

Acute myelitis of children with positive anti-GM1 antibody

Case series and literature review

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Abstract

Rationale: To explore the clinical features, treatment, and prognosis of acute myelitis (AM) of children with positive blood anti-ganglioside (GM1) antibodies.

Patient concerns: Two cases of AM of children with positive anti-GM1 antibody were retrospectively collected and followed up for 6 months. Two cases had positive helicobacter pylori IgG antibody, and Case 2 also had positive mycoplasma IgM antibody.

Diagnoses: Two cases had typical symptoms of myelitis, abnormal spinal magnetic resonance imaging (MRI), and positive serum anti-GM1 IgM.

Interventions: They were treated with steroid, immunoglobulin and rehabilitation.

Outcomes: Symptoms of AM were relieved after treatment. After 6 months of follow-up, case 1 was fully recovered and case 2 was partially recovered. Summarizing previous reports in literature and our 2 cases, AM with positive anti-GM1 antibody can be induced by multiple pathogen infections. About 35.7% were fully recovered, 42.9% had mild sequelae, and 21.4% had severe sequelae.

Lessons: Post-infection immune injury plays an important role in the pathogenesis of AM with positive anti-GM1 antibody. H pylori and Mycoplasma pneumoniae infection may also induce AM with positive anti-GM1 antibody. Screening and treatment of pathogens were required and only 21.4% patients had severe sequelae after treatment.

Abbreviations: ADEM = acute disseminated encephalomyelitis, AM = acute myelitis, AQP4 = aquaporin-4 NMO = neuromyelitis optica, CRP = C-reactive protein, CSF = cerebrospinal fluid, GM1 = ganglioside 1, H. pylori = helicobacter pylori, MRI = magnetic resonance imaging, MSG = muscle strength grade, PLT = platelet, TGAb = antithyroglobulin antibody, TPOAb = thyroid peroxidase antibody, TSH = thyroid-stimulating hormone, WBC = white blood cell.

Keywords: acute myelitis, anti-GM1 antibody, post-infection immunity

1. Introduction

Acute myelitis (AM) is an acute inflammatory demyelination or necrosis of spinal cord caused by various autoimmune reactions.^[1] Current studies show that approximately 30% to 60% of AM are related to post-infection immune injury, and

most of them have upper respiratory tract infection or gastrointestinal infection and systemic disease before onset of illness.^[2–4] There were many reports of positive anti-GM1 antibody in immune-mediated neurologic diseases^[5–7] and more likely to be found in Guillain–Barre syndrome, but there have been few reports of AM with positive anti-GM1 antibody. We report clinical data, treatment, and follow-up of 2 patients who were treated in our hospital in 2016 to 2017.

Editor: N/A.

Key research and development project of Sichuan Provincial Science and Technology Department (2018FZ0054, 2017SZ0153) and National Science Foundation of China (81170607).

Ethical approval was obtained from the ethics committee of West China Second University Hospital, Sichuan University. Written informed consent was obtained from the parents of the patient for publication of this case report and any accompanying images.

The authors have no conflicts of interest to disclose.

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Medicine (2018) 97:20(e10796)

Received: 20 January 2018 / Accepted: 25 April 2018

<http://dx.doi.org/10.1097/MD.0000000000010796>

2. Methods

This study retrospectively analyzed the clinical data of AM with positive anti-GM1 antibody who were admitted between 2016 and 2017 in the West China Second University Hospital and followed up for 6 months. This study was approved by the Ethics Committee of the West China Second University Hospital and written informed consent was obtained from parents of 2 children.

The patients were follow-up for 6 months. Motor outcomes were divided into 4 categories according to scheme described by Defresne et al^[8]: full recovery; minor sequelae: able to walk independently but unable to run; mild sequelae: gait disturbances and walking with support; and severe sequelae: unable to walk independently.

3. Results

A total of 2 children with acute myelitis with positive anti-GM1 antibody is included in this series.

