



Northampton
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Unusual presentation of Diabetic ketoacidosis in non-diabetic pregnant woman at 17 weeks' gestation.

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Introduction :

Diabetic ketoacidosis (DKA) is a dangerous medical and obstetric emergency that was once only seen in those with type 1 diabetes. Although it is an uncommon illness, DKA during pregnancy can be harmful to both the mother and the foetus (1). Pregnancy-related metabolic alterations make DKA more likely to develop, although it is rare, DKA can arise during pregnancy even when there is near normoglycaemia. Diabetic patients with type 2 and gestational diabetes mellitus (GDM) have also been documented to experience DKA (2). Modern diabetes management should avoid DKA during pregnancy. Since DKA does not usually appear with the typical presenting symptoms or laboratory findings during pregnancy, a diagnosis can be more difficult. Optimizing maternal and foetal outcomes requires prompt diagnosis and treatment (3,4,5). This case presents an interesting case of DKA occurring in a 17-week pregnant woman with no previous history of diabetes.

Case report :

30-year-old Caucasian women, who was 17 weeks pregnant, gravida 4 para 2, with no antenatal care attended A&E due to a week history of epigastric pain, nausea, vomiting, watery loss, and a day history of per vaginal bleeding on the 30th of December 2022. A private ultrasound scan showed an intrauterine live pregnancy of 16+6-week gestation on 24th December 2022. She had a background of infective endocarditis, mitral valve repair in June 2022, depression, and alcohol abuse. Recently, she was switched to aspirin from warfarin by her cardiologist. There was no history of diabetes or gestational diabetes in her previous pregnancies, or any family history of diabetes noted. Moreover, it was unwanted pregnancy and was also not on any safe contraception.

She presented with vomiting and hematemesis, mild vaginal bleeding, and rupture of membranes. She was tachycardiac with a heart rate of 120bpm, other vitals were a respiratory rate of 20, 100% oxygen saturation on room air, temperature of 36.5°C and a blood pressure of 118/78. Laboratory tests showed white blood cells of 33.5 x10⁹/L (normal value: 4–10 x10⁹/L), Neutrophils 32.29 x10⁹/L (normal value: 1.8–7.4 x10⁹/L), reticulocytes 89x10⁹/L (normal value: 10–100 x10⁹/L), eGFR 71mL/min, creatinine 93µmol/L (normal value: 45–84µmol/L), serum sodium 137 mmol/L(normal value: 133–146mmol/L), serum potassium 5 mmol/L (normal value: 3.5–5.3mmol/L), chloride 109mmol/L and glucose of 15.6. Her serum HbA1c was 31mmol/mol. Venous blood gas showed pH 7.034, glucose 16, lactate 3.1, base excess -25.9 and ketones of 5.6 mmol/L (normal value: 0.6–1.5 mmol/L).

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DKA protocol was commenced with 0.9% sodium chloride maintenance fluids and a fixed rate insulin infusion and antiemetics. venous blood gases were used to monitor the trend of acidosis and glycaemia. Blood cultures were put out on suspicion of sepsis, and meropenem started due to a penicillin allergy. However, cultures were negative.

Blood sugars and ketones normalised within a few hours. The following day she passed her foetus spontaneously and was given Misoprostol for retained products of conception. Diabetic team Review suggested gestational diabetes and the patient was discharged on day 3 with a course of antibiotics and booked for an outpatient diabetic clinic review.

Discussion: Diabetic ketoacidosis (DKA) remains a life threatening complication in pregnancy which is mostly seen in type-1 diabetes. DKA poses a risk to both mother and foetus. However, it is a rare entity, particularly in patients who are not known to have any co-morbidities. Numerous studies have shown that DKA can cause foetal neurological complications and even foetal demise (6).

Prevalence of DKA is higher in pregnant women compared to non-pregnant women (8.9% vs 3.1%) (7). This is due to various physiological changes that occurs during pregnancy. These include Increased insulin resistance from the human placental lactogen, insulin, progesterone, and subsequently increased insulin demand in mid pregnancy, along with reduced glycogen storage (8). Highest insulin resistance is seen in the late second and third trimester, which contradicting in our case report as she developed the DKA in her early second trimester.

