Sensory Guillain–Barré syndrome

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Article abstract—*Objective:* To report eight cases of sensory Guillain–Barré syndrome (GBS). *Background:* The concept of sensory equivalent to ascending paralysis of GBS was raised in 1958, and the diagnostic criteria for a sensory loss and areflexia variant of GBS were proposed in 1981. However, clinical cases meeting these criteria have been relatively scarce. *Methods:* During a 13-year period between 1986 and 1999, the authors collected eight cases of an acute sensory demyelinating neuropathy that met most of the proposed diagnostic criteria of a sensory variant of GBS. *Results:* In all patients, sensory neuropathy was sudden at onset and peaked to maximal deficit within 4 weeks. In five (63%) cases, there was an antecedent viral illness. All patients had objective sensory loss and diminished or absent reflexes. None showed any muscle weakness. In all four patients in whom the spinal fluid was examined during the first 4 weeks, there was albuminocytologic dissociation. All of the patients had electrophysiologic evidence of demyelination in at least two nerves. Demyelination was demonstrated in motor nerve conduction in seven patients and in sensory nerve conduction in one, indicating that motor nerve conduction studies were the key for the diagnosis of demyelinating neuropathy. All patients had sensory nerve conduction abnormalities in at least one nerve. Three patients responded to immunotherapies. All had a favorable outcome, with a monophasic course of disease and no sign of relapse. *Conclusion:* The current study confirms the existence of sensory GBS.

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Guillain–Barré syndrome (GBS), or acute inflammatory demyelinating polyradiculoneuropathy, is characterized by ascending motor paresis peaking within 4 weeks, diminished or absent muscle stretch reflexes, sensory symptoms with minimal objective sensory loss, electrophysiologic evidence of a demyelinating neuropathy, and CSF albuminocytologic dissociation.[1] Variants of this syndrome include pharyngeal–cervical–brachial, Miller–Fisher, paraparetic, ptosis without ophthalmoplegia, acute motor and sensory axonal neuropathy, and acute motor axonal neuropathy.[2,3]

In 1958, Wartenberg[4] discussed the concept of a sensory equivalent to the ascending paralysis of GBS. Although Asbury[1] provided diagnostic criteria for a sensory loss and areflexia variant in 1981, reported clinical cases meeting these criteria have been scarce. Thus, there has been some question as to whether sensory GBS exists.[5] We reported four cases of sensory GBS in 1990 in abstract form.[6] In the current article, we report eight cases of an acute, monophasic, sensory neuropathy accompanied by reduced muscle stretch reflexes, high spinal fluid protein, and nerve conduction features of demyelination, which meets most of the diagnostic criteria of a sensory variant of GBS.

Materials and methods. During a 13-year period between 1986 and 1999, we observed eight patients with an acute demyelinating sensory neuropathy at the University

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of Alabama at Birmingham. All of these patients met the following eight diagnostic criteria: 1) acute onset of symmetric loss; 2) peak deficit achieved within 4 weeks; 3) diminished or absent reflexes; 4) normal motor strength; 5) nerve conduction evidence of demyelination in at least two nerves; 6) monophasic course; 7) no other known cause for neuropathy; and 8) no family history of neuropathy. The ninth diagnostic criterion—elevated CSF protein during the acute phase of disease—was met in four cases. Neurologic evaluation of each of the eight patients was verified by the same clinician (S.O. and G.C.). In 1999, a follow-up interview was conducted via telephone by one of the authors (C.L.) regarding the patients' recovery status, recurrence of any novel neurologic symptoms, and any interval medical diagnoses that might explain an episode of acute sensory neuropathy, such as malignancies, endocrine/nutritional abnormalities, HIV positivity, or connective tissue disorders.

Routine nerve conduction studies (NCS) were performed using standard surface electrode placement as previously described.[7] All patients underwent testing of sural, peroneal, posterior tibial, median, and ulnar nerves. A monopolar needle electromyogram (EMG) was performed in all patients on a minimum of three leg muscles, and four patients had H-reflex testing.[7] In Patient 8, the near-nerve needle sensory NCS of the plantar nerve was performed.[8] Electrophysiologic criteria of demyelination were based on the following: 1) prolongation of terminal latency and F-wave by more than 150% of the normal mean; 2) nerve conduction velocity (NCV) slowing by more than 40% below the normal mean; 3) conduction block (more than 50% drop in the amplitude and area in the proximal compound muscle action potential); and 4) dispersion phenomenon (abnormal compound muscle action potential [CMAP] or