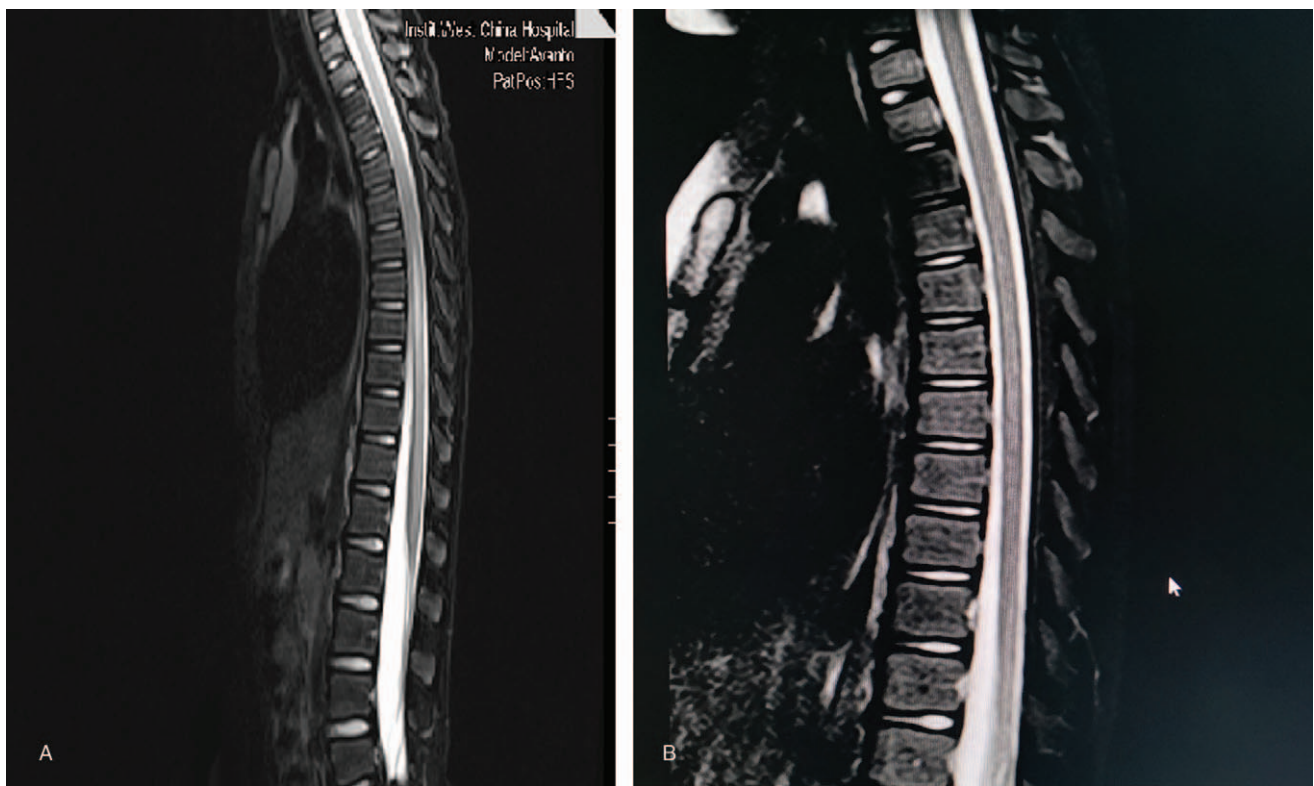


Figure 1. Spinal magnetic resonance imaging (MRI) changing of 2 cases in these reports. Case 1: (A) Thoracolumbar MRI showed long T1 and T2 signal of the spinal cord below T7 level. (B) Thoracolumbar MRI recovered completely at 1 month after discharge.

