



THE UNIVERSITY OF
CHICAGO
MEDICINE &
BIOLOGICAL
SCIENCES

“39 year old pregnant woman
with ketosis”



39 year old pregnant woman with ketosis

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JUNE 11, 2020

Objectives

Discuss DKA in pregnancy

Discuss accelerated starvation

Discuss euglycemic DKA and its causes

Discuss euglycemic DKA in pregnancy

39 year old G4P1112 at 30w6d and recently diagnosed diabetes presents with

- 6-7 day history of shortness of breath and cough
- 2-3 day history of decreased appetite
- Subjective fevers at home
- Denies nausea or vomiting
- Has not taken insulin for the last 2-3 days

Initial Presentation

Other History

Past Medical History:

Diagnosed with diabetes mellitus at 8 weeks gestation

Gestational diabetes during 1st and 2nd pregnancy

Morbid obesity

Past Surgical History:

Right sided salpingectomy and oophorectomy in 2018

Social History:

- Married with 2 children
- Denies tobacco, alcohol, and illicit substances

Family History:

- Father, paternal aunt and grandparents – type 2 diabetes

Other History

Medications:

- Aspirin 81mg
- Prenatal vitamin
- Lantus 35 units daily
- Humalog 8 units with meals

Allergies: NKDA



Review of Systems:

Constitutional: **+fevers, decreased appetite**; No chills, activity change, weight change

HEENT: No visual disturbance, hearing loss, congestion, sore throat, neck pain

Resp: **+cough, shortness of breath**

CV: **+palpitations**; No chest pain, LE edema.

GI: No nausea or vomiting. No abdominal pain, d/c, or blood in stool.

MSK: No myalgias.

Skin: No rashes or ulcers.

Neuro: No seizures, syncope, headache, lightheadedness

Endo: No heat/cold intolerance. No hair/skin changes noted.

Heme: No adenopathy

Psych: Mood stable

Physical Exam:

Vitals: 116 kg, BMI 44, Temp 97.9, **HR 110**, RR 20, BP 125/74, **SpO2 99% on 2L O2 NC**

General: No apparent distress. Appears stated age.

HEENT: No pharyngeal erythema. PERRL, EOMI.

Neck: No neck tenderness. No thyromegaly or thyroid nodules appreciated.

Cardiovascular: **tachycardic**; regular rhythm. No peripheral edema.

Pulmonary/Chest: nonlabored respirations; **scattered wheezes**

Gastrointestinal: **obese, gravid**, nontender

Musculoskeletal: normal range of motion of joints.

Neurological: Alert & oriented, no focal deficits

Skin: No apparent bald spots. No acanthosis nigricans.

Psychiatric: normal mood, thought content, appropriate.

Initial Labs and Imaging

Glucose, Ser/Plasma	129 *	▲
Sodium	134	
Potassium, Ser/Plasma	3.2	▼
Chloride	104	
Carbon Dioxide	13	▼
Anion Gap	17	▲
BUN	3	▼
Creatinine	0.4	▼
eGFR, Non-African ...	129 *	
eGFR, African Amer...	149 *	
Calcium	9.2	
Inorganic Phosphate		
Magnesium		
Total Protein	6.7	
Albumin	3.5	
Bilirubin, Total	0.7	
Bilirubin, Conjugated		
Bilirubin, Unconju...		
Alk Phos, Serum	101	
AST (SGOT)	22	
ALT (SGPT)	17	

Beta-Hydroxybutyrate	2.74	▲
Lactic Acid	0.9	
ARTERIAL BLOOD GAS		
FIO2	21	
pH (Arterial)	7.34	▼
PCO2 (Arterial)	21	▼
PO2 (Arterial)	88	
HCO3- (Arterial)	11.3	▼
Base Excess (Arter...	MINUS 12 *	
SO2 (Arterial)	99.2 *	▲
Temperature	37.0	
pH (Temp Corr Art)	7.34	▼
PCO2 (Temp Corr Art)	21	▼
PO2 (Temp Corr Art)	88	Re
Hemoglobin	10.1	▼
Oxyhemoglobin	96.7 *	
Deoxyhemoglobin	<1.0	▼
Carboxyhemoglobin	1.3	
Methemoglobin	1.2	

A1c 6.0%

A1c 6.7% at diagnosis

SARS-CoV 2 RNA - POSITIVE

Chest x-ray - bilateral opacities present

CT chest – ground glass opacities bilaterally



Hospital Course

Covid positive

- Respiratory status remained stable during hospital stay
- Required supplemental oxygen the first day of admission
- On subsequent days, O2 sats >95% on room air

Anion gap metabolic acidosis

- Initially ketosis thought to be from starvation ketosis
- Appetite much improved on day 1, back to normal by day 2



