Hyperglycemic Hyperosmolar Syndrome

Other names:

- Hyperosmolar Hyperglycemic nonketotic syndrome (HHNK)
- Hyperosmolar coma
- nonketotic Hyperosmolar coma
- Hyperosmolar nonketotic syndrome
- Hyperosmolar Hyperglycemic nonketotic coma
- Nonketotic Hyperglycemic Hyperosmolar coma

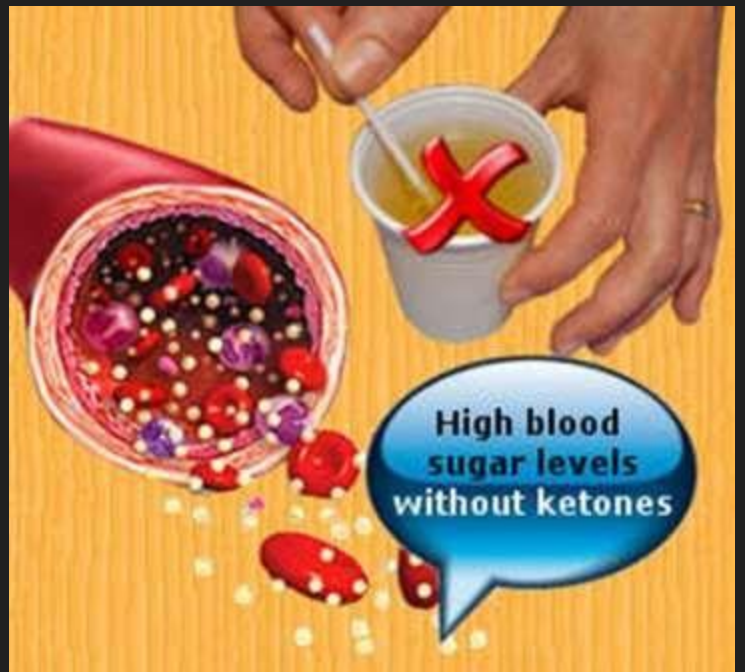
Definition :

- Is a life threatening emergency resulting from a lack of effective insulin, or severe insulin resistance, causing extreme hyperglycemia.
- HHS is precipitated by a stressor such as trauma, injury, or infection that increases insulin demand.
- Serum glucose:

  From 400 to 1800 mg/Dl

Pathophysiology:

- **there is enough insulin to prevent acidosis and formation of ketone bodies at the cellular level, but there is not enough insulin to facilitate transportation of all the glucose into the cells.**



Pathophysiology :

- glucose molecules accumulate in the blood stream, causing serum hyper osmolality with resultant osmotic diuresis and simultaneous loss of electrolytes, most notably potassium, sodium, and phosphate.

Pathophysiology:

- Patients may lose up to 25% of their total body water. Fluids are pulled from individual body cells by increasing serum hyper osmolality and extracellular fluid loss, causing intracellular dehydration and body cell shrinkage.

- The patient with HHNKC has severe hyperglycemia and azotemia without ketoacidosis. The intravascular volume is contracted, and the patient shows signs and symptoms of hypovolemia and severe dehydration. Both diffuse and focal central nervous system deficits may occur. These may include hallucinations, aphasia, nystagmus, hemianopsia, hemiplegia, hemi sensory deficits, and focal or grand mal seizures. Coma may ensue

As extracellular volume decreases……

- blood viscosity increases, causing slowing of blood flow.
- Thromboemboli are common (increased blood viscosity, enhanced platelet aggregation and adhesiveness, and possibly patient's immobility).
- Cardiac workload is increased and may lead to MI.
- Renal blood flow is decreased, potentially resulting in renal impairment or failure.
- Stroke may result from thromboemboli or decreased cerebral perfusion.

- Mortality rate of HHS ranges from 10%-50%, which is higher than that of DKA (1.2%-9%).

- Unlike DKA, in which ketoacidosis produces severe symptoms requiring fairly prompt hospitalization, symptoms of HHS develop more s l o w l y …. and often are nonspecific.

