Short Communication
A King Complication: Diabetic Ketoacidosis

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Abstract: Diabetic Ketoacidosis is a serious short term complication and complex metabolic state characterized by hyperglycemia, ketoacidosis, and ketonuria. It usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones i.e., glucagon, cortisol, growth hormone, epinephrine. Accumulation of ketone bodies causes severe complications and lead to many life threatening clinical manifestations.

I. INTRODUCTION

Diabetic Ketoacidosis (DKA) is a treacherous impediment faced by clients with diabetes which happens in the means of insulin deprivation. It is also called as DKA or Ketoacidosis can upshot in coma or even death if it is not treated hastily (American Diabetes Association, 2015). Diabetic Ketoacidosis is a serious short term complication and complex metabolic state characterized by hyperglycemia, ketoacidosis, and ketonuria. It usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones i.e., glucagon, cortisol, growth hormone, epinephrine. This type of hormonal imbalance enhances hepatic gluconeogenesis, glycogenolysis, and lipolysis. DKA is most commonly associated with type 1 diabetes, however, people with type 2 diabetes that produce very little of their own insulin may also be affected (Medical Care in Diabetes, 2016).

According to WHO, Diabetes mellitus is a leading cause of death and disability worldwide. Its global prevalence was about 8% in 2011 and is predicted to rise to 10% by 2030. Nearly 80% of people with diabetes live in low- and middle-income countries. In the World 171,000,000 (cases in 2010) 366,000,000 cases in 2030. The WHO also estimates that 80 per cent of diabetes deaths occur in low and middle-income countries and projects that such deaths will double between 2016 and 2030 (WHO, 2012).

India is called as Diabetes Capital of the World since 50 million people suffering from Diabetes. India accounts for most of the children with T1DM in South-East Asia. According to the 6 th edition of the International Diabetes Federation diabetes atlas, India has 3 new cases of T1DM/100,000 children of 0-14 years. The prevalence of diabetes in India is variable, and three sets of data show 17.93 cases/100,000 children in Karnataka, 3.2 cases/100,000 children in Chennai, and 10.2 cases/100,000 children in Karnal (Haryana). The bottom line remains that T1DM is quite prevalent and common (Aguiree F etal, 2015, Kalra S et al, 2010).

The estimate of the actual number of diabetics in India is around 40 million. The prevalence of impaired glucose tolerance IGT is thought to be around 8.7 per cent in urban areas and 7.9 per cent in rural areas, although this estimate may be too high. It is thought that around 35 per cent of IGT sufferers go on to develop type 2 diabetes, so India is genuinely facing a healthcare crisis (Omics India).

II. ETIOLOGY

The most common scenarios for Diabetic Ketoacidosis (DKA) are underlying or concomitant infection (40%), missed or disrupted insulin treatments (25%), and newly diagnosed, previously unknown diabetes (15%). Other associated causes make up roughly 20% in the various scenarios (Bowden SA et al, 2008). DKA has also been reported in people with type 2 diabetes treated with sodium-glucose cotransporter-2 (SGLT2) inhibitors (Taylor SI et al, 2015).

Imperative Causes of Diabetic Ketoacidosis

Idiopathic	Medical & Emotional stress
Brittle Diabetes	Lack of Security
Omission or reduced daily injections	Pregnancy
Dislodgement/occlusion of insulin pump catheter	Hyperthyroidism
Infection	Substance abuse (Cocaine)
Medications: Steroids, thiazides, antipsychotics, sympathomimetics	Heat-related illness
Brain attack	Gastrointestinal Bleeding
Intercurrent illness : Myocardial infarction	Pulmonary embolism
Pancreatitis	Major trauma
Bacterial infection and intercurrent illness (E.g, Urinary Tract Infection [UTI])	Surgery

III. PATHO-PHYSIOLOGY

It occurs due decreased insulin secretion and altered glucose metabolism in the body. Body starts metabolizing fats in the absence of glucose and produces ketone bodies (acetone, acetoacetic acid and β-hydroxy butyric acid). Accumulation of ketone bodies causes severe complications and lead to many life threatening clinical manifestations.

IV. CLINICAL FEATURES OF DKA

The initial symptoms of DKA include anorexia, nausea**,** vomiting, polyuria, and thirst. Abdominal pain, altered mental function, or frank coma may ensue. Classic signs of DKA include Kussmaul respirations and an acetone odor on the pt's breath. Volume depletion can lead to dry mucous membranes, tachycardia, and hypotension. Fever and abdominal tenderness may also be present.