	4/16/2020 0554	4/16/2020 1013	4/16/2020 1156	4/16/2020 1410	4/16/2020 2327	4/17/2020 0430	4/17/2020 1429	4/17/2020 1826
Carbon Dioxide	13 ▾	12 ▾		13 ▾	13 ▾	13 ▾	15 ▾	16 ▾
Anion Gap	17 ▲	18 ▲		15	17 ▲	18 ▲	17 ▲	15
Beta-Hydroxybutyrate	2.74 ▲	2.94 ▲		1.71 ▲	2.41 ▲	2.65 ▲	1.54 ▲	0.41 ▲



Ketosis persisted, despite appetite being back to normal and patient eating 100% of her meals and being restarted on insulin

	4/18/2020 0446	4/18/2020 0446	4/18/2020 1253	4/18/2020 1833	4/19/2020 0334	4/19/2020 0334	4/19/2020 0746	4/19/2020 1214
Carbon Dioxide	17 ▾	16 ▾	19 ▾	19 ▾	17 ▾	17 ▾	17 ▾	19 ▾
Anion Gap	15	15	12	12	14	13	14	14
Beta-Hydroxybutyrate	1.51 ▲	1.49 ▲	0.34 ▲	0.10	0.59 ▲	0.59 ▲	0.23	0.10



Patient was transferred to ICU for insulin gtt given concern for euglycemic DKA



Transitioned off insulin gtt to lantus 35 units daily and Humalog 8 units with meals

	4/19/2020 2000	4/20/2020 0450	4/20/2020 1701	4/21/2020 0718	4/21/2020 1814	4/22/2020 0945	4/22/2020 1426
Carbon Dioxide	18 ▾	18 ▾	18 ▾	18 ▾	17 ▾	17 ▾	18 ▾
Anion Gap	15	13	14	16 ▲	16 ▲	15	14
Beta-Hydroxybutyrate	0.76 ▲	0.97 ▲	0.30 ▲	1.00 ▲	1.03 * ▲	0.57 ▲	0.46 ▲



Blood sugars ranged 80s to 160s throughout hospital stay

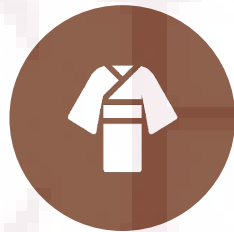
Patient discharged



DKA in pregnancy



CONSIDERED A MEDICAL AND
AN OBSTETRIC EMERGENCY



OCCURS IN APPROXIMATELY 1
TO 3 PERCENT OF PREGNANT
WOMEN WITH DIABETES



HIGHER INCIDENCE IN THE
SECOND AND THIRD
TRIMESTERS



INCREASED INSULIN
RESISTANCE AND
ACCELERATED KETOSIS
PREDISPOSE THE PREGNANT
WOMAN WITH DIABETES TO
DKA



ASSOCIATED WITH RISKS TO
BOTH THE MOTHER AND THE
FETUS



Incidence

Table 1. Incidence of diabetic ketoacidosis in pregnancy

	Time Interval	Incidence, % (No.)	Perinatal Mortality Rate, % (No.)
Lufkin et al. (1)	1950–1979	7.9 (18/228)	27.8 (5/18)
Kilvert et al. (2)	1971–1990	1.7 (11/635)	22
Montoro et al. (3)	1972–1987	3.9 (22/560)	35 (7/20)
Chauhan et al. (4)	1976–1981	22	35
	1986–1991	3	10
Cullen et al. (5)	1985–1995	2 (11/520)	9 (1/11)

Crit Care Med 2005 Vol. 33, No. 10 (Suppl.)

Precipitating factors

Infection

Inadequate insulin management or inappropriate insulin cessation

Acute illness

Stress

Dehydration

Intractable vomiting

β -sympathomimetic use

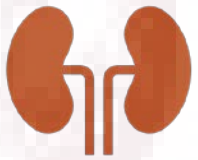
Steroid administration for fetal lung maturation

Montoro et al. - 15-year series of cases of DKA in pregnancy,

- 40% of cases due to cessation of insulin therapy
- 20% of cases due to infection
- 30% of cases were previously undiagnosed diabetes



Physiologic changes in pregnancy



Compensated primary respiratory alkalosis

pH is 7.43, PCO₂ is 30 mm Hg, and bicarbonate is 19–20 mEq/L.

