Guillain-Barré Syndrome

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Guillain-Barré syndrome (GBS) is a group of autoimmune syndromes consisting of demyelinating and acute axonal degenerating forms of the disease. Nerve conduction study helps differentiate the heterogeneous subtypes of GBS. Patients exhibit a progressive paralysis that reaches a plateau phase. In most patients, resolution is complete or near complete. Mortality from GBS most often is associated with dysautonomia and mechanical ventilation. GBS usually is associated with an antecedent infection by one of several known pathogens. Cross-reactivity between the pathogen and the nerve tissue sets up the autoimmune response. Treatment consists of supportive care, ventilatory management (in about one third of patients), and specific therapy with intravenous immunoglobulin or plasmapheresis. Consultation with a neurologist is suggested. (Am Fam Physician 2004;69:2405-10. Copyright© 2004 American Academy of Family Physicians)

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See page 2321 for levels of evidence definitions.

Guillain-Barré syndrome (GBS) is an eponym for a heterogeneous group of immune-mediated peripheral neuropathies. A feature common in all GBS variants is a rapidly evolving polyradiculoneuropathy preceded by a triggering event, most often an infection.[1] GBS generally manifests as a symmetric motor paralysis with or without sensory and autonomic disturbances.

Population-based surveys attempting to document the annual incidence of GBS have been conducted in various countries worldwide and generally are in agreement on a rate of 1 to 3 per 100,000 persons annually.[2,3] GBS occurs in all age groups, although rarely in infants, and the incidence varies. From birth to 30 years, the annual incidence is fairly uniform at 1.3 to 1.9 per 100,000. Peaks are noted in late adolescence and young adulthood, as well as in the elderly. The first peak likely correlates with increased risk of cytomegalovirus and *Campylobacter jejuni* infection. The reason for the peak in the elderly is unknown but is postulated to be caused by failing immune suppressor mechanisms.[2] Another variation in incidence is found in pregnant and postpartum women. According to a Swedish epidemiologic study,[4] the incidence appears to be lower during pregnancy with an increase in the months immediately after delivery.

The patient with GBS typically presents with weakness accompanied by tingling dysesthesias in the extremities. This weakness is prominent in the proximal muscles; legs are more often affected than arms. Paresthesias occur, spreading proximally but seldom extending past the wrists and ankles. Deep tendon reflexes disappear within the first few days of symptom onset.

The progressive phase of the syndrome lasts from a few days to four weeks. About 73 percent of patients reach a nadir of clinical function at one week and 98 percent at four weeks.[5] The progressive phase is followed by a plateau phase of persistent, unchanging symptoms. Improvement will begin within days of the plateau. The time to resolution of symptoms varies among patients.

Cranial nerve involvement may affect airway maintenance, facial muscles, eye movements, and swallowing. Patients should be hospitalized for observation. Approximately 30 percent of patients will require ventilatory assistance at some time during the illness.[2] Poor outcomes primarily are associated with the increasing severity of disease, with a mortality rate as high as 20 percent occurring primarily in patients who require mechanical ventilation.[6]

Pain, another common feature of GBS, is seen in approximately one half of all patients and is sometimes described as severe, occur-

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ring with even the slightest of movements. Pain is most severe in the shoulder girdle, back, and posterior thighs.[7,8] Patients complain of a deep aching pain in the weakened muscles that is similar to the muscular discomfort experienced following exercise. Pain may be accompanied by muscle cramps, and it is most severe at night.

Diagnosis

GBS is the most common type of rapidly evolving, generalized peripheral nervous system disorder. However, several disorders can be mistaken for GBS, particularly early in its course *(Table 1)*.[7]

The diagnosis of GBS is based on typical clinical features; electrodiagnostic examination and examination of the cerebrospinal fluid (CSF) can aid in the diagnosis *(Table 2)*.[7,9] Electrodiagnostic findings suggestive of GBS include an absent H reflex, low amplitude or absent sensory nerve action potentials, an abnormal F wave, and other less frequent abnormalities. These findings may allow earlier intervention with specific treatments, but a definitive diagnosis is usually not possible until the fifth day after the onset of symptoms.[10]

Characteristic CSF findings consist of elevated protein

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TABLE 1
Differential Diagnosis and Characteristics Differing from Guillain-Barré Syndrome

Basilar artery occlusion (asymmetric limb paresis)
Botulism (descending paralysis)
Heavy metal intoxication (confusion, psychosis, organic brain syndrome)
Hypophosphatemia (irritable, apprehensive, hyperventilation, normal cerebrospinal fluid)
Metabolic myopathies (cerebral and cerebellar symptoms)
Myasthenia gravis (weakness and fatigue that improves with rest)
Neoplastic meningitis (asymmetric spastic paralysis)
Neurotoxic fish poisoning (spontaneous recovery within 24 hours)
Paraneoplastic neuropathy (chronic)
Poliomyelitis (purely motor disorder with meningitis)
Polymyositis (chronic, affects proximal limb muscles)
Spinal cord compression (asymmetric)
Tick paralysis (sensory changes absent, normal cerebrospinal fluid)
Transverse myelitis (abrupt bilateral leg weakness, ascending sensory)
Vasculitic neuropathies (mononeuropathy)

Information from Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992;326:1130-6.

