Progression of gestational diabetes mellitus to pregnancy-associated fulminant type 1 diabetes: a case report

Qun Ji[1]^, Haiwei Liu[1], Fei Wang[1], Lan Gao[1], Kaining Chen[1], Huibiao Quan[1], Min Lu[2]

[1]Department of Endocrinology, Hainan Hospital Affiliated Hospital of Hainan Medical University/Hainan General Hospital, Haikou, China; [2]Department of Obstetrics, Hainan Hospital Affiliated Hospital of Hainan Medical University/Hainan General Hospital, Haikou, China

Contributions: (I) Conception and design: Q Ji, H Liu; (II) Administrative support: H Liu; (III) Provision of study materials or patients: Q Ji; (IV) Collection and assembly of data: H Liu, F Wang, L Gao, K Chen, H Quan; (V) Data analysis and interpretation: M Lu; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Haiwei Liu, MD. Department of Endocrinology, Hainan Hospital Affiliated Hospital of Hainan Medical University/Hainan General Hospital, 19 Xiuhua Road, Xiuying District, Haikou 570311, China. Email: hndxlhw@126.com.

Background: Pregnancy-associated fulminant type 1 diabetes (PF) occurs during pregnancy or within 2 weeks of delivery. Although it occurs infrequently, it is associated with high fetal mortality rate. Few studies have examined whether PF is associated with gestational diabetes mellitus (GDM).

Case Description: A 29-year-old woman diagnosed with GDM at 24 weeks of gestation developed a fever, sore throat, nausea and vomiting at 29 weeks of gestation. Ketoacidosis was considered based on her blood ketone and glucose levels and the results of a blood gas analysis. Since the patient's islet function declined rapidly, fluid replacement, insulin therapy, and other treatments were administered. The patient was ultimately diagnosed with PF, and has required ongoing insulin therapy. She delivered a healthy baby girl by elective cesarean section at 37-week gestation. Her blood glucose has been satisfactorily controlled over the 12 months since her acute presentation.

Conclusions: PF is characterized by poor maternal and infant outcomes and a high stillbirth rate. Blood glucose should be regularly monitored in pregnant women with GDM. A sudden increase in blood glucose may indicate the possibility of PF, which needs to be managed in a timely manner to avoid adverse pregnancy outcomes.

Keywords: Pregnancy-associated fulminant type 1 diabetes (PF); gestational diabetes mellitus (GDM); case report

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Introduction

Pregnancy-associated fulminant type 1 diabetes (PF) occurs during pregnancy or within 2 weeks of delivery, has a low incidence, and is characterized by a rapid decline in islet cell function and the onset of ketoacidosis (1). It is a highly dangerous condition associated with a high incidence of stillbirth. The cause of PF is not yet clear. Gestational diabetes mellitus (GDM) occurs during pregnancy and is harmful to both the mother and fetus (2). Unfortunately, few studies have examined the possible correlation between GDM and PF.

The current patient was diagnosed with GDM in her second trimester, but subsequently represented with PF. The fetus survived after active treatment. This report summarizes the clinical features of this patient along with review of the relevant literature with the aim to extend understanding about PF and to prevent its misdiagnosis and mistreatment. We present this article in accordance

^ ORCID: 0009-0002-1104-8846.

Table 1 General data of the patient

Parameters	Value
Age (years)	29
Gravida	1
Family history of diabetes	Denied
History of drug allergies	Denied
Maternal pre-pregnancy BMI (kg/m^2)	22.3

BMI, body mass index.

with the CARE reporting checklist (available at https://acr.amegroups.com/article/view/10.21037/acr-24-52/rc).

Case presentation

All the procedures performed in this study were conducted in accordance with the ethical standards of Hainan General Hospital and the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Chief complaints

A 29-year-old female in her 29th gestational week was admitted to hospital on January 23, 2021. Over the 3 days prior, she had experienced fever, fatigue, mild sore throat, and cough without any obvious trigger. She also reported one day of nausea and vomiting and stated that fetal movements appeared to have decreased over the preceding 12 hours (*Table 1*).

Five weeks earlier, she had been diagnosed with GDM after an oral glucose tolerance test (OGTT), which revealed a 1-hour postprandial glucose of 10.6 mmol/L (*Table 2*) [diagnostic criteria for GDM developed by the International Association of Diabetes and Pregnancy Study Group (IADPSG) in 2010: GDM is diagnosed by meeting or exceeding at least one of the following indicators: fasting plasma glucose (FPG) ≥5.1 mmol/L, glucose level at 1-hour postprandial glucose (1 h PG) ≥10.0 mmol/L and (or) at 2 hours postprandial glucose after OGTT (2 h PG) ≥8.5 mmol/L]. Subsequent to this, her blood glucose had been satisfactorily controlled with diet and exercise alone.

