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SCIENTIFIC ARTICLE - INTEGRATIVE REVIEW

IMMUNE-MEDIATED RHEUMATIC DISEASES AND THEIR PERIPHERAL NEUROPATHY MANIFESTATIONS

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ABSTRACT

Immune-mediated rheumatic diseases (IMRDs) frequently involve neurological complications, with peripheral neuropathy being a relevant but underdiagnosed manifestation. This integrative review evaluated evidence on the prevalence, clinical patterns, and associated factors of neuropathy in rheumatoid arthritis, Sjögren's disease, systemic lupus erythematosus, and systemic vasculitis. A literature search was conducted in the PubMed database, including studies published between 2010 and 2025. Findings show that peripheral neuropathy is highly prevalent in rheumatoid arthritis, often subclinical, and in vasculitis, where it presents acutely and with severe symptoms. In Sjögren's disease, specific sensory forms and small fiber neuropathy may precede classic symptoms. In systemic lupus erythematosus, neuropathy occurs early and in association with disease activity and responds well to immunosuppressive therapy. The study highlights the importance of early identification of peripheral neuropathy in autoimmune diseases, allowing more effective interventions and improved functional prognosis.

Keywords: Autoimmune diseases, Early diagnosis, Inflammation, Peripheral neuropathy, Rheumatology.

INTRODUCTION

Immune-mediated rheumatic diseases (IMRDs) comprise a group of chronic disorders characterized by dysregulation of the immune system, inflammation, and tissue destruction. Several systems and tissues are affected. Among the neurological manifestations, peripheral neuropathies stand out, as they may cause pain, paresthesia, loss of strength, and sensory or motor dysfunctions, leading to impaired quality of life. The rheumatic diseases with the highest prevalence of neuropathies are: rheumatoid arthritis (RA), Sjögren's disease (SD), systemic lupus erythematosus (SLE), and systemic vasculitides.[1]

Rheumatoid arthritis is a chronic inflammatory autoimmune disease that mainly affects small joints, leading to pain, stiffness, and joint deformities. In more advanced stages or in cases of high inflammatory activity, RA may present with extra-articular manifestations, including peripheral neuropathy. It is estimated that up to 75% of RA patients present some degree of neuropathy, even if subclinical. The most common manifestations include asymmetric sensorimotor neuropathy,

multiple mononeuropathy, and carpal tunnel syndrome.[2]

Sjögren's disease mainly affects exocrine glands, causing ocular and oral dryness, but it may also present significant extraglandular manifestations, such as joint, renal, pulmonary, and neurological involvement. Peripheral nervous system involvement in SD occurs in 15% of cases, and the most common forms are distal sensory axonal polyneuropathy and sensory neuronopathy (ganglionopathy), in addition to small fiber neuropathy — often associated with severe pain and autonomic dysfunction. In many cases, neurological symptoms precede the clinical diagnosis of SD, hindering early identification of the disease.[3]

Systemic lupus erythematosus is a multisystemic autoimmune inflammatory disease characterized by the production of autoantibodies and immune complex formation, with the potential to compromise any organ, including the nervous system. Peripheral neuropathy in SLE is frequently associated with inflammatory disease activity, and the most commonly observed forms are axonal sensorimotor polyneuropathy, multiple mononeuropathy, and, more rarely, cranial neuropathies. The pathogenic mechanisms include peripheral vasculitis, immune complex deposition, and immune-mediated neuronal damage.[4]

Systemic vasculitides comprise a heterogeneous group of diseases characterized by inflammation of blood vessels, which may affect arteries, venules, and capillaries of different calibers. When the vasa nervorum is involved, ischemia and tissue necrosis may occur, leading to the development of multiple mononeuropathy, the most common clinical form of vasculitic neuropathy. Vasculitides such as polyarteritis nodosa (PAN), granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and cryoglobulinemic vasculitis are most frequently associated with this type of involvement.[5],[6]

Although pathophysiological mechanisms may vary and clinical manifestations may be similar, there are specific forms of neuropathy in each condition: sensory neuronopathy and small fiber neuropathy in SD; sensorimotor axonal patterns in SLE; carpal tunnel syndrome and subclinical neuropathy in RA; and painful multiple mononeuropathy in vasculitides. Recognizing the different clinical presentations of neuropathy in patients with IMRDs is essential for treatment, providing these patients with better quality of life. This review aims to analyze peripheral neuropathies observed in IMRDs (rheumatoid arthritis, Sjögren's disease, systemic lupus erythematosus, and vasculitis), considering their main clinical, diagnostic, and associated aspects.