3.1. Case 1

A 5-year-old previously healthy female was admitted to our hospital with weakness of lower extremity and acute urinary retention for 1⁺ day, and denied history of trauma, infection, and vaccination. The lower limb muscle tension decreased and muscle strength grade (MSG) was 1/5. Temperature sensation of lower extremity below the knee was abnormal and the pain, tactile, and position sensation were normal. Bilateral knee tendon reflex decreased. Babinski sign was suspicious positive, incontinence of urine. Laboratory test: white blood cell (WBC) $18.16 \times 10^9/L$ ($3.6\text{--}9.7 \times 10^9/L$), N 81.2% (23.6–75%), Hb 134 g/L (110–146 g/L), platelet (PLT) $435 \times 10^9/L$ ($100\text{--}450 \times 10^9/L$), C-reactive protein (CRP) 1.77 mg/L (0–8 mg/L); serum liver and kidney function and electrolyte are normal; erythrocyte sedimentation rate 27.0 mm/h (<21 mm/h); T3: 1.33 nmol/L (1.6–4.1 nmol/L), T4: 124.8 nmol/L (93–200 nmol/L), thyroid-stimulating hormone (TSH): 1.453 mIU/L (0.64–6.27 mIU/L), FT3: 5.27 pmol/L (5.1–10.1 pmol/L), FT4: 22.74 pmol/L (12–22 pmol/L), antithyroglobulin antibody (TGAb): 89.8 IU/mL (<60 IU/mL), thyroid peroxidase antibody (TPOAb): 130.0 IU/mL (<60 IU/mL); auto-antibody, anti-cardiolipophospholipid antibodies, and anti-neutrophil cytoplasmic antibodies were negative; virus screening were negative; mycoplasma and chlamydia antibodies were negative. *Helicobacter pylori* IgG antibody was positive. Stool and urine routine tests were normal. Cerebrospinal fluid (CSF) test was normal. CSF and serum were checked at Peking Union Medical College Hospital and blood anti-GM1 IgG was weak-positive and anti-GM1 IgM was positive. Serum GD1b, GQ1b, AQP4 and NMO IgG antibodies were negative and CSF GM1, GD1b, GQ1b antibodies were negative. Head and thoracolumbar magnetic resonance imaging (MRI) showed long T1 and T2

signal of the spinal cord below T7 level and normal brain parenchyma (Fig. 1A). After ceftazidime, immunoglobulin (1 g/kg \times 2d) and methylprednisolone (400 mg/kg \times 3d) were used, and prednisone orally was continued, the patient's condition was improved. She was able to walk slowly on her own at the time of discharge and MSG of lower extremity was 4/5. Bilateral knee tendon reflex was induced and limbs sensation was recovered. Thoracolumbar MRI recovered completely (Fig. 1B) and the girl can walk normally at 1 month after discharge. The girl fully recovered after 3 months of discharge and prednisone was gradually discontinued. Thyroid function and blood GM1-IgG and IgM were retested and result was normal.

3.2. Case 2

A 5-year-old previously healthy boy was admitted because of fever accompanied with limb weakness for 1 day. The boy had a history of respiratory infection previous week, denied history of trauma and vaccination. He can answer to the point, but volume of the speech was small and had mild dysphagia. Double eyelids were slightly drooped, pharyngeal was hyperemia, and bilateral amygdale were I^o enlargement. He had tachypnea and weakened abdominal breathing; biceps reflex, triceps reflex, knee tendon reflex, and abdominal wall reflex did not elicit. MSG of lower extremity and left upper limb were 1/5, MSG of right upper limb was 3/5, limb muscle tension was decreased, and bilateral Babinski sign was positive. Sensation below both nipple levels was disappeared. Bowel and bladder were dysfunction. Laboratory examination: WBC $7.6 \times 10^9/L$ ($3.6\text{--}9.7 \times 10^9/L$), N% 72.8% (23.6–75%), Hb 125 g/L (110–146 g/L), PLT $319 \times 10^9/L$ ($100\text{--}450 \times 10^9/L$), CRP 2.0 mg/L (0–8 mg/L); liver and

