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Clinical characterization of anti-GQ1b antibody syndrome in Korean children

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ABSTRACT

Anti-GQ1b antibody syndrome encompasses Miller Fisher syndrome and its related disorders. We retrospectively identified 11 pediatric patients (5.4–18 years old) with anti-GQ1b antibody syndrome. Diagnoses of patients included acute ophthalmoparesis (n = 6), classical Miller Fisher syndrome (n = 2), Miller Fisher syndrome/Guillain-Barré syndrome (n = 1), acute ataxic neuropathy (n = 1), and pharyngeal-cervical-brachial weakness (n = 1). Nine patients (81.8%) fully recovered. Maturational change in GQ1b antigen expression and the accessibility of anti-GQ1b antibodies might be the cause of the difference of clinical manifestations in children with anti-GQ1b antibody syndrome.

1. Introduction

Miller Fisher syndrome (MFS) is an acute demyelinating polyneuropathy and an atypical variant of Guillain-Barré syndrome (GBS). The clinical triad of MFS includes ophthalmoparesis, ataxia, and areflexia. MFS is a rare condition with an annual incidence of less than one patient per one million population (Aranyi et al., 2012). It accounts for around 5% of adults with GBS (McGrogan et al., 2009) and 1.6% of children with GBS (Buompadre et al., 2006).

Since Chiba et al. discovered the increased activity of IgG anti-GQ1b antibodies in six patients with typical MFS (Chiba et al., 1992), the role of anti-GQ1b antibodies in MFS and its subtypes has been reported in multiple publications. At present, anti-GQ1b antibodies are sensitive biomarkers of MFS, and about 85% of patients with MFS are positive for IgG anti-GQ1b antibodies (Nishimoto et al., 2004; Odaka et al., 2001). Additionally, anti-GQ1b antibodies have been reported to be associated with incomplete form of MFS, acute ophthalmoparesis (AO), Bickerstaff's brainstem encephalitis (BBE), and pharyngeal-cervical-brachial weakness (PCBW) (Bickerstaff and Cloake, 1951; O'Leary et al., 1996; Odaka et al., 2001). The common autoimmune mechanisms of MFS and its variants can be explained by the presence of anti-GQ1b antibodies. The "anti-GQ1b antibody syndrome" encompasses different conditions with common mechanisms and different phenotypes (Odaka, Yuki, 2001). Although differences in fine specificity of anti-GQ1b antibodies are associated with diverse clinical features (Fukami et al., 2016), the role of anti-GQ1b antibodies is still not clear.

Because of the rarity of MFS and its subtypes in children, the study of anti-GQ1b antibody syndrome in children has been reported only through case reports or case series. This study aimed to identify the clinical and laboratory characteristics of anti-GQ1b antibody syndrome in Korean children. In addition, we described featured cases with atypical features of MFS.

2. Materials and methods

2.1. Patients

We reviewed 124 patients below 19 years of age, who had anti-ganglioside antibody panel tests performed at Pusan National University Hospital between November 2008 and February 2017. The reason for testing antiganglioside antibodies was acute peripheral neuropathies, including flaccid paralysis, sensory ataxia, ophthalmoparesis, and unexplained focal weakness. We identified patients who had increased levels of IgG anti-GQ1b titer of 50 or more. Their clinical records were retrospectively reviewed for the following information: sex, age at onset of symptoms, diagnosis, preceding illnesses, initial

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Table 1
Diagnostic criteria of anti-GQ1B antibody syndrome in this study.

Classification	Core clinical features	Incomplete form
GBS	Weakness and areflexia/hyporeflexia in all four limbs	
Pharyngeal-cervical-brachial weakness	Oropharyngeal, neck and arm weakness and arm areflexia/hyporeflexia Leg weakness (−)	Acute oropharyngeal palsy: arm and neck weakness (−) Acute cervicobrachial weakness: pharyngeal palsy (−)
MFS	Ophthalmoparesis, ataxia, and areflexia/hyporeflexia Limb weakness (−) Hypersomnolence (−)	Acute ophthalmoparesis: ataxia (−) Acute ataxic neuropathy: ophthalmoparesis (−)
BBE	Hypersomnolence and ophthalmoparesis and ataxia Limb weakness (−)	Acute ataxic hypersomnolence: ophthalmoparesis (−)

BBE: Bickerstaff's brainstem encephalitis; GBS: Guillain-Barré syndrome; MFS: Miller Fisher syndrome.

symptoms, neurologic signs and symptoms, laboratory findings including cerebrospinal fluid (CSF) examinations, neuroimaging results, and outcomes.