Table 1 *Summary of clinical and laboratory features*

Patient no.	Age, y/ sex	AI	Onset site	Time to peak deficit	Sensory loss	Reflexes	CSF protein, mg/dL:cells, WBC/mm^3 (time of lumbar puncture)	Treatment/outcome (duration of follow-up)
1	73/M	−	Leg	7 d	Pinprick, vibration	Absent AJ	71:2 (1 mo)	Asymptomatic in 3 mo (4.5 y)
2	26/M	+	Feet, hand	4 d	Pinprick, vibration	Decreased	40:0 (4 mo)	Asymptomatic in 6 mo (2.5 y)
3	65/M	−	Lower leg	6 d	Light touch, vibration	Absent AJ, decreased others		Asymptomatic in 2 mo (10.5 y)
4	21/M	+	Lower leg	7 d	Vibration	Decreased AJ/ triceps reflex	114:7 (3 wk)	Asymptomatic with steroid in 1 mo (1.5 y)
5	70/M	−	Toes	2 d	Pinprick, vibration	Absent		Static sensory loss (3.5 y)
6	50/M	+	Hands	2 wk	Pinprick, vibration, position, sensory ataxia	Absent	107:10 (2 wk)	Improved with IVIG, failed with steroid, paresthesia in the feet 1 y (2 y)
7	48/M	+	Feet	3 wk	Pinprick, vibration, position, sensory ataxia	Absent	106:8, high IgG, (1 wk)	Improved with PE, failed with IVIG and steroid, paresthesia in the feet 7 mo (2 y)
8	41/M	+	Toes	10 d	Pinprick, light touch, vibration	Decreased AJ		Improved, paresthesia in the toes 7 mo (2 y)

AI = antecedent illness; WBC = white blood cells; AJ = ankle jerk; PE = plasma exchange; IVIG = IV immunoglobulin.

compound nerve action potential [CNAP] with multiple phases and prolonged duration).[7]

In four patients, CSF was obtained by lumbar puncture for diagnostic purposes within the first 4 weeks of disease. One patient had a lumbar puncture 3 months after onset, one patient refused the test, and in two others, who were evaluated 7 and 10 months after onset of disease, the CSF evaluation was not performed. All of these patients had basic peripheral neuropathy workups, which included thyroid and rheumatology profiles, vitamin B12 and folic acid, hemoglobin A1C, erythrocyte sedimentation rate, and immunoelectrophoresis of serum protein by immunofixation. Additional studies were performed in some patients, depending on the findings.

Reports of three illustrative cases. Patient 4 was a 21-year-old man who was well until 3 weeks before evaluation, when he developed diffuse pruritus after a day of hiking in the local woods. The next day, he developed rhinorrhea, malaise, a nonproductive cough, and a low-grade fever. One week later, he experienced the acute onset of prickly dysesthesias from his knees distally. Over the ensuing week, his symptoms ascended to his proximal thighs and arms. He also experienced transient "pins-and-needles" dysesthesia across his anterior thorax. This evolved to hyperesthesia over the following week, at which time he presented for evaluation. His neurologic evaluation was remarkable only for mild symmetric vibratory loss in the toes and diminution of Achilles and triceps reflexes. Laboratory workup was remarkable only for elevation of CSF protein (table 1). Initial electrophysiologic examination (table 2) demonstrated a demyelinating distal motor neuropathy, with a normal EMG. After refusing IV immunoglobulin treatment, the patient was treated with one course of prednisone (60 mg/day for 1 week, gradually

tapering over the next 3 weeks) and had subsequent complete resolution in symptoms and signs within 4 weeks.

Patient 6 was a 50-year-old man who experienced 1 week of fever, chills, and malaise about 3 months before evaluation for neuropathy at the University of Alabama at Birmingham. Two weeks after his constitutional symptoms, he had onset of bilateral medial hand numbness. A few days later, he developed paresthesia on the soles of his feet. Over the next 2 weeks, he had a progression of the symptoms to his distal forearms and thighs symmetrically. At that time, his local neurologist performed an extensive initial diagnostic workup before prescribing 20 mg of prednisone daily. Because of little improvement after 1 month, he was given 5 days of a standard-dose IV immunoglobulin. He gradually improved over the following 3 weeks, despite developing a deep vein thrombosis in the right calf. Neurologic exam at this time was notable for diminished distal pinprick/vibratory sensation bilaterally and loss of toe proprioception, with a concomitant Romberg sign in the lower extremities. His gait was ataxic and he had global areflexia. NCS was indicative of widespread demyelination with conduction block in the posterior tibial nerves (see table 2). He received no further immunosuppressants and reported a gradual recovery, such that 1 year after onset of symptoms, he reported paresthesia from the mid-feet distally.

