Scientific Research Publishing

Neuroscience and Medicine, 2025, 16(4), 261-271
https://www.scirp.org/journal/nm
ISSN Online: 2158-2947
ISSN Print: 2158-2912

Peripheral Neuropathies Associated with Gammopathies in the Outpatient Clinic of the Neurology Department at the University Hospital Center of Libreville: Study of Two Cases and Literature Review

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How to cite this paper: Diouf Mbourou, N., Nsounda, A., Mboumba Mboumba, C., Gnigone, P., Nyangui Mapaga, J., Mambila, G., Saphou Damon, M.A., Camara, I., Ndao Eteno, M., Mialoundama, C., Guarisco, M., Ondimba, K. and Ndouongo, P.K. (2025) Peripheral Neuropathies Associated with Gammopathies in the Outpatient Clinic of the Neurology Department at the University Hospital Center of Libreville: Study of Two Cases and Literature Review. *Neuroscience and Medicine*, **16**, 261-271.
https://doi.org/10.4236/nm.2025.164025

Received: November 9, 2025
Accepted: December 23, 2025
Published: December 26, 2025

Abstract

Introduction: Peripheral neuropathies associated with gammopathies are common after the age of 55 years. However, serum protein electrophoresis is not systematically performed in the etiological workup of neuropathy. **Objective:** to describe cases the clinical profiles of patients with peripheral neuropathy and co-occuring gammopathy and highlight associated diagnostic challenges in our clinical setting. **Methodology:** This retrospective and prospective descriptive study, conducted in the neurology department of CHUL, reported observations of two female patients, aged 73 and 62 years, respectively, who presented in consultation with clinical signs suggestive of peripheral neuropathy, subsequently confirmed by electroneuromyography, and whose investigations revealed gammopathy. **Results and Conclusion:** Peripheral neuropathies associated with gammopathies should be systematically investigated, as they are not uncommon. They primarily affect elderly women, who often present with bilateral carpal tunnel syndrome, with or without an associated neuropathy. Etiological evaluation, in our context, is often limited once additional diagnostic tests are pursued. However, in the presence of certain gammopathies, it is essential to rule out malignant hematological disorders.

Keywords

Peripheral Neuropathy-Gammopathy-Libreville

1. Introduction

Peripheral neuropathies are relatively common conditions in Gabon, with diverse etiologies. A population-based study in two municipalities reported a neuropathy prevalence of 2.8% [1]. Regarding the prevalence of monoclonal gammopathies, it increases with age: 1% at 50 years, 3% beyond 70 years, and 10% beyond 80 years [2]. Furthermore, it is not uncommon to detect a gammopathy upon reviewing the results of serum protein electrophoresis profiles. We therefore investigated the association between this gammopathy and peripheral neuropathy, the clinical and electrophysiological profiles of patients presenting with associated peripheral neuropathy, and the frequency of electrophoresis testing in the department, with the aim of refining the diagnostic approach, etiological evaluation, and therapeutic prospects. We also wanted to highlight the diagnostic difficulties in these neuropathies in which only gammopathy is found.

2. Patients and Methods

We reported several cases of peripheral neuropathies observed in outpatient consultations at the neurology department of CHUL from June 2024 to June 2025. Only patients who presented with peripheral neuropathy, had undergone electroneuromyography, and exhibited an electrophoretic profile indicative of monoclonal gammopathy were included.

The records were collected based on the ENMG registry of the functional explorations in the Neurology Department.

All patients presenting with a clinical picture suggestive of an alteration in one or more peripheral nerves and a DN4 score of at least 4 were considered to have peripheral neuropathy.

All neuropathies were included, notably both focal and diffuse neuropathies. We recorded whether or not an electrophoretic analysis had been performed in patients seen in the outpatient neurology clinic at CHUL. Gammopathy was diagnosed based on the elevation of the gamma fraction in serum, even if the subunits had not been reported.

In total, 266 patients presenting with peripheral neuropathy confirmed by electromyography (ENMG) were identified; only 8 of them had undergone serum protein electrophoresis, and two female patients exhibited monoclonal gammopathy.