The diagnoses of MFS, GBS, BBE, and AO were based on established clinical criteria (Wakerley et al., 2014) and are summarized in Table 1. MFS was diagnosed when ophthalmoparesis, ataxia, and areflexia/hyporeflexia were observed without limb weakness and hypersomnolence. Absence of certain features indicates incomplete MFS: patients without ataxia have "AO," and those without ophthalmoparesis have "acute ataxic neuropathy" (AAN). For patients who present a single feature, "acute ptosis" for patients with ptosis only or "acute mydriasis" for those with mydriasis only can be diagnosed. A patient with limb weakness, ophthalmoparesis, ataxia, and areflexia was diagnosed with MFS/GBS. Patients who show hypersomnolence, ophthalmoparesis, and ataxia were considered to have BBE. PCBW was diagnosed when patients have oropharyngeal, neck, and arm weakness and arm areflexia/hyporeflexia without leg weakness.

2.2. Antiganglioside antibody test

Serum samples obtained from patients were tested for antibodies against GQ1b, GM1, and GD1b by using enzyme-linked immunosorbent assay (ELISA; BÜHLMANN GanglioCombi™ Light ELISA, BÜHLMANN Laboratories AG, Schönenbuch, Switzerland). Calibrator, controls, and patient sera were incubated for 2 h at 2–4 °C. Antiganglioside antibodies in the samples bound to the immobilized gangliosides at the pre-coated microtiter plate. After washing off unbound substances, the antibodies were detected with horseradish-peroxidase-labelled antibodies against human IgG and IgM. Following a second washing step in which unbound enzyme label was removed, a color substrate was added. The intensity of the color was measured at 450 nm. The color intensity was proportional to the titer of antibodies present in each sample. Results were attributed to one of the clinically validated titer categories: negative (< 30%), borderline (30–50%), positive (50–100%), and strongly positive (> 100%).

2.3. Standard protocol approval

This study was approved by the Ethics Committee of Pusan National University Hospital.

3. Results

3.1. Clinical features of 11 patients with anti-GQ1b antibody syndrome

We identified 11 patients with positive results for IgG anti-GQ1b antibodies. The patients' demographic and clinical characteristics are summarized in Table 2. The mean age at onset was 15.0 years (range 5.4–18 years), and there were eight boys (72.7%) and three girls (27.3%). Eight patients (72.7%) had a preceding infection, and five of them (41.7%) had gastrointestinal symptoms, such as diarrhea. Diagnoses of patients were as follows: AO (n = 6), MFS (n = 2), MFS/GBS (n = 1), AAN (n = 1), and PCBW (n = 1).

Table 2
Demographic and clinical characteristics of enrolled patients.

Demographics	Number of Patients (Total N = 11)
Age of onset, years	14.6 ± 3.7
Sex, male, N (%)	8 (72.7)
Preceding infection	
Gastrointestinal	5 (45.5)
Upper respiratory	3 (27.3)
None	3 (27.3)
Initial Symptoms	
Diplopia	8 (72.7)
Dysarthria	1 (9.1)
Ataxia	1 (9.1)
Blepharoptosis	1 (9.1)
Symptoms during illness	
Diplopia	8 (72.7)
Dysesthesia	6 (54.5)
Ataxia	4 (36.4)
Limb weakness	3 (27.3)
Blepharoptosis	3 (27.3)
Dysarthria	1 (9.1)
Dysphagia	1 (9.1)
Neurologic signs during illness	
External ophthalmoparesis	8 (72.7)
Areflexia	7 (63.6)
Sensory ataxia	5 (45.5)
Internal ophthalmoparesis	2 (18.2)
Decreased muscle power	2 (18.2)
Paresthesia	2 (18.2)
Blepharoptosis	2 (18.2)
Bulbar palsy	1 (9.1)

Initial symptoms were diplopia (n = 8), blepharoptosis (n = 1), ataxia (n = 1), and dysarthria (n = 1). Subjective symptoms during the clinical course were diplopia (n = 8), dysesthesia (n = 6), gait ataxia (n = 4), limb weakness (n = 3), blepharoptosis (n = 3), dysarthria (n = 1), and dysphagia (n = 1). Abnormal neurologic findings during the clinical course were external ophthalmoparesis (n = 8), areflexia (n = 7), sensory ataxia (n = 5), internal ophthalmoparesis (n = 2), decreased muscle power (n = 2), paresthesia (n = 2), blepharoptosis (n = 2), and bulbar palsy (n = 1).