METHOD

This study is an integrative literature review, aimed at gathering and critically analyzing the available evidence on the occurrence of peripheral neuropathies in immune-mediated rheumatic diseases.

Data collection was carried out through a search in the PubMed database, between May and July 2025. The search strategy involved combining the descriptor "peripheral neuropathy" with the English names of the following autoimmune diseases: systemic lupus erythematosus, Sjögren's syndrome, rheumatoid arthritis, and systemic vasculitis. Thus, four independent searches were performed, using the following pairs of descriptors: "peripheral neuropathy" AND "systemic lupus erythematosus," "peripheral neuropathy" AND "Sjögren's syndrome," "peripheral neuropathy" AND "rheumatoid arthritis," and "peripheral neuropathy" AND "systemic vasculitis." Finally, a fifth independent search was conducted combining the descriptors "peripheral neuropathy" AND "rheumatic diseases," in order to cover the broader theme.

In each of these searches, only articles that contained both descriptors in the title were selected, ensuring that peripheral neuropathy was directly related to the autoimmune disease investigated. A temporal filter was also applied, including only publications from the last 15 years (2010 to 2025), in order to guarantee the timeliness of the evidence analyzed.

Article screening was carried out based on the reading of titles and abstracts, considering relevance to the topic. Studies considered relevant were then evaluated in full. Original articles, systematic reviews, and narrative reviews published in English, Portuguese, or Spanish were included. Duplicate articles, those without access to the full text, or those addressing non-peripheral neurological manifestations were excluded.

Each set of studies, corresponding to one of the autoimmune diseases investigated, was analyzed separately. The selected articles were evaluated based on their scientific relevance and methodological quality. The extracted information included: type of peripheral neuropathy described, frequency, clinical presentation, diagnostic methods, pathophysiological mechanisms, and therapeutic approaches.

The results were organized descriptively, by autoimmune disease, to allow a clear comparison between the findings of each group.

DISCUSSIONS

The main results of the articles included in this review are presented in Table 1:

Table 1. Main results of the selected articles

Titles	Authors/Year	Main results
Clinical characteristics of rheumatoid arthritis patients with peripheral neuropathy and potential related risk factors.	Li et al.[2]	Prevalence of PN: 50%; Types: 63.6% sensorimotor, 18.2% pure sensory, 13.6% pure motor; 22.7% with carpal tunnel syndrome; Risk factors: total protein < 63 g/L, anti-CCP < 285.7 U/mL, elevated CRP and platelets in severe cases; High association with neurological symptoms and loss of reflexes.

Prevalence and patterns of peripheral neuropathy in patients of rheumatoid arthritis.	Kaeley et al.[7]	Prevalence of PN: 75.3%; 50.7% with subclinical neuropathy; Types: 33.7% sensorimotor, 22.4% pure motor, 8.9% multiple mononeuropathy, 8.9% compressive; Association with age, longer disease duration, higher DAS-28, ESR and CRP; Worse functionality (HAQ-DI) and greater pain (VAS) in patients with PN.
Primary Sjögren syndrome-related peripheral neuropathy: A systematic review and meta-analysis.	Liampas et al.[8]	Prevalence of PN: 15% (in >5,600 patients); Most common type: distal axonal polyneuropathy (80%); Second most common form: sensory neuronopathy (20%); Mononeuropathy (e.g., carpal tunnel) in 12.8%; Trigeminal neuropathy in 3.9%; Associated factors: advanced age and vasculitis; Neurological symptoms often precede the diagnosis of SD.
Relation of Sensory Peripheral Neuropathy in Sjögren Syndrome to Anti-Ro/SSA.	Scofield et al.[9]	31% of patients with SD presented sensory peripheral neuropathy; Significant association with anti-Ro/SSA and anti-La/SSB detected by immunodiffusion (66.7% of those with both antibodies had PN); Isolated anti-Ro: 48.1% with PN; No significant association with vitamin B12; More sensitive