TABLE 2
Diagnostic Criteria for Typical Guillain-Barré Syndrome

Features required for diagnosis
Progressive weakness in both arms and legs
Areflexia
Features strongly supporting diagnosis
Progression of symptoms over days, up to four weeks
Relative symmetry of symptoms
Mild sensory symptoms or signs
Cranial nerve involvement, especially bilateral weakness of facial muscles
Recovery beginning two to four weeks after progression ceases
Autonomic dysfunction
Absence of fever at onset
High concentration of protein in cerebrospinal fluid, with fewer than 10 cells per cubic millimeter
Typical electrodiagnostic features
Features excluding diagnosis
Diagnosis of botulism, myasthenia, poliomyelitis, or toxic neuropathy
Abnormal porphyrin metabolism
Recent diphtheria
Purely sensory syndrome, without weakness

Reprinted with permission from Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992;326:1130-6 and Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27(suppl):S21-4.

TABLE 3
Subtypes of Guillain-Barré Syndrome

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
Autoimmune disorder, antibody mediated
Is triggered by antecedent viral or bacterial infection
Electrophysiologic findings demonstrate demyelination.
Inflammatory demyelination may be accompanied by axonal nerve loss.
Remyelination occurs after the immune reaction stops.
Acute motor axonal neuropathy (AMAN)
Pure motor axonal form of neuropathy
Sixty-seven percent of patients are seropositive for campylobacteriosis.
Electrophysiologic studies are normal in sensory nerves, reduced or absent in motor nerves.
Recovery is typically more rapid.
High proportion of pediatric patients
Acute motor sensory axonal neuropathy (AMSAN)
Wallerian-like degeneration of myelinated motor and sensory fibers
Minimal inflammation and demyelination
Similar to AMAN except AMSAN affects sensory nerves and roots
Typically affects adults
Miller Fisher syndrome
Rare disorder
Rapidly evolving ataxia, areflexia, mild limb weakness, and ophthalmoplegia
Sensory loss unusual, but proprioception may be impaired.
Demyelination and inflammation of cranial nerve III and VI, spinal ganglia, and peripheral nerves
Reduced or absent sensory nerve action potentials, tibial H reflex is usually absent.
Resolution occurs in one to three months.
Acute panautonomic neuropathy
Rarest of all the variants
Sympathetic, parasympathetic nervous systems are involved.
Cardiovascular involvement is common (postural hypotension, tachycardia, hypertension, dysrhythmias).
Blurry vision, dry eyes, and anhydrosis
Recovery is gradual and often incomplete.
Often combined with sensory features

Information from references 2, and 12 through 16.

(higher than 0.55 g per dL [5.5 g per L]) without pleocytosis (abnormal number of cells in the CSF). CSF is often normal when symptoms have been present for less than 48 hours, but by the end of one week the level of CSF protein is elevated. An increased white blood cell count in CSF (10 to 100 per mm^3 [10 to 100 $\times$ 10^6 per L]) in a patient with typical GBS symptoms increases the possibility of Lyme disease, neoplasia, human immunodeficiency virus (HIV) infection, sarcoid meningitis, or other diseases.[7,11]

GBS has five distinct subtypes: acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), Miller Fisher syndrome, and acute panautonomic neuropathy. The axonal forms are generally thought to have poorer prognoses, indicating a need for determining the specific subtype. These subtypes are distinguished electrodiagnostically and pathologically *(Table 3)*.[2,12-16]

The diagnosis of Guillain-Barré syndrome is based on typical clinical features, electrodiagnostic examination, and examination of the cerebrospinal fluid.

Antecedent Events

Acute infectious illnesses are well-known antecedent events in two thirds of patients who have GBS. Cross-reactivity between the pathogen and the nerve tissue sets up the autoimmune response. Patients commonly report a respiratory tract infection or gastroenteritis that resolved when the neuropathy began. Campylobacteriosis is the most common precipitant in GBS.

