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REVIEW

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The spectrum of autoimmune disorders in chronic immune-mediated neuropathies

Joumana Freiha[a], Young Gi Min[b], Yusuf A. Rajabally[c,d] and Chinar Osman[a]

[a]Department of Neurology, Wessex Neurological Centre, University Hospital Southampton, Southampton, UK; [b]Department of Neurology, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea; [c]Aston Medical School, Aston University, Birmingham, UK; [d]Department of Neurology, Inflammatory Neuropathy Clinic, University Hospitals Birmingham, Birmingham, UK

ABSTRACT

Introduction: Autoimmune disorders frequently cluster within individuals, a phenomenon known as polyautoimmunity, yet its scope and implications in chronic immune-mediated neuropathies remain underexplored.

Areas covered: This review examines the association between chronic immune-mediated neuropathies and broader systemic autoimmunity to highlight the immunopathological mechanisms driving these associations, and clinical implications for diagnosis, prognosis, and treatment. A comprehensive literature search was conducted using PubMed and Embase databases for studies published up to October 2025 employing terms related to autoimmune neuropathies, polyautoimmunity, and associated systemic autoimmune diseases.

Expert opinion: While autoimmune comorbidities in CIN are often viewed as confounding conditions for diagnosis, they may indicate a broader, systemic immune dysregulation. Adopting this perspective has direct clinical relevance. Proactive screening for associated autoimmune disorders is essential, as their presence can shape disease trajectory and modify treatment responsiveness. Furthermore, uncovering shared pathological pathways between CIN and these coexisting conditions may open avenues for therapeutic strategies that simultaneously target both neuropathic and systemic manifestations. To advance this field, future research may allow discovery of biomarkers that could stratify patients based on their distinct underlying immune drivers, which may pave the way for a precision medicine approach in this clinically heterogeneous population.

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Autoimmune disorders; chronic immune-mediated neuropathies; spectrum of polyautoimmunity; systemic immune dysregulation; comorbidity screening; treatment personalization

1. Introduction

Autoimmune disorders (AID) often coexist within the same individual, a phenomenon termed polyautoimmunity. This clustering of immune-mediated disorders suggests shared pathogenic mechanisms, including genetic predisposition, environmental triggers, and dysregulated immune responses [1].

A critical perspective in evaluating these associations is distinguishing frequent comorbidity from proven causality, as some co-occurring conditions may reflect coincidence or shared risk factors rather than a direct pathological link.

Chronic immune-mediated neuropathies (CINs) – such as chronic inflammatory demyelinating polyneuropathy (CIDP), multifocal motor neuropathy (MMN), anti-myelin-associated glycoprotein (anti-MAG) neuropathy, chronic ataxic neuropathy with ophthalmoplegia, M-protein, agglutinins, and disialosyl antibodies (CANOMAD), and autoimmune nodopathies are no exception. These neuropathic disorders may occur alongside other autoimmune diseases, suggesting that immune dysregulation may extend beyond the peripheral nervous system (PNS), in a subset of affected subjects.

In this review, we explore the spectrum of polyautoimmunity in CINs. We examine the prevalence of associated autoimmune conditions, potential shared immunological pathways, and the clinical implications of these overlaps for diagnosis and management. Understanding these connections may provide deeper insights into disease mechanisms and guide more personalized therapeutic approaches.

2. Chronic inflammatory demyelinating polyradiculoneuropathy

CIDP is the most common chronic autoimmune PNS disorder. The European Academy of Neurology/Peripheral Nerve Society (EAN/PNS) guidelines published in 2021 have provided up-to-date guidance on CIDP diagnosis and treatment [2]. Current clinical data on the co-occurrence of autoimmune diseases in CIDP primarily derive from uncontrolled case series and small case-control studies in tertiary care settings. Nevertheless, a temporal association between CIDP and various autoimmune comorbidities has been observed proposing shared pathogenic mechanisms (Figure 1).

A key challenge is interpreting these associations is the absence of robust epidemiological data on baseline autoimmune disease coexistence in the general population. As a result, it remains unclear whether the clustering of autoimmune conditions in CIDP patients reflects a true immunological link or merely coincidental overlap in individuals with inherent immune susceptibility.

CONTACT Yusuf A. Rajabally y.rajabally@aston.ac.uk Aston Medical School, Aston University, Aston Triangle, Birmingham B4 7TE, UK

Article highlights

- Chronic immune mediated neuropathies frequently occur alongside other autoimmune diseases, reinforcing the concept that immune dysregulation extends beyond the peripheral nervous system.
- In most cases, these conditions share a common pathogenic mechanism including genetic predisposition, and immune pathway dysregulation.
- The frequent association between immune-mediated neuropathies and other autoimmune diseases has important diagnostic, prognostic, and therapeutic implications for patient management.
- Understanding the spectrum of polyautoimmunity in neuropathic disorders paves the way for more targeted, mechanism-based therapeutic approaches.
- Investigating the links between chronic inflammatory neuropathies and systemic autoimmunity offers new opportunities to unravel disease mechanisms and develop innovative treatments.

2.1. Diabetes mellitus

While Type 1 Diabetes mellitus (DM) results from autoimmune destruction of pancreatic β-cells, Type 2 DM has traditionally been viewed as a metabolic disorder, however, emerging evidence suggests significant inflammatory and autoimmune components. The current understanding is that Type 2 DM now encompasses a vicious cycle where aging and obesity trigger adipose tissue dysfunction, initiating systemic inflammation through dysregulated secretion of adipokines and cytokines. This inflammatory milieu not only exacerbates insulin resistance but also promotes autoimmune activation, evidenced by β-cell autoantibodies, self-reactive T cells, and regulatory T-cell defects [3].

Contradictory reports have previously been made in the last two decades on the relationship between DM and CIDP. While early non-population-based studies reported an, up to elevenfold elevated CIDP risk in diabetic patients [4,5], epidemiological studies had subsequently failed to replicate this association [6,7]. More recent studies of subjects with CIDP from the UK, Serbia, Italy, and the Netherlands, however, consistently demonstrated a two-fold elevated risk of DM in CIDP patients [7].