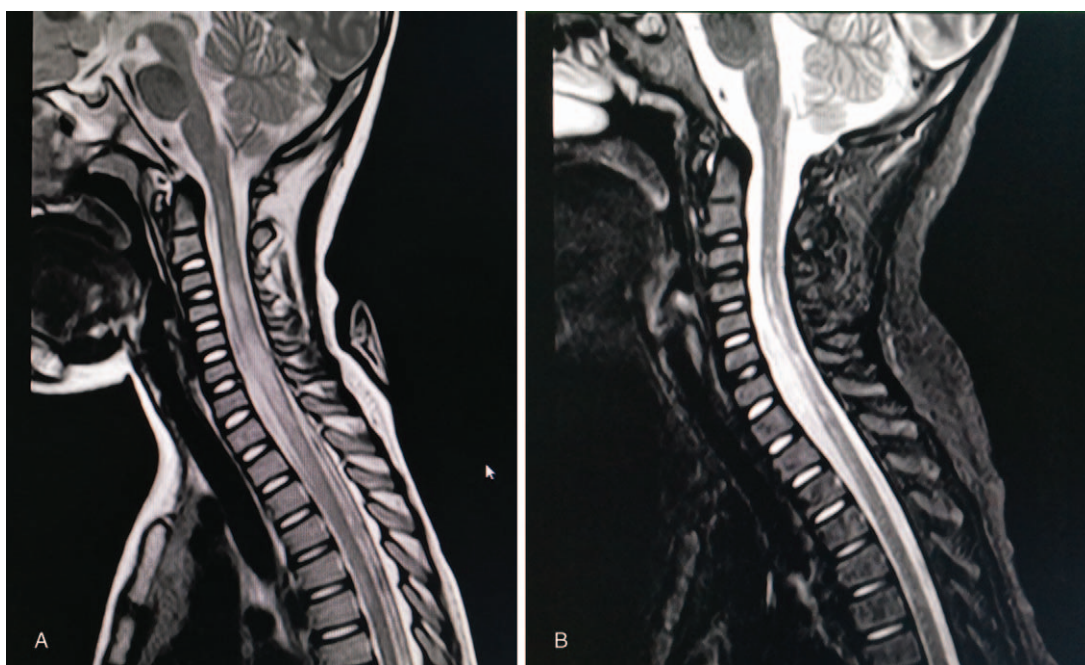


Figure 2. Case 2: (A) Cervical and thoracolumbar enhanced magnetic resonance imaging (MRI) showed abnormal intramedullary enhancement edema of spinal cord from C3 to T4. (B) Spinal MRI showed signal abnormal from C4 to T1, and the localized spinal cord was atrophy at 3 months after discharge.

kidney work, electrolyte, and blood transfusion immunity were normal; stool and urine routine tests were normal. Erythrocyte sedimentation rate 32.0 mm/h (<21 mm/h); T3 0.72 nmol/L (1.6–4.1 nmol/L), T4 45.7 nmol/L (93–200 nmol/L), TSH 3.937 mIU/L (0.64–6.27 mIU/L), FT3 2.34 pmol/L (5.1–10.1 mIU/L), FT4 11.15 pmol/L (12–22 pmol/L), TGAb 221.1 IU/mL (<60 IU/mL), TPOAb 374.8 IU/mL (<60 IU/mL). Autoantibody, anti-cardiolipin phospholipid antibody, and anti-neutrophils cytoplasmic antibody were negative. *H. pylori* antibody IgG and *Mycoplasma pneumoniae* antibody were positive (titer 1:320). Virus screening was negative. CSF test was normal. Serum anti-GM1 IgG was negative and anti-GM1 IgM was positive. Brain MRI was normal. Cervical and thoracolumbar enhanced MRI showed abnormal intramedullary enhancement and edema of spinal cord from C3 to T4 (Fig. 2A). Visual evoked potential showed prolonged latency, but fundus examination and facial nerve function test were normal. He had repeated fever for more than 10 days after admission and treatment with ceftriaxone and acyclovir and gamma globulin (2 g/kg). Methylprednisolone (500 mg/kg × 3d) was used and then oral prednisone was continued, but the boy still had recurrent fever. After azithromycin used to anti-mycoplasma infection, temperature gradually stabilized and limb movement gradually improved. When discharged, the boy had no dysphagia, pharyngeal reflex was recovered and eyelids drooping were partly improved. Moreover, left upper limb MSG is 2/5, right upper limb MSG is 3/5, lower extremity MSG is 1/5, and limb muscle tension was still decreased. Knee tendon reflexes and abdominal wall reflexes did not elicit. Bilateral Babinski sign was positive, ankle clonus was positive, and sensation below bellybutton level was disappeared.