For the review, the terms "peripheral neuropathy" and "gammopathy" were used in the search engine Google Scholar with a selection criterion (last 15 years, their presence in the title).

3. Observations

3.1. Observation 1

We report the case of a 73-year-old woman with occasional alcohol consumption, no history of diabetes, but under follow-up for arterial hypertension.

The patient presented with paresthesias characterized by cramps, accompanied by a sensation of "mud" felt over the entire body, and swelling of the feet, all evolving over 3 years and initially starting in the hands. Furthermore, she complained of right knee pain requiring the use of elbow crutches.

Upon examination, atrophy of the thenar eminences was noted, predominantly on the right. Segmental muscle strength was 4/5 in the right first dorsal interosseous muscle; muscle strength was normal elsewhere. Sensory examination revealed hypoesthesia to protopathic touch on the lateral aspect of the right forearm. In the lower limbs, bilateral edema was present, predominantly associated with varus deformity of the knees. The Hoffmann sign and Babinski sign were absent. Deep tendon reflexes were present in the upper limbs and difficult to assess in the lower limbs due to pain.

Paraclinical evaluation revealed normal complete blood count, blood glucose level, transaminase and gamma-GT levels, serum electrolyte panel, calcium level, and renal function tests. HIV retroviral serology and hepatitis B and C serologies were negative.

The electrophoretic profile was consistent with a monoclonal gammopathy at 37.83 g/L. Quantitative assay of immunoglobulins revealed IgA at 2.09 g/L and IgM at 0.48 g/L, both normal, and elevated IgG at 29.2 g/L. Bence Jones proteinuria was negative, with a level below 0.04 g/L.

Electroneuromyography revealed axonal sensory-motor involvement of the peroneal nerves, tibial nerves, and median nerves. These findings were suggestive of a length-dependent axonal sensory-motor polyneuropathy associated with severe carpal tunnel involvement of the median nerves at the wrists (see **Figure 1**).

Motor Nerve Conduction Studies								
MNCS								
Nerve	Lat		Amp		CV		F-M Lat	
	ms	Ref.Dev	mV	Ref.Dev	m/s	Ref.Dev	ms	Ref.Dev
Medianus Moteur Gauche								
Wrist - APB	20.0		--					
Medianus Moteur Droit								
Wrist - APB	19.4		--					
Peroneus Moteur Gauche								
Ab. knee-Fib. head	4.70		**1.26**		64.5			
Peroneus Moteur Droit								
Ab. knee-Fib. head	5.60		**1.62**		41.9			
Tibialis Moteur Gauche								
Ankle - Abd hal	--		--					
Tibialis Moteur Droit								
Ankle - Abd hal	--		--					
Ulnaris Moteur Gauche								
Wrist - ADM	2.56		10.2				25.5	
Ab. elbow-Wrist	7.79		8.8		52.6			
Ulnaris Moteur Droit								
Wrist - ADM	2.51		10.1				30.8	
Ab. elbow-Wrist	10.2		8.3		42.3			

Sensory Nerve Conduction Studies						
SNCS						
Nerve	Peak Lat		Amp		CV	
	ms	Ref.Dev	uV	Ref.Dev	m/s	Ref.Dev
Medianus Sensitif Gauche						
Dig II - Wrist	--		--			
Dig III - Wrist	--		--			
Palm - Wrist	--		--			
Medianus Sensitif Droit						
Dig II - Wrist	--		--			
Dig III - Wrist	--		--			
Palm - Wrist	--		--			
Radialis Sensitif Gauche						
EPL tendon - Wrist	1.96		27.3		57.7	
Radialis Sensitif Droit						
EPL tendon - Wrist	2.19		23.1		51.3	
Suralis Sensitif Gauche						
Mid. lower leg - Lat. Malleolus	--		--			
Suralis Sensitif Droit						
Mid. lower leg - Lat. Malleolus	--		--			
Ulnaris Sensitif Gauche						
Dig IV - Wrist	3.28		10.8		56.8	
Ulnaris Sensitif Droit						
Dig IV - Wrist	--		--			
Dig V - Wrist	3.34		10.0		46.3	
Peroneus superfic Sensitif Droit						
Calf - Med. Dor. Cutan.	8.56		**5.3**		--	

Figure 1. EMG patient 1.