DPN is a progressive symmetrical length-dependent, axonal polyneuropathy primarily affecting distal sensory and autonomic fibers. Its insidious onset typically presents with gradually worsening sensory deficits – including numbness, paresthesia, and neuropathic pain in a characteristic glove-stocking distribution, alongside variable autonomic involvement. While small fibers are affected first, large fiber involvement emerges with disease progression [8]. In contrast, typical CIDP presents as a symmetric, progressive, or relapsing sensorimotor polyneuropathy affecting both proximal and distal limb muscles over a more rapid fashion [2]. Approximately 50% of CIDP cases present with the typical phenotype, while the remaining half exhibit CIDP variants such as focal, purely sensory or motor, and distal-predominant phenotypes which can be a diagnostic challenge [2].

Patients with CIDP-DM typically present at an older age [7,9] and more frequently exhibit the typical CIDP phenotype compared to non-diabetic CIDP patients [7]. Clinically, CIDP-DM manifests with greater neuropathic severity, including prominent proximal weakness, gait impairment, and imbalance [10], the latter being a distinguishing complaint, alongside higher rates of dysautonomia and sensory deficits [11]. Electrophysiology is helpful in separating CIDP from DPN, by demonstrating typical features of demyelination, absent in DPN [12]. However, therapeutic decision-making with regard to an eventual trial of immunomodulatory therapy should exclusively be based on clinical suspicion.

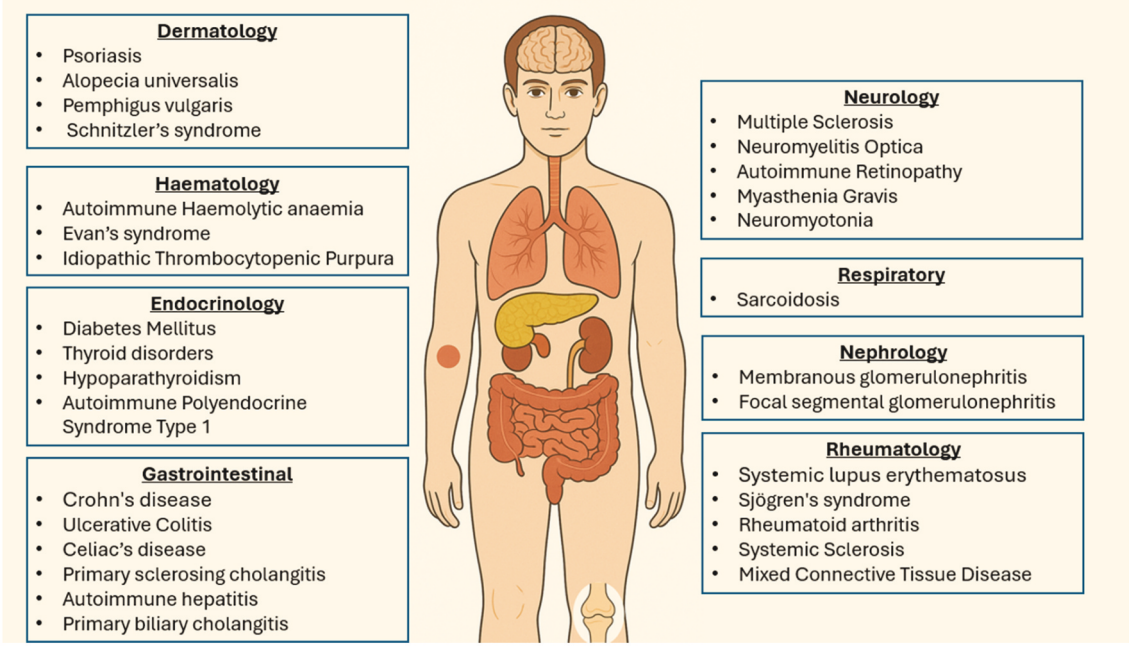

Figure 1. A summary of all reported autoimmune disorders grouped by systems in CIDP.

2.2. Thyroid disorders

An association between hypothyroidism and PNS dysfunction has reported prevalence rates ranging from 10% to 72% [13,14]. Similarly, electrophysiological studies have identified peripheral neuropathy in hyperthyroid patients, with incidence rates varying from 19% to 66% [13,15]. These neuropathies are broadly categorized as either mononeuropathies or polyneuropathies, though available data do not specify whether their underlying pathology involves inflammatory mechanisms.

The Italian CIDP database study group conducted a comprehensive analysis of comorbidities in 294 CIDP patients, identifying thyroid disorders in 11% of the total cases (42/294 patients) [6]. While most reports of CIDP-associated thyroid conditions remain limited to isolated case studies [16,17], their clinical significance, including potential effects on disability progression and treatment responsiveness may require further investigation.

Whether mechanisms involved in CIDP in these subjects may at least in part relate to the autoimmune thyroid pathology, is not demonstrated.

2.3. Hypoparathyroidism

While muscle weakness, fatigue, tetany, and dementia represent well-documented neurological manifestations of hypoparathyroidism, PNS involvement occurs only exceptionally and typically manifests as predominantly axonal neuropathies [18,19]. To date, just one reported case describes an association between hypoparathyroidism and CIDP [20]. In this unique instance, a patient with a six-year history of hypoparathyroidism subsequently developed CIDP, as evidenced by characteristic electrodiagnostic findings, elevated CSF protein levels, and a clinically meaningful response to intravenous immunoglobulin (IVIg).

2.4. Autoimmune polyendocrine syndrome type 1

Autoimmune Polyendocrine Syndrome Type 1 (APS-1) also known as Autoimmune Polyendocrinopathy-Candidiasis-Ectodermal Dystrophy (APECED), or Multiple Autoimmune Syndrome Type 1, is a monogenic autoimmune disorder caused by biallelic loss-of-function mutations in the Autoimmune Regulator (Aire) gene. The syndrome is classically defined by a triad of major clinical manifestations: chronic mucocutaneous candidiasis, chronic hypoparathyroidism, and Addison's disease, along with various endocrine and non-endocrine manifestations. Notably, CIDP has been reported in pediatric APS-1 patients, including one case in a large Sardinian cohort followed for over 25 years [21]. Valenzise et al. also identified two additional CIDP cases in their APS-1 patient cohort [22,23].

Mechanistic insights on this very rare association may come from studies in non-obese diabetic mice carrying a dominant-negative Aire mutation, which develops spontaneous autoimmune peripheral neuropathy, suggesting that CIDP may represent another rare but clinically significant feature of APS-1. The underlying immune dysregulation in these mice involves defective central tolerance to peripheral myelin protein zero [24].