In 1995, results of a case-control study of 103 patients with GBS found that 26 percent of affected persons were positive for *C. jejuni*, compared with 2 percent of household controls and 1 percent of age-matched controls. Seventy percent of the patients with *C. jejuni* infection reported a diarrheal illness within 12 weeks of neurologic sequelae.[17] Other antecedent infections include cytomegalovirus, HIV, Epstein-Barr virus, and varicella-zoster virus.[2]

Electrophysiologic classification demonstrated that the subtypes AMAN and AMSAN occurred more frequently in the *C. jejuni*-infected patients with GBS than the subtype AIDP.[17] *C. jejuni* infection is associated with slower recovery, axonal degeneration, and severe residual disability.[18]

Recent influenza immunization also has been associated with GBS. One study reviewed the cases of GBS during the 1992-1993 and 1993-1994 influenza seasons and found an adjusted relative risk of 1.7 cases per 1 million influenza vaccinations.[19] Therefore, the risk of developing GBS after receiving the influenza vaccine was one to two cases per 1 million persons immunized.

The Vaccine Adverse Event Reporting System, which is administered by the Centers for Disease Control and Prevention and the U.S. Food and Drug Administration to monitor reports of suspected adverse events after immu-

Treatment with plasmapheresis or high-dose intravenous immunoglobulin should be initiated soon after diagnosis.

nization, concluded in its 2003 surveillance summary that the risk of developing vaccine-associated GBS is less than the risk of severe influenza. In addition, despite increasing doses of administered vaccine during the four previous influenza seasons, the number of GBS reports has remained stable.[20]

Treatment

Treatment of GBS has two components: supportive care and specific therapy. Supportive care remains the cornerstone of therapy. If patients advance past the acute phase of illness, most will recover function. However, the neuropathy can advance so rapidly that endotracheal intubation and mechanical ventilation may be necessary within 24 hours of symptom onset.[2]

For this reason, all patients who have GBS should be admitted to a hospital for close observation for respiratory compromise, cranial nerve dysfunction, and autonomic instability. Autonomic nervous system dysfunction may manifest as fluctuations in blood pressure, cardiac dysrhythmias, gastrointestinal pseudo-obstruction, and urinary retention.[1] Prophylaxis for deep venous thrombosis should be provided because patients frequently are immobilized for many weeks.

As respiratory muscles weaken, elective endotracheal intubation should be considered. Progression to respiratory failure can be predicted using measurable respiratory parameters *(Table 4)*.[21] Patients who are unable to demonstrate this minimal lung function require intubation. Frequent reassessment with serial lung function testing for rapid progression is critical. Additional predictors of subsequent mechanical ventilation include the following: (1) time from GBS onset to hospital admission of less than seven days, (2) inability to lift the elbows or head above the bed, (3) inability to stand, (4) ineffective coughing, and (5) increased liver enzyme levels. Predictors of mechanical ventilation in patients who had a previously determined vital capacity included time from GBS onset to admission of less than seven days, an inability to lift the head, and a vital capacity less than 60 percent predicted.[22]

One retrospective study[21] demonstrated a 40 percent decrease from predicted vital capacity, compared with a 60 percent decrease reported in another study.[22] This discrepancy may be related to different study methods and the larger number of patients enrolled in the latter study.

Pain and psychologic stress should be treated. Narcotics should be used with caution because risk of ileus is already increased. Physical therapy, including gentle massage, passive range-of-motion exercises, and frequent position changes may provide pain relief. Carbamazepine[23] (Tegretol) and gabapentin[24] (Neurontin) have been used as adjuncts in pain management in GBS. Patients who were treated with these medications required less narcotic analgesia with fewer narcotic side effects and minimal sedation compared with those who received placebo. Patients are paralyzed by the illness, but mentally alert and fearful. Reassurance and discussion about the phases of illness and recovery can help reduce psychologic stress.

Specific treatment should be initiated soon after diagnosis. High-dose intravenous immunoglobulin (IVIg; 400 mg per kg daily for five days) or plasmapheresis (five exchanges over five to eight days) can be initiated. To determine whether IVIg was as effective as plasma exchange in treating patients with GBS, a large multicenter trial[25] was designed to compare plasma exchange and IVIg and the combination of both treatments for GBS. The study followed 150 patients over four weeks. There were no statistically significant differences in the disability rating between the two treatment groups. IVIg and plasmapheresis were found to be equally effective therapies.[25,26] [References 25 and 26—Evidence level A, randomized controlled trials (RCTs)]

TABLE 4
Indications to Consider Intubation

Forced vital capacity <20 mL per kg
Maximal inspiratory pressure <30 cm H_2O
Maximal expiratory pressure <40 cm H_2O
Progression is noted with reduction of more than 30 percent in vital capacity, maximal inspiratory pressure, or maximal expiratory pressure

Information from Lawn ND, Fletcher DD, Henderson RD, Wolter TD, Wijdicks EF. Anticipating mechanical ventilation in Guillain-Barré syndrome. Arch Neurol 2001;58:893-8.

