Diabetic ketoacidosis (DKA)

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Definition of DKA: (ISPAD Guidlines 2018 and BSPED)

1- Hyperglycemia (BG> 200mg /dl)

2- Venous PH < 7.3 or serum HCO3 <15mmol/L.

3- Ketonemia or ketonuria

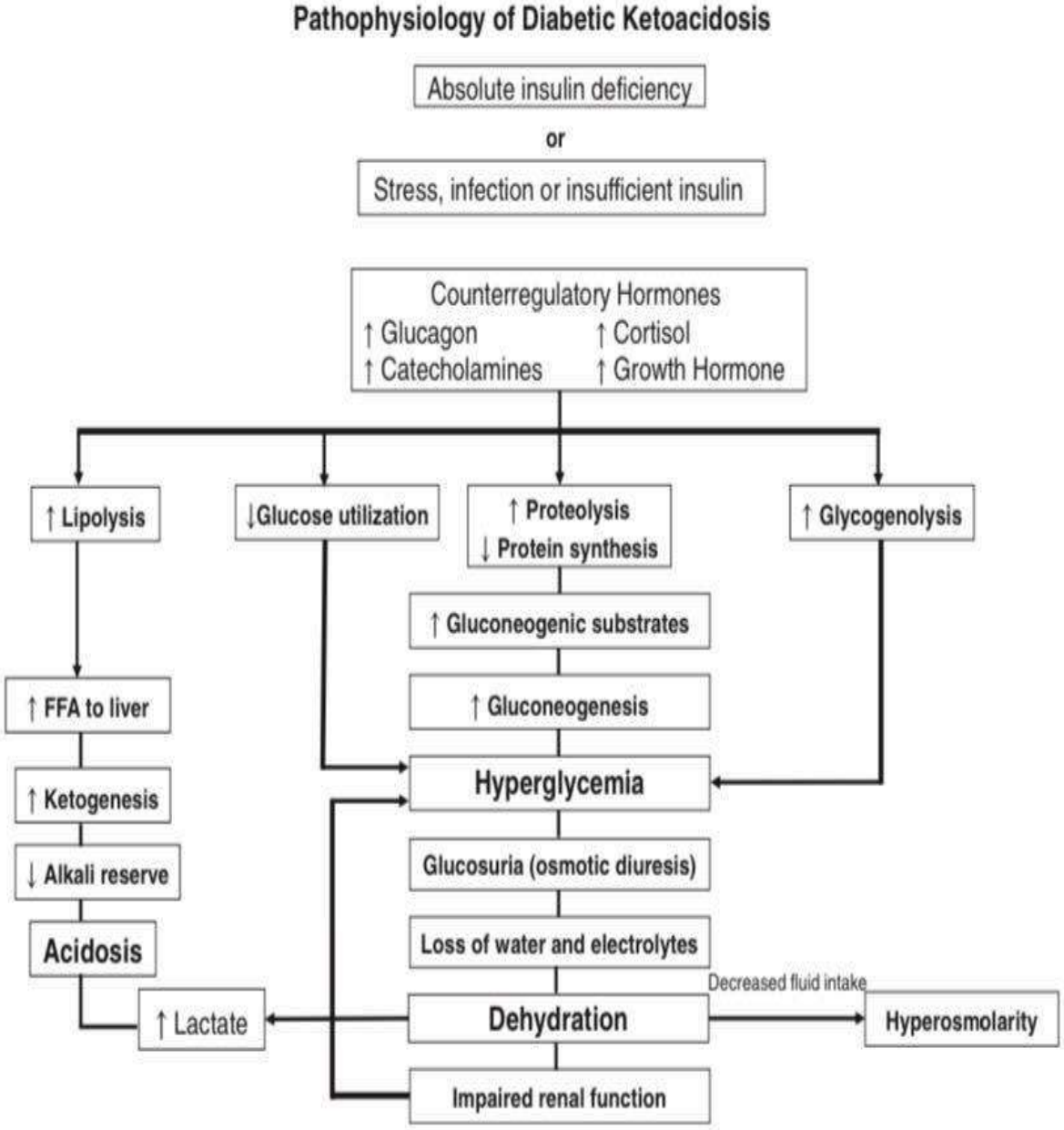

FIGURE 1 Pathophysiology of diabetic ketoacidosis. Copyright© 2006 American Diabetes Association. From diabetes care, Vol. 29, 2006:1150-1159. Reprinted with permission of *The American Diabetes Association*

* SEVERITY OF DKA

1-Mild : vPH less than 7.3 or serum HCO3 less than 15 mmol/L , assume 5% dehydration

2-Moderate: PH less than 7.2 , serum HCO3 less than 10 mmol/L , assume 7% dehydration

3-Severe : PH less than 7.1 , serum HCO3 less than 5 mmol/L , assume 10% dehydration

Frequency of DKA:

The risk of DKA in established T1D is 1% to 10% per patient per year.