Oral prednisone and rehabilitation are continued after discharged. A month later, spinal MRI was rechecked and indicated that the range of cervical myelin lesion was significantly reduced and other areas had not significantly altered. Three months after discharge, muscle strength of upper limb was

restored and MSG of lower limb is 2/5. However, he cannot stand and sensation below the knee did not recover. Spinal MRI was rechecked and showed signal abnormal from C4 to T1, and the localized spinal cord was atrophy (Fig. 2B). *M. pneumoniae* antibody titer decreased (1:160), T3 1.28 nmol/L, T4 72.4 nmol/L, TSH 1.037 mIU/L, FT3 4.72 pmol/L, FT4 22.24 pmol/L, TGAb 17.1 IU/mL, TPOAb <28 IU/mL. At present, he has been discharged from hospital for 6 months, and MSG of lower limb is 2 to 3/5, but still unable to stand.

4. Discussion

The AM is characterized with paralysis of spinal cord lesion, sensory disturbance, and autonomic nervous dysfunction,^[1–3] including acute partial transverse myelitis and longitudinal extensive transverse myelitis.^[9] Main pathologic changes were myelin swelling, demyelination, peripheral lymphocyte proliferation, axonal degeneration, perivascular inflammatory cell infiltration. Childhood AM is often characterized by longitudinal extensive transverse myelitis,^[2,10–12] including MRI lesions that span over at least 3 vertebral segments. Two children in this report have different degrees of limb paralysis, bowel and bladder dysfunction and sensory disturbance with abnormal spinal MRI signals over 3 vertebral segments.

Two children in this report were accompanied by positive blood anti-GM1 antibody and thyroid antibody, indicating that the immune response played an important role in the pathogenesis of AM. Ganglioside is a kind of acid glycolipid, which is one of the main components of the mammalian nerve cell membrane. Ganglioside can regulating nerve cell function and also serves as the target of autoantibody. In vitro experiments, Kanda et al^[13] confirmed that the anti-GM1 antibody had damaged the integrity of the blood-nerve barrier, exposed axon and caused injury. The emergence of anti-GM1 antibody is associated with post-infection immunity, which has been

reported in many immune-mediated neurologic diseases, including Guillain–Barre syndrome, poliomyelitis syndrome, and cerebellar ataxia. So far, there have been few reports of myelitis, and more frequently seen in intestinal bacterial infection, especially after *Campylobacter* infection (Table 1). After infection of *Campylobacter*, an immune response is produced, which stimulates producing of serum anti-GM1 antibody, resulting in neuroimmune injury.^[13–15] Baaret et al^[15] reported that a 17-year-old girl presented myelitis after intestinal *Campylobacter jejuni* infection accompanied with high titer anti-GM1 antibody, and the anti-GM1 antibody titer was reduced, and the prognosis was good after the treatment of *C jejuni*. Gaig et al^[16] and Huber et al,^[17] respectively, reported 2 patients of acute disseminated encephalomyelitis (ADEM), with positive anti-GM1 antibody. But Kalra et al^[10] reported 15 children with acute transverse myelitis, although 46% had anti-GM1 IgG positive and 33% had anti-GM1 IgM positive, there was no relationship between *Campylobacter* and anti-GM1 antibody. Two patients in this group did not do stool culture for *C jejuni*, but they had no diarrhea, vomiting, and other digestive tract symptoms and routine stool test was normal, so possibility of intestinal *C jejuni* infection was less likely.

In addition, anti-GM1 antibody can also be caused by other pathogen infections, including cytomegalovirus, leptospirosis, hepatitis E virus, pinworm, etc.^[18–20] Drulovic et al^[21] reported a case of transverse myelitis with positive anti-GM1 antibody after pinworm infection and indicated that the lipid compound of the pinworm contained GM1 and sulfatin, leading to molecular simulation mechanism. Rinsho Shinkeigaku^[22] reported that human T-lymphocytic leukemia virus infection causes chronic B lymphocytic leukemia and myelopathy accompanied by anti-GM1 antibody positive. Chong et al^[23] reported that acute flaccid myelitis was caused by viral infection, 28% patients had positive anti-GM1 IgM, which was similar to the positive rate in Guillain–Barre syndrome. Farbu et al^[24] also reported that the anti-GM1 antibody was present in 14% patients with polio syndrome. Our 2 patients had negative virus screening, and no parasitic ovum was tested in stool. However, both patients had positive serum *H pylori* IgG antibody, so it could not be excluded from the infection of *H pylori*.

