DIABETIC EMERGENCIES

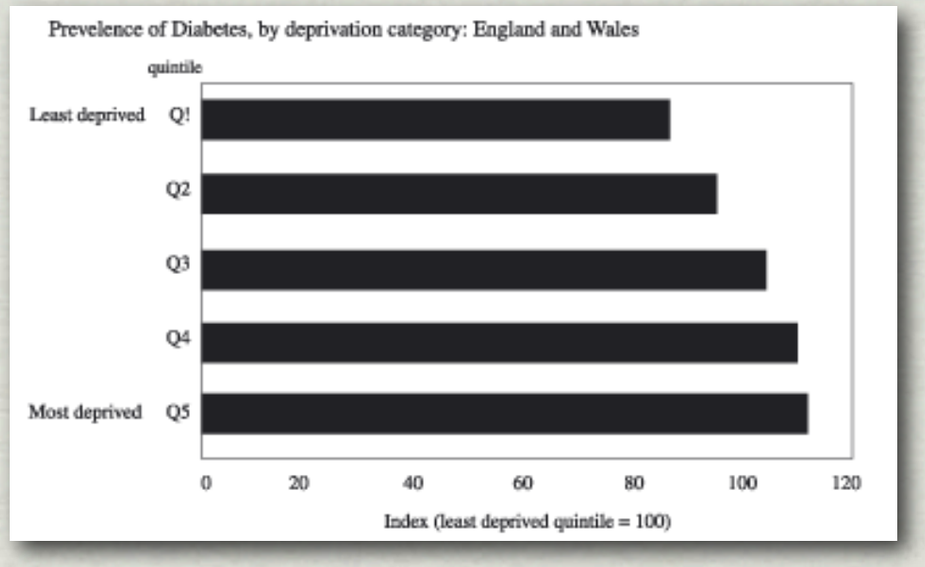
MR COLIN DIBBLE, CONSULTANT IN EMERGENCY MEDICINE, NMGH

Objectives

Understand:

- Pathophysiology
- Clinical presentation
- Hypoglycaemia
- DKA
- HONK
- Complications

