Award - Winning Extended Abstracts

246 | Leptospirosis Presenting as Acute Motor and Sensory Axonal Neuropathy (AMSAN): A Rare Form of Neuroleptospirosis

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Case Report

A 42 - year - old previously healthy woman developed symptoms in September 2024, beginning with severe calf pain (left > right), arthralgias, and erythematous palmar–plantar lesions. In the following days, she evolved with dark urine and jaundice, consistent with Weil's syndrome.

Concurrently, she experienced progressive lower - limb weakness, distal sensory loss, and neuropathic pain characterized by burning and electric shock sensations in the calves and dorsum of the feet. Within three weeks, neurological involvement progressed to the upper limbs, with a motor nadir around day 21.

In October 2024, at another healthcare facility, leptospirosis was suspected and subsequently confirmed through positive anti-Leptospira IgM serology (22.10 U/mL). She started amoxicillin, which led to improvement of systemic features (cutaneous and musculoskeletal symptoms), but neuropathic pain, sensory deficits, and weakness persisted. She was started on pregabalin in late October, with partial symptom control. Between November and December 2024, she showed slow but progressive motor recovery, though significant functional impairment remained.

The patient was first evaluated at our neuromuscular clinic (HUCFF/UFRJ) only in July 2025, nearly ten months after symptom onset, already with a prolonged history of neuropathic pain, partial response to pregabalin, and residual weakness.

At her initial neurological examination at HUCFF, she presented with:

- Steppage gait, worse on the left;
- generalized areflexia;
- distal hypoesthesia in both lower limbs, more pronounced on the left;
- predominantly distal weakness in the lower limbs (approximately MRC 2/5 on the left and 3–4/5 on the right), with proximal strength around 4/5;
- upper-limb strength largely preserved, except for mild distal involvement.

An electroneuromyography (ENMG) performed in March 2025 at another institution showed:

- markedly reduced CMAP amplitudes in bilateral fibular (worse on the left) and tibial nerves;
- conduction velocities reduced but not within demyelinating range (left fibular nerve at the lower limit);
- absent SNAPs bilaterally in superficial fibular nerves;
- findings consistent with a motor–sensory axonal polyneuropathy, compatible with Acute Motor and Sensory Axonal Neuropathy (AMSAN).

During follow-up at HUCFF, she maintained regular motor physiotherapy. Because of difficulties in obtaining pregabalin, medication was switched to amitriptyline in July 2025, with excellent symptomatic improvement. By August 2025, she demonstrated significant motor recovery, complete resolution of neuropathic pain, independent ambulation, and mild residual proximal weakness in the lower limbs (left > right). Generalized areflexia persisted, though without functional impact. She successfully returned to work and reported high satisfaction with her recovery.

DISCUSSION

Leptospirosis presents in three classical forms: anicteric (~90%), icteric/Weil's disease (5–10%), and atypical forms (<5%). Neurological manifestations can occur in any form and represent 5–15% of symptomatic cases, including aseptic meningitis, encephalitis, mononeuritis multiplex, and, more rarely, Guillain–Barré syndrome (GBS). Among GBS variants, AMSAN is exceedingly rare and accounts for <5% of neurological presentations of leptospirosis. This case is notable because the patient arrived at our service only in the chronic–subacute phase, months after the initial infection, limiting early diagnostic and therapeutic intervention. Her ENMG findings clearly reflected an axonal process rather than demyelination, consistent with AMSAN.

The pathophysiology of leptospirosis-associated neuropathy is believed to involve molecular mimicry. Leptospira membrane components, particularly lipeoligosaccharides (LOS), can trigger anti-ganglioside antibodies (anti-GM1, anti-GD1a, anti-GD1b), analogous to those seen in axonal GBS variants following Campylobacter jejuni. These antibodies target axolemmal gangliosides and nodes of Ranvier, activating complement and leading to axonal degeneration.

The patient's electrophysiological pattern aligns with this mechanism.

Published cases by Panicker (2001)[1], Mathew (2006)[2], Dian (2023)[3], Akdoğan (2022)[4] and Soni (2024)[5] highlight AMSAN as a possible yet exceptionally uncommon complication of leptospirosis. Recovery in AMSAN tends to be slower than in AIDP, but meaningful improvement is achievable, particularly with prolonged rehabilitation, as demonstrated here Early, intense neuropathic pain, concomitant with sensory loss, is also typical of axonal neuropathies and was a prominent feature in this case. The patient's favorable response to amitriptyline aligns with established management strategies for neuropathic pain in peripheral axonopathies.

