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Ganglioside Antibodies in Children with Guillain-Barré Syndrome: A Narrative Review

Short title: Ganglioside Antibodies in Pediatric GBS

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Abstract

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy of the peripheral nervous system and one of the leading causes of acute flaccid paralysis in children. Although pediatric GBS often has a more favorable prognosis than adult disease, severe cases may cause respiratory failure, autonomic instability, prolonged disability, and rarely death. Antiganglioside antibodies are increasingly recognized as important contributors to GBS immunopathogenesis, clinical heterogeneity, diagnostic classification, and prognosis. These antibodies are commonly generated after antecedent gastrointestinal or respiratory infections through molecular mimicry and may target gangliosides expressed on peripheral nerves. In children, antiganglioside antibodies have been associated with acute motor axonal neuropathy, Miller Fisher syndrome, Bickerstaff brainstem encephalitis, and the pharyngeal-cervical-brachial variant. This narrative review summarizes the role of antiganglioside antibodies in pediatric GBS, focusing on immunopathogenesis, clinical associations, diagnostic value, therapeutic implications, limitations, and future research priorities. Although antibody testing can support diagnosis and subtype classification, interpretation remains limited by assay variability, incomplete sensitivity, and insufficient pediatric prognostic validation. Further pediatric-focused studies are needed to standardize testing, clarify prognostic significance, and support development of targeted therapies. **[GMJ.2026;15:e4155]**
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Introduction

Guillain-Barré syndrome (GBS) is an acute autoimmune neuropathy of the peripheral nervous system characterized by rapidly progressive weakness, diminished or absent reflexes, and variable sensory, cranial nerve, and autonomic involvement [1]. Since its original description by Guillain, Barré, and Strohl in 1916, GBS has remained a major focus of clinical and immunological research because of its diverse presentations and potential for severe neurological compromise [2]. GBS is best understood as an acute inflammatory polyradiculoneuropathy in which immune-mediated injury affects peripheral nerves, nerve roots, or both [3]. The pathological process may involve demyelination,

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axonal injury, or mixed patterns depending on the subtype and immune mechanism involved [3]. The global incidence of GBS is estimated to range from approximately 0.89 to 1.89 per 100,000 person-years, with variation according to age, geographic region, infection exposure, and case definition [4, 5]. Although GBS can occur at any age, pediatric disease differs from adult disease in clinical course, subtype distribution, recovery pattern, and long-term outcomes [6]. Children often show better functional recovery than adults, but residual symptoms such as fatigue, paresthesia, impaired coordination, concentration difficulty, and mild neurological deficits may persist after the acute illness [6, 8]. Severe pediatric cases may require intensive supportive care, including respiratory monitoring and, in selected cases, plasma exchange or intravenous immunoglobulin (IVIG) [7, 11].

Postinfectious pediatric GBS has been reported after several infectious triggers, including emerging viral infections such as severe acute respiratory syndrome coronavirus 2 [9]. More broadly, antecedent infections are central to the immunopathogenesis of antibody-mediated GBS through molecular mimicry and cross-reactive immune responses [12]. Antiganglioside antibodies have emerged as important immunological markers in GBS [10]. These antibodies recognize gangliosides located on neuronal and glial membranes and may initiate nerve injury through complement activation, disruption of nodal architecture, interference with nerve conduction, and antibody-mediated cellular injury [10]. The relevance of these antibodies is particularly strong in axonal forms of GBS and related variants, including Miller Fisher syndrome (MFS), Bickerstaff brainstem encephalitis (BBE), and pharyngeal-cervical-brachial (PCB) variant [10, 13-17]. Current immunotherapies, including IVIG and plasma exchange, are thought to act partly by neutralizing or removing pathogenic antibodies and modulating complement-mediated injury [7, 11]. Although the pathogenic role of antiganglioside antibodies has been extensively studied in adults, pediatric evidence remains comparatively limited. This narrative review summarizes the role of antiganglioside antibodies in children with GBS, with particular attention to pathogenesis, clinical subtype classification, diagnosis, prognosis, and treatment.