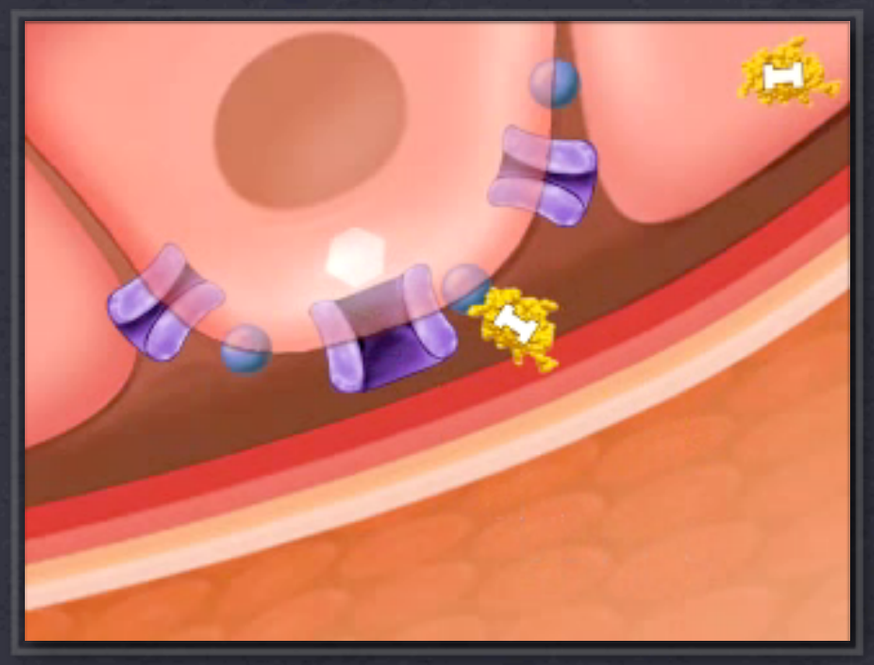
Introduction



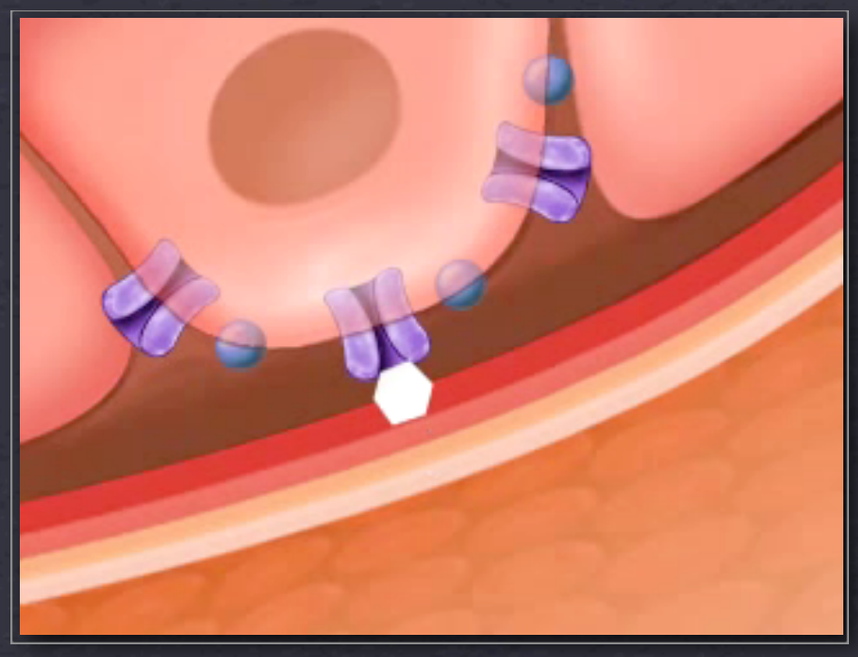
- Common, affects 1.3 million people and increasing, 2-3% population, 25-30/100000 kids, 9% of hospital costs
- Life threatening if not acted on promptly and correctly
- More common in ethnic minorities/older age groups, in less affluent, men
- There is a NSF for diabetes