FINAL REMARKS

This case represents a rare neurological complication of leptospirosis, manifested as Acute Motor and Sensory Axonal Neuropathy (AMSAN). The patient's course underscores the need for early recognition of neuroleptospirosis and rapid initiation of multidisciplinary care, including adequate antimicrobial therapy, neuropathic pain management, and sustained physiotherapy.

Although the patient presented to our center only in a late phase of the disease, appropriate management resulted in a favorable functional outcome, with minimal residual deficits and full social reintegration.

Clinicians in endemic regions should maintain a high index of suspicion for GBS-spectrum disorders following leptospirosis, especially when confronted with progressive weakness, sensory deficits, and generalized areflexia.

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657 | Population-Based Prevalence of Neuromyelitis Optica in Rio de Janeiro Using Advanced Statistical Methods

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Introduction

Acute neuromyelitis optica (NMO), originally described by Eugène Devic in 1894, is an autoimmune inflammatory disorder of the central nervous system characterized predominantly by severe and acute episodes of optic neuritis and transverse myelitis. Long considered a severe monophasic variant of multiple sclerosis, NMO was redefined as a distinct entity after the recognition of its relapsing form (Wingerchuk et al., 1999) and the discovery of the anti–aquaporin-4 autoantibody (AQP4-IgG) by Lennon et al. (2004), which demonstrated its primarily astroglial pathology. This breakthrough led to a conceptual reformulation of the disease and culminated in the 2015 international consensus diagnostic criteria, which formally replaced the term NMO with neuromyelitis optica spectrum disorders (NMOSD), encompassing both AQP4-IgG–positive and seronegative presentations (Wingerchuk et al., 2015).

In South America, the first well-characterized cases were reported in Brazil and predominantly involved Afro-descendant women presenting with recurrent and severe episodes of optic neuritis and paraplegia—features that differed from patterns observed in European and Asian populations (Papais Alvarenga et al., 2002). These observations were subsequently reinforced by a multicenter Latin American study demonstrating that NMOSD is more frequent in regions with higher proportions of African ancestry (Papais Alvarenga et al., 2015).

Despite the historical relevance of Brazil in the clinical characterization of NMOSD, the prevalence of this condition in the state of Rio de Janeiro—where the disease was first described—remains unknown. The Volta Redonda – Resende Intermediate Geographic Region, comprising 17 municipalities with high admixture and a substantial Afro-descendant population, offers favorable conditions for epidemiological research due to its extensive neurological network and specialized clinical services. Addressing this gap, the present study aimed to estimate regional prevalence using complementary statistical approaches—direct prevalence, capture–recapture analysis, and Bayesian modeling—and to contextualize the findings through comparison with global prevalence data.

OBJECTIVES

To estimate the point prevalence of NMOSD as of December 31, 2022, in the 17 municipalities of the Volta Redonda–Resende Intermediate Geographic Region.

To apply capture – recapture estimators and Bayesian modeling to correct for potential underreporting.

To compare regional prevalence estimates with global data through systematic review and meta-analysis.

To discuss ethnic and historical determinants associated with the global distribution of NMOSD.

METHODS

The study was conducted across the 17 municipalities of the Volta Redonda–Resende Intermediate Geographic Region, with a combined population of 985,051 inhabitants (IBGE, 2022). Eligible participants included individuals diagnosed with NMOSD according to the 2015 international criteria, residing in the region for at least one year, and alive on December 31, 2022.

Active case ascertainment was performed between 2023 and 2025 using multiple independent data sources: structured interviews with 61 neurologists practicing in the region; the local NMOSD patient association; records from the Reference Center for Demyelinating Diseases at the Federal Hospital of Lagoa; and verification through specialized public pharmacies responsible for dispensing high-cost immunosuppressive therapies. Duplicate cases were manually reconciled.

Direct prevalence was calculated as the number of confirmed cases divided by the resident population of municipalities in which at least one case was identified. To address underreporting, four classical capture–recapture estimators were applied (Lincoln–Petersen, Chapman, Bailey, and Chao). Bayesian modeling was performed using Markov chain Monte Carlo (MCMC) simulations with 50,000 iterations and a burn-in of 10,000, with convergence assessed using the Gelman–Rubin diagnostic.

A systematic review (PROSPERO) was conducted to identify population-based NMOSD prevalence studies published between 2007 and 2025. A total of 44 eligible studies were included and analyzed using a random-effects meta-analytic model, grouped by continent. The protocol was prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO; CRD42022359519).

RESULT

Twenty-four NMOSD cases were identified across nine of the 17 municipalities included in the study. The direct prevalence estimate was 3.07 per 100,000 inhabitants, with a marked predominance of women (87.5%) and Afro-descendant individuals (62.5%).