Overview of Guillain-Barré Syndrome in Children

Pediatric GBS usually presents as acute or subacute progressive weakness, often beginning in the lower limbs and ascending proximally [6]. Loss of deep tendon reflexes is a key clinical feature, while sensory complaints, pain, cranial nerve involvement, autonomic symptoms, and respiratory muscle weakness may occur depending on disease severity and subtype [6]. GBS encompasses several electrophysiological and clinical subtypes, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor-sensory axonal neuropathy (AMSAN) [6, 17]. Related immune neuropathy phenotypes include MFS, BBE, and the PCB variant [17]. Approximately two-thirds of patients report an antecedent infection before neurological symptom onset [12]. Common triggers include Campylobacter jejuni, Mycoplasma pneumoniae, cytomegalovirus, Epstein-Barr virus, and other respiratory or gastrointestinal pathogens [12]. These infections may initiate molecular mimicry, in which microbial antigens resemble host peripheral nerve gangliosides [12]. The resulting immune response may cross-react with gangliosides on peripheral nerves, producing antibody-mediated nerve injury [12]. Children often recover more favorably than adults, likely because of differences in immune response, nerve regeneration capacity, and comorbidity burden [6]. Nevertheless, pediatric GBS can still cause major morbidity, particularly when diagnosis or treatment is delayed [6]. Early recognition, respiratory monitoring, electrophysiological assessment, cerebrospinal fluid analysis, and timely immunotherapy remain central to management [6, 7].

Gangliosides and the Immune Response

Gangliosides are sialic acid-containing glycosphingolipids that are highly expressed in

neuronal membranes [13]. They contribute to membrane stability, cell signaling, axonal-glial interactions, ion channel regulation, and synaptic function [13]. Common gangliosides implicated in GBS include GM1, GD1a, GD1b, GT1b, and GQ1b [14]. Their distribution varies across peripheral nerves, nerve roots, neuromuscular junctions, and cranial nerves, which may partly explain the clinical specificity of certain antibody-associated GBS variants [14]. The immune response to gangliosides is usually triggered by antecedent infection [12, 14]. Some microbial surface structures, especially lipooligosaccharides of Campylobacter jejuni, resemble human gangliosides and may induce cross-reactive antibodies through molecular mimicry [12, 14]. Once bound to gangliosides, antibodies may induce nerve injury through complement activation, membrane attack complex formation, disruption of nodes of Ranvier, impaired sodium channel clustering, macrophage recruitment, and axonal degeneration [10, 25]. These mechanisms are especially important in axonal subtypes, where antibodies target gangliosides concentrated at motor nerve terminals and nodal regions [10]. The proposed immunopathogenic sequence linking antecedent infection to clinical GBS phenotype is summarized in Figure-1.

The type of antiganglioside antibody often correlates with clinical phenotype [15]. Anti-GM1 and anti-GD1a antibodies are frequently associated with AMAN, while anti-GQ1b antibodies are strongly linked to MFS and BBE [15, 22]. Although these associations are better established in adults, pediatric studies increasingly suggest similar immunological patterns in children [16, 17].

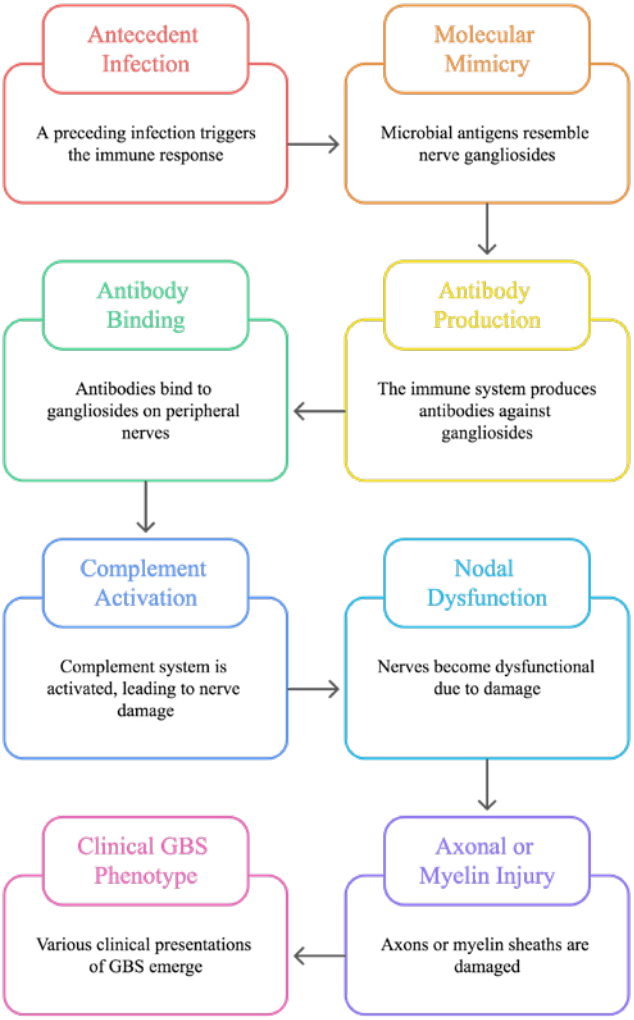


Figure 1. Proposed pathophysiological pathway of antiganglioside antibody-mediated pediatric Guillain-Barré syndrome.

