

Clinical Practice Guideline for Evaluation and Management of Peripheral Nervous System Manifestations in Sjögren's Disease

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Objective. Sjögren's disease is an autoimmune disorder that can impact multiple organ systems, including the peripheral nervous system (PNS). PNS manifestations, which can exist concurrently, include mononeuropathies, polyneuropathies, and autonomic nervous system neuropathies. To help patients and providers in the decision-making process, we developed an evidence-based clinical practice guideline for the evaluation and management of PNS manifestations in patients with Sjögren's disease.

Methods. A Topic Review Group, comprising experts in rheumatology, neurology, and guideline methodology, developed Patient, Intervention, Comparison, and Outcome questions and conducted a systematic review to identify the current best evidence on management of PNS manifestations of Sjögren's disease. PubMed and Embase were searched for evidence published up to July 22, 2025. Literature screening, data extraction, and critical appraisal were performed in duplicate. Six case series, one retrospective cohort, and two prospective cohort studies lacking a comparison group met the inclusion criteria.

Results. We developed an aligned nomenclature of PNS terms that can be used across disciplines, 31 good practices for evaluation of suspected PNS manifestations, and 20 evidence-based treatment recommendations, the latter of which were rated as conditional or strong based on the Grading of Recommendations Assessment, Development, and Evaluation methodology. Because of the scarcity of high-level evidence, this guideline predominantly derives from expert opinion.

Conclusion. This clinical practice guideline on PNS manifestations of Sjögren's disease provides clinicians a rigorous, evidence-based resource, developed through an expert consensus-based process, for the assessment, diagnosis, and treatment of peripheral neuropathy in patients with Sjögren's disease. Recommendations were rated as strong when the benefits significantly outweighed potential harms, creating a scenario in which most patients would prefer the advised action.

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SIGNIFICANCE & INNOVATIONS

- An evidence-based clinical practice guideline was developed to assist in the evaluation and management of peripheral nervous system (PNS) manifestations in Sjögren's disease, including mononeuropathies, polyneuropathies, and autonomic nervous system (ANS) neuropathies.
- A multidisciplinary team of rheumatologists, neurologists, and guideline methodologists conducted a systematic review and developed standardized nomenclature, 31 good practices for evaluation, and 20 treatment recommendations for PNS and ANS manifestations in Sjögren's disease.
- The guideline also uses a unified terminology and framework to facilitate multidisciplinary collaboration and improve patient outcomes in the management of Sjögren's disease-related PNS manifestations.
- The available evidence, primarily from case series and cohort studies without comparison groups, necessitated reliance on expert consensus for many recommendations, although strong recommendations were made when potential benefits clearly outweighed risks.

INTRODUCTION

Sjögren's disease (SjD), a complex autoimmune disorder, impacts multiple organ systems including the peripheral nervous system (PNS). Reports on the prevalence of PNS manifestations in SjD range from 8% to 60%.^{1–11} Evidence suggests the prevalence of PNS manifestations in SjD is significantly underestimated.¹² Contributing to underreporting are neuropathies that are asymptomatic in early stages and/or underrecognized as a feature of SjD.⁹ The mean age of diagnosis of PNS manifestations is 59 years, often preceding the SjD diagnosis in up to 90% of patients.^{2,6,11,13} The consequences of PNS manifestations in SjD significantly diminish patients' quality of life (QOL) while presenting diagnostic and management challenges for health care providers.^{9,14–17}

PNS manifestations encompass a spectrum of neurologic abnormalities, including cranial neuropathies (trigeminal neuropathy and facial neuropathy), polyneuropathies (large fiber neuropathy, small fiber neuropathy, and demyelinating polyradiculoneuropathy), and autonomic nervous system (ANS) neuropathies (postural tachycardia, orthostatic hypotension, and autonomic dysfunction).^{2,11,18–20} Additionally, some patients with SjD may develop vasculitic neuropathy or ganglionopathy, which can be rapidly debilitating.^{21,22} Autonomic disorders contribute to reduced QOL and function, which can be overlooked or misdiagnosed as anxiety or functional disorders. Patients with SjD may experience various multiple peripheral neuropathies concurrently. Understanding the specific subtype of peripheral neuropathy and the underlying pathophysiologic mechanisms is essential

to effectively diagnosing and treating comorbid conditions. Therefore, a collaborative, multidisciplinary approach to patient evaluation involving neurologists, rheumatologists, and other specialists proves invaluable.

In response to the growing recognition of PNS involvement and management uncertainties, the Sjögren's Foundation convened a Topic Review Group (TRG) of neurologists and rheumatologists to develop an evidence-based guideline on PNS manifestations in patients with SjD. The guideline incorporates aligned nomenclature of PNS terms that can be used across disciplines, which was used in developing good practices for the evaluation of suspected PNS manifestations and evidence-based treatment recommendations. This guideline provides rheumatologists, neurologists, primary care providers, and other members of the health care team with an evidence-based and consensus-based resource for collaborative assessment, diagnosis, and treatment of PNS manifestations in SjD.

METHODS

We developed this evidence-based guideline based on the methodology of the American College of Rheumatology (ACR),²³ which was previously used for other Sjögren's Foundation guidelines.^{24,25} We used principles of the American Society of Clinical Oncology, the US Preventive Services Taskforce, and the American Dental Association to formulate the methodology.^{26–29} During the process of developing the recommendations, we transitioned to the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system for rating the strength of each recommendation.^{29–32}

We assembled the PNS TRG, consisting of six neurologists (AD, EDS, BPG, SM, PPP, and AV), six rheumatologists (RF, MCB, SD, JKK, GN, and DJW), one member dual-specialized in neurology and rheumatology (GS), and one patient representative (KMH). We invited neurologists with specific experience in SjD and who collaborate with rheumatologists in managing their patients with SjD. Leadership for the PNS TRG included coleaders (a rheumatologist [RF] and a neurologist [AD]), guideline oversight committee members (SC, NC, and RHS), and a guideline expert (JF-H).

The Sjögren's Foundation guideline policy is that TRG members with a direct financial conflict of interest (COI), defined as receiving funds directly from industry related to a treatment or procedure addressed in the guideline, must be recused from engaging in that part of the project, and no more than 49% of the TRG members can have a direct COI. All TRG members signed a COI and disclosed any potential financial conflicts at the project's onset, which was periodically updated. The guideline leadership (SC, NC, RHS, RF, and AD) managed conflicts accordingly. Panelists were aware of disclosures. No direct COIs were identified, and only 40% had indirect COI disclosures

(defined as engagements with industry not directly related to a treatment or procedure addressed in this guideline).

Our systematic review protocol is registered with International Platform of Registered Systematic Review and Meta-analysis Protocols (protocol 2025110020). The TRG defined systematic review parameters, inclusion/exclusion criteria, endpoints (Supplementary Material 1), and the Patient, Intervention, Comparison, and Outcome (PICO) questions. The overarching therapy PICO question for the treatment recommendations was as follows: in patients with SjD and PNS manifestations (mononeuropathies, polyneuropathies, and vasculitis) and ANS manifestations, which therapies are most effective in improving neurologic symptoms or function? The overarching evaluation and monitoring PICO question for the good practice statements was as follows: in patients with SjD and suspected PNS or ANS manifestations, which diagnostic and monitoring strategies are clinically useful for diagnosis and tracking disease progression or treatment response? The full set of PICO questions is available in Supplementary Material 2. We searched PubMed for articles published in English between January 1, 1990, and May 1, 2023 (Supplementary Material 3), and updated the search through July 22, 2025. An Embase literature search was conducted through July 22, 2025. At least two TRG members screened titles and abstracts, and subsequently full-text articles, in duplicate, with discrepancies resolved by mutual discussion. Two TRG members conducted data extraction and quality assessment in duplicate, with discrepancies resolved via discussion. The quality assessment involved rating the overall quality of each study according to criteria related to study design and the risk of bias used in recommendation development.

