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The role of neurologists and diagnostic tests in the management of distal symmetric polyneuropathy

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Abstract

Importance—Distal symmetric polyneuropathy (DSP) is a prevalent condition resulting in high costs from diagnostic testing. However, the role of neurologists and diagnostic tests on patient care is unknown.

Objective—To determine how often neurologists and diagnostic tests influence the diagnosis and management of DSP patients in a community setting.

Design—We utilized a validated case-capture method (ICD-9 screening technique with subsequent medical chart abstraction) to identify patients with a new DSP diagnosis (retrospective cohort). Using a structured data abstraction process, diagnostic testing, diagnoses rendered (before and after testing), and subsequent management were recorded.

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Conflicts of Interest:

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Setting—Community neurologist's outpatient offices in Corpus Christi, Texas.

Participants—Patients meeting the Toronto consensus criteria for probable DSP.

Main Outcome Measure—Changes in etiology and management after diagnostic testing by neurologists.

Results—Between 1/1/2010–3/31/2011, we identified 458 DSP patients followed for mean (SD) 435.3 (44.1) days. Neurologists identified a cause of DSP in 63.5% of cases prior to their diagnostic testing. Seventy-one patients (15.5%) had a new DSP cause discovered after testing by neurologists. The most common new diagnoses were pre-diabetes (N=28), B12 deficiency (N=20), diabetes (N=8), and thyroid disease (N=8). Management changes were common (63.1%), usually related to neuropathic pain management (77.5%). Disease modifying management changes occurred in 24.7% with diabetes management (N=45), starting vitamins (N=39), advising diet/exercise (N=33), and adjusting thyroid medications (N=10) the most common. Electrodiagnostic testing and MRIs of the neuroaxis rarely led to management changes.

Conclusions and Relevance—Neurologists diagnosed the cause of DSP in almost two-thirds of patients prior to their diagnostic testing. Inexpensive blood tests for diabetes, thyroid dysfunction, and B12 deficiency, allowed neurologists to identify a new etiology in 15.5% of patients. In contrast, expensive electrodiagnostic tests and MRIs rarely changed patient care. Neurologists also frequently made pain medication changes utilizing best evidence medications.

Introduction

Disorders of the peripheral nervous system account for 1.5 million visits to neurologists annually, which is over 10% of all visits.[1] Diagnostic testing of these conditions by outpatient neurologists costs $357 million each year with electrodiagnostic tests ($205 million, 57%) and MRIs ($135 million, 38%) accounting for the vast majority of the costs.[1] Peripheral neuropathy is the most common disorder of the peripheral nervous system, with a prevalence of 2–7% in the entire population, which rises to greater than 10% in the elderly.[2–4] Not surprisingly, the evaluation of peripheral neuropathy can be quite costly, with most of the cost driven by electrodiagnostic and MRI testing.[5–7] Given the high aggregate costs associated with this evaluation, determining the value of these diagnostic tests becomes of paramount importance.

Understanding the role of neurologists on the care of peripheral neuropathy patients is also essential. Not all primary care physicians see the benefit of having a neurologist involved in the diagnosis and treatment of common neurologic diseases such as TIA, dementia, and Parkinson's disease.[8] As reimbursement incentives are realigned, such as the cognitive care bonus for primary outpatient care specialties (which excluded neurologists), the need for evidence to support the value of neurologists is evident and critical.[9,10] While data exists to support the role of neurologists in improving patient outcomes in stroke populations, little data exist to define the role of neurologists in the care of peripheral neuropathy populations.[11–13]

Current evidence supports routine testing of fasting glucose, B12, serum protein electrophoresis (SPEP), and a glucose tolerance test in the initial evaluation of distal

symmetric polyneuropathy (DSP), by far the most common subtype of peripheral neuropathy.[14] However, little is known about the value of electrodiagnostic tests and MRIs in this evaluation. Similarly, the value of neurologists in the diagnosis and care of DSP patients has not been previously studied. The aim of this study was to determine the role of neurologists and diagnostic tests on the diagnosis and care of DSP patients in a community setting.

