Outline: Complications in Diabetes

Hypoglycemia

DKA

HHNS: Hyperglycemic Hyperosmolar Nonketotic Syndrome

Macrovascular CAD, HTN, PVD

Microvascular Nephropathy, Retinopathy, Neuropathy

Hypoglycemia

Low blood sugar (60)
Predisposing factors:
too much insulin
erratic absorption of insulin
sudden increase in activity
failure to eat on time
alcohol ingestion
some meds

Signs & Symptoms:
Systemic: shaky, irritable, nervous, > pulse, palpitations,
tremor, hunger, diaphoresis, pallor
Neurologic: HA, slurred speech, blurred vision, confusion,
behavior changes, lethargy, loss of consciousness, coma, death
Treatment Hypoglycemia
Mild 50 - 70 BG
Symptoms: tremors, anxious, tachy, sweaty, hunger, shaky, pallor
Give: 10-15 g. CHO (ie: orange juice) then follow with a complex CHO
(ie cheese/crackers, milk)
Moderate 30 - 50 BG
Symptoms: HA, irritable, drowsy, slurred speech,
blurred vision, double vision
Give 20-30 g CHO or Glucagon 1 mg SQ
or IM; Dextrose 50% 50cc IV push
Severe hypoglycemia < 30 BG
S/S: decreased LOC, coma, seizures, death
Treatment: administer 50% dextrose IVP

Increasing Hyperglycemia

Predisposing factors:

- Inadequate insulin
- Eating too much food
- Infection
- Acute illness
- Inactivity
- Stress

Treatment of Increasing Hyperglycemia

Signs & Symptoms: BG>250 , ↑ urination, ↓appetite,
weakness, fatigue, blurred vision, HA,
N/V, abdominal cramps, dry mouth
Management: give insulin SQ or IV, monitor BG, treat cause
Can lead to DKA

Diabetic Ketoacidosis
Definition: DKA occurs when there is insufficient insulin. Then, the body breaks down fats & proteins for NRG. During this oxidation of fatty acids, highly acidic ketone bodies build up, which are waste products of fat metabolism. This causes a systemic acidosis or Diabetic Ketoacidosis that requires hospitalization to correct.

Onset: Type 1 DM, sudden

Precipitating factors: infection, illness, stress, inadequate insulin

S/S: Polyuria (severe diuresis), polyphagia, polydipsia, elevated ketones, abdominal cramps, N/V, fatigue, dehydration, weight loss, HA, tachycardia, hypotension, fruity breath, Kussmaul respiration

DKA Diagnostic Parameters
Serum glucose: over 250 mg/dL
Serum ketones: positive
Urine ketones: positive
Serum pH: <7.3
Serum HCO3: <15mEq/L
Serum NA: initially low
Serum K: low in severe DKA
BUN: >20 mg/dL, elevated, dehydration

DKA Initial Treatment
BG: Q1hr
Insulin: IV drip, SQ regular insulin prn
IV NS fluids: 500cc hr + with K
After 2-3 hours or when BP normalizes, IV is changed to 0.45 NS at 200-300cc/hr w/ K+.
Dextrose: BG reaches 250 mg/dl dextrose added to avoid drop in glucose (causes cerebral edema).
Frequent BG
Insulin admin: SQ or IV
ICU: monitored bed required due to cardiac dysrhythmias from electrolyte imbalance
ABGs: monitoring acidosis level.

HHNS-Hyperglycemic Hyperosmolar Nonketotic Syndrome
Also called Hyperosmolar Nonketotic Coma
Metabolic condition that occurs in Type 2 DM
Similar to DKA but more severe
Mortality 10-40%

Onset: Type 2 DM, gradual

Precipitating factors: infection, stress, illness, poor fluid intake

S/S: severe osmotic diuresis,
dehydrated-poor turgor, dry skin, dry mucous mem;
tachycardia, hypotension, electrolyte loss-low NA, K, Phos.,
neuro s/sx-lethargy, change LOC, seizures, coma

HHNS Diagnostic Parameters
Serum glucose: 600-1200 mg/dL
Serum ketones: negative, normal
Urine ketones: negative, normal
Serum pH: <7.40, near normal
Serum HCO3: <20mEq/L, near normal
Serum NA: normal to low
Serum K: normal to low
BUN: elevated, dehydration

HHNS Treatment- Similar to DKA
BG is maintained around 250 until fluid deficit is corrected.
Electrolytes replaced
Rapid fluid replacement
Insulin IV used to tx BG (as opposed to reversing acidosis)
Treatment of underlying cause

Comparison of Diabetic Ketoacidosis (DKA) and Hyperglycemic Hyperosmolar Nonketotic Syndrome (HHNS)