Patient 7 was a 48-year-old man who developed a febrile illness with severe skin rash after returning from a trip to Europe. He was treated with doxycycline. The next day, he developed numbness on the soles of both feet, which progressed over the next week to the knees, with walking difficulty due to poor balance. Neurologic examination showed proprioception and vibratory loss up to the knees without pinprick loss below the knees, diffuse

Table 2 *Summary of nerve conduction data*

Nerve conduction parameter	No. of patients
Abnormal NCS, n	8
Abnormal motor nerve conduction*	7
Prolonged terminal latency	7
Slow NCV	6
Low CMAP	5
F-wave prolongation or NP	7
Abnormal sensory nerve conduction†	8
No potential	4
Slow NCV	8
Low CNAP amplitude	6
Abnormal mixed nerve conduction‡	5
No potential	1
Slow NCV	4
Low CNAP amplitude	4
Evidence of demyelination§	
Sensory nerve conduction	2 (5, 8)¶
Mixed nerve conduction	0
Motor nerve conduction	7 (except 8)
Abnormal temporal dispersion	2 (PT in 3 & 7)
Conduction block	3 (PT in 3 & 6; P in 7)
<60% of normal mean NCV	2 (U in 2; PT in 6)
>150% of normal mean terminal latency	7 (except 8)
>150% of normal mean F-wave latency	2 (M in 2; all in 6)

Additional material related to this data can be found on the *Neurology* Web site.

*Median, ulnar, peroneal, and posterior tibial nerves were tested.
† Median, ulnar, and sural nerves were tested.
‡ Median and ulnar nerves are tested.
§ Number and letters in parentheses represent patient number and nerve with abnormal nerve conduction.
¶In Patient 5, sural NCV was 22.7 m/s; in Patient 8, dispersion phenomenon was found in the I digital and I-II interdigital nerves of plantar nerves with the near-nerve needle sensory nerve conduction. NCV in the I digital nerve was 22.6 m/s and in the I-II interdigital nerve, 24.4 m/s.

NCS = nerve conduction study; CMAP = compound muscle action potential; NCV = nerve conduction velocity; NP = no potential; CNAP = compound nerve action potentials; PT = posterior tibial; U = ulnar; P = peroneal; M = median.

areflexia, and sensory ataxic gait. MRI scan and lumbar myelogram were normal. CSF showed 144 mg/dL in protein and eight mononuclear cells. NCS showed demyelinating neuropathy (see table 2). Despite treatment with IV immunoglobulin and prednisone for 3 days, the numbness extended up to his hips and hands. Neurologic exam at the University of Alabama at Birmingham 3 weeks after onset confirmed severe sensory ataxia, with markedly positive Romberg test and abnormality on heel-to-shin tests. With one course of plasma exchange, his symptoms began to improve. This improvement continued over the next 7

months, by which time he was completely asymptomatic except for slight pinprick loss below the right knee.

Results. *Clinical features.* All eight patients were men aged 21 to 73 years (see table 1). A preceding viral illness occurred in five patients. The onset of sensory symptoms (numbness, tingling, or burning sensations) was acute in all patients, occurred solely in the lower extremities in six patients, solely in the upper extremities in one patient, and in all four extremities in one patient. The duration of symptoms to maximum peak ranged from 2 days to 3 weeks. Among sensory signs, there was vibratory loss in all patients, pinprick loss in six, light touch loss in two, proprioceptive loss in two, and sensory ataxia in two. Reflexes were either absent or diminished in all patients: diffusely absent in three, diffusely diminished in two, and three had diminished ankle jerk.

Laboratory findings. All of four patients in whom CSF was collected within the first month of illness had albuminocytologic dissociation (see table 1). In one patient, who had CSF collected in the fourth month of illness, the findings were normal. All patients had essentially normal routine chemistry results, complete blood count, liver function tests, folate, vitamin B12, rapid protein reagent, immunofixation serum protein analysis, erythrocyte sedimentation rate values, and rheumatology and thyroid profiles. Heavy metal results were normal in the three patients assessed. Serum autoantibodies (MAG, GM1, GQ1b, GD1b, anti-Hu, and SGPG) were normal in four tested cases. In individual cases, Lyme antibody, vitamin E, CRP, phytanic acid, and arylsulfatase-A values were normal.