2.5. Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) with PNS involvement is common, with approximately 21–42% of all SLE patients having electrodiagnostic abnormalities [25]. The American College of Rheumatology acknowledges seven PNS manifestations of SLE, although this does not include CIDP [26]. The concurrent diagnosis of systemic SLE and CIDP is rare and may be regarded as coincidental. A systematic review of 16 CIDP and SLE found in the literature prior to 2019 identified a predominantly female cohort (81%) with three distinct disease-onset patterns [27]: prior SLE diagnosis (31%), concurrent CIDP-SLE onset (44%), and CIDP preceding SLE (25%). The cohort exhibited ANA positivity (100%), with anti-DNA (63%), anti-Sm (38%) antibodies, anti-Ro (31.25%), anti-cardiolipin (31.25%), anti-RNP (25%), and anti-B2GPI (6.25%). Electrodiagnostic studies revealed demyelinating features in all cases and CSF analysis of 13 patients demonstrated elevated protein (56%) in nearly all the patients.

Since 2019, several further cases have been reported [28–32]. Notably, these include a novel case of concurrent CIDP and SLE diagnosis during pregnancy [28], a complex multi-autoimmune presentation featuring generalized myasthenia gravis (MG), CIDP, hypothyroidism, and SLE [29], and successful dual-disease remission achieved through cyclophosphamide [30] and Anifrolumab [31]; an unusual CIDP-SLE-Sjögren's syndrome (SS) triad was also reported [32].

A case series described six patients with concurrent CIDP and SLE, whereby half demonstrated significant clinical improvement with IVIg therapy. Treatment responders exhibited features including tetra paresis, severe visceral organ involvement, and multiple SLE-associated autoantibodies [33].

The observed B-cell hyperactivity reflected by multiple SLE-associated autoantibodies and severe visceral involvement may have contributed to peripheral nerve damage through molecular mimicry, where cross-reactive autoantibodies target neuronal antigens like gangliosides or myelin glycoproteins, and complement activation, which can amplify inflammatory nerve injury [34].

2.6. Sjögren's syndrome

In an initial cross-sectional study [35] of 184 polyneuropathy patients identified SS comorbidity in 24% of cases ($n = 44$), with 52% fulfilling definite EFNS/PNS criteria for CIDP and an additional 9% meeting probable criteria, collectively suggesting that SS may drive CIDP-like demyelination in over 60% of affected patients. In a subsequent comparative analysis [36] of 54 CIDP-SS patients versus CIDP-alone controls, two key findings emerged: first, a pronounced female predominance in the CIDP-SS cohort (52% versus 28%), mirroring Sjögren's well-established gender bias; and second, a 2.8-fold increase in cranial nerve impairment (39% versus 14%). Notably, the absence of significant differences in other symptoms, neuropathy severity, electrodiagnostic studies, or CSF profiles argues against classifying CIDP-SS as a distinct entity [36].

The management of CIDP-SS remains poorly characterized, with current strategies largely extrapolated from CIDP treatment paradigms. Roux et al. conducted a retrospective monocentric

study of 28 CIDP patients, including two with SS, demonstrating rituximab efficacy in one SS patient [37]. In contrast, Bregante et al. [38] reported successful treatment of refractory CIDP-SS using intensive immunomodulation with autologous followed by allogeneic hematopoietic stem cell transplantation.

2.7. Rheumatoid arthritis, systemic sclerosis, mixed connective tissue disease

The association between CIDP and other rheumatological conditions is scarce. Current literature reveals no established link between CIDP and rheumatoid arthritis (RA), with only two documented case reports [39,40]. CIDP development in RA patients more frequently correlates with immunomodulatory therapies rather than arising as a direct complication of the rheumatological disease itself. Similarly, systemic sclerosis has been reported in just one instance, occurring two years after CIDP diagnosis in a patient with preexisting anti-nuclear and anti-Scl-70 antibodies [41]. The association with mixed connective tissue disease remains equally rare, with only two cases reported [42,43]. These isolated occurrences, lacking temporal consistency or mechanistic plausibility, could suggest a coincidental, rather than causal, relationships.

2.8. Dermatological disorders

Similar to RA, patients undergoing treatment for psoriasis have also shown a susceptibility to developing other autoimmune conditions, including CIDP. To date, only two cases of non-medication-induced CIDP have been reported with psoriasis, both responding well to Rituximab and Secukinumab [44,45].

Rare dermatological inflammatory conditions have been documented in CIDP patients. However, many of these reports were published in non-English languages, limiting access to full-text articles. These include cases of alopecia universalis, pemphigus vulgaris, and Schnitzler's syndrome [46–49]. The latter being a rare, chronic autoinflammatory disorder characterized by recurrent urticarial rash, fever, and monoclonal IgM gammopathy.

2.9. Sarcoidosis

Sarcoidosis (SAR) is a multisystem granulomatous disorder that can involve the nervous system in approximately 5% of cases, with PNS manifestations occurring in about 14% of cases of neurosarcoidosis [50,51]. The peripheral neuropathies associated with SAR exhibit considerable clinical heterogeneity, ranging from acute or chronic distal symmetric neuropathies to focal or multifocal neuropathies. A retrospective analysis of 57 biopsy-confirmed sarcoid neuropathy cases demonstrated that asymmetric, non-length dependent axonal polyneuropathy represents the predominant pattern, with only three cases (5%) showing demyelinating features, in contrast to classic CIDP which is primarily characterized by demyelination.

A comprehensive French review identified 12 cases of CIDP occurring in association with SAR [52]. Their comparative study of 16 histologically confirmed CIDP-SAR patients versus 17 typical CIDP patients revealed several clinically significant distinctions, though complete data were available for only 14 CIDP-SAR cases. Notably, 70% of CIDP-SAR patients (10/14) developed CIDP within one year of SAR diagnosis. Facial palsy occurred in 36% of CIDP-SAR cases (4/11); and systemic markers including serum ACE levels were elevated in only 20% (2/10) of cases, with hypercalcemia present in just 12% (1/8). Importantly, half of CIDP-SAR patients (8/16) exhibited extra-neurological involvement at diagnosis, and nerve biopsies confirmed granulomatous inflammation in nine cases. Furthermore, the CIDP-SAR cohort exclusively exhibited variant CIDP forms (100% versus 35% in CIDP), which may raise doubt about the diagnosis of CIDP itself, especially with additional differentiating features including weight loss (4/7 cases) and acute/subacute onset (11/14 cases) [52].

