CASE REPORT

Fulminant Type 1 Diabetes Mellitus Presenting 15 Days after Delivery Diagnosed in Cooperation with Obstetricians

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Abstract:
The patient was a 32-year-old Japanese woman who was given a 75-g oral glucose tolerance test at the 35th week of pregnancy and was normoglycemic. She had excessive thirst and polyuria from 15 days after delivery. When she visited for the 1-month postpartum checkup, her plasma glucose level was 479 mg/dL, HbA1c was 7.4%, and urinary C-peptide was 1.1 μg/mL; she was therefore diagnosed with fulminant type 1 diabetes mellitus associated with pregnancy. All physicians should be aware of this disease so as to provide a prompt diagnosis and emergency treatment and consequently improve the maternal prognosis.

Key words: type 1 diabetes mellitus, fulminant type 1 diabetes mellitus, pregnancy, delivery, diabetic ketoacidosis

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Introduction

Type 1 diabetes mellitus (T1DM) is caused by a decline in the insulin secretion function due to the disruption of pancreatic β cells, which are divided into two categories: autoimmune type (type 1A), involving an autoimmune mechanism by pancreatic islet associated autoantibody; and a spontaneous type (type 1B), in which autoimmune involvement has not been proven. Fulminant type 1 diabetes mellitus (FT1DM) is a subtype of type 1B in which the pancreatic islet cells fail rapidly, leading to hyperglycemia and ketoacidosis. FT1DM was first reported by Imagawa et al. in 2000 (1). In Japan, FT1DM accounts for about 20% of acute-onset T1DM (2). FT1DM is often related to pregnancy and can develop during pregnancy and immediately after delivery (3). The major clinical characteristics of FT1DM are a markedly abrupt onset of disease with a very short (<1 week) duration, leading to severe ketoacidosis that requires the initiation of insulin therapy.

We herein report a case of FT1DM developing immediately after delivery, in which a good outcome was achieved by close contact with the obstetrics and gynecology departments.

Case Report

The patient was a 32-year-old woman without a history of pregnancy and delivery and no family history of diabetes. She had been given a 75-g oral glucose tolerance test at the 35th week of pregnancy to rule out gestational diabetes at that time. Her fasting blood glucose was 70 mg/dL, her 1 hour post-prandial blood glucose was 88 mg/dL, and her 2 hours post-prandial blood glucose was 119 mg/dL. She was diagnosed with a normal glucose tolerance.

She delivered a girl (3,034 g, Apgar score 10 points) at 40 weeks and 6 days by natural delivery. However, 15 days after delivery, she began to have excessive thirst, polyuria, and malaise. She did not have flu-like symptoms, such as a sore throat, cough, or nasal discharge. Obstetricians realized her abnormal condition when she visited for the one-month postpartum checkup. She was emergently admitted to our hospital with symptoms of nausea, abdominal pain, and vomiting. Upon admission, the patient was 149 cm tall, weighing 38.6 kg (immediately after delivery, 45.3 kg) with a body temperature of 37.4°C, blood pressure of 123/69...
mmHg, and a respiration rate of 12 times/min. A physical examination revealed spontaneous pain and tenderness in the upper abdomen. As shown in Table 1, she developed diabetic ketoacidosis (DKA), with a random sample glucose of 479 mg/dL, arterial pH of 7.162, PaO₂ of 121 mmHg, PaCO₂ of 15.3 mmHg, BE of -22.6 mmol/L, HCO₃⁻ of 5.2 mmol/L, lactate of 1.1 mmol/L, anion gap of 16.3 mmol/L, 3+ urinary ketone bodies, and serum ketone of 10,710 μmol/L. Despite the presence of DKA, the HbA₁c value was 7.4%, which was relatively low compared to the blood sugar level. In addition, the serum lipase level was slightly increased to 73 IU/L, but neither amylase nor elastase-1 levels were within the normal range. The plasma C-peptide (CPR) level progressively decreased day by day: 0.11 ng/mL on day 2, and <0.03 ng/mL on day 7. In a glucagon stimulation test, the ΔCPR was <0.01 ng/mL, and the urinary CPR was 1.1 μg/day. Antibody against glutamic acid decarboxylase (GADA) was 9.8 (<5.0) μU/mL. Neither islet cell antibody (ICA) nor anti-insulina-associated antigen-2 (IA-2Ab) antibodies were detected. Human leukocyte antigen (HLA) class II haplotypes were DRB1*09:01-DQB1*03:03 and DRB1*04:03-DQB1*03:02. Paired serum examinations for viral infections, such as Coxsackie, human herpesvirus 6 (HHV-6), and Epstein-Barr virus (EBV), which are known to be associated with the development of FT1DM, showed no clear evidence of a viral infection. The thyroid function was normal, and anti-thyroid autoantibody group was negative.