Furthermore, radiography of the dorso-lumbar spine and pelvis revealed findings consistent with degenerative disc disease. Radiography of the limbs and skull showed, respectively, advanced gonarthrosis with inflammatory edema of the soft tissues. A hematology consultation was recommended. The diagnosis of benign monoclonal gammopathy was confirmed based on serum protein levels below 30 g/L, a clonal plasma cell percentage below 10% on bone marrow biopsy analysis, and the absence of CRAB criteria.

Therapeutically, the patient had primarily benefited from the treatment of neuropathic pain.

The patient has been lost to follow-up for 5 months.

3.2. Observation 2

The second patient was a 62-year-old woman with hypertension since 2007, treated with Exforge HCT (Amlodipine/Valsartan/Hydrochlorothiazide), who presented to us on June 19, 2025, with complaints of paresthesias manifesting as cramps affecting the feet and hands. She also described burning sensations in the feet and toes and reported occasionally losing her shoes while walking. Motor examination revealed no amyotrophy, and segmental muscle strength was preserved; sensory

examination, however, demonstrated hypo-pallesthesia extending to the level of the anterior tibial tuberosity but predominant distally and on the right.

An ENMG of the four limbs had been recommended and revealed sensory axonal involvement of the median and ulnar nerves. Severe sensory-motor axonal involvement was observed in the lower limbs and distally. The ENMG findings were compatible with a severe length-dependent sensory-motor polyneuropathy in the lower limbs (see Figure 2).

The laboratory workup revealed a normal complete blood count, as well as normal fasting blood glucose and glycated hemoglobin levels. The vitamin B12 level was normal at 324 pmol/ml. Serologies for hepatitis B and C and for retroviral infections were negative.

Serum protein electrophoresis revealed a moderate polyclonal increase in the gammaglobulin fraction, with the presence of a monoclonal-appearing peak at 5.5 g/L. The profile suggests an early gammopathy.

The IF workup was completed with normal quantitative IgM and IgA levels, and slightly elevated IgG at 19.3 g/L (normal range 7 - 16). The patient was reviewed, and we recommended a hematology consultation (See Figure 3).

Motor Nerve Conduction Studies								
MNCS								
Nerve	Lat		Amp		CV		F-M Lat	
	ms	Ref.Dev	mV	Ref.Dev	m/s	Ref.Dev	Ms	Ref.Dev
Medianus Moteur Gauche								
Wrist - APB	4.06		9.4				27.0	
Elbow-Wrist	8.33		8.4		54.3			
Medianus Moteur Droit								
Wrist - APB	3.13		10.6				24.9	
Elbow-Wrist	7.67		9.2		55.1			
Peroneus Moteur Gauche								
Ankle - EDB	6.25		**0.32**				40.0	
Fib. head-Ankle	3.28		**1.79**		--			
Ab. knee-Fib. head	4.98		**2.6**		42.4			
Peroneus Moteur Droit								
Ankle - EDB	18.3		**0.015**				42.9	
Fib. head-Ankle	2.58		**2.3**		--			
Ab. knee-Fib. head	4.46		**3.3**		48.4			
Tibialis Moteur Gauche								
Ankle - Abd hal	--		--					
Tibialis Moteur Droit								
Ankle - Abd hal	19.1		--					
Ulnaris Moteur Gauche								
Wrist - ADM	2.27		8.3				26.6	
Ab. elbow-Wrist	7.90		6.9		51.5			
Ulnaris Moteur Droit								
Wrist - ADM	2.14		7.9				26.5	
Ab. elbow-Wrist	7.65		7.5		58.1			