The TRG engaged in collaborative efforts to unify terminology and define the nomenclature used to describe PNS manifestations. The unified nomenclature provided the structure and categories for the subsequent good practices and treatment recommendations.³³

The TRG intended to develop evidence-based recommendations for the evaluation of PNS manifestations in patients with SjD. However, the identified studies provided information on which tests were used but did not address sensitivity, specificity, positive predictive values, negative predictive values, and likelihood ratios in an SjD population. Thus, we used a good practices approach for the evaluation portion of the project. For this, we categorized the most common diagnostic tests reported and used expert opinion to develop good practice statements for evaluating patients with SjD who present with PNS symptoms.

The TRG drafted recommendations on therapies for patients with SjD with PNS manifestations. Each recommendation was rated based on two factors: (1) the level of the available evidence and (2) the strength of the recommendation based on confidence that the recommendation offers the best current guidance for practice (Supplementary Material 4). For each recommendation, the TRG evaluated the balance between desirable and

undesirable effects, the quality of evidence, patients' values and preferences, and feasibility, including costs.³⁴ Based on GRADE methodology, a recommendation was considered strong, even if the quality of evidence is low or very low, when the potential benefit significantly outweighed the potential harm, aligning with the majority of patients' preferences, and when the potential harms of inaction were unacceptable to patients. A conditional recommendation was given when the potential benefit slightly outweighed the potential harm, leading to a more varied preference among patients. We followed an anonymous Delphi-type process to ascertain the level of agreement from the Consensus Expert Panel (CEP)—composed of 69 members, including 39 rheumatologists, 20 neurologists who actively work with patients with SjD, and 10 additional members, all of whom were patients with SjD or family of patients (Appendix A; Supplementary Material 5). At least 75% agreement was required for inclusion in the manuscript, with repeated rounds of revision by the TRG and voting until the consensus threshold was met. The formal consensus process was deemed critical in view of the lack of high-quality evidence.

The guideline process adhered to the Appraisal of Guidelines for Research and Evaluation II instrument (Supplementary Material 6). For the systematic review component, we followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses checklist (Supplementary Material 7). This work did not require institutional review board approval.

RESULTS

Systematic review

The TRG developed 102 clinical questions on SjD-related PNS manifestations (Supplementary Material 2). These questions spanned mononeuropathy, polyneuropathy, and autonomic neuropathy and addressed diagnostic/evaluation procedures or therapeutic interventions.

The literature search yielded 13,848 articles. After duplicates were removed, 11,174 articles remained. Of these, 10,918 articles were excluded based on title/abstract review, and 247 were excluded based on full-text review (Figure 1). A list of studies excluded at the full-text review stage, with the reason for exclusion, is provided in Supplementary Material 8.

Nine studies that addressed therapeutic interventions met the inclusion criteria and were used to develop evidence-based recommendations. Six were case series, one was a retrospective cohort study, and two were prospective cohort studies lacking a comparison group. Data extraction and quality assessment are provided in Supplementary Material 9. Because of the lack of high-quality evidence, most of the treatment recommendations are based on expert opinion, informed by balancing benefits, harms, and patient values.

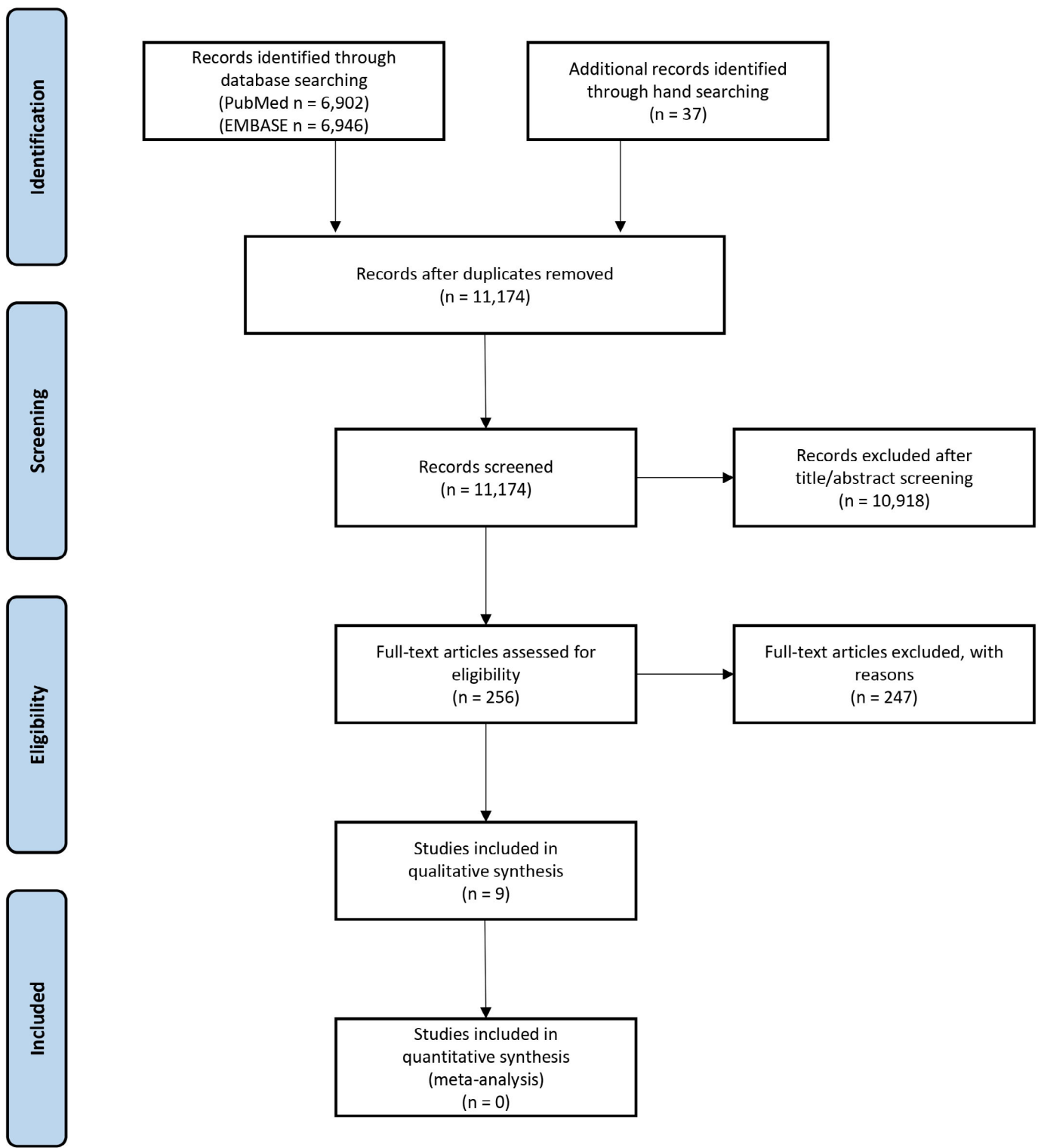


Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) diagram.

PNS nomenclature and definitions

The guideline development process highlighted a critical communication gap between rheumatology and neurology clinicians because of differing terminologies used by the two

specialties that posed challenges to collaboration in clinical management and guideline formulation.^{5,6,35} We aimed to bridge this gap by establishing consensus on the nomenclature among neurologists and rheumatologists for PNS involvement in SjD. A unified nomenclature that formed the

basis of the guideline is available in Supplementary Material 10.³³

Good practices and recommendations

The most common tests reported for evaluation of PNS manifestations in patients with SjD were used to inform good practice statements (Supplementary Material 11). Evidence from the systematic review informed the recommendations (Supplementary Material 9). Four rounds of anonymous consensus Delphi voting occurred, with modifications to statements made based on comments received from the survey as well as journal reviewers. There was 74.3% to 95.7% participation in each round (Supplementary Material 12).

