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Influence of antibody detection on diagnosis and treatment of recurrent optic neuritis and myelitis

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Abstract

Purpose Identify the clinical features of recurrent optic neuritis and myelitis, improve the detection of related antibodies, make an early diagnosis, and give reasonable treatment. Materials and MethodsThe clinical data of 2 patients with recurrent optic neuritis and myelitis in our hospital were collected, including basic information, onset characteristics, signs of visual impairment, neurological signs, imaging features of the central nervous system, laboratory results, treatment and effects. Results Both patients were female, one patient with late onset and another patient with early onset. Visual impairment was decreased visual acuity and visual field defect. Spinal cord damage was segmental, resulting in sensorimotor disturbances in the limbs and sensory disturbances in the trunk. The early-onset patient was accompanied by brain damage. Serum AQP4-IgG positive in the late-onset patient, no antibody testing in the early-onset patients. Glucocorticoid therapy was effective in the acute phase. Glucocorticoid side effects were found in the early-onset patient. ConclusionsRecurrent optic neuritis and myelitis are common in MS, NMOSD and MOGSD.Early diagnosis requires identification of clinical manifestations and imaging features. At the same time, early detection of serum AQP4 antibodies, MOG antibodies and other autoimmune-related antibodies is required.

Keywords

Antibody; recurrence;optic neuritis; myelitis.

1. Introduction

Recurrent optic neuritis and myelitis are common in MS (multiple sclerosis), NMOSD (neuromyelitis optica spectrum disorder), and MOGAD (myelin oligodendrocyte glycoprotein-associated disorder). These diseases are associated with demyelination of the central nervous system. Although the clinical manifestations and imaging features are similar, the pathogenesis, treatment, and prognosis are different.Optic neuritis causes visual impairment. Myelitis causes sensory-motor impairment of limbs.The patient's quality of life is affected. In order to improve prognosis and reduce functional impairment, early diagnosis is required.

2. Materials and Methods

2.1. Data

Clinical data of 2 patients with recurrent optic neuritis and myelitis.

2.2. Methods

The basic information, onset forms, visual characteristics, neurological signs, imaging features of the central nervous system, Laboratory results, treatment and effects of 2 patients were collected.

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3. Results

3.1. Basic information, Onset characteristics, Signs of visual impairment, Neurological signs

Both patients were female. The disease course of late-onset patient was more than 1 year, and the disease course of early-onset patient was more than 20 years.The optic neuritis and myelitis recurred. Features of visual impairment included vision loss, visual field defect,increased pupil diameter, sluggish or absent light reflexes,optic atrophy. Features of spinal cord damage included decreased limb muscle strength and sensation,sensory disturbances in the trunk plane,active tendon reflexes in the extremities,positive pyramidal tract signs. See Table 1 and Figure A.

3.2. Imaging features of the central nervous system and Laboratory results

Spinal cord MRI showed short segment lesions of the cervical or thoracic cord, long T2 signal, STIR high signal.MRI showed different abnormal signals in the brain. Serum AQP4 antibody in late-onset patient was positive and autoimmune antibodies were negative. Serum AQP4 antibody and autoimmune antibodies were not detected in the early-onset patient. See Table 2 and Figures B, C, D, and E.

3.3. Treatment and Effects

Two patients were treated with glucocorticoids in the acute phase, and their symptoms improved. The early-onset patient had side effects such as hyperglycemia, hypertension, and hyperlipidemia after repeated use of glucocorticoids. No immune drugs were used for prevention during the remission period.

Table 1: basic information and clinical features

	early-onset patient	late-onset patient
Gender	female	female
First onset	36 years old	56 years old
Last onset	57 years old	57 years old
Disease course	21 years	1.5 years
Optic neuritis frequency	3 times	2 times
Myelitis frequency	7 times	1 time

Table 2: Imaging features of the central nervous system and Laboratory results

		early-onset patient		late-onset patient
Time		December 2021	October 2021	September 2020
MRI position	Spinal cord level of vertebralbodies	Cervical 2-4 Thoracic 3-5	Cervical 5-6	Thoracic 2-3
	Brain	bilateral paraventricular		right internal capsule
Serum AQP4 antibody		no detection		positive
Autoimmune antibodies		no detection		negative

