Guillain-Barré syndrome: An update

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1. Introduction

Guillain-Barré syndrome (GBS) is one of the acute flaccid paralysis syndromes in humans. First described in 1916 in two soldiers by French neurologists Georges Guillain, Jean-Alexandre Barré and Andre Strohl, a distinguishing feature from the then most prevalent cause of acute flaccid paralysis, poliomyelitis, was the finding of elevated cerebrospinal fluid (CSF) protein with a normal cell count, the now classic albumino–cytologic dissociation. Since the original description, different subtypes producing the clinical picture of GBS have been described including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), acute sensory neuronopathy, acute pandysautonomia and the Fisher syndrome.

The most frequent subtype of GBS in North America and Europe is AIDP, which accounts for 90% of GBS cases, while in Asia, South and Central America, the axonal form of GBS constitutes 30% to 47% of cases. Only about 5% to 10% of patients in North America and Europe have an axonal subtype. Autonomic involvement is a common feature of GBS, but rarely does GBS manifest with pure autonomic dysfunction. In 1956, the American neurologist Charles Miller Fisher described a syndrome consisting of ataxia, areflexia and ophthalmoplegia, which has proved to be a variant of GBS. In the present review, we discuss the epidemiology, clinical features, diagnosis, pathogenesis and treatment of GBS.

2. Epidemiology

Based on well-controlled population-based studies the incidence of GBS in Europe is 1.2–1.9 cases per 100 000, while worldwide, the incidence is 0.6–4 cases per 100 000. Atypical presentations, such as the Fisher syndrome, are much less frequent, with an incidence of 0.1 per 100 000. Men are 1.5 times more likely to be affected than women, and the incidence increases with age from 1 per 100 000 in those aged below 30 years, to about 4 cases per 100 000 in those older than 75 years. In China, the incidence in adults is 0.66 cases per 100 000.

About two-thirds of GBS cases have an antecedent infection within 6 weeks prior to symptom onset, generally an upper respiratory tract infection or gastroenteritis. Although the pathogenic organism is not often identified, the usual infectious agents associated with subsequent GBS include Epstein-Barr virus, Mycoplasma pneumoniae, Campylobacter jejuni and cytomegalovirus. In China, summer epidemics of the AMAN form of GBS were found to be secondary to infection with Campylobacter jejuni.

In addition to antecedent infections, GBS develops after vaccination. Concerns about vaccine-induced GBS were first raised following the 1976–77 influenza vaccinating season, when a statistically significant increased risk of GBS was reported within 6–8 weeks of receiving the A/New Jersey “swine flu” vaccine. Subsequently, studies that investigated the relationship between GBS
and influenza immunisation reported low relative risks that were not statistically significant. Combined analysis of the 1992–93 and 1993–94 vaccine campaigns in the USA reported a marginally increased risk of GBS (1 extra case of GBS for every 1 million vaccinees) following influenza vaccination during the 6 weeks following immunisation, recently confirmed in a Canadian study. Further, GBS was reported after immunisation with the hepatitis vaccine and the meningococcal conjugate vaccine (MCV4). However, the incidence of GBS after immunization was not different from the background incidence of GBS, thereby precluding any firm conclusions about the significance of these findings. However, because of the close temporal association of GBS with selected vaccines, the risks and benefits of immunisation merit individual review by the clinician and patient.

GBS has also been reported following surgery and head trauma. The mechanisms that link GBS with surgery or trauma remain unclear. However, several postulates have been proposed. Surgery and trauma may alter both cellular and humoral immunities. Specifically, head trauma imparted by injury or surgery may be associated with depressed cell-mediated immunity and production of antitymelyn antibodies. Furthermore, major stress of head trauma or surgery may result in activation of latent processes that would in turn affect the immunological system, as has also been documented following spinal cord injury. Surprising in this regard is that GBS has not been linked to peripheral nerve injury in which one might postulate that exposure of peripheral nerve to the circulation would allow for the creation of autoantibodies against nerve tissue and thus stimulate GBS.