- Risk is increased in:

- Children who omit insulin
- Children with poor metabolic control or previous episodes of DKA
- Gastroenteritis with persistent vomiting and inability to maintain hydration
- Children with psychiatric disorders, including those with eating disorders

Cont.

- Children with difficult or unstable family circumstances (eg, parental abuse)
- Peripubertal and adolescent girls
- Binge alcohol consumption
- Children with limited access to medical services

Clinical manifestations of DKA:

- Nausea, vomiting, and abdominal pain that may mimic an acute abdominal condition
- Dehydration
- Tachypnea; deep, sighing (Kussmaul) respiration (acetone)
- Confusion, drowsiness, progressive obtundation, and loss of consciousnes

*Managment of DKA :

* In emergency room

* In pediatric ICU

In emergency department

1- immediate BG and BOHB by urine dispstick if available

2- Weigh the pt

3- Assess the degree of dehyration

4-Assess level of consciousness (Glasgow coma scale)

5- Give oxygen to patients with circulatory impairment or shock.

6- A cardiac monitor should be used for continuous ECG monitoring to assess T-waves for evidence of hyper- or hypokalemia.

7- Insert IV access for blood sampling and management

8- Obtain blood sample

9- Give bolus 0.9% saline 10ml/kg over 1h

Clinical and biochemical monitoring:

1- hourly vital sign

2- hourly level of consciousness assessment

3- Amount of administered insulin

4- hourly fluid input and output

5- hourly glucocheck and 2 hourly VBG

6- every 2-4 h serum electrolyte and KFT

7-measure body weight every morning

8- calculate the anion gap

Steps of managment

-Goals of therapy:

- **Correct acidosis and reverse ketosis**
- **Correct dehydration**
- **Restore blood glucose to near normal**
- **Monitor for complications of DKA and its treatment**
- **Identify and treat any precipitating events**

Resuscitation fluid :

- For patients who are volume depleted but not in shock, volume expansion (resuscitation) should begin immediately with 0.9% saline to restore the peripheral circulation.

- The volume administered typically is 10 mL/kg infused over 30 to 60 minutes

- patients with DKA in shock, rapidly restore circulatory volume with isotonic saline in 20 mL/kg boluse infused as quickly as possible through a large bore cannula with reassessment of circulatory status after each bolus

Fluid therapy

- Fluid therapy should begin with deficit replacement plus maintenance fluid requirements.
- Deficit replacement should be with a solution that has a tonicity in the range 0.45% to 0.9% saline, with added potassium chloride, potassium phosphate or potassium acetate

- Clinical assessment of hydration status and calculated effective osmolality are valuable guides to fluid and electrolyte therapy.
- The aim is gradually to reduce serum effective osmolality to normal.
- There should be a concomitant increase in serum sodium concentration as the serum glucose concentration decreases (sodium should rise by 0.5 mmol/L for each 1 mmol/L decrease in glucose concentration).

TABLE 1 Losses of fluid and electrolytes in diabetic ketoacidosis and maintenance requirements in normal children

	Average (range) losses per kg	24-hour maintenance requirements	
Water	70 mL (30-100)	*≤10 kg	100 mL/kg/24 h
		11-20 kg	1000 mL + 50 mL/kg/24 h for each kg from 11 to 20
		>20 kg	1500 mL + 20 mL/kg/24 h for each kg >20
Sodium	6 mmol (5-13)	2-4 mmol†	
Potassium	5 mmol (3-6)	2-3 mmol	
Chloride	4 mmol (3-9)	2-3 mmol	
Phosphate	0.5-2.5 mmol	1-2 mmol	

Insulin therapy

- **Although rehydration alone frequently causes a marked decrease in blood glucose concentration, insulin therapy is essential to:**

1- restore normal cellular metabolism

2- to suppress lipolysis and ketogenesis

3- to normalize blood glucose concentrations

- **Start insulin infusion at least 1 hour after starting fluid replacement therapy; that is, after the patient has received initial volume expansion**