The initial laboratory test results (normal range) showed capillary blood glucose: 19 mmol/L (FPG 3.9–6.1 mmol/L); urine ketone bodies: 4+ (negative); pH: 7.1 (7.35–7.45); base excess (BE): –22 (–3 to 3); and urine amylase: 735.5 U/L (4–32 U/dL). A diagnosis of diabetic ketoacidosis (DKA) was considered. She received fluid replacement and glucose-lowering treatment [0.9% sodium chloride infusion (500 mL) + 10% potassium chloride (15 mL) + one-off human insulin (6–8 IU)], intravenous (IV) drip in the obstetrics department and was then transferred to the Endocrinology ward for ongoing care.

History of illness

The patient had a previous history of α-thalassemia [Southeast Asian (SEA) deletion] and denied a family history of diabetes. She previously had regular menstrual cycles, and the first day of her last menstrual period was June 30, 2020. Her childbearing history was G_1P_0 (*Table 1*).

Physical examination

General observations demonstrated body temperature: 36.7 ℃; heart rate: 103 beats per minute; respiratory rate: 20 breathes per minute; and blood pressure: 122/72 mmHg. Maternal pre-pregnancy body mass index (BMI) was 22.3 kg/m^2. She was alert and cooperative during the physical examination. She had normal vesicular breath sounds bilaterally with no added sounds on auscultation. Her heart rhythm was regular, with normal findings on examination of the praecordium. Longitudinal oval bulges were observed

Highlight box

Key findings

- Gestational diabetes mellitus (GDM) and gestational pregnancy-associated fulminant type 1 diabetes (PF) have different pathogenesis, and only a few patients have GDM before PF.

What is known and what is new?

- Fulminant type 1 diabetes mellitus (FT1DM) that occurs during pregnancy or the perinatal period is known as PF.
- PF is always without history of abnormal glucose metabolism. Here, we present a patient who developed PF during treatment but were first diagnosed with GDM.

What is the implication, and what should change now?

- Body mass index was not a predictor of PF; patients with GDM need to monitor their blood sugar regularly during pregnancy, and a sudden rise in blood sugar may alert them to turn into PF, which requires high attention and timely medical treatment to avoid adverse pregnancy outcomes.

Table 2 Examination/test results before the development of PF

Time to diagnosis of GDM	OGTT results in the second trimester (mmol/L)			Fasting insulin levels in the second trimester (4–23 pmol/L)	HbA1c in the second trimester (4–6%)	Insulin use
	0 h (<5.1 mmol/L)	1 h (<10 mmol/L)	2 h (<8.5 mmol/L)			
At the 24th gestational week	4.8	10.6	8.0	66.5	5.1%	Not used

Data in parentheses are ranges of normal values. PF, pregnancy-associated fulminant type 1 diabetes; GDM, gestational diabetes mellitus; OGTT, oral glucose tolerance test; HbA1c, glycated hemoglobin.

on abdominal inspection. Visible peristaltic waves were not present. There was no peripheral edema. Her upper and lower limb muscle strength and muscle tone were normal.

Gynecological examination showed uterine height: 25 cm; abdominal circumference: 85 cm; cephalic presentation; left occiput anterior position; head not engaged; palpable irregular uterine contractions; and fetal heart sounds: regular (136 beats/minute). A colposcopy showed that the cervix was not dilated.

Laboratory examinations

The ancillary test results (normal range) of the patient were as follows—white blood cell count: 14×10^9/L ($3.5–9.5\times10^9$/L); neutrophils (percentage): 80.9% (40–75%); serum glucose: 22.5 mmol/L (≤7.8 mmol/L); D3-hydroxybutyric acid: 8.1 mmol/L (0.03–0.3 mmol/L); lactic acid: 0.97 mmol/L (0.6–2.2 mmol/L); pH: 7.1 (7.35–7.45); and BE: 22 (–3 to 3). The routine urine tests showed urine glucose: 4+ (negative); and urine ketone bodies: 4+ (negative). There were no obvious abnormalities on routine stool tests. The test results for influenza A and B antibodies were negative, as was a nucleic acid test for severe acute respiratory syndrome coronavirus.

