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Case Report

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THE TOGETHERNESS OF GUILLAINE-BARRE SYNDROME AND DIABETICAUTONOMIC NEUROPATHY; CASE REPORT; AND REVIEW OF THE LITERATURE

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ABSTRACT

Guillain-Barre Syndrome is an inflammatory immune peripheral neuropathy disease. The incidence of Guillain-Barre Syndrome in patients who are followed-up with diabetic ketoacidosishas increased in recent years. In this paper, Guillain-Barre Syndrome developed in a 31-year-old male patient who was followed with diabetic autonomic neuropathy. The Guillain-Barre Syndromediagnosis neuron message studywas based on cerebrospinal fluid and clinical findings. As a conclusion, the probability of the incidence of Guillain-Barre Syndrome in diabetic patients with bad glycemic control being a coincidence must be ignored. In our opinion, we consider that bad glycemic control triggered the development of Guillain-Barre Syndrome.

KEYWORDS: Guillain-Barre Syndrome, diabetes, neuropathy.

INTRODUCTION

Neuropathy in diabetic patients may present with autonomic, sensory and motor symptoms. If the patients have atypical neurological symptoms and especially motor neuropathy clinic, non-diabetic neuropathies must be considered in such patients even if they are diabetic patients.[1] Among these, Guillaine-Barre Syndromemust be handled in the first place as the differential diagnosis. In this case report, we are presenting a patient with Type-1 Diabetes Mellitus diagnosed with diabeticautonomic neuropathy, who developed Guillaine-Barre syndrome. We would like to remind this rare case.

The Case

31-year-old male case was being followed-up with Diabetes Mellitus Type-1 for 11 years, and with diabetic Autonomic neuropathy for the last 2 years. He applied to our clinic with the increased complaints like weakness in hands and feet, which developed for the last 1 week after influenza, not being able to move his feet and hands, diarrhea and urinary incontinence. In the physical examination, the blood pressure values were arterial 90/60 mmHg, pulse 110 beat/min, temperature 37°C. He had symmetrical paralysis in bilateral alt extremity, loss of strength, and areflexia.Respiratory distress developed in the patient during our follow-ups. The laboratory findings were as follows; glucose: 252 mg/dL, HbA1c: %10, Na:142 mEq/L, K: 4.8 mmol/L, creatine: 1.1 mg/dL, AST: 14 IU/L, CK: 60 U/L;urine test: ketone (++).In the electromyography, decrease in the motor unit action potential,reduced speed in the nerve conduction, and acute motor and sensory axonal neuropathy was detected. In the spinal fluid obtained with lumbar puncture; mononuclear cell: 11/mm^3, protein: 80 mg/dL, glucose: 72 mg/dL. The cervicalandbrainMagnetic Resonance Imaging (MRI) were determined as being within normal limits. The patient was consulted with the Neurology Clinic. Guillaine-Barre syndrome was considered about the patient in thelight of these findings. Intravenousimmunoglobulin was given to the patient in the treatment. The clinical picture of the patient started to recover on the 6-10th day of the treatment. He started to walk without aid in 3 months.

DISCUSSION

Diabetic neuropathy is characterized with sensory and motor symptoms. However, even in motor neuron involvement, paralysis and extremity weakness are not frequent.[2] Guillain-Barre Syndrome(GBS), on the other hand, is the acute andinflammatory-immune disease of the peripheralnerves and nerve roots. GBS usually proceeds with progressive flask paralysis and reduced deep tendon reflex. It is an acquired acute poly-radiculo-neuropathycharacterized with widespread sensory, motor

and autonomic symptoms.[3] Our case was characterized with autonomic neuropathyandacute motor neuropathy symptoms.

Cerebrovascular event in cases presented with peripheralneuropathicclinic, peripheralvascularinsufficiency, spinal diseases, periodicparalysis, myasthenia gravis, transvers myelitis, toxicneuropathy, diabeticperipheralneuropathyand GBS must be considered in differential diagnosis.[4] Our case had received diabeticautonomousneuropathydiagnosis 2 years ago. He arrived at our clinic withacuteperipheralneuropathy symptoms. The patient received GBS diagnosis by eliminating clinical and laboratory findings and otherperipheralneuropathiccauses.

There are no reports on diabetic patients having high risk for GBS. However, very few GBS cases, which were related with diabeticketoacidosis were reported.[5] Bad glycemic control and ketone positivity in urine were detected in our case. In the light of previous case reports, we can suggest that bad glycemic control might trigger GBS.

GBS is usually a disease with good prognosis, and the majority of the patients recover with full recovery or with slight motor losses. It was reported in previous case reports that 62-92% of the patients showed full functional recovery.[6] Following and supporting the vital findings in early symptomatic cases is the basis of the treatment. In previous studies, plasma change and intravenousimmunoglobulinare the most effective treatment methods.[1] As the treatment in our patient, we administered a 5-day-long 0.4 gr/kg/day intravenousimmunoglobulin. Our case started to walk without aid after 3 months, which is consistent with the literature findings. Nearly full functional recovery was observed in later follow-ups.

As a conclusion, diabetic neuropathyand GBS are very rarely determined together. The togetherness with diabeticketoacidosis was detected in previous studies. We consider that bad glycemiccontrolmight be a risk factor for GBS.

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