Methods

Population

We attempted to capture all patients with a new diagnosis of DSP seen by community neurologists in Nueces County, Texas (retrospective cohort). Most of the county population lives in the city of Corpus Christi, which is more than 140 miles from tertiary care centers in Texas. The geographic characteristic of this community means that patients with common medical conditions are likely to receive their medical care within the county. Of the eleven practicing neurologists in Nueces County, nine agreed to participate and two declined (one of these is retiring and the other does not regularly schedule neuropathy patients). This study was approved by the University of Michigan Institutional Review Board.

Distal Symmetric Polyneuropathy Definition

Patients were required to meet the Toronto consensus panel definition of probable neuropathy: ≥ 15 2 of the following criteria: neuropathic symptoms (self-report of pain, numbness, and/or tingling in the feet and/or legs), decreased distal sensation on neurologic examination, or decreased or absent ankle jerks.[15] We also required documentation of a diagnosis of neuropathy in the medical record. We excluded patients who were seen in the hospital only, had electrodiagnostic testing only, or were previously diagnosed with neuropathy by a Corpus Christi neurologist.

Case Capture Method

We used a previously validated DSP case-capture method which involves screening all new patient visits for ICD-9 neuropathy symptom and diagnostic codes (250.60; 356.0,1,2,4,8,9; 357.1–7,82,89,9; 729.5 (pain in limb); and 782.0 (disturbance of skin sensation)) using the outpatient office's billing database followed by medical record abstraction to confirm that they met our DSP definition.[16] We have previously demonstrated that this case-capture method has a sensitivity of 100% and a specificity of 88% for the classification of DSP patients when compared with a neuromuscular specialist.[16] The case capture method was used from 4/1/2010 to 3/31/2011.

Medical Record Abstraction

Medical records were abstracted by a trained research coordinator using the entire outpatient medical record, from the initial visit and any subsequent follow-up visits within the next 1–2 years. Information abstracted included demographics, clinical characteristics (the three criteria for the Toronto consensus definition of probable neuropathy, the time since symptom onset, family history of neuropathy, pain, weakness on examination, and five warning signs of an atypical neuropathy (acute/subacute or relapsing presentation,

asymmetry, non-length dependent, motor predominant, prominent autonomic features), and all diagnostic tests ordered by the neurologist. We also documented the suspected etiologies of the neurologist at the time of the initial evaluation and at last follow up as recorded in the medical record. All management changes were documented from the first evaluation to the last neurologist visit. Potential disease modifying management changes that were recorded included the following: recommending improved diabetes management, starting vitamins such as B12, encouraging diet and exercise, changing thyroid medications, recommending alcohol cessation, stopping medications thought to be neurotoxic, and prednisone. Ongoing quality control comparing abstractions done by the trained research coordinator and a neuromuscular specialist was performed throughout the study period.

Results

We screened 4,890 patient charts, and 831 had a neuropathy ICD-9 code. Of the 831 patients identified by the screen, 86 were excluded because the patients were seen in the hospital only, had electrodiagnostic testing only, or were previously diagnosed with neuropathy by a Corpus Christi neurologist. We also excluded 287 patients who did not meet the Toronto consensus DSP criteria. Thus the final population was 458 DSP patients. Population demographics are presented in Table 1. The mean (SD) age was 65.8 (12.9) and 56.3% were female. Medical records were available for a mean of 2.4 (1.6) visits per patient and 435.3 (44.1) days after the initial neurology evaluation. In the entire population, neurologists ordered electrodiagnostic testing in 353 (77.1%) patients and MRIs of the neuroaxis in 65 (14.2%). Of the four AAN-recommended tests, neurologists ordered B12 levels in 177 (38.6%), fasting glucose levels in 56 (12.2%), SPEP evaluations in 127 (27.7%), and GTTs in 20 (4.4%). TSH levels were measured in 144 (31.4%) patients.

