Journal of Biosciences and Medicines, 2023, 11, 55-64
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Diagnosis and Treatment of Diabetic Ketoacidosis Mellitus with *Guillain-Barré* Syndrome: A Case Report

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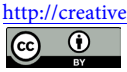
How to cite this paper: Zhong, J.W., Huang, L.F. and Xue, J.L. (2023) Diagnosis and Treatment of Diabetic Ketoacidosis Mellitus with *Guillain-Barré* Syndrome: A Case Report. *Journal of Biosciences and Medicines*, **11**, 55-64.
https://doi.org/10.4236/jbm.2023.1111006

Received: October 8, 2023
Accepted: November 5, 2023
Published: November 8, 2023



Abstract

This article reports the diagnosis and treatment of a case of *Diabetic ketoacidosis* with *Guillain-Barré syndrome*. *Diabetic ketoacidosis* (DKA) is the most common acute diabetes mellitus, often diabetes and infection, insulin withdrawal or interruption of the history of triggers, with hyperglycemia, ketoacidosis, and acid poisoning as the main symptoms, rapid onset of illness, and serious illness. *Guillain-Barré syndrome* (GBS) is an autoimmune-mediated peripheral neuropathy with frequent respiratory or gastrointestinal tract infections and low clinical incidence before 1 - 3 weeks. This case was characterized by a headache with vomiting acute onset, a relatively clear diagnosis of *diabetic ketoacidosis*, a symmetrical decrease in muscle strength in the extremities, and recovery of consciousness after aggressive correction of environmental disturbance in electrolytes, but very poor muscle strength in the extremities, protein-cell segregation in cerebrospinal fluid (CSF). Considering *Guillain-Barré syndrome*, the patient's muscle strength gradually recovered after treatment with *proglobulin* shock. At present, the pathogenesis of the two is not clear, but because of its rapid progress, clinicians should raise awareness of *diabetic ketoacidosis* combined with *Guillain-Barré syndrome*, early diagnosis, and active treatment. Inform consent has been obtained from the patient for this report.

Keywords

Diabetic Ketoacidosis, *Guillain-Barré* Syndrome, Case Report

1. Introduction

Diabetic ketoacidosis (DKA) is a common acute complication of diabetes melli-

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tus [1], which can be manifested as anorexia, nausea and vomiting, peripheral weakness, and other symptoms, and is often accompanied by neurological symptoms. Common neurological have varying degrees of consciousness disorder, unresponsiveness, and even coma. *Guillain-Barré syndrome* (GBS) is generally recognized as an autoimmune-related peripheral neuropathy caused by infection [2]. Clinical manifestations are characterized by progressive limb weakness and motor neuron disease. Clinical manifestations include a monophasic course, limb weakness, cerebrospinal fluid showing protein-cell separation, and neural electrophysiologic demyelination of peripheral nerves. The concomitant occurrence of GBS in DKA is relatively rare, and the incidence of the disease is low in the clinic. Due to the complexity of the pathogenetic mechanism, which has not yet been fully elucidated, the clinician usually pays close attention to the patient's glucose and ketone levels, as well as to the cardiovascular, pulmonary, and cerebral levels, and the infections and other complications. DKA patients are often combined with disorders of consciousness, muscle strength examination is more difficult, and the lack of understanding of concomitant *Guillain-Barré syndrome* can easily lead to missed diagnosis. The clinical data of one patient with DKA complicated by *Guillain-Barré syndrome*, which was diagnosed in our hospital in January 2023, are reported as follows, combined with relevant literature, to deepen primary care doctors' understanding of the disease and improve the diagnosis and treatment level of primary care hospitals.