Therapeutic responses differed substantially between the two groups. Corticosteroids demonstrated 100% efficacy in CIDP-SAR (13/13 patients), while IVIg showed benefit in only 14% (1/7) of cases, contrasting with the response rate reported in typical CIDP. Plasmapheresis was ineffective in the single CIDP-SAR case where it was attempted. Furthermore, the CIDP-SAR cohort exclusively exhibited variant CIDP forms (100% versus 35% in CIDP), with additional differentiating features including weight loss (4/7 cases) and acute/subacute onset (11/14 cases) [52].

These clinical and therapeutic differences likely reflect distinct underlying pathophysiological mechanisms. In cases diagnosed as 'CIDP-SAR,' multiple mononeuropathies may result primarily from granulomatous inflammation, in effect representing an alternative pathology to that of CIDP. The frequent cranial nerve involvement and multifocal involvement may stem from the patchy distribution of granulomatous inflammation and the good response to corticosteroids but poor response to IVIg and plasmapheresis further supports that 'CIDP-SAR' represents a granulomatous infiltrative neuropathy rather than a primary autoimmune demyelinating disorder, i.e. CIDP per se. These findings underscore the importance of mononeuritis multiplex presentations in SAR as a distinct entity, different to CIDP, and requiring different diagnostic approaches (including consideration of nerve biopsy) and therapeutic strategies (favoring immunosuppression).

2.10. CNS demyelinating disorders: multiple sclerosis, neuromyelitis optica spectrum disorder

While MS globally affects about 23.9 cases per 100,000 population [53], CIDP occurs in only about 3 per 100,000 [54]. Between 1980 and 2013, 133 co-existing MS polyneuropathy cases were identified, in a retrospective chart review of Mayo Clinic patients, 11 of which were CIDP [55]. There have been subsequent cases of CIDP in patients with MS [56,57]. Conversely, CIDP patients showed MRI evidence of central demyelination appearing in 17–37% of patients [5,58]. Particularly striking are reports of extensive thoracic spinal cord lesions in CIDP [59]. Additionally, visual evoked potential studies have demonstrated optic nerve involvement in 47% (8/17) of patients with CIDP, in one small study, consistent with subclinical optic neuritis [60] Combined central and peripheral demyelination (CCPD) may be a distinct clinical entity, with emerging evidence, in a proportion of cases, particularly implicating neurofascin-155 (NF155) autoantibodies in its pathogenesis, this will be further discussed in the autoimmune nodopathy section.

Neuromyelitis optica spectrum disorder (NMOSD) is another inflammatory CNS disorder that is associated with serum aquaporin-4 immunoglobulin G antibodies [61]. To date there have been few reported of cases of co-occurrence of both CIDP and NMOSD [62,63].

2.11. Autoimmune retinopathy

A single case of concurrent CIDP and autoimmune retinopathy has been reported to our knowledge, characterized by progressive photoreceptor degeneration, confirmed by funduscopic examination, optical coherence tomography, and electrophysiological studies [64]. Notably, the condition demonstrated significant clinical improvement following treatment with systemic corticosteroids and IVIg.

2.12. Myasthenia gravis

A total of 16 cases of CIDP-MG overlap have been reported in the literature [29,65–79]. The characteristics of 14 cases analyzed are here detailed (Figure 2).

Out of the 14 analyzed cases, 8 (57.1%) were male, and 6 (42.9%) were female. The age at which CIDP was diagnosed varied widely, ranging from 10 to 76 years, with a mean age of approximately 47 years. The overwhelming majority of cases (13 out of 14, 92.9%) presented with generalized MG, while only one case (8.1%) exhibited ocular MG. The sequence in which MG and CIDP developed varied among patients. Six cases (40%) had a concomitant presentation. In three cases (20%), MG appeared first, while CIDP preceded MG in four cases (20%). Acetylcholine receptor antibodies (AChR) were present in all 14 cases (100%). Additionally, three cases (20%) had other antibodies, including voltage-gated potassium channel, striatal, and titin antibodies. CSF analysis was available for 12 cases. Elevated protein levels were observed in 10 cases (83.3%). Four cases reported other overlap AID, including thyroiditis, autoimmune hepatitis (AIH), and glomerulonephritis. Additionally, two concurrent cases were triggered by infections. First-line therapy varied depending on the disease onset sequence and the publication date of the case reports. Among the cases with treatment details, six out of twelve required second-line therapies, with two cases achieving remission only after further treatment with cyclophosphamide or rituximab. Thymoma and subsequent thymectomies was reported in seven out of eleven cases with thymic pathology either thymomas or hyperplasia.

Shared genetic susceptibility factors, such as HLA class II alleles (e.g. HLA-DRB1) may explain the presence of other AID and may predispose individuals to broader immune dysregulation [29]. While the exact Th1/Th2 balance in CIDP-MG overlap remains unresolved, the coexistence of these conditions underscores the heterogeneity of autoimmune mechanisms. The involvement of both cellular (Th1/T-cell) and humoral (Th2/B-cell) immunity suggests that therapeutic strategies targeting multiple immune pathways, such as B-cell depletion (rituximab) or broad immunosuppression may be necessary in refractory cases.

2.13. Neuromyotonia

Acquired neuromyotonia is an extremely rare disorder, most likely driven by autoimmune mechanisms. Though it typically