With a diagnosis of FT1DM, the patient was treated with intravenous insulin infusion. With the improvement of her ketoadicosis, her symptoms, such as upper abdominal pain, nausea, and malaise, also improved. After confirming that HCO₃⁻ was almost normalized by a blood gas analysis, she started oral ingestion and shifted to multiple daily injections with insulin lispro and degludec (Figure). Thereafter, her plasma glucose level was satisfactorily controlled, and she was discharged.

**Table 1. Laboratory Data on Admission.**

| CBC    | TSH    | Diabetes          |
|--------|--------|-------------------|
| WBC 6,200 μL | F-T3 | 0.50 μU/mL        |
| RBC 553x10⁴ μL | F-T4 | PG               |
| Hb 16.4 g/dL | Serum ketone | 479 mg/dL        |
| Ht 49.3 % | 10,710 μmol/L | PF               |
| Plt 38.1 μL | BGA | 0.21 ng/mL        |

Biochemistry

- Na 132 mEq/L
- Cl 104 mEq/L
- K 4.6 mEq/L
- GOT 17 U/L
- GPT 22 U/L
- LDH 194 U/L
- γ-GTP 15 U/L
- CK 95 U/L
- BUN 12 mg/dL
- CRE 0.57 mg/dL
- T-Bil 6.0 mg/dL
- TP 8.2 g/dL
- Alb 5.0 g/dL
- CRP 0.17 mg/dL
- Amy 101 U/L
- Lipase 59 U/L
- Elastase-1 73 ng/mL
- T-Chol 247 mg/dL
- HDL-Chol 55 mg/dL
- TG 126 mg/dL

- pO₂ 121 mmHg
- pCO₂ 15.3 mmHg
- HCO₃⁻ 5.2 mmol/L
- BE -22.6 mmol/L
- Anion Gap 16.3 mmol/L

- pH 7.162
- Coagulation
- Coa 24H U-CPR
- Lactate 1.1 mmol/L
- D-dimer <1.0 μ/mL
- Urinalysis
- C-reactive protein (CRP) 1.1 μg/mL
- Albumin (Alb) 4.7
- Creatinine (Cr): 8.0
- Electrolytes (EIA): 8.0
- Erythrocyte sedimentation rate (ESR): 4.8
- Human leukocyte antigen (HLA): 2.8
- Epstein-Barr virus (EBV): 2.6
- Human herpesvirus 6 (HHV-6): 1.1 μg/mL
- Anti-thyroid antibodies: <4
- Anti-insulina-associated antigen-2 antibody: <4
- Anti-insulinoma antibodies: <4
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Generally manifests late in pregnancy (4). The present patient, who developed FT1DM 15 days after parturition but was diagnosed with FT1DM associated with pregnancy (PF) based on the clinical course. FT1DM that develops during pregnancy or immediately after delivery (<2 weeks) is referred to as PF (5). It is suggested that pancreatic β cells are destroyed rapidly, resulting in the rapid depletion of endogenous insulin secretion in cases of PF. Our present patient had undergone a 75-g oral glucose tolerance test at the 35th week of pregnancy (45 days before FT1DM onset), which confirmed that her glucose tolerance was normal at that time. In addition, the rapid progressive depletion of endogenous insulin secretion (the plasma CPR level was 0.11 ng/mL on day 2, and <0.03 ng/mL on day 7) was observed. In

Table 2. Criteria for Definite Diagnosis of Fulminant Type 1 Diabetes Mellitus (2012)(3).

| Criteria                                                                 | Value                                     |
|-------------------------------------------------------------------------|-------------------------------------------|
| pH                                                                      | 7.162, 7.335                              |
| pO2 (mmHg)                                                              | 121, 101.7                                |
| pCO2 (mmHg)                                                             | 15.3, 30.0                                |
| HCO3- (mmol/L)                                                          | 5.2, 15.6                                 |

**Figure.** The clinical course of this case. Multiple daily injections were started after intravenous insulin infusion. The plasma glucose level was controlled satisfactorily, and she was discharged.

