Case 14-2011: A Woman with Asymmetric Sensory Loss and Paresthesias

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Presentation of Case

Dr. Sheila L. Arvikar (Medicine): A 52-year-old woman was seen in the neurology clinic at this hospital because of asymmetric sensory loss and paresthesias in both arms and legs as well as impaired dexterity.

The patient had been well until 5 months earlier, when her hands and feet gradually began to feel as though they were wrapped tightly in bandages. This sensation was most prominent in the right hand and arm and the left foot and leg. Approximately 2.5 months before this evaluation, the patient noted difficulty with fine motor control and recognizing objects by feel in both hands. Several weeks later, a sensation of imbalance occurred when the patient closed her eyes in the shower. She began to feel unstable while walking, although she did not fall. At about this time, numbness and tingling developed in both hands and arms, and an intermittent shooting pain in the hands that had no clear provocation or alleviation also developed. The symptoms progressed proximally in the right arm and left leg and affected the left arm and right leg in a milder fashion. She noted stiffness in her hands that was worse in the morning but never resolved completely. She was unable to discern whether the stiffness was confined to the joints or located elsewhere in the hands.

The symptoms increased in severity over the following weeks, and the patient had difficulty keeping her balance while dressing. She lost approximately 17 kg (37 lb) and saw a gastroenterologist. Esophagogastroduodenoscopy showed mild gastritis but was otherwise normal. Pathological examination of biopsy specimens of the stomach showed chronic gastritis; biopsy specimens of the antrum and the duodenum were normal. Chest radiography and contrast-enhanced computed tomography (CT) of the abdomen and pelvis were normal. A complete blood count, chemistry panel, and levels of serum electrolytes, thyrotropin, and C-reactive protein were normal. An antinuclear antibody (ANA)–based multiplex assay was positive (titer unknown), as was an assay for rheumatoid factor (381 IU per milliliter; normal value, <30). The erythrocyte sedimentation rate was 65 mm per hour (normal rate, <20), and the levels of vitamin B₁₂ and carcinoembryonic antigen were normal. A test for antibodies to *Borrelia burgdorferi* was negative.

The patient was referred to the neurology clinic at this hospital. She attributed her weight loss to a decreased appetite and difficulty feeding herself; she reported
no fevers, chills, nausea, vomiting, night sweats, diarrhea, or constipation. Three years before this evaluation, she had been evaluated by a neurologist at another facility because of episodes of numbness and tingling over the shoulders, extending up into the neck and below both breasts; these episodes lasted for weeks at a time. On the examination at that time, muscle strength, tendon reflexes, gait (including tandem gait), and coordination were normal. There was patchy loss of sensation of pinprick in the arms that did not conform to a dermatomal pattern and a loss of pinprick sensation on the back, from C2 to T6 on the right side and from T1 to C4 on the left. Sensation of light touch, joint position, and temperature was intact. Magnetic resonance imaging (MRI) of the spine with contrast medium was normal. No treatment was prescribed, and the symptoms resolved.

The patient had had mild Raynaud’s phenomenon for years, with no digital ulceration. She was married, lived with her husband, and worked in an office. She had a 25-pack-year history of smoking but had stopped smoking 1 year earlier, drank alcohol in moderation, and reported no use of illicit drugs or exposures to metals, toxins, or large doses of vitamin supplements. Her mother was 83 years of age and had a history of diabetes and breast cancer. Her father had died from cardiac disease at 81 years of age. Her brother and sister had ulcerative colitis, and her sister had myasthenia gravis. There was no other family history of neurologic diseases.