Antiganglioside Antibodies in Pediatric Guillain-Barré Syndrome

The detection of antiganglioside antibodies in children with GBS provides insight into disease immunology and may support subtype classification [16]. Anti-GM1, anti-GD1a, anti-GD1b, and anti-GQ1b antibodies have been reported in pediatric GBS, particularly in axonal and cranial nerve-predominant variants [16, 17]. Anti-GM1 and anti-GD1a antibodies are most often associated with AMAN [16]. This subtype is characterized by motor weakness with electrophysiological evidence of axonal involvement and relative preservation of sensory nerve function [16, 17].

Anti-GQ1b antibodies are closely associated with MFS, which is classically defined by ophthalmoplegia, ataxia, and areflexia [22]. These antibodies may also appear in BBE, a related condition characterized by ophthalmoplegia, ataxia, and impaired consciousness [22-24]. The shared anti-GQ1b antibody profile supports the concept of an anti-GQ1b antibody spectrum that includes MFS, BBE, and overlapping GBS phenotypes [22]. The major antiganglioside antibodies and their commonly reported pediatric clinical associations are summarized in Table-1.

In contrast, antiganglioside antibodies appear less consistently in AIDP [16, 17]. This difference suggests that demyelinating and axonal forms of GBS may involve partially distinct immune pathways [17].

In AIDP, immune responses may target Schwann cell or myelin-associated antigens more prominently than gangliosides, whereas axonal forms show stronger associations with antiganglioside antibodies [17, 25].

Despite their diagnostic value, antiganglioside antibodies have variable sensitivity [16]. A negative antibody test does not exclude GBS, especially early in the disease course, in mild disease, or in demyelinating forms [16]. Therefore, antibody testing should complement, rather than replace, clinical examination, electrophysiological studies, and cerebrospinal fluid analysis [6, 16].

The prognostic value of antiganglioside antibodies in children remains uncertain [6, 16, 17].

Some studies suggest that antibody-positive axonal disease may be associated with more severe weakness and longer recovery, whereas other studies have not confirmed consistent pediatric prognostic associations [6, 17].

Table 1. Common antiganglioside antibodies and their clinical associations in pediatric Guillain-Barré syndrome

Antiganglioside antibody	Commonly associated subtype or variant	Main clinical features	Diagnostic relevance
Anti-GM1	AMAN	Acute motor weakness, reduced or absent reflexes, and relative sensory sparing	Supports axonal motor subtype, especially AMAN
Anti-GD1a	AMAN; PCB variant	Motor weakness with possible bulbar, neck, or upper limb predominance	May indicate axonal motor involvement
Anti-GD1b	PCB variant; sensory ataxic or overlap forms	Ataxia, weakness, sensory symptoms, or bulbar involvement	May support variant or overlap classification
Anti-GQ1b	MFS; BBE	Ophthalmoplegia, ataxia, areflexia, and altered consciousness in BBE	Strong marker of the anti-GQ1b antibody spectrum
Multiple antiganglioside antibodies	Overlap syndromes	Mixed motor, sensory, cranial nerve, autonomic, or brainstem features	May suggest a complex or overlapping phenotype

AMAN: Acute Motor Axonal Neuropathy; **BBE:** Bickerstaff Brainstem Encephalitis; **GBS:** Guillain-Barré Syndrome; **MFS:** Miller Fisher Syndrome; **PCB:** Pharyngeal-Cervical-Brachial.

Pharyngeal-Cervical-Brachial Variant in Children

The PCB variant is a rare form of GBS characterized by acute weakness involving the oropharyngeal, neck, and upper limb muscles [18]. Children may present with dysphagia, nasal speech, difficulty swallowing, neck weakness, and proximal upper limb weakness [19, 20]. Because the lower limbs may be relatively spared early in the disease, this variant can mimic myasthenia gravis, botulism, brainstem stroke, or other neuromuscular disorders [18, 20]. The clinical course is typically acute, with progression over days to one or two weeks [20]. Cerebrospinal fluid may show albuminocytologic dissociation, while nerve conduction studies may support the diagnosis by demonstrating features compatible with GBS [20]. Early recognition is important because bulbar weakness can progress to respiratory compromise [20].