- The cardinal symptoms of polyuria and polydipsia are noted first but may be ignored by older persons or their families.

- Neurologic deficits may be mistaken for senility. The similarity of these symptoms to those of other disease processes common to this age group may delay differential diagnosis and treatment, allowing progression of pathophysiologic processes with resultant hypovolemic shock and multiple organ failure.
- As shock progresses, lactic acidosis may ensue due to poor perfusion.

HEALTH CARE SETTING

- Acute care (usually intensive care) is necessary .

Note:

- Patients with HHS may be olderthan 50 yr and have preexisting cardiac or pulmonary disorders.
- On the other hand ,The incidence of type 2 DM is increasing in children, teenagers, and young adults, so younger people are now presenting with HHS.
- Assessment results often cannot be evaluated based on accepted normal values.
- Evaluate results based on what is normal or optimal for the individual patient.
- Central venous pressure (CVP), heart rate (HR), and blood pressure (BP) should be evaluated in terms of deviations from the patient's baseline and concurrent clinical status.

DIAGNOSTIC TESTS

- *Serum glucose:* From 400 to 1800 mg/Dl
- *Serum chemistry:*
- • *Na+:* 125-160 mEq/L. Although the patient has lost large quantities of Na+, osmotic diuresis causes abnormally high blood concentration. The Na+ value may appear high despite probable Na+ deficits.
- • *K+:* less than 3.5 mEq/L.
- • *Cl–:* less than 95 mEq/L.
- • *Phosphorus:* less than 1.7mEq/L.
- • *Magnesium:* less than 1.5mEq/L.

- A1C of 8.5% (69 mmol/mol) equates to an estimated average glucose of ~200 mg/dL (11.1 mmol/L). Looser A1C targets above 8.5% (69 mmol/mol) are not recommended as they may expose patients to more frequent higher glucose values and the acute risks from glycosuria, dehydration, hyperglycemic hyperosmolar syndrome, and poor wound healing.

Clinical Findings: Comparison of Diabetic Ketoacidosis (DKA) & Hyperglycemic Hyperosmolar Syndrome (HHS):

	DKA	HHS
Type of diabetes	Usually type 1	Usually type 2
Signs, symptoms/physical assessment	Symptoms are a result mainly of hyperglycemia, intracellular hypoglycemia, hypotension or impending hypovolemic shock, and fluid-electrolyte imbalance with possible acid-base imbalance	Same as DKA
Neurologic	Altered LOC (confusion, lethargy, irritability, coma), strokelike symptoms (unilateral/bilateral weakness, paralysis, numbness, paresthesia), fatigue	Same as DKA; also possible seizures and tremors
Respiratory	Deep, rapid Kussmaul's respirations	Shallow, rapid (tachypneic) breathing
Cardiovascular	Tachycardia, hypotension, ECG changes	Same as DKA

Clinical Findings: Comparison of Diabetic Ketoacidosis (DKA) & Hyperglycemic Hyperosmolar Syndrome (HHS)—*cont'd*

	DKA	HHS
Metabolic/GI/endocrine	Polyuria, polyphagia, polydipsia, fruity "acetone" breath, abdominal pain, weight loss, fatigue, generalized weakness, nausea, vomiting	Polyuria, polyphagia, polydipsia, fatigue, generalized weakness, nausea, vomiting
Integumentary	Dry, flushed skin; poor turgor; dry mucous membranes	Same as DKA
VS monitoring	BP low (more than 20% below normal), HR more than 100 bpm, CVP less than 2 mm Hg (less than 5 cm H_2O), temperature normal	BP low (more than 20% below normal), HR more than 100 bpm, CVP less than 2 mm Hg (less than 5 cm H_2O), temperature possibly elevated
Diagnostic tests/laboratory values	Values reflect dehydration/metabolic acidosis (ketosis) secondary to hyperglycemia, abnormal lipolysis, and osmotic diuresis; fluid loss 6.5 L or more. Anion gap: more than 10	Values reflect dehydration secondary to hyperglycemia, osmotic diuresis, and possible lactic acidosis from hypoperfusion; fluid loss 9 L or more. Anion gap: normal
Hgb/Hct	Elevated	Same as DKA
Serum BUN/creatinine	Elevated	Same as DKA
Serum electrolytes	Initially elevated, then decreased	Same as DKA
Serum glucose	250-800 mg/dL (+ ketones)	400-1800 mg/dL (– ketones)
Serum ketones	Elevated	Normal; rarely slightly elevated
ABGs	pH 6.8-7.3, HCO_3^- 12-20 mEq/L, CO_2 15-25 mEq/L	pH 7.3-7.5, HCO_3^- 20-26 mEq/L, CO_2 30-40 mEq/L
Serum osmolality	300-350 mOsm/L	More than 350 mOsm/L