3. Clinical features

The dominant clinical feature of AIDP is generalised muscle weakness with sensory symptoms (excluding pain) being a relatively minor feature. In most patients, the symptoms ascend from the lower to upper limbs; however, in about one-third of cases, all limbs may be involved simultaneously and in about 10% the upper limbs may be affected first. Classically, both proximal and distal muscles are involved in AIDP simultaneously. Weakness of facial muscles is common, occurring in 50% of cases and is frequently bilateral. Respiratory muscle weakness may be severe enough to warrant artificial ventilation in about 25% of patients and portends a poor prognosis. Dysautonomia is well documented in AIDP, occurring in 15% of patients, and includes cardiac arrhythmia, hypertension or hypotension, ileus and urinary retention.

The weakness reaches a nadir at 2 weeks to 4 weeks after symptom onset, with progressive recovery over weeks to months. Although GBS is a monophasic illness, about 7% to 16% of patients suffer recurrent episodes of worsening after an initial improvement.

Although the axonal forms of GBS appear similar to AIDP, there are important clinical differences. Specifically, the axonal forms of GBS exhibit a more rapid and severe course, with frequent respiratory involvement and ventilator dependence, along with cranial nerve involvement and infrequent and mild involvement of the autonomic nervous system. The AMAN form is a pure motor syndrome, with rapid onset of muscle weakness and absent reflexes, while AMSAN is clinically characterised by the presence of both motor and sensory deficits.

The Fisher syndrome is clinically characterised by the presence of ophthalmological abnormalities, ataxia and areflexia. Ophthalmological abnormalities may vary and include acute and chronic ophthalmoplegia. Internuclear ophthalmoplegia, Parinaud’s syndrome, convergence failure or spasm, divergence paralysis, optic neuritis, ptosis or lid retraction, isolated abducens nerve palsies and areflexic mydriasis. In addition, patients with Fisher syndrome may exhibit facial and bulbar nerve palsies. Although Fisher syndrome is self-limiting, there have been patients progressing to respiratory failure requiring mechanical ventilation. Further, other serious complications including coma, autonomic cardiomyopathy and generalised ballism have been described.

4. Diagnosis

4.1. How is GBS diagnosed?

The diagnosis of GBS may be challenging and given an extensive differential diagnosis (Table 1), a thorough medical assessment of patients may be needed to exclude “mimic disorders”. The clinical features of ascending weakness and sensory loss, along with hyporeflexia or areflexia, should raise suspicion of GBS. Nerve conduction studies (NCS) and CSF analysis are important investigations that help confirm the diagnosis of GBS.

4.2. Neurophysiological studies

NCS and electromyography (EMG) are important investigations to establish the diagnosis of GBS, and different neurophysiological diagnostic criteria have been proposed. NCS may support a suspected clinical diagnosis of GBS, identify the GBS subtype and help to exclude mimic disorders.

NCS rely on abnormalities in motor nerves to identify features of demyelination (Table 2), with sensory nerve conduction studies helping to differentiate different forms of axonal GBS, that is AMAN from AMSAN. The diagnostic yield of NCS is increased by studying at least three sensory and four motor nerves, in addition to F-waves and H-reflexes. The classical findings on NCS include the presence of a partial motor conduction block (Fig. 1A), abnormal temporal dispersion of motor responses (Fig. 1A), prolonged distal motor and F-wave latencies, and reductions in maximum motor conduction velocity. Diagnostic criteria, typically used for research purposes, use a combination of these findings (Table 2). Although in over 85% of patients NCS reveal demyelination consistent with the AIDP form of GBS, in up to 13% of cases the initial
NCS is normal but repeat testing in 1 week to 2 weeks might be required to confirm the diagnosis.50–62

NCS may be unrevealing when patients studying early in the disease course, especially within days of symptom onset. At this early stage, the absence or prolongation of tibial nerve H-reflex responses is a frequent neurophysiological finding,63,64 followed by abnormalities of F-wave responses, increased temporal dispersion of distal compound muscle action potential (CMAP) responses, prolonged distal motor and F-wave latencies and reductions of motor conduction velocity in the non-demyelinating range.64 In addition, the presence of A-waves (Fig. 1B) and abnormal blink reflexes (Fig. 1C) may be an early feature of GBS.63–65 Electromyography should be performed to assess for the degree of axonal loss, but this occurs later in the disease process.

