Fulminant Type 1 Diabetes Mellitus Complicated with a Life-threatening Electrolyte Abnormality and Abnormal Electrocardiogram Findings

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Abstract:
Fulminant type 1 diabetes mellitus (T1DM) is idiopathic T1DM with the rapid destruction of pancreatic β-cells. We herein report a 48-year-old man who developed fulminant T1DM complicated with a life-threatening electrolyte abnormality and abnormal electrocardiogram findings. He had no remarkable medical history, but one day, he developed general fatigue. His blood glucose level and HbA1c were 806 mg/dL and 6.3%, and his insulin secretion was markedly suppressed. He had ketoacidosis, hyponatremia and hyperkalemia. Furthermore, a life-threatening abnormality was noted on electrocardiogram. After fluid infusion and insulin therapy, the abnormality disappeared. In conclusion, we should bear in mind the possibility of fulminant T1DM in patients complaining of general malaise.

Key words: fulminant type 1 diabetes mellitus, diabetic ketoacidosis, hyperkalemia, a life-threatening electrocardiographic abnormality

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Introduction

Type 1 diabetes mellitus (T1DM) is classified as autoimmune type (type 1A) or idiopathic type (type 1B). Fulminant T1DM is a type 1B diabetic condition characterized by the acute onset of diabetic ketoacidosis soon after the development of typical diabetic symptoms and the markedly acute progression of insulin deficiency (1-4). Histologically, the infiltration of macrophages and T-cells into the pancreatic islets and the complete destruction of pancreatic β-cells are observed in the pancreas of subjects with fulminant T1DM. Therefore, we must be very careful not to make a misdiagnosis, which can lead to an unfavorable situation for the patient.

We herein report a 48-year-old man who developed fulminant T1DM complicated with a life-threatening electrolyte abnormality and abnormal electrocardiogram findings.

Case Report

We encountered a 48-year-old man who developed fulminant T1DM complicated with a life-threatening electrolyte abnormality and abnormal electrocardiogram findings. He had no remarkable medical history, and there were no abnormalities on a medical checkup. One day, he developed general malaise, nausea and vomiting, none of which are necessarily specific for hyperglycemia. Three days later, he found himself unable to walk due to severe general fatigue, and he was hospitalized.

On admission, he had severe dry mouth. His height and body weight were 169.1 cm and 62.8 kg, respectively, and his blood pressure and heart rate were 112/69 mmHg and 109 bpm, respectively. His body temperature was 37.1°C.

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Table. Laboratory Data on Admission in This Subject.

| Peripherial blood | Diabetes marker | Electrolyte |
|------------------|----------------|-------------|
| RBC 369x10^6/μL | Plasma glucose 806 mg/dL | Na 120 mEq/L |
| Hb 13.1 g/dL | HbA1c 6.3% | K 7.2 mEq/L |
| WBC 20,720/μL | GA 22.5% | Cl 99 mEq/L |
| Seg 85.0% | IRI <1.0 μU/mL | Ca 6.5 mg/dL |
| Stab 2.0% | Serum CPR 0.1 ng/mL | IP 3.9 mg/dL |
| Myelo 1.0% | Urinary CPR 0.6 μg/day | |
| Eos 0% | Ketone body 14,594 μmol/L | |
| Baso 0% | 3-OHAc 11,781 μmol/L | |
| Mono 6.0% | Glucagon test | |
| Lymph 5.0% | | |
| Platelet 29.6x10^4/mL | CPR 0 min 0.1 ng/mL | |
| | CPR 6 min 0.1 ng/mL | |
| | GAD ≤1.3 U/mL | |
| | ICA (-) | |
| | Insulin Antibody (-) | |
| Blood biochemistry | ACTH 88.7 pg/mL | |
| Total Protein 6.7 g/dL | Cortisol 23.2 μg/dL | |
| Albumin 4.2 g/dL | DHEA-S 130 μg/dL | |
| Globulin 2.5 g/dL | Renin activity 1.7 ng/mL/hr | |
| AST 50 U/L | Aldosterone 109 pg/dL | |
| ALT 37 U/L | TSH 10.41 μU/L | |
| γ-GTP 118 U/L | FT3 0.99 ng/dL | |
| LDH 300 U/L | FT4 2.29 pg/mL | |
| ALP 477 U/L | | |
| Total bilirubin 0.9 mg/dL | | |
| ChE 241 U/L | | |
| Creatinine 1.58 mg/dL | | |
| BUN 67 mg/dL | | |
| UA 13.2 mg/dL | | |
| CRP 2.79 mg/dL | | |
| CK 1,694 U/L | LDL-chol 95 mg/dL | |
| P-amylase 30 U/L | HDL-chol 74 mg/dL | |
| Elastase 602 ng/dL | Triglyceride 138 mg/dL | |
| Lipase 28 U/L | Total-chol 215 mg/dL | |

