Diabetic ketoacidosis Noushin Rostampour Assistant professor of Pediatric

Endocrinology

Outline

- Risk factors for DKA
- Definition
- Clinical manifestations
- Diagnosis
- Management

DKA

• DKA :absolute or relative deficiency of circulating insulin and the combined effects of increased levels of the counterregulatory hormones

• Leading cause of morbidity and mortality in children with T1DM

High risk for DKA

- Children with poor metabolic control (higher HbA1c)
- Gastroenteritis with vomiting and dehydration
- Peripubertal and pubertal adolescent girls
- Children with a history of psychiatric disorders or unstable family circumstances

High risk for DKA

- Children with limited access to medical care
- Inadvertent or intentional omission of insulin, including failure of an insulin pump

High risk for DKA at initial presentation

- Young age (<5 years of age and especially <2 years)
- Low socioeconomic status or lack of health insurance
- Delayed diagnosis of diabetes
- Children living in countries with low prevalence of type 1 diabetes

PRECIPITATING FACTORS

- Poor metabolic control or missed insulin doses
- Illness
- Medication :such as corticosteroids, atypical antipsychotics, tacrolimus, L-asparaginase, and diazoxide
- Drugs and alcohol

Definition

- Plasma glucose > 200 mg/dl
- Venous pH < 7.30
- Bicarbonate level < 15 mEq/1
- Ketonemia(BOHB)> 3 mmol/L
- Moderate or large ketonuria

Clinical manifestations

- Dehydration
- Kussmal respiration
- Nausea, vomiting, abdominal pain
- Loss of consciousness
- Fever when infection is present
- Leukocytosis with left shift

Clinical presentations

- The clinical presentation are related to:
- Degree of hyperosmolarity
- Volume depletion
- Severity of acidosis

Clinical assessment

- Vital sign
- Weight
- Estimate degree of dehydration
- Neurologic assessment

Laboratory evaluation

- BS
- BOHB
- Electrolyte
- BUN/Cr
- Ca,Ph,Mg
- VBG
- CBC
- U/A,B/C if fever or localizing sign of infection are present
- ECG

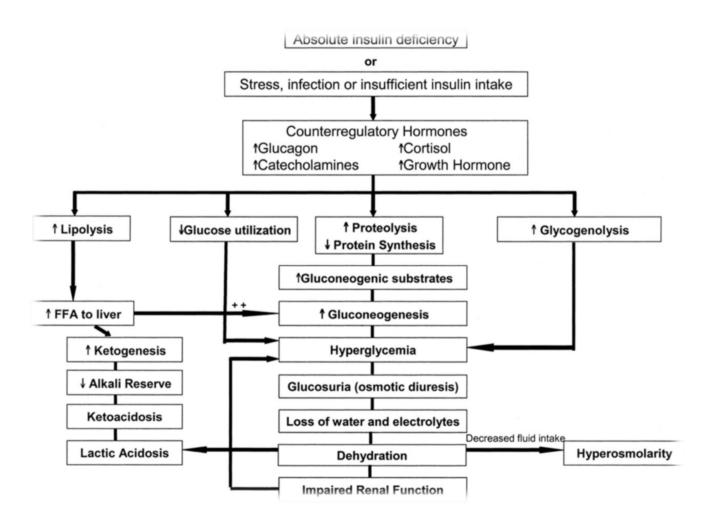
Assessment of severity

	Mild	Moderate	Severe
PH	7.2-7.3	7.1-7.2	< 7.1
HCO3	10-15	5-10	< 5

Classification

	Mild	Moderate	Severe
Volume (%) deficit	3-5	6-8	8-10
BS	200-400	400-600	≥ 600
BUN	≤25	≤30	≥ 30
Na	NL	NL	NI or Hi

Pathophysiology



Management

Case study

کودک ۶ ساله ای با وزن ۲۰ کیلوگرم از دو هفته قبل دچار پر ادر اری و پر نوشی شده است.

HCO3= 4

.PH=6.9 BS=624 9FBS=482 •

Na=135, K=5.5

- با توجه به ازمایشات فوق چه درجه ای از DKA مطرح است؟
 - میزان و نوع مایع دریافتی اولیه؟
 - کنتر ل قند خون با انسولین چگونه است؟
 - درمان با انسولین را از چه زمانی آغاز می کنید؟
 - در ادامه چه نوع مایعی به بیمار می دهید؟

Goals of therapy

- Improvement of circulatory volume and tissue perfusion
- Reduction of serum glucose and plasma osmolarity
- Correction of electrolyte imbalance
- Resolution of ketosis
- Identification and prompt treatment of comorbid precipitating causes

Treatment

- 1-Fluids
- 2-Insulin
- 3-Electrolytes replacement

Fluid therapy

- Initial therapy
- Maintenance
- Deficit

Initial therapy

- 1st hour 10-20 ml/kg
- Using isotonic saline or Ringer's lactate
- If circulating volume is still compromised after the initial bolus is complete, additional IV bolus infusions of 10 to 20 mL/kg can be given
- Initial reexpansion should not exceed 50 ml/kg over the first 4 h of therapy

Initial therapy

- Repeated 10ml/kg if:
- Shock
- Hypotension
- Delay capillary refilling
- Decrease tissue perfusion