Capture–recapture estimators yielded prevalence values ranging from 3.5 to 3.9 per 100,000. Bayesian modeling produced an adjusted prevalence estimate of 3.87 per 100,000 (95% CrI: 2.9–5.0), representing an approximate 25% increase compared with the direct estimate.

The global meta - analysis demonstrated a worldwide mean prevalence of 2.02 per 100,000. The Americas exhibited the highest prevalence (4.18 per 100,000), followed by Asia (3.07), Europe (1.32), and Oceania (0.37). The Volta Redonda–Resende region therefore falls within one of the highest global prevalence ranges. The systematic review identified only one prior prevalence study from Brazil, conducted in Goiânia, which estimated a prevalence of 0.7 per 100,000.

DISCUSSION

Although NMOSD is a rare disease globally, the findings indicate a comparatively high prevalence in the Volta Redonda–Resende region. This pattern is consistent with the region's demographic profile, characterized by substantial African ancestry, and aligns with epidemiological data from Martinique, French Guiana, Colombia, and the United States, where populations of African descent exhibit increased susceptibility to NMOSD.

The concordance among direct prevalence, capture–recapture estimators, and Bayesian modeling supports the methodological rigor of the study. The use of multiple independent data sources strengthened case ascertainment sensitivity and mitigated the risk of underestimation—an important consideration in the epidemiology of rare diseases.

The absence of NMOSD-specific epidemiological systems within municipal health departments and the lack of available treatments for this severe central nervous system disorder within the public health system underscore the public health relevance of these findings. This study provides an evidence-based foundation for expanding access to serological testing, specialized care, and immunological therapies.

CONCLUSION

This study provides the first population-based estimate of NMOSD prevalence in the state of Rio de Janeiro and demonstrates elevated prevalence in the Volta Redonda–Resende Intermediate Geographic Region—four times higher than the only previously reported Brazilian estimate from Goiânia. The findings align with the epidemiological pattern observed in the Americas, which exhibit the highest global prevalence rates, and reinforce the role of African ancestry in NMOSD susceptibility. The combined use of direct prevalence, capture–recapture methods, and Bayesian modeling ensures robust estimations and offers a methodological framework applicable to future rare disease research in Brazil.

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536 | Technological advances and application of rapid and automated tests using the multiplex PCR methodology to aid in the diagnosis of nosocomial meningitis in the state of Rio de Janeiro.

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Objectives

Infectious meningitis/encephalitis (ME) are pathological conditions associated with high morbidity and mortality rates, for which cerebrospinal fluid (CSF) examination is essential for diagnosis. However, conventional CSF tests are not rapid (cultures) and often have low sensitivity. These limitations are being overcome through the development of molecular tests for the simultaneous detection of infectious agents. To evaluate the clinical utility of the HS12 and Filmarray platforms for the simultaneous detection of nosocomial pathogens in CSF.

METHODS

Sample selection was performed consecutively from 2019 to 2025. CSF samples were collected and sent to the Neurolife Laboratory immediately after lumbar puncture for diagnostic investigation. CSF was analyzed using conventional and molecular tests. Panel screening was performed based on clinical indications, epidemiology, and patient history.

Results 1,241 CSF samples were analyzed. The Xgen SEPSIS Panel (HS12) detected 15 different pathogens in 117 CSF samples. The BCID Panel (Filmarray), specific for nosocomial infectious agents, detected 14 different pathogens in 51 CSF samples. Furthermore, these panels also evaluated resistance genes (RG), with 23 samples identified with RG (figure 1), the most common being mecA and KPC. The pathogens and RG detected through the panels showed good agreement with the patients clinical suspicion and other findings in the CSF examination, especially culture and antimicrobial susceptibility testing.

CONCLUSION

The use of rapid multiplex PCR tests in CSF allowed the rapid and accurate diagnosis of the etiological agent in nosocomial meningitis, as well as predicting phenotypic resistance through the detection of RG, even before bacterial growth, possibly contributing to the early initiation of appropriate treatment and de-escalation of antibiotics. The correlation between the multiplex PCR panels and culture (gold standard) was practically 100% in all positive cases.