Table shows the clinical characteristics of this subject on admission. His blood glucose level was 806 mg/dL. His HbA1c and glycoalbumin were 6.3% and 22.5%, respectively. His insulin secretion was markedly suppressed: immune reactive insulin (IRI) <1.0 μU/mL and serum cardiopulmonary resuscitation (CPR) 0.1 ng/mL. Ketoic bodies were increased: 3-hydroxybutyric acid, 11,781 μmol/L and acetooacetic acid, 2,813 μmol/L. On an arterial blood gas examination, his pH was 7.194. Anti-glutamic acid decarboxylase (GAD) antibody was negative, but anti-IA-2 antibody was 1.4 U/mL. In addition, he had Kussmaul breathing, which is often observed in subjects with diabetic ketoacidosis. Based on these findings, we diagnosed him with fulminating T1DM and diabetic ketoacidosis. Renal dysfunction, likely due to dehydration, was observed; creatinine, 1.58 mg/dL; blood urea nitrogen (BUN), 67 mg/dL. Other data were as follows: Na, 120 mEq/L; K, 7.2 mEq/L; Cl, 99 mEq/L; white blood cells, 20,720/μL; Seg, 85.0%; C-reactive protein, 2.79 mg/dL; and creatinine kinase (CK), 1,694 U/L. Thyroid-related data were as follows: anti-thyroid peroxidase (TPO) antibody, 569.1 U/mL; anti-Tg antibody, 156.7 U/mL; thyroid stimulating hormone (TSH), 10.41 μU/L; FT4, 0.99 ng/dL; and FT3, 2.29 pg/mL. The increased CK was suspected of having facilitated the hyperkalemia and acute renal failure. No abnormalities were noted on abdominal computed tomography.

Although there were no baseline abnormalities in heart in this patient, a life-threatening abnormality of a wide QRS complex was noted on electrocardiogram (Fig. 1A). In addition, a tall, sharp T wave was also observed, probably due to severe hyperkalemia, which could lead to a lethal situation. Figure 2 shows a possible process through which such an abnormality might have occurred. We immediately started fluid infusion and insulin therapy and administered calcium gluconate to increase the stability of the cell membranes and prevent arrhythmia. After these treatments, the potassium level decreased to 4.5 mEq/L, and the life-threatening electrocardiographic abnormalities disappeared (Fig. 1B). The blood glucose level was also decreased the next day to approximately 200 mg/dL. We failed to evaluate the heart condition on admission precisely with echocardiogram due to severe palpitation, but there were no specific findings on echocardiogram after the patient had recovered.

We confirmed that he was not infected with cytomega-

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Figure 1. Electrocardiogram findings obtained before (A) and after the treatment of hyperkalemia (B). On admission, a life-threatening abnormality of a wide QRS was observed (A). However, this abnormality disappeared after insulin and calcium gluconate therapy in addition to fluid replacement (B).

Figure 2. Possible mechanism underlying the abnormal electrocardiogram findings.

Discussion

We herein report a patient who developed general malaise, nausea and vomiting, none of which are necessarily specific for hyperglycemia, and ultimately was diagnosed with fulminant T1DM accompanied by a life-threatening electrolyte abnormality as well as abnormal electrocardiogram findings. Given that the typical symptoms of hyperglycemia are thirst, polyuria and decreased body weight, the symptoms in this case may be considered atypical. As such...
severe life-threatening abnormalities in electrolyte levels and/or electrocardiogram findings are quite rare, which factors and/or conditions are involved in the development of such a severe situation—especially in the absence of typical symptoms for hyperglycemia—is unclear. Further analyses in a larger number of subjects with conditions similar to the present case will be needed to clarify this point. However, we feel that the findings in this case will still prove important from a clinical point of view.

The onset of fulminant T1DM is sometimes accompanied by sudden death or cardiac arrest (5). Previous studies have shown that subjects with sudden death or cardiac arrest tend to be younger; have a higher rate of impaired consciousness, hyperglycemia, hyponatremia, hyperkalemia, and hypochloremia; have more severe acidosis; and have a higher serum blood urea nitrogen level, a higher serum creatinine level, and a higher plasma osmolality level than those without such outcomes (5). In multiple logistic regression analyses, the plasma glucose level was found to be positively associated with sudden death or cardiac arrest. The present case was relatively young and had severe acidosis, hyperglycemia, hyponatremia, hyperkalemia, and high serum creatinine and blood urea nitrogen levels. Taken together, the present and previous findings suggest that physicians should be on guard against sudden death or cardiac arrest at the onset of fulminant T1DM in patients with the above risk factors.

In addition, the life-threatening abnormal findings on electrocardiogram in this subject may simply have been due to electrolyte abnormalities, as such electrocardiographic abnormalities which are not necessarily specific to the pathogenesis of FT1DM as seen in this subject. We must therefore be very careful when interpreting electrocardiogram findings of subjects with electrolyte abnormalities, regardless of their underlying disease.

In conclusion, we should bear in mind the possibility of fulminant T1DM in patients who complain of general malaise, as fulminant T1DM rapidly induces life-threatening electrolyte abnormalities and abnormal electrocardiogram findings.

Informed consent was obtained from the patient regarding his inclusion in the study.

The authors state that they have no Conflict of Interest (COI).

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