4.3. Cerebrospinal fluid examination

In addition to NCS and EMG, CSF analysis may confirm a diagnosis of GBS. A raised CSF protein concentration is present in 80% of patients, with the mononuclear cell count being either normal (albuminocytologic dissociation) or < 50 cells/mm.3–7 The CSF may be normal in the first week of the illness.42 Other investigations that may be helpful in diagnosing GBS are outlined in Table 3.

5. Pathology and pathogenesis

In the AIDP form of GBS, pathological studies reveal patchy multifocal mononuclear cell infiltrates throughout the peripheral nervous system, with the distribution of inflammation often corresponding to the pattern of clinical deficit.56,67 Activated macrophages invade intact myelin sheaths resulting in myelin damage and demyelination.69 The immunological mechanisms underlying the macrophage-mediated invasion of nerves remain elusive, although two potentially complementary mechanisms have been proposed. According to the first hypothesis, activated helper (CD4) T cells react against specific antigens on the surface of Schwann cells or the myelin sheath thereby directing activated macrophages to this region. Inflammatory mediators released by the activated macrophages, such as matrix metalloproteinases and other toxic mediators, underlie Schwann cell injury and subsequent invasion of the peripheral nerve. Recently, dysregulation of T cell function was reported in GBS, further supporting a role of T cells in the pathogenesis of GBS.83

An alternative hypothesis proposes the importance of humoral immunity in AIDP, especially in the early stages of the disease, whereby antibodies bind to epitopes on the outer surface of Schwann cells inducing complement activation and subsequent myelin destruction prior to macrophage invasion.69 In severe cases of AIDP, inflammatory mediators and cells may induce axonal damage, a process referred to as secondary or “bystander” degeneration.7

In AMAN, the pathological features differ from AIDP in that macrophages invade the space between the Schwann cell and axon, leaving the myelin sheath intact.7,70 Given that the rate of recovery in some AMAN patients may be rapid, the pathological process either blocks axonal conduction or results in distal axonal degeneration.71 In very severe cases, axons may be damaged at the ventral nerve roots, resulting in protracted recovery.7

5.1. The role of Na+ channels

Although demyelination and axonal loss may account for chronic symptoms of GBS, the rapid improvement in clinical features with immunomodulatory therapy, often over hours to days, suggests that antibodies directed against specific nerve antigens are important in the pathogenesis of GBS. Of particular interest is the presence of antibodies against gangliosides, a family of acid glycolipids whose lipid portion is located in the cell membrane and the sugar residues are exposed on the extracellular surface. Gangliosides are abundantly expressed in the nervous system and antibodies against specific gangliosides have been identified in different forms of GBS.75,76–78 High titres of antibodies directed against the GM1 ganglioside (anti-GM1 antibodies) have been reported in 10% to 42% of patients with the AMAN form of GBS.75,77

While the role of anti-GM1 and other anti-ganglioside antibodies in the pathogenesis of GBS remains controversial, there is increasing evidence that anti-GM1 antibodies cause reversible dysfunction of voltage-gated Na+ channels at the nodes of Ranvier, thereby resulting in conduction failure.78–81

Voltage-gated Na+ channels are located at the nodes of Ranvier and underlie the generation of action potentials (Fig. 2).82–84 These voltage-gated Na+ channels consist of a transmembrane alpha (α) subunit associated with auxiliary beta (β) subunits.85,86 The α subunits are organised in four homologous domains (I–IV), each of which contains six transmembrane α helices (S1–S6) and an associated pore loop located between the S5 and S6 segments that acts as a selectivity filter. The pore loops line the outer, narrow entry to the pore, whereas the S5 and S6 segments line the inner wider exit from the pore. The S4 segment functions as a voltage sensor. Although the pore-forming α subunit is sufficient for functional expression, the β subunit modifies the kinetics and voltage dependence of channels. The β subunit is involved in channel localisation and interacts with cell adhesion molecules, extracellular matrix and the intracellular cytoskeleton.86

### Table 2

Neurophysiological criteria for Guillaine-Barré syndrome (GBS) according to Van den Bergh and Pieret (2004)57