Pregnant woman more susceptible to metabolic acidosis, particularly DKA



Relative insulin resistance, enhanced lipolysis, elevated free fatty acids, and ketogenesis

Ketone bodies can be seen in the serum and urine of normal pregnant women throughout the antepartum period



Prone to develop more severe and rapidly progressive episodes of DKA and at lower glycemic levels after 20 weeks of gestation



Physiologic changes in pregnancy

Human placental lactogen, progesterone, and cortisol, impair the action of maternal insulin and further contribute to a diabetogenic state

- Progesterone decreases the gastrointestinal motility and enhances the carbohydrate absorption which promotes hyperglycemia

Insulinase from the placenta depletes maternal insulin

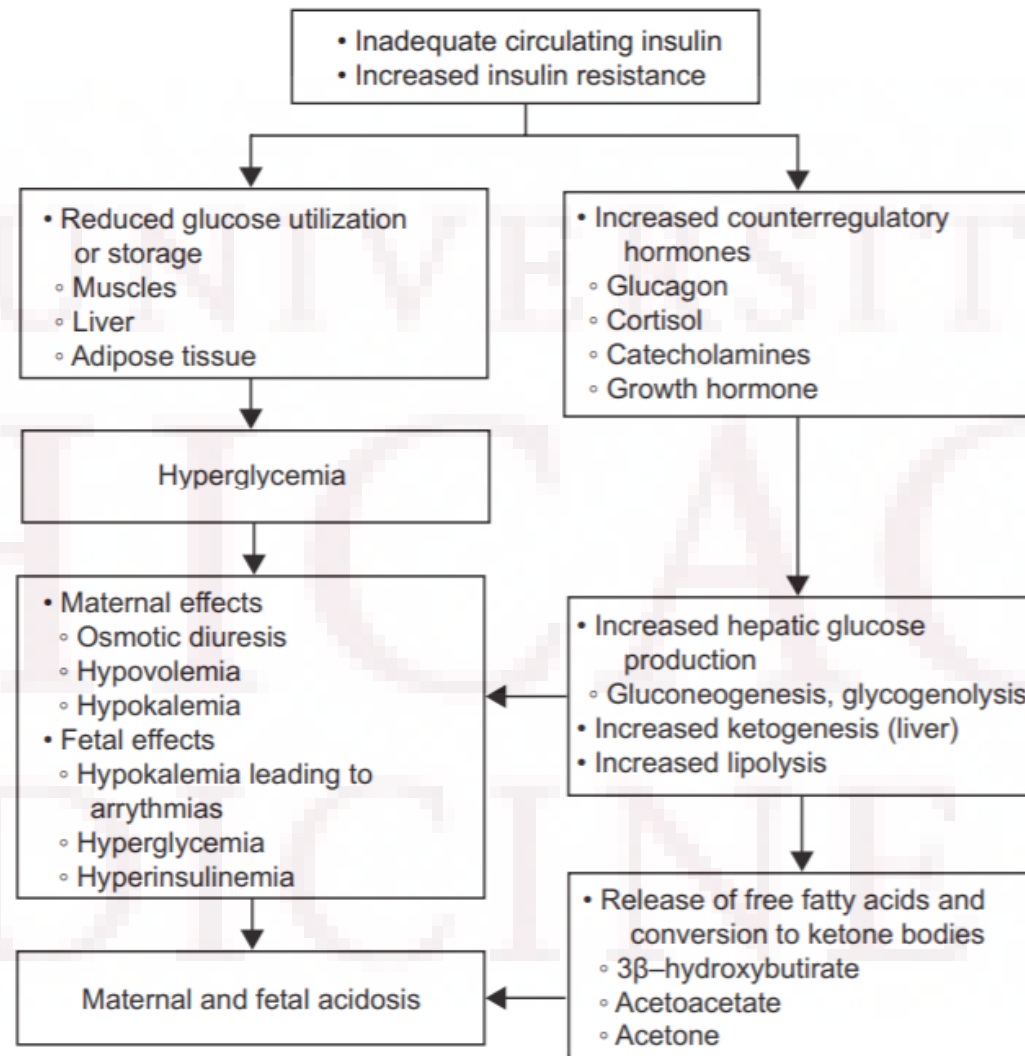


Fig. 1. Pathophysiology of diabetic ketoacidosis in pregnancy. Modified from Sibai BM. Management of acute obstetrics emergencies: female pelvic surgery video, atlas series. 1st ed. Philadelphia (PA): Saunders, Elsevier; 2011. p. 140.

Sibai. *Diabetic Ketoacidosis in Pregnancy.* Obstet Gynecol 2014.

Maternal concerns

Considered a medical emergency

Severe dehydration can lead to hypotension

Acidosis can cause organ dysfunction

Electrolyte imbalance can cause cardiac arrhythmias

Maternal mortality related to DKA is <1 percent



Fetal outcomes

Morrison et al. Retrospective cohort study of pregnant women with type 1 diabetes and at least one episode of DKA between 1996 and 2015 at one of three teaching hospitals in Boston

- 77 DKA events in 64 pregnancies in 62 women
- Mothers presented in DKA between 5 and 38 weeks of gestation
- Fetal demise occurred in 15.6% of the pregnancies
- Preterm birth occurred in 46.3% of the pregnancies
- Neonatal intensive care unit (NICU) admissions in 59% of the pregnancies



Fetal concerns

The same metabolic disturbances that occur during maternal DKA are induced in the fetus

Maternal acidemia decreases uterine blood flow -> decrease in placental perfusion leading -> decreased fetal oxygen delivery

