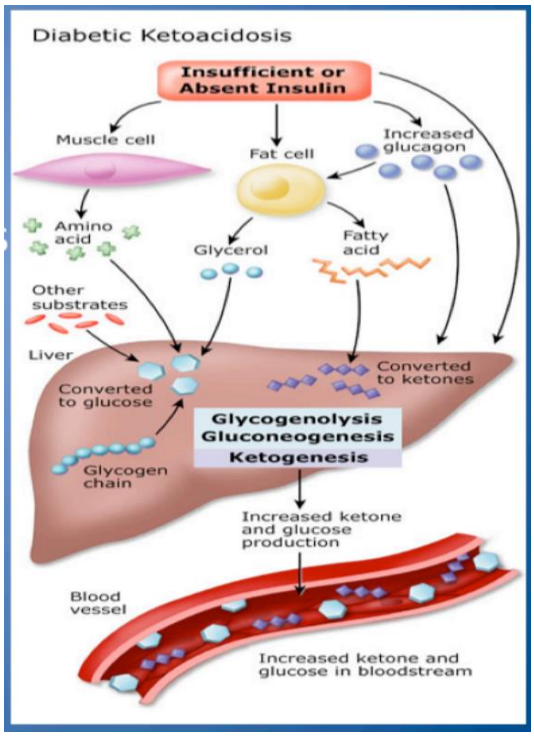
RESUSCITATION

DIABETIC EMERGENCIES

DIABETIC KETO ACIDOSIS (DKA)

DKA is a potentially life threatening complication of Diabetes Mellitus (DM). It is most commonly associated with Type 1, or insulin dependent (IDDM). It can be caused by inappropriate insulin administration, disease processes e.g. infection/ stroke/ MI, or medications such as corticosteroids.



- Lack of insulin prevents glucose uptake
- Stimulates glucagon release which causes glycogenolysis and gluconeogenesis.
- Elevated blood glucose acts as an osmotic diuretic
- Glucagon and lack of insulin stimulated gluconeogenesis causes lipolysis.
- Free fatty acids produced are converted to ketone bodies
- Bicarbonate buffering overwhelmed by acidic ketones
- Body attempts to compensate by respiratory mechanism and vomiting to expel acidic CO2 & HCl

Symptoms usually develop over 24 hours and include:

- Polydipsia
- Polyuria
- Sweating
- Fatigability
- Abdominal pain*
- Vomiting
- Confusion
- Symptoms of causative illness

DM is often first diagnosed in younger patients when they present in DKA.
In the elderly it is not uncommon for a 'silent' MI to precipitate DKA.
*Abdominal pain may be so severe and the abdomen rigid to examination that an 'Acute Abdomen' is suspected

Signs:

- Raised BM/ ketonaemia/ ketonuria
- Ill appearance
- Dry skin & mucous membranes
- Kussmaul respiration (gasping tachypnoea)
- Ketotic 'fruity' breath
- Confusion
- Tachycardia
- Hypotension
- Abdominal tenderness/ rigidity

Diagnosis:

- Usually initially suspected by **beside testing revealing a high blood glucose**
- **Urinalysis** (or serum biochemistry) shows **ketonuria**/ ketonaemia
- **Venous blood gas shows metabolic acidosis H+ >45 or HCO3- <18**

Management:

1. Mainstay of treatment is a **controlled IV insulin infusion and IV fluid rehydration**
2. B.M, Blood Gases and KCL should be regularly checked
3. Consider catheterisation to monitor urine output and consider central venous access
4. Screen for infection, check renal function and ECG

