LUMEN ET VIRTUS

Follow-up of a patient diagnosed with Guillain-Barré Syndrome with sensory-motor axonal injury (AMSAN)

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ABSTRACT

Guillain-Barre syndrome (GBS) is a rare inflammatory disease of the peripheral nervous system, with a prevalence of 1-4 cases per 100,000 population. It is characterized by acute flaccid paralysis with ascending motor and sensory symptoms, and can be electrophysiologically classified into subtypes such as acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute sensory-motor axonal neuropathy (AMSAN). GBS, usually triggered by viral infections, can lead to serious complications like respiratory failure due to phrenic nerve palsy. The reported case study describes a patient with AMSAN, a severe and rare form of the disease, who presented with progressive weakness and areflexia, with a diagnosis confirmed by electroneuromyography and successful treatment with intravenous immunoglobulin. The prognosis is usually favorable with appropriate treatment, but may include complications and the need for prolonged rehabilitation.

Keywords: Guillain-Barre Syndrome, Acute Sensorimotor Axonal Neuropathy, Polineuropatia.

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INTRODUCTION

Guillain-Barre syndrome (GBS) is an inflammatory disease of the peripheral nervous system (PNS) that affects about 1-2 patients per 100,000 inhabitants, and can reach up to 4 per 100,000 inhabitants, which does not absolve the fact that it is a rare disease. It presents ascending characteristics, with symptoms of motor and sensory dysfunction, and is electrophysiologically classified into subtypes according to the usually symmetrical neuronal lesion1,4.

Presentations of Guillain-Barre Syndrome involve acute flaccid paralysis and may be typical, atypical, or variant variants of the pathology. Such a disease should be considered in cases of involvement of the lower limbs that will present with symmetrical weaknesses, without involvement of the central nervous system (CNS) or other obvious causes, progressing to the arms and cranial muscles, establishing its typical form1. In its atypical form, this involvement is described bilaterally, but simultaneously affecting the extremities of the arms and legs, damaging sensory and motor neurons, and may be asymmetrical. Of the variants of the disease, cranial nerve involvement, pure motor neuron involvement (without sensory signs), weakness without sensory signs, pharyngeal-cervical-brachial weakness, paraplegic variant (affects lower limbs) and Miller-Fisher Syndrome were listed in the literature. Miller-Fisher syndrome, in turn, manifests itself in ataxia, areflexia, and ophthalmoplegia1,2.

According to the electrophysiological study, Guillain-Barre subtypes can be combined into acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN), the first two being the most common2,3.

The pathophysiology of Guillain-Barre syndrome is not very well understood, but it is known that it affects more men than women, in the 20-40 age group, with any age and may undergo immunological activation. That said, it is understood that this syndrome arises from immune-mediated acute polyradiculoneuropathy, developing from a previous viral infection, usually of intestinal and respiratory origin1,2,3. Some scarce literature still suggests the emergence of such a pathology triggered in the postoperative period11. Thus, there is a destruction of peripheral nerve fibers by autoimmune mechanism, leading to symptoms such as numbness, tingling, weakness, complicating loss of motor skills4.

About 20% of patients who develop Guillain-Barre syndrome progress to acute respiratory failure1, this occurs due to paralysis of the phrenic nerve, responsible for the diaphragm muscle. Due to this specific characteristic, patients will hardly present respiratory distress in the evolution of the insufficiency.

Given the infections associated with GBS, prosaically said to be of intestinal origin, it was observed that lipooligosaccharides present in the outer membrane of *Campylobacter jejuni* They are similar to compound glycosides in peripheral nerves, so cross-reacting to fight infection would react

to host nerves3,4. Periods of higher incidence of GBS were observed in 2013 after the outbreak of Chikungunya and Zika virus in Latin America4.

GBS is considered monophasic, i.e., periods of remission and reactivation would be uncommon for this pathology, presenting 1 to 6 weeks after the triggering infection4. The diagnosis of the disease is very well elucidated in the literature, and this clinical diagnosis is associated with nervous symptoms with proteins present in cerebrospinal fluid (CSF) collection[1]. However, little is known about the evolution of the disease, except for the fact that the most common complication previously mentioned in respiratory failure is the most common complication. Therefore, the scientific gap is the objective of this report, which aims to bring to light the characteristic hospital of a patient diagnosed with the most uncommon form of Guillain-Barre syndrome: AMSA.