Prior to diagnostic testing by the neurologist, neurologists were able to determine the etiology of DSP in nearly two-thirds (63.5%) of the population. The most common etiology prior to diagnostic testing was diabetes (50.9%), followed by thyroid condition (6.8%), alcohol (3.1%), chemotherapy (2.0%), and B12 deficiency (1.7%) (Table 2). A total of 167 (36.5%) patients had no clear etiology prior to testing by the neurologist. Of these 167 patients, diagnostic testing by neurologists revealed a new etiology in 45 (9.8% of total population). Including all patients, the neurologist discovered a new etiology in 71 (15.5%) patients. The most common new diagnosis was pre-diabetes (6.1%), followed by B12 deficiency (4.4%), diabetes (1.7%), and thyroid disorder (1.7%) (Table 3). Other new conditions diagnosed by laboratory abnormalities included paraproteinemia, anemia, and folic acid deficiency. The neurologist identified a new etiology in 8 cases based on history alone, including DSP attributed to toxic medications, alcohol, inherited neuropathy, peripheral vascular disease, poliomyelitis, and steel toed shoes. An additional 4 new etiologies were based on history and/or laboratory abnormalities including renal disease, hypoglycemia, and the metabolic syndrome. Of note, two patients were no longer considered to have neuropathy after diagnostic testing.

A total of 289 (63.1%) patients had a least one management change at or after the initial neurologist evaluation (Table 4). The most common management change was altering medication regimens (57.2%). Two hundred twenty four (48.9%) patients had a change to

their neuropathic pain medications with the vast majority of the changes involving calcium channel agonists (N=145) such as gabapentin and pregabalin, tricyclic antidepressants (TCAs, N=53), and serotonin norepinephrine reuptake inhibitors (SNRIs, N=49). A potential disease modifying management change was made in 113 (24.7%) patients. Improved diabetes management was recommended for 45 (9.8%), starting vitamins for 39 (8.5%) with most involving B12 (N=33), encouraging diet and exercise for 33 (7.2%), and changing thyroid medications for 10 (2.2%). The next most common disease modifying management changes were recommending alcohol cessation (1.7%) and stopping medications thought to be neurotoxic (0.9%). The only other potentially disease modifying therapy was prednisone which was started in 2 patients, one of whom also received methotrexate. Of these two patients, one had a known mixed connective tissue disease for which the patient was taking prednisone and had a chronic DSP of unclear duration. A sural nerve biopsy revealed a mild axonal neuropathy with no features suggestive of vasculitis. The prednisone was increased with little change in the neuropathy although the patient only had 18 days of follow up. The other patient had the subacute onset of DSP and was diagnosed by the referring physician with Sjogren's syndrome based on an elevated sedimentation rate and SS-A and SS-B antibodies. The electrodiagnostic study revealed an axonal sensory motor neuropathy. The neurologist started the patient on prednisone with substantial improvement of the neuropathic symptoms. Over time the patient was started on methotrexate as he was unable to tolerate weaning off the prednisone.

Electrodiagnostic studies (N=368) led to a change in etiology and/or management in 2 patients. In both of these patients the change in etiology was from a neuropathy diagnosis to a non-neuropathy diagnosis. Neither of the two patients that received disease modifying therapy with prednisone had a change in management based on electrodiagnostic studies. One did not have a study performed and the other had the non-specific finding of an axonal symmetric sensory motor neuropathy. MRIs of the neuroaxis did not lead to a change in management in any case, outside of alteration in pain medications. Diagnostic testing that most frequently led to a change in management included B12, thyroid studies, and testing for diabetes (Table 3).

Discussion

Prior to diagnostic testing, neurologists were able to determine the cause of DSP in nearly two-thirds of new DSP patients presenting to community neurologists. After diagnostic testing, neurologists identified a new etiology of DSP in an additional 15% of cases, with the most common new diagnoses being pre-diabetes, B12 deficiency, diabetes, and thyroid disease. Therefore, neurologists, with the aid of their clinical evaluation and a few simple blood tests, were able to diagnose the underlying cause of DSP in nearly three-fourths of this population. Moreover, neurologists frequently made management changes with the vast majority of these being alterations in neuropathic pain medication regimens. Other common management changes were recommending improved diabetes management, starting vitamins (usually B12), encouraging diet and exercise, and altering thyroid medications. On the other hand, other disease modifying management changes were rare and did not result from the findings of electrodiagnostic tests or MRIs of the neuroaxis. In fact, electrodiagnostic tests

and MRIs rarely changed the management of new DSP patients presenting to community neurologists.

