

Guidelines in the management of Diabetic Ketoacidosis

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Do we have international Guidelines

- **American Diabetes Association (2004)**
- **British Society of Paediatric Endocrinology and Diabetes (2004)**
- **International Society for Pediatric and Adolescent Diabetes (2007)**

**Treatment of DKA could be summarized into fluid, Insulin and
K therapy**

**HOWEVER, DETAILS WILL BE ASSOCIATED
WITH MANY CONTROVERSIAL ISSUES**



The 1st pitfall



How to diagnose DKA ?

- **Urine testing for ketone bodies is a simple bedside test that rapidly confirm diagnosis.**
- **However, Nitroprusside test measures only acetoacetate and acetone but not B-hydroxybutric acid.**
- **In some patients with DKA B-hydroxybutric acid is the major ketone bodies. Specific tests for B-hydroxybutric acid are present but not widely used.**

Serum betaOHB levels

- ☐ > 3.0 mmol/l in children**
- ☐ > 3.8 mmol/l in adults**

in the presence of uncontrolled diabetes can be used to diagnose DKA and may be superior to the serum HCO(3) level for that purpose.

- Diabetes Care. 2008 Apr;31(4):643-7.**



The key message (1)

DKA is confidently excluded if serum beta-hydroxybutyrate is less than 3 mmol/l.

SO we should have meters that measure B-hydroxybutric acid



The 2nd pitfall



How to start insulin therapy?

Large versus small dose regimens

lower doses given at frequent intervals effectively lower blood glucose and are more safe than previously adopted regimens.

Loading dose of Insulin

Using loading dose of insulin also makes no difference because the initial blood glucose response is largely dependent on rehydration.

Still recent debate

- To assess the efficacy of an insulin priming dose with a continuous insulin infusion versus two continuous doses without priming (0.07 & 0.14U U/kg/h)
- priming dose in low-dose insulin therapy in DKA patients is unnecessary if using an adequate dose of 0.14U of regular insulin per kg/Bwt/h (about 10U/h in a 70kg patient).
- Kitachi et al: Is A Priming Dose Of Insulin Necessary In A Low Dose Insulin Protocol For The Treatment Of Diabetic Ketoacidosis?. Diabetes Care. 2008 Aug 11

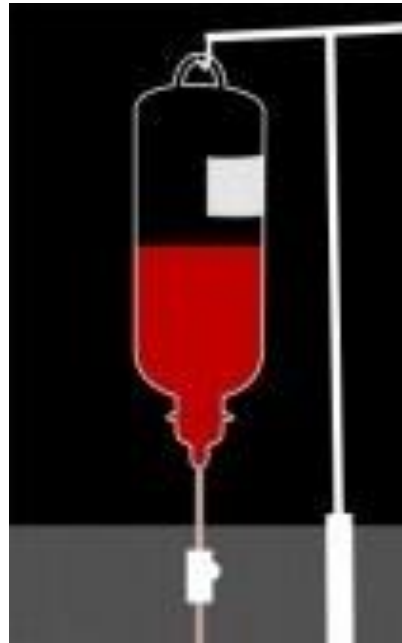


The key message (2)

- **Insulin therapy should be started with low doses at frequent intervals (0.1 unit / kg/ hr) regular insulin by IV infusion or IM injection**
- **But the response to insulin should be monitored. The predicted response to insulin is 10% reduction from its initial level (about 75 mg drop in blood glucose) every hour. If this is not achieved after 2 hr of therapy double the dose of insulin .**



The 3th pitfall



Do we need rapid vigorous correction of dehydration?

Fluid therapy

- **Type of fluid needed (Quality)**
- **Amount of fluid needed (Quantity)**
- **The rate of infusion**

Amount of fluid needed (Quantity)

Maintenance fluid + fluid deficit

Maintenance fluid (1500 ml/m² /24hr)

+

Fluid deficit

(Fluid deficit in patients > 2yrs)

=

30ml/ kg for mild deficit

60ml/kg for moderate deficit

90ml/kg for severe deficit

The rate of infusion

- **Fluid resuscitation by 10-20 ml /kg in the first hour. In severe dehydration this dose may be repeated but the initial re- expansion should not exceed 50 ml/kg over the first 4 hr of therapy.**
- **Then give the remaining total volume evenly over the next 48 hours .**



The key message (3)

- **Start fluid therapy with 10-20 ml /kg isotonic saline in the first hour. In severe dehydration this dose may be repeated but the initial re- expansion should not exceed 50 ml/kg over the first 4 hr of therapy.**
- **Use glucose 5% instead of saline if BG dropped below 250mg%**