A Cochrane Database Review combined the largest trials into a meta-analysis and also concluded that IVIg is as effective as plasma exchange in hastening recovery from GBS in patients who required assistance to walk.[27] [Evidence level A, meta-analysis]

In another multicenter RCT of approximately 380 patients,[28] there was no additional benefit from the combination of therapies, and the rate of relapse was not statistically significant.[26,28] [References 26 and 28—Evidence level A, RCTs] Immunotherapy should be initiated early after motor symptoms appear but is unnecessary in mild cases where no motor symptoms are exhibited.

Patients treated early with plasmapheresis required less mechanical ventilation, and hospitalization time was decreased.[29] Plasmapheresis removes or dilutes the circulating immune factors implicated in the pathogenesis of GBS. The French Cooperative Group on Plasma Exchange in GBS concluded that patients with mild symptoms of GBS at admission benefit from two plasma exchanges while patients with more severe symptoms may require two additional plasma exchanges. There is some disagreement regarding the optimal number of plasma exchanges, but all of the studies showing a benefit used four to six exchanges. Plasma exchange is the only treatment that is superior to supportive treatment alone.[30] Furthermore, plasma exchange is most effective for ambulant patients when initiated within two weeks of disease onset but is still beneficial in nonambulant patients up to 30 days after onset.[26,30] [References 26 and 30—Evidence level A, RCTs]

Disadvantages of plasmapheresis include its rare complications, such as sepsis, that are believed to be caused by depletion of immunoglobulins.[31] [Evidence level A, RCT] If fresh frozen plasma is used as replacement fluid, there is a risk of acquiring viral infections such as hepatitis and HIV.

IVIg treatment has advantages over plasmapheresis because it is easier to administer, has significantly fewer complications, and is more comfortable for the patient. IVIg is recommended for patients with GBS who cannot ambulate without assistance within two to four weeks of neuropathic symptom onset.[26,28] Modulation of the immune system is thought to occur through multiple mechanisms involving the constant and variable regions of immunoglobulin class G (IgG), as well as receptors on macrophages and B cells. Pathologic antibodies might be bound by the IgG increasing their clearance. In addition, $CD8^+$ T cell function is enhanced by an unknown mechanism.[32]

Despite its benefits, there are side effects from this IVIg therapy. IVIg expands the plasma volume so it must be administered with caution in patients with congestive heart failure and renal insufficiency. Patients may develop fever, myalgia, headache, nausea, and vomiting, but these "influenza-like" symptoms are self-limiting.[1] Patients also may develop aseptic meningitis, neutropenia, and hypertension. A history of previous anaphylaxis to IVIg is a contraindication to repeat treatment. The risk of serious hepatitis C infection transmission has been reduced because of changes in preparation and purification.[32]

Corticosteroids were once believed to be useful in the treatment of GBS because of its immune-mediated inflammatory mechanism. However, a Cochrane Database Review of randomized trials, which included 195 patients treated with corticosteroids compared with controls, showed no difference in outcome.[33] [Evidence level B, systematic review] Corticosteroids no longer have a role in GBS treatment.[26] [Evidence level A, RCT]

Prognosis and Recovery

Approximately 85 percent of patients with GBS achieve a full and functional recovery within six to 12 months. Recovery is maximal by 18 months past onset. However, some patients have persistent minor weakness, areflexia, and paresthesia. Approximately 7 to 15 percent of patients have permanent neurologic sequelae including bilateral footdrop, intrinsic hand muscle wasting, sensory ataxia, and dysesthesia.[7] The mortality rate is less than 5 percent in tertiary care centers with a team of medical professionals who are familiar with GBS management.[2] Causes of death include adult respiratory distress syndrome, sepsis, pulmonary emboli, and cardiac arrest.[34]

Several factors during the acute phase of illness predict subsequent poor recovery. These factors include age older than 60 years; severe, rapidly progressive disease; and low nerve conduction amplitudes on distal stimulation, which suggests axonal loss.[6] In addition, prolonged mechanical ventilation for more than one month and preexisting pulmonary disease predict a poor outcome. In general, a poor long-term prognosis is directly related to the severity of the acute episode and delay in onset of specific treatment.

Relapse occurs in a small percentage of patients. One multicenter trial[18] of 229 patients showed a relapse rate of 3 to 5 percent. In that study, the relapse rate was not significantly affected by treatment type or any other factor tested.

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