## Management of Diabetic Ketoacidosis

# Introduction of Diabetic Keto Acidosis

It is a disorder mainly in patient with type 1 Diabetes Mellitus, but may occur in Type 2 Diabetes Mellitus also. It may result from increased insulin requirements in Type 1 Diabetes mellitus during infection, trauma, Myocardial Infarction, or surgery. It is a life-threatening medical emergency with mortality rate <5% individual under 40 years of age, but more severe prognosis in older people with mortality rate >20%.

### Essential criteria for diagnosis of Diabetic Ketoacidosis

- Hyperglycaemía (Blood glucose level >250 mg/dl).
- Metabolic acidosis-blood pH <7.3 g serum HCO₃-<15 mg/L.</li>
- Ketone bodies +ve in serum.

### Therapeutic Goal

- To restore plasma volume.
- To reduce blood glucose level and osmolality.
- To correct acidosis.
- To replenish electrolyte losses.
- To identify and treat precipitating factors.

#### Treatment

- Admít in ICU.
- A- Maintain patency of airway.
- B-Breathing, sp02 >90%. Intubation and mechanical ventilation.
- C- Circulation, IV-line access.
- D- Drugs (Insulin).
- E- Electrolytes replacement.
- F- Fluid replacement.

## Fluid replacement

- Fluid deficit 4 to 5 L.
- Fluid of choice 0.9% NS.
- Should be started in emergency department as soon as diagnosis is established.
- Should be infused @1 L/hour over the first 1 to 2 hour.

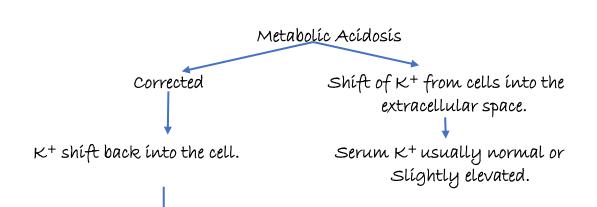
- After the first 2 L of fluid have been given, IV fluid infusion should be @ 300 to 400 ml/hour.
- Use 0.9% NS unless the serum sodium is >150mEq/L, then 0.45% of NS should be used.
- The volume status should be monitor Clinically.
- If volume replacement < 3 to 4 L in 8 hours, difficult to restore normal perfusion.
- If volume replacement > 5 litre in 8 hours, Acute Respiratory Distress Syndrome (ARDS) and cerebral oedema may occur.
- When blood glucose levels fall to Approx. 250 mg/dl, the fluid should be changed to 5% glucose containing solution to maintain serum glucose in the range of 250 to 300 mg/dl. This will prevent development of hypoglycaemia and cerebral oedema due to rapid decline of blood glucose level.

### Insulin replacement

- Immediately after initiation of fluid replacement.
- · Type of insulin-HIR
- Loading dose- 0.1 Unit/Kg IV bolus prime the tissue insulin receptors.
- Followed by **0.1 unit/kg/hour** continuously IV infused to replace the deficit of insulin.
- If blood glucose level fails to fall at least 10% in the first hour, repeat loading those (0.1 unit per kg) recommended.
- The insulin dose should be adjusted to lower the blood glucose level by about 50 to 70 mg/dl/hour.

#### Potassium

 Total body potassíum loss from polyuría and vomíting may be as hígh as 200mEq.



Hypokalaemía.

Need K+ replacement.

- Potassíum replacement should be started as soon as metabolíc acídosís starts to resolve.
- Potassíum Chloride (KCl)-10 to 30mEq/hour should be infused during 2nd and 3rd hours after beginning of therapy.
- Food high in potassium content should be prescribed when the patient has recovered sufficiently to take food orally.
- Tomato juice has 14mEq of K+/240 ml, and a medium sized banana contains about 10mEq of potassium.

### Sodium Bicarbonate (NaHCO3-)

The use of sodium bicarbonate in the management of DKA has been questioned because of the following potentially harmful consequences:

- Development of **hypokalaemía** from rapid shift of K+ into the cells due to overcorrected metabolic acidosis.
- Tissue anoxia from reduced dissociation of oxygen from haemoglobin when metabolic acidosis is rapidly reversed.
- Cerebral acidosis resulting from lowering of CSF pH.

Therefore, it is recommended that  $HCO_3$ -should be administered in DKA, if the arterial blood pH is  $\leq$  7.0

One or two ampoules of NaHCO3- (1 ampoule contains 44mEq/50ml) should be added to 1 L of 0.45% saline with 20mEq KCl and infused over 1 to 2 hour.

## Phosphate

- Phosphate requirement is seldom required in treating DKA.
- However, if severe hypophosphatemia of less than 1mEq/dl develops during insulin therapy, or small amount of phosphate can be replaced per hour as the potassium salt.

# Hyperchloremic acidosis during therapy

• A portion of the bicarbonate deficit is the placed with chloride ions infused in large amounts as NS to correct the dehydration.

• Plasma lyte solution (pH-7.4, Cl-98mEq/L) instead of NS (pH-5.5, Cl-154mEq/L) should be used to prevent hyperchloremic acidosis.

## Treatment of associated infections

• Antibiotics should be prescribed as causative organisms identified.

## Transition to subcutaneous Insulin regimen

• Once the DKA is controlled and the patient is awake and able to eat, subcutaneous insulin therapy can be initiated.

References: - CMDT.

íple of 1: Harrison's Principle of Internal Medicine.

Katzung, Basíc & Clínical Pharmacology.