**Discussion**

According to the diagnostic criteria for FT1DM published from The Japan Diabetes Society (Table 2) (3), this case was diagnosed with FT1DM by satisfying three of the diagnostic criteria. It has been reported that FT1DM is more likely to develop during pregnancy and immediately after delivery (3). Almost all patients who develop T1DM during pregnancy or immediately after delivery have FT1DM, accounting for 22.7% of all FT1DM cases developing in women of childbearing age (13-49 years), and the disease generally manifests late in pregnancy (4). The present patient developed FT1DM 15 days after parturition but was diagnosed with FT1DM associated with pregnancy (PF) based on the clinical course. FT1DM that develops during pregnancy or immediately after delivery (<2 weeks) is referred to as PF (5). It is suggested that pancreatic β cells are destroyed rapidly, resulting in the rapid depletion of endogenous insulin secretion in cases of PF. Our present patient had undergone a 75-g oral glucose tolerance test at the 35th week of pregnancy (45 days before FT1DM onset), which confirmed that her glucose tolerance was normal at that time. In addition, the rapid progressive depletion of endogenous insulin secretion (the plasma CPR level was 0.11 ng/mL on day 2, and <0.03 ng/mL on day 7) was observed. In
In response to a viral infection, antigen-presenting cells, such as dendritic cells and macrophages, activate T cells via Toll-like receptor 3/4 (TLR 3/4). At the same time, inflammatory cytokines, such as interferon, are produced by the infected cells and delivered to pancreatic β cells. However, Regulatory T cells (Tregs), as a defense system against cytotoxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of toxic T cells, suppress the effect of cytotoxic T 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Compared to the previous cases, the duration of hyperglycemic symptoms before the diagnosis was longer and the levels of serum and urinary CPR much lower in the present case. Although the detailed mechanisms underlying the occurrence of FT1DM in relation to pregnancy have not yet been clarified, HLA and viral infection are considered to be involved. HLA class II genotypes are estimated to account for 50% of all cases of acute-onset T1DM (13, 14). Similarly, a HLA class II genotype strongly sensitizes the development of FT1DM (4, 15, 16). HLA class II haplotypes contribute to the development of PF or FT1DM that is not associated with pregnancy (NPF), although the relevant HLA class II haplotypes appear to differ between PF and NPF (4, 15). FT 1DM has been reported to be associated with DRB1*04:05-DQB1*04:01 in Japan (17). DRB1*04:05-DQB1*04:01 is significantly more frequent in NPF than in PF, while DRB1*09:01-DQB1*03:03 is significantly more frequent in PF than NPF (18). The involvement of HLA class II haplotypes is suggested to vary depending on the presence or absence of pregnancy. In the present patient, the HLA II haplotypes were DRB1*09:01-DQB1*03:03 and DRB1*04:03-DQB1*03:02. Thus, this patient had a genetic background that made her susceptible to developing PF.

Because FT1DM often precedes flu-like symptoms, the involvement of a virus infection has been suggested (2, 5). However, our patient did not have any flu-like symptoms, and no significant increase in viral antibody was observed. In response to a viral infection, antigen-presenting cells, such as dendritic cells and macrophages, activate T cells via Toll-like receptor 3/4 (TLR 3/4). At the same time, inflammatory cytokines, such as interferon, are produced by the infected cells and delivered to pancreatic β cells. However, Regulatory T cells (Tregs), as a defense system against cytotoxic T cells, suppress the effect of cytotoxic T cell and Tregs have an effect which prevents destruction of β cells by cytotoxic T cells; therefore, if Tregs are sufficiently active to suppress cytotoxic T cell activity, the onset of FT1DM is suppressed. The number of Tregs varies during pregnancy (19); Somerset et al. reported that the number increases from early pregnancy and decreases after reaching a peak in the middle of pregnancy. After the end of pregnancy, cytotoxic T cells are more likely to be activated by virus infection because the number of Tregs decrease from the late pregnancy stage, and this makes β cells more susceptible to damage by cytotoxic T cells, in other words, the normal defense mechanism may deteriorate, thus leading to the onset of FT1DM. In addition, interleukin-21 (IL-21), which induces immunoglobulin production from B cells, is produced from follicular helper T cells and is reported to be associated with autoimmune T1DM (20). The IL-21 level was found to be significantly lower in the third trimester of pregnancy than in non-pregnant women (21), which may be related to FT1DM being more likely to develop during preg-
nancy and immediately after delivery than autoimmune type 1 diabetes.

In conclusion, no significant differences in the age of onset, BMI, family history, disease duration, CPR, or GADAb were noted between PF and NPF, but the arterial blood pH of PF was significantly lower than that of NPF (18); therefore, PF may have a poorer prognosis than NPF, even after delivery. This case had extremely low arterial blood pH; as such, if the diagnosis had been delayed, the patient might have died. All medical professionals, including obstetricians, must recognize that FT1DM is likely to develop during pregnancy or immediately after delivery. If a pregnant woman or a woman immediately after delivery suddenly develops hyperglycemic symptoms, such as thirst, polydipsia, or polyuria, it is necessary to bear in mind the possibility of FT1DM.

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

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