The results of objective investigations of patients are listed in Table 3. Cerebrospinal fluid examination was performed in seven patients. Two (28.6%) of them showed albumino-cytologic dissociation, and no patient showed pleocytosis and oligoclonal band. In ELISA for antiganglioside antibodies of anti-GM1 and anti-GD1b, only patient 5 showed positivity for IgG anti-GD1b. Nerve conduction study was performed in nine patients. The results of eight patients (88.9%) were normal, and only patient 8 showed prolonged terminal latency. Brain magnetic resonance imaging (MRI) was performed in nine patients to rule out ophthalmoparesis and bulbar palsy of central origin. Patient 7 showed enhancement of bilateral abducens nerves (Fig. 1), and patient 8 showed focal encephalitis on the left internal capsule and thalamus (Fig. 2).

Table 3
Descriptions of patients' diagnoses, laboratory findings, treatment, and prognosis.

Patient No.	Diagnosis	CSF exam		NCS		Brain MRI	Treatment	Prognosis
		Days after onset[a]	ACD	Days after onset[b]	Result			
1	AAN	3	Yes	3	Normal	Normal	IVIG (400 mg/kg/d, 5 days)	CR
2	AO	ND	ND	8	Normal	Normal	Dexamethasone (0.1 mg/kg/d, 6 days)	CR
3	AO	11	No	12	Normal	Normal	ND	CR
4	MFS/GBS	1	Yes	3	Normal	ND	IVIG (400 mg/kg/d, 5 days)	CR
5	MFS	ND	ND	ND	ND	Normal	ND	CR
6	MFS	8	No	ND	ND	ND	ND	CR
7	AO	36	No	36	Normal	6th CN enhancement	IVIG (400 mg/kg/d, 5 days)	CR
8	PCBW	5	No	14	Prolonged terminal latency	White matter lesion	IVIG (400 mg/kg/d, 5 days)	PR
9	AO	ND	ND	4	Normal	Normal	ND	PR
10	AO	ND	ND	7	Normal	Normal	IVIG (400 mg/kg/d, 5 days)	CR
11	AO	8	No	8	normal	Normal	IVIG (400 mg/kg/d, 5 days)	CR

AAN: acute ataxic neuropathy; ACD: albumino-cytologic dissociation; AO: acute ophthalmoparesis: CN: cranial nerve; CR: complete recovery; CSF: cerebrospinal fluid; GBS: Guillain-Barré syndrome; IVIG: intravenous immunoglobulin; MRI: magnetic resonance imaging; MFS: Miller Fisher syndrome; NCS: nerve conduction study; ND: not done; PCBW: pharyngeal-cervical-brachial weakness; PR: Partial recovery.

[a] Time latency to CSF examination from the onset of symptoms.

[b] Time latency to NCS from the onset of symptoms.

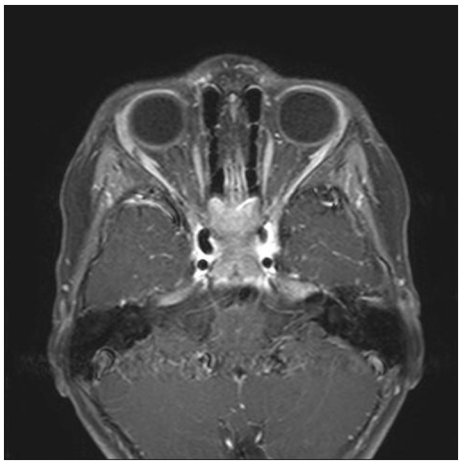

Fig. 1. Patient 7. Axial T1-weighted, gadolinium-enhanced brain MRI showing enhancement of bilateral abducens nerves (arrows).

