A CASE STUDY ON GESTATIONAL DIABETES MELLITUS

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Objectives of Case study Presentation

- • To share experience and knowledge with participants.
- • To get feedback from the participants for further improvement.
- • To provide brief note on Gestational Diabetes mellitus and its complications

GESTATIONAL DIABETES MELLITUS

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.** 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. Unfortunately, few studies have examined the possible correlation between GDM and PF.

Incidence and prevalence of DM

- **Global Increase:**
- The global incidence of pregnancy-associated fulminant type 1 diabetes (PF) is not precisely known due to its rare and infrequent nature, Studies from Japan suggest that PF accounts for approximately **21% of all fulminant type 1** diabetes cases, though this proportion varies geographically.
- **India's Burden:**
- India has a high overall diabetes burden, and the risk of gestational diabetes is significant, with recent estimates ranging from **(0.80%) to (13%)** depending on the study, which could be a risk factor for PF development.

Patient Information

- **Name**: Mrs . ABCD
- **Age**: 32 years
- **Gender**: Female
- **Date of admission**: 12/10/25
- **Date of discharge**: 22/10/2025
- **Chief complaints**: 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.

- **Present health history**:
- • Patient has no history of DM previously.

- 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.

- **Past medical history**:
- 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** [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.

- **Past medical history**:

- 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 29, 2024.
- Her childbearing history was G_1P_0

- **Past surgical history: NA**

- **Family history:**

• Not Having history of Hypertension, Diabetes and cardio vascular diseases in family for past 2 generations.

Personal history:

- Smoking :- NA
- Alcohol : -NA
- Food habit :- patient is having mixed meal pattern
- Bowel and bladder :- Regular bowel and bladder habit
- Sleeping Pattern :- having regular 8-10 hours of sleep during both day time and night time.

- **P HYSICAL EXAMINATION**

General Inspection:

- Gait : Coordinated
- Body Build : General
- Consciousness : Conscious and alert
- Weight : 70kgs

- **Vital signs**
- Temperature : 98f
- Pulse : 103 b/minute
- Respiration : 20 /minute, regular
- Blood Pressure : 150/90 mm Hg in both arms (supine)

- **General examination**
- • Pallor Absent
- • Icterus absent
- • Lymph node not palpable
- • Clubbing, cyanosis absent
- • Pedal edema not present
- • Dehydration present
- • Skin dry

- **Physical examination**
- Examination of head, face and neck
- 1.**Head**
- Hair colors and texture normal, clean hair no any injury
- 2.**Eyes**
- No watering of the eyes and no redness of the eye lid
- 3. **Ears**
- No discharge

- 4. **Nose**
- No discharge.
- 5.**Mouth**
- Redness of oral cavity.
- 6. **Neck**
- No enlarged lymph node and thyroid gland, normal neck mobility is present
- 7. **Extremities**
- No Edema present in lower limbs
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- **SYSTEMIC EXAMINATION**

- **Respiratory examination**
- • Inspection- normal
- • Palpation- Non tender
- • Percussion- Resonant in all side of the chest.
- • Auscultation- Normal breath sound in both site (20 breaths/min)
- **Cardiovascular system**
- Inspection: Normal
- Palpation: Non tenderness.
- Auscultation: regular heart beat 103 beats/ min.

- **Abdominal examination**:
- **Inspection**- Longitudinal oval bulges were observed on abdominal inspection. Visible peristaltic waves were not present. no scar marks.
- **Palpation**- uterine height: 25 cm; abdominal circumference: 85 cm; cephalic presentation; left occiput anterior position; head not engaged; palpable irregular uterine contractions.
- **Percussion**- Dullness present
- **Auscultation**-fetal heart sounds: regular (136 beats/minute).
- A colposcopy showed that the cervix was not dilated.

- **CNS examination**

- • Mental function is adequate
- • Motor examination eg position of limbs normal, no atrophy.
- • No abnormal movement.
- • Sensory normal
- Numbess and tingling sensations in the both foot after prolonged sitting

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 $\times 10^9$/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

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

Parameters (range and unit of normal value)	Value at the 29th gestational week
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.

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.

Treatment:

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 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 psychiatry departments** were 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 proteins, 53 g of fats, and the proportion of breakfast, lunch and dinner respectively. And the fetal condition was assessed by the obstetricians. The mother was diagnosed with mild depression by the psychiatry 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

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.

Discussion

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, 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. Consistent with that observation, although the current case initially had a fever and symptoms of an upper respiratory tract infection, viral testing was negative.

Discussion

According to the literature, most pregnant women deny a history of GDM before the occurrence of PF. 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.** Islet autoimmunity may be involved in the development of PF, but the specific mechanism needs to be further studied.

Discussion

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. 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., 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.

Recommendations (Informal Health Teaching)

1. Treatment for DKA should be commenced **as early as possible**, even before the diagnosis of PF is confirmed.
2. 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.
3. 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. recently reported that induced pluripotent stem cells may be a new therapeutic strategy for PF.