Numerous studies have shown that *H pylori* had been involved in immunologic diseases, including multiple sclerosis, optical neuromyelitis, and so on.^[25] Long et al^[26] reported that *H pylori* antibody was found in 90.4% optical neuromyelitis, 95.8% high-risk optical neuromyelitis, 73.8% multiple sclerosis, and 59.3% control. Yoshimura et al^[27] showed that *H pylori* infection was associated with positive anti-aquaporin-4 antibody. In addition, Guillain–Barre syndrome^[28] and acute immune polyneuropathy patients had positive *H pylori* IgG antibody.^[29] Meanwhile, studies have shown that *H pylori* inhibited combination of GM1 and cholera toxin,^[30] which indicated that *H pylori* and GM1 have common antigenic epitopes. But until now there is no report of myelitis with *H pylori* infection. Our case 2 also had *M pneumoniae* infection, and the literature reported that *M pneumoniae* infection could result in myelitis,^[31,32] which could be combined with anti-neuronal antibody. However, no positive anti-GM1 antibody has been reported in myelitis, inducing by *M pneumoniae*, and we suspected case 2 had a mixed infection of *H pylori* and *M pneumoniae*. Whether *H pylori* or *M pneumoniae* can induce myelitis need to further study. Our study is a retrospective case series, and only 2 patients included, so we need much more cases to further study.

Table 1
Summary of acute myelitis with positive anti-GM1 antibody reported in literature.

Case no	Age (Y), sex	Diagnosis	Prior infection	Lower limb deficit	Upper limb deficit	Sensory loss level	Sphincter dysfunction	MRI	GM1 IgG	GM1 IgM	Immunotherapy	Antipathogen therapy	Outcome
1	Kalraet al ^[10] 6 M	Myelitis	N	Partial	N	N	N	D10–D12	+	-	MP	-	Full recovery
2	Kalraet al ^[10] 11 M	Myelitis	N	Complete	Partial	N	Y	C1–D12	+	+	MP	-	Full recovery
3	Kalra ^[10] 8 M	Myelitis	Diarrhea	Partial	N	T6	Y	D4–D8	-	+	MP	-	Mild sequelae
4	Kalraet al ^[10] 4 M	Myelitis	URI	Partial	N	?	Y	D4–D8	+	-	MP	-	Full recovery
5	Kalraet al ^[10] 3.5 F	Myelitis	URI	Partial	N	T10	Y	D3–D9	+	+	MP	-	Severe sequelae
6	Kalraet al ^[10] 9 M	Myelitis	N	Complete	N	T6	Y	C1–DD10	+	-	Dexa	-	Mild sequelae
7	Kalraet al ^[10] 10 M	Myelitis	Diarrhea	Complete	N	T12	Y	D5–DD10	+	+	MP	-	Severe sequelae
8	Kalra ^[10] 6 M	Myelitis	Diarrhea	Partial	Partial	-	Y	D2–DD10	-	+	MP	-	Mild sequelae
9	Baaret et al ^[15] 17 F	Myelitis	Diarrhea	Partial	Partial	T4	Y	C2–C7 brainstem	+	+	MP	Levofloxacin oral	Full recovery
10	Drulovic et al ^[21] 40 F	Myelitis	pinworm	Partial	N	T12	Y	Normal	+	+	MP	Mebendazole	Mild sequelae
11	Gaig et al ^[16] 38 F	ADEM	Diarrhea	Partial	N	Perianal	Y	Conus medullaris medulla	+	?	MP	Ceftriaxone	Mild sequelae
12	Huber et al ^[17] 23 M	ADEM	CJ Diarrhea V accination	Partial	N	N	N	cerebellar peduncle, pons, basal ganglia	+	-	MP	Amoxicillin acyclovir	Mild sequelae
13	Case 1 5 F	Myelitis	N	Partial	N	L1	Y	T7	+	+	MP, IVIG	ceftriaxone	Full recovery
14	Case 2 5 M	Myelitis	URI	Complete	Partial	T4	Y	C6	+	+	MP, IVIG	Ceftriaxone azithromycin	Severe sequelae

ADEM = acute disseminated encephalomyelitis, CJ = *Campylobacter jejuni*, Dexa = dexamethasone, F = female, IgG = immunoglobulin G, IgM = immunoglobulin M, IVIG = intravenous immunoglobulin, M = male, MP = methylprednisolone, MRI = magnetic resonance imaging, N = no, PE = plasma exchange, URI = upper respiratory infection, Y = yes.