Pathophysiology to develop ketosis remains the same in both groups (pregnant vs non-pregnant). However, time taken to develop ketosis is shorter and at a lower blood sugar threshold in pregnant women. Starvation or uncontrolled nausea and vomiting can also elicit ketosis in pregnancy. This could be an explanation into why our patient with no previous risk factors for diabetes but had prolonged episodes of nausea and vomiting developed ketosis. Moreover, she had multiple risk factors that could have trigger the condition. It includes unbooked pregnancy, stress, alcohol abuse, infection and vomiting. Most of them could have prevented by just providing early antenatal care and psychological support for her mental health condition. In addition to that proper selection of contraception method particularly in women with under lying heart disease.

DKA is associated with different maternal and foetal complications, most of them can be prevented with prompt diagnosis and management. Severe complications include maternal mortality (<1%) or foetal demise (9-36%) (9). In this case, we successfully treated the mother but unfortunately the foetus had demised already, and patient eventually spontaneously miscarried. This could be either due to the gestation age, associated anhydramnios due to spontaneous rupture of membrane and underlying foetal academia. Spontaneous rupture of membrane carries good prognosis in foetus with advance gestation age. However, prognosis found to be poor if foetus has not crossed the period of viability. We also suspected sepsis in our patient in view of prolonged leaking, for which the patient was started on broad spectrum antibiotics.

Conclusion

DKA is a life threatening emergency bringing significant risk to both mother and foetus. Prompt diagnosis and treatment are the key factors to reducing associated comorbidities for the mother and unborn child. However, suspicious of developing DKA in non-diabetic mother is quite challenging particularly with a lesser gestational age (<20weeeks). In both situations management plan remains the same which include IV fluids, bicarbonate, and insulin to correct the academia and ketonemia. Our case highlights the importance of suspecting DKA even in a non-diabetic mother with underlying risk factors and associated complications such as sepsis and foetal demise. Additionally, it is also learning point for modern health care workers, how we can prevent it to happen in future by providing contraception counselling, early antenatal care and psychological support to intended mother.

Reference: **1.**Sibai BM, Viteri OA. Diabetic ketoacidosis in pregnancy. Obstet Gynecol. 2014 Jan;123(1):167-178. doi: 10.1097/AOG.0000000000000060. PMID: 24463678. **2.**Dalfrà MG, Burlina S, Sartore G, Lapolla A. Ketoacidosis in diabetic pregnancy. J Matern Fetal Neonatal Med. 2016 Sep;29(17):2889-95. doi: 10.3109/14767058.2015.1107903. Epub 2015 Nov 23. PMID: 26461169. **3.**Long B, Lentz S, Koyfman A, Gottlieb M. Euglycemic diabetic ketoacidosis: Etiologies, evaluation, and management. Am J Emerg Med. 2021 Jun;44:157-160. doi: 10.1016/j.ajem.2021.02.015. Epub 2021 Feb 16. PMID: 33626481. **4.** Eshkoli T, Barski L, Faingelernt Y, Jotkowitz A, Finkel-Oron A, Schwarzfuchs D. Diabetic ketoacidosis in pregnancy - Case series, pathophysiology, and review of the literature. Eur J Obstet Gynecol Reprod Biol. 2022 Feb;269:41-46. doi: 10.1016/j.ejogrb.2021.12.011. Epub 2021 Dec 21. PMID: 34968873. **5.**Mohan M, Baagar KA, Lindow S. Management of diabetic ketoacidosis in pregnancy. The Obstetrician & Gynaecologist. 2017;19(1):55–62. **6.** Jaber JF, Standley M, Reddy R. Euglycemic diabetic ketoacidosis in pregnancy: a case report and review of current literature. Case Rep Crit Care. 2019;2019: 8769714 –5. **7.** Metzger BE, Vileisis RA, Ravnikaar V, Freinkel N. "accelerated starvation" and the skipped breakfast in late normal pregnancy. The Lancet. 1982;319(8272):588–92. **8.** Dargel S, Schleußner E, Kloos C, Groten T, Weschenfelder F. Awareness of euglycaemic diabetic ketoacidosis during pregnancy prevents recurrence of devastating outcomes: A case report of two pregnancies in one patient. BMC Pregnancy and Childbirth. 2021;21(1). **9.** Dhanasekaran M, Mohan S, Egan A. Diabetic ketoacidosis in pregnancy: An overview of pathophysiology, management, and pregnancy outcomes. EMJ Diabetes. 2022.