1. A. Ketogenesis: Ketogenesis, or formation of ketone bodies, is an alternative catabolic pathway for active acetates. The amount of ketone bodies is small in normal individuals, but their levels become important in certain metabolic conditions. Acetoacetate, 3- hydroxybutyrate, and acetone are all ketone bodies.

B. Ketoanaemia: Presence of unrecognizable concentration of ketones present in plasma

C. Ketonuria: excretion of abnormally large amount of ketone bodies in the urine, characteristics if diabetes mellitus, starvation or other medical conditions

1. A. The "keto flu"

B. Diarrhoea

C. Reduced athletic performance

D. Ketoacidosis

E. Weight regain

F. Less muscle mass, decreased metabolism

1. The management of patients presenting with DKA includes a full clinical assessment, while regular monitoring of vital signs and consciousness levels using the Glasgow Coma Scale is essential (JBDS, 2013). Key areas in the management of DKA include:

-Restoring circulatory volume;

-Insulin therapy (fixed-rate intravenous insulin infusion)

-Correcting metabolic acidosis and electrolyte imbalances;

-Identifying and treating precipitating factors;

-Early involvement of the diabetes specialist team

 Key management points

-Fluid resuscitation with 0.9% sodium chloride

-Insulin infusion (fixed-rate intravenous insulin infusion) at 0.1 unit/kg/hr

-Close monitoring of vital signs, blood glucose, ketones, electrolytes and blood gases

-Continue FRIII until DKA has resolved before converting to subcutaneous insulin

-Give background insulin alongside IV to prevent rebound hyperglycaemia

-Involve the diabetes specialist team as soon as possible

**Restoring circulatory volume**

Fluid replacement is one of the most important initial therapeutic interventions in the management of DKA. Patients are usually dehydrated and correcting this deficit will result in significant metabolic improvement. The aims of fluid resuscitation

* Restore circulatory volume
* Clear ketones
* Correct electrolyte imbalance

Pulse and blood pressure should be used to assess the severity of dehydration, as hypotension (systolic BP<90mmHg) is likely to be due to low circulatory volume. Other causes such as heart failure, sepsis and factors such as age, sex and medication history should also be taken into consideration. Normal saline (0.9% sodium chloride) is recommended for fluid resuscitation. Rapid fluid replacement is usually required in the first few hours of treatment; most patients require between 500ml and 1L to be given rapidly. However, the rate of fluid replacement must be tailored to patients' clinical situation. Special attention must be paid to fluid balance in patients at high risk of complications -these include older people, pregnant women, children and young people (18-25 years), and those with heart and kidney failure.

**Insulin therapy**

The aim of insulin therapy in DKA management is to suppress ketogenesis, reduce blood glucose and correct electrolyte imbalance. Insulin therapy increases peripheral glucose use and decreases hepatic glucose production, thereby lowering blood glucose concentration. It inhibits the release of free fatty acids from adipose tissues and decreases ketogenesis

A continuous fixed-rate intravenous insulin infusion (FRIII) of 0.1 units/kg/hr is recommended. The recommendation for preparation of insulin infusion is 50 units of human soluble insulin made up with 50ml normal saline (0.9% sodium chloride). FRIII should continue until DKA is resolved. When the blood ketones are <0.6mmol/L, pH >7.3 and the patient is able to eat and drink, an appropriate subcutaneous insulin regimen should be recommenced background insulin should be continued along with the IV insulin infusion to reduce the risk of rebound hyperglycaemia when the IV insulin infusion is discontinued. If background insulin is discontinued, a subcutaneous dose must be given before the lIV insulin infusion is discontinued. The conversion to the subcutaneous insulin regimen should be planned around a mealtime; subcutaneous short- acting insulin should be given at the meal and then IV insulin discontinued one hour later.

**Correcting metabolic acidosis and electrolyte Imbalance**

Recommended the following metabolic treatment targets for DKA:

* Reduction in blood ketones of at least 0.5mmol/L/hr;
* Increase in venous bicarbonate by 3mmol/L/hr;
* Reduction in capillary blood glucose by 3mmol/L/hr;
* Maintenance of serum potassium at 4-5.5 mmol/L.

Blood glucose, ketones, electrolytes, including bicarbonate, and venous pH, should be monitored closely at or near the bedside. If the above targets for blood ketones and/or bicarbonate are not reached, the rate of the IV insulin infusion should be increased by 1 unit every hour until metabolic targets are achieved.

**Potassium**

Maintaining normal serum potassium and prevention of hypoglycaemia are important in the management of DKA as hypokalaemia (low potassium level) and hyperkalaemia (high potassium level) are both life-threatening conditions and common complications. Serum potassium is often high on admission but falls rapidly with insulin treatment, so regular monitoring is

essential and potassium should be added to IV infusions if serum potassium is <5.5mmol/L.

**Capillary blood glucose**

Prevention of hypoglycaemia is vital, so bedside blood glucose monitoring should be performed every 1-2 hours. It is sometimes necessary to give dextrose infusions to stabilise blood glucose levels;

this should be given concurrently with the sodium chloride infusions used to correct circulatory volume. To avoid complications related to rapid infusion it is important to monitor fluid balance and electrolytes closely. Regular assessment for complications such as cerebral oedema and fluid overload is vital.