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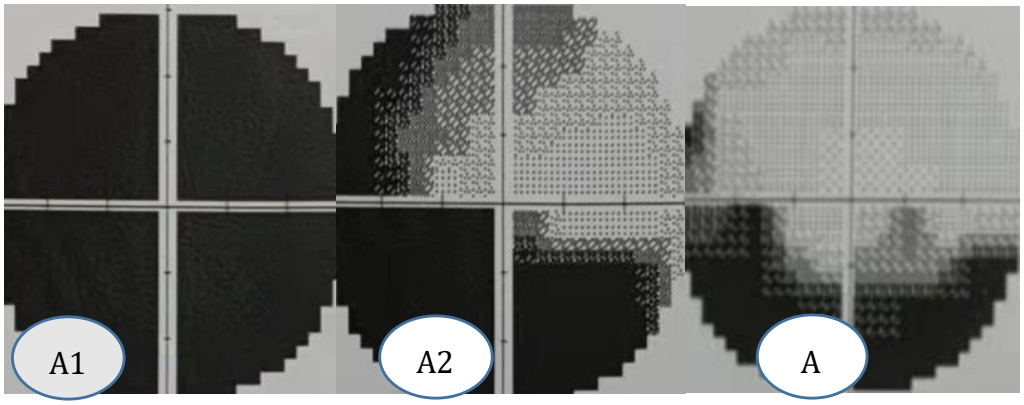

Figure A (the early-onset patient) : A1,Right eye, visual field defect(December 2021). A2, Left eye, visual field defect (December 2021). A3, Left eye, visual field defect(June 2016).

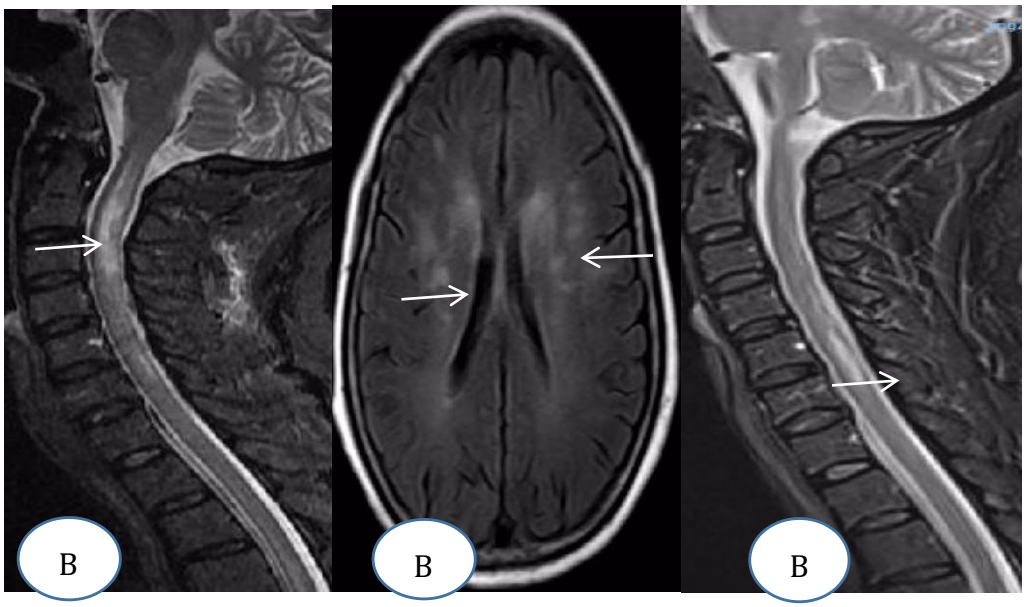

Figure B (the early-onset patient): B1, PatchySTIR highsignal in cervical spinal cord (December 2021). B2,Dotted FLAIR highsignal in head (December 2021). B3,Long stripSTIR highsignal in cervical spinal cord (October 2021).

