Case Report

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Guillain-Barré Syndrome With Nine Positive Anti-Glycolipid Antibodies A Case Report

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1.Abstract

1.1. Background: Objective: to investigate the clinical characteristics of nine glycolipid antibody-positive patients. Method: the clinical data of a patient with Guillain-Barré syndrome were retrospectively analyzed. Result: A 69-year-old woman presented with dysphasia, cough when drinking water, and weakness of the extremities after diarrhea. Physical Examination: Dysarthria, shallowness of the right nasolabial groove, weakness of uplift over the soft palate on the right side, and weakness of the right pharyngeal reflex. Muscle strength was grade 2 proximal, grade 3 distal, and grade 4 inferior.

The muscle tone of the extremities was reduced, and bilateral biceps, biceps, radial, knee tendon, and Achilles reflexes were not elicited. Electromyogram: multiple peripheral nerve lesions: motor nerve involvement; axonal, demyelinating lesions coexist. Serum autoimmune peripheral nerve antibody profiles suggested anti-Sulfatide IgG(+), anti-GD1a IgG(+), IgM(+), anti-GT1a IgG(+), IgM(+), anti-GT1b IgG(+), IgM(+), anti-GQ1b IgG(+), IgM(+).Weakness of the extremities recovered after gamma globulin treatment. Result: The purpose of this study was to gain a better understanding of the clinical features of Guillain-Barré syndrome in a small number of patients who were positive for nine anti-glycemic antibodies and did not exhibit typical symptoms corresponding to a single glycemic antibody.

2. Keywords:
Guillain-Barré syndrome. Gangliosides. Sulfates. glycolipid antibodies

3. Introduction

Guillain-Barré syndrome (GBS) is an autoimmune-mediated peripheral nerve disorder that affects the cranial nerve and also the spinal root and peripheral nerves [1]. Since 1988, when Ilyas et al [2]. first reported the detection of sarcosine antibodies in GBS patients, subsequent studies have shown that these antibodies are associated with the pathogenesis of GBS [3]. The gangliosides are distributed mainly in the cell membrane, especially in the nervous system. Anti-ganglionic antibodies are produced mainly because the lipopolysaccharide structure resembles that of human peripheral ganglion lipids, leading to the production of anti-ganglionic antibodies [4]. Differences in sarcosine content may influence the clinical presentation of anti-sarcosine antibody-mediated disease [5]. Foreign studies by Kaida et al. suggest the presence of NAD-C in the serum of patients with GBS, suggesting that NAD-C may play a role in the clinical manifestations [6] of GBS and serve as a useful marker for various clinical features and histologic subtypes, but not determined [7]. More recently, antibodies to the GQ1b/GT1a complex have been reported concomitantly, with no significant antibody response to single nucleosides (including GQ1b and GT1a) [6].

4. Clinical date

A 69-year-old woman was admitted on 2020-9-22 for “three days of dysphasia with progressive weakness of the extremities.”He developed headaches and diarrhea after cooling in 2020-9-12. He was speechless and vague, but able to hear. He coughed when drinking water. He had a poor effect after taking cold medicine. He felt weakness in his upper limb when combing the head in the morning of 2 days ago. The main manifestations are the inability of the upper limb to lift above the head and the inability of the hands to grasp and lift objects, followed by the weakness of the lower limb, and the ability to stand and walk, requiring assistance. the symptoms continued to worsen the day before admission. He has a previous history of hypertension for 8 years. Positive neurological signs: clear mind, dysarthria. The right nasolabial sulcus becomes shallow, the soft palate on the right is weakened, and the right pharyngeal reflex is weakened. Muscle strength was grade 2 proximal, grade 3 distal, and grade 4 inferior. The muscle tone of the extremities is reduced and there is no atrophy of the extremities. Bilateral biceps reflex, triceps reflex, radial periosteal reflex, knee tendon reflex, and Achilles tendon reflex were not elicited. The bilateral nasopharyngeal test and the tibiofemoral test did not cooperate. Adjunctive tests: blood, urine, stool, biochemistry, lipids, preoperative eight items, rheumatic immunity, tumor markers, cerebrospinal fluid, biochemistry, and cytology were no abnormalities. CSF immunoglobulin (IgG):50.80mg/L(reference range 0-34mg/L). Serum autoimmune peripheral nerve antibody profiling revealed anti-Sulfatide IgG(+), anti-GD1a IgG(+), IgM(+), anti-GT1a IgG(+), IgM(+),