Good practices and recommendations for each PNS category are provided below, and a decision tree is provided in Supplementary Material 13. The rationales for each good practice as well as the rationale, balance of desirable and undesirable effects, quality of evidence, values and preferences, and feasibility for each recommendation are provided in Supplementary Material 14. Additionally, Table 1 provides a list of questions for the clinician to ask when the diagnosis of SjD is being considered.

Mononeuropathy. Various mononeuropathies are seen in individuals with SjD, with a prevalence of 12% to 50%.¹¹ These include trigeminal neuralgia, trigeminal neuropathy, facial

neuropathy, and upper-extremity neuropathies. Trigeminal neuralgia, characterized by severe facial pain, and trigeminal neuropathy, presenting with numbness or tingling, are common manifestations. Facial neuropathy results in weakness of the muscles of the upper and lower face. Upper-extremity neuropathies include median neuropathy at the wrist (carpal tunnel syndrome) and ulnar neuropathy.

When considering SjD as the etiology of a mononeuropathy, it is important to exclude alternative causes, such as demyelinating lesions, neoplastic involvement, or infectious processes. Prompt evaluation and treatment can mitigate symptoms and may prevent long-term complications. Management includes symptomatic medications for pain relief and treatments aimed at the underlying pathology and preserving nerve function. The specific treatment will be dictated by the type and severity of the mononeuropathy to optimize outcomes and enhance QOL. Good practices and recommendations for the evaluation and management of specific mononeuropathies in patients with SjD are listed in Table 2, Supplementary Material 13A, and Supplementary Material 14.

Polyneuropathy. Polyneuropathies encompass a spectrum of neurologic disorders affecting peripheral nerves with a published prevalence of 10% to 20% in patients with SjD.^{11,35–38} These polyneuropathies include large fiber neuropathies, small fiber neuropathies, demyelinating polyradiculoneuropathies, ganglionopathies, and vasculitic neuropathies. The first step in

Table 1. Screening questions for potential Sjögren’s disease in patients with peripheral nervous system symptoms*

Systemic symptoms	Oral symptoms	Ocular symptoms
• Do you experience extreme fatigue? • Do your joints or muscles ache when you are not sick (arthralgias, myalgias)? • Do you have gland swelling in your face or along the jawline (swollen parotid and/or submandibular glands)? • Do you often experience heartburn, a sour or bitter taste in your mouth, especially when lying down or bending over? • Do you experience irritable bowel, frequent diarrhea, constipation, bloating, pain, or nausea (gastrointestinal dysmotility)? • Do you notice your fingers turning pale or blue in the cold (Raynaud disease)? • Do you have issues with memory, difficulty concentrating on tasks, or experiencing mental fatigue? • Do you have shortness of breath, a persistent cough, chest discomfort, or recurrent respiratory infections? • Do you have dryness of the vagina and/or is intercourse painful? (women) • Do you have chronic prostatitis? (men) • Do you have dry, itchy, or flaking skin?	• Does your mouth feel dry? • Do you need liquids to swallow dry foods? • Do you frequently sip/drink water? • Do you have a burning sensation in your mouth (burning mouth syndrome)? • Do you have painful sores or red patches at the corners of the mouth (angular cheilitis)? • Do you get frequent dental cavities and/or cavities at the gumline? • Do your teeth tend to chip, crack, and/or erode on the surfaces? • Do you experience gum inflammation or receding gums (gingivitis)? • Do foods high in acid or heat (spicy) feel irritating or painful?	• Do your eyes frequently feel dry, irritated, itchy, or painful? • Do you have a sensation that there might be a foreign body in your eye? • Are your eyes light-sensitive? • Do you frequently use eye drops for irritation or dryness? • Is your vision frequently blurry, or do you have unexplained vision changes? • Are you prone to getting eye infections? • Do your eyes feel “gummy” and eyelids stick together, especially upon waking?

* Symptoms should prompt further serologic evaluation and/or rheumatology consultation. An estimated 30% to 40% of patients with Sjögren’s disease are negative for the anti-SSA autoantibody, so a patient can still have Sjögren’s disease without a positive test for this antibody. Source: Reprinted with permission from the Sjögren’s Foundation. Copyright © 2024 Sjögren’s Foundation. All rights reserved.

Table 2. Good practices and recommendations for mononeuropathies in patients with Sjögren's disease

Screening good practices for mononeuropathies			
Good practice 1	Sjögren's patients with persistent facial pain or numbness should be assessed for dysfunction of cranial nerve V which can manifest as either trigeminal neuralgia or trigeminal neuropathy. - Physical examination: assess for numbness or pain of the face in the distribution of one or more branches of the trigeminal nerve, unilateral or bilateral, by testing for loss of light touch and either pin prick or cold sensation. - If the distribution of symptoms or findings on physical examination is consistent with trigeminal neuralgia or trigeminal neuropathy, a contrast-enhanced brain MRI with attention to brainstem/skull base and exiting cranial nerves should be performed to exclude structural or other etiologies of trigeminal dysfunction. - Consider electrophysiological testing, including blink reflex.		
Good practice 2	Sjögren's patients with unilateral facial weakness should be assessed for facial (cranial nerve VII) neuropathy. - Symptoms and physical examination findings: weakness of the upper and ipsilateral lower face. Upper facial weakness manifests as an inability to raise the eyebrows, inability to wrinkle the forehead, weakness of eye closure, or a widened palpebral fissure. Involvement of the lower facial muscles manifests as lip closure weakness, and facial droop. Some patients experience a loss of taste on the anterior two-thirds of the tongue and/or hyperacusis (hearing sensitivity) on the affected side. - Physical examination findings that may suggest an alternative etiology include coexisting cranial mononeuropathies, ear canal vesicles, neck stiffness, headaches, and fevers. These findings should prompt an evaluation for Lyme disease, herpes zoster, sarcoidosis, or variants of Guillain-Barré Syndrome (GBS), which may include lumbar puncture. - Perform a contrast-enhanced brain MRI with attention to the brainstem/skull base and exiting cranial nerves to exclude structural or other etiologies of facial neuropathy. - Consider electrophysiological testing with nerve conduction study, including blink reflex, and electromyography (EMG). - Facial neuropathy rarely presents bilaterally. If the manifestations are bilateral or recurrent, a thorough evaluation for more generalized systemic disease should be pursued.		
Good practice 3	Sjögren's patients who present with cranial mononeuropathies of other cranial nerves should be evaluated for alternative etiologies. Work-up should include referral to the appropriate specialist, such as a neurologist, otolaryngologist, ophthalmologist, speech pathologist, or gastroenterologist for evaluation.		
Good practice 4	Sjögren's patients with numbness, pain, or weakness of the hand should be evaluated for median or ulnar neuropathy. - Physical examination: Median neuropathy presents with sensory loss of the lateral (radial) three and ½ digits, atrophy of the thenar eminence, and weakness of thumb abduction. Ulnar neuropathy presents with sensory loss of the medial one and ½ digits, atrophy of intrinsic hand muscles, a “claw-hand” appearance, and weakness of the fingers. - If a median or ulnar neuropathy is suspected, consider electrodiagnostic studies and/or nerve ultrasound. - Referral to a hand surgeon or other specialist who evaluates and treats carpal tunnel syndrome or ulnar neuropathy should be considered. - If carpal tunnel surgical release is performed, a tenosynovial biopsy may be obtained to exclude amyloidosis.		
Therapy recommendations for mononeuropathies		Evidence rating	Strength of recommendation
Recommendation 1	For patients with Sjögren's-related trigeminal neuropathy, we recommend a short course of oral glucocorticoids for suspected acute inflammatory etiology.	Very Low	Conditional
Recommendation 2	For patients with Sjögren's-related trigeminal neuralgia (neuropathic pain), we recommend: - First-line treatment with carbamazepine or oxcarbazepine. - Second-line treatments, either as alternatives or adjuncts, may include gabapentin, pregabalin, baclofen, lamotrigine, lacosamide, or phenytoin.	Very Low	Conditional
Recommendation 3	For patients with Sjögren's who develop an acute facial neuropathy, we recommend: - A short course of glucocorticoid treatment for at least 10 days. - Consider the addition of valacyclovir.	Very Low	Conditional