Antiganglioside antibodies may be detected in some cases of PCB variant, including anti-GD1a and anti-GD1b antibodies [19]. The presence of these antibodies supports an immune-mediated mechanism involving regional vulnerability of cranial, cervical, and brachial motor pathways [19, 20].

Treatment generally follows standard GBS management principles, including IVIG or plasma exchange when clinically indicated [6, 7]. Although the prognosis is often favorable with early treatment, some children may experience residual weakness or prolonged recovery [20].

Bickerstaff Brainstem Encephalitis in Children

Bickerstaff brainstem encephalitis is a rare immune-mediated disorder considered part of the broader GBS spectrum [21, 22]. It is characterized by acute ophthalmoplegia, ataxia, and altered consciousness and may also include facial weakness, bulbar dysfunction, pyramidal signs, and limb weakness [22, 23].

Although uncommon in children, pediatric cases have been documented and may present with headache, vomiting, diplopia, gait disturbance, dysarthria, fever, and progressive disturbance of consciousness [24]. The pathophysiology is thought to involve autoimmune inflammation affecting the brainstem and related peripheral structures [22, 23].

Anti-GQ1b antibodies are frequently implicated, linking BBE with MFS and other anti-GQ1b antibody-mediated disorders [22, 23]. This antibody association supports the view that BBE and MFS are related conditions within a continuous immunological spectrum [22].

Diagnosis is primarily clinical but is supported by cerebrospinal fluid analysis, neurophysiological testing, and magnetic resonance imaging [23, 24]. Cerebrospinal fluid may show elevated protein with normal white cell count, although this finding may be absent early in the disease [23].

Treatment usually involves immunotherapy, particularly IVIG or plasma exchange [6, 7]. Early intervention is important to reduce complications and support recovery, and most pediatric patients improve substantially, although residual neurological deficits may persist in some cases [24].

Pathophysiological Implications

The presence of antiganglioside antibodies in pediatric GBS provides strong evidence for antibody-mediated immune injury [10, 14]. After binding to gangliosides on peripheral nerves, these antibodies can activate complement and contribute to membrane attack complex formation on axonal or Schwann cell membranes [10, 25].

This mechanism is especially relevant in AMAN, where nodal and paranodal injury may impair conduction and produce rapid motor weakness [10, 16]. Experimental and translational data support the biological plausibility of antibody-mediated nodal dysfunction, complement activation, and axonal injury in antiganglioside antibody-associated GBS [10, 14, 25].

In children, the pathophysiological impact of antiganglioside antibodies may be modified by developmental differences in immune regulation and peripheral nerve repair [6, 17]. Pediatric nerves may have greater regenerative potential than adult nerves, which may con-

tribute to better long-term recovery in many children [6].

However, severe antibody-mediated axonal injury can still result in prolonged weakness, rehabilitation needs, and persistent disability [6, 8]. These observations highlight the importance of early diagnosis and immunotherapy [6, 11]. If antibody-mediated complement activation and axonal injury are interrupted early, irreversible nerve damage may be reduced [10, 11]. This provides a biological rationale for rapid initiation of IVIG or plasma exchange in clinically significant pediatric GBS [6, 7, 11].

Therapeutic Implications

The identification of antiganglioside antibodies has therapeutic implications in pediatric GBS, although treatment decisions remain primarily clinical [6, 11]. Intravenous immunoglobulin and plasma exchange remain the main disease-modifying treatments [7, 11].

Intravenous immunoglobulin acts by neutralizing pathogenic antibodies, inhibiting complement activation, modulating Fc receptor activity, suppressing inflammatory cytokines, and altering B-cell and T-cell responses [11]. These mechanisms are particularly relevant in antibody-mediated GBS subtypes [10, 11].

Children with antiganglioside antibody-associated GBS may respond favorably to IVIG, although response can vary according to disease severity, timing of treatment, and degree of axonal injury [6, 11]. Plasma exchange removes circulating pathogenic antibodies, immune complexes, and complement components [7]. Plasma exchange may be useful in severe cases, rapidly progressive weakness, or respiratory compromise, but pediatric use may be limited by vascular access challenges, hemodynamic instability, availability, and procedural complications [7]. Real-world treatment patterns also vary according to setting, access, and clinician experience [26].

Emerging treatments targeting complement activation and specific immune pathways may offer future therapeutic benefit in antiganglioside antibody-positive GBS [28, 29]. Complement inhibitors are of particular interest because complement-mediated nodal and axonal injury is central to antibody-associated pathogenesis [10, 29].

