric Characteristics

The study population included 19 women (59.4%) and 13 men (40.6%), with a sex ratio of 0.7. The mean age was 57.1 ± 8.9 years, with a range of 41 to 79 years. Table 1 presents the participant's occupational categories. Regarding body mass index (BMI), 12 patients (37.5%) had normal BMI, 10 (31.3%) were underweight, 6 (18.8%) were overweight, and 4 (12.5%) were classified as obese (12.5%).

Table 1. Occupational categories of patients.

| | n | % |
|---|---|---|
| Manual workers | 17 | 53.1 |
| Office workers | 5 | 15.6 |
| Retirees | 7 | 21.9 |
| Unemployed | 3 | 9.4 |

### 3.2. History of Human Immunodeficiency Virus Infection

The mean duration of HIV infection was 13.3 ± 5.2 years (range: 3 - 23 years). In five patients (15.6%), HIV was discovered incidentally, whereas in 27 patients (84.4%), it was identified during investigations for digestive disorders (68.8%), respiratory disorders (9.4%), or skin lesions (6.3%).

HIV-1 type was confirmed in 30 patients (93.8%), while in two patients (6.2%), serology indicated HIV infection, but the specific viral type was not recorded in the OTC database. All patients had an undetectable viral load (<50 copies/ml). With respect to clinical staging, six patients (18.8%) were at stage 1, three (9.4%) at stage 2, sixteen (50%) at stage 3, and seven (21.9%) at stage 4. No opportunistic infections were observed.

The mean duration of ART was 12.2 ± 5.9 years, ranging from 8 to 16 years. First-line regimens were used in 25 patients (78.1%), second-line in 6 patients (18.8%), and third-line in 1 patient (3.1%). Among those on first-line ART, 60% received a combination of tenofovir (TDF), lamivudine (3TC) or emtricitabine (FTC), and dolutegravir (DTG), while 40% were given TDF, 3TC/FTC, and efavirenz (EFV). In the second-line group, 83.3% received TDF, 3TC/FTC, and DTG, whereas 16.7% were switched to zidovudine (AZT), 3TC, plus lopinavir (LPV) or ritonavir (RTV). The one patient on third-line therapy receiving raltegravir (RAL) and darunavir (DRV) boosted with RTV.

Nine patients (28.1%) had been exposed to neurotoxic ARVs. Moreover, the study found that 16 patients (50%) demonstrated high treatment adherence, 13 (40.6%) showed moderate adherence, and three (9.4%) exhibited low adherence.

### 3.3. Characteristics of Peripheral Neuropathies

Among the 32 patients diagnosed with PN, 18 (56.3%) had subclinical PN, whereas 14 (43.7%) presented clinically symptomatic neuropathy.

- **Clinical profile**

Previous subjective neuropathic symptoms were reported by 18 (56.3%) patients; however, no clinical or electrophysiological investigations were undertaken to explore these symptoms.

Current subjective neuropathic complaints were documented in 15 (46.9%) patients, of whom 12 (80%) had sensory disorders (primarily paresthesia, see Figure 1) and 3 (20%) had motor disorders motor (manifesting mainly as cramps). No instances of neuropathic pain were noted. In 14 patients (93.3%), these symptoms had been present for more than six months, while one patient (6.7%) reported a duration of less than four weeks. Five patients (33.3%) had previously experienced subjective neuropathic symptoms and had a history of neurotoxic ARV use.

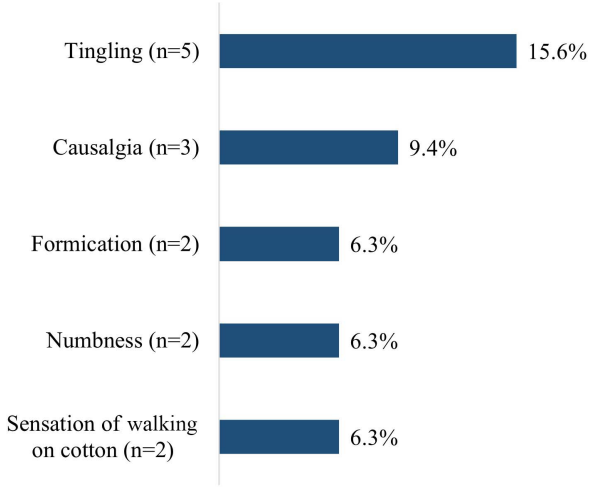


**Figure 1.** Type of paresthesia.

Neurological examination was abnormal in 14 (43.8%) patients. Among them, 1 (7.1%) had hypoesthesia, and all 14 (100%) showed decreased osteotendinous reflexes. These 14 patients also had subjective neuropathic symptoms.