Electrophysiologic findings. The NCS was performed within 4 weeks of symptom onset in four patients, within 12 to 16 weeks in two, and over 28 weeks later in two (see table 2) (additional material related to this article can be found on the *Neurology* Web site. Go to www.neurology.org and scroll down the Table of Contents for the January 9 issue to find the title link for this article). There was electrophysiologic evidence of demyelination in at least two nerves in all cases. Evidence of demyelination was observed in motor nerve conduction in seven patients and in the sensory nerve conduction in two patients. Near-nerve needle sensory NCS of the plantar nerves was needed to document demyelination in Patient 8 because the routine NCS was normal.

Motor NCS was abnormal in seven patients. Evidence of demyelination was most prominently observed in the terminal latency—in the peroneal nerve in seven patients, in posterior tibial and median nerves in four patients, and in the ulnar nerve in three patients. Though a slow NCV was observed in posterior tibial and peroneal nerves in five patients and in median and ulnar nerves in three patients, evidence of demyelination was observed in only two patients. Conduction block (>50% drop in the proximal amplitude compared with the distal CMAP amplitude) was present in posterior tibial nerves in two patients and in the peroneal nerve in one patient.[7] Dispersion phenomenon was found in the posterior tibial nerve in two patients. In Patients 6 and 7, evidence of demyelination was widespread and easily recognized, whereas in other five patients it was spotty and confined to the terminal latency.

Sensory nerve conduction was abnormal in at least two nerves in seven patients and in one nerve in one patient.

Mildly slow NCV was the most common finding, and was observed in all cases. Sensory CNAP was absent in four cases and low CNAP amplitude were seen in six cases. Mixed nerve conduction was abnormal in five patients, in the ulnar nerve in four patients, and in the median nerve in three patients. F-wave latency was prolonged in ulnar and median nerves in four patients and in peroneal and posterior tibial nerves in seven patients. Only two patients had an F-wave latency in the range of demyelination. H-reflex was abnormal in all of four tested patients. Needle EMG studies were essentially normal in all cases, except for a high-amplitude motor unit potential in some tested muscles in three cases.

Treatment response and outcome. Three patients were treated with immunotherapies. Patient 4 had a course of prednisone with complete recovery. Patient 6 received small-dose (20 mg/day) prednisone initially without any benefit, but then had satisfactory improvement with one course of IV immunoglobulin. Patient 7 initially received steroid and IV immunoglobulin therapy without any benefit but began to improve with plasma exchange, with subsequent satisfactory recovery. Of five patients who received no immunotherapy, three completely recovered and two have had a stable deficit. No patients experienced a relapse of neuropathy during follow-up periods of 2 to 11 years.

Discussion. Among the classic diagnostic criteria of GBS, progressive motor weakness of more than one limb has been a feature.[1] Thus, none of our cases meet these criteria. Asbury[1] described the following diagnostic criteria for GBS variants with sensory loss and areflexia: 1) the onset must be rapid; 2) the distribution must be widespread and symmetric; 3) recovery must be complete or nearly so; 4) CSF protein must be elevated with few or no cells; and 5) electrodiagnostic results must be characteristic of a demyelinating process in the peripheral nerve.

Four of our cases met all the diagnostic criteria for the sensory variant GBS,[1] and the other four met four of the five diagnostic criteria. In all four patients who had CSF collected in the first 4 weeks, albuminocytologic dissociation, the most helpful diagnostic laboratory feature in GBS, was observed. All had electrophysiologic evidence of demyelination, the other helpful diagnostic laboratory feature, in at least two nerves. Demyelination was most convincing in motor nerve conduction in seven patients and in sensory nerve conduction in one, indicating that motor NCS is the key for the diagnosis of demyelinating neuropathy. Even in motor NCS, however, the obvious evidence of demyelination was recognized in only two patients. In others, evidence of demyelination was confined to the terminal latencies, as observed in previously reported cases.[9-11] This is understandable, given the nature of the clinical presentation of sensory GBS. The difficulty in demonstrating demyelination in sensory nerve conduction is due to the technical limitation of sensory NCS performed with surface electrodes.[7] Certainly, this can only be achieved by the near-nerve sensory nerve conduction as demonstrated in Patient 8, and

in some patients with chronic sensory demyelinating neuropathy (CSDN).[12] In general, our diagnostic criteria of sensory GBS were much stricter and better defined than those of Asbury.[1]

Following the traditional nosologic designation based on clinical features, we believe that the designation of sensory GBS in these cases is justified, even in the electrophysiologic presence of motor fiber demyelination. This is because the electrophysiologic data do not necessarily correlate with neuropathic symptoms and deficits,[7,13] and GBS is often placed in the category of motor neuropathy, despite the fact that 58 to 76% of patients have sensory NCS abnormalities.[14,15]

A chronic counterpart of sensory GBS already exists in CSDN as a sensory variant of chronic inflammatory demyelinating polyradiculoneuropathy. This condition is a sensory neuropathy characterized by subacute or chronic (>4 weeks) development of neuropathy, demyelination in the NCS, a high CSF protein in 75% of cases, and responsiveness to immunotherapy.[16,17] Evidence of demyelination is easily found electrophysiologically in motor nerves and pathologically in sural nerves. Thus, in CSDN, motor fiber demyelination is a common finding and a key for diagnosis of demyelinating neuropathy, as noted as in our cases.