V. PINNACLE '5' CLINICAL DEPICTS OF DKA

Hyperglycemia: Blood glucose =250 - 800 mg/dL
Rx - Insulin IV (Serum osmolarity 300 - 350mosm/L)

Dehydration and electrolyte loss: Polyuria and polydipsia
Rx - 0.9% N/S is administered at a rapid rate, usually 0.5 to 1 L per hour for 2 to 3 hrs (6-10 L of fluids per day) + Potasium (Q. 2- 4 hrs)

Acidosis: Acetone breath, low pH (< 7.3) & HCo_3- < 15 meq/l

- "Fruity" odor on breath
- Kussmaul's respirations (deep, non labored)
- Rx - Insulin IV (5u/hr) + Antibiotics? + Hourly blood glucose assessment

Hyperkalemia initially then hypokalemia
Rx - Potassium must be infused Q.2-4hrs even if the plasma potassium level is normal + Insulin
Mild hyponatremia

VI. DIFFERENTIAL DIAGNOSIS FOR DKA

- Alcoholic Ketoacidosis
- Starvation Ketoacidosis
- Renal failure
- Lactic Acidosis
- Ingestions eg.Salicylates, Ethylene & Methanol

VII. GOALS OF DKA MANAGEMENT

- Fluid resuscitation
- Reversal of the acidosis and ketosis
- Reduction in the plasma glucose concentration to normal
- Replenishment of electrolyte and volume losses
- Identification the underlying cause

VIII. EMERGENCY & CRITICAL CARE MANAGEMENT OF DKA

- Confirm diagnosis (↑ plasma glucose, positive serum ketones, metabolic acidosis).
- Admit to hospital; intensive-care setting may be necessary for frequent monitoring or if pH < 7.00 or unconscious.
- Assess: Serum electrolytes like K+, Na+, Mg2+, Cl–, bicarbonate, phosphate and Acid-base status-pH, HCO3 –, PCO2, β-hydroxybutyrate Renal function (creatinine, urine output).
- Replace fluids: 2–3 L of 0.9% saline over first 1–3 h (10–15 mL/kg per hour); subsequently, 0.45% saline at 150–300 mL/h; change to 5% glucose and 0.45% saline at 100–200 mL/h when plasma glucose reaches 14 mmol/L, (250 mg/dL).
- Administer short-acting insulin: IV (0.1 units/kg) or IM (0.3 units/kg), then 0.1 units/kg per hour by continuous IV infusion; increase 2- to 3-fold if no response by 2–4 h. If initial serum potassium is < 3.3 mmol/L (3.3 meq/L), do not administer insulin until the potassium is corrected to > 3.3 mmol/L (3.3.meq/L).
- Assess patient: What precipitated the episode (noncompliance, infection, trauma, infarction, cocaine)? Initiate appropriate workup for precipitating event (cultures, CXR, ECG).
- Measure capillary glucose every 1–2 h; measure electrolytes (especially K+, bicarbonate, phosphate) and anion gap every 4 h for first 24 h.
- Monitor blood pressure, pulse, respirations, mental status, fluid intake and output every 1–4 h.
- Replace K+: 10 meq/h when plasma K+ < 5.5 meq/L, ECG normal, urine flow and normal creatinine documented; administer 40–80 meq/h when plasma K+ <3.5 meq/L or if bicarbonate is given.
- Continue above until patient is stable, glucose goal is 150–250 mg/dL, and acidosis is resolved. Insulin infusion may be decreased to 0.05–0.1 units/kg per hour.
- Administer intermediate or long-acting insulin as soon as patient is eating. Allow for overlap in insulin infusion and subcutaneous insulin injection (Tintinalli's Emergency Medicine Manual, 2012-16).

IX. POSSIBLE TREATMENT COMPLICATIONS: This includes Hypoglycemia, Hypokalemia, Crebral Edema, Fetal & Infant morbidity & Mortality in case of pregnancy and Unconscious or lead to fatal.

X. CONCLUSION: The severity of DKA depends on the magnitude of the decrease in arterial pH, serum bicarbonate levels, and the mental state rather than the magnitude of the hyperglycemia. It is mainly results from a relative insulin deficiency and counter-regulatory hormone excess causing hyperglycemia andn ketonemia.

XI. REFERENCES

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