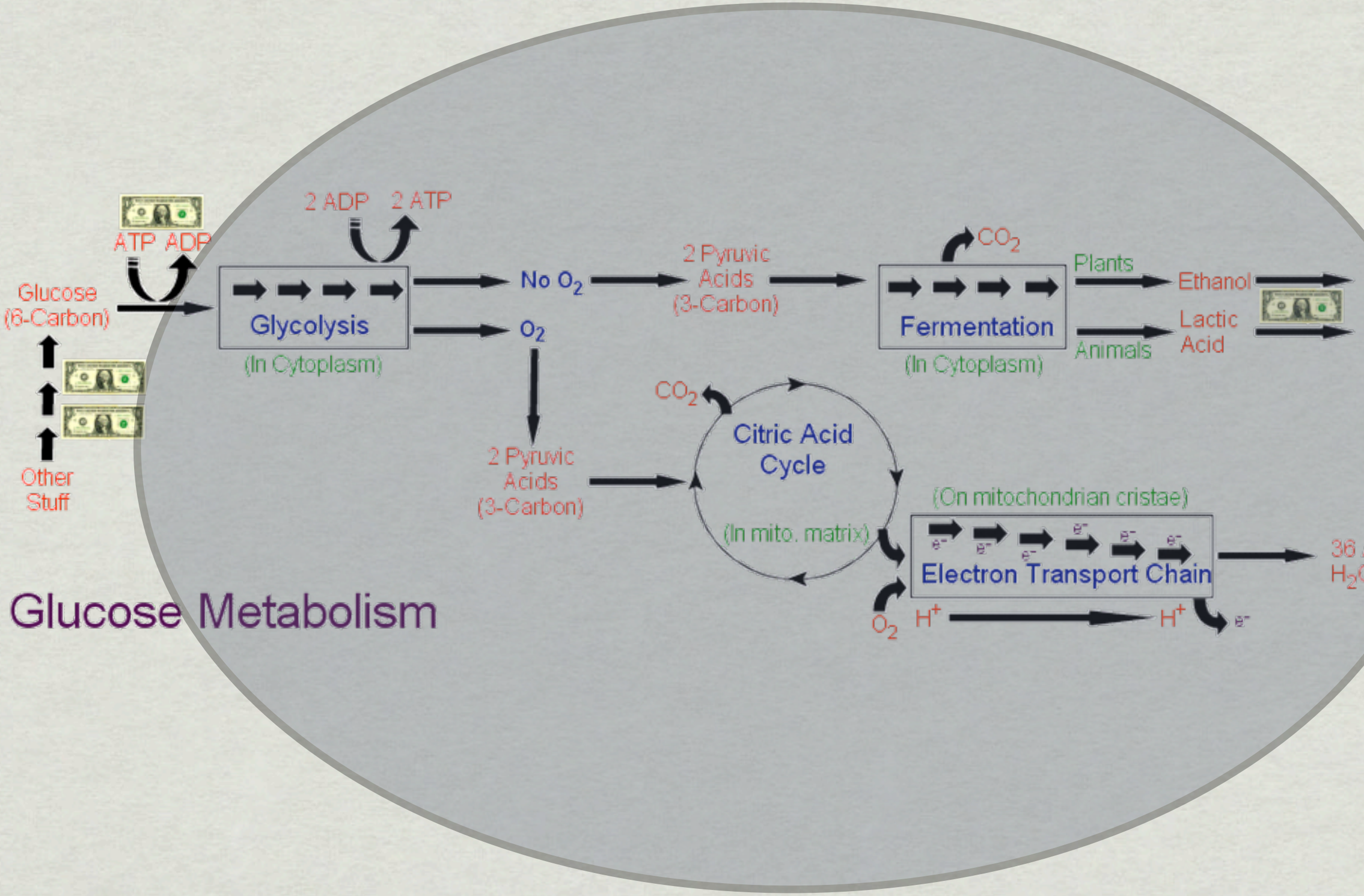
Pathophysiology

With Insulin



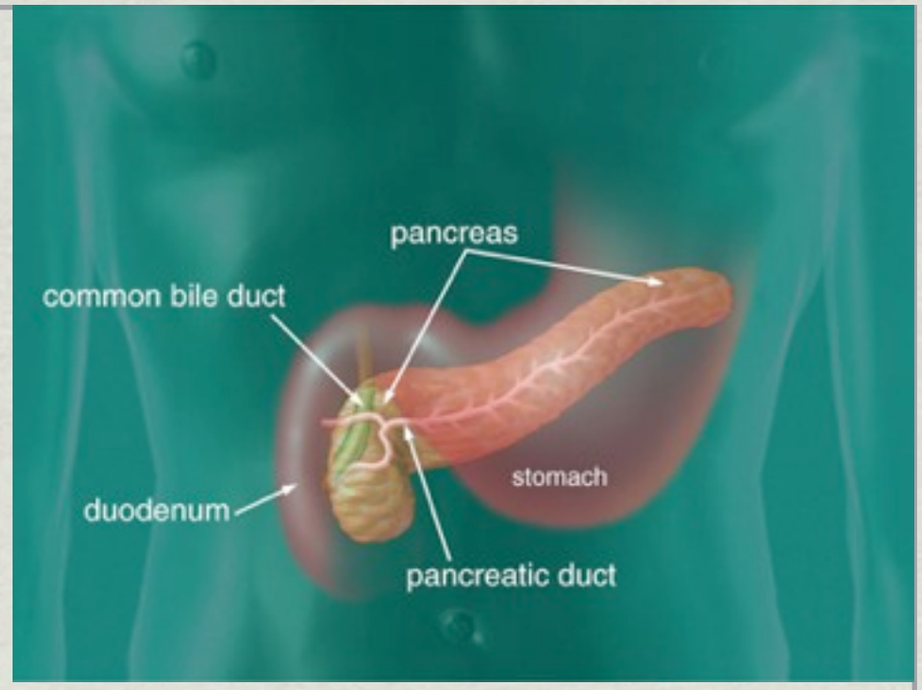
Without Insulin




ATP ADP
Glucose
(6-Carbon)
Other
Stuff
2 ADP 2 ATP
Glycolysis
(In Cytoplasm)
No O_2
O_2
2 Pyruvic
Acids
(3-Carbon)
2 Pyruvic
Acids
(3-Carbon)
CO_2
Fermentation
(In Cytoplasm)
Plants
Ethanol
Animals
Lactic
Acid
CO_2
Citric Acid
Cycle
(In mito. matrix)
(On mitochondrian cristae)
Electron Transport Chain
36 ATP
H_2O
O_2
H^+
H^+
Glucose Metabolism

Insulin Effects

- Reduced release of glucose by liver
- Increased uptake of glucose eg in skeletal muscle
- Inhibit triglyceride breakdown to glycerol and free fatty acids (& stimulate its formation)
- Inhibits protein breakdown to free amino acids (which can themselves increase hepatic production)



- Type 1: IDDM, usually juvenile onset, damage to pancreatic β cells, always need insulin, more prone to DKA. 30% twin concordance
- Type 2: NIDDM, usually adults >40yrs, more common in men, asians, obesity, lack of exercise, high calorie diet. Decreased insulin and insulin resistance. 80% twin concordance.
- Lack of insulin results in failure of uptake and utilisation of glucose in cells causing rise in blood glucose. Long term risks of CVS/renal/eye disease.
- Acute complications DKA, HONK, hypoglycaemia. Long term in DM: IHD, Blindness, renal failure, arterial dz-amputations

Clinical Presentation

- History polydispia, polyuria, weight loss with increased appetite
- May have reduced GCS, sweaty, clammy, tachycardia and fits (hypo)
- May be acidotic, shocked, coma (DKA). Coma without acidosis, raised osmolarity- HONK
- fasting BS ≥7mmol/l, random ≥11.1 mmol/l

Hypoglycaemia

=glucose <3mmol/l

- Neonates or DM missed meals or overdosed insulin/oral hypoglycaemic agents, increased activity
- EXPLAIN
  - EXogenous drugs
- Pituitary insufficiency
- Liver failure
- Addison's disease
- Islet cell tumours
