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DIABETIC KETOACIDOSIS IN PREGNANCY WITH RESPIRATORY AND LATENT SYPHILIS INFECTION: A CASE REPORT

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Abstract

Introduction: DKA in pregnancy is a rare and life-threatening condition. In general, this case occurs more often in patients with a history of Type I Diabetes Mellitus (DM) compared to Type II DM or gestational DM.

Case: A 33-year-old pregnant woman (G2P1A0) 20 weeks and 6 days of gestation complaints of shortness of breath accompanied by fever, dry cough, and nausea and vomiting. She had not felt fetal movements since one day before admission. The patient had a history of type II DM. From examination, a respiratory rate of 28 breaths per minute with Kussmaul breathing, a heart rate of 92 beats per minute, body temperature of 37.7°C, and tenderness in the epigastrium. Complete blood count showed WBC 20.21 x 10^3/µL with blood gas analysis was acidosis metabolic, urinalysis showed ketones +4, Glucose +4, Protein +1, and TPHA reactive. Chest X-ray examination found bronchitis and abdominal USG showed no fetal heartbeat and fetal movement. The patient showed significant improvement after administration of therapy.

Discussion: Physiologically, pregnancy carries a risk for DKA due to increased insulin resistance and ketogenesis. This risk will increase with the presence of precipitating factors such as infection. The infection will trigger the release of pro-inflammatory cytokines and counter-regulatory hormones such as cortisol or adrenaline which trigger ketoacidosis.

Conclusion: A multidisciplinary approach to managing DKA is crucial to minimizing maternal and infant mortality. Pre-pregnancy education and counseling should be provided to all women planning to become pregnant, whether with or without a history of diabetes.

Keywords: Diabetic ketoacidosis, pregnancy, infection, intrauterine fetal death

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Introduction

Diabetic ketoacidosis (DKA) is a severe and potentially life-threatening complication of diabetes mellitus (DM). This condition is characterized by acute hyperglycemia, ketosis, and metabolic acidosis, and it is more commonly seen in patients with Type 1 DM than in those with Type 2 DM or gestational diabetes [1]. A study conducted in the United Kingdom in 2021 estimated the incidence of DKA to be 16.6 per 1000 women with Type 1 DM who gave birth and 1.1 per 1000 women with Type 2 DM who gave birth [2]. Although cases of DKA in Type 2 DM and gestational diabetes are relatively rare, the risk is expected to rise with the increasing prevalence of Type 2 DM and gestational diabetes.

Physiologically, pregnancy carries a risk for DKA due to increased insulin resistance, and ketogenesis [3]. This risk can be exacerbated by factors such as infection, inadequate treatment, dehydration, acute kidney insufficiency, acute thrombosis or vascular events, trauma, other endocrine disorders, and the use of certain medications (e.g., thiazides, SGLT-2 inhibitors, antipsychotics, steroids). Infections, particularly respiratory and urinary tract infections, are the most common triggers for DKA [4].

Previous studies have shown that maternal mortality due to DKA is relatively low, less than 1%, but perinatal morbidity and mortality are high. Dhanasekaran et al. reported no maternal deaths, but fetal death occurred in 10 pregnancies (17.2%), with 6 miscarriages and 4 stillbirths. Among the live-born infants, 16 (33.3%) were diagnosed with large for gestational age, 4 (8.3%) had shoulder dystocia, and 5 (10.4%) were diagnosed with congenital anomalies [5].

DKA in pregnancy is a rare occurrence, but its consequences can be severe for both the mother and the fetus. Education, early detection, and appropriate management are key to

preventing DKA and reducing the risks of morbidity and mortality. We report a case of a 33-year-old female with DKA at 20 weeks and 6 days of gestation, intrauterine fetal death, bronchitis, and syphilis infection.

