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Review Article

Guillain Barre Syndrome in Pediatrics

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Abstract

Guillain-Barré syndrome in pediatrics. This paper reviews the current knowledge about Guillain-Barré syndrome (GBS). GBS is defined as an acute, areflexic, flaccid paralysis, which is classified into 4 subgroups: acute inflammatory demyelinating polyneuropathy (AIDP), acute motor-sensory axonal neuropathy (AMSAN), acute motor axonal neuropathy (AMAN) and Miller-Fisher syndrome (MFS). AIDP is associated in 30-50% of cases with cranial nerve involvement, which is not observed in AMAN. MFS is characterized by ataxia, ophthalmoplegia and areflexia, but it may also present cranial nerve dysfunction. Recent data on the pathology and pathophysiology of GBS emphasize the important role of *Campylobacter jejuni* infection in generating anti-ganglioside antibodies (GM1 in AIDP, GQ1b in MFS and GD1a in AMAN), which damage myelin in AIDP and MFS and axons in AMAN. The differential diagnosis must rule out other disorders of the central nervous system (encephalitis, encephalomyelitis, myelitis), myasthenic syndromes, toxic neuropathies induced by heavy meals, drugs, chemical substances or animal toxins, and myopathic conditions, especially acute benign infectious myositis and neuromyopathy of the intensive care unit patient. It is important the treatment with immuneglobulin, at a total dose of 2 grams per kilogram administered over 48 hours. Plasmapheresis can be equally effective. GBS has a good prognosis in children with a total recovery in 85% of cases. Rehabilitation is crucial to attain a more rapid and global improvement.

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- Acute motor axonal neuropathy (AMAN)
- Miller-Fisher syndrome (MFS)
- Campylobacter jejuni; Immunoglobulin

INTRODUCTION

Guillain Barre Syndrome (GBS) is classically defined as an acute acquired sensitive-motor polyrradiculoneuropathy post infectious, immunologically mediated, usually of demyelinating nature [1]. It is the leading cause of acute flaccid paralysis in developed countries, in which polio has been eradicated [2].

The first cases were described in 1859 by Landry, who noted that the disease could produce motor and sensory engagement, which committed the distal portion of extremities and in some cases

Guillain Barre Syndrome progressed in caudocephalic or ascending direction to generalized involvement [3]. In 1916, Guillain and Barre emphasized the importance of albumin-cytological dissociation for clinical diagnosis [4].

For decades, these pictures were explained pathophysiological as inflammatory disorders produced by immunological attack, whose sole target was peripheral myelin antigens. So they called acute inflammatory demyelinating polyneuropathy (AIDP) to the acute form of SGB and chronic demyelinating polyneuropathy to inflammatory sensitive-motor chronic or recurrent motor polyneuropathy (CIDP) [5]. There are other two clinical forms described decades ago as subtypes of GBS: acute motor-sensitive axonal neuropathy (AMSAN) and Miller Fisher syndrome (MFS) Finally was added acute motor axonal neuropathy (AMAN) more recently described [6-8]. AMAN complicates the ancient concept of immune injury in GBS, as in this picture only commit the motor axons and myelin remains intact [9].

In most GBS patients (60-70%) the disease is preceded by a viral or bacterial infection [10]. Paralysis occurs acutely in a period of 1-28 days and, frequently, especially in AIDP a significant autonomic involvement is associated.

In most pediatrics patients evolution is good, although in 10% of cases sequelae is observed. The mortality is low (3-4%). The best outcome of GBS in children has been linked to the most appropriate management of critically ill patients and the use of intravenous immunoglobulin [5-10].

EPIDEMIOLOGY

GBS is a relatively rare disorder, with an incidence ranging between 0.5 to 1.5 cases per 100,000 individuals in the population of 0-17 años [11-14]. Is a well-recognized disease in all world's countries and it is the most frequent cause of acute flaccid paralysis in countries where polio vaccination has allowed polio eradication [15-17].

GBS affects patients of all ages, from the time of infant to old age, but is less common in children. It affects both sexes in a ratio M/F 1.5-1 [18].

The GBS is frequently associated with a history of recent infection. *Campylobacter jejuni* gastroenteritis is the most common pathogen history in the GBS, especially in axonal type [19,20]. On several series represents 23-41% of sporadic cases [21-22].

Viral agents are also frequently associated with GBS: 8-22% cytomegalovirus, Epstein-Barr 10.02% and herpes zoster in 5% of the cases [23,24] .The GBS has also been associated with *Mycoplasma pneumoniae* [25] and *Haemophilus influenza* [26].