On examination, the patient was alert, cooperative, and articulate. The blood pressure was 100/75 mm Hg in a sitting position, the pulse 90 beats per minute and regular without orthostatic change, and the weight 57 kg (126 lb). Mood, memory, language, and cranial-nerve examinations were normal. There was very mild atrophy of the intrinsic muscles of both hands, without evidence of muscle atrophy or fasciculations elsewhere; muscle tone and strength were normal and symmetric. There was a subtle pronator drift of the right arm. The finger-to-nose and heel-to-shin tests were normal. Successive hand tapping on a table and finger tapping of the index finger on the interphalangeal joint of the thumb were both slightly slowed on the right side as compared with the left. The reflexes of the biceps, triceps, brachioradialis, and finger flexors were absent on the right side but 2+ on the left; patellar reflexes were 1+ on the right and absent on the left; ankle reflexes were absent bilaterally; and the plantar reflexes were flexor. The gait was normal, with a normal base. On Romberg examination, the patient swayed slightly, without faltering, and she reported feeling unsteady. She required support with tandem walking but was able to walk normally on both heels and toes. Her sense of vibration was severely diminished in the fingers and forearms on the right side, and less severely diminished on the left. The sense of joint position was severely impaired at the interphalangeal joint of the right thumb and was intact on the left. The sense of vibration was absent in the left great toe and trace in the right great toe, and it was severely reduced at the left ankle and nearly normal at the right ankle. She had difficulty identifying coins with either hand when her eyes were closed; this difficulty was more pronounced on the right side than the left. Pinprick sensation was diminished throughout the arms and legs but present on the trunk. Facial sensation of light touch and cold was normal. The nasal tickle was intact.

Diagnostic tests were performed.

**Differential Diagnosis**

Dr. David A. Chad: I cared for this patient and am aware of the diagnosis. This 52-year-old woman presented with a diffuse, progressive sensory disorder of 5 months’ duration, which involved her arms more than her legs. She had tightness, pins and needles (paresthesias), and “shooting pains,” a set of positive sensory symptoms that correlate with spontaneous discharges from sensory nerves. She also described a loss of feeling in her hands and feet, an inability to recognize objects by feel, diminished dexterity, and poor balance, all considered to be negative sensory symptoms correlating with the loss of sensory-nerve function or structure or both.

**Localizing the Neurologic Lesion**

Because of the predominant sensory symptoms, I considered disturbances in both the peripheral and central nervous systems. The physical examination did not provide support for a central localization of the lesion (i.e., one in the spinal cord, brain stem, or cerebrum). There was no evidence of a spinal sensory level, sphincter dysfunction, or long-tract findings such as hyperreflexia, spasticity, or Babinski signs. There were also no cranial-
nerve signs to suggest involvement of the brain stem and no mental-status or cognitive abnormalities to implicate the cerebral hemispheres. I therefore focused on the peripheral nervous system. I began the process of localization of the lesion by reviewing the organization of the peripheral component of the sensory system (Fig. 1A).

The sensory neurons, which reside in the dorsal-root ganglion, may be large cells with heavily myelinated axons or smaller cells with unmyelinated or thinly myelinated axons. Both types have one process directed centrally into the spinal cord and the other projecting to the periphery. The central projection of large dorsal-root ganglion neurons passes into the posterior columns, where it connects with second-order neurons in the medulla (nucleus grácilis and cuneatus), which project in turn to the thalamus. The central processes of smaller neurons enter the substantia gelatinosa and connect with a second-order neuron with axons that make their way to the thalamus along the spinothalamic pathway. The peripheral component of the large fibers travels to a joint or skin receptor (a pacinian corpuscle) (Fig. 1) to mediate the sensations of touch, pressure, and vibration; proprioception; and kinesthesia. The peripheral elements of small fibers terminate as free endings in the skin, mediating the sensations of pain and temperature. A peripheral-nerve lesion would be associated with evidence of lower motor-neuron dysfunction (weakness and atrophy), sensory loss (due to large- and small-fiber dysfunction), diminished tendon reflexes, and possibly dysautonomia, because some post-ganglionic sympathetic fibers reside within peripheral nerves.

Our patient had sensory loss stemming from dysfunction of both large and small nerve fibers. The sensory deficits were bilateral and asymmetric, with the greatest loss of all sensory capabilities in the right hand and the left foot. Her loss of tendon reflexes was strikingly asymmetric, and although she reported weakness, muscle atrophy and reduced strength were conspicuously absent. Rather, she had a subjective sense of weakness that stemmed from impairment in the sensory functions of proprioception and kinesthesia. There was no evidence of dysautonomia; for example, orthostatic hypotension and pupil abnormalities were absent. Finally, the sensory involvement affected her hands more than her feet, with prominent asymmetries. Thus, rather than the familiar pattern of graded, symmetric sensory loss — with sensory function improving in a distal-to-proximal direction and referred to as a “dying back” or length-dependent neuropathy — this patient had the picture of a neuropathy that was not length-dependent.