Case Illustration

A 33-year-old pregnant woman (G2P1A0) presented to the emergency department with complaints of shortness of breath that began the previous night and worsened by this morning. Four days before hospital admission, the patient experienced intermittent fever, dry cough, nausea, and vomiting 3-4 times, with food content. She had not felt fetal movements since one day before admission. Intermittent abdominal pain, vaginal discharge, and vaginal bleeding were denied. She denied any genital lesions or warts and red spots on her body. The patient was diagnosed with Type 2 DM approximately 4 years ago and had experienced DKA about one year ago. She was regularly using insulin therapy, including 14 units of Glargine once daily and 12 units of Lispro three times daily. The patient is currently a housewife, but she has previously worked at a spa.

Upon examination, the patient was alert and conscious (GCS E4V5M6), with vital signs showing a blood pressure of 138/84 mmHg, respiratory rate of 28 breaths per minute with Kussmaul breathing, heart rate of 92 beats per minute, body temperature of 37.7°C, and oxygen saturation of 98% on 3 L/min via nasal cannula. From physical examination it was found bronchovesicular breath sounds and tenderness in the epigastric region. Obstetric examination showed a fundal height corresponding to the umbilicus, absent fetal heart beat, negative signs of uterine contractions (his), and no vaginal discharge.

Additional diagnostic investigations were performed, including laboratory tests, urinalysis, chest X-ray, blood gas analysis, and abdominal ultrasound. Laboratory results showed: Complete blood count: HGB 15.0 g/dL, HCT 44.8%, WBC 20.21 x 10^3/µL, PLT 327 x 10^3/µL. Liver function: SGOT 17 U/L, SGPT 26 U/L. Kidney function: BUN 34 mg/dL, SC 1.3 mg/dL. Random blood glucose: 530 mg/dL. Electrolytes: Sodium 133 mmol/L, Potassium 5.1 mmol/L, Chloride 99 mmol/L. Blood gas analysis: pH 7.01, pCO2 10 mmHg, pO2 123 mmHg, cHCO3 3 mmol/L, SO2 97%. Urinalysis: Leukocyte esterase 25, Ketones +4, Glucose +4, Protein +1. Triple elimination test: HIV negative, hepatitis B negative, TPHA reactive. Chest X-ray: bronchitis. Abdominal ultrasound: No fetal heartbeat or fetal movement detected and estimated gestational age at 20 weeks and 6 days.

The patient was diagnosed with DKA, intrauterine fetal death (IUFD), bronchitis, and latent syphilis. Initial management included oxygen supplementation via nasal cannula at 3 L/min and intravenous fluid resuscitation with 1500 cc of NaCl0.9%, followed by 30 drops per minute. A urinary catheter was inserted for fluid balance monitoring. The patient also received insulin drip (Lispro) at 4 units per hour with hourly blood glucose monitoring. The insulin drip was gradually reduced as blood glucose levels improved and then stopped. Other treatments included cefotaxime 1 gram IV every eight hours, omeprazole 40 mg IV every 24 hours, ondansetron 4 mg IV every eight hours, and sodium bicarbonate infusion (50 mEq in 0.9% NaCl) over 8 hours with an infusion pump. The patient was treated in the Intensive Care Unit (ICU).

After 6 hours of insulin drip, the patient's blood glucose had decreased to 167 mg/dL, and the drip was continued until the following day, at which point it was switched to subcutaneous Lispro 4 units three times daily. After 4 hours of sodium bicarbonate infusion, repeat blood gas and electrolyte tests showed improvement in metabolic acidosis, with pH 7.29, pCO2 25 mmHg, cHCO3 12 mmol/L, SO2 99%, sodium 136 mmol/L, potassium 3.5 mmol/L, and chloride 103 mmol/L. After the patient's condition is stable, the obstetrics and gynecology team terminated the pregnancy using misoprostol, followed by dilatation and curettage. The patient was discharged on day 5 of hospitalization with improved symptoms and stable blood glucose levels. Further evaluation and treatment for syphilis will be provided during the follow-up at the dermatology and venereology clinic.