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n	idade	sexo	resultado patógenos (PCR)	resultado genes resistência (PCR)	plataforma	L	H	ptn	gli	lac	Bacterioscopia (+/-) GRAM	cultura (+/-)
1	4	F	Staphylococcus spp. ; Staphylococcus aureus	mecA	HS12 auto	1.024	169	121	20	58	Presença de cocos e diplococos gram positivos intra e extracelulares	Staphylococcus aureus
2	53	F	Candida albicans; Klebsiella pneumoniae	Carbapenemase KPC	HS12 auto	5.688	1.280	128	20	58	-	Klebsiella pneumoniae ssp pneumoniae
3	47	M	Staphylococcus spp.	mecA	HS12 auto	5.461	3	131	20	66	-	-
4	40	F	Klebsiella pneumoniae	Carbapenemase KPC, Extended-spectrum B-lactamase CTX-M	HS12 auto	181	4.180	125	35	44	-	-
5	54	M	Enterobacteriaceae; Klebsiella pneumoniae	Carbapenemase KPC, Extended-spectrum B-lactamase CTX-M	HS12 auto	39	16	50	106	N.A.	-	-
6	33	M	Enterobacteriaceae; Klebsiella pneumoniae	Carbapenemase KPC	HS12 auto	485	133	153	20	121	Presença de cocobacilos gram negativos intra e extracelulares	Klebsiella pneumoniae
7	19	F	Enterobacteriaceae	Extended-spectrum B-lactamase CTX-M	HS12 auto	32	2	105	20	13	Presença de cocobacilos gram negativos	Enterobacter cloacae complex
8	19	F	Enterobacteriaceae	Extended-spectrum B-lactamase CTX-M	HS12 auto	52	1	48	44	19	Presença de cocobacilos gram negativos	Enterobacter cloacae
9	66	F	Streptococcus pneumoniae; Staphylococcus spp. ; Staphylococcus aureus	mecA	HS12 auto	2.560	2.986	150	20	103	Presença de cocos e diplococos gram positivos	Staphylococcus aureus
10	2	F	Acinectobacter baumannii	Carbapenemase OXA23_like; OXA51_like	HS12 auto	37	1.024	72	22	15	Presença de cocos e diplococos gram negativo intra e extracelular	Acinectobacter baumannii
11	57	M	Staphylococcus aueus	mecA	HS12 auto	37	1.946	127	41	32	-	Staphylococcus aureus
12	51	M	Enterobacteriaceae, Klebsiella pneumoniae	Extended-spectrum B-lactamase SHV, Extended-spectrum B-lactamase CTX-M	HS12 auto	199	213	160	57	43	-	Staphylococcus epidermidis/Klebsiella pneumoniae
13	61	F	-	mecA	HS12 auto	27	61.440	129	104	76	-	Staphylococcus haemolyticus
14	36	F	Enterobacteriaceae, Klebsiella pneumoniae	Carbapenemase KPC, Extended-spectrum B-lactamase SHV	HS12 auto	8	3.669	116	33	33	Presença de bastonetes gram negativo	-
15	79	M	Staphylococcus spp.	mecA	HS12 auto	124	6	51	67	24	-	-
16	86	M	-	Carbapenemase GES	HS12 auto	192	12.800	124	26	40	-	-
17	8	M	-	Carbapenemase KPC	HS12 auto	773	1.363	148	20	72	-	Pseudomonas putida
18	79	M	Enterobacteriaceae, Klebsiella pneumoniae	Carbapenemase KPC, Extended-spectrum B-lactamase SHV	HS12 auto	2.645	19.370	137	20	88	-	Klebsiella pneumoniae
19	40	M	Staphylococcus spp., Staphylococcus aureus	mecA	HS12 auto	6.826	50	106	20	71	-	Staphylococcus aureus
20	39	F	Streptococcus pneumoniae, Streptococcus spp.	Carbapenemase GES	HS12 auto	3.115	208	132	36	52	-	-
21	3	M	Stenotrophomonas maltophilia, Acinetobacter baumannii, Enterobacteriaceae	Carbapenemase KPC, GES, NDM, OXA51_like	HS12 auto	4	2	55	5	13	Presença de bacilo gram negativo intra e extracelular	Citrobacter braakii
22	67	F	Enterobacteriaceae, Klebisiela pneumoniae	Carbapenemase KPC, NDM, Extended-spectrum B-lactamase SHV, CTX-M	HS12 auto	11520	1280	177	7	143	-	Klebsiella pneumoniae
23	65	M	Enterobacterales	IMP, KPC, NDM	filmarray BCID	411	584	71	51	70	bacilos gram -	Providencia rettgeri

Figure 1. Positive cases with resistance genes detected. The table shows a comparison of the results of resistance genes detected by molecular platforms and the results obtained by direct bacterioscopy and culture growth and identification by antibiogram (TSA). Legend: L (leukocytes); H (red blood cells); ptn (total protein); gli (glucose); lac (lactate).