Fetal oxygen delivery is further compromised by a shift of the maternal oxyhemoglobin dissociation curve caused by acidemia

Fetal hypokalemia may lead to fetal arrhythmias and cardiac arrest

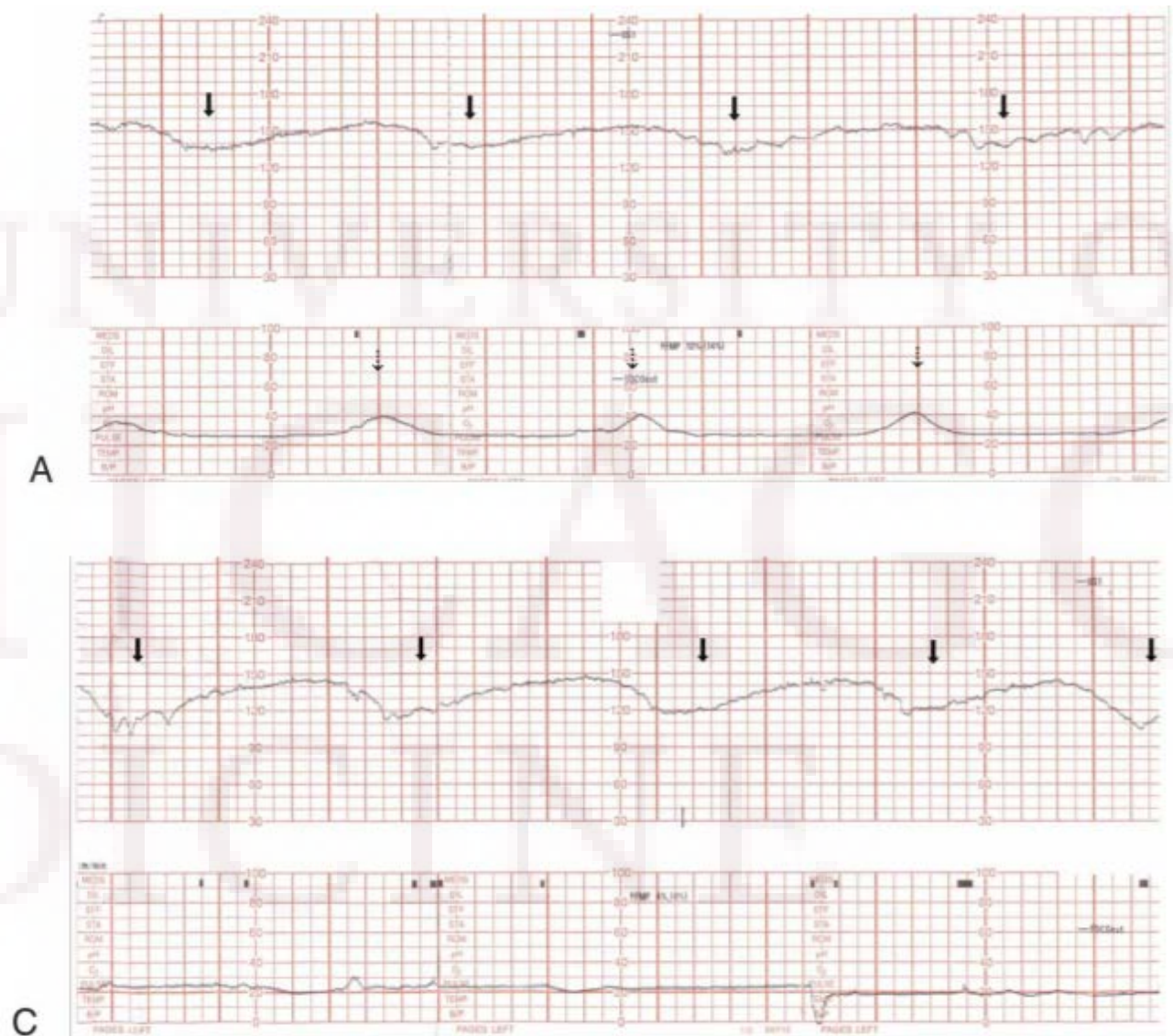
Severe acidosis results in decreased fetal heart rate variability

Maternal phosphate deficits may contribute to fetal hypoxia

Maternal hyperglycemia results in fetal hyperglycemia and fetal osmotic diuresis and volume depletion

Fetal heart rate tracings in pregnant women with DKA can show:

- Decreased or absent variability
- Absent accelerations
- Late decelerations



Sibai et al. OBSTETRICS & GYNECOLOGY. 2014



Long term effects

Effects of DKA during pregnancy have not been well studied

Some studies have shown an association between the presence of ketosis in pregnant women with diabetes and lower IQ scores

An association has been reported between ketonuria detected during prenatal visits and adverse neurobehavioral outcomes even in nondiabetic pregnancies

Acidemia has been associated with poor myelination and poor cortical connectivity