2. Case Information

A 32-year-old female was admitted to the hospital on January 6, 2023, with headache vomiting for 10 days, and impaired consciousness for 5 days. On December 28, 2022, the patient had a headache with a fever (37.8˚C) and poor appetite without any obvious cause and took oral flu medicine on herself, but her symptoms were not relieved. she went to the local clinic for treatment on December 30 (details unknown), and her fever improved, but she continued to have headaches, thirst (drinking water and vomiting, and the amount of the contents was not known), heartburn, and poor appetite, and went to a hospital for a CT scan of her chest, which showed slight lung changes, and she did not undergo any special treatment. December 31 still local clinic to continue treatment, delirium, and dyspnea in the evening to the outside hospital, the specific diagnosis and treatment process is not known. On January 2, 2023, the doctor checked the patient for the *Novel Coronavirus* nucleic acid test which showed positive, so her diagnosis was considered to be *viral pneumonia* (*severe*). Then the patient is transferred to our hospital to continue treatment. Since the onset of the disease, the patient was in fair spirits, had a poor diet, uncharacteristic urination and defecation, decreased physical strength, and no significant change in weight. She had a history of *type 2 diabetes mellitus* (T2DM) for more than 5 years and was taking *dagliflozin* 10 mg/times once a day and *repaglinide* 1.0 mg/times once in the morning and once in the evening, and her blood glucose fluctuated between 7.0 - 9.0 mmol/L at random on her fingers. She was hospita-

lized in June 2021 for "*ketoacidosis*". Denies history of coronary artery disease, hypertension, hepatitis, tuberculosis, schistosomiasis, and drug and food allergies. She has received 3 doses of the *Novel Coronavirus* vaccine. Physical examination: T 37.8˚C, P 130 times/min, R 20 times/min, BP 127/75 mmHg (*norepinephrine* maintenance), height 168 cm, weight 60 kg, BMI 21.26 kg/m^2, lethargy, physical examination does not cooperate, the whole body of the skin and sclera did not see yellow staining, no enlargement of superficial lymph nodes, the lungs respiratory sounds clear, did not hear obvious dry and wet rales, the cardiac rhythm is regular, did not hear obvious pathological murmurs, the abdomen is flat, not heard a pathological murmur. There was no obvious pathological murmur, the abdomen was flat and soft, the liver and spleen were not touched under the rib margins, there was no edema in both lower limbs, and pathological reflexes were not elicited. After admission, she completed the following: Blood routine: white blood cell count 17.52 × 10^9/L, *neutrophilic granulocyte percentage* 84.50%, *hemoglobin* 104.0 g/L. *Urine routine*: glucose 4+, *urinary ketone bodies* 2+, *bilirubin* negative, occult blood 1+, microscopic *erythrocytes* 8.0/μL; Liver function: *direct bilirubin* 10.0 μmol/L, *total bilirubin* 21.5 μmol/L, total protein 54.1 g/L, albumin 33.1 g/L. *Renal function*: urea 14.54 mmol/L, *creatinine* 81.6 μmol/L. *Coagulation function*: *prothrombin time* 13.70 seconds. *Plasma D-dimer* (turbidimetric method) 997.00 ng/ml; *DIC* complete set: *prothrombin time* 13.50 sec, *prothrombin time* 1.28, *plasminogen activity* 76.00%, *antithrombin activity* 87.00%, *plasma fibrin* (pro) *degradation product* 5.89 μg/ml. Sepsis test: *calcitonin gene* 0.7 ng/ml, *C-reactive protein* 89.2 mg/L, *interleukin-6* 7.23 pg/mL, *amyloid A* 183.4 mg/L; no obvious abnormalities were seen in the blood transfusion complete set, BNP, and cardiac enzyme profile. *Novel Coronavirus* IgG antibody was positive. *Novel Coronavirus* nucleic acid (COVID-19) was positive; *ECG* showed: sinus tachycardia—right deviation of electrical axis—T wave abnormality. Bedside cardiac ultrasound showed left heart hypoplasia with EF 43%. A bedside chest radiograph showed a possible infection in the lower lungs. Lower extremity deep vein ultrasound and renal ultrasound showed no obvious abnormality. *Cerebrospinal fluid* biochemistry: sugar 6.75 mmol/L, chloride 140.1 mmol/L, cerebrospinal fluid protein 135.9 mg/dl; *Cerebrospinal fluid* routine: colorless, transparency, transparency, coagulability, no clots, Penn's test 1+, total cell count 7 × 10^6/L, leukocyte count 1 × 10^6/L ; cytology of *cerebrospinal fluid*: light microscopy, centrifugal smear, a few red blood cells and nucleated cells, including lymphocytes (like cells), which *lymphocytes* (like) cells accounted for 1/5, *monocytes* accounted for 2/5, neutrophils accounted for 2/5, no other special . She was admitted to the hospital with the diagnosis of *type 2 diabetic ketoacidosis* and *Novel Coronavirus* infection.