		techniques (ELISA, BioPlex) showed no correlation with PN.
Peripheral Neuropathy in Patients with Systemic Lupus Erythematosus.	Florica et al.[4]	Prevalence of PN: 13.5%; 60.3% of cases attributed to SLE; Most common forms: sensory or sensorimotor polyneuropathy, multiple mononeuropathy, cranial neuropathy; Predominance of asymmetric and distal involvement; 74% with axonal pattern on electroneuromyography; SLEDAI significantly higher in cases attributed to SLE; Favorable therapeutic response in 66% of cases.
Short- and Long-Term Outcome of Systemic Lupus Erythematosus Peripheral Neuropathy: Bimodal Pattern of Onset and Treatment Response.	Fargetti et al.[10]	Prevalence of PN attributed exclusively to SLE: 1.8%; Most cases occur within the first 5 years of disease; Most common type: axonal sensorimotor polyneuropathy (71.1%); Main symptoms: paresthesia, pain, weakness, gait disturbance; 92.1% with clinical improvement after 1 year; 89.3% in remission after 5 years; High frequency of immunosuppressant and corticosteroid use.
Neuropathy associated	Graf and Imboden[5]	Frequency of PN: PAN

with vasculitis.		(85%), EGPA (60–80%), MPA (40–50%), GPA (20–25%); Predominant clinical form: painful and asymmetric multiple mononeuropathy; Most affected nerves: deep fibular, ulnar; Nonsystemic vasculitic neuropathy (NSVN) in 25% of cases; Histological lesions: fibrinoid necrosis, transmural infiltrate, and vascular occlusion.
Peripheral neuropathy in systemic vasculitis and other autoimmune diseases – a report of five cases.	Rodrigues et al.[11]	Study of 5 cases with PN due to systemic vasculitis: MPA, HBV-PAN, EGPA, and an undetermined case; Pattern: asymmetric sensorimotor axonal neuropathy; Progression to multiple mononeuropathy with overlapping pattern; Diagnosis confirmed by nerve/skin biopsy; Treatment with corticosteroid, cyclophosphamide, and antivirals; Good clinical response and variable prognosis.

Source: authors' elaboration.

This integrative literature review highlighted peripheral neuropathy as a relevant manifestation in several immune-mediated rheumatic diseases (IMRDs), with clinical, pathophysiological, and therapeutic characteristics that vary according to the underlying etiology. The results obtained from the analysis of the selected studies identified patterns of prevalence, clinical presentations, risk factors, and outcomes associated with PN in the IMRDs evaluated: rheumatoid arthritis (RA), Sjögren's disease (SD), systemic lupus erythematosus (SLE), and systemic vasculitis. The high frequency and heterogeneity of PN hinder its detection and clinical management in these conditions.

1. Rheumatoid Arthritis

In rheumatoid arthritis, PN proved to be highly prevalent, identified in about 75.3% of patients in Kaeley et al.[7] and in 50% in the study by Li et al.[2] An interesting finding of Kaeley et al.[7] is that, among the affected patients, approximately half of the PN cases were subclinical, asymptomatic on

neurological clinical evaluation, requiring complementary tests, such as electroneuromyography, for detection. The most frequently observed clinical patterns in these studies were asymmetric axonal sensorimotor neuropathies, multiple mononeuropathy, carpal tunnel syndrome, and pure motor neuropathy. This diversity of clinical manifestations in RA suggests different mechanisms, including mechanical compression (as in carpal tunnel syndrome), systemic inflammatory activity, and neural damage secondary to the progression of joint disease.

In the study by Li et al.[2], among the 44 patients with PN, 28 presented multiple nerve involvement (11 with polyneuropathy and 17 with multiple mononeuropathy) and 16 presented single nerve involvement (10 with carpal tunnel syndrome). In cases with symptoms, the most frequent were numbness (97.7%), pain (54.5%), paresthesia (45.5%), and weakness (36.4%). Objective findings included loss of deep reflexes (84%), especially in knees and ankles, and sensory alterations such as decreased analgesia (65.9%), apselaphesia (61.3%), and thermesthesia (40.9%).

