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Research Article

Acute Guillain Barre Syndrome in Hemodialysis Patients with Diabetic Nephropathy: A Case Report

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Abstract: Acute Guillain Barre syndrome is a common type of autoimmune mediated acute peripheral neuropathy. Its initial symptoms are symmetrical limb weakness, sensory disturbance, pain or other symptoms. This paper reports a case of acute Guillain Barre syndrome in a uremic patient with diabetic nephropathy and long-term regular hemodialysis, in order to further explore the clinical manifestations and differential characteristics of uremic patients with acute Guillain Barre syndrome, improve the early diagnosis rate of uremic patients with acute Guillain Barre syndrome, make the patients get timely treatment, so as to reduce the disability of such patients To improve the prognosis of the disease.

Key words: Acute Guillain-Barre syndrome; Uremia; Diabetic peripheral neuropathy

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1 Case data

A 51 years old male patient with a history of type 2 diabetes mellitus for 9 years and hypertension for 1 year had poor control of blood glucose and blood pressure. The varicose veins of the right lower extremity were operated on 3 years ago. Five years ago, the patient's physical examination found that the urine test was abnormal, and the urine routine examination was positive for urine protein. He was diagnosed as diabetic nephropathy, and was treated regularly, but not regularly reviewed. More than one year ago, his renal function test showed that creatinine was about 150 umol/L, and he took Chinese medicine treatment. 11 months ago, his renal function test showed that creatinine increased significantly, rising to about 500 umol/L, so he took internal venous fistula plasty, and suggested hemodialysis vascular access. After the internal fistula was completed, his renal function test showed that creatinine increased to about 900umol / L again, so he started hemodialysis treatment, in the local hospital three times a week, the condition is still stable, but no hemoperfusion or hemodiafiltration treatment. More than 20 days before admission, the patient had no obvious cause of lower limb pain, followed by numbness of both feet and toes, resulting in weakness of both lower limbs and difficulty in walking and standing. In the local hospital, the brain MRI showed multiple lacunar cerebral infarction, and nutritional nerve therapy was given. The patient's condition continued to worsen and developed to numbness and weakness of limbs. He came to our hospital for further diagnosis and treatment. Physical examination on admission: T 36.6 ℃ , P 76 times / min, R 20 times / min, BP 162/106 mmHg. There was no edema in both eyelids, no pallor in eyelid conjunctiva, equal size and equal circle in both pupils, about 3mm in diameter, thick respiratory sounds in both lungs, no dry or wet rales, heart rate of 76 beats / min, arrhythmia, no pathological murmur in auscultation area of heart valves. The abdomen was flat and soft without tenderness, rebound pain and muscle tension. The liver and spleen were not touched under the ribs. The bowel sounds were normal. The scar of arteriovenous fistula operation could be seen in the left forearm. The vascular

tremor could be touched above the scar. The vascular murmur could be heard by auscultation. There was no edema in both lower limbs. The cranial nerves were normal, the muscle strength of both upper limbs was grade 4, the muscle strength of both lower limbs was grade 3, the muscle tension of the limbs was normal, the tendon reflex of the limbs was not elicited, the pain of the distal segment, sole and toe of both upper limbs was decreased, the Chaddock sign was negative on both sides, the Kernig sign was negative, and the Babinski sign was negative on both sides. Auxiliary examination after admission: Routine blood test:The red blood cell count was 4.08 * 10^{12}/L, hemoglobin was 125g /L, ESR was 17mm /h. Biochemical routine test: Urea 28.56mmol/l, creatinine 1004umol/ L, Cystatin C 7.96mg/l, blood glucose 6.12mmol/l. Immunization: SSA antibody (±), antinuclear antibody 1:100 positive, karyotype (nuclear granular type, cytoplasmic granular type).Serological examination: Vitamin B12 > 2000.0 pg /ml, troponin 60.140 ng/ L, myoglobin 470.400 ng / ml, parathyroid hormone 228.80 pg/ml, N-terminal pro-B-type natriuretic peptide 1626 pg/ml, procalcitonin 1.010 ng / ml, 25 hydroxyvitamin D 28.45 ng / ml. ECG: Sinus rhythm, abnormal T wave. Electromyography: multiple severe peripheral nerve injuries. The results of autoimmune peripheral neuropathy series antibody test showed that anti-GD1a antibody IgM (+), anti-GD1b antibody IgM (++), anti-GD2 antibody IgM (+), anti-Sulfatide antibody IgG (+), IgM (+). Six serum test results of autoimmune encephalitis-related antibodies were not abnormal. The cerebrospinal fluid examination was not completed because the patient and his family refused to perform a lumbar puncture biopsy. After consultation in the Department of Neurology, the patient was considered as acute Guillain Barre syndrome, and was given neuronutrition, hemodialysis, hemoperfusion, and hemodiafiltration treatment. He was transferred to the Department of neurology to receive glucocorticoid pulse therapy and intravenous injection of human immunoglobulin. At the same time, he was given physical therapy such as Electroacupuncture stimulation and physical factors. After treatment, the condition did not worsen significantly, and the condition stabilized and the patient left hospital.