Discussion

Diabetic ketoacidosis (DKA) is a medical emergency caused by an absolute or relative deficiency of insulin, accompanied by increased activity of counter-regulatory hormones. It is defined by the triad of hyperglycemia, metabolic acidosis, and ketosis[1].Insulin deficiency triggers lipolysis, breaking down triglycerides into free fatty acids (FFAs). These FFAs are subsequently converted into ketone bodies through beta-oxidation in the mitochondria. Because ketone bodies are acidic, excessive production of ketone bodies will deplete the body's buffer reserves and reduce blood pH, a condition known as ketoacidosis. The activation of counter-regulatory hormones further exacerbates hyperglycemia through glycogenolysis and gluconeogenesis, which in turn leads to progressive volume depletion and electrolyte loss [6]. DKA in pregnancy is relatively rare, typically occurring in patients with a history of Type 1 DM. A study conducted in South Africa found 48 pregnant women with DKA, 31 of whom (66%) had a pre-existing history of diabetes, while 16 patients (34%) were newly diagnosed with diabetes. Among the 31 patients with pre-existing diabetes, 22 had Type 1 DM and 9 had Type 2 DM[7]. Similar findings were reported in another study, where 58 patients with DKA were reviewed: 82.8% had Type 1 DM, while 17.2% had Type 2 DM[5]. In the present case, the patient had a history of Type 2 DMand was routinely using insulin therapy with Glargine and Lispro.

The initial evaluation of a patient with hyperglycemia includes a metabolic assessment, evaluation for infectious diseases, and screening for complications or comorbidities. Patients typically present with symptoms of hyperglycemia, including polyphagia, polyuria, polydipsia, nausea, vomiting, loss of appetite, and abdominal pain. Dehydration signs may also be present, such as dry mouth, tachycardia, decreased sweat and urine output, skin turgor loss, delayed capillary refill time, hypotension, and even altered consciousness. A decrease in blood pH leads to respiratory compensation, where the body tries to expel excess carbon dioxide, often resulting in deep, labored breathing (Kussmaul respiration) or fruity-smelling breath due to the presence of acetone. In patients with an infection trigger, fever or hypothermia, along with symptoms related to the source of the infection, may be present. Initial

diagnostic workup should be performed based on clinical indications and setting, starting with blood glucose measurements and extending to tests for serum ketones, electrolytes, serum osmolality, blood gas analysis, kidney function, amylase and lipase levels, HbA1c, and urinalysis. To identify potential infection triggers, further tests such as a complete blood count, chest X-ray, and urine and blood cultures may be indicated[8,9]. In this case, the patient presented with shortness of breath accompanied by Kussmaul breathing, abdominal pain, and fever. The laboratory findings met the criteria for diabetic ketoacidosis (DKA) according to the American Diabetes Association (ADA) (as shown in Figure 1), with a blood glucose level of 503 mg/dL, urine ketones +4, and blood gas analysis showing metabolic acidosis (pH 7.01, pCO2 10 mmHg, pO2 123 mmHg, cHCO3 3 mmol/L, SO2 97%). Based on these findings, the patient was classified as having severe DKA.

Severity	Glucose (mg/dl) (mmol/l)	Arterial or venous pH	Bicarbonate (mmol/l)	Urine or serum ketones (nitroprusside test)	β-hydroxybutyrate (mmol/l)	Anion gap (mmol/l)	Mental status	Ref.
American Diabetes Association criteria for adults								
Mild	>250 (13.8)	7.25–7.30	15–18	Positive	>3.0	>10	Alert	8
Moderate	>250 (13.8)	7.24–7.0	10–15	Positive	>3.0	>12	Alert/drowsy	
Severe	>250 (13.8)	<7.0	<10	Positive	>3.0	>12	Stupor/coma	
Joint British Diabetes Societies for Inpatient Care								
NA	>200 (11.1)	<7.30[a]	<15	Positive	>3.0	NA	NA	130
International Society of Pediatric and Adolescent Diabetes								
Mild	>200 (11.1)	<7.30[a]	<15	Positive	>3.0	NA	NA	22
Moderate	>200 (11.1)	<7.2[a]	<10	Positive	>3.0	NA	NA	
Severe	>200 (11.1)	<7.1[a]	<5	Positive	>3.0	NA	NA	

