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Case Report

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Meningitis? (Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody-related disease) A clinical analysis

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Abstract

Objective: *By analyzing meningitis? (Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody-related diseases) clinical diagnosis and treatment methods to improve clinicians' understanding of autoimmune encephalitis.*

Methods: *Collect 1 case of meningitis? (Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody-related disease) patient's clinical data, combined with literature review, to analyze the diagnosis and treatment methods of this case.*

Results: *The patient, male, 26 years old, was admitted to the hospital mainly because of "headache for 12 days and convulsions once". After imaging examination, hematology examination, body fluid examination, and repeated confirmation of the clinical treatment plan through remote consultation, hormones, immunosuppressants, immunoglobulins, anti-inflammatory and anti-viral treatments were given. He was discharged from the hospital and re-examined 2 months later. His condition became stable and his indicators improved.*

Conclusion: *For patients with autoimmune encephalitis, in addition to actively doing antibody detection and adjuvant therapy, it is necessary to adhere to the treatment plan mainly focusing on the treatment of autoimmune encephalitis, and do not focus too much on the relevant antibody indicators, so as to avoid deviations in the formulation of treatment plans, resulting in The condition fluctuates, and the patient's condition persists.*

Case Introduction

The patient, male, 26 years old, was admitted to the hospital mainly because of "headache for 12 days and convulsions once".

Past history: past health, denied hypertension, diabetes, coronary heart disease, cerebrovascular disease, infectious disease history.

History of present illness: The patient developed headache without obvious incentive on November 20, 2021, mainly in the right temporal region, pulsatile, with intermittent vomiting. Facial twitching appeared on the 24th, and went to the second hospital of the city. The head CT examination was normal, and no special treatment was given. On the 25th, there was no obvious cause for shouting, hitting hard objects with the head, unconsciousness, and involuntary shaking of the limbs, patients with fecal incontinence (specific episode manifestations unknown) were unable to recall these manifestations. During the attack, the patient was sent to the ICU of the Second City Hospital by his family, and his consciousness recovered after 2 hours. After two times of cerebrospinal fluid extraction, "14 paraneoplastic syndromes" were sent out. The second result showed "anti-myelinating oligodendrocytes" Cellular glycoprotein immunoglobulin G antibody (MOG antibody)" was positive with a titer of 1:32. Intracranial pressure is too high to measure. Both cerebrospinal fluid white blood cell counts were high at 165*10^6/L (range 0-8*10^6/L). Symptomatic treatment such as "C ball, debakin, antibiotics" was given. Since then, the patient has become conscious, has no more convulsions, and still has symptoms of headache and vomiting. At present, the patient has no visual rotation, no diplopia, no falls, no chest tightness, shortness of breath, and no dyspnea. Today, I came to our hospital for systematic treatment. Since the onset of the disease, the general state of the patient was acceptable, and there was no obvious abnormality in the second stool, and the sleep was acceptable. After admission, the patient had a low-grade fever (37.4-37.5 degrees Celsius) in the afternoon, which could be relieved by physical cooling. The patient had been vaccinated against COVID-19 before January. During the headache, the symptoms of cold medicines such as ibuprofen and Xinkangtec have been improved. No obvious abnormality was found in clinical examination. On December 2, he applied for a remote consultation from the Neurology Department