PATHOLOGICAL ANATOMY

Pathological anatomy varies according to clinical subtype. In the classic form of GBS (AIDP) commitment of motor and sensory nerve fibers is observed, although dominates the commitment of motor roots as the adyacents plexuses [10]. There is a marked segmental demyelination, with significant mononuclear infiltration, predominantly T-lymphocytes and macrophage at all levels of the peripheral nervous system, including sympathic chains and ganglia and cranial nerves [27].

Secondarily is observed Schwann cell proliferation, expression of reparative mechanism. In the MFS findings are similar to classic demyelinating GBS [28].

In axonal forms of GBS there are no inflammatory changes and the primary lesion is observed at nodes of Ranvier level, which usually produces axonal degeneration [28].

PHYSIOPATHOLOGY

The GBS is actually a group of clinical pictures more than an isolated entity, which is also reflected in the physiopathology. Myelin injury in classic GBS and MFS occurs with participation of humoral and cellular immunity. There is a cross reaction of antibody against GM1 ganglioside in demyelinating GBS and they have shown axonal and similar epitopes to gangliosides in *Campylobacter jejuni*, especially 019 and 041 serotypes, whose polysaccharides resemble gangliosides of peripheral nerve, causing in most cases direct axonal damage, but also demyelination in a significant percentage of cases [29].

MFS pathogenesis is similar, but in this entity responsible ganglioside is GQ1b present in myelin of cranial nerve which is attacked by specific antibodies cross-reaction against *Campylobacter jejuni* [30]. Currently it is considered to ganglioside GQ1b is considered a marker of ophtalmoplejic GBS [31,32], and antibody anti-GT1 a marker of GBS with bulbar cranial nerves comminment [33].

Conversely, the finding of ganglioside antibodies N-acetylgalactosaminyl GD1a (GalNAc-GD 1a) shows a high correlation with clinical forms of GBS without cranial nerves involvement [34-36].

This anti-GD1a antibody is also more specific in the AMAN and is not observed in cases of demyelinating GBS (AIDP) [34-36] (Table 1).

The mechanism of action of viral agents means similar to that described for *Campylobacter jejuni* in the classic GBS (AIDP) [28,35,36].

Table 1: Subtype Guillain-Barre syndrome (GBS) and antibody related antiganglioside GBS Antibody Subtype.

AIDP	**UNKNOWN**
AMSAN	**GM1, GM1b, GD1a**
AMAN	**GM1, GM1b, GD1a, GalNAc-GD1a**
MFS	**GQ1b, GT1a**
Acute sensitive neuronopathy	**GD1b**
Orofacial variant	**GT1a**
Overlap MFS/GBS	**GQ1b, GM1, GM1b, GD1a, Gal,Nac-GD1a**

Modified Lancet 2005; 366: 1653-1666 (ref. 35)

CLINICAL MANIFESTATIONS

In most cases GBS is expressed with homogenic clinical characteristics, but there are more atypical presentations as persistence of deep tendon reflexes or hyperreflexia.

The existence of extensor plantar extensor reflex (Babinski) and papiledema [37] are exceptional and must be ruled out other etiologies [35-37].

In our experience, in isolated cases initially diagnosed as GBS, the further development of extensor plantar reflex allowed suspect CNS involvement and diagnose acute disseminated encephalomyelitis.

The existence of a locked-in syndrome or pseudo brain death with no reactive pupils was another experience lived in our clinical practice, in a child with fast progression and infection of all cranial nerves has also been described in literature [38-40].

Acute inflamatory demyelinating polyradiculoneuropathy (AIDP)

AIDP is the prototype of GBS and represents 85-90% of cases in North America, Europe and most developed countries [28,35]. In children with AIDP the clinical pictures develops 2-4 weeks after a respiratory or gastrointestinal infection. They are frequent paraesthesia of the fingers and toes, followed by distal symmetrical lower limbs weakness, progressing in hours or days to engage the upper extremities and in the severe cases, the respiratory muscles [2,4,28,36]. The cranial nerves are affected in 30-40% of cases at any time of the evolution [2,4,28,36]. Bilateral facial paralysis for VII pair involvement, is the most common cranial neuropathy in GBS [2,4,18, 28,35,36]

Pain is a common symptom in GBS. In a series of 26 children with acute GBS under 6 years hospitalized, 79% accused pain, usually of lower extremities or lumbar location [18,28,36,41]. The neurological examination shows symmetric weakness of lower extremities (and upper if the picture has progressed), with deep tendon reflexes diminished or absent. The sensory involvement is mild and deep sensory impairment predominates. The autonomic symptoms are observed in 50% of the cases: cardiac dysrhythmias, orthostatic hypotension, transient or persistent hypertension, paralytic ileus, bladder dysfunction and impaired sudoration [18,28,36].