Nursing Management

NURSING CARE PLAN

ASSESSMENT	DIAGNOSIS	PLANNING	IMPLEMENTATION	EVALAUTION
Subj. DATA: Patient Says her appetite has Increased. OBJ. DATA: Blood glucose levels: FBS - 155 mg/dl PP - 204 mg/dl HbA,C - 9.3	Altered nutri -tional status less than body requirement r/t inability to utilize nutrients approximately [imbalance be -tween glucose intake and glucose utili -zation.	• To weigh client on each prenatal visit. • To Asses Calorie intak -e and dietary patte -rn using 24 hours re Call. • To review Importance of regularity of meals and snacks when taking Insulin. • To Discuss dosage, sche -dule, type of Insulin. • To adjust diet or Insulin regimen to meet Individual needs. • To Refer to registered dietician to Individuals diet pattern. • To monitor Serum glu Cose levels. • To Ascertain results	• client weight is 56kg. • Advised the patient to eat well balanced diet. It should be highly nu tritious and easily digestable. • Take meals after 15 minutes of Insulin admi -nistration. • Diet with 2000 - 2500kcal/ day for normal women weight and restriction to 1200 - 1800 kcal/day for overweight women is recommended. High protei -n diet. • Avoid food containi ng excess of Carbohydrates like Sweets, honey, sugar, fried foods, Cold drinks. • Avoid Foods rich in fats and calori	• patient mai -ntains fasting Serum glucose level between 80 - 100mg/dl and follows the diabetic diet chart

Subj. DATA : patient c/o frequent and Excessive urination. OBJ. DATA: patient looks lethargic and dehydrated. Total Urine Output is 2700ml/day.	Risk For fluid Volume deficit r/t loss of fluids from body duto to polyuria.	• To assess the hydration status of client and observe the skin turgor. • Take assess Intake and Out put daily. • To monitor Vital Signs. • To discuss Signs and Symptoms of hypoglycemia and hyperglycemia • To Recommend monitoring urine for ketones on awakening and when meal	• Examined the skin turgor and mucous membrane. • monitored the intake and output of client • Vitals monitored. • Advised patient to have adequate fluid consumption and small frequent meals at regular interval • Mild edema present over lower Extremities. • Signs and Symptoms of hypoglycemia & hyperglycemia. • Recommended monitoring urine for ketones on awakening and when a meal is delayed	Hydration Maintained and Vitals are normal.

Primary Medical Dx and pathophysiology: Diabetes Mellitus - Acute Complications

Assessment Noticing / Recognizing cues what do I see, hear, feel?	Nursing Diagnosis Analysing cues / prioritize hypothesis (what's the problem?)	Plan Generate Solutions what am I going to do about it?	Implementation Take action what problem am I solving? why would I do this?	Evaluation Reflection [How will I know what I am doing is working?]
DKA: • underlying causes are infection and illness metabolic syndrome, missed dose. • Can lead to seizures, coma, and death. S/S: • Hypersmolarity (hypertonic). • Acetone breath related to metabolic acidosis. • GI Sympto n/v/ adb pain) because of acidosis Component (acetone)	Extreme Dehydration for both DKA and HHS: • Cells collapse so blood will be thick with glucose, electrolytes, ketones. Priority Focus: • Near circulatory collapse • Severe dehydration.	We need to hydrate with fluids, then address hyperglycemia with insulin therapy. For electrolytes imbalance we're going to focus on potassium levels (normal to high) e.g. place pt in cardiac monitor. For DKA, we need to treat the ketoacidosis with bicarbonate therapy only used in extreme cases of ph of 7.0 or lower. Some providers choose to not use it because it's hard to reverse.	Reasses / Monitor: • Blood glucose hourly. • check electrolytes before giving fluids. • check potassium levels before giving insulin.. (hypokalemia) • monitor their Vital signs every 15 mins until stable and then every hour, and then every 4 hours.	• Blood glucose levels < 200mg/dl • Acidosis resolves • Vital Signs Stabilize. • Electrolyte imbalance resolve • urine Output within normal limits. • Treatment is effective if blood glucose is <200 and acidosis is resolved.

- polyuria can lead to dehydration.
- polydipsia.
- polyphagia.
- Tachycardia
- BP goes down (hypotension)
- Altered mental status.
- kussmaul respirations the longer acidotic state continues.
- weight loss
- blurred vision, headache, weakness.

Goal: Low Serum glucose to 50-75:

- First check blood glucose and then potassium.
- First: The IV bottle dose will be 0.1 unit/kg/hr
- Second: move to continuous IV infusion of 0.1 unit/kg/hr.
- Third: Give Subcu when po intake is possible & ketosis stopped.

Tx: [Meds, diet, activity level]

Fluids

- Infuse Isotonic Solution (0.9) every 1-3 hours to restore blood volume.
- Then shift fluid to hypotonic Solution (0.45) to begin rehydrating the blood cells.
- Treat client with insulin IV boules to get it in system Fast to bring down glucose levels until it reaches 250mg and then start IV solution of D5.45 Nacl because we don't want blood glucose.

Conclusion:

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. 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. **Islet autoimmunity** may be involved in the development of PF, but the specific mechanism needs to be elucidated.

"It all starts in the mind. Just keep reminding yourself that you can beat this"