anti-GT1b IgG(+), IgM(+), anti-GQ1b IgG(+), IgM(+), and negative serum and cerebrospinal fluid antibodies. electrocardiogram, abdominal ultrasound, echocardiogram, and cranial MRI+ diffusion did not show abnormalities. EMG: Motor nerves: Left common peroneal motor conduction velocity decreased and amplitude decreased markedly; Right common peroneal motor conduction velocity decreased and amplitude decreased markedly with CB and fibula head stimulation; Both posterior tibial motor conduction velocities decreased and amplitude decreased with popliteal stimulation; Right median motor conduction velocity decreased and amplitude decreased markedly, and distal latency period increased markedly; Right ulnar motor conduction velocity decreased and amplitude decreased. Sensory nerves were normal. Left median nerve F wave with decreased amplitude; Bilateral posterior tibial nerve F wave with prolonged latency and decreased amplitude.Posterior left tibial nerve H reflex, not elicited.

Diagnostic advice suggests multiple peripheral nerve lesions: motor nerve involvement; and axonal, demyelinating lesions coexist. Taken together, consideration of GBS is highly likely, and the specific type is unknown. Gamma globulin (0.4 g·kg·d-1) and vitamin B1 and B12 neurotrophins were administered for 5 days. Speech improved after treatment, as did limb weakness, and neurologists at discharge: right nasolabial groove was superficial, and bilateral pharyngeal reflexes were diminished. Muscle strength was grade 3 in the proximal upper limb, grade 4+ in the distal limb, and grade 5 in the lower limb. After half a month, the patient's serum autoimmune peripheral nerve antibody profile showed anti-GT1a IgG(+), IgM(+), anti-GT1b IgM(+), and anti-GQ1b IgM(+), all of which were negative. The patient's symptoms and muscle strength improved. After 1 month, muscle strength of the extremities was of grade 5, and the patient had no recurrence to date.

5. Discussion

Gangliosides and sulpholipids, the glycolipid antibodies, are important components of peripheral nerves. Ganglioside is a salicylated glycocomplex widely present in the central nervous system and is associated with Guillain-Barré syndrome. The most commonly encountered gangliosides are antibodies to GM1, GM2, GM3, GD1a, GD1b, GD3, GT1a, and GQ1b [8]. Sulfatide, an acidic glycolipid with sulfuric acid residues on the myelin sheath, is involved in the formation of the neurosphingotic membrane and is associated with its physiological function, mainly in peripheral neuropathies [9]. Anti-sulfatide antibodies predominantly affect sensory nerve axons [10], are highly positive in autoimmune-mediated chronic multiple peripheral neuropathy[11], and may be detected even in healthy individuals. The presence of anti-sulfatide antibodies in tissues may be associated with a widespread distribution of anti-sulfatide antibodies [12]. However, no specific clinical effects have been reported [13], and there is considerable controversy regarding the type of clinical effects and their specificity [14-15]. Anti-GD1a antibodies primarily affect locomotion [16-17] and are associated with lung cancers caused by acute motor axonal neuropathy, acute motor sensory axonal neuropathy, and paraneoplastic peripheral neuropathy. The anti-GD1a antibodies are specific for acute motor axonal neuropathy (pure motor) populations. The anti-GD1a antibodies are associated with disease severity and the need for adjuvant ventilation after facial palsy. and are associated with axonal mutations [18]. Anti-GT1a antibody is associated with diarrhea and medulla oblongata paralysis [19-21]. Antibodies are generally persistently positive after treatment for pharyngeal-brachial variant GBS and fulminant GBS [22]. The presence of anti-GT1b antibody may be a useful predictor of mechanical ventilation or severe GBS. Primary involvement of ophthalmoplegia in anti-GQ1b-positive patients [23,24]. It is associated with melanoma due to Miller-Fischer syndrome, Bickerstaff encephalitis, and paraneoplastic peripheral neuropathy and is specific to the population with Miller-Fischer syndrome. It generally has a high titer of antibody in the acute phase, a low titer in the remission phase, and a negative titer 3–4 weeks later [25], with cross-reactivity with anti-GT1a antibodies present at the same time. Cross-reactions occur when anti-GT1a antibody is present simultaneously. Multiple antiganglioliplid and antiglycolipid antibodies are present in patients with Guillain-Barré syndrome, and antiganglipid antibodies are composed of two distinct ganglionolipid complexes, suggesting that the ganglionolipid complexes can form new antibody binding sites [26].