Neurological involvement was also correlated with longer RA duration, higher inflammatory disease activity (elevated CRP and DAS-28), presence of joint erosions, subcutaneous nodules, and muscle atrophy. In addition, these patients presented higher scores of functional disability (HAQ-DI) and greater pain intensity (VAS). Laboratory tests showed relevant inflammatory and immunological alterations in patients with PN compared to others, such as hypoalbuminemia, thrombocytosis, leukocytosis, and low anti-CCP levels, especially in more severe cases. These findings support the relationship between chronic inflammation and peripheral neurological involvement in RA.

2. Sjögren's Disease

In Sjögren's disease, peripheral neuropathy is one of the main extraglandular manifestations, with a prevalence of approximately 15%, according to the meta-analysis by Liampas et al.[8] In contrast to RA, in which neuropathy is generally silent, in pSS neuropathic symptoms, when present, are evident and may even precede the diagnosis of the disease. These include burning pain, allodynia, hypoesthesia, tingling, loss of balance, and muscle weakness. Pain tends to worsen at night and can be disabling. Motor involvement, although less common, is also reported in more severe cases. The most frequent clinical form is distal axonal polyneuropathy (80%), length-dependent, usually painful, with paresthesia, allodynia, and hypoesthesia, especially in the lower limbs. Next, sensory neuronopathy (20%) stands out, presenting asymmetry and not length-dependence, with sensory ataxia and sometimes cerebellar involvement. Less frequent forms include multiple mononeuropathy, pure motor neuropathies, and polyradiculoneuropathies.

Regarding the presence of small fiber neuropathies (SFN), associated with burning pain, pruritus, autonomic dysfunction, and allodynia, few studies have investigated their occurrence in SD using different diagnostic approaches. According to Liampas et al.[8], the prevalence of pure SFN in patients with SD was 9.2% in the only study that followed well-established criteria. Its manifestation is associated with a higher prevalence of restless legs syndrome and also with reduced electrochemical conductance of the skin, revealing possible concomitant involvement of both large and small nerve fibers. This neuropathy is difficult to study due to the challenges in its diagnosis, which is established by determining small fiber density in a skin biopsy, as reported by Scofield et al.[9], since electroneuromyography is normal in these cases.

Peripheral mononeuropathies have also been observed in SD, such as carpal tunnel syndrome, with a prevalence of 12.8%, and cranial neuropathies, mainly of the trigeminal nerve, which affect 3.9% of

patients with SS, possibly related to a generalized ganglionopathy.[8]

The results of the study by Scofield et al.[9] revealed a significant association between certain immunological factors and the occurrence of peripheral sensory neuropathy in patients with SD, suggesting a possible pathogenic role of these markers. The frequency of neuropathy was substantially higher (66.7%) among individuals with anti-Ro (SSA) and anti-La (SSB) autoantibodies detected by immunodiffusion, compared to only 25% in those without these antibodies ($p = 0.0036$). Furthermore, even the isolated presence of anti-Ro was significantly associated with neuropathy (48.1% versus 23%, $p = 0.018$). These findings reinforce the clinical relevance of identifying these autoantibodies in the context of correlating them with neuropathic involvement, particularly when assessed by immunodiffusion. However, more sensitive methods, such as ELISA and BioPlex, did not show a significant correlation with neuropathy, which may indicate differences in the specificity of the methods or in the detection of distinct autoantibody subpopulations. This suggests the need for standardization and caution in the interpretation of serological tests when evaluating neuropathies, although they should certainly be taken into account due to their aforementioned relevance.

The study also excluded any association between serum vitamin B12 levels and the presence of neuropathy in SD, suggesting that this is not a relevant factor in this context.

3. Systemic Lupus Erythematosus

In systemic lupus erythematosus, there was relevant variation in the prevalence of PN among the studies analyzed, due to the difficult characterization stemming from the diversity of clinical presentations and the multiplicity of possible causes. While Florica et al.[4] reported 13.5%, Fargetti et al.[10], using more restrictive exclusion criteria and electrophysiological confirmation, considering only cases attributed exclusively to SLE, reported only 1.8%. Most cases manifested within the first five years of the disease, with an emphasis on early-onset cases (36.6%) (within the first year), which showed high inflammatory activity (SLEDAI = 21.3) compared to late-onset cases (SLEDAI = 3.9). The most commonly observed pattern was axonal sensorimotor polyneuropathy, but there were also reports of mononeuropathies, polyradiculoneuropathy, and cranial neuropathy.