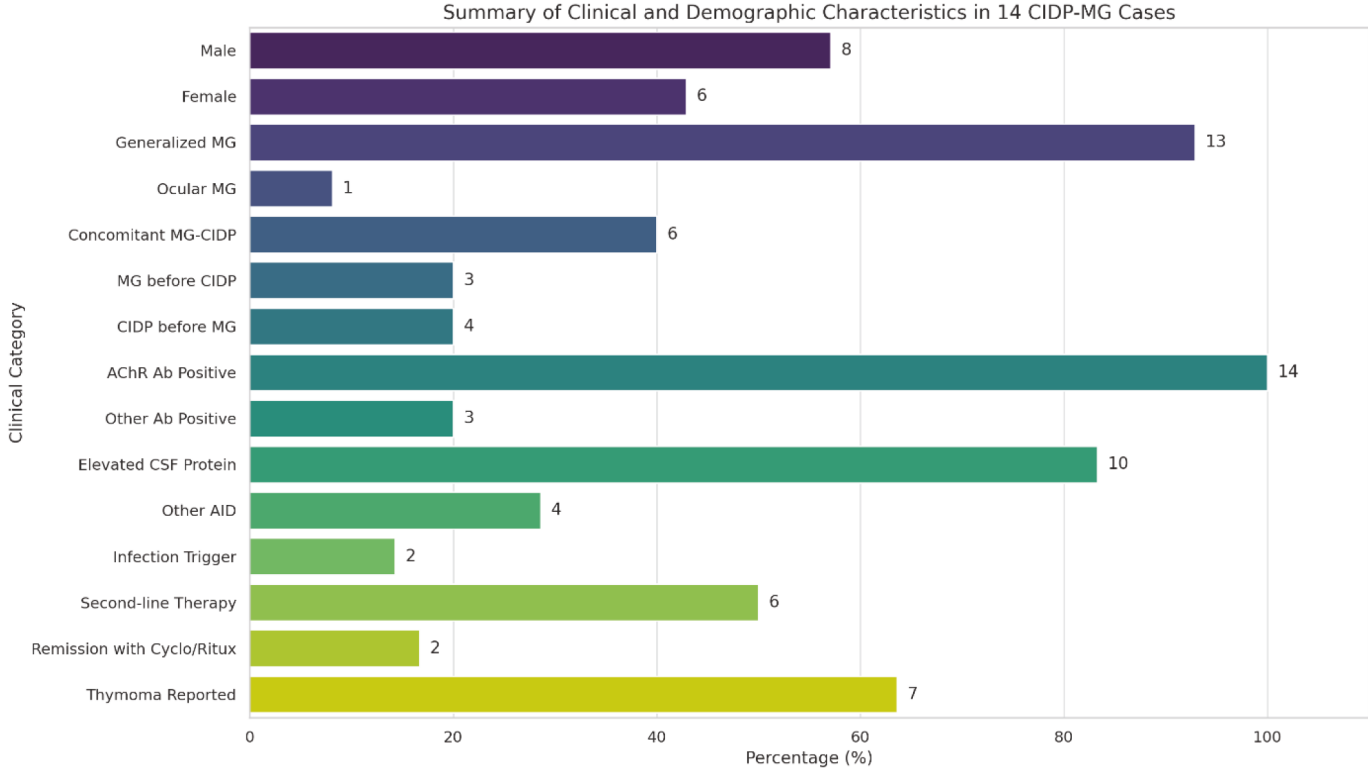

Figure 2. Features of the 14 CIDP-MG cases analyzed.

manifests as an isolated syndrome, its co-occurrence with CIDP has only been reported by two cases to date. The first documented case involved a patient with neuropathic symptoms, including muscle twitching, contractures, and focal hypertrophy, alongside electrophysiological confirmation of neuromyotonia. The second case featured a CIDP patient who developed intermittent binocular diplopia over four months, consistent with ocular neuromyotonia. Notably, despite comprehensive antibody testing yielding no clear biomarkers, both patients experienced complete resolution of neuromyotonic symptoms following IVIg therapy [80,81]. The dramatic response may suggest a potential pathophysiological link between the two conditions.

2.14. Autoimmune hemolytic anemia

Autoimmune hemolytic anemia (AIHA) is a rare immune-mediated disorder characterized by the premature destruction of red blood cells through autoantibodies, which can be classified as warm-reactive or cold-reactive based on their thermal amplitude [82]. While AIHA may occur as an isolated condition, it can also manifest as part of broader autoimmune syndromes. Evans syndrome represents a distinct clinical entity characterized by the concurrent presence of immune-mediated thrombocytopenia and AIHA, typically associated with benign conditions and historically managed with splenectomy [83]. Several case reports describe an association between CIDP and AIHA and autoimmune hematologic conditions [83,84]. A 39-year-old man initially presented with AIHA that responded to corticosteroids but subsequently developed CIDP with elevated anti-ganglioside antibodies. Similarly, an 85-year-old male experienced simultaneous onset of CIDP and AIHA that improved in parallel with steroid treatment [85]. More complex presentations include a 50-year-old male whose CIDP progressed to include Evans syndrome. While conventional therapies failed, B-cell depletion with rituximab achieved sustained remission in both conditions [83]. Additionally, a 78-year-old male developed refractory CIDP post-splenectomy for AIHA, which only resolved after discovery and resection of pancreatic adenocarcinoma [86]. This case of CIDP and AIHA co-occurring as paraneoplastic phenomena from a solid tumor indicates the importance of malignancy screening in refractory CIDP.

2.15. Idiopathic thrombocytopenic purpura

Several case reports highlight an association between CIDP and Idiopathic Thrombocytopenic Purpura (ITP), demonstrating that both conditions often respond similarly to immunomodulatory therapies, particularly IVIg and rituximab [87,88]. The shared therapeutic response underscores the plausibility of a common immunological pathway, involving CD20+ B cells and Fc receptor-mediated platelet and nerve injury. Some cases suggest that CIDP and ITP may arise sequentially [89] or concurrently [87,88], with ITP often preceding CIDP by months to years, hinting at a progressive breakdown of immune tolerance. One case was preceded by an infection [87], while another was associated with a malignancy [90].

2.16. Inflammatory bowel disease

Inflammatory bowel disease (IBD) refers to a group of chronic inflammatory disorders of the gastrointestinal tract, primarily categorized as Crohn's disease (CD) and ulcerative colitis (UC). Both conditions are associated with extraintestinal manifestations, including neurological complications, which exhibit a widely reported incidence ranging from 0.25% to 47.5% [91]. Among these, peripheral neuropathy is one of the most frequently observed, with potential etiologies including immune-mediated inflammation, micronutrient deficiencies (e.g. vitamin B12, vitamin D, copper), and iatrogenic factors (e.g. metronidazole, TNF-α antagonists).