The biochemical results (normal range) of the patient were as follows—blood sodium: 131 mmol/L (137–147 mmol/L); and normal liver/kidney function. The pancreatic enzyme results (normal range) of the patient were as follows—blood amylase: 346 U/L (35–135 U/L); pancreatic amylase: 331 U/L (30–220 U/L); lipase: 320 U/L (150–200 U/L); islet function: C-peptide (0') [CP (0')], 0.017 nmol/L (0.37–1.47 nmol/L); and C-peptide (120') [CP (120')], 0.09 nmol/L (0.37–1.47 nmol/L). The glycated hemoglobin (HbA1c%) was 6.1% (4–6%), and the islet cell antibodies, antibodies to glutamate decarboxylase, and insulin autoantibodies were negative (*Table 3*).

Imaging examinations

Computed tomography (CT) of the upper abdomen revealed possible cholestasis of the gallbladder, but no obvious abnormalities were observed in the liver, pancreas, or spleen. Abdominal color ultrasound revealed thickened and unevenly distributed echoes in the liver parenchyma, but no sign of pancreatitis was observed. An electrocardiogram showed sinus tachycardia.

Final diagnoses

The patient's diagnoses were as follows: (I) PF; (II) DKA; (III) GDM.

Treatments

DKA treatment was started immediately after the identification of DKA, and included active fluid replacement [0.9% sodium chloride infusion (500 mL) + 10% potassium chloride (15 mL) + one-off human insulin (6–8 IU)], alternating with 5% glucose + 0.9% sodium chloride infusion (500 mL) + 10% potassium chloride (15 mL) + one-off human insulin (8–12 IU), IV insulin pump [human insulin (50 IU) + 0.9% sodium chloride (50 mL), infused at a rate of 2–5 mL/h], once every hour blood glucose monitoring, electrocardiogram monitoring, and fetal heart rate monitoring.

After DKA was corrected, a subcutaneous insulin pump was used (Medtronic, MMT-712EWS, Minneapolis, USA) with a total baseline dose of insulin aspart injection 26 IU/d (0:00–3:00 0.8 IU/h, 03:00–7:00 1.4 IU/h, 07:00–12:00 1.1 IU/h, 12:00–17:00 1.0 IU/h, 17:00–22:00 1.1 IU/d, 22:00–24:00 0.9 IU/h) and bolus dose before meals of 10–14 IU to maintain a stable blood glucose fluctuation range as follows: fasting: 4.5–5.7 mmol/L; 2-hour postprandial: 6–9 mmol/L.

In addition, a multidisciplinary team of experts from the nutrition, obstetrics, and psychology departments was established. Nutritious meals were provided after the correction of ketoacidosis, The nutritious meals were: 1,800 kcal of total calories, 250 g of carbohydrates, 80 g of

Table 3 Clinical features and examination/test results after the development of PF

Parameters (range and unit of normal value)	Value at the 29th gestational week
Interval between GDM and PF (weeks)	5
Clinical symptom	Fever, fatigue, sore throat, cough, and vomiting
Routine blood test (WBC, 3.5–9.5 $\times10^9$/L; NE%, 40–75%)	WBC, 14×10^9/L; NE%, 80.9%
Blood glucose (≤7.8 mmol/L)	22.5
Serum D3-hydroxybutyric acid (0.03–0.3 mmol/L)	8.1
pH value (7.35–7.45)	7.1
FCP (0.37–1.47 nmol/L)	0.017
PCP (0.37–1.47 nmol/L)	0.09
HbA1c (4–6%)	6.1%
Islet antibodies	Negative
PG/HbA1c (<3.3)	3.68
SCr (57–97 μmol/L)	41
CK (50–310 U/L)	65
Lactic acid (0.6–2.2 mmol/L)	0.97
Serum amylase level (35–135 U/L)	346
Pancreatic amylase level (30–220 U/L)	331
Serum lipase level (150–200 U/L)	320
Abdomen ultrasound or CT	No signs of pancreatitis
Fetal outcomes	Survived
Delivery mode	Cesarean section
Long-term treatment options	Insulin pump

PF, pregnancy-associated fulminant type 1 diabetes; GDM, gestational diabetes mellitus; WBC, white blood cell count; NE%, neutrophils (percentage); FCP, fasting C-peptide; PCP, 2-hour postprandial C-peptide; PG, plasma glucose; HbA1c, glycated hemoglobin; SCr, serum creatinine; CK, creatine kinase; CT, computed tomography.

proteins, 53 g of fats, and the proportion of breakfast, lunch and dinner is 1/5, 2/5, and 2/5, respectively. And the fetal condition was assessed by the obstetricians. The mother was diagnosed with mild depression by the psychology department, and psychological counseling was offered.