3. Diagnostic Process

Upon admission, the patient was reported to be in critical condition, fasting, intensive care, monitoring blood pressure, respiration, oxygen saturation, and completing relevant auxiliary examinations. Immediately, arterial blood gases

were checked: PH 7.10, potassium 6.5 mmol/L, GLU 29.8 mmol/l. Combined with the patient's history of *type 2 diabetes mellitus*, the patient was considered to be in *diabetic ketoacidosis* and was admitted to the hospital with a poor mental state, and was immediately placed on endotracheal intubation with respiratory assisted ventilation, while rehydration (glucose-salt injection), anti-infection, maintaining blood pressure, and maintaining the stability of the internal environment were all handled. After 1 hour, arterial blood gas was rechecked: PH 7.34, PO_2 153 mmHg, PCO_2 26 mmHg, GLU 22.1 mmol, potassium 2.6 mmol/l, sodium 150 mmol/l. Potassium supplementation treatment was started immediately, and arterial blood gas was rechecked 2 hours later: PH 7.35, GLU 26 mmol/l, potassium 2.6 mmol/l, sodium 150 mmol/l, potassium 2.6 mmol/l, sodium 2.6 mmol/l, potassium 2.6 mmol/l, sodium 150 mmol/l. 7.35, GLU 26.5 mmol/l, potassium 4.0 mmol/l, sodium 157 mmol/l, subcutaneous insulin 12 U was given. On the second day of admission, the patient's finger blood glucose was fluctuating between 15.0 - 23.5 mmol/l, sodium 161 mmol/l, potassium 3.3 mmol/l, and she was treated with sodium restriction, nasal feeding of 1000 ml of lukewarm water (70 ml/h), potassium supplementation, and intermittent potassium supplementation.), potassium supplementation, intermittent subcutaneous injection of insulin 6 U - 10 U. On the 3rd day of admission, the patient was comatose and developed high fever, with the highest temperature reaching 41˚C, finger blood glucose fluctuating at 11.3 - 20.2 mmol/l, leukocyte count of 12.51×10^9/L, neutrophil % 82.70%, potassium 4.21 mmol/l, sodium 173.0 mmol/L, *calcitoninogen*, *C-reactive protein*, *interleukin-6* were all elevated. Infectious fever, urgent examination of adult bilateral blood cultures, please infectious disease consultation, and actively reduce fever, fluid replacement, infectious disease consultation recommended the use of *bisopenem* anti-infection, in addition, the patient's blood sodium progressive rise, the bedside hemofiltration to reduce sodium, the lowering of glucose regimen will be adjusted to the subcutaneous insulin pump continuous insulin, initially 4 - 6 u/h. Night hemofiltration process hemofiltration interface alarm prompts the extreme negative value of the input, stops the bedside hemofiltration, rechecks the blood sodium, and then stops the hemofiltration. Paracentral hemofiltration was performed, blood sodium was rechecked and decreased to 150 mmol/l level, and CRRT therapy was suspended. On the fifth day of admission, the patient was lethargic, with intermittent fever, the highest temperature of 39˚C, found poor muscle strength in the limbs, but the examination did not cooperate with the inability to check the muscle strength, finger blood glucose fluctuations in 9.6 - 17.0 mmol/l, review of potassium 4.17 mmol/L, sodium 150.5 mmol/L, anti-influenza virus B IgM antibody weak positive, adjust the speed of the subcutaneous insulin pump to 2 - 4 u/h, and the neurology consultation to find a solution to the problem. A neurology consultation was also requested to find the cause of the patient's lethargy. On the 6th day of admission, the patient's mental state was better than before, and she could respond to instructions, but the muscle strength of the limbs was significantly reduced, and the neurologist suggested improving the cranial DWI ex-