**CAUSES OF NON–LENGTH-DEPENDENT PERIPHERAL NEUROPATHY**

Several diagnostic possibilities came to mind. The first is a patchy or multifocal, acquired demyelinating process (a variant of chronic inflammatory demyelinating polyneuropathy described by Lewis and colleagues); the second is a vasculitic neuropathy; and the third is a dorsal-root ganglionopathy.

**The Lewis–Sumner Syndrome**

The Lewis–Sumner syndrome is a chronic asymmetric, demyelinating sensorimotor neuropathy. It initially affects the arms and later spreads to distal nerves in the legs, conforming to discrete peripheral-nerve distributions. It is insidious and slow in its tempo; pain and paresthesias are common. In the Lewis–Sumner syndrome, the arms are more involved than the legs, the deep-tendon reflexes are absent in the arms but preserved in the legs, and the sensory loss affects large-fiber functions such as proprioception with relative sparing of the sensations of pain and temperature. A defining attribute of this syndrome is demyelination, manifested as conduction block that can be identified by means of electrodiagnostic testing.

**Vasculitis**

A second consideration was vasculitis. Vasculitic neuropathy is characterized by an asymmetric, patchy pattern of peripheral-nerve deficits. Mononeuritis multiplex, the simultaneous or sequential involvement of individual, noncontiguous nerve trunks, evolves over a period of days to weeks and usually affects more than one limb. Untreated vasculitic processes lead over time to confluent and symmetric features, evolving into a distal, symmetric, sensorimotor, glove-and-stocking polyneuropathy. Careful examination is required to reveal asymmetries between limbs in the sensory and motor deficits. In some patients, vasculitic neuropathy is manifested as a painful, distal, small-fiber polyneuropathy. In this respect, vasculitic neuropathy offers a plausible explanation for this patient’s presentation. However, the complete absence of motor involvement would be unusual.
Dorsal-Root Ganglionopathy

The third diagnostic possibility, a dorsal-root ganglionopathy, is a purely sensory disorder in which the sensory neuron residing in the dorsal-root ganglion is targeted. If there were damage to these neurons, then wallerian degeneration (disruption of axons and myelin) would occur in the axonal extensions of these neurons, in the dorsal columns, and in the peripheral nerves (Fig. 1B). A dorsal-root ganglionopathy that affected the right cervical segments more than the left and the left lumbosacral segments more than the right could have caused the sensory deficits and tendon reflex asymmetries observed in this patient.

The first neurologic study performed to help refine the diagnosis was electrodiagnostic testing.

ELECTRODIAGNOSTIC TESTING

The striking finding revealed by the nerve-conduction studies was the marked attenuation of the
sensory responses (Table 1). Motor function was spared: median, ulnar, peroneal, and tibial motor-conduction studies, including F-wave responses, were uniformly normal. Moreover, the needle examination of selected upper- and lower-limb muscles was normal. There was no evidence of acquired demyelination that might have supported the Lewis–Sumner syndrome and no compelling evidence of multiple mononeuropathies that would have suggested a vasculitic neuropathic process.

The sensory deficits were notable for a non–length-dependent pattern, which is consistent with the physical-examination findings: the sensory amplitudes of the upper extremities (median, ulnar, and radial nerves) were as diminished as or more so than those of the lower extremities (sural nerves). By contrast, in a length-dependent polyneuropathy, one would expect sural-nerve responses to be affected to a greater extent than median-, ulnar-, or radial-nerve sensory responses. Therefore, these electrodiagnostic findings were consistent with a sensory neuronopathy.

The next diagnostic test was MRI, which was required to rule out concomitant disease in the central sensory pathways of the brain stem, cerebral hemispheres, and cerebellum. May we see the neuroimaging studies?