We believe that the diagnosis of sensory GBS is important because it should help the clinician in planning potential immunotherapies, as noted in three of our cases, and offers a generally favorable prognosis to patients, in contrast to the slow but steady progression usually associated with idiopathic sensory neuropathy.[18,19] The differentiation of sensory GBS from acute sensory neuronopathy is also important in view of the poor recovery rate in the latter.[5,20] Sensory neuronopathy can be differentiated by the classic pattern of nerve conduction abnormality—absent sural nerve action potentials in the presence of normal motor nerve conduction.[7] The quick onset and lack of progression should allow differentiation between paraneoplastic sensory neuronopathy and sensory GBS.[21]

Historically, there has been ongoing debate concerning this disease since 1946.[22-24] In three reports, 12 cases were cited as examples of sensory GBS. However, these cases hardly meet the current diagnostic criteria of the sensory variant of GBS according to our review.

In 1980, three patients with acute sensory neuronopathy were reported. This sensory neuronopathy was characterized by sudden numbness and pain over the entire body a few days after initial antibiotic treatment for a febrile illness, profound sensory ataxia, areflexia, absent sensory nerve potentials, a high CSF protein, and severe static, residual sensory deficits.[20] The authors concluded that this represented an acute sensory neuronopathy with lesions in the dorsal root ganglia, rather than a demyelinating neuropathy.

In 1988, a patient who presented with severe sen-

sory loss and ataxia, total areflexia, elevated CSF protein with pleocytosis, and terminal latency in the demyelinating range in the median motor nerve was reported.[10] At autopsy, there was extensively lymphocytic infiltration of nerves and posterior roots, with sparing of the anterior roots. Teased fiber preparation of nerve showed a demyelinating lesion. This case was cited as the first case of a sensory GBS with autopsy in the subsequent literature. However, the patient also had mild weakness in the triceps, finger extensors, intrinsic muscles of the hands, hip flexors, and foot dorsiflexors. Thus, though histologic and electrophysiologic criteria of GBS were met, this case was not an example of sensory GBS because of presence of mild motor weakness.

In 1990, we reported four cases of sensory GBS in an abstract.[6] In the same year, 42 patients who had acute or subacute sensory neuropathy were reported.[5] Their cases were characterized by an absence of sensory CNAP potential, normal CSF findings, loss of large myelinated fibers, and axonal atrophy without inflammation in the sural nerve biopsy. The authors concluded that these cases represented an immune-mediated or vascular sensory neuropathy or radiculopathy, rather than sensory GBS.

In 1992, a case of sensory GBS was reported.[9] The patient developed sensory ataxia with areflexia and proprioception loss over 2 to 3 days following a viral illness, high CSF protein, absent sensory SNAP but demyelinating value of terminal latency, and a 39% decrease in the CMAP on proximal stimulation of the median nerve. The patient had a complete recovery in 1 month. This case clearly meets the strict diagnostic criteria of the sensory variant of GBS as outlined above.[9] In 1996, two cases of acute sensory neuropathy with prolonged terminal latency in the demyelinating range and good recovery in a few months were reported. CSF protein was not examined in these cases.[11]

From this review, it is clear that acute sensory neuropathy represents two clinical syndromes: acute sensory neuronopathy involving the dorsal root ganglia, and sensory GBS, an acute demyelinating neuropathy that presents clinically with only sensory peripheral nerve involvement. The former entity is recognized by the sensory neuronopathy pattern (absent sensory CNAP in the presence of normal motor nerve conduction) in the NCS, and the latter predominantly by the demyelinating motor nerve conduction, especially in the terminal latency. CSF protein is not helpful in distinguishing sensory neuronopathy from sensory GBS because it may be elevated in both disorders.[20] The most important distinction between these two entities lies in the clinical outcome: in acute sensory neuronopathy the recovery rate is poor, whereas in sensory GBS the recovery is good, as noted in our cases.

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