In this paper, we summarized 14 cases of myelitis and ADEM with positive anti-GM1 antibody (Table 1).^[10,15–17,21] Among them, 3 cases (1 case of myelitis, 2 cases ADEM) had jejunum *Campylobacter* infection and had no severe sequelae after treated with antibiotics and steroid. One patient was infected with pinworm and had no severe sequelae after treatment with antiparasitic and steroid. Our case 2 also had clinical symptoms improved after anti-mycoplasma therapy. All cases were treated with steroid therapy (1 case was used with dexamethasone, the other cases used with methylprednisone). One case had poor effect with steroid therapy and then plasma exchange was used; Intravenous immunoglobulin was used in our 2 patients. In 14 cases, 12 cases (85.7%) had positive anti-GM1 IgG, 9 cases (64.3%) had positive anti-GM1 IgM, and 7 cases (50%) had both positive GM1 IgM and IgG. After treatment, 5 cases (35.7%) were fully recovered, 6 cases (42.9%) had mild sequelae, and 3 cases (21.4%) had severe sequelae (2 cases were anti-GM1 IgM and IgG antibody double positive).

5. Conclusion

To sum up, the children with positive anti-GM1 antibody AM is rare, myelitis and ADEM with positive anti-GM1 antibody in pediatric may be induced by multiple pathogens including *H pylori* and *M pneumoniae* infection. Screening for pathogens and anti-pathogens treatment may alleviate the sequel. Among all of this cases reported before, only 21.4% had severe sequelae after treatment.

Acknowledgment

The authors thank the patient's parents who provided their information.