More than 90% of patients reach the maximum neurological

commitment at 2-4 weeks of clinical evolution, returning slowly to normal function in weeks or months. The chronic form (CIDP) is generally considered as a different entity to acute GBS [18,28,36]. The clinical course of the disease is shorter in children and recovery is more complete than in adults.

Acute sensorimotor axonal neuropathy (AMSAN)

The AMSAN is a more serious disorder that causes sensory and motor axonal degeneration with absent or minimal demyelination. It was described by Feasby et al. [42], in patients with clinical features of GBS, in inexitable peripheral nerves and absence of demyelination in the anatomopatological study [42,43]. AMSAN shows a slower recovery than the classic GBS, and the sensory and motor sequelae are frecuents [36,42,43].

Acute motor axonal neuropathy (AMAN)

AMAN represents 10-20% of cases of GBS in the Western world and 60-70% of cases of GBS in North China [6-9,36,41]. Several years later was described in South America [44]. It is associated most often associated with *Campylobacter jejuni* infection.

The clinical picture is not necessarily severe and it depends on the extent of axonal injury. Characteristically, in contrast with AIDP, tendon reflex are preserved, and even it may have hiperreflexia [45]. In cases with exclusive distal involvement recovery is quick and complete [18,36,38,43,46].

Miller-Fisher syndrome (MFS)

MFS was described by Fisher in 1956 [47] and constitutes about 3-5% of cases of GBS in occidental countries [22]. It is characterized by the association of ataxia, areflexia and ophthalmoplegia, it presented usually within one week [28]. The first sign is usually diplopia and facial diparesis, seen in 50% of cases. The external ophthalmoplegia usually starts at upper rectus, followed by lateral rectus palsy and ends within inferior rectus. Often Bell phenomenon is observed despite voluntary gaze palsy [48]. MFS differentiation of brainstem Bickerstaff encephalitis is controversial. While some argue that the MFS shows only peripheral nervous system (PNS) involvement [49] others remarks which it is a combination of PNS and CNS impairment [36]. In practice, the peripheral engagement in MFS is clear and is based on the existence of arreflexia and caracteristic neurophysiological changes. The peculiarity of cranial nerve involvement with total external ophtalmoplegia and mild or absent eyelid ptosis, often differentiates MFS of Bickerstaff encephalitis and acute disseminated encephalomyelitis, entities with tipical neuroimaging which shows brainstem involvement and associated with severe ptosis (personal experience). The frequent absence of eyelid ptosis and the Bell's phenomenon with superior voluntary gaze palsy suggest the existence of both peripheral and central involvement (nuclear and supranuclear) in MFS [50].

MFS shows mild limb and ventilatory involvement, without respiratory insufficiency or severe limb paresis. Recovery is complete in a period of months, when the external ophthalmoplegia disappears, especially paralysis of upward gaze, the sign which shows more slow recovery [36,49].

GBS variants

In addition to MFS, now classified as a subgroup and not GBS variant, the most known variant is multiple cranial neuropathy or cranial polyneuropathy. It is characterized by acute palsy of several cranial nerves (except the optic nerve) associated with severe sensitive involvement, [49] usually preceded by CMV infection [18,51]. In 1994, Ropper described almost all variants of GBS known today: a) weakness faringo-cervical-brachial (DFCB), b) Paraparesia, c) eyelid ptosis without ophthalmoparesis, d) and facial diplegia paresthesias and e) Combination of MFS and DFCB [51,52]. Recently, Buompadre et al., reported 11% variants in a casuistry of 179 pediatric patients [53].

In addition to presenting the full range of relevant cases to the variants described by Ropper, they described a case of saltatory type, corresponding to exclusive lower limb involvement associated with cranial nerves impairment without affection of upper extremities, variant not previously reported in the literature.

Cronic inflamatory demyelinating polyneuropathy (CIDP)

It is an entity initially indistinguishable AIDP with AIDP, but differs by the progression of signs and symptoms during a period of more than 28 days, followed by rapid progression or repeated recurrences of the picture. Usually clinical features of patients take more than 2 months to develop the complete picture, which usually has greater asymmetry than AIDP. Occasionally they affect more the upper limbs, but generally predominates involvement of the lower extremities. Dystal sensitive impairment may be significative [54].

The largest difference with GBS is the excellent response of CIDP to treatment with corticosteroids (which is not the case with AIDP), although it should be kept for years to avoid relapses [36,54]. Immunoglobulin is a validated treatment for CIDP and validation of Rituximab is yet in study [55]. Currently, CIDP is considered a different entity from the SGB.