**NEUROIMAGING STUDIES**

Dr. Rajiv Gupta: MRI of the brain, performed with the administration of contrast medium, was normal, with no evidence of a demyelinating condition or abnormal enhancement. MRI of the cervical spine without contrast enhancement was performed. After a localizer sequence, sagittal T₁-weighted, T₂-weighted, and short-tau inversion recovery (STIR) sequences were obtained, followed by axial T₁-, T₂-, and T₂*–weighted images. T₂*-weighted images use gradient-echo sequences as opposed to the spin-echo sequences used for T₁- and T₂-weighted images. T₂*-weighted images allow for the use of thinner slices and can be acquired faster.

On a sagittal image through the cervical spine (Fig. 2A), where we have suppressed the signal in the paraspinal soft tissues and accentuated the cord parenchyma, there is a patchy signal abnormality involving the cord parenchyma. There is a suggestion that the areas of abnormal high intensity are primarily posterior in location and extend from approximately the C3 level down to the C6 and C7 levels. Axial T₂*-weighted images (Fig. 2B, 2C, and 2D) confirm that the cord parenchyma is abnormal. The signal abnormality mainly involves the posterior columns, with equivocal involvement of the central gray matter. Within the posterior columns, the distribution is non-confluent, is asymmetric from left to right, and does not affect the entire length of the dorsal columns. At some levels, both the fasciculus cuneatus and fasciculus gracilis are involved, whereas at other levels, only one of these two columns is affected. The degree of hyperintensity is also variable from level to level. Although there are several foci in the midcervical spine superiorly, close to the cervicomедullary junction, the cord parenchyma is essentially normal.

This abnormal T₂* hyperintensity does not inform us about a single underlying disease. The

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* The anatomical snuff box contains the cephalic vein, the radial artery, and the superficial radial nerve. The concavity of the snuff box is most pronounced during thumb extension.
Differential diagnosis includes subacute combined degeneration; demyelinating conditions such as multiple sclerosis; transverse myelitis; tabes dorsalis; myelopathies associated with the human immunodeficiency virus (HIV), sarcoidosis, or a connective-tissue disorder such as Sjögren’s syndrome; cord infarction; and dorsal-root ganglionopathy with secondary involvement of the posterior columns. Most of these disorders can be eliminated on the basis of the distribution of the signal abnormality and the clinical history. An absence of B₁₂ deficiency rules out subacute combined degeneration. The preferential involvement of the posterior columns and normal brain MRI findings make multiple sclerosis unlikely. From the clinical history, one can also rule out tabes dorsalis and myelopathies associated with HIV and sarcoidosis. The patchy, posterior distribution of the signal abnormality is not consistent with transverse myelitis or cord infarction. Dorsal-root ganglionopathy, with secondary involvement of the posterior columns, is a possibility. In this process, an insult to the dorsal-root ganglion containing the cell bodies of the unipolar first-order neurons results in central wallerian degeneration leading to patchy T₁- and T₂*-weighted signal abnormality confined to the posterior columns. Therefore, disease processes that can

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**Figure 2. Sagittal Short-Tau Inversion Recovery (STIR) and Axial T₂*-Weighted Images through the Parenchyma of the Cervical Spinal Cord.**

The STIR image (Panel A) shows patchy high-intensity signal abnormality involving the spinal cord, with a suggestion that the abnormal areas are primarily posterior in location. They extend from approximately C3 down to C6 level (lines at 1, 2, and 3 indicate levels corresponding to Panels B, C, and D, respectively). Axial T₂*-weighted images show patchy hyperintense signal abnormality involving the posterior columns (Panels B, C, and D; arrows). In comparison, a T₂*-weighted axial image from a normal spinal cord (Panel E) shows clear delineation of central gray matter, which is more hyperintense (light) than the surrounding white matter (dark).
affect the dorsal-root ganglia should be considered.

Dr. Chad: The clinical, electrophysiological, and neuroimaging studies all implicate a lesion in the dorsal-root ganglia.