In this case, the patient had a history of prodromal dysarthria, clinical manifestations of dysphasia with weakness of the extremities, and physical findings of dysarthria. The right nasolabial groove becomes shallow, the soft palate on the right is weakened, and the right pharyngeal reflex is weakened. Muscle strength was grade 2 proximal, grade 3 distal, and grade 4 inferior. The muscle tone was decreased in the extremities and the bilateral biceps, biceps, radial, knee tendon, and Achilles reflexes disappeared. Glucolipid antibody 9 positive, serum: Anti-sulfatide antibody IgG (+), anti-GD1A antibody IgG (+), IgM (+), anti-GT1A Antibody IgG (+), IgM (+), anti-GT1B antibody IgG (+), IgM (+), anti-GQ1b antibody IgG (+), IgM (+), and multiple positive glycolipid antibodies were found at the same time. Considering multiple groups of cranial nerve damage and movement related, the patient did not show typical clinical manifestations of GD1a, GT1a, GT1b, GQ1b antibody positive, which was consistent with relevant reports[25]. It is an atypical clinical manifestation of the combination of multiple antibodies, and the specific cause has not been reported. After treatment, the patient's anti-GD1A antibodies IgG (+) and IgM (+) turned negative, indicating the recovery of motor function. The GD1a positive antibody of the patient turned negative after treatment, and a longitudinal study showed that the titer of the anti-ganglioside antibody of the patient decreased with the clinical course of the disease [27,28]. Looking at the literature, serum was detected with high titer IgG anti-Galac-GD1A antibody, which decreased after treatment. It is believed that this antibody may be related to axonal neuropathy [29,30] Both anti-GD1A antibody and anti-GalNAc-GD1a antibody are related to acute motor axonal neuropathy. Therefore, whether GD1a turns negative, like anti-GalNAc-GD1a antibody, is related to neuropathy needs further study.

In conclusion, nine glycolipid antibodies were positive and none exhibited the typical features of a single antibody. The prognosis was excellent, and

the patient's symptoms, signs, adjuvant examination, and the Guidance on the diagnosis and treatment of Guillain-Barré syndrome in China (Guidelines on the diagnosis and treatment of Guillain-Barré syndrome in China, 2019) [1] provided experience in the diagnosis and treatment of GBS with clinically rare polyglycolipid antibodies.

