DIABETIC KETOACIDOSIS TREATMENT

FOR MORE DETAILS OF MANAGEMENT SEE DKA IN PREGNANCY PROTOCOL
TENETS OF MANAGEMENT:

- Aggressive hydration, use normal saline
- IV insulin (may need to give D5 in order to facilitate insulin administration in euglycemic DKA, which is more common in pregnancy)
- Correction of underlying etiology

Fluid resuscitation
- Fetal assessment – Classical teaching is to not intervene while patient is in DKA
- Aggressive hydration, use normal saline
- 1 L in first hour
- Hours 2-4 0.5-1L/hour
- Thereafter: give 250 mL/h 0.45NS until 80% deficit corrected
- Once BG < 300 mg/dL, change IV fluids to D51/2NS and follow intrapartum IV insulin algorithm (Refer to Veciana & Evans 2007.

Insulin
- Loading dose of 0.1-0.4 units/kg
- Maintenance of 2-10 units/hour (start with insulin gtt in labor protocol and adjust as necessary). Double insulin infusion rate if BG does not decrease by 20% in first 2 hours if hyperglycemic
- Continue insulin therapy until bicarbonate/anion gap normalize (serum GB/potassium/anion gap

Potassium replacement
- If K is initially normal or reduced, consider an infusion of K of up to 15-20 mEq/h
- If K is elevated, do not add supplemental K until levels are normal, then 20-30 mEq/L
- Phosphate – consider replacement if serum phosphate < 1.0 mg/DL or cardiac dysfunction present or patient obtunded

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The risk of fetal demise after DKA during pregnancy has decreased over time but remains substantially higher than the baseline risk (2–3%) in women with type 1 diabetes (3). Risks of preterm birth and NICU admissions were also elevated compared with the general population of pregnant women with diabetes (33% [4] and 47% [5], respectively). Factors associated with increased risk of fetal demise were primarily characteristics of the DKA event severity (e.g., maternal ICU admission and higher serum osmolality), lending support to a direct causal relationship. Factors associated with increased risk of preterm birth and NICU admissions, on the other hand, were more indicative of the mother's overall health status and health behaviors. This finding suggests that the observed increased risk of preterm birth among women with DKA during pregnancy could be due to the higher prevalence of risk factors in this population.

DKA during pregnancy poses a risk to the fetus both at the time of the event and following. Further research is needed to identify effective methods for prevention, early recognition, and timely treatment of DKA in pregnancy to mitigate risk of fetal demise and other adverse fetal outcomes.

Diabetes Care 2017;40(7):e77–e79

56

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Among the 77 DKA events in 64 pregnancies in 62 women included in the study, fetal demise, preterm birth, and neonatal intensive care unit (NICU) admissions occurred in 15.6%, 46.3%, and 59% of pregnancies, respectively. Mothers presented in DKA between 5 and 38 weeks of gestation. Fetal demise occurred at the time of or within 1 week of the DKA event and between 1 and 11 weeks afterward in 60% and 40% of cases, respectively.

Maternal ICU admission ($P = 0.024$) and higher serum osmolality ($P = 0.045$) during the DKA event were associated with increased risk of fetal demise (Table 1). Maternal smoking ($P = 0.0005$) and higher pre-DKA HbA_{1c} levels ($P = 0.032$) were associated with higher risk of preterm birth. Maternal smoking ($P = 0.0077$), preeclampsia during pregnancy ($P = 0.031$), higher anion gap during the DKA event ($P = 0.019$), and preterm birth ($P = 0.0003$) were associated with higher risk of NICU admission.

57

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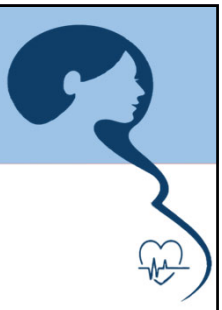
"Euglycemic" DKA: still a metabolic emergency



- "euglycemic DKA" type characterized by metabolic acidosis and increased total body ketone concentration, but with glucose levels ≤250 mg/dL, occurring in approximately 10% of patients with DKA

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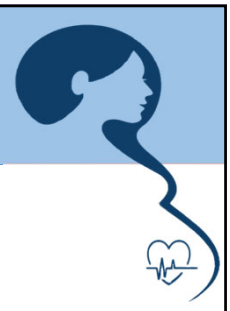
J Clin Endocrinol Metab. 2022 Nov; 107(11): 3137–3143

58

58

ENDOCRINE

Hyperglycemic hyperosmolar syndrome (HHS)



- Findings:
  - altered consciousness, varying from confusion or disorientation to coma
  - extreme dehydration
  - with or without prerenal azotemia,
  - hyperglycemia, and hyperosmolality. In contrast to diabetic ketoacidosis,
  - focal or generalized seizures and transient hemiplegia may occur.
- The fluid deficit can exceed 10 L
- Acute circulatory collapse is a common cause of death.
- Widespread thrombosis is a frequent finding on autopsy and in some cases
- Bleeding may occur as a consequence of disseminated intravascular coagulation
- Maternal mortality up ***to 20%***
- FLUIDS: Normal saline, potassium; no insulin until osmolality stops falling
- Remember to correct serum sodium for serum glucose by adding 1.6 mEq/L (1.6 mmol/L) for each 100 mg/dL (5.6 mmol/L) elevation of serum glucose over 100 mg/dL (5.6 mmol/L)
- Target rate of change of sodium is no faster than 0.5 mEq/hour
- Target reduction of serum glucose is no faster than 90 mg/dl/hour
- Reduction in serum osmolality target range is 3.0–8.0 mOsm/kg/h, using fluids and judicious insulin
- Too rapid reduction in osmolality risks cerebral edema and osmotic demyelination

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Brutsaert EF. HHS. MD Manuals, see references *Mustafa OG,et al. Diabet Med. 2023 Mar; 40(3): e15005*

59

59

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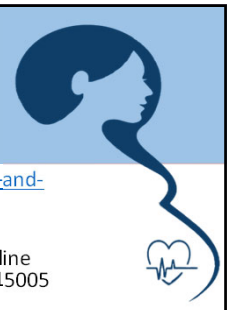

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60

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61

61

Thank you for all your support over the years

"In this life, everything has a beginning and an end. And I think it's an appropriate time to put an end to a career that has been long and much more successful than I could have ever imagined." *Rafael Nadal 2024*

"The names of the patients whose lives we save can never be known. Our contribution will be what did not happen to them. And, though they are unknown, we will know that mothers and fathers are at graduations and weddings they would have missed, and that grandchildren will know grandparents they might never have known, and holidays will be taken, and work completed, and books read, and symphonies heard, and gardens tended that, without our work, would never have been."
Don Berwick, MD,2004 IHI's 16th Annual National Forum on Quality Improvement in Health Care

62

62