Outcome and follow-up

After active treatment, the patient's mental status improved remarkably. Her nausea and vomiting resolved, and her appetite improved. Her blood glucose after DKA correction was well controlled (fasting: 4.5–5.7 mmol/L; 2-hour postprandial: 6–9 mmol/L). Fetal heart rate monitoring revealed no obvious abnormality.

Two months subsequent to her presentation with DKA (at 37 weeks of gestation), the mother delivered a healthy baby girl by elective cesarean section. Over the subsequent 12 months, she maintained stable glucose control using an insulin pump (fasting blood glucose 4.5–7 mmol/L; 2-hour postprandial glucose 6–11 mmol/L). Her most recent HbA1c was recorded to be 7.6%. She has not had recurrence of episodes of DKA.

Discussion

Fulminant type 1 diabetes mellitus (FT1DM) is a new subtype of type 1 diabetes mellitus (T1DM) that was first proposed by Imagawa and colleagues in 2000 (3). FT1DM

diagnosis was proposed by the Japan Diabetes Association in 2016 Standard (4). It is more common in Asian populations, and is especially common in Japan, South Korea, and China. FP is defined as FT1DM that occurs during pregnancy or within 2 weeks of delivery. FP is the most common type of FT1DM (5) and typically occurs in the third trimester. However, few studies have examined the possible correlation between GDM and PF.

The current report summarizes the clinical features of a woman diagnosed with PF. She had been diagnosed with GDM in the second trimester, and then experienced an acute onset of hyperglycemia and ketoacidosis in the third trimester. Her blood glucose was ≥16.0 mmol/L, but her HbA1c was not high (<8.7%). Her fasting and postprandial serum C-peptide levels were almost undetectable. There were no abnormal findings on the pancreatic ultrasound or CT (to determine the presence of pancreatitis, CT was performed with the patient's informed consent). These findings represented the typical clinical and laboratory features for PF and she met the diagnostic criteria for PF.

Both the 2021 American Diabetes Association standards (6) and the 2019 World Health Organization guidelines (7) mention hyperglycemia during pregnancy. These descriptions include GDM, overt diabetes diagnosed in pregnancy, and pre-conceptional diabetes, but do not include PF. This suggests that the pathogenesis and clinical features of PF are completely different from any other type of hyperglycemia during pregnancy.

Both GDM and overt diabetes diagnosed in pregnancy occur due to the increased insulin resistance caused by insulin-antagonizing hormones secreted by adipocytes and placental tissues after pregnancy, the low-level inflammatory responses, and the reduced sensitivity to insulin of pregnant women (8). They can be regarded as compensatory and decompensated manifestations that help maintain the normal physiological glucose metabolism (8). After the delivery of the fetus and placenta, insulin resistance is alleviated and blood sugar improves or even returns to normal; thus, insulin can be used in much lower dosages or stopped. However, for person with PF, postpartum blood glucose is difficult to control due to the complete loss of islet function, and lifelong insulin use is consequently required.

The common etiologies of PF include viral infections, human leukocyte antigen (*HLA*) gene susceptibility and autoimmunity. Various viral infections have been described: these include coxsackievirus (9), herpes virus, and influenza virus. These viruses can directly and rapidly destroy β cells and also initiate autoimmunity by exposing the antigens. In one Chinese report, however, few patients were shown to have a viral trigger (10). Consistent with that observation, although the current case initially had a fever and symptoms of an upper respiratory tract infection, viral testing was negative.

The presence of specific HLA class II genes, notably *HLA DR* and *DQ* genes is closely related to the occurrence of PF (11). Next, with the patient's informed consent, we intend to improve the testing of the woman's related genes.

Autoimmunity also plays a key role in the pathogenesis of PF (12). While most individuals with PF have negative antibodies, a small proportion may have islet autoantibodies. Given the low rate of seropositivity, this is not regarded as a diagnostic criterion for PF. However, the autoantibodies in the current case were negative, which is consistent with most individuals with PF.

The pancreatic histopathology of patients with FT1DM is characterized by the rapid destruction of both α and β cells, which differs from classic T1DM, in which only β cells are destroyed at a relatively low rate of destruction. In addition, the elevated pancreatic enzymes in the current patient were consistent with the reported elevated exocrine pancreatic indicators in patients with PF (10). There were, however, no signs of pancreatitis on imaging. This combination of results might be explained by lymphocyte infiltration of the exocrine pancreas without pancreatic edema (6).