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References

- Transverse Myelitis Consortium Working Group Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology* 2002;59:499–505.
- Pidcock FS, Krishnan C, Crawford TO, et al. Acute transverse myelitis in childhood: center-based analysis of 47 cases. *Neurology* 2007;68:1474–80.
- Paine R, Byers R. Transverse myelopathy in childhood. *AMA Am J Dis Child* 1953;85:151–63.
- Jeffery DR, Mandler RN, Davis LE. Transverse myelitis: retrospective analysis of 33 cases with differentiation of cases associated with multiple sclerosis and parainfectious events. *Arch Neurol* 1993;50:532–5.
- Volta U, De Giorgio R, Granita A, et al. Anti-ganglioside antibodies in celiac disease with neurological disorders. *Dig Liver Dis* 2006;38:183–7.
- Pestronk A, Adams RN, Clawson N, et al. Serum antibodies to GM1 ganglioside in amyotrophic lateral sclerosis. *Neurology* 1988;38:1457–61.
- Shill HA, Alaedini A, Bushkara KO, et al. Anti-ganglioside antibodies in idiopathic and hereditary cerebellar degeneration. *Neurology* 2003;60:1672–3.
- Defresne P, Hollenberg H, Husson B, et al. Acute transverse myelitis in children: clinical course and prognostic factors. *J Child Neurol* 2003;18:401–6.
- Pittock SJ, Lucchinetti CF. Inflammatory transverse myelitis: evolving concepts. *Curr Opin Neurol* 2006;19:362–8.
- Kalra V, Sharma S, Sahu J, et al. Childhood acute transverse myelitis: clinical profile, outcome, and association with antiganglioside antibodies. *Child Neurol* 2009;24:466–71.
- Defresne P, Hollenberg H, Husson B, et al. Acute transverse myelitis in children: clinical course and prognostic factors. *Child Neurol* 2003;18:401–6.
- Aberle J, Kluwe J, Pawlas F, et al. Severe myelitis following infection with *Campylobacter* enteritis. *Eur J Clin Microbiol Infect Dis* 2004;23:134–5.
- Kanda T, Iwasaki T, Yamawaki M, et al. Anti-GM1 antibody facilitates leakage in an in vitro blood-nerve barrier model. *Neurology* 2000;55:585–7.
- Jacobs BC, Hazenberg MP, Van Doorn PA, et al. Cross-reactive antibodies against gangliosides and *Campylobacter jejuni* lipopolysaccharides in patients with Guillain-Barré or Miller Fisher syndrome. *J Infect Dis* 1997;175:729–33.
- Baaret I, Jacobs BC, Govers N, et al. *Campylobacter jejuni*-induced acute transverse myelitis. *Spinal Cord* 2007;45:690–4.
- Gaig C, Valdeoriola F, Saiz A. Acute disseminated encephalomyelitis associated with *Campylobacter jejuni* infection and antiganglioside GM1 IgG antibodies. *J Neurol* 2005;252:613–4.
- Huber S, Kappos L, Fuhr P, et al. Combined acute disseminated encephalomyelitis and acute motor axonal neuropathy after vaccination for hepatitis A and infection with *Campylobacter jejuni*. *J Neurol* 1999;246:1204–6.
- Khalili-Shirazi A, Gregson A, Gray I, et al. Antiganglioside antibodies in Guillain-Barré syndrome after a recent cytomegalovirus infection. *J Neurol Neurosurg Psychiatr* 1999;66:376–9.
- Lepur D, Himbele J, Klinar , et al. Antiganglioside antibodies-mediated leptospiral meningomyeloencephalopolyneuritis. *Scand J Infect Dis* 2007;39:472–5.
- Maurissen I, Jeurissen A, Strauven T, et al. First case of anti-ganglioside GM1-positive Guillain-Barré syndrome due to hepatitis E virus infection. *Infection* 2012;40:323–6.
- Drulovic J, Dujmovic I, Stojisavljevic N, et al. Transverse myelopathy in the antiphospholipid antibody syndrome: pinworm infestation as trigger? *J Neurol Neurosurg Psychiatry* 2000;68:249.
- Akamatsu MM, Mitsui Y, Kihara M, et al. A case of polyneuropathy with B-CLL and HTLV-I associated myelopathy (HAM). *Rinsho Shinkeigaku* 1997;37:701–3.
- Chong PF, Kira R, Mori H, et al. Clinical features of acute flaccid myelitis temporally associated with an enterovirus D68 outbreak: results of a Nationwide Survey of Acute Flaccid Paralysis in Japan, August–December 2015. *Clin Infect Dis* 2018;66:653–64.
- Farbu E, Rekand T, Tysnes OB, et al. GM1 antibodies in post-polio syndrome and previous paralytic polio. *J Neuroimmunol* 2003;139:141–4.
- Smyk DS, Koutsoumpas AL, Mytilinaiou MG, et al. *Helicobacter pylori* and autoimmune disease: cause or by stander. *World J Gastroenterol* 2014;20:613–29.
- Long Y, Gao C, Qiu W, et al. *Helicobacter pylori* infection in neuromyelitis optica and multiple sclerosis. *Neuroimmunomodulation* 2013;20:107–12.
- Yoshimura S, Isobe N, Matsushita TY, et al. Distinct genetic and infectious profiles in Japanese neuromyelitis optica patients according to anti-aquaporin 4 antibody status. *J Neurol Neurosurg Psychiatry* 2013;84:29–34.
- Chiba S, Sugiyama T, Matsumoto H, et al. Antibodies against *Helicobacter pylori* were detected in the cerebrospinal fluid obtained from patients with Guillain-Barré syndrome. *Ann Neurol* 1998;44:686–8.
- Nevo Y, Pestronk A. Acute immune polyneuropathies: correlations of serum antibodies to *Campylobacter jejuni* and *Helicobacter pylori* with anti-GM1 antibodies and clinical patterns of disease. *J Infect Dis* 1997;176(Suppl 2):S154–6.
- Sack DA, Lastovica AJ, Chang SH, et al. Microtiter assay for detecting *Campylobacter* spp. and *Helicobacter pylori* with surface gangliosides which bind cholera toxin. *J Clin Microbiol* 1998;36:2043–5.
- Goebels N, Helmchen C, Abele-Horn M, et al. Extensive myelitis associated with *Mycoplasma pneumoniae* infection: magnetic resonance imaging and clinical long-term follow-up. *J Neurol* 2001;248:204–8.
- Tsiodras S, Kelesidis T, Kelesidis I, et al. *Mycoplasma pneumoniae*-associated myelitis: a comprehensive review. *Eur J Neurol* 2006;13:112–24.