patients with suspected polyneuropathy is to characterize the particular type, as further evaluation varies based on this classification (Supplementary Material 10).³³ Neurology expertise can assist in the accurate classification of polyneuropathies. The type and severity of polyneuropathy will determine treatment strategies. Again, early recognition and management can improve outcomes and QOL. Clinicians should be aware that polyneuropathies can be the initial manifestation of SjD, and identifying the etiology or the pathophysiologic cause of the neuropathy may be challenging. Good practices and recommendations for the evaluation and

management of polyneuropathies in patients with SjD are listed in Table 3, Supplementary Material 13B, and Supplementary Material 14.

Autonomic neuropathy. ANS impairment can occur in up to 50% of patients with SjD^{2,11} with the potential to affect various autonomic systems and ranging from mild to severe autonomic failure. Symptoms of ANS impairment in SjD typically result from autonomic neuropathy or ganglionopathy and may include orthostatic intolerance, vasomotor dysfunction, secretomotor

Table 3. Good practices and recommendations for polyneuropathies in patients with Sjögren's disease

Screening good practices for polyneuropathies		
Good practice 5	Recognize that neuropathies can have many causes, including Sjögren's disease. Potential alternative etiologies should be identified, such as diabetes, hypothyroidism, B-12 deficiency, monoclonal gammopathies, infectious etiologies, toxins, and medications. Labs related to these etiologies should be ordered.	
Good practice 6	Recognize that Sjögren's patients can present with a wide range of symptoms, including diverse presentations of peripheral neuropathies. For abnormal sensations suspected of being neuropathic, ask the following questions to characterize the neuropathy: 1. Do you have numbness, tingling, pins and needles or buzzing sensation, burning pain, other abnormal sensations, and/or weakness? 2. Are the symptoms symmetric or asymmetric? 3. Where is it worse—hands, feet, or all over? 4. Where did you notice it first? (distal onset?) 5. How long have symptoms been present? 6. Do you have any gait disturbances/abnormalities? Additional questions that may be useful in screening are: 7. Was the onset of neuropathy gradual or abrupt? 8. How have symptoms changed? (worse? persistent? intermittent? more proximal?) 9. Do symptoms awaken you from sleep? 10. Under what circumstances did symptoms begin (i.e. Did they start before or after new medication or infection?) 11. Have there been any exposures to medications, supplements, complementary and alternative medicine, or occupational exposures that could be neurotoxic?	
Good practice 7	Sjögren's patients who have symptoms suggesting a peripheral neuropathy should have a comprehensive neurological examination.	
Good practice 8	For Sjögren's patients who have history and physical examination findings suggesting a peripheral neuropathy, consider referral to a neurologist.	
Good practice 9	For Sjögren's patients with persistent neuropathic symptoms, electrodiagnostic studies (nerve conduction studies [NCS] and electromyography [EMG]), should be performed by an experienced electromyographer. The study is best timed at 1-4 weeks from the onset of symptoms, depending on the type, progression, and severity of the neuropathy.	
Good practice 10	For Sjögren's patients with suspected small fiber neuropathy, consider evaluation of epidermal nerve fiber density with punch skin biopsy to support this diagnosis.	
Good practice 11	For Sjögren's patients with suspected peripheral neuropathy and electrodiagnostic studies (NCS/EMG) warranting additional evaluation or when there is no response to treatment, the following should be considered: • Fat pad and/or minor salivary gland biopsy if amyloid is suspected. • Nerve and muscle biopsy if vasculitis, amyloid, or other infiltrative processes are suspected. • Lumbar puncture should be reserved for assessing suspected cases of inflammatory demyelinating polyradiculoneuropathy (including acute inflammatory demyelinating polyradiculoneuropathy [AIDP] and chronic inflammatory demyelinating polyradiculoneuropathy [CIDP]), ganglionopathy, neoplastic diseases, or infection.	
Good practice 12	For Sjögren's patients with gait disturbance due to weakness, sensory loss, ataxia, or imbalance, consider assessments for gait and balance training and for assistive devices by physical therapy, physical medicine rehabilitation (PMR), and/or other related healthcare professionals.	
Good practice 13	For Sjögren's patients with polyneuropathy with neuropathic pain who have not benefited from initial treatments for neuropathic pain, referral to a neurologist and/or a pain specialist may be warranted.	
Good practice 14	For Sjögren's patients with confirmed polyneuropathy, treatment choice depends on the type and location of neuropathy, disease severity, and degree of systemic involvement (e.g., vasculitis).	
Good practice 15	Vasculitic neuropathy should be considered in Sjögren's patients with polyneuropathy and the following clinical scenarios: Acute, painful, rapid onset, or stepwise progression of neurological deficit, clinical and electromyographic evidence of multiple mononeuropathies, evidence of single-organ or systemic vasculitis, or immunologically active disease as defined by cryoglobulinemia, hypocomplementemia, monoclonal gammopathy, positive rheumatoid factor (RF), and elevated inflammatory markers.	
Good practice 16	We recognize the technical difficulties in correctly identifying serum cryoglobulins. Therefore, other surrogate markers associated with the presence of cryoglobulinemia (i.e., positive RF and C4 hypocomplementemia), in the appropriate clinical context, may also be suggestive in those negative for serum cryoglobulins.	
Good practice 17	For Sjögren's patients with a suspicion of vasculitic neuropathy, EMG and NCS should be performed. The timing of this study is based on the severity and tempo of presentation, typically 1-4 weeks after the onset of symptoms.	
Good practice 18	For Sjögren's patients with clinical and electrodiagnostic findings suggestive of a vasculitic etiology, a nerve and muscle biopsy is warranted.	
Good practice 19	For Sjögren's patients with vasculitic neuropathy with cryoglobulinemia, repeat testing for serum cryoglobulin as a biomarker for therapeutic response, along with complement C4 and RF as markers of biological response.	
Therapy recommendations for polyneuropathies		
	Evidence rating	Strength of recommendation
Recommendation 4	Very Low	Strong ^a
For Sjögren's patients with neuropathic pain in the setting of a peripheral neuropathy, we recommend first-line symptomatic treatment that includes gabapentin, pregabalin, or serotonin-		

(Continued)

Table 3. (Cont'd)