amination. On the 8th day of admission, the patient had no change in his mental state compared to the previous day, still had a low-grade fever, poor tolerance to de-ventilator, and the head MRI + DWI results returned: symmetrical abnormal signal in bilateral occipital lobe area, cortex of bilateral frontal lobe area, and hemi-ovoid centers, which was considered to be an ischemic-hypoxic encephalopathy? Infectious lesion? Other? Complete lumbar puncture and electromyography. On the 9th day of admission, the patient was clear, the muscle strength of the limbs was very poor (symmetrical decrease in muscle strength of the limbs), the blood glucose of the fingers fluctuated at 7.0 - 13.1 mmol/l, the speed of the subcutaneous insulin pump was adjusted to 1 - 3 u/h, the blood test was restored to normal, and the sodium was still at the level of 150 mmol/L, the results of the blood cultures were returned negative, and the results of the cerebrospinal fluid were returned with the phenomenon of protein-cell separation, and *electromyography* showed that peripheral nerve injury in extremities (lower extremity more obvious than upper extremity), and the diagnosis of *Guillain-Barré syndrome* was considered. On the 10th day of admission, the patient's consciousness was clear, the muscle strength of the limbs was grade 2, the blood sodium, blood potassium, and blood chloride did not show any obvious abnormality, the urine glucose was 4+, the urine ketone body was micro, and the sepsis test did not show any abnormality, and the patient was started to undergo the shock treatment of *immunoglobulin* 20 g per day. After 5 days of continuous shock therapy, the patient's muscle strength gradually recovered and body temperature improved. During this period, finger blood glucose fluctuated at 7.7 - 14.2 mmol/l, and the speed of the subcutaneous insulin pump was 2 - 4 u/h. In the third week of admission, the patient's sputum culture detected *Aspergillus flavus* and *Aspergillus fumigatus*, alveolar lavage was performed, and the culture fluid detected *Aspergillus flavus*, *Aspergillus fumigatus*, and *Acinetobacter baumanii*, which was considered to be a fungal infection of lungs, and the patient was placed in bedside isolation, and the patient was given nebulizer inhalation and vibratory lung excretion of sputum, and then, after consulting with the department of infectious diseases, that began to be given anti-infective treatments (*voriconazole* and *liposomal amphotericin B*) was started. During this period, the insulin pump speed was maintained at 2 - 4 u/h, and blood glucose and electrolyte control were monitored. In the 5th week of admission, the patient's consciousness was clear, the muscle strength of the limbs was grade 3, the proximal muscle strength was poor, the blood glucose of the fingers fluctuated at 10.0 - 13.8 mmol/l, the antibody spectrum of autoimmune encephalitis was negative, the patient's respiratory muscle strength had not been fully recovered, and could not be completely taken off the ventilator, so the patient was again given *gamma globulin* shock treatment for 5 days, and the subcutaneous insulin pump was still maintained at 2 - 4 u/h. In the 7th week of admission, the patient's respiratory condition improved, and the patient tolerated it after a trial of off the ventilator. On the 7th week of admission, the patient's respiratory condition improved, and the patient tolerated the trial off the ventilator. Considering that the patient's in-