- Non-pancreatic neoplasms

Hypoglycaemia

- Think of in any coma, may have confusion, personality change appear drunk, sweaty, tachycardia, fits, focal signs
- Check BM and formal sugar but treat
- Oral if awake
- SC/IV/IM glucagon 1mg NOT in liver disease, alcohol or malnourished or sulphonyl urea OD. Doesn't work if on beta blockers
- IV dextrose 50ml 50%, 250ml 10% (5ml/kg)

Hypoglycaemia

- If cause found, corrected and patient well, discharge
- If not responding further investigate eg CT and admit
- If OD eg sulphonyl ureas, admit (long duration of action). May need continuous infusion

DKA: Introduction

- More common in children, life threatening. May have developed over 2-3 days. Often compliance issues.

- Excess glycogenolysis with high glucagon levels and low insulin and raised sugar levels results in acidosis, fluid loss (osmotic diuresis), hypotension and shock

- Caused by four ‘I’s (Infection, Infarction, Insufficient insulin and Intercurrent illness)

- Death from hypokalaemia, cerebral oedema, & aspiration pneumonia

DKA: Features

- S/S of DM, plus D&V and abdominal pain
- Hyperventilation (Kussmaul) and acetone breath
- Altered GCS, dehydration and +/- shock
- Urinary glucose/ketones
- ↓pH, ↑K+, ↓Na, ↑Glucose, ↑urea/creatinine
- High anion gap: $(Na^+ + K^+ - HCO_3^- - Cl^-)\{>14\text{-}18\}$
- Check urine, CXR, blood cultures for infection

DKA: Management I

- **A**: may need RSI if airway risk from coma/vomiting etc
- **B**: 0_2 100% by mask
- **C**: IV access and initial resuscitation bolus fluids of Normal saline 1-2 litres (10ml/kg aliquots up to 30ml/kg). Get routine bloods/cultures/gases. May need CVP.
- **D**: RSI if less than 8, (also may need CT)

DKA: Management II

- Add potassium after bolus resuscitation fluid, 40mmol/l unless anuric
- Strict fluid record, Paeds: Maintenance plus deficit correction **over 48 hours** once resuscitated.