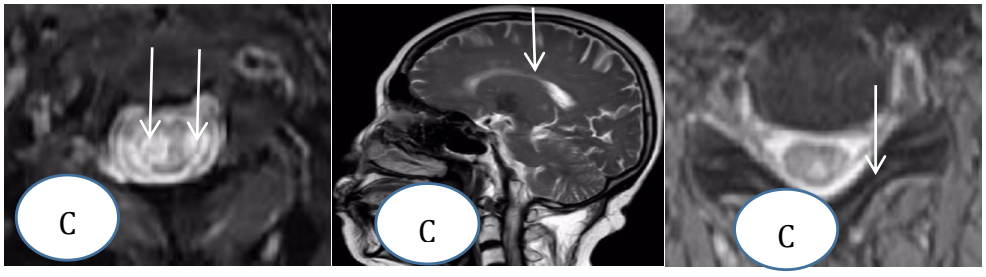

Figure C(the early-onset patient): C1, STIR highsignal in the cross section of the cervical spinal cord (December 2021) . C2, Dotted FLAIR highsignal in the sagittal plane of the head (December 2021). C3, STIR highsignal in the cross section of the cervical spinal cord (October 2021).

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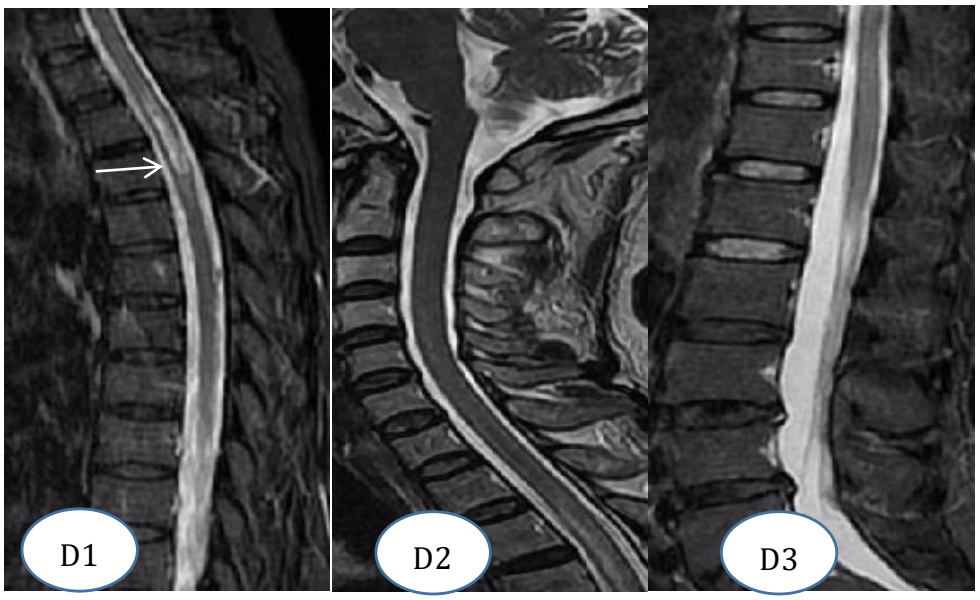

Figure D(the late-onset patient) (April 2020): D1,Long strip STIR high signal of thoracic spinal cord . D2, Normal cervical spinal cord. D3,Normal lumbar spinal cord.

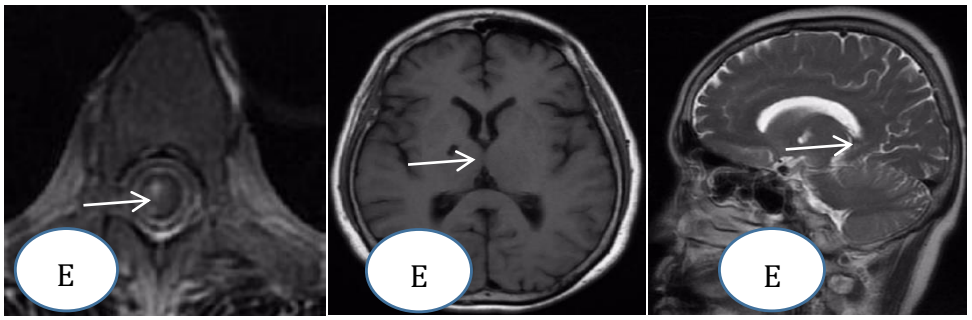

Figure E ((the late-onset patient) (April 2020): E1, STIR high signal of thoracic spinal cord cross-section. E2, FLAIR low signal surrounding ring high signal in head. E3, Long T2 signal of head sagittal.