<table>
<thead>
<tr>
<th>GBS subtype</th>
<th>Distal CMAP amplitude (mV)</th>
<th>Conduction block</th>
<th>Temporal dispersion</th>
<th>Motor conduction velocity (m/s)</th>
<th>Distal motor latency (ms)</th>
<th>F-wave latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDP</td>
<td>Normal or reduced</td>
<td>Proximal: distal ratio of CMAP amplitudes</td>
<td>&gt; 30% increase in proximal negative peak CMAP duration</td>
<td>&lt; 70% Lower limit of normal</td>
<td>&gt; 150% Upper limit of normal</td>
<td>&gt; 120% Upper limit of normal</td>
</tr>
<tr>
<td>AMSAN</td>
<td>Absent or reduced</td>
<td></td>
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<tr>
<td>AMAN</td>
<td>Absent or reduced</td>
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AIDP = acute inflammatory demyelinating polyneuropathy, CMAP = compound muscle action potential, AMSAN = acute sensory and motor axonal neuropathy, AMAN = acute motor axonal neuropathy.

1 Although prolongation of F-wave latency is a diagnostic feature of AIDP, absence of F-wave responses in two or more nerves, when the CMAP amplitude is > 20% of the lower limit of normal, is suggestive of a proximal block.

2 For diagnosis of probable conduction block, a > 30% reduction in the proximal to distal CMAP amplitude ratio is required, while definite conduction block is diagnosed when there is a > 50% reduction in proximal to distal CMAP amplitude. If the CMAP amplitude is markedly reduced (<1 mV), conduction block cannot be diagnosed.

3 For the AMSAN form of GBS, the sensory potentials are either absent or reduced markedly. Further, there should be no features of AIDP, except for the presence of one demyelinating feature in a single nerve, if the distal CMAP amplitude is < 10% of the lower limit of normal.

4 In AMAN the sensory responses are within normal limits. In AIDP the abnormalities must be present either in two or more nerves, or if only one nerve is excitable and the distal CMAP is > 10% of the lower limit of normal, then at least two neurophysiological abnormalities must be present in one nerve.
In myelinated axons, transient voltage-gated Na⁺ channels (Nat) are clustered at high densities (about 1000/m²) at the node of Ranvier. This high density of Na⁺ channels reflects the need of saltatory conduction for a large inward current at the node. When the nodal membrane is depolarised, an inward Na⁺ current is established. The Na⁺ conductance is voltage sensitive and regenerative, increasing with depolarisation. This, in turn, results in greater depolarisation as well as the spread of depolarisation to neighbouring nodes. The explosive process is terminated by inactivation of these voltage-gated Nat channels.

Blocking of voltage-gated Nat channels is thought to underlie the clinical deficits in GBS, in addition to demyelination. Specifically, the rapid improvement in clinical deficits following immunomodulatory treatment, often within hours, cannot be explained by axonal remyelination, but possibly by removal of antibodies or other circulating factors that interfere with Na⁺ channel function. Inactivation of Na⁺ channels results in a conduction block and slowing of conduction velocity. In GBS patients, Na⁺ channel blocking factors have been demonstrated in the CSF. Consistent with this notion of Na⁺ channel blockade are findings that GM1 gangliosides, immunological targets in GBS, are localised to the nodes of Ranvier where Nat channels are clustered.

Animal studies have provided evidence that supports a role for anti-GM1 in the pathogenesis of GBS. Passive transfer of anti-GM1 antibodies into the rat sciatic nerve has resulted in a conduction block, along with deposition of immunoglobulin at the node of Ranvier and axonal demyelination. In addition, sensitisation of Japanese white rabbits with a bovine brain ganglioside mixture containing the GM1 gangliosides induced acute flaccid paralysis and development of anti-GM1 immunoglobulin G antibodies. Further anti-GM1 antibodies cause complement-mediated blockade of voltage-gated Na⁺ channels at the nodes of Ranvier, as well as reversible structural dysfunction of the voltage-gated Na⁺ channels. More recently, axonal excitability studies in patients with GBS using novel threshold tracking techniques have reported an impaired refractory period of transmission distal to the wrist in the AMAN form of GBS. Given that this refractory period of transmission reflects Na⁺ channel function, it was hypothesised that the blockade of Na⁺ channels by anti-ganglioside antibodies (anti-GM1 and anti-GalNac-GD1a, evident in 90% of AMAN...
patients tested) was responsible for the impaired refractory period of transmission and, thereby, conduction failure. The rapid resolution of clinical features, with concurrent improvement of the CMAP amplitude with immunomodulatory treatment in these patients, would further support a role of antibodies against Na\textsubscript{v} channels in AMAN. However, other studies have failed to demonstrate that antibodies to the GM1 ganglioside mediate conduction block or a Na\textsuperscript{+} channel blockade.