However, pediatric data remain limited, and further studies are needed to determine safety, efficacy, timing, and patient selection [6, 30].

Overall, antiganglioside antibody detection has potential clinical value in pediatric GBS, but its role should be interpreted within the broader clinical context [6, 16].

The diagnostic and therapeutic implications of antibody testing are summarized in Table-2.

Table 2. Diagnostic and therapeutic implications of antiganglioside antibody testing in pediatric Guillain-Barré syndrome

Clinical area	Potential role of antibody testing	Main limitation
Diagnosis	Supports the diagnosis of immune-mediated GBS when clinical and electrophysiological findings are compatible	Negative antibody testing does not exclude GBS
Subtyping	Helps distinguish AMAN, MFS, BBE, and PCB variant	Antibody overlap may occur between variants
Prognosis	May suggest axonal involvement, severe weakness, or prolonged recovery in selected cases	Pediatric prognostic evidence remains inconsistent
Treatment planning	Supports the biological rationale for early immunotherapy, including IVIG or plasma exchange	Treatment decisions remain primarily clinical
Research	Helps identify immune pathways for future targeted therapies	Assay standardization and pediatric validation are still needed

AMAN: Acute Motor Axonal Neuropathy; BBE: Bickerstaff Brainstem Encephalitis; GBS: Guillain-Barré Syndrome; IVIG: Intravenous Immunoglobulin; MFS: Miller Fisher Syndrome; PCB: Pharyngeal-Cervical-Brachial.

Challenges and Future Directions

Despite growing evidence supporting the role of antiganglioside antibodies in pediatric GBS, several challenges limit routine clinical application [16, 30]. First, antibody testing methods vary across laboratories, including assay type, antigen preparation, antibody isotype detection, cutoff values, and interpretation criteria [16].

Standardization is essential before antiganglioside antibody panels can be reliably integrated into routine pediatric diagnostic algorithms [16, 30]. Second, the sensitivity of antiganglioside antibodies remains incomplete, and some children with clinically definite GBS test negative, particularly in demyelinating disease or early testing [16].

Therefore, negative serology should not delay treatment when clinical suspicion is high [6, 16]. Third, the prognostic significance of these antibodies in children remains uncertain [6, 17].

Although anti-GM1 and anti-GD1a antibodies may indicate axonal disease and potentially slower recovery, available pediatric studies are not sufficiently consistent to support firm prognostic conclusions [6, 17]. Larger multicenter pediatric studies are needed to determine whether antibody type, titer, persistence, or isotype predicts severity, ventilation risk, recovery time, or long-term disability [6, 27].

Fourth, mechanisms underlying antibody specificity remain incompletely understood. Genetic susceptibility, geographic variation, infection type, microbial strain, host immune response, and developmental immune differences may influence which antibodies develop and which clinical phenotype emerges [12, 30].

Finally, antiganglioside antibodies may represent future therapeutic targets. Current treatments are broadly immunomodulatory rather than antibody-specific [11, 29]. Future strategies could include therapies that block antibody binding, inhibit complement activation, remove pathogenic antibody subsets, or prevent molecular mimicry-related immune activation [29, 30].

Conclusion

Antiganglioside antibodies play an important role in the pathogenesis and clinical expression of GBS in children. Through molecular mimicry after antecedent infection, these antibodies may target gangliosides on peripheral nerves and initiate complement-mediated nerve injury.

Their presence is particularly relevant in axonal GBS variants, MFS, BBE, and the PCB variant. In pediatric practice, antiganglioside antibody testing can support subtype classification and may provide useful diagnostic information when interpreted alongside clinical findings, electrophysiological studies, and cerebrospinal fluid analysis.

However, clinical utility is limited by variable sensitivity, inconsistent testing methods, and incomplete pediatric prognostic data. A negative antibody result does not exclude GBS, and treatment should not be delayed when clinical suspicion is high.

Intravenous immunoglobulin and plasma exchange remain the mainstays of therapy, while emerging complement-targeted and antibody-specific approaches may offer future benefit. Further pediatric-focused research is needed to standardize antibody detection, clarify prognostic significance, and develop personalized therapeutic strategies.

Conflict of Interest

The author declares no conflict of interest related to this manuscript.

AI Disclosure Statement

During the preparation of this manuscript, AI-based language assistance (ChatGPT, OpenAI) was used to support language polishing, grammar correction, manuscript restructuring, and improvement of clarity and flow. The author reviewed, edited, and approved all AI-assisted content and takes full responsibility for the accuracy, integrity, and final version of the manuscript.

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