The 4th pitfall



Potassium assessment and replacement

Potassium Assessment

- **Assessment of K status is an important item in the management of DKA.**
- **If you didn't have reliable lab. ECG can give some hint about s K.**

Potassium replacement (In the absence of reliable lab)

Give 20 mEq K/hr

Stop K infusion:

- If the patient is anuric
- If ECG revealed tall T wave

Potassium replacement (In the presence of reliable lab)

- *If serum K is < 2.5 mEq/L give 60mEq K/ liter of fluids and stop insulin temporarily.*
- *If serum K is 3 mmol/L give 40 mEq K /hr*
- *If serum K is $3 -4$ mmol/L give 30 mEq K/hr*



The key message (4)

Even if you didn't have reliable lab you should give K by small dose *20 mEq K/hr and follow T wave changes.*



The 5th pitfall



**Cerebral oedema as a complication of
DKA treatment**

- **Cerebral oedema is a fatal complication of treatment of DKA, It accounts for 40 % of DKA deaths.**
- **Rapid decline in plasma osmolality as a result of rapid decline of blood glucose or excessive infusion of hypotonic saline will lead to cerebral oedema.**
- **The pathogenesis appears complex and is poorly understood. Dehydration and hypocapnia diminish cerebral perfusion, resulting in mild brain ischaemia and subsequent cytotoxic and vasogenic cerebral oedema.**

Diagnosis of cerebral oedema

- **Cerebral oedema should be suspected if consciousness start to be disturbed while metabolic status is improving.**
- **Brain stem herniation may be rapid so that papilledema is not found.**
- **About 40% of cases showed no acute abnormalities on their initial CT exams, emphasizing that CE in the context of DKA is a clinical, not a radiological, diagnosis.**

**Neurological Deterioration in Diabetic
Ketoacidosis – Is it Cerebral Edema or
Something Else?**

Cerebral edema is the most likely cause of acute neurological deterioration in DKA, though in about 20% of acute neurological episodes other causes should be suspected e.g.

- **Cerebral venous thrombosis**
- **Viral encephalitis**
- **Meningitis (TB, streptococcal, meningococcal)**

Treatment of CE

- **The current treatment for suspected CE is immediate infusion of mannitol 0.5-1 g/kg over 20 minutes and to restrict fluids.**

- **Endotracheal intubation may be needed to protect the airway of the comatose patient, but the value of hyperventilation is questionable.**

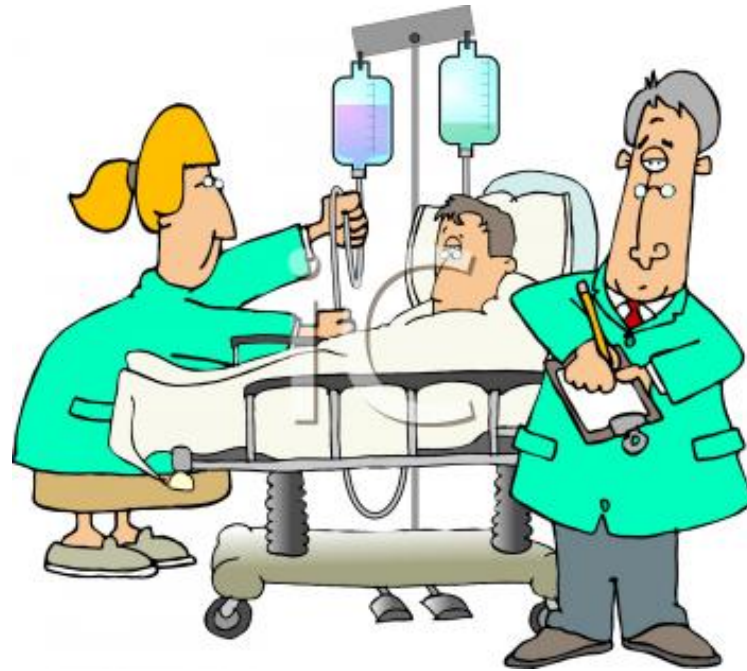


The key message (5)

Early diagnosis and treatment of CE will markedly reduce mortality in DKA



The 6th pitfall



Do we need a follow up sheet for DKA?