Interestingly, axonal excitability studies have been reported to be normal in the AIDP form of GBS. However, none of the AIDP patients exhibited anti-ganglioside antibodies, thereby arguing against anti-ganglioside mediated Na\textsubscript{v} channel blockade in AIDP. Antibodies directed against other nerve targets (possibly other ion channels) might partly underlie the pathophysiology of AIDP. In addition, other circulating factors, such as interleukin-2, which is elevated in GBS and induces Na\textsuperscript{+} channel blockade, may contribute to the clinical deficits.

The pathology of Fisher syndrome remains unclear as only a few autopsy cases have been published. One study reported the presence of segmental demyelination with scanty inflammatory cell infiltration in sensory and motor spinal nerve roots, as well as the 3rd, 7th, 10th and 11th cranial nerves. Although Fisher syndrome is regarded as a variant of GBS, the central nervous system (CNS) involvement remains controversial. Some studies report the presence of inflammatory lesions in the brainstem, whereas others fail to demonstrate any CNS involvement in Fisher syndrome.

As with other forms of GBS, in most cases Fisher syndrome is associated with an antecedent infection, thereby suggesting that molecular mimicry is a major pathogenic mechanism. Antecedent infections, particularly infections with Campylobacter jejuni, are associated with production of IgG antibodies against gangliosides, especially GQ1b. Q1b gangliosides are abundantly expressed in the paranodal myelin sheets of extraocular motor nerves, the neuromuscular junction and dorsal root ganglia. Anti-Q1b IgG antibodies are strongly associated with Fisher syndrome, and correlate with clinical features of ophthalmoplegia and ataxia. The neurological effects of anti-Q1b antibodies are induced by complement-mediated destruction of both perisynaptic Schwann cells and axonal terminals, resulting in neuromuscular junction blockade. Patch clamp techniques have shown that anti-Q1b antibodies inhibit presynaptic Ca\textsuperscript{2+} inflow and interact with proteins of the exocytotic apparatus, thereby interfering with neurotransmitter release, which prevents activation of postsynaptic neurons and ultimately results in muscle weakness. In addition, the binding of anti-ganglioside antibodies to paranodal myelin sheets may disrupt the paranodal axo–glial junction, resulting in loss of ion channel clustering and conduction failure.

6. Prognosis

Although GBS reaches a nadir at 2 weeks to 4 weeks, with most patients recovering from this debilitating illness, 10% to 20% of patients are left with disabling motor deficits and 4% to 15% of patients die by 1 year after onset. Up to one-third of GBS patients need to make substantial changes in their job, hobbies or social activities due to the residual functional deficit. Adverse prognostic factors include older age at disease onset (>50 years); severe disease at nadir as indicated by being either bed bound or requiring artificial ventilation; rapid onset of disease; infection with either Campylobacter jejuni or cytomegalovirus; evidence of axonal loss as demonstrated by neurophysiological testing and possibly elevated CSF neuronfilament levels.

7. General treatment of GBS

Treatment of GBS patients requires a multidisciplinary approach. General supportive treatment includes monitoring and controlling pulse rate and blood pressure because 5% to 61% of GBS patients may suffer wide fluctuations in blood pressures and cardiac arrhythmias due to autonomic involvement. Vital capacity should be monitored because about 25% of GBS patients require artificial ventilation, which should be considered once the vital capacity falls below 15 mL/kg to 20 mL/kg. In non-ambulant patients, deep venous thrombosis prophylaxis with subcutaneous heparin and compression stockings is beneficial. Other complications requiring treatment in the acute setting include urinary retention, bowel dysfunction and pain.