Sensory Nerve Conduction Studies						
SNCS						
Nerve	Peak Lat		Amp		CV	
	ms	Ref.Dev	uV	Ref.Dev	m/s	Ref.Dev
Medianus Sensitif Gauche						
Dig II - Wrist	4.44		**6.1**		41.3	
Dig III - Wrist	4.33		**14.1**		44.0	
Palm - Wrist	4.23		**0**		--	
Medianus Sensitif Droit						
Dig II - Wrist	4.13		**4.8**		36.3	
Dig III - Wrist	--		--			
Palm - Wrist	--		--			
Radialis Sensitif Gauche						
EPL tendon - Wrist	1.96		23.0		73.5	
Radialis Sensitif Droit						
EPL tendon - Wrist	2.00		34.2		61.1	
Suralis Sensitif Gauche						
Mid. lower leg - Lat. Malleolus	5.76		**0**		--	
Suralis Sensitif Droit						
Mid. lower leg - Lat. Malleolus	3.93		9.3		42.7	
Ulnaris Sensitif Gauche						
Dig IV – Wrist MED	--		--			
Dig iV - Wrist	3.73		**3.2**		52.1	
Ulnaris Sensitif Droit						
Dig IV – Wrist mED	--		--			
Dig IV - Wrist	--		--			
Peroneus superfic Sensitif Gauche						
Calf - Med. Dor. Cutan.	3.06		**−0.77**		34.8	
Peroneus superfic Sensitif Droit						
Calf - Med. Dor. Cutan.	3.62		**4.7**		43.7	

Figure 2. EMG patient 2.

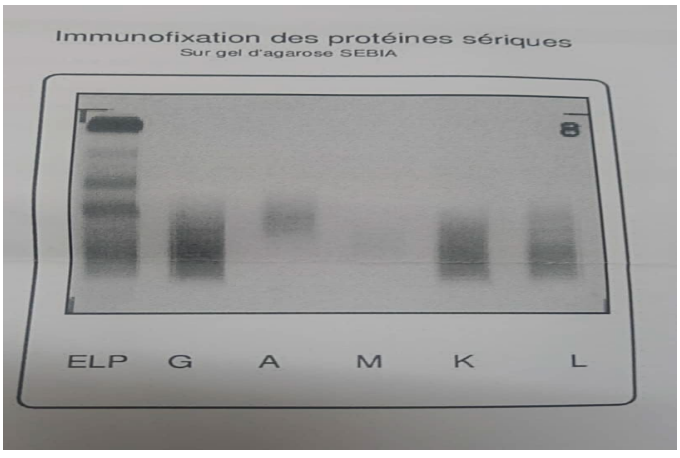


Figure 3. Immunofluorescence of serum proteins.

4. Discussion

Peripheral neuropathies are common conditions in clinical practice but pose chal-

lenges for etiological diagnosis. Their prevalence in the population ranges from 2.4% to 8%, depending on age [3]. In the classification, several types are described, including focal forms and circumscribed forms. The latter can be divided into length-dependent forms, which are the most common (polyneuropathies), and non-length-dependent forms, including polyradiculoneuropathies, neuronopathies, and multiple mononeuropathies. Indeed, numerous possible causes are observed, the most frequent of which are diabetes, alcohol-related and nutritional deficiencies, renal insufficiency, and hereditary causes [4]. In our observations, carpal tunnel involvement was noted in most of our female patients and was associated with polyneuropathy.

Gammopathies are more frequent after the age of 50 years [5]. In our small series, the patients had a mean age of 67.5 years. In the study by Nouha Hamza *et al.*, conducted over 17 years, 7 cases of neuropathy associated with monoclonal gammopathy were reported, with a mean age of 60.1 years [6].

Furthermore, the diagnosis of monoclonal gammopathy is possible only through protein electrophoresis with immunofixation, sometimes supplemented by Bence Jones proteinuria. A practical approach is to first investigate cryoglobulinemia when neuropathy is associated with MGUS. Additionally, the clinical presentation and, in some cases, the measurement of specific antibodies (anti-MAG, anti-ganglioside) will guide the identification of associated neuropathy (anti-MAG, CANOMAD, POEMS). The purpose of the workup is also to rule out or confirm other causes, particularly toxic, infectious, metabolic, inflammatory, or paraneoplastic etiologies, especially when a non-IgM immunoglobulin or MGUS is identified [7].