Correction of insulin deficiency

- Dose: 0.05 to 0.1 unit/kg/h.
- Route of administration IV
- An IV bolus should not be used at the start of therapy; it is unnecessary, may increase the risk of cerebral edema, can precipitate shock by rapidly decreasing osmotic pressure, and can exacerbate hypokalemia

- The dose of insulin should usually remain at 0.05 to 0.1 unit/kg/h at least until resolution of DKA (pH >7.30, serum bicarbonate >15 mmol/L, BOHB<1 mmol/L or closure of the anion gap)which in variably takes longer than normalization of blood glucose concentration

- monitor PH and BOHB Q2H to ensure steady improvement of biochemical parameters

- To prevent an unduly rapid decrease in plasma glucose concentration and hypoglycemia, 5% glucose, initially, should be added to the IV fluid when the plasma glucose falls to approximately 14 to 17 mmol/L (250-300 mg/dL), or sooner if the rate of fall is precipitous.

Potassium replacement

- K may be high, normal or low.
- The starting potassium concentration in the infusate should be 40 mmol/L(4mmol/100ml).
- Subsequent potassium replacement therapy should be based on serum potassium measurements
- If potassium is given with the initial rapid volume expansion, a concentration of 20 mmol/L should be used.

- Potassium phosphate may be used together with potassium chloride or acetate;
- Administration of potassium entirely as potassium chloride contributes to the risk of hyperchloremic metabolic acidosis, whereas administration entirely as potassium phosphate can result in hypocalcemia

Phosphate replacement ???

Acidosis

- Severe acidosis is reversible by fluid and insulin replacement; insulin stops further ketoacid production and allows ketoacids to be metabolized, which generates bicarbonate.
- Treatment of hypovolemia improves tissue perfusion and renal function, thereby increasing the excretion of organic acids. Controlled trials have shown no clinical benefit from bicarbonate administration.
- Bicarbonate therapy may cause paradoxical CNS acidosis and rapid correction of acidosis with bicarbonate causes hypokalemia.

* complication of therapy :

- Cerebral edema
- Hypokalemia
- Hyperchloremic acidosis
- Hypoglycemia
- Inadequate rehydration

Morbidity and mortality of DKA

- Cerebral injury is the major cause of mortality and morbidity and cerebral edema accounts for 60% to 90% of all DKA deaths

Cerebral edema

- **Factors associated with an increased risk of cerebral edema include:**
  - Younger age
  - New onset diabetes
  - Longer duration of symptoms These risk associations may reflect the greater likelihood of severe DKA.
  - Increased serum urea nitrogen at presentation
  - More severe acidosis at presentation

- Bicarbonate treatment for correction of acidosis
- A marked early decrease in serum effective osmolality
- An attenuated rise in serum sodium concentration or an early fall in glucose-corrected sodium during therapy
- Greater volumes of fluid given in the first 4 hours
- Administration of insulin in the first hour of fluid treatment

- **Signs and symptoms of cerebral edema include**:

- Onset of headache after beginning treatment or progressively worsening headache.
- Change in neurological status (irritability, confusion, inability to arouse, incontinence).
- Specific neurological signs (eg, cranial nerve palsies, papilledema).
- Cushing's triad (rising blood pressure, bradycardia, and respiratory depression) is a late but important sign of increased intracranial pressure.
- Decreased O2 saturation.

Treatment of cerebral edema

- Adjust fluid administration rate as needed to maintain normal blood pressure while avoiding excessive fluid administration that might increase cerebral edema formation. Assiduously avoid hypotension that might compromise cerebral perfusion pressure
- Hyperosmolar agents should be readily available at the bedside.
- Give mannitol, 0.5 to 1 g/kg IV over 10 to 15 minutes.247–249 The effect of mannitol should be apparent after ~15 minutes, and is expected to last about 120 minutes. If necessary, the dose can be repeated after 30 minutes.

- Hypertonic saline (3%), suggested dose 2.5 to 5 mL/kg over 10 to 15 minutes, may be used as an alternative to mannitol, or in addition to mannitol if there has been no response to mannitol within 15 to 30 minutes.
- Hypertonic saline (3%) 2.5 mL/kg is equimolar to mannitol 0.5 g/kg.
- Elevate the head of the bed to 30 and keep the head in the midline position.
- Intubation may be necessary for the patient with impending respiratory failure due to severe neurologic compromise.

Prevention of recurrent dka ??

Thank you