- These findings are associated with greater deficits in expressive language among children diagnosed with autism spectrum disorder 96



Treatment

DKA is treated similarly in pregnant and nonpregnant patients. This includes:

- Administration of insulin
- Replacement of intravascular volume
- Repleting electrolytes

DKA alone is generally not an indication for delivery

The timing of delivery should be individualized based on gestational age, maternal condition, fetal condition

If fetal condition does not improve or if the maternal condition continues to deteriorate despite aggressive therapy, emergency delivery may be warranted



Accelerated starvation

Term coined by Norbert Freinkel in 1965

Exaggerated response to fasting with increased ketosis compared to non-pregnant women

- Lipolysis and ketogenesis are simulated during pregnancy by pregnancy related hormones

Has been observed with overnight fasting or when feeding is delayed by only a few hours

One study showed maternal ketone levels during fasting are elevated by 33% during the third trimester as compared with the postpartum state (Sibai et al)

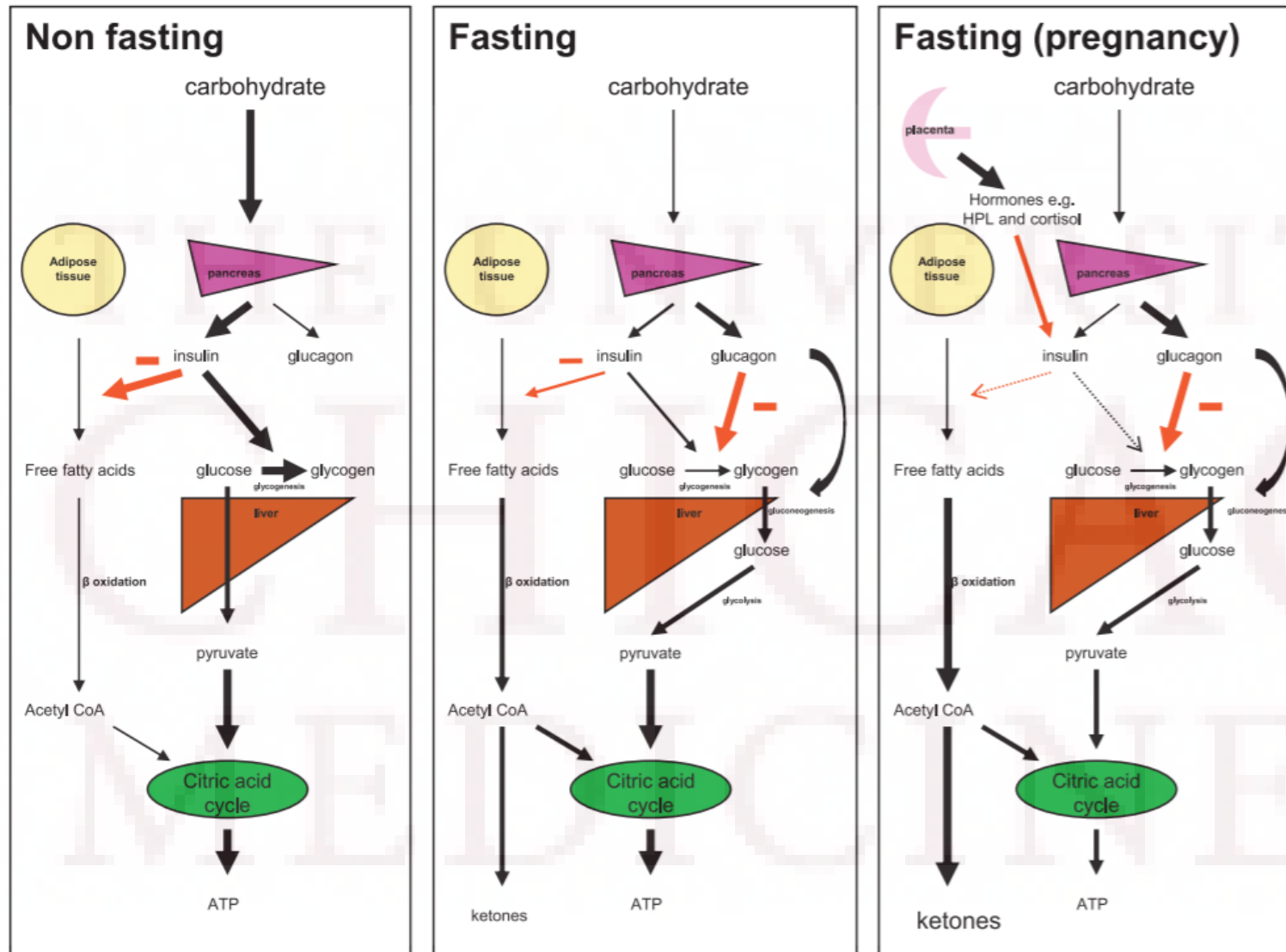


Fig. 1. Metabolic changes in the fasting and non-fasting state.