METHODOLOGY

This is a case report study, whose information was collected through a review of medical records. In parallel, to support the ideas discussed in this article, a literature review was carried out in scientific databases such as PubMed and UptoDate. The production of this scientific article followed the regulations proposed by the National Research Council (CONEP).

CASE REPORT

A 43-year-old male patient reported that he had developed severe low back pain 10 days earlier. She reports that concomitantly with the pain, about 7 days ago, she started to have paresthesia of the lower limbs and tongue. When orthopedists were consulted, the hypothesis of paravertebral muscle weakness was raised, since magnetic resonance imaging of the spine ruled out alterations and the patient was emaciated. Treatment with physiotherapy and vitamin agents was proposed, evolving without improvement, with motor failure, decreased muscle strength and areflexia. When consulting a neurologist, the absence of superficial reflexes was observed on physical examination and deep reflexes on simple tests.

When investigating antecedents, the patient reported flu-like illness for 10 days, bariatric surgery for 20 years, smoker for 25 packs.

Hospitalization was proposed with physical examination at entry showing areflexia, cerebellar gait, absence of sensitivity in the lower and upper limbs, decreased strength grade 4, with worsening on the second day of hospitalization. The collection of cerebrospinal fluid showed significant proteinuria, spirometry without alterations, magnetic resonance imaging of the skull without alterations, electroneuromyography identifying sensory-motor axonal lesion suggestive of DS. Guillain-Barre (AMSA).

The patient was transferred to the intensive care unit, where pulse therapy was performed with immunoglobulin 50 mg/ml, 6 ampoules of 100 ml for 5 days. On the third day, the patient showed improvement in muscle tone, repeated spirometry without alterations, evolving with improved sensitivity on the fourth day, and was discharged on the sixth day of hospitalization.

Title: Electrophysiological study of motor conduction of the patient.

Condução Motora (Direita)

Pto. Estim.	Lat., ms	Ampl., mV	Dur., ms	Dist., mm	Vel., m/s
direita, Abdutor curto do polegar, Mediano, c6-t1					
punho	7,4	2,2	11,0	80	
fossa	13,6	2,7	10,4	270	43,7
direita, Abductor do mínimo, Ulnar, C8 T1					
punho ADM	3,4	4,9	9,21		
ab. do cotovelo ADM	7,9	3,6	9,48	240	53,9
acm. do cotovelo ADM	9,4	3,6	10,6	100	64,1
direita, Extensor curto dos dedos, Fibular, l4 L5 S1					
tornozelo EDB	6,8	3,4	11,0	80	
abx cab fib EDB	13,9	4,0	11,1	360	50,7
acm cab fib EDB	15,5	3,9	11,9	100	65,3
direita, Abdutor do hálux, Tibial, l4 L5 S1					
tornozelo AH	6,6	5,4	11,5	70	
fossa AH	16,1	3,9	12,4	400	41,8

Condução Motora (Esquerda)

Pto. Estim.	Lat., ms	Ampl., mV	Dur., ms	Dist., mm	Tempo, ms	Vel., m/s
esquerda, Abdutor curto do polegar, Mediano, c6-t1						
punho	6,6	3,8	10,8	80		
fossa	12,1	3,5	10,5	270	5,47	49,4
esquerda, Abductor do mínimo, Ulnar, C8 T1						
punho ADM	3,5	7,4	11,0			
ab. do cotovelo ADM	8,0	5,3	12,2	240	4,44	54,1
acm. do cotovelo ADM	9,3	4,7	12,4	100	1,31	76,3
esquerda, Extensor curto dos dedos, Fibular, l4 L5 S1						
tornozelo EDB	7,7	4,5	11,2	80		
abx cab fib EDB	16,0	3,9	12,4	360	8,21	43,8
acm cab fib EDB	17,1	4,0	12,8	100	1,16	86,2
esquerda, Abdutor do hálux, Tibial, l4 L5 S1						
tornozelo AH	7,1	3,5	10,8	70		
fossa AH	15,9	3,3	12,7	400	8,76	45,7

Source: The authors.