Clinically, mononeuropathy was observed in 3 (21.4%) patients, and polyneuropathy was noted in 11 (78.6%).

- **Electrical profile**

Among the 32 patients with ENMG-confirmed PN, 15 (46.9%) had mononeuropathy, 13 (40.6%) had polyneuropathy, and 4 (12.5%) presented with a combination of both. All clinically suspected cases of neuropathy were confirmed by ENMG. In addition, 18 patients (56.2%) reported previous subjective neuropathic symptoms, while 15 (46.9%) described current symptoms.

***Characteristics of mononeuropathy***

Of the 19 mononeuropathies identified, 16 (84.1%) involved the median nerve. Ulnar nerve involvement, tibial nerve involvement, and combined median-ulnar nerve involvement were each observed in one (5.3%). In all instances, median nerve involvement was consistent with carpal tunnel syndrome, and the lesion mechanism involved was compressive. Details of the mononeuropathies are presented in Table 2.

**Table 2.** Characteristics of mononeuropathy.

| | Median N = 17 | | Ulnar N = 2 | | Tibial N = 1 | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| Type of fibers affected | | | | | | |
| Sensitive | 3 | 17.6 | 2 | 100 | - | - |
| Motor | 5 | 29.4 | - | - | - | - |
| Sensory-motor | 9 | 53.0 | - | - | 1 | 100 |
| Laterality of involvement | | | | | | |
| Left | 2 | 11.8 | 1 | 100 | 1 | 100 |
| Right | 6 | 35.2 | - | - | - | - |
| Bilateral | 9 | 53.0 | 1 | 100 | - | - |

### *Characteristics of polyneuropathy*

As shown in Table 3, the characteristics of polyneuropathies include their topography, the specific nerve fibers affected, and the underlying lesion mechanisms.

**Table 3.** Characteristics of polyneuropathy.

| | n | % |
|---|---|---|
| Topography | | |
| Lower limbs | 8 | 47 |
| Four members | 9 | 53 |
| Type of fibers affected | | |
| Sensitive | 14 | 82.4 |
| Sensory-motor | 3 | 17.6 |
| Mechanism of lesion | | |
| Axonal damage | 16 | 94.1 |
| Demyelination | 1 | 5.9 |

## 4. Discussion

### 4.1. Prevalence of Peripheral Neuropathies

Involvement of the peripheral nervous system in early and often subclinical stages is a well-documented phenomenon in HIV infection [28]. In the present study, the prevalence of PN was 50%, aligning with findings by Konchalard *et al.* [29], who used both clinical and electrophysiological evaluations in treated PLHIV. However, the literature presents variable rates of PN, influenced by the heterogeneity of study population and the use of different diagnosis criteria.

For instance, Ndouongo *et al.* [24] and Mehta *et al.* [30] reported lower prevalence rates of 27.5% and 36%, respectively, based on clinical criteria using the brief

peripheral neuropathy screen (BPNS). Similarly, Puplampu *et al.* [8] found a prevalence of 17.7% using a biothesiometer, a semi-quantitative device measuring vibratory perception thresholds with a 128 Hz tuning fork. These approaches, relying primarily on clinical criteria, may underestimate the true prevalence of PN. Conversely, Pillay *et al.* [31] observed a low prevalence (19%) in cohort of ARV-naïve patients who are evaluated clinically after six months of treatment, *i.e.*, a relatively short exposure period. In contrast, Simpson *et al.* [13] documented a higher prevalence (62%) by using a combination of clinical assessments, nerve conduction studies, and skin biopsy to quantify epidermal nerve fiber density—an approach that significantly increases diagnostic sensitivity.

It is imperative to note that epidermal nerve fibers density assessment through skin biopsy, combined with neurophysiological investigations, remains the most sensitive method for diagnosing PN. However, these techniques are often less accessible in many African settings due to high costs and limited availability, leading to more frequent reliance on clinical criteria in these regions.

In this study, a considerable number of patients were diagnosed with HIV clinical stage 3 or 4. At the time of the current investigation, all patients had undetectable viral load. The presence of PNs may be related to the advanced clinical stage at diagnosis, as PN commonly manifests in later stages of HIV infection due to immunosuppression [6] [13] [32]. It is hypothesized that peripheral nerve damage emerges during these stages yet remains subclinical in many cases, consistent with the finding that 56.2% of patients in this study exhibited subclinical PNs. Simpson *et al.* [13] and Morgello *et al.* [33] reported lower frequencies of subclinical PN (10% and 26.3%, respectively), while Skopelitis *et al.* [34] observed a rate of 13% in ENMG-detected subclinical forms. Histopathological evaluation is crucial for clarifying the underlying mechanisms, as well as the timing and progression of subtle ENMG changes before they become clinically evident. Nevertheless, ENMG remains a highly sensitive and specific tool for detecting neuropathies, and its diagnostic yield surpasses that of clinical examination alone [35] [36]. Its sensitivity, however, is reduced in cases involving small sensory fibers [37].