2 Discussion

Guillain Barre syndrome, also known as inflammatory demyelinating polyneuropathy, was first discovered by two biologists guillan and Barre in 1916. It is characterized by acute onset, disappearance of tendon reflex, quadriplegia and cerebrospinal fluid protein cell separation. It can also involve cranial nerve and autonomic nerve. In severe cases, respiratory muscle paralysis may occur and cause death. The pathological features were infiltration and destruction of inflammatory cells in nerve roots and peripheral nerves. According to the location and form of nerve root injury, acute Guillain Barre syndrome is divided into three subtypes, namely acute inflammatory demyelinating polyneuropathy, acute motor axonal neuritis and Miller Fisher syndrome[2]. The typical clinical manifestation of acute Guillain Barre syndrome is progressive bilateral or relatively symmetrical limb weakness, mostly from the distal end, gradually involving the proximal end. Some patients have cranial nerve involvement, and can also have symptoms or signs of sensory ataxia and autonomic nerve involvement.

For the immunotherapy of acute Guillain Barre syndrome, plasma exchange, glucocorticoid and immunoglobulin are generally considered to be effective. In principle, the earlier the immunotherapy, the better. Plasma exchange can clear nonspecific antibodies, complement and inflammatory factors in patients. Immunoglobulin can neutralize antibodies and inhibit antibody and complement activation. In addition, early rehabilitation treatment can restore motor and sensory function to a certain extent, which has a positive effect on reducing the disability rate of patients with acute Guillain Barre syndrome.

The exact pathogenesis of Guillain Barre syndrome is still unclear, but most studies believe that its pathogenesis is related to autoimmune factors, and is mostly secondary to some precursor diseases[2]. It is generally believed that patients have a history of nonspecific infection before the onset of the disease, in which bacterial and viral infections are more common. Studies have found that Campylobacter jejuni infection is an important factor in inducing Chinese Guillain Barre syndrome[3]. In addition, cytomegalovirus, Epstein Barr virus, hepatitis B virus, HIV and Mycoplasma pneumoniae infection can be involved in inducing acute Guillain Barre syndrome[4]. Patients usually get sick after 1-2 weeks of infection, progress rapidly within 12 hours, and reach the peak within 2-4 weeks. For Guillain Barre syndrome, the

diagnostic examination is cerebrospinal fluid protein cell separation phenomenon[5]. In this case, it is a pity that the cerebrospinal fluid examination can not be improved due to the refusal of the patient and his family members to carry out lumbar puncture biopsy. However, the serum ganglioside antibody found in the patient has a key role in the diagnosis of Guillain Barre syndrome, and it suggests that it may be acute Motor axonal neuropathy. For this patient, the basic diseases were type 2 diabetes mellitus and diabetic nephropathy, and regular hemodialysis treatment had been performed. In the diagnosis and treatment of uremia, peripheral neuropathy was easily misdiagnosed as peripheral neuropathy.

Diabetes is often complicated with a variety of chronic complications, diabetic nephropathy, diabetic peripheral neuropathy and diabetic retinopathy are known as "diabetic triad"[6]. The most common type of diabetic peripheral neuropathy is distal symmetric polyneuropathy, with the involvement of sensory and motor nerves in the distal part of hands and feet. The other common type is focal mononeuropathy, which can involve the brain nerve or spinal nerve, but the oculomotor nerve, median nerve and popliteal nerve are the most common. In addition, there are asymmetric multiple focal neuropathy and multiple radiculopathy. In this case, the clinical manifestation of the patient is similar to that of diabetic peripheral neuropathy, and the history of diabetes is long, which is easy to cover up the condition of Guillain Barre syndrome and cause misdiagnosis.

The symptoms of uremia are various, and complications of multiple organs and systems can occur. Long term hemodialysis patients, combined with nervous system damage is also more common. Uremic nervous system damage can involve the central nervous system, peripheral nervous system and autonomic nervous system, with a variety of clinical manifestations, and it is also difficult to distinguish from primary nervous system diseases. More than 80% of patients with long-term hemodialysis have neuropathy, and the degree of neuropathy has nothing to do with dialysis age and gender[7]. It is generally believed that uremic neuropathy is associated with excessive accumulation of neurotoxins, especially with middle molecular toxins with relative molecular weight of 500-5000 daltons[8]. The most common clinical manifestation of paresthesia is hypoesthesia or hypoesthesia. With the development of the disease, autonomic nerve damage often occurs. However, the general hemodialysis treatment has no obvious improvement on uremic nerve damage[9]. Compared with the general hemodialysis, the blood purification method with better clearance effect on large and medium molecular toxins can significantly improve uremic nerve damage.

The early clinical manifestations of acute Guillain Barre syndrome are various. With the increase of basic diseases, the misdiagnosis of Guillain Barre syndrome is caused. Improving the clinical experience of clinicians is helpful to reduce the misdiagnosis rate of the disease. At present, the diagnosis and treatment of Guillain Barre syndrome is still a relatively difficult challenge for doctors and patients. Early diagnosis and timely treatment have a positive impact on the prognosis of Guillain Barre syndrome. In the process of clinical diagnosis and treatment, for patients with a variety of basic diseases that can cause peripheral nerve damage at the same time, when limb weakness, sensory disorders and other symptoms suddenly occur, we need to be alert whether there is Guillain Barre syndrome, so as not to delay the treatment and affect the prognosis.

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