Persistent fatigue is a frequent problem in the chronic setting, perhaps resulting from axonal loss. A multidisciplinary rehabilitation program, including input from a physiotherapist and an occupational therapist, is an important aspect of therapy. Patients may also benefit from joining a patient support organisation such as the GBS/CIDP Foundation International or the Guillain-Barré Syndrome Association of New South Wales, both of which provide information and support to those affected by the GBS.
8. Immunotherapy

8.1. Plasmapheresis and intravenous immunoglobulin (IVIg)

Plasmapheresis has been the accepted as the gold standard of treatment for GBS for almost 20 years. In four trials that included 585 participants, plasmapheresis resulted in significant improvement in clinical features within 4 weeks of randomisation, as indicated by an improvement in disability and increased proportion of patients recovering to full strength within 1 year.133–136 In a further five trials with 623 participants, plasmapheresis reduced the percentage of patients requiring ventilation at 4 weeks from 27% to 14%.7 Plasmapheresis was beneficial within 4 weeks of symptom onset, and the benefit was greatest when the treatment was given early.133,137 The usual regime is to exchange 4 to 6 plasma volumes over 2 weeks,133,134 with four plasmapheresis treatments being more effective than two exchanges.134 Plasmapheresis has proved to be a safe treatment and the costs associated with plasmapheresis are recovered by savings made from reduced intensive care and hospital stay.138,139

In 1992, a Dutch group demonstrated that intravenous immunoglobulin (IVIg) was at least as effective as plasmapheresis in the treatment of GBS.140 Subsequently, four randomised, controlled trials, including a total of 536 subjects, demonstrated equal efficacy of IVIg and plasmapheresis in terms of reducing the duration of mechanical ventilation, improving disability at 4 weeks, reducing residual disability and preventing death.141–144 Although the most frequently used IVIg regime is 0.4 g/kg per day for 5 days, a trial of 50 children found no significant difference in outcome when IVIg was given over 2 days rather than 5 days.145 Trials combining IVIg with either plasmapheresis or immunoadsorption have failed to demonstrate additional benefits when compared to treatment with IVIg alone.142,146

8.1.1. Which GBS patients will benefit from therapy?

Recent American Academy of Neurology practice guidelines recommend treatment with either plasmapheresis or IVIg for GBS patients who were considered immobile.147 The treatment of mildly affected GBS patients, that is those who were ambulant, remains uncertain.148 A French study of mildly affected and ambulant GBS patients reported benefit with plasmapheresis.149 There are no trials assessing the efficacy of either plasmapheresis or IVIg in children younger than 12 years.148

8.1.2. Treatment timing

Trials assessing the efficacy of plasmapheresis have established that plasmapheresis is effective within the first 4 weeks of illness148 whereas trials assessing the efficacy of IVIg have included patients within 2 weeks of symptom onset, so that the efficacy of IVIg beyond this time remains uncertain.7 Patients who have not responded to initial IVIg treatment may benefit from a second course of IVIg.149

8.2. Corticosteroids

Unlike plasmapheresis and IVIg, corticosteroids are largely ineffective in GBS.150 Six trials, studying 587 participants, have failed to demonstrate improvement in disability after 4 weeks of treatment with steroids,151–154 and have reported less improvement in GBS patients treated with oral steroids compared to placebo.151–154 In contrast, GBS patients treated with a combination of intravenous methylprednisone and IVIg tended to improve more rapidly compared to IVIg treatment alone,156 thereby suggesting a possible role of intravenous steroids in GBS. The lack of benefit of oral corticosteroids in GBS is surprising, but may be explained partly by inhibition of macrophages responsible for the clearance of myelin debris.7

9. Conclusions

Although the pathophysiological mechanisms and efficacy of different immunomodulatory treatments have been studied extensively over the last 20 years, about 20% of GBS patients are left with a functional disability and 60% report severe fatigue at 12 months.7,157,158 Further research is required in understanding the underlying pathophysiology of different GBS subtypes that may result in development of novel therapies. For example, if the T cells were of prime importance in AIDP, then agents that block the egress of T cells across the blood–nerve barrier and agents that antagonise T cell cytokine-induced priming of macrophages may prove efficacious. In GBS subtypes with detectable anti-ganglioside antibodies, extracorporeal immunoadsorption, whereby the anti-ganglioside antibodies bind to specific ligands, may prove useful.159 Protection of axons by blocking persistent sodium channels may prevent axonal degeneration, an adverse prognostic feature in GBS.160 More recently, inhibitors of complement activation were shown to exert neuroprotective effects at the presynaptic nerve terminal in mouse models of Fisher syndrome.161,162 Given that complement activation is an important immunopathological factor in GBS,160,163 inhibitors of complement activation may yet prove useful in clinical trials of GBS.

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References