BUCHANAN ET AL. "ACCELERATED STARVATION IN LATE PREGNANCY: A COMPARISON BETWEEN OBESE WOMEN WITH AND WITHOUT GESTATIONAL DIABETES MELLITUS." 1990.

METZGER ET AL. ACCELERATED STARVATION IN PREGNANCY: IMPLICATIONS FOR DIETARY TREATMENT OF OBESITY AND GESTATIONAL DIABETES MELLITUS. 1987.

Pregnant women in third trimester of pregnancy

- Obese women with normal glucose tolerance (n = 10)
- Age- and weight-matched women with gestational diabetes mellitus (n = 10)

3-Hydroxybutyrate levels were similar in the two groups at 12 hour and 18 hour fasts

Pregnant women with and without gestational DM

14 hour and 18 hour of dietary deprivation

No significant difference between the two groups in terms of ketosis

Montelongo et al.

12 age-matched PGDM women, 9 GDM women, and 12 healthy control subjects

1st, 2nd, and 3rd trimester of gestation and at postpartum and post-lactation

FFA and 3-hydroxybutyrate levels were higher in both PGDM and GDM patients than in control subjects during gestation but not after postpartum

TABLE 3
Plasma lipidic components in normal and diabetic women during pregnancy, postpartum, and postlactation

	FFA (μM)	β -OH-butyrate (μM)	TG (mM)	Cholesterol (mM)
Normal control subjects				
1st trimester	353.1 \pm 49.1 (a)	74.8 \pm 36.6 (a)	0.68 \pm 0.10 (a)	4.48 \pm 0.18 (a)
2nd trimester	328.2 \pm 35.4 (a)	76.4 \pm 11.3 (a)	1.17 \pm 0.10 (b)	6.02 \pm 0.21 (b)
3rd trimester	314.1 \pm 33.9 (a)	110.6 \pm 43.6 (a)	2.03 \pm 0.26 (c)	6.69 \pm 0.36 (c)
Postpartum	355.5 \pm 44.1 (a)	84.1 \pm 33.6 (a)	0.95 \pm 0.18 (ab)	5.86 \pm 0.33 (b)
Postlactation	319.6 \pm 36.0 (a)	78.1 \pm 21.8 (a)	0.62 \pm 0.06 (a)	4.81 \pm 0.35 (a)
PGDM women				
1st trimester	599.8 \pm 83.2 (a)*	455.3 \pm 114.5 (a)†	0.58 \pm 0.05 (a)	4.18 \pm 0.16 (a)
2nd trimester	483.8 \pm 62.2 (a)*	378.1 \pm 115.3 (a)*	1.22 \pm 0.10 (b)	5.81 \pm 0.36 (b)
3rd trimester	545.1 \pm 54.8 (a)†	366.5 \pm 95.5 (a)*	1.92 \pm 0.20 (c)	6.21 \pm 0.45 (b)
Postpartum	554.6 \pm 120.5 (a)	313.3 \pm 126.6 (a)	0.85 \pm 0.08 (d)	6.09 \pm 0.40 (b)
Postlactation	457.1 \pm 107.3 (a)	566.8 \pm 204.9 (a)	0.68 \pm 0.09 (acd)	4.82 \pm 0.20 (ab)
GDM women				
1st trimester	642.4 \pm 63.9 (a)‡	444.9 \pm 97.9 (a)‡	0.87 \pm 0.10 (a)	4.67 \pm 0.37 (a)
2nd trimester	474.6 \pm 38.5 (bd)*	370.3 \pm 116.8 (ab)†	1.54 \pm 0.22 (b)	5.90 \pm 0.35 (ab)
3rd trimester	499.6 \pm 36.3 (ab)†	348.3 \pm 101.3 (ab)*	2.06 \pm 0.23 (c)	6.30 \pm 0.33 (b)
Postpartum	319.7 \pm 30.9 (c)	50.0 \pm 7.3 (c)	1.05 \pm 0.15 (ab)	6.07 \pm 0.35 (ab)
Postlactation	371.9 \pm 23.8 (d)	49.5 \pm 11.6 (abc)	0.99 \pm 0.23 (abc)	5.46 \pm 1.39 (ab)

Values are means \pm SE. Letters in parenthesis correspond to statistical comparison by Student's *t* or postgestational stages studied: the same letter within one parameter means no statistical difference between the groups, whereas different letters indicate significant differences between the corresponding groups ($P \leq 0.05$). Statistical comparisons of PGDM or GDM women versus control subjects were calculated by Student's *t* independent test.

* $P < 0.05$.

† $P < 0.01$.

‡ $P < 0.001$.