Similar to previous studies, we found that diabetes is the most common cause of DSP accounting for more than half of the cases.[17,18] However, compared to studies performed in tertiary care clinics and hospital settings, an even higher proportion of patients in this community sample have DSP attributable to diabetes (53% compared to 20–30%). The second most common diagnostic category was idiopathic DSP, which accounted for over one-third of cases prior to diagnostic testing and more than one-fourth after all tests were performed. Several past investigations have also described a similar proportion of idiopathic neuropathy although none have been performed in a community neurology setting.[17–19] On the other hand, alcohol was a much less frequent cause of neuropathy in our sample compared to other populations.[17,18] Other common causes found in this population included thyroid dysfunction, pre-diabetes, and B12 deficiency.

Almost 10% of patients with an unclear diagnosis prior to evaluation had a new etiology discovered during their diagnostic evaluation. Therefore, not only are neurologists able to diagnose the cause of DSP in almost two-thirds of patients with history and examination alone, but they are also able to diagnose a new cause in an important proportion of patients after diagnostic testing. Neurologists identified the cause of neuropathy in such a large proportion despite the fact that few patients received all of the AAN recommended tests. These results highlight the need to increase guideline adherence testing as has been demonstrated in previous nationally representative studies.[6,7] The most common new etiology was pre-diabetes followed by B12 deficiency, diabetes, and thyroid dysfunction. All of these new etiologies have the potential to lead to changes in management and are diagnosed with inexpensive laboratory tests. Other new etiologies that were discovered were quite rare and usually the result of new historical information and/or simple laboratory tests. The exception is that two patients were considered to have DSP prior to electrodiagnostic testing, but after these tests the patients were not considered to have neuropathy. Community neurologists identify the cause of three-fourths of patients with DSP either through their clinical evaluation or through inexpensive laboratory tests.

By far, the most common management change that a community neurologist makes for DSP patients is adjustment of neuropathic pain medications. Neurologists recommended changes in the pain medication for almost half of the DSP patients, which emphasizes both the importance of the neurologist in the care of these patients and the frequency of pain in this prevalent condition. Furthermore, 85% of the changes in pain medication made by neurologists involved the three classes of neuropathic pain medications with the best levels of evidence to support their use (SNRIs, TCAs, and calcium channel agonists).[20,21] Neurologists rarely prescribed NSAIDs or narcotics for neuropathic pain. Neuropathic pain is often under-recognized and undertreated.[22] Moreover, the management of neuropathic pain requires expertise and comfort with multiple medications classes. Neurologists can aid general practitioners not only in the discovery of the underlying cause of neuropathy, but also in the management of the pain that is often the patient’s most debilitating symptom.

In contrast to pain management alterations, disease modifying interventions were less commonly initiated by community neurologists. The most frequent interventions included recommending enhanced diabetes management, starting vitamins such as B12, encouraging diet and exercise, and adjusting thyroid medications. Less common interventions included counseling to limit or abstain from alcohol or to stop toxic medications. The only other disease modifying therapy that was prescribed in this population was prednisone in two patients. While no firm conclusions can be made based on these two patients, neither of these patients had a change in management based on an electrodiagnostic test. In fact, the only changes in this study that could be attributable to electrodiagnostic testing were the reclassification of two patients to non-neuropathy diagnoses after testing. Similarly, MRIs of the neuroaxis did not lead to significant changes in etiology or management in this population. While neurologists make important management changes such as addressing diabetes, pre-diabetes, B12 deficiency, and thyroid dysfunction, other management changes are rare and not influenced by electrodiagnostic tests or MRIs.

Limitations of the study include the retrospective cohort design and the use of medical record abstraction for data collection. Our ICD-9 case capture technique may have missed DSP cases; however, we have previously shown this technique captures almost 95% of cases in this community.[16] We were unable to study potential changes in etiology and management that occur years after seeing a neurologist as the mean follow up was only 435 days. Similarly, we do not know what the referring physician considered the cause of the DSP or what they would have done without the neurologists being involved; therefore, neurologists likely did not make the initial etiologic diagnoses in all cases. We were also unable to validate the neurologist's diagnosis or assess the quality of the electrodiagnostic testing. This study was not designed to demonstrate whether diagnostic tests that changed the patient's etiology and/or management led to improved patient outcomes or if DSP patients who see neurologists have better outcomes than those that do not. Of note, our study population had a low proportion of patients with warning signs of atypical neuropathy; these patients likely require more extensive testing than patients without these features, which should be left to the discretion of the neurologist. Specifically, patients with concern for inherited, vasculitic, or demyelinating neuropathy likely need electrodiagnostic testing. Likewise, some patients with DSP likely need MRI testing for other indications such as concerns for spinal stenosis. Future studies are needed to define the clinical scenarios and circumstances in which electrodiagnostic tests and MRIs are likely to change the etiology and/or management of DSP patients. While many studies support pre-diabetes as a cause of neuropathy, controversy remains.[23–28] Furthermore, how the results generalize to other communities and clinical settings, such as tertiary referral centers, is unclear.