CAUSES OF DORSAL-ROOT GANGLIONOPATHY

There is a short list of conditions that cause loss of sensory neurons: toxic neuronopathy, paraneoplastic sensory neuronopathy, the acute sensory neuronopathy syndrome, chronic idiopathic ataxic neuronopathy, and the neuronopathy associated with Sjögren’s syndrome. There was no indication in the patient’s history of a possible toxic neuronopathy (e.g., that induced by cisplatin or by pyridoxine at megavitamin levels). Similarly, there was no evidence to support a diagnosis of paraneoplastic sensory neuronopathy, which is typically characterized by an acute (days) to subacute (months) asymmetric onset, often in an upper limb, progressing to all limbs, the trunk, and the face. Approximately 90% of patients with paraneoplastic sensory neuronopathy have small-cell lung carcinomas, and in approximately 80% of these patients, tests for anti-Hu antibodies are positive. A chest radiograph and a test for anti-Hu antibodies were negative in our patient. A syndrome positive. A chest radiograph and a test for anti-Hu antibodies were negative in our patient. A syndrome can present with the subacute onset of acute sensory neuronopathy after antibiotic treatment, or a long-standing ataxic process, she did have findings consistent with connective-tissue disease (an elevated erythrocyte sedimentation rate and positive tests for rheumatoid factor and ANA). We suspected a link between the neuronopathy and an immunologic pathogenesis; specifically, Sjögren’s syndrome was proposed to explain the neuropathic features, imaging findings, and laboratory abnormalities. Indeed, patients with Sjögren’s syndrome can present with the subacute onset of non–length-dependent loss of kinesthesial and proprioception (often with autonomic insufficiency). Many also have non–length-dependent neuropathic pain. We referred the patient to Dr. Stone in rheumatology for further evaluation.

Sensory ganglionopathy associated with a connective-tissue disorder, suggestive of Sjögren’s syndrome.

THON Speedwell: Sensory ganglionopathy associated with a connective-tissue disorder, suggestive of Sjögren’s syndrome.

PATHOLOGICAL DISCUSSION

Dr. John H. Stone: This patient with a dorsal-root ganglionopathy also has positive tests for ANA and rheumatoid factor, an elevated erythrocyte sedimentation rate, and a normal C-reactive protein level. Although these serologic findings are compatible with a connective-tissue disorder, they are far from specific, and the patient had few elements in her history to implicate a specific autoimmune condition. Her Raynaud’s phenomenon had been relatively mild, without digital ulcers or ischemic crises. She had no history of sicca symptoms, rash, or arthralgias.

On physical examination in the rheumatology clinic, however, more telling findings were noted. The patient had telangiectasias over the bridge of her nose that blanched for several seconds on pressure (Fig. 3A). She had similar lesions on her lips (Fig. 3B) and fingers (Fig. 3C) and furrowing around her mouth. These telangiectasias and oral furrowing raised the question of systemic sclerosis, particularly the so-called limited subtype.

A complete serologic evaluation provided the answer. An ANA assay performed by means of immunofluorescence on HEp-2 cells showed a classic speckled pattern. In patients with connective-tissue disease, this pattern of immunofluorescence is generally caused by an antibody to one of four antigenic targets: the Ro, La, Sm, or ribonucleoprotein antigens. Further testing showed strongly positive anti-Ro antibodies (57.30 optical density [OD] units; reference value, <19.99). Although anti-Ro antibodies also occur in systemic lupus erythematosus, inflammatory muscle disease, systemic sclerosis, and overlapping connective-tissue diseases, this finding was also consistent with Sjögren’s syndrome. My rheumatologic diagnosis is an overlap syndrome, with features of Sjögren’s syndrome and limited systemic sclerosis.

Sjögren’s syndrome and limited systemic sclerosis.