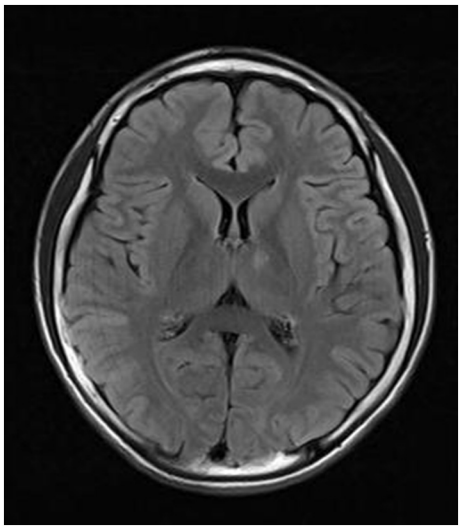

Fig. 2. Patient 8. Brain MRI T2 FLAIR axial image showing hyperintense signal changes involving the posterior limb of the left internal capsule and left thalamus (arrow).

Six patients (54.5%) were treated with intravenous immunoglobulin (400 mg/kg/day for five days), and one patient (18.2%) received dexamethasone (0.1 mg/kg/day for six days). Within three months, nine patients (81.8%) fully recovered, while two (18.2%) showed partial recovery.

3.2. Case reports

3.2.1. Patient 7 – illustrative typical case of AO

The patient was a four-year-old girl with a history of fever two weeks ago and esotropia for two weeks. Neurologic examination revealed bilateral lateral gaze palsy and bilateral mydriasis. Deep tendon reflexes of both knees and ankles were decreased. The patient was alert and did not have ataxia and weakness of extremities. Her cranial nerve examinations and cerebellar function tests were normal. Initial laboratory tests, including white blood cell count, hemoglobin, platelet, C-reactive protein, liver/kidney function test, and urine analysis, were normal. CSF studies did not exhibit pleocytosis, oligoclonal band, and albumino-cytologic dissociation. Nerve conduction studies were normal. Brain MRI showed the enhancement of bilateral abducens nerves (Fig. 1). The IgG anti-GQ1b antibody titer was strong positive (> 100%). The patient was diagnosed with AO and received a five-day course of intravenous immunoglobulin therapy (400 mg/kg/day). Her esotropia started to improve 14 days after the treatment and fully recovered three months after the onset.

3.2.2. Patient 8 – illustrative typical case of PCBW

The patient was a 15-year-old boy with history of dysarthria and numbness of both hands for one day after flu-like symptoms a week ago. His gag reflex and deep tendon reflex of both biceps and triceps were decreased. However, his other cranial nerve examinations were normal. Focal high signal intensity on the posterior limb of the left internal capsule was noticed in T2 FLAIR axial image of brain MRI, and focal encephalitis was suspected (Fig. 2). Nerve conduction studies of upper extremities showed prolonged terminal latency. CSF studies were normal. The result of anti-GQ1b antibody test was strong positive (> 100%). Intravenous immunoglobulin was administered (400 mg/kg/day for five days). The patient gradually improved. At three months after onset, he still had mild dysarthria.

4. Discussion

Miller Fisher described the clinical triad of acute ophthalmoparesis, areflexia, and ataxia as a variant of GBS in 1956 (Fisher, 1956). IgG

anti-GQ1b antibodies has been frequently found in sera of patients with MFS, GBS with ophthalmoparesis, AO, and BBE (Boylu et al., 2010; Fukami et al., 2016). In previous studies of anti-GQ1b antibody syndromes in adults, MFS and MFS/GBS were the most common diagnoses in 57–59% and 8–17% of patients, respectively (Fukami et al., 2016; Odaka et al., 2001). AO, BBE, AAN, and PCBW were less frequently diagnosed than MFS and MFS/GBS. Anti-GQ1b antibody is a biomarker of MFS and found in 80% of patients with MFS (Nishimoto et al., 2004). Based on our results, anti-GQ1b antibody syndrome in Korean children and adolescents presented diverse neurological manifestations. There were higher incidence of AO and lower incidence of MFS and MFS/GBS in this study than in previous reports. Of 11 patients with anti-GQ 1b antibody syndrome, six (54.5%) were diagnosed with AO, only two (18.1%) with MFS, and one (9.1%) with MFS/GBS.