Maintenance & deficit

1-12 hr	½ M+1/2 D
12-36 hr	M+1/2 D

Type of fluid

- BS > 300 mg/dl : N/S
- 200 < BS < 300 mg/dl : D/W 5% in 75 meq/L Na
- 100 <BS< 200 mg/dl : D/W 7.5% in 75 meq /L Na
- BS <100mg/dl : D/W 10% in 75 meq /L Na

Fluid therapy

• Max fluid:3 -4lit/m2/day

Potassium replacement

- Patients with DKA have a total body deficit of k
- K :normal or even increased
- k routinely decline during DKA treatment due to:
- Insulin-stimulated transport to the intracellular space
- Exchange for intracellular hydrogen ions with correction of acidosis

Electrolyte replacement

```
K: 2 th hr
K>5.5 No kcl
K=3.5-5.5 40 meq/L IV
K=2.5-3.5 0.5-1meq/kg kcl 15% IV (1 hr) 40-60 meq/L IV Monitor K hourly
```

K < 2.5-3

- 1 meq/kg KCL 15% IV within 1 hour
- Withhold insulin infusion
- Monitor K hourly

Insulin therapy

- Since 2th hr 0.1 U/kg/hr
- $K < 2.5 3 \rightarrow$ Insulin hold

Insulin therapy

• If the patient shows marked sensitivity to insulin, as in some younger or malnourished children, it may be necessary to decrease the insulin infusion rate to avoid hypoglycemia (eg, to 0.05 units/kg/hour)

Insulin therapy

- If facilities to administer intravenous insulin are not readily available, SC or IM insulin can be used as initial therapy
- However, the combination of volume depletion and secondary sympathetic activation decrease local perfusion, initially leading to inconsistent absorption

Clinical &biochemical monitoring

- Hourly (or more frequently as indicated) vital signs (heart rate, respiratory rate, and blood pressure)
- Hourly (or more frequently as indicated) neurological observations for warning signs and symptoms of cerebral edema
- Amount of administered insulin

Clinical &biochemical monitoring

- Hourly (or more frequently as indicated) accurate fluid input (including all oral fluid) and output
- Capillary blood glucose should be measured hourly
- Laboratory tests: serum electrolytes and blood gases should be repeated every 2–4 h

Vital Signs					
temp					
pulse					
blood pressure					
respiratory rate					
GCS					
pupII size					
pupil reaction					
cap refill time					
Laboratory					
glucose					
pH					
sodium					
potassium					
chloride					
CO2					
HCO3					
BUN					
creatinine					
ketones serum					
ketones urine					
βOHbytyrate					
osmolality measured					
osmolality calculated					
calclum					
phosphorus					
magnesium					
Fluids and Insulin					
type					
potassium mEq/L					
rate					
Intake					
urine output					34
Insulin units/hour	l				

Mild DKA

- Older children and adolescents with established diabetes and mild DKA can frequently be managed in the emergency department
- These patients often improve substantially after IV fluid therapy and subcutaneous insulin administration
- Rapid-acting insulin can be given at an initial dose of 0.1 units/kg every one to two hours, with close monitoring of blood glucose
- Regular insulin (given every four hours) has also been used in these circumstances

Bicarbonate therapy

- life-threatening hyperkalemia
- Severe acidosis: PH < 6.9 and
- Hypotension
- Shock
- Arrhythmia

Bicarbonate therapy

- Decrease the acidemic stimulus for hyperventilation: rise in partial pressure of carbon dioxide (pCO2) and causing a paradoxical fall in cerebral pH
- Slow the rate of resolution of ketosis
- Development of cerebral injury
- Hypokalemia

Criteria for resolution of DKA

- Serum anion gap reduced to normal $(12 \pm 2 \text{ meq/L})$
- Venous pH >7.30 and/or the serum HCO3 >15 meq/L
- Plasma glucose <200 mg/dL
- Tolerating oral intake

Stop IV insulin

• IV insulin infusion should be discontinued 15 to 30 minutes after the first injection of rapid-acting insulin

Urine ketone

- Nitroprusside test strip reacts with acetoacetate and acetone but not BOHB, which makes up 75 percent of circulating ketones
- During recovery from DKA, BOHB is converted to acetoacetate and acetone, which are excreted in urine for several hours after the serum BOHB concentration has returned to normal

Time of feeding

- The patient wishes
- Conscious
- No vomiting

Persistent acidosis

- Infection
- Dehydration
- Inadequate insulin
- Out of date insulin

COMPLICATIONS AND MORTALITY

- Reported mortality rates for DKA: 0.15 to 0.51 percent
- Cerebral edema accounts for the majority of deaths (60 to 90 percent)

warning signs and symptoms of cerebral edema

- Headache
- Inappropriate decrease in heart rate
- Recurrence of vomiting
- Changes in neurologic status
- Rising blood pressure
- Decreased oxygen saturation

Risk factors for brain edema

- An attenuated rise in measured serum sodium concentrations during therapy
- Severity of acidosis
- Bicarbonate treatment
- Greater hypocapnia at presentation
- Elevated serum urea nitrogen at presentation
- Greater volumes of fluid (first 4 hr)
- Administration of insulin in the first hour of fluid treatment

Treatment

- Manitol 0. $5 1 \frac{gr}{kg}/10-15 \min$
- Adjust fluid administration as indicated to maintain normal blood pressure and optimize cerebral perfusion
- Hypertonic saline(3%) 2.5-5 ml/kg/15 min