العلاج Treatment

1 = متيقظ alert = 2 = مدروخ لكن يستجيب بسرعة للتعبئة lethargic = 3 = مدروخ لكن يستجيب ببطء للتعبئة stuporous = 4 = غيبوبة coma
 حرق الشفوي: طبيعي، حرق، سطحي



The key message (6)

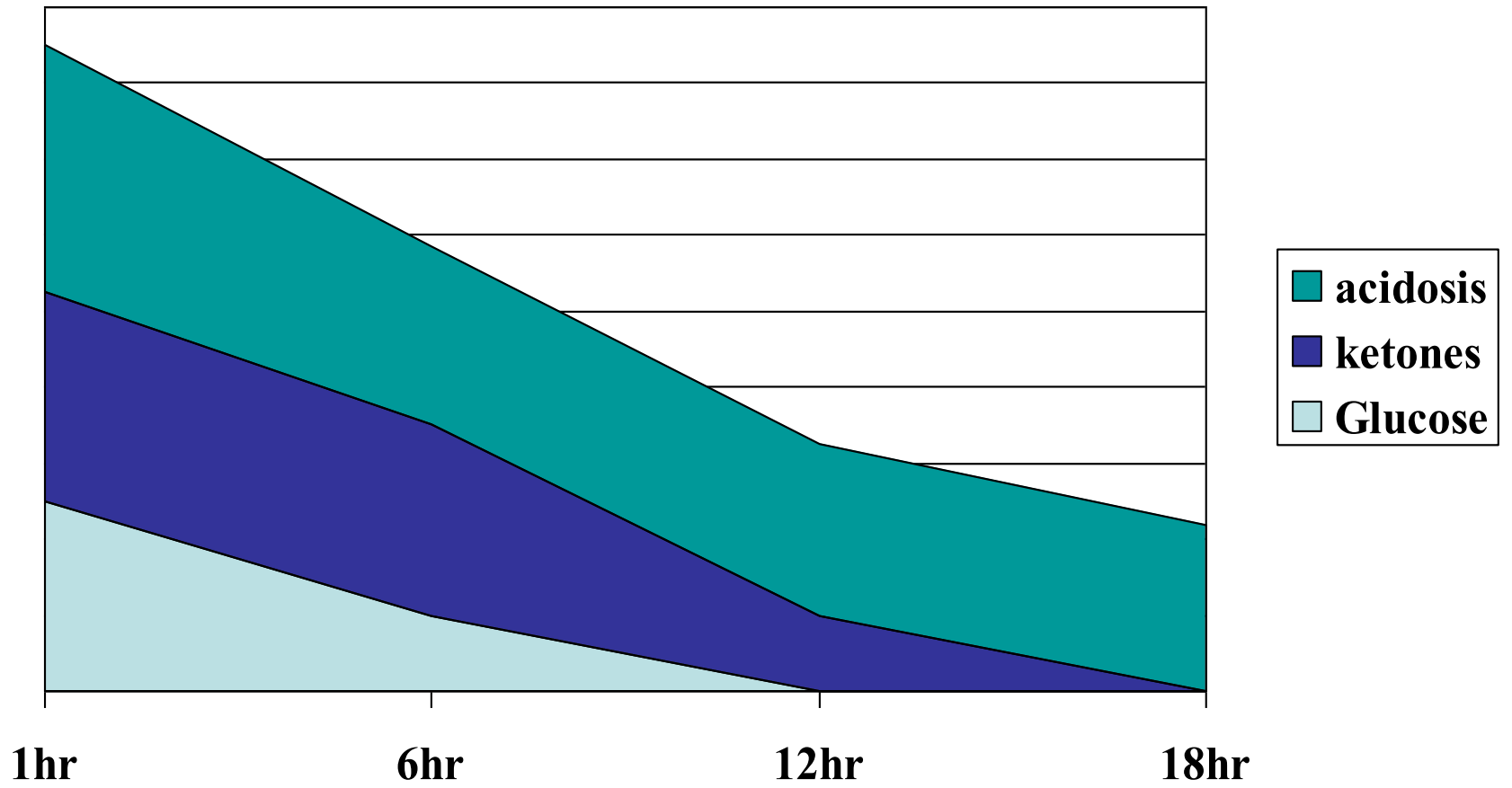
Patient with DKA should be carefully monitored to ensure adequate response to therapy and to early diagnose CE



The 7th pitfall



When Diabetic Keto acidosis is considered resolved ?





The key message (7)

DKA is considered resolved when 2 of the following criteria were met:

- 1. $\text{HCO}_3^- > 18 \text{ mEq/L}$**
- 2. Venous pH > 7.32 (venous pH is 0.03 U lower than arterial pH)**
- 3. Calculated anion gap less than 14 mEq/L.**

**For one mistake done in not
knowing, ten mistakes are done
in not doing**

Thank you