Neurologists influence the care of patients with DSP in important ways including discovering new etiologies that result in potentially disease modifying therapies such as treatment of pre-diabetes, diabetes, B12, and thyroid medications. Neurologists are also instrumental in the treatment of neuropathic pain and frequently use the medications with the best evidence to support their use. Importantly, the neurologist's clinical history and examination and a few simple blood tests provide the clues for identifying the underlying cause of most neuropathies. However, electrodiagnostic tests and MRIs, which account for

the vast majority of the expenditures in the evaluation of DSP,[5–7] rarely lead to changes in etiology and management, and therefore the role of these tests requires further study.

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Dr. Callaghan had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The funding organizations had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, and approval of the manuscript; or decision to submit the manuscript for publication.

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Table 1

Demographics and clinical features of the distal symmetric polyneuropathy (DSP) population

Variable	N (%) Unless otherwise specified
Age, mean (SD)	65.8 (12.9)
Female	258 (56.3%)
Insurance status	
HMO, PPO, Private	244 (53.3%)
HMO, PPO, Private and Medicare	117 (25.5%)
Medicare, Medicaid, or both	82 (17.9%)
VA/Tricare/Champus	11 (2.4%)
Not insured	2 (0.4%)
Nueces County Indigent Health Care Program	2 (0.4%)
Toronto criteria	
Neuropathic symptoms	327 (71.4%)
Abnormal sensory examination	445 (97.2%)
Decreased reflexes	378 (82.5%)
2/3 criteria	224 (48.9%)
3/3 criteria	234 (51.1%)
Time since onset of neuropathy in months, mean (SD)	39.2 (49.6)
Family history of neuropathy	8 (1.7%)
Neuropathic pain	218 (47.6%)
Weakness or atrophy on examination	77 (16.8%)
Warning signs of an atypical neuropathy	
Acute/subacute/relapsing presentation	40 (8.7%)
Motor predominant	1 (0.2%)
Asymmetric	35 (7.6%)
Non-length dependent	6 (1.3%)
Prominent autonomic involvement	5 (1.1%)
Number of neurology visits, mean (SD)	2.4 (1.6)
Length of follow up (days), mean (SD)	435.3 (44.1)