The presence of preceding infections in MFS and GBS has been reported previously. In a case-control study, serology results showed that *Campylobacter jejuni* and *Haemophilus influenzae* infections were evident in 21% and 8% of MFS patients, respectively.(Koga et al., 2005) Molecular mimicry between GQ1b and lipo-oligosaccharides extracted from *C. jejuni* strains in MFS patients was demonstrated (Koga et al., 2005; Yuki et al., 1999). *C. jejuni* infection may provoke the production of anti-GQ1b antibodies in patients with MFS, and these antibodies bind to the paranodes and neuromuscular junction of the ocular, trochlear, glossopharyngeal, and vagus nerves. In this study, five patients (45.5%) had a history of gastrointestinal infection, but there was no patient with evidence of *C. jejuni*. GT1a is also known to be related with MFS, and anti-GT1a and anti-GQ1b antibodies showed cross-reactivity (Nagashima et al., 2004). In addition, GD1b also showed cross-reactivity with anti-GQ1b antibodies and is associated with deep sense impairment (Susuki et al., 2001b). In the present study, only patient 5 showed positivity for IgG anti-GD1b antibodies.

GQ1b is mainly expressed at the paranodal regions of the extramedullary portion of oculomotor, trochlear, and abducens nerves (Chiba et al., 1993). Additionally, muscle spindles, glossopharyngeal nerve, vagal nerve, and spinal anterior roots may express GQ1b (Fukami et al., 2016). The location of GQ1b expression is closely related with the clinical manifestations of anti-GQ1b antibody syndrome, such as external and internal ophthalmoparesis, ptosis, ataxia, bulbar palsy, and muscle weakness. However, the reason for the difference of clinical manifestations among individuals with anti-GQ1b antibody syndrome is unknown. Some researchers hypothesized that differences in fine specificity of anti-GQ1b antibodies are related to various clinical manifestations due to the different expression of gangliosides in different parts of the nervous system (Fukami et al., 2016; Susuki et al., 2001a). Individual difference of the expression sites of GQ1b and the accessibility of anti-GQ1b antibodies may be the cause of different clinical symptoms. Cross-reactivity of anti-GQ1 antibodies with other ganglioside antibodies differs among clinical symptoms and associated anatomic locations (Fukami et al., 2016, Susuki et al., 2001a). Maturational change of the expression of GQ1b and the accessibility of anti-GQ1b antibodies has not been studied. Differences of anti-GQ1b antibody syndrome among races have not been reported either. However, the high incidence of AO in anti-GQ1b antibody syndrome in our study may be associated with maturational or age-related factors and racial differences of the expression of GQ1b and the accessibility of anti-GQ1b antibodies.

Diagnostic tests, including CSF studies, nerve conduction study, and neuroimaging, were supportive, but clinical and neurological characteristics and the presence of anti-GQ1b antibodies were most helpful for the diagnosis of anti-GQ1b antibody syndrome. Only two patients (18.1%) showed albumino-cytologic dissociation in CSF studies, and one (11.1%) showed abnormal nerve conduction study results. Neuroimaging results were abnormal in two patients (18.1%). In patient 8, focal encephalitis at the left internal capsule was observed (Fig. 1). The patient did not present any symptom related with the lesion. It was considered as an accompanied lesion in the central nervous system without a significant symptom.

Intravenous immunoglobulin and plasma exchange have been used for the treatment of MFS and its subtypes. A total of six patients (54.5%) were treated with intravenous immunoglobulin (400 mg/kg/day for five days), and one (9.1%) received intravenous dexamethasone (0.1 mg/kg/day for six days). Among four patients without any treatment, three fully recovered and one showed persistent strabismus. The overall prognosis of anti-GQ1b antibody syndrome in Korean children was good. Nine patients (81.8%) recovered completely, while two (18.2%) partially recovered three months after the onset. Patient 8 had mild dysarthria, and patient 9 showed persistent strabismus and received corrective surgery one year after the onset. No patient had recurrence or relapse.

AO was the most common diagnosis in Korean children with anti-GQ1b antibody syndrome. Maturational changes or racial differences of the expression of GQ1b antigen and the accessibility of anti-GQ1b antibodies may be the reason for the disparity with previous reports in adults. Careful history taking, neurologic examinations, and anti-GQ1b antibody testing are important for prompt diagnosis in suspected patients. This study has several limitations, including a small simple size due to the rarity of anti-GQ1b antibody syndrome in children and the short follow-up periods. Prospective multicenter studies and longer follow-up periods are needed to clarify the characteristics of anti-GQ1b antibody syndrome in Korean children.

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