Several papers have reported single cases of CIDP associated with either CD or UC [92,93]. However, Gondim et al. investigated 33 patients with IBD-related neuropathy after excluding secondary causes and identified CIDP in 3 CD patients and 4 UC patients [94]. The study revealed that demyelinating changes were less pronounced in UC compared to CD, and UC patients also exhibited less severe weakness than their CD counterparts with demyelinating neuropathy. Treatment responses differed between the two subtypes. In CD patients, immunomodulatory therapy – including plasmapheresis, IVIg, etanercept, cyclophosphamide, azathioprine, and fludarabine – resulted in improvement in 80% of cases. In contrast, UC patients were treated exclusively with IVIg, achieving improvement in 100% of cases.

Growing evidence indicates that gut dysbiosis may be a key factor in the development of IBD [95]. The disease arises from a maladaptive immune reaction to gut microbes in genetically predisposed individuals, with the Th17 immune pathway playing a central role. This response triggers the release of proinflammatory cytokines and chemokines, leading to persistent intestinal inflammation marked by T-cell accumulation, mucosal injury, and vascular inflammation. These mechanisms may also contribute to systemic complications, such as CIDP. Elevated fecal calprotectin levels, a sign of gut inflammation, have been observed even in CIDP patients without clinical IBD, supporting the idea of interconnected gut-immune-neural pathways in both disorders [95].

2.17. Celiac disease

In a large nationwide Swedish study, the risk of neuropathy was examined in patients with biopsy-confirmed Celiac disease [96]. Comparing 28,232 Celiac disease patients to 139,473 matched controls, they discovered a 2.5-fold increased risk of neuropathy overall. Strikingly, Celiac disease patients were nearly three times more likely to develop CIDP. These findings may underscore the importance of screening for celiac disease in patients with CIDP

2.18. Autoimmune liver disease

Autoimmune liver conditions linked to CIDP are rare, manifesting as primary sclerosing cholangitis (PSC), AIH, or primary biliary cholangitis (PBC). One case involved a patient with a long-dormant history of PSC, whose disease suddenly flared alongside the onset of CIDP [87]. Similarly, Murata et al.

reported the case of a 36-year-old female whose chronic neuropathy symptoms led to a CIDP diagnosis, only for further testing to unveil elevated liver enzymes, anti-mitochondrial M2 antibodies, and biopsy-confirmed PBC [97]. The association between CIDP and AIH has also been reported. In one instance, a patient presenting with CIDP and abdominal distension was found to have cirrhosis, alongside anti-LKM-1 antibodies [98]. Another case, tested positive for ANA, ANCA, and smooth muscle antibodies [99]. More recently, anti-dihydrolipoamide S-acetyltransferase autoantibodies have been described in 2 CIDP patients with concurrent PBC and AIH [100].

2.19. Nephrotic syndromes

Since the first documented case in 1987 [101], concurrent presentations of CIDP and NS have suggested a potential autoimmune-mediated mechanism targeting both peripheral nerve myelin and renal podocytes. Among the reported CIDP patients with NS, MN is the most frequently observed, with at least 27 documented cases [102]. Of these, four patients exhibited simultaneous onset of CIDP and MN, 16 developed CIDP prior to MN, and two presented with MN before neurological manifestations. Focal segmental glomerulosclerosis represents the next most common renal pathology [103,104]. Renal involvement was often suspected in patients presenting with hypertension or edema, though proteinuria was occasionally detected incidentally. This diagnostic variability raises the possibility that many cases of concurrent neuro-renal disease may remain undiagnosed, highlighting the need for heightened clinical vigilance.

Contemporary research has characterized distinct autoantibodies targeting paranodal and nodal proteins, leading to the recognition of autoimmune nodopathies (AN) as a separate entity from CIDP. The classification status of previously reported cases (described prior to this discovery) remains uncertain, as their antibody profiles were not characterized using current diagnostic criteria.

3. AID in multifocal motor neuropathy

MMN is a rare pure motor immune-mediated neuropathy, characterized by multifocal motor deficits affecting predominantly the upper limbs [105]. Electrophysiologically, motor conduction blocks are detected, and IgM anti-ganglioside antibodies to GM1 are reported in 30–50% of affected subjects [106]. One case report described a patient with MMN-like symptoms who tested positive for CASPR2 antibodies – an autoantibody more commonly seen in conditions like neuromyotonia and autoimmune encephalitis. The patient's improvement following IVIg suggests that similar autoimmune pathways may underlie a subset of MMN cases [107]. Conversely, Rosier et al. reported a case of MMN with clinical and electrodiagnostic features of neuromyotonia, with a negative antibody panel [80].

The association between MMN and other autoimmune diseases has been suggested in few case reports [35,94,108,109]. Examples involve the co-occurrence of Hashimoto's thyroiditis [109], SS [35] and inflammatory bowel disease (IBD) [94]. More broadly, a Dutch case-control study found that patients with MMN who also had first-degree relatives with the condition had significantly higher rates of AD, including type 1 DM and celiac disease, compared to controls [108]. These patients also had a higher prevalence of HLA-DRB1 × 15, a genetic marker associated with other autoimmune conditions, suggesting possible immunogenetic factors contributing to the development of MMN and its comorbid autoimmune conditions [108].

4. AID in CANOMAD/CANDA

Chronic ataxic neuropathy with ophthalmoplegia and disialosyl antibodies (CANOMAD), or its related clinically more restricted variant, chronic ataxic neuropathy with disialosyl antibodies (CANDA) is a rare, immune-mediated neuropathy characterized by chronic sensory ataxia, possible ophthalmoplegia, and presence of IgM autoantibodies targeting disialosyl gangliosides [110]. There has been limited evidence in the literature to suggest an association between CANOMAD and other AID. Toussirot et al described co-occurrence of CANOMAD in a patient with longstanding positive anti-CCP RA [111]. Sanvito et al. reported two CANOMAD patients who subsequently developed optic neuropathies associated with high titers of anti-GQ1b/GT1b antibodies, suggesting cross-reactivity with optic nerve tissues [112]. Moreover, Delval et al. described an association with extramembranous glomerulopathy, likely mediated by IgM paraprotein deposition in the glomerular basement membrane, triggering complement activation and immune injury and leading to renal dysfunction [113].

These observations collectively suggest that while CANOMAD primarily affects the PNS, its IgM autoantibodies may occasionally target other tissues through molecular mimicry or immune complex deposition (Figure 3).

5. AID in anti-MAG neuropathy

Anti-MAG neuropathy is a chronic immune-mediated demyelinating neuropathy, most often associated with IgM monoclonal gammopathy [114]. While traditionally limited to the PNS, few reports suggest that anti-MAG neuropathy can co-occur with a variety of other systemic autoimmune diseases (Figure 4), which may suggest in some cases, a broader immune dysregulation [115,116].