The American Diabetes Association criteria recommend the use of arterial or venous pH for diagnosis and to evaluate the need for bicarbonate therapy and to measure resolution. Note that the severity of diabetic ketoacidosis (DKA) is defined by the degree of acidosis and level of consciousness, not by the degree of hyperglycaemia or ketonaemia. NA, not applicable. [a]Venous pH can be used to diagnose DKA. Data from REFS[130,131].

Figure 1. Diagnostic Criteria for Diabetic Ketoacidosis (DKA) [10]

Physiologically, pregnancy causes changes in the metabolic environment that can increase the risk of DKA even in conditions of mild hyperglycemia. Several mechanisms are thought to be the pathogenesis of DKA in pregnancy. During pregnancy, there is an increase in insulin antagonist hormones such as human placental lactogen, estrogen, and prolactin, which results in a reduction of insulin sensitivity by more than 50%, particularly in the third trimester. Additionally, during the second and third trimesters, there is an increase in relative hunger. This is because the fetus requires more glucose so the mother's fasting glucose becomes lower. In addition to relative insulin insufficiency, there is an increase in lipolysis and the production of ketone bodies, even after a relatively short fasting period.An increase in the human chorionic gonadotropin hormone in early pregnancy can trigger nausea, vomiting, and dehydration resulting in the formation of ketones. Stress, fasting, and dehydration will increase insulin antagonist hormones which increase ketoacidosis. Third, pregnancy also increases minute alveolar ventilation which causes respiratory alkalosis with compensation for increased renal bicarbonate excretion. Thus, the buffer capacity decreases when there is an increase in acids such as ketones [3,11].

This physiological condition can be aggravated by the presence of triggering or precipitating factors, one of the most common of which is infection. Respiratory and urinary tract infections are the most common types of infections. Several other factors that can trigger DKA are emesis, dehydration, inadequate treatment, acute renal insufficiency, acute thrombosis or vascular events, trauma, other endocrine diseases, use of drugs (thiazides, SGLT-2 inhibitors, antipsychotics, steroids), and several other conditions [11]. Research in the United Kingdom in 2021 showed that the most common causes of DKA were infection (21%), emesis (21%), steroid use (13%) and inadequate treatment or poor compliance (10%)[2]. However, other studies have shown that the main triggering factors are non-compliance with treatment (32.3%), followed by gastrointestinal symptoms (14.7%), pump failure (8.8%), infection (7.3%), drug use (2.9%), other causes (11.7%), and unknown (22%)[12].This is following the case where the trigger for DKA in this patient was an infection consisting of respiratory infection and syphilis. The infection will trigger the release of pro-inflammatory cytokines and counter-regulatory hormones such as cortisol or adrenaline which trigger ketoacidosis.

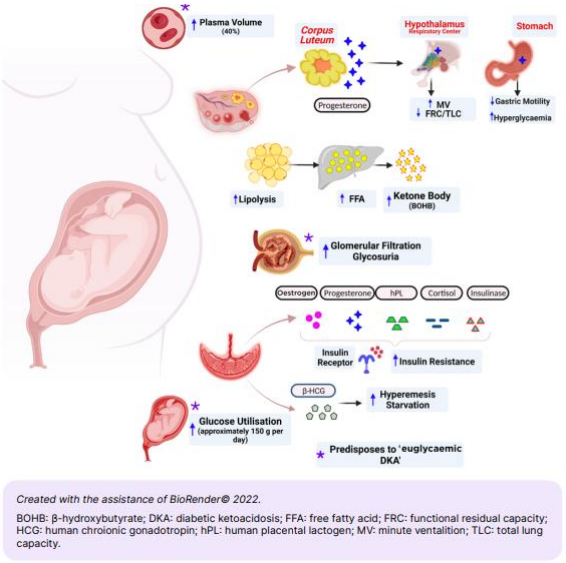

Figure 2. Pathophysiological Mechanism of DKA in Pregnancy [4]