Regarding symptoms, the most frequently reported were paresthesia (81.6%), pain (57.9%), muscle weakness (52.6%), and gait disturbances (42.1%). All patients presented lower limb involvement, and about one-third (31.5%) also showed upper limb involvement. Electroneuromyography revealed a predominance of axonal lesions, with mixed or demyelinating patterns in some cases. Regarding therapy, all patients received corticosteroid treatment, with dose escalation (76.3%) and pulse therapy (55.2%) required in most cases. Immunosuppressants were initiated in 97.3% of patients, particularly intravenous cyclophosphamide (50%) and azathioprine (42.1%). There was a high treatment response, with complete or partial clinical remission after one year in the majority of cases (92.1%), and maintenance of this response after five years, with 89.3% still in remission. These findings demonstrate the potential reversibility of neuropathic involvement in SLE, provided that there is early recognition and adequate treatment.

The Canadian study by Florica et al.[4] evaluated 207 patients with PN among 1,533 patients with SLE, with 60.3% of PN cases attributed to SLE. The most common clinical form was sensory or sensorimotor polyneuropathy, with a predilection for distal and asymmetric involvement (59%), mainly of the sural, fibular, median, and ulnar nerves. Less common forms included multiple mononeuropathy (9.2%), cranial neuropathy (12.5%), CIDP (5.3%), and AIDP (1%). Electroneuromyography showed axonal neuropathy in

74% of patients with PN attributed to SLE.

Patients with PN attributed to SLE presented higher disease activity (SLEDAI-2K = 11) compared with patients with PN from other causes (SLEDAI = 5). Multiple mononeuropathy was exclusive to cases attributed to SLE, reinforcing its association with active vasculitis. The most common treatment was oral corticosteroids or pulse therapy, with a favorable clinical response in about 66% of patients.

Both studies reinforce that PN in SLE may arise at different stages of the disease and is frequently associated with systemic inflammatory activity, especially when onset is early. Immunological markers associated with PN include lymphopenia, cutaneous vasculitis, and anti-Sm positivity, particularly in the Brazilian study.

4. Systemic Vasculitides

In systemic vasculitides, peripheral neuropathy is observed as one of the most prevalent and striking manifestations, particularly in polyarteritis nodosa (PAN), ANCA-associated vasculitis (granulomatosis with polyangiitis – GPA, microscopic polyangiitis – MPA, and eosinophilic granulomatosis with polyangiitis – EGPA), and cryoglobulinemic vasculitis associated with HCV. Vasculitic neuropathy may also occur in a form limited to the peripheral nervous system, called nonsystemic vasculitic neuropathy (NSVN). Graf and Imboden[5] described PN in up to 85% of patients with PAN, 60–80% in EGPA, 40–50% in MPA, and 20–25% in GPA. The most dominant form is multiple mononeuropathy, with asymmetric, acute or subacute, painful involvement, especially of lower limb nerves such as the deep fibular (causing foot drop) and upper limb nerves such as the ulnar. Pathophysiologically, there is involvement of transmural inflammation of the vasa nervorum with fibrinoid necrosis, perivascular infiltration, and vascular occlusion. In cases of NSVN, the inflammatory process is restricted to the peripheral nerve, often without systemic symptoms, making diagnosis difficult. It is estimated that 25% of vasculitic neuropathies are NSVN, with a more insidious and progressive presentation and a less aggressive course.

The study by Rodrigues et al.[11] detailed clinical cases of vasculitic neuropathy, including HBV-associated PAN, MPA, EGPA, and vasculitis without specific definition. Most patients presented progressive asymmetric sensorimotor deficits, characterizing multiple mononeuropathy evolving into distal asymmetric polyneuropathy. In all cases, electroneuromyography revealed an asymmetric axonal pattern, with skin or nerve biopsy being fundamental for diagnostic confirmation. Typical histological findings included asymmetric loss of nerve fibers, perivascular inflammatory infiltrate, fibrinoid necrosis, and signs of vascular recanalization. The presence of constitutional symptoms (weight loss, low-grade fever, asthenia), skin lesions, and systemic signs aided in etiological differentiation. Regarding treatment, corticosteroid therapy with immunosuppressants (cyclophosphamide or azathioprine) was used, in addition to antivirals in HBV-associated vasculitis. Clinical response was favorable in most patients, with significant neurological recovery within weeks or months. The study reinforces the importance of early etiological characterization of PN, as therapeutic approaches vary according to the underlying vasculitis.

