Case Conference

2010.03.23 R1徐英洲/VS蕭蔚全 **DKA** : Definition

 hyperglycemia (>200 mg/dL), anion gap metabolic acidosis (pH<7.3, HCO3<15 meq/L), and ketonemia.

		DKA				
		Mild	Moderate		Severe	
Plasma glucose (mg/dL)		>250	>250	>250	>250	
Arterial pH		7.25-7.30	7.00-7.24	<7.00	<7.00	
Serum bicarbonate (mEq/L)		15-18	10 to <15	<10	<10	
Urine ketones*		Positive	Positive Positiv			
Serum ketones*		Positive	Positive Positi			
ffective serum osmolality (mOsm/kg)*		Variable	Variable	Variable		
inion gapa		>10	>12 >12			
Alteration in sensoria or mental obtundation		Alert	Alert/drowsy	Stupor/co	oma	
		Mild	Moderat		Severe	
Defi	ining features		1			
Ve	moves pH	7.2-7.3	7.4-7.3		<7.1	
	rum bicarbonate	10-15	5-10		<5	

Recurrent DKA

- Risk factors :
 - Higher A1C levels and higher reported insulin requirements
 - Female adolescents, with the highest risk in female adolescents over 13 years of age
 - Children over 13 years of age, who are underinsured and/or have a history of psychiatric disorders
 - Longer duration of DM

~Predictors of acute complications in children with type 1 diabetes JAMA 2002 May 15;287(19):2511-8.

DKA : Clinical Manifestation1

- earliest symptoms are related to hyperglycemia.
- polyuria (due to the glucose-induced osmotic diuresis), polydipsia (due to the increased urinary losses), and fatigue, weight loss.
- abdominal pain (unusual in HHS) : associated with the severity of the metabolic acidosis

DKA: Clinical Manifestation2

- Hyperventilation and deep (Kussmaul) respirations : the respiratory compensation for metabolic acidosis.
- Neurologic findings : from drowsiness, lethargy, and obtundation to coma, are related to the severity of hyperosmolality and/or to the degree of acidosis.

DKA management

- Assess severity :
 - Neurologic status : potential risk for cerebral edema
 - Acid-base status : venous pH > serum bicarbonate concentration > respiratory rate
 - The anion gap : A high anion gap may also reflect decreased renal perfusion, which limits ketoacid excretion.
 - Assessment of volume depletion status
 - Duration of symptoms : long duration of symptoms, as well as depressed level of consciousness or compromised circulation, is evidence of severe DKA

DKA management : Fluid repletion1

- Assess degree of dehydration in children with DKA is DIFFICULT →less likely to show the classic signs of hypovolemia (ex : dry oral mucous membranes or decreased skin turgor)
- To reduce risk of cerebral edema →accomplishing the volume expansion gradually and with isotonic fluids
- Initial fluid management : assumption of a 5 to 7 percent deficit for moderate DKA, and 10 percent dehydration for severe DKA

DKA management : Fluid repletion2

- moderate to severe DKA : usually begun with an infusion of 10 to 20 mL/kg over one hour.
- If hemodynamic is stable → slowly administration of isotonic saline for 4 to 6 hours → then switch to one-half isotonic saline (may add Potassium)
- Based upon body surface area, the volume should be no greater than 2500 mL/m2 for 24 hours (may increase risk of cerebral edema).

DKA management : insulin1

- suppress glucose and ketone production, and stimulate peripheral glucose and ketone metabolism.
- After the initial fluid bolus → insulin infusion is begun at a rate of 0.1 unit/kg per hour.
- IV bolus of insulin is **NOT** recommended in the first hour before initiation of fluid therapy.

~The UK case-control study of cerebral oedema complicating diabetic ketoacido in children. Diabetologia. 2006 Sep;49(9);2002-9, Epub 2006 Jul 18.

DKA management : insulin2

- F/S: 250 to 300 mg/dL→ shift saline to 5 % dextrose in isotonic saline or lactated Ringer's solution (continued administration of insulin→ correct ketoacidosis)
- (continued administration of msumi > correct retoracidosis)
- Ideal goal → gradual decrease glucose about 50 to 100 mg/dL
 For younger children → keep serum glucose around 150 to 200 mg/dL or
 - For older children \rightarrow keep 100 to 150 mg/dL
 - \rightarrow Then switch to subcutaneous insulin

ide evaluation of neurological state of children with DKA

DKA management : Sodium

- Usually low measured serum sodium→ osmotic effect of hyperglycemia.
- Reversing the hyperglycemia with insulin → lower the plasma osmolality, cause water to move from the extracellular fluid into the cells → Na ↑
- If Na \rightarrow or \downarrow : may predict risk of cerebral edema

Mered mentation/Ruchating level of consciousness Sostaned heart rate deceleration (decline more than 20 bpm) not attributable to improved intravascular valume or sleep state Ager-naporposite montheme Winor orbital Userhamp or being not easily aroused from sleep Detatiots biod pressure >90 mm/tg Ager <5 years Diagnostic criteria Ager <5 years Diagnostic criteria Commit equipy (especially III, Nr, and VI) Abommit more criteria pattern (eq. guntage, Echypenes, Cheyne-Stokes respiration, apneuse) Cerebral edcema is diagnosed if any of the diagnostic criteria is present. And it is also likely if two major criteria OR one major and two minor criteria are present.

~Cerebral edema in childhood diabetic ketoacidosis: natural history, radiographic findings, and early identification. Diabetes Care 2004; 27:1541.

DKA management : Potassium

- most DKA patients $: K \rightarrow or \uparrow$
- After initial IV expansion : $K \downarrow$ (to normal range)
 - If $K \rightarrow$: potassium replacement should be given with the start of insulin therapy
 - If K↓ : potassium replacement should be started immediately,
 - If $K \uparrow$: potassium replacement should be initiated when the serum potassium falls to normal

DKA management : Metabolic Acidosis

- Insulin : promotes the metabolism of ketoacid anions, resulting in the generation of bicarbonate, and stops the ongoing production of new ketoacids.
- sodium bicarbonate : NO clinical benefit from the routine administration.
 - Maybe a risk factor for cerebral edema
- patients with severe acidemia (arterial pH <6.90) or severe hyperkalemia → may use sodium bicarbonate

~ESPE/LWPES consensus statement on diabetic ketoacidosis in children and adolescents.

DKA management : Discontinuing the insulin infusion

- Serum anion gap reduced to normal $(12 \pm 2 \text{ meq/L})$
- Venous pH is >7.30 or serum HCO3 is >15 meq/L
- Plasma glucose <200 mg/dL
- Tolerating oral intake
- a persistent normal anion gap acidosis is NOT a contraindication for switching the patient to subcutaneous insulin

Thanks for your attention !