ternal environment had been corrected, and the muscle strength of the limbs had gradually recovered to grade 4, the patient could be continuously taken off the ventilator. During this period, sputum culture was reviewed several times, suggesting *Acinetobacter baumaii*, which was considered to be colonizing bacteria, and no special treatment was performed. In the 8th week of hospitalization, the *Novel Coronavirus* nucleic acid test was negative, and the infection foci were obviously absorbed in chest CT, and the patient was gradually transitioned to diet, and the glucose-lowering regimen was adjusted to 12 U of insulin to be injected subcutaneously before three meals, and 30 U of diethyl insulin to be injected subcutaneously before bedtime, and the patient was monitored to have good glycemic control, and his limb muscular strength was restored to grade 4+, and she was transferred to the lower hospital to continue the follow-up rehabilitation treatment. After 6 months, the patient's fasting blood glucose was controlled at 6.8 - 8.5 mmol/l, 2-hour postprandial blood glucose was at 8.0 - 12.1 mmol/l, and limb muscle strength was restored to grade 5.

4. Discussion

Diabetic ketoacidosis (DKA) is one of the most serious acute complications of diabetes mellitus, which in severe cases may lead to death or leave sequelae such as neurological deficits, and therefore the disease must be highly vigilant and closely observed. The patient had a history of type 2 diabetes mellitus and the diagnosis of *diabetic ketoacidosis* was established on admission with blood gas analysis showing acidosis, positive urine ketone bodies, and arterial blood gas GLU 29.8 mmol/l. During the disease, there was a change in mental status and symmetrical weakness of the proximal extremities. After correction of *diabetic ketoacidosis* and ionic disorders, mental status improved, but the muscle strength of the extremities was extremely poor, electromyography suggested that the peripheral nerves of the extremities were impaired (motor and sensory fibers were involved, and axonal damage was more pronounced in the lower extremities than the upper extremities), and the cerebrospinal fluid showed a protein-cell separation. This was consistent with the diagnosis of GBS [3].

The cause of *diabetic ketoacidosis* complicating *Guillain-Barré syndrome* is unknown, and it has been previously documented that about two-thirds of patients with GBS have a history of respiratory or gastrointestinal infections within 1 - 3 weeks, most often associated with *Campylobacter jejuni*, *E-B virus*, *Mycoplasma,* and *cytomegalovirus infections* [4], and there are also reports of *rabies virus*, *human papillomavirus*, *influenza A virus*, [5], *seasonal influenza vaccine* [6] and postoperative patients [7] [8] [9] have been reported about the triggering of GBS. In recent years, GBS has been successively reported to be induced by infection with *novel coronaviruses* [10] [11] [12]. *Novel coronavirus* infection (COVID-19) has also been reported as a predisposing factor for DKA in diabetic patients [13] [14] [15], but it is more common in patients with *type 1 diabetes mellitus*. There are three main theories about the mechanism of correlation between DKA and GBS: 1) GBS is considered to be an autoimmune-associated

disease, and DKA is most commonly seen in patients with *type 1 diabetes mellitus*, who often have insulin-associated antibodies, and therefore may be caused by some combined immune mechanisms that produce an immune response to certain components of peripheral nerves, resulting in demyelination [16]; 2) Some diabetic patients who are negative for diabetes-associated antibodies have DKA [17]; and 3) Some diabetic patients who are negative for diabetes-associated antibodies have DKA [18]. This case is a patient with type 2 diabetes mellitus, who had nausea and vomiting at the onset of the disease, with DKA manifestations, and was found to be positive for the nucleic acid of *novel coronaviruses* after a complete examination, and there were no other obvious triggers of DKA before the onset of the disease. So, it was hypothesized that DKA might be induced by COVID-19, and the diagnosis of GBS was gradually clarified in the course of the treatment, but it was not clear whether GBS was already present at the time of the onset of the disease. Therefore, the mechanism of the occurrence of the present case was hypothesized, and the mechanism might be Therefore, it is hypothesized that the mechanism of this case may be that COVID-19 induced DKA, which in turn induced GBS, leading to nerve damage, or that a common mechanism triggered by COVID-19 induced a related immune response that induced both DKA and GBS and that the exact mechanism needs to be further investigated in a follow-up study.