The principles of DKA management include five things, namely: correction of dehydration with fluid resuscitation, correction of hyperglycemia with insulin, correction of acid-base and electrolytes, identification and management of triggering factors, and routine monitoring to prevent complications. In emergency conditions, crystalloid fluid resuscitation is crucial to improve tissue perfusion, correct electrolyte imbalances, and simultaneously reduce blood sugar and counter-regulatory hormone concentrations. In shock patients, 1-2 liters or 15-20 mL/kgBW/hour of 0.9% NaCl infusion can be given for the first hour. If the shock has been treated or the patient's condition is stable, 500 mL can be given for 4 hours, continued with 250 for 4 hours or 250-500 mL/hour until fluids are sufficient. If blood sugar reaches 200-250 mg/dL in DKA, fluids can be changed to D5% infusion 100-200 mL/hour, and insulin drip can be reduced to 1-2 units/hour. This aims to prevent hypoglycemia but still treat DKA by providing enough insulin[8].The patient received 1500 ml of NaCl 0.9% fluid resuscitation followed by 30 drops per minute.

Simultaneously, DKA patients are also given insulin. Contraindications for insulin administration are when the potassium concentration is below 3 mEq/L. Insulin therapy is generally started with an IV bolus of 0.1 unit/kgBW, then continued with an insulin drip starting from 0.05-0.1 unit/kg/hour. The IV insulin drip infusion setting is adjusted according to the results of hourly blood sugar monitoring. The

target for reducing serum blood sugar is 50-75 mg/dL/hour (the decrease is expected not to be too massive, especially in the early stages because it can cause brain edema)[8].After the resolution is achieved and the patient can eat, insulin drip can be replaced with subcutaneous insulin. The criteria for DKA resolution include: blood sugar <200 mg/dl and two of the following criteria: serum bicarbonate levels ≥18 mmol/L, blood pH>7.3, or plasma/capillary ketones <0.6 mmol/L[13].The third management of DKA is electrolyte correction. Generally, patients with DKA will experience mild or moderate hyperkalemia, if hypokalemia is found it is generally associated with a worse prognosis. Insulin administration will cause intracellular potassium shifts and decrease potassium concentrations resulting in severe hypokalemia. Therefore, patients with potassium less than 3.5 mmol/L need to be corrected and insulin administration delayed until above 3.5 mmol/L to prevent arrhythmias, cardiac arrest, and respiratory muscle weakness[8,10]. The patient received insulin drip per protocol starting at 4 units/hour. The patient's potassium level was 5.1 mmol/L so there were no contraindications to insulin administration and no additional KCL therapy was required.

Administration of bicarbonate is still controversial because it does not provide significant benefits and has harmful effects on the mother and fetus such as hypokalemia, worsening intracellular acidosis, delayed keto-anion metabolism, and the development of paradoxical cerebral acidosis. However, several authors and the latest ADA recommendations indicate that bicarbonate can be given to patients with severe acidosis (pH less than 7) or patients with cardiac dysfunction, sepsis or shock. In this case, the patient was given bicarbonate because of the severe acidosis condition. In addition, the patient has been confirmed to have IUFD so the side effects of bicarbonate on the fetus can be ruled out[13,14]. Next is the management related to the triggering factors. In this case, DKA was triggered infection, so the patient was given broad-spectrum antibiotic (cefotaxime 1 gram IV every 8 hours). However, further evaluation and treatment of syphilis will be carried out through the polyclinic. The last is monitoring complications such as symptoms, fluid balance, blood sugar, vital signs, and in cases of pregnancy is monitoring and confirmation of fetal viability. Termination of pregnancy is carried out after the patient is confirmed to have experienced intrauterine fetal death[13,14].