In a case series by Pascual-Goñi et al, two patients with anti-MAG neuropathy were also diagnosed with type 2 DM and RA [115]. Another recent case identified a co-occurrence of Hashimoto's thyroiditis and Anti- MAG neuropathy [116].

Another report identified the concurrent presence of anti-MAG and anti-MOG antibodies in a patient presenting with both a central nervous system demyelinating disorder and a peripheral demyelinating polyneuropathy, suggesting potential convergence between peripheral and central autoimmune processes [117].

A connection between anti-MAG neuropathy and associated autoimmune conditions may reside within the MYD88 gene. MYD88 is a central adaptor protein that transduces pro-inflammatory signals, such as those from the IL-1β receptor, converging on the NF-κB pathway to amplify inflammatory mediator production, a key mechanism in conditions like

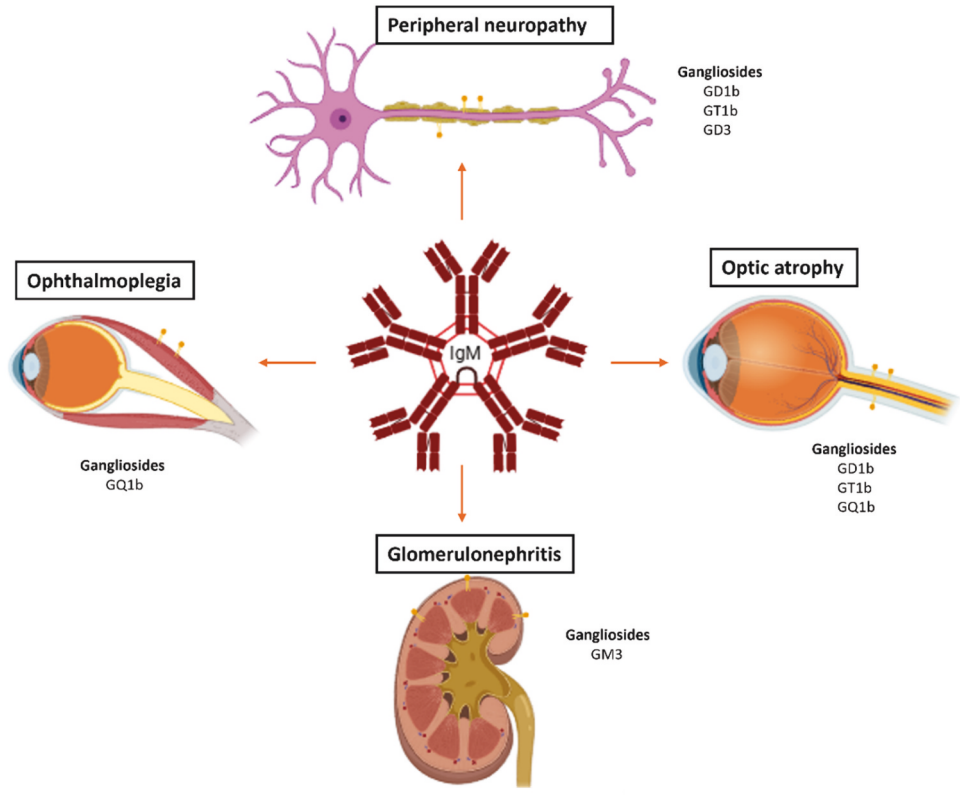

Figure 3. Pathophysiology of CANOMAD and reported autoimmune disorders, in addition to main ganglioside target of IgM.

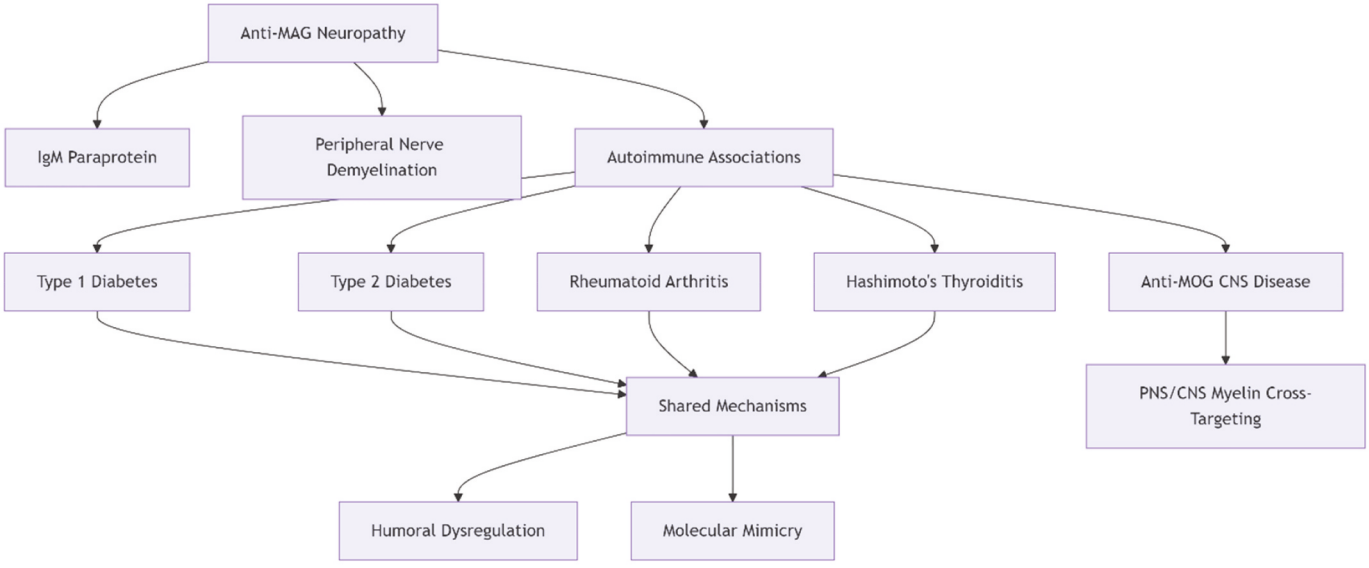

Figure 4. Anti-MAG neuropathy autoimmune associations and possible underlining pathophysiology.

rheumatoid arthritis. Significantly, the MYD88 somatic mutation (MYD88^{L265P}) is found in approximately 60% of patients with anti-MAG neuropathy and is also strongly linked to severe inflammatory arthritis [118].

