Name: Law-Adepoju Inumidun Adejumoke

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 Group 2 assignment

1a. Ketogenesis is the biochemical process through which organisms produce ketone bodies through breakdown of fatty acids and ketogenic amino acids. This process supplies energy under circumstances such as fasting or caloric restriction to certain organs, particularly the brain, heart and skeletal muscle. Insufficient gluconeogenesis can cause hypoglycemia and the excessive production of ketone bodies ultimately leading to a life threatening condition known as ketoacidosis. The 3 ketone bodies are; acetoacetate, acetone, beta-hydroxyl-butyrate.

B. Ketonaemia; is the presence of an abnormally high concentration of ketone bodies in the blood.

C. Ketonuria: is the excretion of abnormally large amounts of ketone bodies in the urine. Causes include; metabolic abnormalities such as diabetes, renal glycosuria, or glycogen storage disease., dietary conditions such as starvation, fasting, low carbohydrates diet, prolonged vomiting, and anorexia caused by hyperemesis gravidarum.

2. Consequences of ketosis

Headache, fatigue, brain fog, increased hunger, poor sleep, nausea, decreased physical performance.

3. Management of ketoacidosis

 The main aims in the treatment of diabetic ketoacidosis are replacing the lost fluids and electrolytes while suppressing the high blood sugars and ketone production with insulin. Admission to an intensive care unit (ICU) or similar high-dependency area or ward for close observation may be necessary.

###  Fluid replacement

The amount of fluid replaced depends on the estimated degree of dehydration. If dehydration is so severe as to cause shock (severely decreased blood pressure with insufficient blood supply to the body's organs), or a depressed level of consciousness, rapid infusion of saline (1 liter for adults, 10 ml/kg in repeated doses for children) is recommended to restore circulating volume. Slower rehydration based on calculated water and sodium shortage may be possible if the dehydration is moderate, and again saline is the recommended fluid. Very mild ketoacidosis with no associated vomiting and mild dehydration may be treated with oral rehydration and subcutaneous rather than intravenous insulin under observation for signs of deterioration. Normal saline  (0.9% saline) has generally been the fluid of choice. There have been a few small trials looking at balanced fluids with few differences

###  Insulin

Some guidelines recommend a bolus (initial large dose) of insulin of 0.1 unit of insulin per kilogram of body weight. This can be administered immediately after the potassium level is known to be higher than 3.3 mmol/l; if the level is any lower, administering insulin could lead to a dangerously low potassium level (see below). Other guidelines recommend delaying the initiation of insulin until fluids have been administered. It is possible to use rapid acting insulin analogs injections under skin for mild or moderate cases.

 Potassium

Potassium levels can fluctuate severely during the treatment of DKA, because insulin decreases potassium levels in the blood by redistributing it into cells via increased sodium-potassium pump activity. A large part of the shifted extracellular potassium would have been lost in urine because of osmotic diuresis. Hypokalemia (low blood potassium concentration) often follows treatment. This increases the risk of dangerous irregularities in the heart rate. Therefore, continuous observation of the heart rate is recommended, as well as repeated measurement of the potassium levels and addition of potassium to the intravenous fluids once levels fall below 5.3 mmol/l. If potassium levels fall below 3.3 mmol/l, insulin administration may need to be interrupted to allow correction of the hypokalemia.

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###  Sodium bicarbonate

The administration of sodium bicarbonate solution to rapidly improve the acid levels in the blood is controversial. There is little evidence that it improves outcomes beyond standard therapy, and indeed some evidence that while it may improve the acidity of the blood, it may actually worsen acidity inside the body's cells and increase the risk of certain complications. Its use is therefore discouraged, although some guidelines recommend it for extreme acidosis (pH<6.9), and smaller amounts for severe acidosis (pH 6.9–7.0).

###  Cerebral edema

Cerebral edema, if associated with coma, often necessitates admission to intensive care, artificial ventilation, and close observation. The administration of fluids is slowed. The ideal treatment of cerebral edema in DKA is not established, but intravenous mannitol and hypertonic saline (3%) are used—as in some other forms of cerebral edema—in an attempt to reduce the swelling.

###  Resolution

Resolution of DKA is defined as general improvement in the symptoms, such as the ability to tolerate oral nutrition and fluids, normalization of blood acidity (pH>7.3), and absence of ketones in blood (<1 mmol/l) or urine. Once this has been achieved, insulin may be switched to the usual subcutaneously administered regimen, one hour after which the intravenous administration can be discontinued