In the case reports of DKA combined with GBS over the past few years [17] [18] [19], the patients were all females aged 25 - 64 years, all of whom presented with impaired consciousness at the onset of the disease, which improved and ketosis disappeared after treatment with insulin and correction of electrolyte-intra-environmental disturbances. However, there were cumulative peripheral [16] [17] [18] [19] and autonomic [17] manifestations of acidosis and correction of electrolyte internal environmental disturbances within about 3 - 14 days. It has been reported in the literature that patients with GBS treated with *immunoglobulin* shock therapy for 5 days or plasma exchanged improve within 8 weeks with a definitive efficacy, but 10% of patients will *re-exacerbate* and the situation improves with the *re-administration* of *immunoglobulin* [20] [21]. The present case is consistent with the above clinical and morbid characteristics, the patient developed symmetrical limb weakness in all four limbs within a week of DKA correction, and the diagnosis of GBS was gradually clarified in the course of treatment of DKA. Since there is no separate treatment plan for GBS patients with combined DKA, the patient was given *immunoglobulin* for 5 days and hormone shock therapy by the general treatment plan for GBS, but the efficacy was not exact. Although the correlation with fungal infection in the lungs was considered to be high, it could not be ruled out that it was due to the combination of DKA. Therefore, it is necessary to pay more attention to the prevention of infection in the course of treatment, and it is more favorable to evaluate whether there is a difference in the efficacy of *gammaglobulin* between GBS patients with and without combined DKA after the exclusion of the infection factor. *Gammaglobulin* shock therapy and rehabilitation training were given again

for 5 days, and after about 6 weeks, the patient's clinical symptoms improved, blood glucose control was stable, and limb muscle strength returned to normal, with a relatively fair prognosis. The long treatment time in this case was mainly due to the slow recovery of the patient's respiratory muscle strength, and after the gradual recovery of limb muscle strength, it was still difficult to get off the ventilator. It was considered that the patient had a combination of fungal infection in the lungs and multidrug-resistant bacterial colonization, but patients with severe DKA may also suffer from multiorgan damage leading to the difficulty of getting off the ventilator.

When DKA is the onset of disease, there are mostly changes in consciousness, physical examination is not cooperative, it is difficult to examine the muscle strength of the limbs, and endocrinologists do not know much about GBS, so it is easy to miss the diagnosis of GBS when DKA is combined with GBS. Combining the characteristics of this case and the review of literature, the authors believe that in the clinical work: firstly, patients with a history of infection before the onset of DKA should be vigilant about the possibility of combining with GBS; secondly, in the course of the disease in DKA patients, attention should be paid to the prevention of infection, not only limited to bacterial infection, but also attention to viral infection and special pathogenic bacterial infection. Second, in the course of DKA patients, attention should be paid to the prevention of infection, not only limited to bacterial infection, but also attention to viral infections, fungal infections, and special pathogenic bacterial infections, DKA patients, once the complication of infection, will prolong the treatment time and increase the difficulty of the treatment; Third, in the daily checkup, in addition to pay attention to the patient's blood glucose, electrolytes, cardiac, renal damage, but also pay attention to the neurological checkups and changes, especially when the internal environmental disorders have been corrected, and the patient is gradually improved, but still the patient off the machine, and the patient has no change. After the gradual improvement, patients still have difficulty in getting off the machine or the muscle strength of the limbs does not see obvious recovery, we should actively carry out the relevant examinations to determine whether the GBS is combined or not, to avoid the development of the disease. In subsequent studies, it is necessary to further investigate whether there are clues for early recognition of GBS in the course of DKA, as well as clinical indicators suggesting prognosis.

Although the number of patients infected with the *Novel Coronavirus* is significantly lower than before, there are still a small number of people infected with the *Novel Coronavirus*. In the process of receiving and managing patients with mental changes, especially those with a history of diabetes, special attention should be paid to the possibility of neurological diseases in patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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