Maintenance requirements	Age	0 - 2 yrs	80 ml/kg/24 hrs
		3 - 5	70 ml/kg/24 hrs
		6 - 9	60 ml/kg/24 hrs
		10 - 14	50 ml/kg/24 hrs
		adult (>15)	30 ml/kg/24 hrs

- Adults: start NS 2L/2hrs, then 2 → 4 hourly. 5% dextrose when BM<11
- Insulin 6u/hr (0.1u/kg/hr) IV. NO loading dose

Clinical History
- polyuria
- polydipsia
- weight loss
- abdominal pain
- weakness
- vomiting
- confusion

Clinical Signs
- assess dehydration
- deep sighing respiration (Kussmaul)
- smell of ketones
- lethargy, drowsiness

Biochemical Signs
- ketones in urine or blood
- elevated blood glucose (>11mmol/l)
- acidaemia (pH<7.3)
- take blood also for electrolytes, urea
- perform other investigations if indicated

Confirm Diagnosis
Diabetic Ketoacidosis
Call Senior Staff

Shock
Reduced peripheral pulse volume
Reduced conscious level
Coma

Dehydration > 5%
Clinically acidotic
Vomiting

Dehydration < 5%
Clinically well
Tolerating fluid orally

Resuscitation
- Airway ± N/G tube
- Breathing (100% 0_2)
- Circulation (10ml/kg of 0.9% saline repeated until circulation restored, max 3 doses)

Intravenous therapy
- calculate fluid requirements
- correct over 48 hours
- 0.9% saline
- add KCL 20 mmol every 500 ml
- **insulin** 0.1U/kg/hour by infusion

Therapy
- start with s.c insulin
- give oral fluids

No improvement

Observations
- hourly blood glucose
- neurological status at least hourly
- hourly fluid input:output
- electrolytes 2 hours after start of IV-therapy, then 4-hourly

No improvement

Re-evaluate
- fluid balance + IV-therapy
- if continued acidosis, may require further resuscitation fluid
- check insulin dose correct
- consider sepsis

Neurological deterioration
Warning signs :
headache, irritability, slowing hea rate, reduced conscious level, specific signs raised intra-cranial pressure

blood glucose
< 15 mmol\L

exclude
hypoglycaemia
is it
cerebral oedema ?

Intravenous therapy
- change to 0.45% saline + dextrose 5%
- continue monitoring as above
- consider reducing insulin 0.05/kg/hour, but only when pH>7.3

Management
- give mannitol 1.0 g/kg
- call senior staff
- restrict I.V. fluids by 2/3
- move to ITU
- CT Scan when stabilised

Improvement
- clinically well, drinking well, tolerating food
- urine ketones may still be positive

Insulin
start subcutaneous insulin **then** stop intravenous insulin 1 hour later

HONK

Hyperosmolar non-ketotic

- Usually in older patients
- Calculate osmolality: [2(Na^++K^+)+Urea+Glucose], Osmolar Gap= Calculated-Actual (NR: 15-20)
- Similar treatment to DKA, (initially NS). Consider 0.45% NS if osmolarity >320mmol/l

Complications

- Cerebral Oedema
- Shock
- Hypokalaemia
- Aspiration pneumonia
- Pulmonary oedema

Questions?

Summary

- Insulin lack causes hyperglycaemia, high triglycerides and protein breakdown
- There is Type I (IDDM) & Type II (NIDDM)
- Hypoglycaemia can mimic drunkenness
- Glucagon not in malnourished for hypo
- DKA, look for infection and focus on fluids/ potassium and then insulin. Avoid rapid drop in glucose