MANAGEMENT OF HYPEROSMOLAR NONKETOTIC COMA (HONK)

Aetiology

- Dehydration syndrome due to hyperglycaemia
- Due to insufficient insulin to prevent hyperglycaemia but enough to prevent significant ketoacidosis
- Often develops during severe illness or physiologic stress in elderly patients either with no history of diabetes or with a history of NIDDM

Clinical Features

- Typical patient is an elderly nursing home resident
- Constitutional – malaise, weakness, myalgia
- Abdominal pain, nausea and vomiting
- Polyuria, polydipsia and loss of weight
- Signs of volume depletion and hypoperfusion +/- hypovolaemic shock
- Hyperventilation Kussmaul's breathing in severe acidosis), ketotic breath, pyrexia
- Pyrexia
- Seizures or focal neurologic signs, stupor, coma
- Evidence of precipitating causes
    - Sepsis eg UTI, respiratory tract infection, bacterial meningitis, retropharyngeal abscess, hepatobiliary sepsis. Examine for sources of sepsis include "hidden" sites eg scalp, back, auditory meatus, perianal region
    - Noncompliance with diet or insulin therapy
    - Trauma
    - AMI
    - CVA
    - Pancreatitis
    - Parenteral glucose admin, TPN
    - Drugs: glucocorticoids, thiazides, phenytoin, β-blockers, calcium channel blockers

Laboratory Findings

- Severe hyperglycaemia (BSL>20mmol/L)
- Plasma hyperosmolality >320mmol/L; may cause neurologic symptoms
  Plasma osmolality = 2 x (Na + K) + glucose (mmol/L) + urea (mmol/)
- Marked free water deficit 6-18 litres(urea:creatinine ratio increased)
- Secondary glycosuria
- Absence of significant ketoacidosis
    - Metabolic acidosis absent or mild
    - Serum anion gap normal or slightly elevated
- HypoNa/hyerpNa
- $HypoNa^+/hyperNa^+$

  - Hyponatraemia may be real (dilutional from osmotic effect of hyperglycaemia initially)
  - Or expected as a result of hyperglycaemia (correction factor: ΔNa = -0.016 x (glucose – 100)
- HypoK/HypoPO4/HypoMg

Investigations
- CBC, U/Cr/Na/K/Cl/PO_4/MgGlucose, ABG, serum osmolality
- Urine microscopy and culture
- Blood culture+/- lumbar puncture in a septic patient
- ECG
- CXR

Principles of Management
1. Aggressive fluid management
2. IV soluble insulin
3. Aggressive K replacement except in renal failure
4. Look for precipitating factors

Treatment
1. Fluid therapy
   - Aggressive fluid replacement about 2 L in 4 h, 1-2 L in the next 4 h; about 4-7 L in total within the first 24 h
   - NS is preferred because it helps to maintain intravascular volume and maintain peripheral perfusion. It may also reduce risk of cerebral oedema when sugar is lowered
   - ½ NS is used if serum Na >150-155mmol/L
   - Change to D5% with additional NaCl when blood sugar is <14mmol/L
   - Avoid alternating NS with dextrose drip as this will cause fluctuations in blood sugar
   - CVP/PCWP monitoring in patient with history of heart failure or renal impairment

2. Insulin therapy
   - Continuous IV insulin infusion (CI) through a pump is preferred as it offers smooth control. A more gradual normalization in the first 12-24 h period is preferred to avoid cerebral oedema. Loading dose of IV insulin does not result in better clinical outcome
   - Dilute 50 U of soluble insulin in 50 ml of gelofusine in a syringe and deliver it by an infusion pump
   - The average initial CI rate is about 0.1U/kg/h. It should preferably not exceed 10U/h for patient weight >70kg

- In severely insulin resistant cases eg morbid obesity, severe sepsis, high dose glucocorticoid treatment and TPN, a sliding scale with an initial rate of >10/h may be required
- Blood sugar should be monitored at 2-h intervals. Depending on whether the blood sugar improves 4 h later, the sliding scale may be switched to one with a higher initial CI rates (eg scale 1 to scale 3)

BSL (mmol/L)	CI Scale 1 (U/h)	Scale 2	Scale 3	Scale 4	Scale 5	Scale 6	Scale 7
≥ 22	3.0	4.0	5.0	6.0	7.0	8.0	10.0
18 -	2.5	3.5	4.0	5.0	6.0	6.0	8.0
14 -	2.0	3.0	3.0	4.0	5.0	5.0	6.0
12 -	1.5	2.5	2.5	3.0	4.0	4.0	4.0
10 -	1.0	2.0	2.0	2.0	3.0	3.0	3.0
8 -	1.0	1.5	1.5	1.5	2.0	2.0	2.5
6 -	0.5	1.0	1.0	1.0	1.5	1.5	2.0
4 -	0.5	0.5	0.5	0.5	1.0	1.0	1.5
< 4	Stop iv insulin infusion and inform doctor						

3. Potassium replacement
   - Aggressive IV K^+ infusion should be given in initial phase because of the shift of K^+ with glucose into the intracellular compartment with hydration and insulin treatment
   - In presence of ECG changes of hypokalaemia, 30 mmol of K^+ diluted in 1 litre NS or ½ NS should be given over 1 h before laboratory confirmation is available. Checking of K^+ usually available in our ICU – use ABG machine
   - Replacement may be given according to the scale below:

K^+ level (mmol/L)	< 3.0	3 -	4 -	5 -	> 5.5
IV K^+ (mmol/h)	30	20	10	5	0

   - Review K^+ infusion rate when blood sugar is normalizing (<14mmol/L). K^+ level should be measured every 4 h within the first 24 h

4. Bicarbonate/phosphate/magnesium
   - If measured phosphate or magnesium is low, replacement can be given
   - Mg^{++} may be considered if arrhythmias occur

5. Venous thromboembolism prophylaxis
   - Look under relevant chapter in the ICU manual

6. Termination of CI
   Continuous insulin infusion can be stopped when:
   - Blood sugar has been stabilized to ≤ 10 mmol/L on a steady insuling infusion rate for 12 h. The usual time frame is about 24-48 h of CI treatment
   - Dehydration is almost corrected
   - Precipitating factor is under control. If patient is still acutely ill, insulin resistance will be high and rebound in blood sugar is likely