**Hyperosmolar hyperglycemic syndrome(HHS)-高滲透高血糖症候群處理流程**

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# General principle

1. Also termed hyperosmolar hyperglycemia nonketotic coma (HHNK)or hyperglycemia dehydration syndrome.

2. Typical often occurred in middle-aged or elderly patients, has no DM history or mild DM.

3. Characterized by severe hyperglycemia (usually > 600 mg/dl), hyperosmolar ( usually> 350 mOsm/dl) and dehydration

4. Lack of severe acidosis (bicarbonate > 18 meq/L) and ketosis (ketone body<2+ )

# Predisposing factors

1. Occur spontaneously in about 5-7 % patients.
2. 90 % patients, some degree of renal insufficiency coexist
3. Infection
4. Drugs: steroid, potassium-wasting diuretics, phenytoin (inhibit insulin secretion), propnanolol, azathioprine, and diazoxide
5. Other medical conditions: eg.: CVA, subdural hemorrhage, acute pancreatitis, severe burn et al.
6. Use of concentrated glucose solutions
7. Endocrine disorders, such as Acromegaly, Cushing disease and thyrotoxicosis.

# Clinical presentation

1. **Signs and symptoms**
	1. Polyuria, polydipsia, weight loss and progress changes in state of conciousness over several days to weeks. (50 % patients)
	2. Those who present in coma (30 %) tend to have serum Osmo>340 mosm/L
	3. Other causes of coma should keep in mind
	4. Seizures occur in 5 % patients.
2. **Physical examination**
	1. Severe dehydration
	2. Various degree of neurologic deficits (eg. coma, transient hemiparesis, hyperreflexia and generalized areflexia) are common present.
	3. Findings associated with coexisting medical problems (eg. renal disease, heart disease or CVA) may be evident.

# Laboratory findings

1. Blood glucose from 600-2000 mg/dl are common
2. Markedly elevated serum Osmo (at least 320 mOsm/L and usually >350 mOsm/L)

 mOsm/L=2(Na) + blood glucose/18 + BUN/2.8

1. BUN can be elevated higher (70-90 mg/dl) than seen in DKA
2. Serum ketones are usually not detectable and not acidotic
3. Serum Na may be high, normal or low ( Na deficits in HHS are 5-10 mEq/kg )

For every 100 mg/dl rise in serum glucose, serum Na tends to be lower by 1.6 meq/L.

1. K deficiency also exists, calculated at about 5 mEq/kg

# Treatment

1. **General management technique**

**a**. A medical emergency

**b**. Flow sheet that includes data on glucose, serum ketones, electrolytes, BUN, Cr

Ca, P, blood gas, ketones should be used.

**c**. If the patient is in shock, stupor or coma,--keep airway ,use NG tube and foley

**d**. Frequent assessment of electrolytes

**e.** Careful observation of neurologic status (detect uncommon cerebral edema)

1. **Hydration and electrolyte therapy**

**1). Fluid replacement**

* 1. It is important to remember that it is the severe hyperglycemia and water shift from intracellular to the intravascular compartment that prevent s vascular collapse—so too rapid correction of hyperglycemia, potential hypovolemic shock may occur.
	2. Estimate fluid deficit (in liters) = 24L(1 – patient’s osm/280 mOsm/L)
	3. Generally half of the loss is replaced in the first 12 hours and the rest in the subsequent 24 hours.
	4. Immediate infusing NS to reexpand the extracellualr space ---- the recommended infusion rate 1L/hr (Fig.40.2) until blood pressure and pulse are stable and urine output is adequate; then if Na is normal or high,1/2 NS is recommended; if Na is low, NS is recommended (at a rate of 4-14ml/kg/h)
	5. Patients who required large volume replacement or have coexisting cardiac or renal disease require CVP or wedge pressure mearsurements.

**2). Electrolytes**

* 1. Once urine flow has been reestablished, K+ should be added ---KCL 20-40 mEeq/L is recommended with adjustments according to K+ measurement
	2. If patient oliguric and K+ is low ---KCL 10-30 mEq/L slowly over 1-2 hours with EKG monitor.
	3. K+ can be given by KCL and phosphate salt.
1. **Insulin therapy**
	1. Insulin is given as a continuous infusion at 0.1 U/kg per hour; some give an IV bolus of 0.1-0.15 U/kg first.
	2. Because insulin cause glucose fall, water shifts into cells and hypotension & oliguria can be further aggravated, thus some advocate delaying insulin therapy while infusing NS until vital signs improved.
	3. If additional stress is present (MI, CVA, or severe burns…), insulin dose might be increased (doubled q4h) if appropriate glucose reduction not noted.
	4. If glucose reach 200-250 mg/dl, shift to 5% dextrose or D5S.
	5. Usually, once stabilized, most patients can treat with diet or combination of diet and OHA.

# F. Complications

1. Mortality to be 10% - 40 %. It has been associated with convulsions, deep vein thrombosis, pulmonary embolus, pancreatitis, and renal failure.
2. Death usually attributes to an associated severe medical condition and not to the hyperosmolarity.