The clinical manifestations and metabolic disorders of patients with PF are more severe than those of patients with FT1DM who are not pregnant. Clinically, it is manifested as a rapid onset (typically within 1 week). PF also has the following clinical features: (I) influenza-like symptoms or gastrointestinal symptoms before onset; (II) a high blood glucose level and a nearly normal HbA1c level; (III) elevated exocrine enzymes of the pancreas; (IV) no signs of pancreatitis on imaging; and (V) an onset during pregnancy or within 2 weeks of delivery. In addition, unlike pre-GDM complicated by gestational DKA, PF has a much higher fetal mortality rate. In one report, stillbirth occurred in eight (89%) of nine cases of PF (13). In contrast, the fetal mortality rate of pre-pregnancy T1DM complicated by DKA is only about 9–36% (14).

Emaciation is a common characteristic of patients with classic T1DM and non-pregnancy-associated FT1DM. Conversely, the body weight of patients with PF is typically quite different from that of patients with non-pregnancy-associated FT1DM. Based on the body weight of several

other patients with PF admitted to our department and the cases described by Peking Union Medical College Hospital, we noted that PF is more likely to occur in those who are overweight or obese (personal unpublished observation). In one of our previous studies (15), the preconception BMI of patients with PF did not differ from that of those with GDM. However, due to the small sample sizes, the reliability of these conclusions needs further verification.

According to the literature, most pregnant women deny a history of GDM before the occurrence of PF (11). In contrast, our patient had GDM during pregnancy. Thus, the question arises as to whether GDM is directly associated with the development of PF. It has been reported that patients with GDM are at risk of developing T1DM diabetes and a variety of autoimmune diseases (16). Islet autoimmunity may be involved in the development of PF, but the specific mechanism needs to be further studied.

In the current case, blood glucose fluctuations were revealed by close monitoring, which was important in identifying the cut-off time point at which GDM turned into PF. If blood glucose suddenly becomes difficult to control and deteriorates rapidly in a woman with GDM whose lifestyle interventions and insulin doses remain unchanged, the risk of PF should be considered. In the current case, the patient received regular blood glucose monitoring after the diagnosis of GDM. Before PF occurred, her blood glucose was basically well controlled. However, she later suffered from a sudden blood sugar increase and sought medical treatment promptly. Her fetus ultimately survived.

Early detection is especially important for PF. GDM turned into PF in the current case, which indicates that blood glucose should be monitored regularly in women with GDM until after delivery. According to Liu *et al.* (17), a PG/HbA1c ratio with a threshold of ≥3.3 can be used as a cut-off point in predicting PF from DKA in China. An elevated PG/HbA1c ratio at the time of diagnosis is predictive of more severe insulin secretion dysfunction and a poor prognosis. In our case, a sudden increase of blood glucose for unknown reasons and a PG/HbA1c ratio of ≥3.3 are highly suggestive of the possibility of PF, and further tests for blood glucose, HbA1c, D3-hydroxybutyric acid, C peptide, and islet antibodies should be performed.

Treatment for DKA should be commenced as early as possible, even before the diagnosis of PF is confirmed. DKA therapies include aggressive fluid replacement, the early insulin administration (via an insulin pump), the maintenance of the water-electrolyte balance, infection control, and fetal heart rate monitoring. In addition, due to the high basal cardiopulmonary stress in pregnant women, special attention should be paid to rehydration rate. After the DKA is corrected, an insulin pump or an intensive insulin regimen may be applied to maintain target blood glucose levels. Hosokawa *et al.* (18) recently reported that induced pluripotent stem cells may be a new therapeutic strategy for PF.

Conclusions

In summary, PF is characterized by poor maternal and infant outcomes and a high stillbirth rate. However, early recognition and treatment of FT1DM is crucial in preventing unfavourable pregnancy outcomes (19). The current patient had been diagnosed with GDM in the second trimester 5 weeks prior to presenting with influenza-like symptoms. She then experienced an acute onset of hyperglycemia, with ketoacidosis and reduced pancreatic islet cell function, but her HbA1c was not elevated. Together, these findings represent the typical clinical and laboratory features of PF. Moreover, patients with GDM are at risk of developing T1DM diabetes and a variety of autoimmune diseases. Islet autoimmunity may be involved in the development of PF, but the specific mechanism needs to be elucidated.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://acr.amegroups.com/article/view/10.21037/acr-24-52/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All the procedures performed in this study were conducted in accordance with the ethical standards of Hainan General Hospital and the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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