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Beta-Hydroxybutyrate	2.74 ▲	2.94 ▲		1.71 ▲	2.41 ▲	2.65 ▲	1.54 ▲	0.41 ▲



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Anion Gap	15	13	14	16 ▲		16 ▲	15
Beta-Hydroxybutyrate	0.76 ▲	0.97 ▲	0.30 ▲	1.00 ▲		1.03 * ▲	0.57 ▲



Characterized by metabolic acidosis, ketosis, blood glucose levels less than 200 mg/dl

Compared to DKA, insulin deficiency and insulin resistance are milder with increased renal glucose clearance

The underlying mechanism may be either due to decreased hepatic production of glucose during the fasting state or increased urinary excretion of glucose caused by an excess of counter-regulatory hormones

Euglycemic DKA

Causes of euglycemic DKA

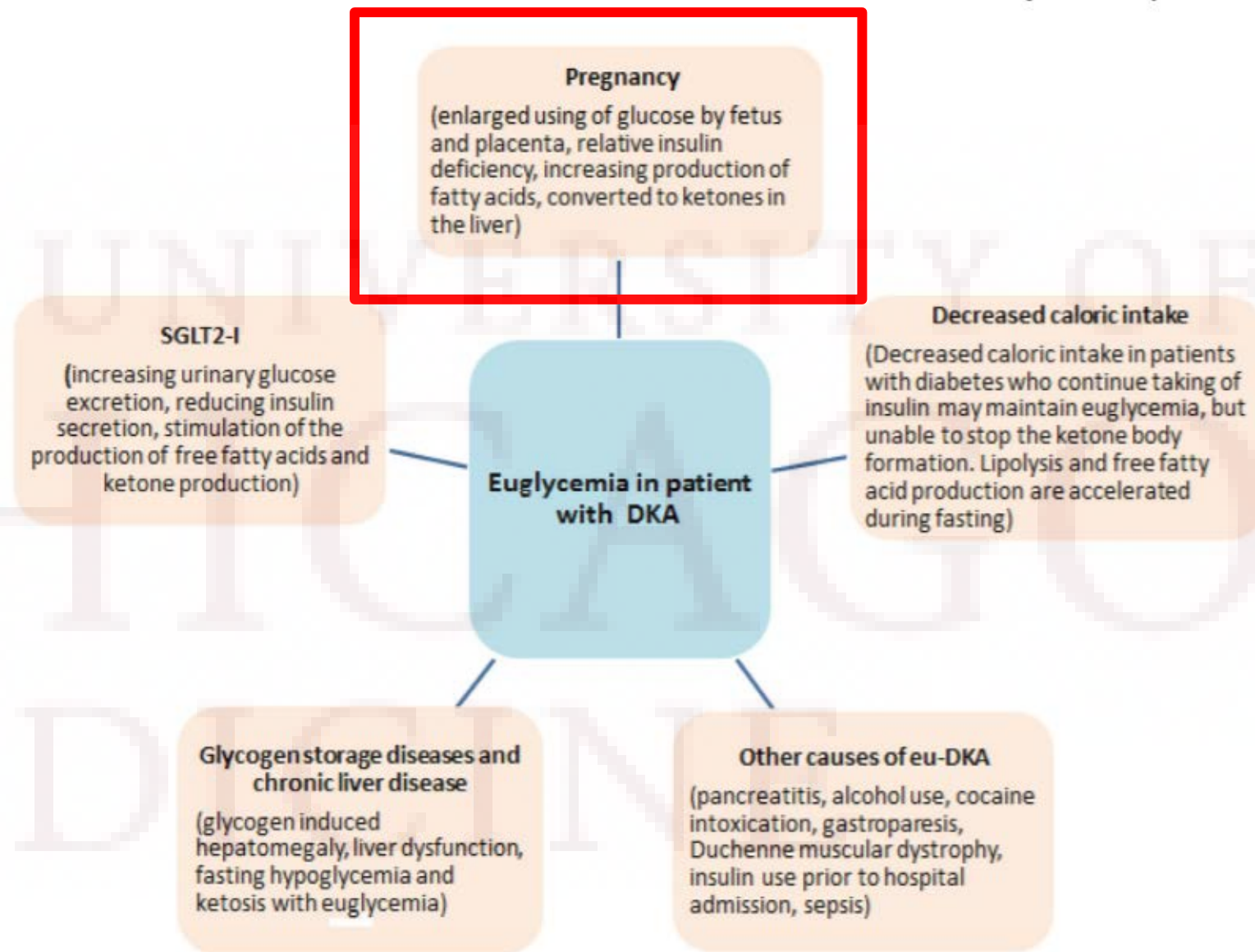


Fig. 2. Possible etio-pathogenetic mechanisms of euglycemic DKA.

Euglycemic DKA in pregnancy

Cullen et al

- 520 pregnant patients with diabetes hospitalized between 1985 and 1995
- 11 episodes (2%) of DKA were diagnosed
- 4 out of 11 had plasma glucose <200mg/dL (36%).