Recommendation 5	norepinephrine re-uptake inhibitors (SNRIs), depending on individual patient tolerance, comorbidities, and side effect risk. For Sjögren's patients with neuropathic pain in the setting of a peripheral neuropathy who are not helped by first-line treatments, we recommend sodium channel blocking agents (carbamazepine, oxcarbazepine, lamotrigine) or tricyclic antidepressants (nortriptyline and desipramine may be better tolerated). For patients who are refractory to, or intolerant of, first- and second-line neuropathic pain medications, referral to a neurologist and/or pain management specialist may be warranted.	Very Low	Conditional
Recommendation 6	For patients with Sjögren's-related polyneuropathy with a progressive or aggressive presentation, we recommend an empiric glucocorticoid treatment trial. The specific dose and length of treatment with glucocorticoids will depend on the individual circumstances. Factors such as the response to treatment, and the potential side effects of the medication should be considered when determining the appropriate duration of treatment.	Very Low	Conditional
Recommendation 7	For Sjögren's-related immune-mediated non-vasculitic large fiber polyneuropathy, we recommend considering intravenous immunoglobulin (IVIG).	Very Low	Conditional
Recommendation 8	For Sjögren's-related immune-mediated small fiber neuropathies that do not respond to symptomatic treatment and have recent onset, severe, or progressive disease, we recommend considering treatment with IVIG for a duration of at least 3 months. Treatment with subcutaneous immunoglobulin (SCIG) is an alternative.	Very Low	Conditional
Recommendation 9	For Sjögren's patients with acquired chronic demyelinating polyradiculoneuropathy, we recommend offering standard treatments for CIDP.	Very Low	Strong ^b
Recommendation 10	For Sjögren's patients with sensory ganglionopathy who manifest significant symptoms, we recommend instituting aggressive and immediate treatment with moderate to high dose glucocorticoids and immunomodulatory/immunosuppressive therapy.	Very Low	Strong ^c
Recommendation 11	For Sjögren's patients with high suspicion of vasculitic neuropathy, we recommend immediate initiation of high-dose systemic glucocorticoids with rituximab as the first line of therapy.	Very Low	Strong ^d
Recommendation 12	For Sjögren's patients with vasculitic neuropathy unresponsive to first-line therapy with glucocorticoids and rituximab, we recommend glucocorticoids and cyclophosphamide as a second line of therapy.	Very Low	Conditional
Recommendation 13	For Sjögren's patients with vasculitic neuropathy with cryoglobulinemia, we recommend maintenance with immunomodulatory therapy.	Very Low	Conditional
Recommendation 14	For Sjögren's patients with vasculitic neuropathy and persistent neuropathic pain, we recommend symptomatic management as discussed in Therapy Recommendations 4 and 5: Polyneuropathies.	Very Low	Strong ^a
Recommendation 15	We do not recommend IVIG as a first line of therapy for Sjögren's patients with vasculitic neuropathy.	Very Low	Conditional

^a This recommendation is rated strong based on a balance of benefits vs harms because most patients would choose the intervention to reduce pain and improve quality of life. Additionally, pain management is the standard of care.

^b This recommendation is rated strong because most patients and clinicians would use standard treatments for CIDP based on strong literature for CIDP in general, because the benefits likely outweigh the risks.

^c This recommendation is rated strong because the profound ataxia of ganglionopathy can be severe, rapidly progressing, and disabling if not treated. Thus, the benefits outweigh the risks of not treating this condition.

^d This recommendation is rated strong because, given the risk of irreversible axonal loss and chronic disability, most physicians and patients would opt for initial immune therapy. Because rituximab has a generally lower risk of adverse events than cyclophosphamide, it is recommended as a first-line treatment for vasculitic neuropathy.

dysfunction, gastrointestinal disturbances, bladder dysfunction, and pupillomotor dysfunction. Recognizing autonomic signs and symptoms in SjD can be challenging because of their nonspecific nature, overlapping symptoms with other aspects of the disease, or patients' adaptation to longstanding symptoms. Referral to specialists such as neurologists, gastroenterologists, cardiologists, urologists, or ANS testing centers may be necessary for comprehensive evaluation and management. Differential diagnostic considerations for autonomic neuropathy in SjD include vitamin deficiencies, autoimmune dysautonomia, and other systemic diseases. A skin biopsy for sweat gland nerve fiber density may aid in diagnosing autonomic small fiber neuropathy. Management strategies for autonomic dysfunction include lifestyle modifications, pharmacotherapy, and, in severe cases, immunomodulatory therapy. Multidisciplinary collaboration is crucial for optimal management and improved QOL for patients with SjD with autonomic neuropathies. Good practices and recommendations for evaluation and management of ANS neuropathies in SjD are listed in Table 4, Supplementary Material 13C, and Supplementary Material 14.

DISCUSSION

The PNS manifestations of SjD include mononeuropathies, polyneuropathies, and ANS neuropathies. The Sjögren's Foundation convened a panel of experts (the PNS TRG) to develop this guideline based on the best available evidence to improve the evaluation and management of these manifestations. Thirty-one good practice evaluation statements were made. Of the 20 recommendations, 14 are rated as conditional and 6 are rated as strong. Clinicians should approach each recommendation with a balanced view, in discussion with the patient, weighing the potential benefits against possible harm as part of shared decision-making.

This guideline is generally consistent with the PNS domain of the EULAR Sjögren's Syndrome Disease Activity Index (ESSDAI) addressed in the 2020 EULAR recommendations for the management of SjD, similarly drawing on glucocorticoids, immunomodulatory agents, and biologic therapies, while providing distinct additional guidance.³⁹ Distinctions include the following: (1) classification of neuropathies aligned between rheumatology and neurology (Supplementary Material 10)³³; (2) localization-specific guidance and addresses autonomic neuropathy, an important but underrecognized dysfunction in SjD; (3) prioritization of specific immunosuppressive agents based on an integration of available evidence and expert neurorheumatology opinion; (4) guidance on ANS dysfunction, small fiber neuropathy, mononeuropathies, and neuropathic pain; and (5) guidance on the use of B cell-targeted therapy, rituximab, for patients with severe or refractory disease, including vasculitic neuropathy and severe, progressive, and treatment-resistant immune-mediated autonomic neuropathy. In alignment with this, two late-phase clinical trials have recently demonstrated positive results for B cell-

targeted therapies in SjD, with ianalumab (anti-B cell activating factor receptor antibody) showing efficacy in Phase 3 and telitacicept (a B lymphocyte stimulator/a proliferation-inducing ligand dual inhibitor approved for systemic lupus erythematosus in China) also meeting its primary endpoints.⁴⁰

We used consensus expert opinion, informed by commonly employed evaluation tests, to develop good practices for evaluating patients with SjD presenting with PNS manifestations. According to the GRADE working group, good practice statements address topics that panels consider essential but are not suitable for formal evidence-quality ratings because they are supported by a substantial body of indirect evidence, the formal appraisal of which would represent an inefficient use of the panel's resources.^{41,42} These good practices emphasize the need for a comprehensive diagnostic evaluation to characterize PNS disorders and exclude other potential etiologies. Early detection and the correct diagnosis are essential to provide the best chance to halt progression and improve QOL.⁹

The treatment recommendations are partially informed by six case series studies and three observational studies, underscoring the scarcity of high-level evidence in this area. Because of the lack of evidence, most of the treatment recommendations are based on expert opinion, informed by balancing benefits, harms, and patient values. As stated in two articles by Djulbegovic and Guyatt (2017 and 2019),^{43,44} all clinical guidelines should be evidence-based, even when the evidence is of very low quality—as in the case of this guideline. They emphasize that evidence-based medicine involves using the best available evidence—whether from randomized trials, observational studies, case series, or the experience of individual clinicians—to guide clinical decision-making and practice guidelines. Distinguishing between “evidence-based” and “consensus-based” guidelines is misleading because interpreting any evidence requires consensus among experts. Therefore, consensus is integral to interpreting and applying evidence in clinical practice, and all guidelines inherently require both evidence evaluation and consensus regardless of the available evidence. Our guideline aligns with these principles, systematically reviewing and assessing evidence, and balancing benefits, harms, and patient values, to provide practical recommendations for managing SjD.