DIAGNOSIS

The diagnostic confirmation is based on the SGB supports in clinical and paraclinical characteristic findings. From the diagnostic criteria established in 1978 by the National Institute of Neurological and Communicative Disorders and Stroke (NINDS) [56], Asbury and Cornblath made an update of these criteria in 1990 [57] (Table 2). Of particular importance is the presence of relatively symmetric progressive weakness associated with hypo or areflexia. However, it is clear that the diagnostic criteria do not cover GBS variants [57].

Albumin-cytological dissociation, an important pillar for diagnostic confirmation of the GBS, it is expressed almost invariably only in the second week in classic disease and in the third week in MFS [58].

The neurophysiological study has higher performance from the second week, mainly to detect H-reflex abolition, which confirms the root involvement and slowing of nervous conduction velocity observed in AIDP and MFS [36,46,58].

However, other important neurophysiological findings, like conduction block and increasing distal latency or abolition of the F wave may appear in the first week of disease progression, thus facilitating earlier diagnosis of GBS [41,58] (Table 3). MRI may demonstrate gadolinium enhancement and enlargement of roots or plexuses which are consistent with ongoing inflammation [59,60].

Differential diagnosis

The differential diagnosis of GBS includes all diseases or disorders that can cause acute flaccid paralysis (Table 4). To help the accurate diagnosis of GBS and other entities presenting as acute flaccid paralysis is essential to make appropriate and necessary studies (Table 5). Brain and spinal MRI can be helpful to differentiate a possible polyneuropathy of myelitis or encephalomyelitis, because sometimes shows similar clinical picture at the beginning period in which neither the study of CSF, or neurophysiological allow to confirm or rule out GBS.

Benign acute infectious childhood myositis is a picture that often is initially interpreted as GBS, in circumstances that only the lower extremities are involved, reflexes are preserved and there are not true pareses but immovility generated by muscle pain. High CPK confirm the diagnosis avoiding more invasive and expensive tests [61].

Initial involvement of oculomotor cranial nerves with eyelid ptosis requires discarding myastenia gravis. If the symptoms occurs in an infant under 6 months old, when GBS is exceptional, botulism should be considered in the differential diagnosis, especially if there are mydriatic hiporreactive pupils [61,62]. Although poliomyelitis has been eradicated in most countries, it should always be considered induced by polio vaccine or other not polio virus, as enterovirus 71 virus and West Nile observed

Table 2: Diagnostic criteria Guillain.
1. Signs required for diagnosis
Progressive motor weakness that involves more than one limb Areflexia or marked hyporeflexia
2. **Signs that strongly support the diagnosis**
Initial absence of fever Progression in days to a few weeks Top recovery 2 to 4 weeks after stop the progression Relatively symmetrical weakness Mild signs and sensory symptoms Cranial nerve involvement Elevated CSF protein after 1st week symptoms Slowing of nerve conduction or extensión the wave F Autonomic dysfunction
Modified of Ann Neurol 1978; 3: 565-6 (ref. 57)

Table 3: Neurophysiological diagnosis of Guillain.
Partial or total motor conduction block
Decreased NCV
Abnormal temporal dispersion
Prolongation of distal latencies
Prolongation of F wave
Abolition of H réflex

Table 4: Differential Diagnosis of Guillain Barre syndrome Barré in relation to the anatomical level of nervous system involvement.
Brain
Meningoencephalitis Brainstem encephalitis (Bickerstaff) Acute disseminated encephalomyelitis(ADEM) Cerebellar post infectious ataxia
Spinal cord
Spinal cord compression Transverse myelitis Anterior spinal artery infarction Acute disseminated encephalomyelitis(ADEM) Optic neuromyelitis (Devic)
Anterior horn motor neuron
Poliomyelitis Enteroviral infection West Nile virus infection
Peripheral nerves
Tick parálisis Paralysis toxin bivalve molluscs (red tide) Paralysis drugs / toxins Diphtheria Porphyria Mitochondrial diseases Polyneuropathy of critically ill patients
Neuromuscular junction
Botulism Myasthenia gravis
Muscle
Acute infectious myositis Autoimmune myositis Metabolic myopathies (glycogenosis, carnitine palmitiltransferase deficiency) Critically ill myopathy and neuropathy Congenital myopathies

Table 5: Diagnostic tests in patients with acute flaccid paralysis (suspected Guillain.
Main
Serum electrolytes Serum creatine phosphokinase(CK) Blood count Chest radiography Electrocardiogram Abdominal ultrasound Cerebrospinal fluid study (cytochemical and viral panel) Electrophysiological of peripheral nerve study MRI brain / spinal cord Stool culture (Campylobacter jejuni) Serology Viral / other (CMV, EB, M. pneumoniae, *B. burgdorferi*) Anti ganglioside antibodies
Others
Heavy metals and toxins detection Drug toxicological panel Urinary porphyrins detection Botulinum toxin (stool, serum)detection Polio virus, enterovirus 71 (depositions)detection Edrophonium or neostigmine test

in recent years [63,64]. One should not forget besides polio in the infant is expressed as symmetrical flaccid paralysis in contrast with typical assymetric involvement in older patients [8,61].