CONCLUSION

Peripheral neuropathies are frequent in IMRDs (rheumatoid arthritis, Sjögren's disease, systemic lupus erythematosus, and systemic vasculitis), and even with differences in the pathophysiology of each disease, they present certain similarities, with varying degrees of pain, paresthesia, weakness, and sensory or motor dysfunctions. Subclinical presentation may occur in rheumatoid arthritis, which suggests investigation even in oligosymptomatic patients. In SD, neuropathy may precede sicca

symptoms, in which case the investigation of anti-Ro antibodies is important for suspicion. In SD, small fiber neuropathy also stands out, which is difficult to diagnose, with normal electroneuromyography, requiring skin biopsy. In systemic lupus erythematosus, PN occurs early, associated with high inflammatory activity, with good prognosis when treated promptly. Finally, in systemic vasculitides, multiple mononeuropathy is the most common and severe form, with biopsy being essential for diagnosis and therapeutic response generally favorable. The recognition and diagnosis of neuropathy in IMRDs are essential for early treatment and improved quality of life for patients.

REFERENCES

1. De Souza JM, Trevisan TJ, Sepresse SR, Londe AC, Franca Junior MC, Appenzeller S. Peripheral Neuropathy in Systemic Autoimmune Rheumatic Diseases-Diagnosis and Treatment. Pharmaceuticals (Basel). 2023;16(4).

2. Li Y, Jiang L, Zhang Z, Li H, Jiang L, Wang L, Li Z. Clinical characteristics of rheumatoid arthritis patients with peripheral neuropathy and potential related risk factors. Clin Rheumatol. 2019;38(8):2099-107.

3. Lima RFRd, Marques RS, Pugliesi A. Doença de Sjögren extraglandular: compreendendo as manifestações do Neuro-Sjögren. Revista Paulista de Reumatologia. 2024;23:36-40.

4. Florica B, Aghdassi E, Su J, Gladman DD, Urowitz MB, Fortin PR. Peripheral neuropathy in patients with systemic lupus erythematosus. Semin Arthritis Rheum. 2011;41(2):203-11.

5. Graf J, Imboden J. Vasculitis and peripheral neuropathy. Current Opinion in Rheumatology. 2019;31:40-5.

6. Kararizou E, Davaki P, Karandreas N, Davou R, Vassilopoulos D. Nonsystemic vasculitic neuropathy: a clinicopathological study of 22 cases. J Rheumatol. 2005;32(5):853-8.

7. Kaeley N, Ahmad S, Pathania M, Kakkar R. Prevalence and patterns of peripheral neuropathy in patients of rheumatoid arthritis. J Family Med Prim Care. 2019;8(1):22-6.

8. Liampas A, Parperis K, Erotocritou MF, Nteveros A, Papadopoulou M, Moschovos C, et al. Primary Sjogren syndrome-related peripheral neuropathy: A systematic review and meta-analysis. Eur J Neurol. 2023;30(1):255-65.

9. Scofield AK, Radfar L, Ice JA, Vista E, Anaya JM, Houston G, Lewis D, Stone DU, Chodosh J, Hefner K, Lessard CJ, Moser KL, Scofield RH. Relation of sensory peripheral neuropathy in Sjögren syndrome to anti-Ro/SSA. J Clin Rheumatol. 2012 Sep;18(6):290-3.

10. Fargetti S, Ugolini-Lopes MR, Pasoto SG, Seguro LPC, Shinjo SK, Bonfa E, Borba EF. Short- and Long-Term Outcome of Systemic Lupus Erythematosus Peripheral Neuropathy: Bimodal Pattern of Onset and Treatment Response. J Clin Rheumatol. 2021;27(6S):S212-S6.

11. Rodrigues R, Branco M, Silva R, Ruano L, Fontão L, Lopes M, Scigliano H, Taipa R, Pires MM, Santos C. Peripheral neuropathy in systemic vasculitis and other autoimmune diseases - a report of five cases emphasizing the importance of etiologic characterization. eNeurologicalSci. 2020 Sep 11;21:100272.

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