The TRG faced situations in which the available evidence was of low or very low quality. Despite this, strong recommendations reflect the expert consensus on what is most beneficial for patients (even with limited evidence), while noting any uncertainties about the evidence quality, after careful consideration of potential benefits and harms, patient values, and the potential risks of inaction. Patients affected by PNS and ANS manifestations frequently report pain and dysfunction that substantially impair their QOL. Available treatment options are consistent with what patients prioritize: relief of suffering, preservation of function, and prevention of worsening disease. Thus, for many of the interventions considered, even low-certainty evidence suggested

Table 4. Good practices and recommendations for autonomic nervous system neuropathies in patients with Sjögren's disease**Screening good practices for autonomic nervous system neuropathies**

Good practice 20	The following screening process is recommended for the initial assessment of potential autonomic nervous system (ANS) involvement in Sjögren's: For primary screening, ask the following questions: 1. Do you experience postural lightheadedness, syncope (fainting), near-syncope, or a racing heart when upright? 2. Do you experience problems with sweating too little, or too much? 3. Do you have chronic GI symptoms or problems, such as: o Difficulty swallowing o Nausea o Abdominal pain o Feel like you fill up more quickly than you ought to when eating a meal (early satiety) o Diarrhea or constipation. If yes, does diarrhea occur at night? o Significant abdominal bloating 4. Do you have genitourinary problems, such as: o Sexual dysfunction o Difficulty emptying your bladder completely or incontinence o Frequent urination, particularly frequent nocturnal enuresis o Urogenital problems and/or pelvic pain
Good practice 21	For Sjögren's patients with suspected ANS involvement, conduct further assessments with the Composite Autonomic Symptom Scale (COMPASS-31) questionnaire and orthostatic blood pressure and pulse measurements (postural vital signs).
Good practice 22	For Sjögren's patients with suspected ANS involvement and abnormal COMPASS-31 or abnormal postural vital sign results, refer to a neurologist, neurogastroenterologist, cardiologist, urologist, or ANS testing center for further evaluation.
Good practice 23	For Sjögren's patients with symptoms of orthostatic intolerance, including postural lightheadedness, syncope, and near-syncope, conduct formal autonomic testing at centers or facilities where this testing is available. Autonomic testing should include tilt table, the Quantitative Sudomotor Axon Reflex Test (QSART), measures of heart rate variability, and assessments of blood pressure changes, including the Valsalva maneuver, to characterize the nature of the ANS involvement.
Good practice 24	If formal autonomic testing is performed, a Composite Autonomic Severity Scale (CASS) score could be calculated to quantify the severity of autonomic nervous system impairment in Sjögren's.
Good practice 25	For Sjögren's patients with significant autonomic neuropathy, the following should be considered to exclude other causes: B-12 and B-6 measurement, hemoglobin A1C, serum immunofixation, serum-free light chains, and an autoimmune dysautonomia panel (screen of antibodies associated with autonomic neuropathy other than Sjögren's, such as acetylcholine receptor ganglionic antibody, ANNA-1, CASPR-2IgG CBA, CRMP-5, DPPX, LGI1-IgG CBA, PCA-2, AP3B2 IFA). Testing for supine and standing catecholamines (norepinephrine) may be considered.
Good practice 26	For Sjögren's patients where there is concern for autonomic neuropathy or symptoms of abnormal sweating, a skin biopsy could be considered to assess sweat gland nerve fiber density.
Good practice 27	Provided the initial study was abnormal, quantitative autonomic testing (such as tilt table, the QSART, measures of heart rate variability, and assessments of blood pressure changes, including the Valsalva maneuver) could be repeated in 6-12 months after institution of immunomodulatory therapy to assess peripheral nervous system remodeling in Sjögren's patients.
Good practice 28	Autonomic failure in conjunction with sensory neuropathy and sensory ataxia may indicate an autonomic ganglionopathy. Providers should recognize that this may require an urgent referral to a neurologist and aggressive treatment.
Good practice 29	For Sjögren's patients with complex gastrointestinal symptoms, refer to a gastroenterologist or neurogastroenterologist for evaluation and treatment. Motility testing to look for esophageal, gastric, or intestinal dysmotility should be considered.
Good practice 30	For Sjögren's patients with genitourinary symptoms, refer to a urologist, urogynecologist, or neurologist for evaluation and treatment.
Good practice 31	Symptoms related to problematic sweating may result from autonomic neuropathy in Sjögren's. Nocturnal sweating, hyperhidrosis, hypohidrosis, and heat intolerance may be reported by patients. A careful history and examination, supplemented by autonomic tests of sweating function (such as QSART) can be helpful in identifying severity and pattern of impairment.

ANNA-1 = anti-neuronal nuclear antibody, type 1

CASPR2-IgG CBA, S = contacting-associated response protein 2

CRMP-5 IgG Western Blot, S = collapsing response-mediator protein – 5

DPPX = dipeptidyl-peptidase-like protein-6 (regulatory subunit neuronal Kv4.2 potassium channel)

LGI1-IgG CBA, S = leucine-rich glioma inactivated 1 protein antibody

PCAB-2 = Purkinje cell cytoplasmic antibody, type 2

AP3B2 IFA Titer, S = adaptor protein 3 beta 2 antibody by IFA (immunofluorescence)

(Continued)

Table 4. (Cont'd)

Therapy recommendations for autonomic nervous system neuropathies		Evidence rating	Strength of recommendation
Recommendation 16	For Sjögren's patients with autonomic neuropathy, we recommend patient education about autonomic dysfunction and non-pharmacologic tools such as lifestyle measures and therapies that target symptoms caused by their disease.	Low	Strong ^a
Recommendation 17	For Sjögren's patients with orthostatic tachycardia, we recommend providing patient education to facilitate lifestyle management and initiating non-pharmacologic approaches such as increased fluid intake, abdominal and lower limb compression garments, increased salt intake, and gentle graded exercise in a seated or supine position. Assess if current medications predisposing to hypotension or tachycardia can be tapered/discontinued (stimulants, diuretics, vasodilators). For patients with severe symptoms of orthostatic intolerance or those who do not respond to lifestyle measures, consider the following: • For heart rate control, use non-selective beta blockers or ivabradine, which is a hyperpolarization-activated cyclic nucleotide-gated (HCN) channel blocker, especially for those patients with prominent heart rate variability. • For individuals with orthostatic tachycardia with low blood pressure thought to potentially contribute to tachycardia, consider vasopressors (midodrine, droxidopa) or fluid expansion (fludrocortisone, IV saline). • Pyridostigmine may be useful in stabilizing heart rate and blood pressure in individuals with orthostatic tachycardia.	Low	Conditional
Recommendation 18	For Sjögren's patients with orthostatic hypotension, we recommend a multidisciplinary team (could include a rheumatologist, cardiologist, and neurologist) for assessment (tilt table, QSART, etc.) and management, depending on services available regionally.	Very Low	Conditional
Recommendation 19	For Sjögren's-related immune-mediated autonomic dysfunction that is severe, progressive, and not responsive to lifestyle and pharmacologic measures, we recommend considering intravenous immunoglobulin (IVIG), with frequency and dosage that manages symptoms.	Very Low	Conditional
Recommendation 20	For Sjögren's-related immune-mediated autonomic dysfunction that is severe and not responsive to IVIG, we recommend rituximab.	Very Low	Conditional

^a This recommendation is rated strong because patients will likely prioritize non-pharmacologic approaches and educational measures that offer control and understanding of their condition without the risks associated with medication.

meaningful improvements in outcomes such as pain reduction, functional ability, and activities of daily living. Importantly, withholding treatment would risk deterioration in patient well-being. In reaching these conclusions, the TRG benefited from a strong suite of rheumatologists and neurologists who conduct research and treat patients with SjD. Taken together, these factors justify the panel's decision to issue strong recommendations despite very low certainty of evidence. This approach reflects the principle that guideline development must consider not only the level of evidence but also the consensus of experts, patient needs and values, and the ethical responsibility to minimize harm. The TRG is confident that the resulting recommendations are consistent with current therapeutic practices and concordant with patient values and preferences, reflecting prevailing clinical consensus.