In all serious child sepsis or with multisystem organ failure, which has mechanical ventilation and which have been administered corticosteroids and neuromuscular plate blockers , and presenting flaccid paralysis, critical ill neuromyopathy should be discarded [65]. Finally, keep in mind that neuromuscular inherited diseases, such as congenital myopathy, congenital muscular dystrophy or spinal muscular atrophy not previously diagnosed, may presents as "acute" flaccid paralysis, in conjunction with intercurrent infection.

TREATMENT

SGB treatment comprises mainly measures proper handling, with preservation of function respiratory and cardiovascular (which may be altered by the autonomic commitment GBS), and maintenance an adequate hydration and nutrition. Special importance is the prevention or early control infections, which can aggravate the course of GBS [66] (Table 6).

Specific treatment in children is the use of immunoglobulin at a dose of 0.4 g per kilo for 5 days or dose of 1 gram per kilo for 2 days (currently considered more effective), always completing a total dose of 2 grams per kilo [67-70]. Currently is recommended in the following cases: rapid progression of muscle weakness, respiratory insufficiency or need of mechanical ventilation, bulbar cranial nerve involvement and inability to independent walking [67]. Plasmapheresis has shown equal efficacy to immunoglobulin but as it is a more invasive treatment, it is reserved only for children's cases who shows intolerance or do not respond to immunoglobulin [68-70]. Corticosteroids are not effective in GBS [68-70].

PROGNOSIS

Prognosis of GBS in children is generally good. Over 90% of cases of AIDP and all the MFS cases recover totally [66,70,71]. The severity of the clinical picture is important as a prognostic factor in GBS. 40% of children affected lose march during acute illness and 15% need to be connected to mechanical ventilation [36]. Those with the most severe forms of GBS will delay 6 months or 1 year in achieving complete recovery. 5-10% of pediatric patients present sensory and/or minor motor sequelae, usually in distal lower extremities. The degree of disability is qualified according to the international scale of disability for GBS (Table 7) [72]. Mortality reported in the literature is 1-5% [73] but today seems to have fallen to very low percentage [36]. In cases of AMAN there are a greater percentage of sequels, but it has been shown that in pure axonal involvement usually has a rapid improvement in cases in which there was only conduction block in nodes of Ranvier without axonal degeneration, allowing rapid reversibility of the clinical picture [41-43]. AMSAN cases, exceptional in children, are those with a more ominous prognosis.

Table 6: Treatment of Guillain.

General
Preservation of ventilatory capacity Cardiovascular monitoring. Treatment of autonomic disorders (arrhythmia, arterial hypertension) Prevention and / or early treatment of infections Adequate nutrition and hydration Preventing skin scars
Specific
IVIG, 2 grams per kilo (total dose) in 2 to 5 days. Plasmapheresis (4 treatments) Neurorehabilitation (motor stabilization, orthotics, splints)

Table 7: Classification of disability Guillain.

0. Health
1. Mild signs and symptoms, its do not impair normal activity
2. Walk more than 10 meters without help
3. Walk more than 10 meters with help or support
4. Confined to bed or wheelchair
5. With mechanical ventilation
Modified Rev Neurol 2002; 35: 269-76 (ref.41) and Phys Med Rehabil Clin N Am 2001; 12: 473-90 (ref.72)

REHABILITATION

Rehabilitation should start early to avoid thrombophlebitis (with mobilization and use of elastic bandages) and joint deformities (using orthotics and splints). In addition, active stimulation of musculature is essential to prevent or reduce the degree of muscle atrophy.

Respiratory physiotherapy is of great importance, especially in children who is still not connected to mechanical ventilation, but it must also be made in those that they are. Motor physiotherapy has as its primary objective diminish the severity of muscle atrophy that occurs as a result of prolonged paralysis; usually it must be kept for several months, or even years in severe cases, to fulfill the second and most important goal is to help restore fully motor function. It occurs always in proximo-distal direction in children with GBS [74].

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