4. Conclusions

4.1. Recurrent optic neuritis and myelitis

In the disease course of 2 patients, Optic neuritis occurred repeatedly with decreased vision in both eyes, blindness, defective field of view. Subsequently, Myelitis also occurred repeatedly with limb sensorimotor disturbances, trunk sensory disturbances and sphincter dysfunction. The late-onset patient was diagnosed with NOMSD due to AQP4-IgG positive. The early-onset patient was diagnosed with MS without antibody test.

Glucocorticoid treatment was effective in most attacks. However, recurrent optic neuritis and myelitis cause functional impairment and reduce quality of life.Recurrence is usually caused by the following factors: whether the diagnosis is correct［1］, whether the treatment in the acute phase is reasonable, and whether preventive treatment is used in the remission phase.

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4.2. Diagnosis

The late-onset patient had recurrent optic neuritis and myelitis. The patient was initially diagnosed with neuromyeloid MS due to the extent of spinal cord lesions involving 2 segments [2].But with the discovery of specific aquaporin-4 (AQP4) antibodies, NMOSD have been proposed. NMOSD occurs in young and middle-aged people, more common in women, common in Asians, with recurrent optic neuritis [3]. Although magnetic resonance imaging (MRI) showed a spinal cord lesion involving less than 3 segments, the AQP4 antibody test was positive, the patient was diagnosed with NMOSD.

The early-onset patient, a woman, had her first onset at a young age. Optic neuritis and myelitis recurred, and the white matter of the brain was also affected.The patient was initially diagnosed with MS because of the temporal and spatial multiple features. However, the patient need to be differentiated not only from NMOSD, but also from MOGAD. Adult MOGAD is mostly manifested as optic nerve-spinal phenotype. Myelin oligodendrocyte glycoprotein (MOG) is present in serum AQP4-negative patients [4]. Detection of serum AQP4 antibodies, MOG antibodies, and other autoimmune-related antibodies is necessary for diagnosis.

4.3. Treatment in the acute phase

According to research, the use of glucocorticoids is different in MS,NMOSD and MOGAD [2] [3] [4]. The 2 patients had recurrent attacks with the reduction of glucocorticoids. It was necessary to analyze whether the treatment was reasonable. Differences in glucocorticoid therapy are related to diagnosis and preventive treatment. The core is a clear diagnosis.

After long-term use of glucocorticoids, patients may experience side effects such as hyperglycemia, hypertension, and hyperlipidemia .At the same time, it is necessary to prevent complications such as upper gastrointestinal bleeding, electrolyte imbalance, osteoporosis, and necrosis of the femoral head. To reduce the side effects of glucocorticoids, prevention of recurrence is necessary.

4.4. Prevention

According to research, prevention is different in MS,NMOSD and MOGAD [2] [3] [4]. Patients with NMOSD or MOGAD can get worse if they receive treatments such as interferon beta, tocilizumab, and fingolimod, which are used to prevent MS. A definitive diagnosis is necessary.

The late-onset patient had a clear diagnosis of NMOSD and had indications for preventive treatment. It is necessary to communicate with the patient's family about the importance of prevention. Uncertainty about diagnosis in the early-onset patient has implications for prevention. It is necessary to communicate with the patient's family repeatedly to detect serum AQP4 antibody, MOG antibody and other autoimmune-related antibodies.

5. Closing remarks

The transition of clinical treatment from standardized treatment to individualized precision treatment requires the improvement of molecular immunopathology [5].Patients with recurrent optic neuritis and myelitis need to detect serum AQP4 antibody, MOG antibody and other autoimmune-related antibodies for early diagnosis. It is worth noting that OB is not only found in MS, but also in NMOSD and MOGAD [2]. AQP4-IgG may be negative at the first attack or at a certain stage of the disease course in patients with NMOSD [3]. MOG antibody is positive in some AQP4 antibody negative NMOSD [4]. The impact of false negative and false positive antibodies on diagnosis cannot be ignored.

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