Reference

1. 中华医学会神经病学分会，中华医学会神经病学分会周围　神经病协作组，中华医学会神经病学分会肌电图与临床神经电生理学组,等. 中国吉兰-巴雷综合征诊治指南2019 - 中华神经科杂志(2019)[2023-08-05].
2. Ilyas A A, Willison H J, Quarles R H, et al. Serum antibodies to gangliosides in guillain-barré syndrome: gbs and ganglioside antigens. Annals of Neurology, 1988, 23(5): 440-447.
3. Kaida K, Morita D, Kanzaki M, et al. Anti-ganglioside complex antibodies associated with severe disability in gbs. Journal of Neuroimmunology, 2007, 182(1-2): 212-218.
4. Shahrizaila N, Lehmann H C, Kuwabara S. Guillain-barré syndrome. Lancet (London, England), 2021, 397(10280): 1214-1228.
5. Ogawa-Goto K, Abe T. Gangliosides and glycosphingolipids of peripheral nervous system myelins—a minireview. Neurochemical Research, 1998, 23(3): 305-310.
6. Kaida K, Morita D, Kanzaki M, et al. Ganglioside complexes as new target antigens in guillain-barré syndrome: antibody to ganglioside complex. Annals of Neurology, 2004, 56(4): 567-571.
7. Goodfellow J A, Willison H J. Antiganglioside, antiganglioside-complex, and antiglycolipid-complex antibodies in immune-mediated neuropathies. Current Opinion in Neurology, 2016, 29(5): 572.
8. 佚名. Antiganglioside complex antibody source nihon rinsho 2013 may 71 5 839 44.pdf.
9. 佚名. The enigmatic role of sulfatides: new insights into cellular functions and mechanisms of protein recognition | springerlink.
10. Pestronk A, Li F, Griffin J,et al. Polyneuropathy syndromes associated with serum antibodies to sulfatide and myelin-associated glycoprotein. Neurology, 1991, 41(3): 357–362.
11. Kaida K. Guillain–barré syndrome[M/OL]. Sango K, Yamauchi J, Ogata T, et al., eds.//Myelin. Singapore: Springer Singapore, 2019: 323-331[2023-08-05].
12. 李晓昶, 杨改清, 胥丽霞, 等. 硫脂抗体阳性与阴性患者的临床特征对比. 医药论坛杂志, 2021, 42(17): 21–24.
13. Esposito S, Longo M R. Guillain–barré syndrome. Autoimmunity Reviews, 2017, 16(1): 96-101.
14. Carpo M, Pedotti R, Allaria S, et al. Clinical presentation and outcome of guillain–barré and related syndromes in relation to anti-ganglioside antibodies. Journal of the Neurological Sciences, 1999, 168(2): 78-84.
15. Alaedini A, Sander H W, Hays A P, et al. Antiganglioside antibodies in multifocal acquired sensory and motor neuropathy. Archives of Neurology, 2003, 60(1): 42.
16. Kusunoki S, Chiba A, Kon K, et al. N-acetylgalactosaminyl gd1a is a target molecule for serum antibody in guillain-barré syndrome. Annals of Neurology, 1994, 35(5): 570-576.
17. Kaida K, Kusunoki S, Kamakura K, et al. GalNAc-gd1a in human peripheral nerve: target sites of anti-ganglioside antibody. Neurology, 2003, 61(4): 465-470.
18. Kim S Y, Kim J K, Suh C K. Polycranial neuropathy and sensory ataxia with igg anti-gd1a antibody as a variant of guillain–barré syndrome. Journal of Clinical Neuroscience, 2013, 20(3): 473-475.
19. Kim J K, Kim B-J, Shin H Y, et al. Acute bulbar palsy as a variant of guillain-barré syndrome. Neurology, 2016, 86(8): 742-747.
20. Saito T. Fulminant guillain-barrè syndrome after campylobacter jejuni enteritis and anti-ganglioside antibody. Internal Medicine, 2002, 41(10): 760-761.
21. Wakerley B R, Yuki N. Pharyngeal-cervical-brachial variant of guillain-barre syndrome. Journal of Neurology, Neurosurgery & Psychiatry, 2014, 85(3): 339-344.
22. Nakamura Y, Motoki M, Hirose T, et al. Fulminant guillain-barré syndrome showing severe pharyngeal-cervical-brachial weakness in the recovery phase: a case report. BMC Neurology, 2019, 19(1): 145.
23. Ito H, Hatanaka Y, Fukami Y, et al. Anti-ganglioside complex antibody profiles in a recurrent complicated case of gq1b-seronegative miller fisher syndrome and bickerstaff brainstem encephalitis: a case report. BMC Neurology, 2018, 18(1): 72.
24. Kaida K, Kusunoki S. Antibodies to gangliosides and ganglioside complexes in guillain–barré syndrome and fisher syndrome: mini-review. Journal of Neuroimmunology, 2010, 223(1–2): 5-12.
25. Koga M. Range of cross reactivity of anti-gm1 igg antibody in guillain-barre syndrome. Journal of Neurology, Neurosurgery & Psychiatry, 2001, 71(1): 123-124.
26. 吕科峰, 陆正齐. 吉兰-巴雷综合征与抗神经节苷脂复合物抗体[J]. 中国神经免疫学和神经病学杂志, 2008(6): 458-460.
27. Yuki N. Acute axonal polyneuropathy associated with anti-gm1 antibodies following campylobacter enteritis. Neurology, 1990, 40[2023–08–05].
28. Anonymous. Severe acute axonal form of guillain-barré syndrome associated with igg anti-gd1a antibodies. 1992.
29. Shimoya K, Ohnishi A, Hashimoto T, et al. [Guillain-barré syndrome with high titers of anti-gm2 and anti-galnac-gd1a antibody following cytomegalovirus hepatitis]. Rinsho Shinkeigaku Clinical Neurology, 1997, 37(2): 106-110.
30. Goto H, Fukudome T, Kusunoki S,et al. Subacute motor axonal neuropathy associated with the igg anti-galnac-gd1a antibody. BMJ Case Reports, 2011, 2011: bcr1120103507.