We used the term “consider” in conjunction with certain recommendations to acknowledge the variability in patient-specific factors, preferences, and clinical scenarios that may influence the appropriateness of a given intervention in the shared

decision-making process. Importantly, the term “consider” should not be construed by insurance providers as a justification for denying coverage but rather as recognition of the need to tailor interventions to the unique circumstances of each individual patient.

The CEP review reached a high level of agreement, with 18 of 20 recommendations and 30 of 31 good practices achieving 90% or higher consensus. Notably, the lowest level of agreement was 79% in the vote for the “strong” rating for the strength of recommendation 7. This recommendation addresses intravenous Ig (IVIG) for patients with nonvasculitic large fiber polyneuropathy. Comments pertaining to this vote included the cost of IVIG, side effects, and strength of the evidence.

In developing this guideline, the TRG acknowledges that management of these manifestations necessitates a multidisciplinary approach to care. To the extent possible, neurologists should play a role in teams managing PNS manifestations in collaboration with primary care physicians and rheumatologists.

Other specialists, such as those in ear, nose, and throat; ophthalmology; speech pathology; cardiology; gastroenterology; neurogastroenterology; physical therapy; urology; and neurology may also be part of the treatment team. Additionally, it is important to note that patients can have more than one co-occurring PNS manifestation. Thus, providers may need to implement more than one treatment modality concurrently and may need to coordinate among multiple specialties. Finally, SjD should be considered in patients presenting with multiple PNS and central nervous system presentations, especially when a singular pattern of localization is lacking.

An important consideration in the management of SjD-related immune-mediated neuropathies is the differing perspectives between neurologists and rheumatologists regarding the use of IVIG therapy. Neurologists often encounter a high volume of idiopathic neuropathies and may be more skeptical about considering IVIG in cases that are not clearly immune-mediated. In contrast, rheumatologists frequently manage patients whose existing neuropathies were not recognized as immune-mediated, delaying appropriate diagnosis and treatment and negatively impacting outcomes. This divergence highlights the need for neurologists to collaborate across disciplines and consider immune-mediated etiologies, such as SjD, in their differential diagnoses when assessing patients with neuropathy. Likewise, rheumatologists should note the potential of immune-mediated neuropathy and SjD.

Acknowledging the significant evidence gap in our understanding of effective therapies for PNS manifestations in SjD is important. A major limitation is the absence of study designs with comparison groups, which increases the risk of bias and limits generalizability. However, for understudied diseases such as SjD, case series can provide crucial initial insights, guiding future research. Another weakness is the lack of current literature supporting a direct link with specific conditions, such as hearing loss, vertigo, and dysfunction of cranial nerves (CNs) IX and X. Thus, we did not make a recommendation regarding these potential manifestations.

Current clinical trials are exploring biologic and small-molecule therapies for systemic features of SjD, including PNS manifestations.^{45–50} These trials may result in multiple potential therapies for more effective management. With this in mind, this guideline will be reviewed and updated in 3 to 5 years or sooner if substantial new evidence becomes available that may impact the recommendations. Corneal neuropathy will be included in the forthcoming update to the Sjögren's Foundation ocular clinical practice guidelines.

There is a dire need to improve the evidence base for patients who have SjD with PNS manifestations. This would include observational studies with comparison groups and, ideally, randomized controlled trials. Because of the difficulties in performing such studies, big data analyses of electronic health care records may provide additional evidence to support causal inferences. Additionally, studies are needed to address the following gaps in clinical knowledge:

Prevalence

1. What is the incidence and prevalence of PNS and ANS manifestations in SjD?
2. What is the prevalence of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) in patients with SjD? Does SjD play a role in the pathophysiology of CIDP?
3. Does COVID-19 exacerbate neurologic morbidities in patients with SjD?
4. What is the true incidence and pathophysiology of other cranial neuropathies, such as CN VIII, CN IX, and CN X, which have been reported to occur in SjD?

Prevention

5. What are the risk factors for PNS and ANS manifestations in SjD?
6. What interventions can prevent PNS and ANS manifestations in SjD?
7. What are the genetics, epigenetics, and pathophysiology of PNS and ANS manifestations in SjD? How can understanding these factors lead to therapies to prevent and treat these disorders?

Diagnosis and Evaluation

8. Is the scale used for interpreting skin punch biopsies when diagnosing small fiber neuropathies in diabetes applicable to SjD, or is a new or modified scale needed?
9. How can we develop improved scales to measure dynamic disabilities associated with ANS neuropathies that effectively capture the temporal variability of symptoms?
10. How can we standardize diagnostically accurate and minimally invasive testing methods, including imaging, biomarkers, and electrodiagnostic techniques, for ANS and PNS manifestations in SjD?
11. What is the sensitivity and specificity of lip biopsy in seronegative sicca-free patients with PNS and/or ANS dysfunction and personal and/or family history of autoimmune disease?
12. How can we develop diagnostic tools or scales to differentiate between progressive PNS/ANS manifestations and irreversible nerve damage?
13. What are the positive and negative predictive values for rheumatoid factor, complement component 4, and complement component 3 for the diagnosis of vasculitis?

Management of PNS and ANS Manifestations

14. What are the optimal first-line, second-line, and maintenance strategies (ie, IVIG or other immunotherapies and pain management treatments) for PNS and ANS manifestations with varying degrees

of symptom severity in SjD, and should treatment decisions be affected by autoantibody (anti-SSA, anti-SSB, rheumatoid factor, and antinuclear antibodies) positivity?

15. What are the effects of pharmacologic interventions versus behavioral interventions in managing neuropathic SjD symptoms?
16. What are the differences in the comparative effectiveness of treatment options for ANS and PNS manifestations between SjD and non-SjD populations?
17. What is the possible role of microbial imprint/dysbiosis in patients with SjD with PNS and ANS manifestations? Does correcting the dysbiosis by lifestyle interventions prevent or ameliorate PNS or ANS symptoms?
18. What are the mechanisms of neuronal injury and disease course of PNS and ANS manifestations in SjD as shown by autopsy studies?

Outcomes

19. How can we establish standardized and defined patient-centered outcomes for measuring PNS and ANS manifestations in SjD, considering their preferences in managing the benefits versus harms of treatment modalities?
20. What strategies can be developed to monitor response to therapy in SjD, including the potential utility of serial electrodiagnostic testing and patient-centered evaluations?
21. How can the ESSDAI and other outcome measures be enhanced to increase sensitivity to identify the spectrum of neurologic and other systemic manifestations?
22. How can standardized outcome measures be used to evaluate treatment effectiveness for PNS and ANS manifestations in SjD?

Guideline Implementation

23. How can implementation science be used to understand the barriers and facilitators in implementing guidelines for SjD in clinical practice, and what strategies can be developed and evaluated to enhance this implementation?
24. What is the impact of educating the health care community about the diverse presentations of PNS and ANS manifestations in SjD?
25. How does engaging interdisciplinary teams affect the diagnosis and management of PNS and ANS manifestations in SjD?

This guideline offers an evidence-based approach to evaluate and manage PNS manifestations of SjD. The recommendations emphasize the need for multidisciplinary care, including

primary care physicians, rheumatologists, neurologists, and other specialists in some cases, and patient-centered decision-making. Given the limited evidence on PNS manifestations in SjD, the guideline also highlights research priorities to propel the field forward and improve our understanding and management of this challenging aspect of SjD.

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AUTHOR CONTRIBUTIONS

All authors contributed to at least one of the following manuscript preparation roles: conceptualization AND/OR methodology, software, investigation, formal analysis, data curation, visualization, and validation AND drafting or reviewing/editing the final draft. As corresponding author, Ms. Hammitt confirms that all authors have provided the final approval of the version to be published and takes responsibility for the affirmations regarding article submission (eg, not under consideration by another journal), the integrity of the data presented, and the statements regarding compliance with institutional review board/Declaration of Helsinki requirements.