10 to 30 percent of cases of DKA in pregnancy have been observed with blood glucose levels <250 mg/dL (Whiteman et al)

Cases reported in type 1 and 2 diabetes, gestational diabetes



PERINATAL/NEONATAL CASE PRESENTATION

Normoglycemic diabetic ketoacidosis in pregnancy

M Chico¹, SN Levine¹ and DF Lewis²

Case Report

Euglycemic Diabetic Ketoacidosis in Pregnancy: A Case Report and Review of Current Literature

Johnny F. Jaber ¹, Matthew Standley,¹ and Raju Reddy ²

Case Report

Euglycemic Diabetic Ketoacidosis in Pregnancy

Nauman Tarif*, Wisam Al Badr**

Department of Medicine, *King Khalid University Hospital, Riyadh,
**King Faisal Specialist Hospital, Riyadh

Case Reports > J Reprod Med. Sep-Oct 2012;57(9-10):452-5.

Diabetic Ketoacidosis Occurring With Lower Blood Glucose Levels in Pregnancy: A Report of Two Cases

Monika Madaan ¹, Kiran Aggarwal, Ritu Sharma, Shubha Sagar Trivedi



Cases of euglycemic DKA in pregnancy

TABLE 1: Summary of literature describing cases of euglycemic diabetic ketoacidosis in pregnant mothers.

Authors	Age (years)	Diabetic history	Gestational age	Admission blood glucose (mg/dL)	Outcome of mother	Outcome of fetus
Bryant et al. [18]	Describes three cases of diabetic ketoacidosis in pregnant patients with admission glucose <200 mg/dL. No specifics about the three cases are given					
Cardonell et al. [19]	33	Type 2 diabetes mellitus	Third trimester, 35 weeks	134	Discharged home	Emergent C-section at 35 weeks
Chico et al. [14]	29	Type 1 diabetes mellitus	Third trimester, 34 weeks	87	Discharged home	Nonemergent C-section at 34 weeks
Clark et al. [20]	34	Gestational diabetes	Third trimester, 36 weeks	140	Discharged home	Nonemergent C-section at 36 weeks
Cullen et al. [21]	Describes four cases of diabetic ketoacidosis in pregnant patients with admission glucose <200 mg/dL. No specifics about the four cases are given					
Darbhamulla et al. [22]	30	Gestational diabetes	Third trimester, 33 weeks	95	Discharged home	Elective C-section at 39 weeks
Franke et al. [15]	23	Gestational diabetes	Third trimester, 32 weeks	127	Discharged home	Delivery at 38 weeks
Frise et al. [23]	40	Gestational diabetes	Third trimester, 35 weeks	52–85	Discharged home	Emergent C-section at 35 weeks
Guo et al. [4]	29	Unknown	Third trimester, 32 weeks	124	Discharged home	Delivery at 38 weeks
Kamalakannan et al. [24]	28	Type 1 diabetes mellitus	Third trimester, 36 weeks	234	Discharged home	Intrauterine fetal demise
Karpate et al. [25]	25	Unknown	Third trimester, 37 weeks	103	Discharged home	Delivery at 37 weeks
Lucero and Chapela [13]	22	Type 1 diabetes mellitus	First trimester, unknown weeks	153	Discharged home	Unknown
Madaan et al. [26]	30	Type 2 diabetes mellitus	Third trimester, 36 weeks	75–155	Discharged home	Elective C-section at 38 weeks
Madaan et al. [26]	23	Gestational diabetes	Third trimester, 34 weeks	89–164	Discharged home	Emergent C-section at 37 weeks
Montoro et al. [27]	Describes two cases of diabetic ketoacidosis in pregnant patients with admission glucose <200 mg/dL. No specifics about the two cases are given					
Napoli et al. [16]	26	Type 1 diabetes mellitus	Third trimester, 34 weeks	211	Discharged home	Elective C-section at 34 weeks
Oliver et al. [28]	29	Type 1 diabetes mellitus	Third trimester, 28 weeks	245	Discharged home	Elective C-section at 34 weeks
Rivas et al. [29]	39	Gestational diabetes	Third trimester, 32 weeks	120	Discharged home	Emergent C-section at 32 weeks
Tarif and Al Badr [30]	37	Type 2 diabetes mellitus	Third trimester, 35 weeks	77	Discharged home	Unknown
Yu et al. [31]	30	Type 2 diabetes mellitus	Third trimester, 28 weeks	121	Discharged home	Elective C-section at 36 weeks

Back to this patient

This patient likely had starvation ketosis initially with accelerated starvation/ketosis with fasting every morning

Blood sugars well controlled

Now 38 weeks pregnant

Followed by MFM, Endo as outpatient

References

Uptodate, Pregestational (preexisting) diabetes mellitus: Glycemic control during pregnancy

Uptodate Critical illness during pregnancy and the peripartum period

Uptodate Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis

Uptodate Pregestational (preexisting) diabetes mellitus: Obstetric issues and management

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