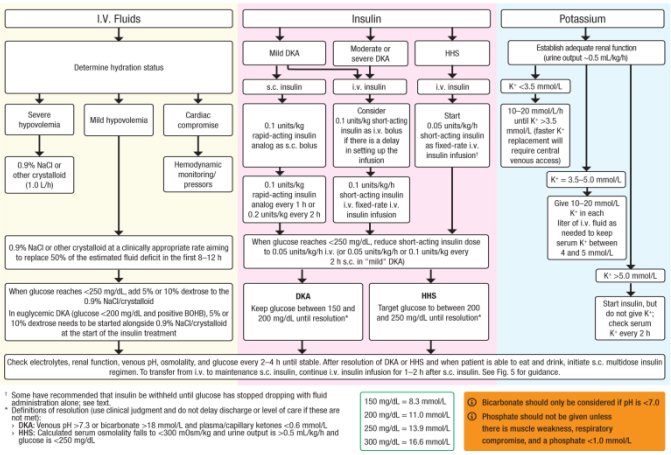

Figure 3. Hyperglycemic Crisis Management Flowchart [13]

DKA can cause maternal complications such as acute kidney failure, respiratory distress syndrome, cerebral edema, coma, and death. Meanwhile, the fetal mortality rate due to DKA ranges from 9% to 36%[11]. Previous studies have shown no maternal deaths but fetal deaths, premature birth, large for gestational age, 5 minutes APGAR Score <7, hypoglycemia, respiratory distress syndrome, jaundice, and infections that require treatment in the neonatal intensive care unit[2].Other studies also showed no maternal deaths but reported miscarriages, stillbirths, and several congenital abnormalities requiring termination. Meanwhile, of the 34 (72%) babies born alive, 27 babies (79%) were born prematurely (<37 weeks)[7]. The mechanism of DKA causing fetal disorders is still unclear. However, several hypotheses have emerged: 1) decreased uteroplacental blood flow due to osmotic diuresis or acidosis resulting in fetal hypoxia, 2) if maternal hypokalemia and severe fetal hyperinsulinemia occur, it will cause myocardial suppression and fatal arrhythmias in the fetus, 3) maternal hypophosphatemia can reduce 2,3-diphosphoglycerate thereby disrupting oxygen delivery to the fetus, 4) fetal hyperinsulinemia will increase fetal oxygen requirements by stimulating the oxidative metabolic pathway[3,10,15,16]. In addition, complications during pregnancy in this patient are further exacerbated by latent syphilis infection. Latent syphilis is a condition in which syphilis is confirmed serologically but does not present with clinical symptoms on the skin or other organs, and has not yet received treatment. This condition is divided into two stages: early latent (occurring less than 1 year, with RPR titer >1:8) and late latent syphilis (occurring more than 1 year, with RPR titer 1:2 or 1:4). Treponema pallidum can cross the placenta and infect the fetus from 9 weeks of pregnancy, although the fetus cannot mount an immunological response before 20 weeks of gestation. Therefore, pathological changes occur after 18-20 weeks of pregnancy. Placental infection and decreased blood flow can lead to fetal death. Pregnant women with untreated latent syphilis may experience the following fetal outcomes: stillbirth, premature, early neonatal death, and congenital syphilis [17,18].

Conclusion

DKA in pregnancy is a rare case but this emergency condition can threaten the lives of the mother and fetus. Multidisciplinary management from internal medicine doctors or endocrinologists, obstetric gynecologists, anesthesiologists, nurses, midwives, and nutritionists is very important. Education, self-monitoring, and pre-pregnancy counseling are ways to prevent DKA in women with DM who are planning a pregnancy.

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Conflict of Interest

The authors declare that there is no conflict of interest.

Informed Consent

Informed consent was obtained prior to performing the procedures, including permission for publication of all data or images included here.

Ethical Statement

No required.

Author Contribution

M.P.A.A designed the project, analyzed the data and wrote the manuscript. D.C.W supervised the project and provided critical feedback.

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