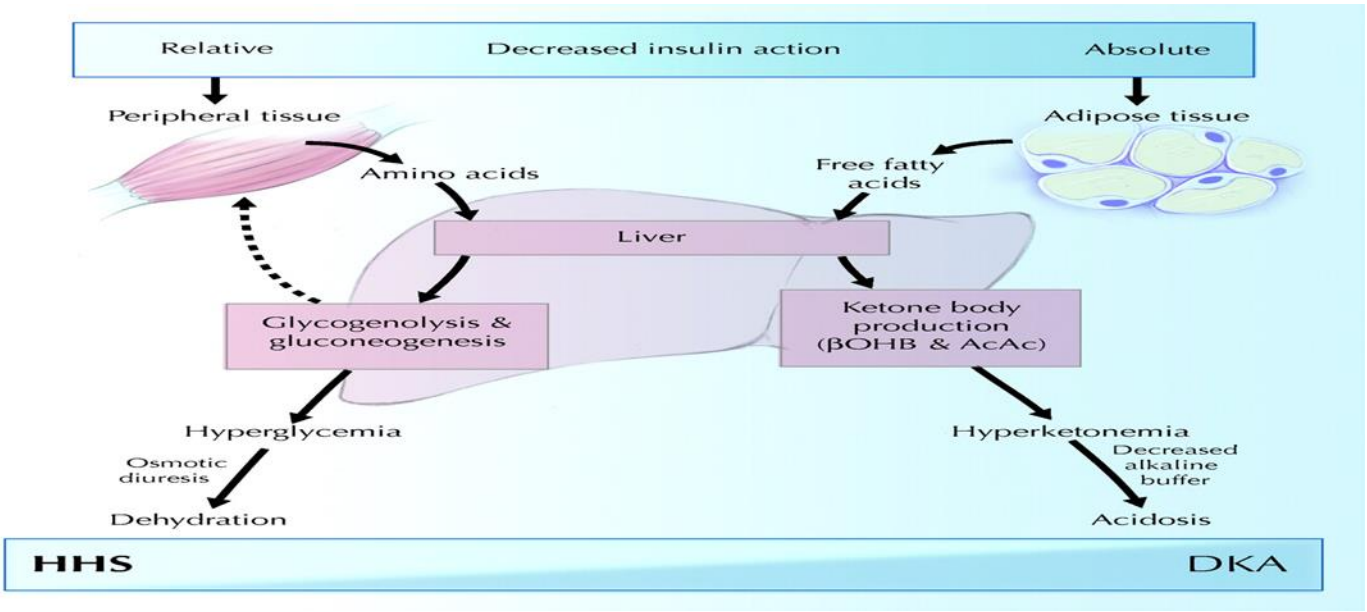
Most patients in DKA are managed in Medical High Dependency, discuss all patients with the Medical Registrar

Hyperosmolar Hyperglycaemic State (HHS)

HHS is a life- threatening complication usually of Type 2 Diabetes (T2DM).
It carries a significant morbidity and mortality, worse than that of DKA, probably due to likely co-morbidities of patients developing this condition.

Pathogenesis

- It is usually precipitated by another illness e.g. infection, MI, stroke
- This increases the body's glucose demand
- However the **relative lack of insulin** leads to hyperglycaemia
- Since **some** insulin is present, lipolysis and **ketogenesis are suppressed**
- However, the significant hyperglycaemia causes **osmotic diuresis** and subsequent dehydration.
- HHS is usually of **insidious onset** over several days
- By the time the patient presents they may already be **profoundly unwell and confused**



Diagnostic criteria:

- **Severe hyperglycaemia > 30mmol/L** (may exceed 40mmol/L)
- **Serum bicarbonate >15mmol/L** (i.e. not profoundly acidotic)
- **Urinary ketones ≤ +**
- **Total osmolality >340mosmol/L** (Osmolality = [2 x (Na + K) + Urea + Glucose)

Management:

- As for DKA the mainstay is IV fluid replacement and insulin
- A patient in HHS can be dehydrated by several litres, but be judicious with rehydration, especially in the elderly and those with cardiac disease.
- They have often taken several days to deteriorate to this level, and will have made some form of compensation to their fluid state. Aim for a slow reduction in blood glucose (2-3mmol/ hour) to prevent cerebral oedema caused by sudden osmotic shifts. This may require >48 hours.
- Monitor serum potassium and aim for a level of 4-5mmol/L