Gammopathy can be observed in the context of monoclonal gammopathy of undetermined significance (MGUS), which requires only clinical and biological monitoring; however, the persistent fear of a malignant hematological disorder, such as multiple myeloma or Waldenström's macroglobulinemia, remains.

In our observations, one patient was able to undergo immunofixation, while the other patient could not, likely due to insufficient financial resources.

Gammopathies can be present in all types of peripheral neuropathies.

Indeed, the concept of monoclonal gammopathy of clinical significance (MGCS) has recently emerged. It encompasses secondary clinical manifestations in the context of asymptomatic monoclonal gammopathy, affecting multiple organs including the peripheral nerves, without meeting CRAB or SLiM criteria [5] [8].

Table 1 and **Table 2** show respectively diagnostic criteria for multiple myeloma and definition of clinical forms.

The MGCS thus represents a broad spectrum, all the more so given the diversity of the underlying pathophysiological mechanisms. In peripheral neuropathy, this may involve an immunological reaction characterized by the presence of antibodies directed against an epitope of the peripheral nerve, cryoglobulinemia, deposition of amyloid substances, or endoneuronal deposits of light chains. The link between gammopathy and neuropathy is not always evident, except in the context of anti-MAG neuropathy, where it is well established that the antibodies exhibit a

tropism for myelin. Moreover, the prevalence of MGUS associated with chronic axonal polyneuropathies is significantly higher than in the general population (10% versus 3% to 5%).

Table 1. IMWG 2014 recommendations: diagnostic criteria for multiple myeloma [Rajkumar].

Organ impairment attributable to monoclonal plasma cell proliferation: 1 CRAB criterion
The symptomatic nature of multiple myeloma, upon which the indication for treatment depends, is based on the presence of clinical symptoms or organ damage defined by at least one of the following abnormalities (CRAB criteria):
C: hypercalcemia > 2.75 mmol/L
R: Renal insufficiency attributable to myeloma with creatinine clearance < 40 ml/min or serum creatinine > 177 µmol/L
A: Anemia < 10 g/dL (normochromic, normocytic, and non-regenerative anemia) attributable to myeloma
B: bone involvement (*Bone*): lytic lesion visible on whole-body low-energy CT scan "*low dose*", or PET-CT
One or more of the following malignancy biomarkers: SLiM CRAB criteria
Bone marrow plasmocytosis ≥ 60%
Serum free light chain ratio (involved/uninvolved) ≥ 100
>1 focal lesion on MRI greater than 5 mm

Table 2. Definition of clinical forms (HAS, 2010; IMWG, 2014) [Rajkumar].

MGUS	Monoclonal immunoglobulin detected but <30 g/L if IgG and bone marrow plasmacytosis <10%	No symptoms (CRAB criteria)
Asymptomatic or indolent multiple myeloma	Detected monoclonal immunoglobulin > 30 g/L and/or bone marrow plasmacytosis ≥10%	No symptoms (neither CRAB criteria nor malignancy criteria)
Symptomatic multiple Myeloma	Monoclonal immunoglobulin detected in the serum and/or urine and/or bone marrow plasmacytosis ≥ 10%	Symptoms: - at least 1 CRAB criterion - and/or 1 SLiM CRAB criterion

All of our patients exhibited axonal involvement of the nerves of interest (median nerves in our case), which is consistent with the literature data [6].

The majority of monoclonal gammopathies are asymptomatic (MGUS, smoldering myeloma), but given the risk of progression to a malignant hematological disorder (1% per year), screening for gammopathy should be systematic in the presence of any type of neuropathy [5].

As comorbidities, we noted hypertension in one of two patients another. In patients with peripheral neuropathy associated with gammopathies, a study revealed that diabetes, renal insufficiency, connective tissue diseases, multiple neoplasms, and polypharmacy are more common comorbidities [7].

