**DIABETES, OBESITY, AND CANCER**

1. **DEFINE THE FOLLOWING TERMS**
* **KETOGENESIS:** Ketogenesis occurs when there is a high rate of fatty-acid oxidation in the liver.

Under metabolic conditions associated with a high rate of fatty-acid oxidation, the liver produces considerable quantities of acetoacetate and d(-)-3-hydroxybutyrate (β-hydroxybutyrate). Acetoacetate continually undergoes spontaneous decarboxylation to yield acetone. These three substances are collectively known as the ketone bodies (also called acetone bodies)

N.B- Ketogenesis is the normal pathway by which ketones are formed in the liver.

* **KETONAEMIA:** Ketonaemia is basically the presence of ketone bodies in the bloodstream, usually seen as a physiological consequence of lipid metabolism.
* **KETONURIA:**  This is a medical condition in which ketone bodies are present in the urine. It is seen in conditions in which the body produces excess ketones as an indication that it is using an alternative source of energy. Situations like starvation, and most commonly diabetes mellitus type 1.
1. **CONSEQUENCES OF KETOSIS:**  Ketosis basically occurs when there is excessive production of ketones due to starvation, which then leads to ketonuria and ketonaemia.

During periods of starvation the body loses its primary source of energy that is carbohydrate coupled with mobilization of free fatty acid (FFA).

This general pattern of metabolism is exaggerated to produce the pathologic states found in diabetes mellitus, the type 2 form. Non-pathologic forms of ketosis are found under conditions of high-fat feeding and after severe exercise in the post absorptive state.

1. **MANAGEMENT OF KETOACIDOSIS**

The clinical management of ketoacidosis involves;

* Fluid and electrolyte replacement therapy should be vigorous.

A 0.9% normal saline solution should be administered, usually 1L initially and then 1L over the next hour and then 2h and repeated at 4h. Monitoring central venous pressure may be useful to assess fluid replacement. Dextrose saline may be used when the plasma blood glucose concentration is less than 15mmol/L. If the plasma glucose concentration is more than 20mmol/L, 10U of insulin should be given.

A sliding insulin scale should be instigated.

Insulin is given either by continuous IV infusion or intermittent IM injections, as soon as the plasma glucose and potassium concentrations are known.

Once patient starts eating, subcutaneous insulin can be given instead.

If the metabolic acidosis (which is one of the clinical representations of diabetic ketoacidosis) is very severe (ph less than 7.0) , biocarbonate may be infused, but only until the blood ph rises to between 7.15-7.20. It is unnecessary and often dangerous to correct plasma biocarbonate concentration completely; it rapidly returns to normal following adequate fluid and insulin therapy.

**N.B the plasma potassium concentration should be measured before insulin is given. It is almost always raised at presentation due to the metabolic acidosis and reduced glomerular filtration rate, although total body potassium maybe reduced.**