of Peking Union Medical College Hospital, and the consultation experts gave a diagnosis of "MOG antibody-related diseases". Administration regimen: 60 mg of prednisone acetate tablets were taken orally once a day, reduced by 1 tablet per week to 30 mg/day, maintained for one month and followed up after a month; antiviral treatment for 2 weeks; control of epileptic seizures. On December 6, the blood culture was negative, and it began to exceed 38 degrees Celsius in the morning, and the physical cooling did not ease significantly, and the body temperature dropped to about 37 degrees Celsius during the day. On December 8, intracranial pressure was 260mmHao (range 80-180mmHao), cerebrospinal fluid white blood cell count was high at $60*10^6$/L (range 0-$8*10^6$/L), blood routine*white blood cell count was 15.2810^9/L On the high side, antibiotics (cefotaxime sodium 2.0 g intravenously twice a day) were treated symptomatically. On the morning of December 10, the body temperature exceeded 38 degrees Celsius, and it persisted. The antibiotic was upgraded to meropenem 0.5 g twice a day, and the body temperature subsequently dropped to normal. The blood routine*white blood cell count on December 11 was high at 17.1710^9/L, and the blood routine*white blood cell count on December 13 was high at 19.7110^9/L. On December 14, the intracranial pressure burst, and the white blood cell count in the cerebrospinal fluid was high at $65*10^6$/L. Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody (MOG) in serum: positive with a titer of 1:100. Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody (MOG) in cerebrospinal fluid: positive with a titer of 1:10. The rest of the examinations, such as ESR, procalcitonin, CRP, and GGM tests, were not abnormal. On December 15th, he applied for a teleconsultation of the Neurology Department of Peking Union Medical College Hospital again. After checking the report in detail, he fully discussed his condition. The initial diagnosis was meningoencephalitis (anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody-related disease). The treatment plan was adjusted to: full-dose hormone therapy, methylprednisolone 80 mg per day, for 7-14 days, and if necessary, C-ball therapy and symptomatic intracranial pressure reduction treatment; lumbar puncture again in 5-7 days, review the results of cerebrospinal fluid, and recommend Second-generation sequencing of cerebrospinal fluid, and then adjust the treatment plan according to the results of cerebrospinal fluid; stop antiviral therapy. To ensure the curative effect, add human immunoglobulin injection 35g/day. On December 16th, the blood routine*white blood cell count was high at 14.3310^9/L. On December 21, a lumbar puncture was performed, the intracranial pressure was 220 mmHao (range 80-180 mmHao), and the white blood cell count in the cerebrospinal fluid was increased by $50*10^6$/L. Routine blood*white blood cell count was high at 19.7110^9/L. Lumbar puncture was performed on December 27, the intracranial pressure was 170 mmHao (range 80-180 mmHao), and the white blood cell count in cerebrospinal fluid was normal at $8*10^6$/L. The patient was discharged after his condition improved. On February 16, the patient returned to the hospital for re-examination, and the blood routine* white blood cell count was high at 12.7510^9/L. A lumbar puncture was performed on February 17, the intracranial pressure was 200 mmHao (range 80-180 mmHao), and the white blood cell count in the cerebrospinal fluid was normal at $5*10^6$/L. Sometimes the hands tremble uncomfortably, and treat them symptomatically. The overall indicators improved compared with before, and he was discharged from the hospital on February 19.

Discussison

Encephalitis is a neurological disorder caused by diffuse or multiple lesions in the brain parenchyma. The pathological changes mainly involve gray matter and neurons, but also white matter and blood vessels. Autoimmune encephalitis (AE) generally refers to a type of encephalitis mediated by autoimmune mechanisms [1]. Since the discovery of NMDAR encephalitis in 2007, a series of autoantibodies against neuronal cell surface or synaptic proteins have been discovered one after another. At present, the prevalence of AE in patients with encephalitis is about 10%-20% [2]. Among them, NMDAR encephalitis is the most common, accounting for about 80% of AE patients [3]. In addition to this, there are many types of AE, including MOG antibody typing. The clinical manifestations of AE include the following manifestations. First, there are prodromal symptoms such as fever and headache[4]. Mental and behavioral abnormalities, cognitive impairment, decreased memory of recent events, seizures, decreased level of consciousness, and coma may follow. Involuntary movements are also common, including orofacial involuntary movements, limb tremors, dance-like movements and even opisthotonos [5]. In addition, the disease can also cause sleep disturbances, limb paralysis, and visual impairment. The clinical manifestations of this patient were basically consistent with the above symptoms, in which involuntary movements appeared after 2 months of treatment. In terms of diagnosis, first of all, a comprehensive analysis of the patient's clinical manifestations, cerebrospinal fluid examination, clinical imaging, EEG and other examinations should be carried out to determine if there are problems with AE, and antibody testing should be carried out. Among them, the number of leukocytes in cerebrospinal fluid should be increased, higher than $5*10^6$/L, or the oligoclonal zone of cerebrospinal fluid should be positive [6]. The above two indicators, the patient has. Second, neuroimaging shows abnormal T2 or FLAIR signal in the limbic system on MRI, and EEG shows focal epilepsy or epileptiform discharges or diffuse multifocal slow-wave rhythms [6]. Contrary to the literature, the patient had no positive findings on imaging. In terms of treatment, the AE Chinese expert consensus recommends three regimens, namely first-line immunization regimen, second-line immunization regimen, and long-term immunotherapy regimen. First-line immunotherapy regimens include glucocorticoids, intravenous immunoglobulin (IVIg), and plasma exchange. Second-line immune drugs, including rituximab and intravenous cyclophosphamide, are mainly used for patients with poor first-line treatment. Long-term immunotherapy, including mycophenolate mofetil and azathioprine, is mainly used for recurrent cases and patients with poor first-line immunotherapy results. Unfortunately, our initial focus was on MOG antibodies, and there was little consideration for AEs. Anti-myelin oligodendrocyte glycoprotein immunoglobulinG antibody