Do these physical examination and serologic findings fit the neurologic picture? Sjögren’s syndrome is associated with a variety of sensory neuropathies, one of which is dorsal-root ganglionitis.17
In some patients with Sjögren’s syndrome, a profound sensory ataxia caused by dorsal-root ganglionitis is the initial manifestation of their underlying disease, occurring well before the classic sicca symptoms develop. Patients with dorsal-root ganglionopathy have a striking loss of proprioception and kinesthesia that often leads to the perception of weakness. Sensations of pain and temperature can also be affected, and autonomic neuropathy can be observed. Some patients have an abrupt onset of symptoms. In others, the progression is more indolent. Pathological examination of the dorsal-root ganglia in such patients has revealed T-cell infiltration and perivascular mononuclear infiltrates without necrotizing arteritis. Mild cases were characterized by degeneration of individual neurons; in very severe cases, there were few neurons left in the dorsal-root ganglia. The condition of approximately half the patients with Sjögren’s syndrome and dorsal-root ganglionopathy stabilizes with immunosuppressive therapy, but others have no improvement. A lack of response to therapy may be partly a function of how advanced the neurologic damage is at the time treatment is instituted.

**Management and Follow-up**

The treatment approach in our patient was intended to halt damage to the dorsal-root ganglia as quickly as possible. We used an empirical regimen comprising both a broad-spectrum immunosuppressive approach with high doses of glucocorticoids and cyclophosphamide, as well as a targeted treatment strategy designed to deplete B cells. We administered a 6-month course of prednisone beginning at a dose of 60 mg per day, oral cyclophosphamide (100 mg per day), rituximab (1000 mg intravenously in two doses, 15 days apart), and pneumocystis prophylaxis with single-strength trimethoprim–sulfamethoxazole. Pain was the most difficult problem, and the patient was followed by staff in the pain clinic. She had little response to gabapentin, had a good response to pregabalin but with unacceptable side effects, and is currently taking nortriptyline and lamotrigine, with hydrocodone as needed for breakthrough pain. The patient had no other adverse effects from the medication, and her symptoms stabilized. Cyclophosphamide was discontinued at 6 months. Her peripheral B cells became depleted promptly, as expected, with rituximab and had not reconstituted by 1 year. The anti-Ro antibody titer became negative (0.71 OD units). Follow-up electrodiagnostic studies performed 1 year after the course of treatment revealed a slight increase in the amplitude of the left radial and left sural responses (Table 1).

**Final Diagnosis**

Sjögren’s syndrome with dorsal-root ganglionitis.

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**Figure 3. Clinical Photographs.**

Telangiectasias were present at multiple sites: over the nasal bridge (Panel A), on the lips (Panel B), and on the fingers (Panel C). The telangiectasias blanched with pressure and refilled within seconds.
**ADDENDUM**

Drs. Chad and Stone: Two years after the onset of the patient’s symptoms, and 7 months after the conference reported here, follow-up MRI of the spine, performed to assess the status of the posterior columns, revealed an incidental right paratracheal mass. Examination of a needle-biopsy specimen, obtained after this Case Record was submitted for publication, showed small-cell lung cancer. After the diagnosis of small-cell lung carcinoma, a repeat assay for anti-Hu antibodies was negative, and careful review of the patient’s initial MRI scan did not reveal any evidence of the paratracheal mass.

When we met the patient for the first time, we considered the possibility of a paraneoplastic process and ordered testing for anti-Hu antibodies, which was negative. Guidelines for the evaluation of patients who have anti-Hu antibodies and are suspected of having a paraneoplastic syndrome are clear and include CT and other imaging studies to detect an underlying tumor. Guidelines for patients with negative tests for anti-Hu antibodies are less clear but suggest evaluation for evidence of another disorder, with imaging studies recommended if no other cause is found and the neurologic condition is worsening. In this case, the strongly positive test for anti-Ro antibodies, approximately 18% are not. The majority of such cases are associated with anti-Hu antibodies, approximately 18% are not. Thus, it is conceivable that the small-cell lung carcinoma contributed in some way to the clinical expression of dorsal-root ganglionopathy in this patient. The resolution of the anti-Ro antibodies coincided with slight improvement in the neurologic examination and sensory-nerve-conduction studies, supporting a neurologic complication of Sjögren’s syndrome as the cause in this case. Patients with paraneoplastic sensory neuronopathies, in contrast, typically have progressive downhill courses. Because much remains unknown about the cause of dorsal-root ganglionitis, however, we thought it important to provide this addendum to the case.

This case was presented at the Rheumatology Grand Rounds. No potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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