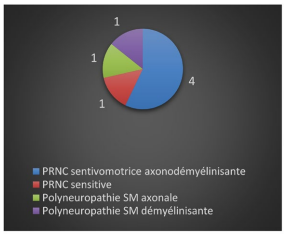
Etiologically, a French multicenter retrospective study identified a cause in 61% of patients with "neurogammopathic" IgM. Dysimmune neuropathies predominated (chronic polyradiculoneuropathy [16%], CANOMAD syndrome [14%], multifocal motor neuropathy [12%], distal form of CIDP [9%], cryoglobulinemic vasculitis [3%]). Among patients without an identified etiology, an evolving ax-

onal polyneuropathy was found in 53% of them. This polyneuropathy was associated with a higher proportion of lymphomas [9]. In another survey, the causes were represented by benign IgG gammopathy (2/7), myeloma (1/7), Waldenström disease (1/7), cryoglobulinemia (1/7), and amyloidosis (1/7) [6].

Among patients seen in our clinic who suffered from neuropathy and in whom gammopathy had been observed, the complementary assessment including immunoglobulin typing was not always performed.

Indeed, patients are frequently lost to follow-up after receiving the results of the initial assessment, which may be attributable to limited financial resources as well as the pursuit of alternative therapeutic practices (alternative and complementary medicine) in response to persistent symptoms. Another possible reason is the progression of the pathology or the development of a disability that prevents or restricts travel to healthcare facilities. Consequently, obtaining an etiological diagnosis for these neuropathies in our setting remains exceptional, although it is well established that, in general, one-third of peripheral neuropathies lack an identified etiology despite comprehensive evaluation [10]. **Table 3** compares our study to the literature.

Table 3. Comparison of our study with other works from the literature.

	Our study	Perlot Q, 2023	Sellier M L, 2025	Hamza N, 2018
Type of study	**Study on 2 cases**	**Study on 2 cases**	**Multicenter cohort**	**Study on 7 cases**
Mean age	67.5 years	80 years		60.1 years
Comorbidity	Hypertension (1/2)	Ischemic heart disease; hypertension, obstructive sleep apnea syndrome,		
Gammopathy	Monoclonal IgG gammopathy (2)	IgM gammopathy without anti-MAG		
ENMG	-A length-dependent axonal sensory-motor polyneuropathy associated with severe carpal tunnel involvement of the median nerves at the wrists (Patient 1). -A severe length-dependent sensory-motor polyneuropathy in the lower limbs. Sensory axonal involvement of the median and ulnar nerves (Patient 2)		PIDC (16%) CANOMAD (14%) multifocal motor neuropathy (12%) distal form PIDC (9%)	
Etiologies	Benign monoclonal gammopathy (1/2)	-Acquired von Willebrand syndrome; -Acquired angioedema	-Vasculitis cryoglobulinemia -Lymphocytic lymphoma	-Benign monoclonal gammopathy IgG (3) -Myeloma (1) -IgM monoclonal gammopathy (1) -Von willebrand disease (1) -Type I cryoglobulinemia (1) -Amyloidosis (1)
Evolution	Lost sight of (1/2)	Not mentioned		Lost Sight of (3/7) Partial improvement (1/7) Myeloma (1/7) Aggravation (3/7)

5. Conclusion

Monoclonal gammopathy in the context of neuropathies, as reported in the literature, is a hallmark of elderly patients. Axonal involvement predominates, and focal neuropathies may also be observed; however, these can sometimes reveal a more diffuse neuropathy. Furthermore, the loss to follow-up of many patients after initial investigations limits etiological diagnosis in our setting. Therefore, more systematic implementation of serum protein electrophoresis with immunotyping could provide substantial diagnostic support, particularly for etiological assessment, and thereby improve patient management.

Contributions

All authors contributed equally to this work. Diouf Mbourou Nelly and Dr. Nsounda drafted the manuscript. Dr. Mboumba, Dr Gnigone, Dr Nyangui, Dr Mambila, Dr Saphou Damon, Dr Camara, Dr Ndao Eteno, Dr Mialoundama, Dr Guarisco, Dr Ondimba conducted the literature review and revised the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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