Title: Electrophysiological study of sensory conduction of the patient.

Condução sensitiva (Direita)

Ptos. de estim. (captação)	Lat. início, ms	Lat. Pico, ms	Ampl., µV	Dur., ms	Dist., mm	Vel., m/s
direita, n. Mediano (punho)						
palma	2,65	3,2	0,9	8,4	80	30,2
direita, n. Mediano III dedo						
punho			**0**			
direita, n. Ulnar V dedo						
punho	2,12	3,12	3,3	4,0	120	56,6
direita, I dedo. Ramo superficial do n. radial, C5 C6						
punho	1,0	1,4	23,7	1,9	100	100
direita, n. Fibular superficial, L4-S1						
fib sup antidromico	2,72	3,52	18,9	2,3	140	51,5
direita, n.Sural, S1-S2						
sural	2,72	3,64	14,2	2,1	140	51,5

Condução sensitiva (Esquerda)

Ptos. de estim. (captação)	Lat. início, ms	Lat. Pico, ms	Ampl., µV	Dur., ms	Dist., mm	Vel., m/s
esquerda, n. Mediano (punho)						
palma	1,46	2,08	8,3	1,7	80	54,8
esquerda, n. Mediano III dedo						
punho	2,6	3,92	3,4	4,5	140	53,8
esquerda, n. Ulnar V dedo						
punho	2,2	3,0	8,0	2,7	120	54,5
esquerda, I dedo. Ramo superficial do n. radial, C5 C6						
punho	1,44	1,92	17,5	2,2	100	69,4
esquerda, n. Fibular superficial, L4-S1						
fib sup antidromico	2,44	3,2	11,4	2,0	140	57,4
esquerda, n.Sural, S1-S2						
sural	2,8	3,6	10,6	4,2	140	50,0

Source: the authors.

Title: electromyogram.

Captação	Ativ Ins	Fib	OAP	Fasc	D.Miot	DCR	Polifasia	Baixa amp	Alta amp	Dur	Recrut	Interf
direita, Deltóide, Axilar, C5 C6	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Bíceps braquial, Músculo-cutâneo, C5 C6	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Tríceps, Radial, c6 C7 C8 T1	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Flexor radial do carpo, Mediano, C6 C7 c8	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, I Interósseo, Ulnar, C8 T1	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Extensor do index, Radial, c6 C7 C8	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Paravertebral Cervical	N	0	0	0	0	0	N	0	0	N	N	N
direita, Glúteo médio, Glúteo superior, L4-S1	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Vasto lateral, Femoral, L2-L4	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Tibial anterior, Fibular, L4 L5 s1	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Gastrocnêmio, Tibial, S1-S2	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Extensor longo do hálux, Fibular, l4 L5 S1	N	0	0	0	0	0	++	0	++	A	D	Rar
direita, Flexor longo dos dedos, tibial, L5 S1	N	0	0	0	0	0	++	0	++	A	D	Rar

Captação	Ativ Ins	Fib	OAP	Fasc	D.Miot	DCR	Polifasia	Baixa amp	Alta amp	Dur	Recrut	Interf
esquerda, Deltóide, Axilar, C5 C6	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Bíceps braquial, Músculo-cutâneo, C5 C6	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Tríceps, Radial, c6 C7 C8 T1	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Flexor radial do carpo, Mediano, C6 C7 c8	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, I Interósseo, Ulnar, C8 T1	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Extensor do index, Radial, c6 C7 C8	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Paravertebral Cervical	N	0	0	0	0	0	N	0	0	N	N	N
esquerda, Glúteo médio, Glúteo superior, L4-S1	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Vasto lateral, Femoral, L2-L4	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Tibial anterior, Fibular, L4 L5 s1	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Gastrocnêmio, Tibial, S1-S2	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Extensor longo do hálux, Fibular, l4 L5 S1	N	0	0	0	0	0	++	0	++	A	D	Rar
esquerda, Flexor longo dos dedos, tibial, L5 S1	N	0	0	0	0	0	++	0	++	A	D	Rar

LEGENDA: Ativ.Ins: atividade de inserção, Fib.: fibrilações, OAP.: ondas positivas, Fasc.: fasciculações, DCR.: descargas complexas repetitivas; Amp.: amplitude, Dur.: duração, Interf.:padrão interferencial, Rar.: padrão rarefeito, Cheio.:padrão interferencial cheio, rap: rápido, n: normal, a: aumentado, d: diminuído, 0: ausente, + a +++ presente com intensidade crescente.