6. AID in autoimmune nodopathy

AN is a rare immune mediated neuropathy characterized by conduction deficits due to autoantibodies targeting the nodes of Ranvier and paranodal junctions of the peripheral nerves. Representative target antigens are neurofascin-155 (NF155), contactin-1 (CNTN1), and contactin associated protein 1 (CASPR1), with the corresponding autoantibodies predominantly of the IgG4 subclass [119]. Clinically, patients typically present with sensory ataxia, tremor, and proximal weakness. Paraclinical findings include distal accentuated conduction slowing rather than conduction blocks, markedly elevated CSF protein levels and nerve hypertrophy. Autoantibodies of

the IgG4 subclass do not activate Fc-dependent mechanisms including complement activation [120]; therefore, patients with AN tend to respond poorly to first-line CIDP treatments like immunoglobulin, while rituximab is considered a promising option [121]. In rare cases, antibodies reactive to all NF isoforms (NF155, 186, and 140, 'pan-NF neuropathy') or specifically to NF186/140 may lead to a fulminant GBS-like presentation with tetraplegia. AN has been frequently associated with nephrotic syndrome (NS) and CNS demyelination.

The co-occurrence of CNTN1 AN and nephrotic syndrome have been recently described [122,123]. A French group found nephrotic syndrome in 5 among 11 patients with CNTN1 AN, with most cases developing concurrently, while renal involvement may precede neurological symptoms by several weeks in some cases [122]. In a UK cohort, 8 out of 10 (80%) patients with CNTN1 AN had nephrotic syndrome [123]. Interestingly, the same study identified 4 seropositive subjects among 295 patients with idiopathic MN, suggesting that CNTN1 autoimmunity may also manifest as isolated MN without neurological involvement [123]. Pathological findings are usually consistent with those of MN, showing characteristic immune complex and complement deposits in glomerular basement membrane.

The neuro-renal syndrome observed in CNTN1 autoimmunity is supported by the expression of CNTN1 in human glomerular podocytes, as well as in the paranodes of peripheral nerves. Protein extracts from human glomeruli show a 125 ~ 135 kDa band (corresponding to CNTN1), which is readily captured by commercial anti-CNTN1 antibodies [123,124]. Factors determining the clinical presentation – whether as neurorenal syndrome or isolated neuropathy – are not fully understood, although antibody titers, which are higher in the former, or preexisting renal injury, e.g. due to diabetes, may contribute to the renal manifestations [124,125].

Neurofascin is another neuronal protein expressed in podocytes. Its expression was first identified through transcriptomic analysis of human glomeruli [126]. Immunofluorescence shows that NF is expressed in the major process of podocytes, and immunoblotting of kidney lysates reveals a band at approximately 185 kDa, which may correspond to the NF186 isoform [127]. Cases of nephrotic syndrome have been reported in association with pan-NF or NF186/140 neuropathies [128,129]. In addition to MN, focal segmental glomerulosclerosis has also been observed in these cases [128,130].

On the other hand, the pathogenesis of CCPD remains unclear, although it has been hypothesized to involve autoimmune responses targeting unknown myelin antigens shared by both the CNS and the PNS. Two Japanese studies reported that 45.5% to 86% of CCPD patients tested positive for anti-NF155 antibodies [131,132]. However, this finding was not replicated in Western cohorts: in the studies by Cortese et al. [133] and Vural et al. [134] studies, none of a total of 21 patients tested were positive, suggesting possible ethnic differences in CCPD presenting in Caucasian and Asian subjects. Other autoantibodies targeting nodal or paranodal proteins have also been proposed, including NF155 IgM, NF186 IgG, MAG IgM, CASPR2 antibodies, and GM1 IgM [135,136]. More recently, PNS involvement has otherwise been observed in some cases of myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease and histological evidence of MOG expression in the PNS has been reported [136,137].

7. Expert opinion

As illustrated by this review, chronic immune-mediated neuropathies have been described in association with multiple other AID.

The hypothesis that CIDP may be a manifestation of systemic immune dysregulation rather than an isolated peripheral nervous system disorder may have significant implications in how the disease is investigated and managed. Relationships between CIDP and diverse systemic conditions, possibly indicative of common pathogenic mechanisms involving shared autoantigens, genetic predispositions, and environmental triggers, although suggested, remain, however, unconfirmed.

It is possible that therapeutic decision-making in CIDP may need to consider incorporating these systemic associations when selecting treatment strategies. Of note, a recent study conducted in our units identified higher immunoglobulin dosing requirements in subjects with CIDP and concurrent AID [138] suggesting therapeutic response implications of such associations.

In practice, it would appear justified to systematically screen for associated systemic AID in the presence of any chronic inflammatory neuropathy. Such evaluation may reveal treatable comorbid conditions that may significantly influence both neurological outcomes and overall prognosis.

Significant knowledge gaps persist. Collaborative studies of large populations are needed particularly in view of the low prevalence of CIDP, to better characterize the epidemiology of inflammatory neuropathy co-existing with other AID. It is crucial to frame this research with appropriate caution. Many reported disease associations are considered coincidental and should not be overinterpreted as causal. Similar caution applies to other proposed links, which often rely on limited, anecdotal, or controversial data. In this regard and as demonstrated by the small numbers of patients described in the papers included in the current literature review, the existing evidence remains greatly limited. Mechanistic studies are otherwise desirable in an attempt to elucidate shared autoantigens across affected organ systems and characterize the complex interplay between genetic predisposition, environmental factors, and immune dysregulation. In conclusion, the association of chronic inflammatory neuropathies and other AID is well-described, but limited, in the literature, with possible implications on diagnostic and management modalities. Given the wide range of reported associations and the low prevalence of inflammatory neuropathies, and hence the paucity of available data, extensive and robust epidemiological studies on described associations are needed, as well as dedicated studies on the pathophysiological and genetic aspects, for the commonest. Future research will determine whether such associations

may in effect have implications from a therapeutic standpoint.

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Generative artificial intelligence (AI)

ChatGPT GPT-4o was used in creating the image of the human in Figure 1. It was also used in adjusting the other graphs after the authors used PowerPoint for the initial blueprint of the image.

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