(anti-myelinoligodendrocyteglycoprotein-IgG,MOG-IgG)-related diseases (MOG-IgG Gassociated Dissor-ders, MOGAD) is a kind of immune-mediated central nervous system proposed in recent years. System (centralnervoussystem, CNS) inflammatory demyelinating disease. Current research suggests that MOG-IgG may be the pathogenic antibody of MOGAD, which is an independent disease spectrum different from multiple sclerosis (MS) and neuromyelitis optica spectrum disease (NMOSD). MOGAD can be monophasic or recurrent course, the main symptoms include optic neuritis (optic neuritis, ON), meningoencephalitis, brain stem encephalitis and myelitis. Glucocorticoids (hereinafter referred to as "hormones") are effective in the treatment of MOGAD, but patients often experience hormone dependence and recurrent attacks. We mainly considered its meningoencephalitis classification. Because in addition to focal localized symptoms in the brain, disturbance of consciousness, cognitive impairment, behavioral changes or seizures are common brain symptoms of MOGAD, which can be accompanied by symptoms of meningitis [7]. Domestic research results show that the proportion of epilepsy in MOGAD ranges from 0.3% to 24.0%. 12% of MOGAD patients have different degrees of meningeal involvement, including headache, nausea, vomiting, and meningeal irritation [8]. MOGAD with meningitis manifestations is often associated with increased intracranial pressure, cerebrospinal fluid (CSF) leukocytes can exceed 100×106/L, and the level of total CSF protein increases. The EEG of MOGAD patients with meningoencephalitis may have slow wave manifestations [8]. This is also consistent with patient performance. In terms of auxiliary examinations, its diagnostic requirements are roughly the same as those of AE. Among them, the literature recommends conventional MRI plain scan plus enhanced scan. Plain scan includes cross-sectional T1/T2/FLAIR and DWI image scanning, and enhanced scan includes cross-sectional and sagittal T1 image. In addition, we also checked the fundus of the patient to rule out visual impairment and disprove the evidence of meningoencephalitis-type MOG antibody disease. In terms of treatment, the main drugs and therapies in the acute phase include hormones, high-dose intravenous immunoglobulin (intravenousimmunoglobulin, IVIg) and plasma exchange (plasmaexchange, PE). Treatment in remission: MOGAD patients who have relapsed should be treated with remission to prevent relapse, and whether long-term immunomodulatory therapy is needed for MOGAD patients with initial onset remains to be seen. and a comprehensive assessment of the duration of positivity. Different immunologic drugs, including low-dose corticosteroids, azathioprine, mycophenolate mofetil, rituximab, and methotrexate, may reduce the risk of recurrence in patients with MOGAD.

Finally, the patient was treated according to the MOGAD remission treatment plan, and there was a significant rebound, with elevated body temperature, continuous increase in cerebrospinal fluid pressure, rapid rebound in leukocytes in cerebrospinal fluid, and an increase in the MOG antibody titer in antibody detection. After thorough discussion, we applied for the remote consultation of AE treatment experts again to determine the AE treatment plan for the patient. The patient's various indicators improved significantly. Although the patient has MOG antibody positive after treatment, it is not premature to consider MOG antibody-related diseases. It should be treated according to AE, and related diseases should be considered after the AE treatment is not good.

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