Most participants in this study were on first-line of ART and exhibited high or moderate adherence to their regimens. Although standard adult dosages were used in accordance with guidelines, detailed records of specific dosages and treatment durations were not consistently available, precluding a more comprehensive analysis of the potential dose-response relationship between ART and PN [38]. Future research would benefit from systematically recording all ART regimens, including precise dosages and treatment durations, to allow for a more nuanced examination of ART-associated neurotoxicity.

### 4.2. Current and Previous Subjective Neuropathic Disorders

The high incidence of subjective neuropathic disorders observed in this study underscores the importance of conducting an ENMG to confirm the presence of PN [27]. These neuropathic disorders were predominantly sensory in nature, and no-

tably, none were accompanied by neuropathic pain. In some cases, neurotoxic ARVs have been implicated in the onset of subjective neuropathic symptoms, which may regress clinically following the discontinuation of these medications. In Congo, "d" drugs were progressively withdrawn in 2009 in compliance with WHO directives [39]. Although such toxic neuropathies may show clinical reversibility, they are not always fully reversible on electrophysiological testing [19].

### 4.3. Characteristics of Peripheral Neuropathies

Polyneuropathy is one of the most common neuropathies among PLHIV. In this study, its prevalence was estimated at 20.3%, primarily manifesting as distal sensory polyneuropathies (DSP) with axonal injury. Reported rates in the literature vary between 37.5% and 64.7% [29] [34] [40]. Morgello *et al.* [33] documented a 53% prevalence of DSP, based on the presence of at least one subjective neuropathic symptom (paresthesia, dysesthesia, or numbness), potentially overestimating true prevalence. Cettomai *et al.* [41] similarly noted a 71% prevalence using subjective screening performed by non-physicians, while a pilot study by a neurologist reported only 20% [42].

Distal sensory polyneuropathy (DSP) and antiretroviral toxic neuropathies (ATN) can be clinically indistinguishable, especially among PLHIV receiving ART. Both pathologies involve the activation of glial and immune cells, resulting in the release of proinflammatory cytokines via distinct pathways [20] [43] [44]. HIV's neurotoxicity is largely indirect: the viral gp120 envelope protein activates chemokine receptor, macrophages, and Schwann cells [43]-[45]. In contrast, the neurotoxic effects of some ARVs—particularly nucleoside reverse transcriptase inhibitors NRTIs like didanosine, zalcitabine, and stavudine—stem from mitochondrial dysfunction caused by inhibition of DNA $\gamma$-polymerase, the enzyme essential for mitochondrial DNA replication and repair [20] [43] [44]. Despite these differing mechanisms, both DSP and ATN share hallmark pathological features, including "dying back" axonal degeneration in distal regions, unmyelinated fibers loss, and macrophage infiltration within peripheral nerves and dorsal root ganglia [20]. Skin biopsy has therefore emerged as a valuable diagnostic tool in PLHIV with suspected PN, revealing reduced intraepidermal nerve fiber density, increased varicosities, and nerve fiber fragmentation [20] [44] [45]. Other HIV-associated neuropathies include acute or chronic inflammatory polyneuropathies—rare presentations typically arising during seroconversion or early HIV stages—and neuropathies secondary to opportunistic infections (e.g., cytomegalovirus, herpes zoster virus), which occur predominantly in advanced AIDS and manifest as mononeuritis multiple or radiculopathies [20].

A noteworthy finding in this study was the high prevalence (25%) of carpal tunnel syndrome (CTS), markedly exceeding the 0.2% reported in the general population of Cotonou (0.2%) [32]. Manfredi *et al.* [46] identified only two cases of CTS in PLHIV without known risk factors, while Mastroianni *et al.* [47] reported a 6.59% prevalence. These observations point to a potential relationship

between HIV infection and CTS, possibly exacerbated by the large proportion of manual workers in our sample.

A principal challenge in this study was patients' reluctance to undergo ENMG, largely due to the procedure's time-consuming and invasive nature, despite detailed explanations of its clinical relevance.

## 5. Conclusion

In our study, peripheral neuropathies affected 50% of people living with HIV, with 56.2% of these cases being subclinical. Sensory complaints were the most common symptoms. Carpal tunnel syndrome and polyneuropathy emerged as the main neuropathic patterns, primarily characterized by sensory and axonal involvement. Systematic ENMG evaluation during the pre-therapeutic work-up could provide a clearer understanding of the relative contributions of antiretroviral therapy and HIV itself to the development of these neuropathies, especially when patients report subjective sensory symptoms.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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