HMO= health maintenance organization, PPO= preferred provider organization

Table 2

The neurologist's documented DSP etiology before and after diagnostic testing

Etiology	Sole Cause Before Testing N (%)	One of the Causes Before Testing N (%)	Sole Cause After Testing N (%)*	One of the Causes After Testing N (%)*
Any known cause	250 (54.6%)	291 (63.5%)	265 (57.9%)	336 (73.4%)
Diabetes	201 (43.9%)	233 (50.9%)	197 (43.0%)	241 (52.6%)
Unclear	167 (36.5%)	167 (36.5%)	122 (26.6%)	122 (26.6%)
Thyroid dysfunction	14 (3.1%)	31 (6.8%)	16 (3.5%)	39 (8.5%)
Alcohol	7 (1.5%)	14 (3.1%)	4 (0.9%)	16 (3.5%)
Chemotherapy	4 (0.9%)	9 (2.0%)	3 (0.7%)	9 (2.0%)
Hereditary	4 (0.9%)	5 (1.1%)	5 (1.1%)	6 (1.3%)
Rheumatoid arthritis	4 (0.9%)	4 (0.9%)	2 (0.4%)	4 (0.9%)
B12 deficiency	3 (0.7%)	8 (1.7%)	4 (0.9%)	28 (6.1%)
Pre-diabetes	3 (0.7%)	4 (0.9%)	23 (5.0%)	32 (7.0%)
Toxic medication	2 (0.4%)	6 (1.3%)	3 (0.7%)	8 (1.7%)
ESRD	2 (0.4%)	4 (0.9%)	1 (0.2%)	6 (1.3%)
Guillain Barre syndrome	2 (0.4%)	2 (0.4%)	1 (0.2%)	2 (0.4%)
Multiple myeloma	2 (0.4%)	2 (0.4%)	2 (0.4%)	2 (0.4%)
Gastric bypass	1 (0.2%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
Sjogren's syndrome	1 (0.2%)	1 (0.2%)	1 (0.2%)	1 (0.2%)
PVD	0 (0.0%)	5 (1.1%)	0 (0.0%)	6 (1.3%)
Cellulitis	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
HCV	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
HIV	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
Hypoglycemia	0 (0.0%)	1 (0.2%)	0 (0.0%)	2 (0.4%)
Paraneoplastic	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
Poliomyelitis	0 (0.0%)	1 (0.2%)	0 (0.0%)	2 (0.4%)
Raynaud's syndrome	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
Smoking	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)
Folic acid	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)
Metabolic syndrome	0 (0.0%)	0 (0.0%)	1 (0.2%)	1 (0.2%)
No neuropathy	0 (0.0%)	0 (0.0%)	2 (0.4%)	2 (0.4%)
Nutritional deficiency	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)
Paraproteinemia	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)
Steel toed boots	0 (0.0%)	0 (0.0%)	1 (0.2%)	1 (0.2%)

DSP=distal symmetric polyneuropathy, ESRD=end stage renal disease, PVD=peripheral vascular disease, HCV=hepatitis C virus, HIV=human immunodeficiency virus

*Of note, not all patients had all of the AAN recommended tests which may have affected the proportion of certain causes of neuropathy

Table 3

New etiologies discovered after diagnostic testing in 458 patients with DSP

New Etiology	N (% of all cases)
Pre-diabetes	28 (6.1%)
B12 deficiency	20 (4.4%)
Diabetes	8 (1.7%)
Thyroid dysfunction	8 (1.7%)
Alcohol	2 (0.4%)
ESRD	2 (0.4%)
No neuropathy	2 (0.4%)
Toxic medication	2 (0.4%)
Paraproteinemia	1 (0.2%)
Folic acid	1 (0.2%)
Hereditary	1 (0.2%)
Hypoglycemia	1 (0.2%)
Metabolic syndrome	1 (0.2%)
Nutrition	1 (0.2%)
PVD	1 (0.2%)
Poliomyelitis	1 (0.2%)
Steel toed boots	1 (0.2%)
Total	**71 (15.5%)**

DSP=distal symmetric polyneuropathy, ESRD=end stage renal disease, PVD=peripheral vascular disease

Table 4

Changes in management for 458 new DSP patients presenting to a community neurologist

Management Change	N (% of all cases)
Any management change	289 (63.1%)
Any medication change	262 (57.2%)
Pain medication change	224 (48.9%)
Best evidence medication	191 (41.7%)
SNRI	49 (10.7%)
TCA	53 (11.6%)
Ca channel agonist	145 (31.7%)
Narcotic	23 (5.0%)
NSAID	21 (4.6%)
Vitamins (includes B12)	39 (8.5%)
Vitamin B12	33 (7.2%)
Thyroid medication adjustment	10 (2.2%)
Stop toxic medication	4 (0.9%)
Prednisone	2 (0.4%)
Other medication change	19 (4.1%)
Non-medication change	95 (20.7%)
Diabetes management	45 (9.8%)
Diet and/or exercise	33 (7.2%)
Physical therapy	9 (2.0%)
Decrease alcohol	8 (1.7%)
Smoking	4 (0.9%)
Anodyne therapy	2 (0.4%)
Change boots	2 (0.4%)
Orthotics	2 (0.4%)
Acupuncture	1 (0.2%)
Referral to pain specialist	1 (0.2%)
Support hose	1 (0.2%)
TENS	1 (0.2%)
Wrist brace	1 (0.2%)

DSP=distal symmetric polyneuropathy, SNRI=selective norepinephrine reuptake inhibitor, TCA=tricyclic antidepressant, NSAID=non-steroidal anti-inflammatory drug, TENS=transcutaneous electrical nerve stimulation