Clinical Findings: Comparison of Diabetic Ketoacidosis (DKA) & Hyperglycemic Hyperosmolar Syndrome (HHS) —*cont'd:*

	DKA	HHS
Urine glucose/acetone	Positive/positive	Positive/negative
Onset	Hours to days	More than 1 day
History/risk factors for development of crisis	Undiagnosed DM, infections, acute pancreatitis, uremia, insulin resistance *Medications:* digitalis intoxication; omission/reduction of insulin dosage; failure to increase insulin to compensate for stress of infections; injury, emotional problems, or surgery	Undiagnosed DM; infections, especially gram-negative; acromegaly; Cushing's syndrome; thyrotoxicosis; acute pancreatitis; hyperalimentation; pancreatic carcinoma; cranial trauma/ subdural hematoma; uremia, hemodialysis, peritoneal dialysis; burns, heat stroke; pneumonia; MI; stroke *Medications:* loop and thiazide diuretics (i.e., hydrochlorothiazide, chlorthalidone, furosemide), diazoxide; glucocorticoids (i.e., hydrocortisone, dexamethasone), propranolol (Inderal); phenytoin (Dilantin), sodium bicarbonate
Mortality	10% or less	10%–25%

Treatment:

- Therapy is primarily directed at replacement of fluid and electrolytes while supportive care is given. Insulin therapy is designed to slowly --over 24 to 48 hours--return the blood glucose level to a near normal range

COMPLICATIONS:

- arterial thrombosis
- stroke,
- renal failure heart failure, multiple organ failure
- cerebral edema,
- malignant dysrhythmias (due to fluid volume deficiency, which prompts poor end-organ perfusion),
- and gram negative sepsis (from infection that may have caused the problem to ensue).

Deficient Fluid Volume/ Risk for Electrolyte Imbalance/Risk for Shock

- Assess for signs and symptoms of hypovolemic shock, including
- changes in VS, Assess for poor skin turgor, dry mucous membranes, Measure I&O accurately and weigh the patient daily. Monitor
- urinary specific gravity and report findings of more than 1.020
- Administer intravenous (IV) fluids as prescribed. This ensures adequate rehydration. Usually, normal saline or 0.45% saline is administered until plasma glucose falls to 200-300 mg/dL. After that, dextrose-containing solutions usually are given to prevent rebound hypoglycemia.
- Initially, IV fluids are administered rapidly (i.e., up to 2000 mL infused during the first 2 hr of treatment and 150-250 mL/hr thereafter until BP stabilizes).

Risk for Infection:

- Immune system dysfunction results from the lack of energy production at the cellular level of all components of the immune system.
- Hyperglycemia indicates glucose has not been transported from the bloodstream into the cells & cellular energy production is impaired.
- All body system functions, including the immune system, are dysfunctional because cells involved with cellular and humoral immunity are unable to do 100% of the work required.
- The inability of glucose to “power” the cells suppresses the immune system, thereby increasing the risk of infection.

Ineffective Peripheral Tissue Perfusion (or risk for same):

- *related to* interrupted venous or arterial flow occurring with increased blood viscosity, increased platelet aggregation/adhesiveness, and patient immobility.

Thank you