Source: the authors.

Title: electrophysiological study of conduction waves.

Onda-F Direita

direita, Abductor do mínimo, Ulnar, C8 T1

Min	Max	Média	Difer.
30,4	34,6	32,9	4,25

Bloqueios F, %	Persist. F, %
0	100

direita, Abdutor do hálux, Tibial, l4 L5 S1

Min	Max	Média	Difer.
61,7	63,6	62,3	1,89

Bloqueios F, %	Persist. F, %
0	100

Onda-F Esquerda

esquerda, Abductor do mínimo, Ulnar, C8 T1

Min	Max	Média	Difer.
33,7	37,6	35,1	3,99

Bloqueios F, %	Persist. F, %
0	100

esquerda, Abdutor do hálux, Tibial, l4 L5 S1

Min	Max	Média	Difer.
56,5	59,3	57,4	2,8

Bloqueios F, %	Persist. F, %
0	100

Source: the authors.

DISCUSSION

Acute sensorimotor axonal neuropathy (AMSAN) is a subtype of Guillain-Barre disease, a variant of pure motor axonal involvement, which is the most severe form of the disease. This has in common with its counterparts the involvement of gangliosides present in the axons of peripheral nerves, whose dysfunction originates from the infiltration of macrophages, inflammation and proliferation of Schwann cells. Electrophysiological studies allow the classification of the pathology according to its subtypes1,2,3,5.

The literature studied agrees that the incidence of Guillain-Barre cases annually varies between 2 per 100,000 inhabitants/year1,2,4,5,7,9, reaching 4-6 people per 100,000 inhabitants/year6,10,11, but few describe the involvement of the AMSAN type. One study, however, classified sensory-motor impairment in 3% of its group2. It is understood, however, that the most common age group of involvement persists between 20 and 40 years, agreeing with this case, whose age error range exceeded only 3 years, differing from the study of Wachira VK et al., (2023)[10], which reports a higher incidence in men over 50 years of age.

GBS is a pathology that was initially described by Gillan, Barre and Strohl, being the first to define progressive areflexia, often symmetrical associated with loss of strength. In addition, the protein count in the cerebrospinal fluid was observed[1,5]. In 2011, the Brighton criteria were described, widely used in everyday life, where the absence of alternative causes of weakness, reduced tendon reflexes of weak muscles, monophasic course, bilateral flaccid weakness, CSF cell count < 50 cells/microL, elevated CSF protein, and clinical consistency with a subtype, grading the degree of certainty from 1 to 4, determine the Guillain-Barre diagnosis3. The clinical findings of this patient are consistent with the syndromic diagnosis of the pathology, whose certainty score was established at grade 4. To understand the subtype of the disease, however, an electrophysiological study was necessary, which showed motor and sensory conduction failure, classifying it as AMSAN.

As classified by the Brighton criteria3, the absence of lesions of the Central Nervous System is essential for the suspicion of an autoimmune disease, since the deficit can be focal as described in GBS variants that cite cranial muscle weakness1. Cranial computed tomography, discarding CNS alterations, was decisive in the diagnosis of the patient in this case, since he presented an important risk factor for the occurrence of strokes, for example, such as smoking. In addition to this fact, the ascending and peripheral clinic was the most striking characteristic for the disease.