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APPENDIX A: CONSENSUS EXPERT PANEL (CEP) MEMBERS

Members of the CEP are as follows: G. Alden Adkins, MD (Rheumatology Fellow, University of Virginia); Brittany Adler, MD (Assistant Professor of Medicine, The Johns Hopkins University School of Medicine); Hossein Ansari, MD (Director of Headache and Facial Pain Clinic, Associate Professor of Neuroscience, Kaizen Brain Center, La Jolla, CA, and University of California, San Diego); Senada Arabelovic, DO (Assistant Professor of Medicine, Brigham and Women's Hospital, Boston, MA); Alan Baer, MD (Director, Jerome Greene Sjögren's Syndrome Clinic, Professor of Medicine, The Johns Hopkins University School of Medicine); Denis Balaban, MD (Instructor in Neurology, Massachusetts General Hospital and Harvard Medical School); Shamik Bhattacharyya, MD (Program Leader, Autoimmune Neurology, Brigham and Women's Hospital and Harvard Medical School); Evelyn Bromet, PhD (Distinguished Professor Emerita, Stony Brook University); Krishna Chaganti, MD, MS (Associate Professor of Medicine, University of California San Francisco); Kamal Chemali, MD (Professor of Neurology, Case Western Reserve University and University Hospitals of Cleveland); Melissa Cortez, DO (Associate Professor, Department of Neurology, Founding Director, Autonomic Physiology Laboratory, University of Utah); Scharless Culpepper, MD (Staff Physician, University of Miami); Paul Dellaripa, MD (Associate Professor, Harvard Medical School, Brigham and Women's Hospital, Boston, MA); Dana Drenzo, MD, MHS (Assistant Professor of Clinical Medicine (Rheumatology), University of Pennsylvania); Daniel El Bogdadi, MD (Assistant Clinical Professor of Medicine, Arthritis & Rheumatism Associates and Georgetown University); Robert Fearon; Mehrez Fischbach, MD (Chief, Division of Rheumatology, Associate Professor of Medicine, National Jewish Health); Judi Furlong, MD (Retired Family Medicine); Christopher Gibbons, MD, MMSc (Professor of Neurology, Harvard Medical School); Rachael Gordon, MD, PhD (Rheumatology Fellow, University of Pittsburgh Medical College); Thomas Grader Beck, MD (Assistant Professor of Medicine, The Johns Hopkins University School of Medicine); Syed Haider, MD (Attending

Cardiologist, Director of Pericardial Disease Program, MedStar Washington Hospital Center and Georgetown University Hospital); Larry Hollenbeck, MD (Neurologist, Meritas Health Neurology, Kansas City, MO); Chadwick R. Johr, MD (Associate Professor of Clinical Medicine, University of Pennsylvania); Stuart S. Kassan, MD (Distinguished Clinical Professor of Medicine, National Jewish Health and University of Colorado School of Medicine); Brian King, MD (Rheumatology Resident, University of Virginia); Octavia Kincaid, MD (Assistant Professor, Rush University Medical Center); Eugene Kissin, MD (Director, Rheumatology Fellowship Program, Clinical Professor of Medicine, Boston Medical Center); Vasileios Kyttaris, MD (Assistant Professor of Medicine, Beth Israel Deaconess Medical Center and Harvard Medical School); Lindsay Lally, MD (Hospital for Special Surgery, New York, NY); Brandon M. Law, MD (Massachusetts General Hospital, Boston, MA); Janet Lewis, MD (Associate Professor of Medicine, University of Virginia); Scott M. Lieberman, MD, PhD (Associate Professor of Pediatrics, Division of Rheumatology, Allergy and Immunology, University of Iowa Carver College of Medicine); Amanda Lusa, MD (Assistant Professor of Rheumatology, University of Virginia); Joseph Lutt, MD (Rheumatologist, Colorado Center for Arthritis & Osteoporosis); Rashmi Maganti, MD (Interim Section Chief, Department of Medicine, Immunology, Allergy & Rheumatology, Rheumatology, Medical Subspecialty Clinic Director, Baylor College of Medicine); Arthur M. Mandelin II, MD, PhD (Associate Professor of Medicine, Northwestern University Feinberg School of Medicine); Sara McCoy, MD, PhD (Assistant Professor, University of Wisconsin School of Medicine and Public Health); Kerry Neall, MD, MPH (Emergency Medicine, Orlando Health, Orlando, FL); Timothy Niewold, MD, FACR (Vice Chair for Research, Department of Medicine, Hospital of Special Surgery, New York, NY); Anne Louise Oaklander, MD, PhD (Associate Professor of Neurology, Assistant in Pathology (Neuropathology), Massachusetts General Hospital and Harvard Medical School); Ruben A. Peredo-Wende, MD (Site Director, Veteran Affairs Medical Center, Albany, NY); Lynn Petrucci, RN, MSN (Retired Registered Nurse); Mark A. Porter, MD (Retired Neurologist); Guada Respicio Duque, MD, MSc (Rheumatologist, Arthritis and Rheumatism Associates); Tania Reyna, MD (Associate Professor, University of Texas Health San Antonio); Nathaniel M. Robbins, MD (Director, MGB Program in Small Fiber and Autonomic Neurology, Harvard Medical School, Massachusetts General Hospital, and Brigham and Women's Hospital); Elliot D. Rosenstein, MD (Rheumatologist, Overlook Medical Center/Atlantic Health System); Laura Rosow, MD (Associate Professor of Neurology, University of California, San Francisco); Breanna Ruthrauff, MD (Retired Neurologist); Amit Sachdev, MD (Director, Neuromuscular Medicine, Assistant Professor, Michigan State University); Nora Sandorfi, MD (Professor of Clinical Medicine, University of Pennsylvania); Sarah Schafer, MD (Medical Educator, Retired Physician); Elena Schiopu, MD (Professor of Medicine, Rheumatology Division, Medical College of Georgia); Chokkalingam Siva, MD (Professor of Clinical Medicine, University of Missouri School of Medicine); Daniel Small, MD (Rheumatologist, Mayo Clinic Healthcare System SW Wisconsin); Sara M. Stern, MD (Assistant Professor, University of Utah School of Medicine and Primary Children's Medical Center); Lauren Stiles, JD (President, Dysautonomia International, Research Assistant Professor, Neurology, Stony Brook University); Susan Stoddard, RN (Research Coordinator in Cardiology); Jinny Tavee, MD (Professor of Medicine, National Jewish Health); Donald Thomas, MD (Rheumatologist, Arthritis and Pain Associates for Prince George's County); Edward L. Treadwell, MD (Professor of Medicine/Rheumatology/Immunology, East Carolina University School of Medicine); Nagagopal Venna, MD (Chief, Division of Neuroimmunology and Neuro-Infectious Diseases, Massachusetts General Hospital, Boston, MA); Steven Vernino, MD (Professor of Neurology, University of Texas Southwestern); Frederick B. Vivino, MD, MS (Director Emeritus, Penn Sjögren's Center, University of Pennsylvania); Brittany Weber, MD, PhD (Director of the Cardio-Rheumatology Clinic, Associate Physician in Prevention Cardiology and Cardiovascular Imaging, Assistant Professor, Brigham and Women's Hospital and Harvard Medical School); Sepideh Yadollahi, MD (Resident Physician, University New Mexico, Kaizen Brain Center); Huiying Yu, MD (Director of Neurodiagnostic Lab, Neuromuscular Section, NYU Langone Hospital Long Island); Scott Zashin, MD (Texas Health, Dallas, Texas).