The Acute inflammatory demyelinating polyneuropathy (AIDP) is the most common subtype of GBS and more common in Western countries. Antigenic epitopes are presented to the T cells of the immune system, causing cross-reaction, releasing cytokines and free radicals that destroy the myelin sheath of neurons6. It can be reasoned that for this reason, it is associated with the outbreak of Zika virus and Chikungunya in Latin America. The AMSAN type varies from the AMAN type,

which in turn is closely related to gastrointestinal tract infections and lipooligosaccharides from the bacterium, with the *Campylobacter jejuni* acting on the axon, but it is possible to cite respiratory tract infections by *Haemophilus influenzae, Sars-Cov-2* and *Mycoplasma pneumoniae*. Having flu-like symptoms as a previous history, it is possible to hypothesize that this episode was responsible for triggering the autoimmune reaction in this reported patient. This flu-like episode occurred 10 days after the pathology, which is in agreement with the literature that describes the onset of symptoms 1-6 weeks after the triggering infection3,4,10.

Of the most well-known complications, 20% of patients present respiratory failure in the course of the pathology. Therefore, a score is presented that estimates the risk of this complication based on the need for mechanical ventilation in hospital, called EGRIS – *Erasmus Guillain-Barre Syndrome respiratory insufficiency Score*, being moderately reliable, according to studies, determining a score of 0-7, taking into account factors such as time of symptom onset, facial/bulbar weakness and the sum of the Medical Research Council (MRC) score. The MRC, in turn, scores between muscle weakness of bilateral shoulder abduction; elbow flexion; wrist extension; hip flexion; knee extension; and ankle dorsiflexion. An EGRIS score of 0-2 indicates a low probability of the need for mechanical intervention, 3-4 intermediate risk, 5 or higher risk1,7,9. In this case, the patient's severity classified him as high risk, but he was responsive to immunoglobulin pulse therapy, evolving without signs of respiratory failure. It is noteworthy that nocturnal mechanical ventilation relieves chronic hypoventilation in the course of the disease and prolongs the patient's survival.

Although there are other prognostic scores for Guillain-Barre syndrome, EGRIS remains the most reliable, according to a study by Busl KM et al. (2023)[7]. Other complications include autonomic involvement such as cardiac arrhythmias, postural hypotension, and bladder disorders5.

For the treatment of GBS, immunomodulatory measures are indisputable. Treatment should be initiated when patients are unable to travel 10 meters without assistance, indicating severe motor impairment, even if the episode is self-limited, due to the risks of unfavorable evolution. Intravenous immunoglobulin 0.4 g/kg in five sessions if symptoms started within two weeks, and plasma exchange 200-250 ml/kg also in 5 sessions when started at 4 weeks is the classic therapy proposed. Oral or intravenous corticosteroids do not present benefits to combat the course of the disease1,3,4,9, but the high cost of immunoglobulin is a solid obstacle to such treatment. The therapy used for this patient is in accordance with that cited in the literature, which emerged in 1992 after a randomized study that helped to elucidate its benefit6. For such therapy, admission to the intensive care unit (ICU) was necessary and complied with scientific criteria for ICU admission, which include: rapid disease progression; development of respiratory symptoms; dysautonomia or severe dysphagia; EGRIS score > 49.

Among the monetary disadvantage cited for the use of immunoglobulin, the adverse effects are also notable. In addition to anaphylaxis, rapid immunoglobulin infusion may trigger imbalance in colloidal osmotic pressure and culminate in cardiac arrest or renal failure. Such adverse effects were not observed in this case in question. Other reactions include stroke, hemolytic anemia, transfusion-related acute lung injury, aseptic meningitis, and venous embolism9. The evolutionary course of the patient submitted to this therapy is notorious, he presented almost complete recovery in 6 days, being discharged from the hospital while walking, contrary to the literature that cites a delay of months for complete recovery8.

It is not clear which etiological agent was responsible for triggering the autoimmune reaction in this patient, but in view of the previous respiratory infection, antiviral or antimicrobial therapy could be considered9,10. The history of bariatric surgery occurred a long time ago to even be considered as a cause, according to studies11. There are no studies that target the line of treatment specifically for AMSAN. Another alternative is long-term motor rehabilitation which, according to Shah N et al., (2022)[8], is effective in reestablishing functional independence after 6 months, reducing fatigue and improving muscle tone.

CONFLICTS OF INTEREST

The authors state that there is no potential conflict of interest that could compromise the impartiality of the information presented in this scientific article.

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