Characteristics	DKA	HHNS
Pts most commonly affected	more common in type 1 diabetes	more common in type 2, esp elderly patients with type 2
Precipitating event	Omission of insulin; physiologic stress (infection, surgery, CVA, MI)	Physiologic stress (infection, surgery, CVA, MI)
Onset	Rapid (<24 h)	Slower (over several days)
Blood glucose levels	Usually >250 mg/dL	Usually >600 mg/dL (>33.3 mmol/L)
Arterial pH level	<7.3	Normal
Serum and urine ketones	Present	Absent
Serum osmolality	300–350 mOsm/L	>350 mOsm/L
Plasma bicarbonate level	<15 mEq/L	Normal
BUN and creatinine levels	Elevated	Elevated
Mortality rate	<5%	10–40%

Management

In addition to treating hyperglycemia, management of DKA is aimed at correcting dehydration, electrolyte loss, and acidosis.

Rehydration

important for maintaining tissue perfusion, enhances the excretion of excessive glucose by the kidneys.

may need as much as 6 to 10 L of IV fluid to replace fluid losses caused by polyuria, hyperventilation, diarrhea, and vomiting.

normal saline (0.45%) solution may be used for pts with HTN or hypernatremia or risk for HF pts. Switch to it after couple hours in any event

When BG level reaches 250 mg/dL, the IV solution may be changed to dextrose 5% in water (D5W) to prevent precipitous decline in the BG

Monitor fluid volume status w/ frequent VS: orthostatic BP and HR, lung assessment, and I/O. Initial urine output lags behind IV fluid intake

Plasma expanders may be necessary to correct severe hypotension that does not respond to IV fluid treatment.

Monitoring for signs of fluid overload is especially important for patients who are older, have renal impairment, or are at risk for heart failure.

Restoring Electrolytes major electrolyte of concern during treatment of DKA is potassium.

initial plasma concentration of K+ may be low, normal, or high. there is a major loss of K+ from body stores and an ICF to ECF shift,. Serum level of K+ decreases as K+ reenters the cells during the course of treatment of DKA; due to rehydration, which leads to increased plasma volume and subsequent decreases in the concentration of serum K+. Rehydration also leads to increased urinary excretion of K+. Insulin administration enhances the movement of K+ from the ECF into the cells.

Cautious but timely K+ replacement is vital to avoid dysrhythmias that may occur with hypokalemia.

Frequent (every 2 to 4 hours initially) ECGs and laboratory measurements of K+ are necessary during the first 8 hours of treatment.

K+ replacement is withheld only if hyperkalemia is present or if the patient is not urinating.

Because a patient's serum K+ level may drop quickly as a result of rehydration and insulin treatment, K+ replacement must begin once K+ levels drop to normal

Reversing Acidosis Ketone bodies accumulate as a result of fat breakdown. The DKA acidosis is reversed with insulin, which inhibits fat breakdown, thereby stopping acid buildup

Regular insulin, the only type of insulin approved for IV use, may be added to IV solutions

insulin is often infused separately from the rehydration solutions to allow frequent changes in the rate and content of the latter.

Insulin must be infused continuously until subcutaneous administration of insulin can be resumed.

Even if blood glucose levels are decreasing and returning to normal, the insulin drip must not be stopped until subcutaneous insulin therapy has been started. Increase the rate or concentration of the dextrose infusion instead

Blood glucose levels are usually corrected before the acidosis is corrected. Therefore, IV insulin may be continued for 12 to 24 hours, until the serum bicarbonate level increases (to at least 15 to 18 mEq/L) and until the patient can eat.).

Nursing Alert: flush the insulin solution through the entire IV infusion set and to discard the first 50 mL of fluid. Insulin molecules adhere to the inner surface of IV infusion sets.

Cardiovascular Complications DM: Macrovascular

- Coronary Artery Disease
- Cerebrovascular Disease
- Atherosclerosis
- 2X more MI & CVA in DM
- Hypertension
- Peripheral Vascular Disease

macrovascular complications result from changes in the medium to large blood vessels.

Blood vessel walls thicken, sclerose, and are occluded by plaque that adheres to the vessel walls.

Eventually, blood flow is blocked

Cerebral blood vessels are similarly affected by accelerated atherosclerosis.

Occlusive changes or the formation of an embolus elsewhere in the vasculature that lodges in a cerebral blood vessel can lead to TIA and strokes

Cardiovascular Complications: Microvascular

characterized by capillary basement membrane thickening.

Two areas affected by these changes are the retina and the kidneys

Diabetic Nephropathy

- S/S: urine albumin, changes in UO, hypertension, proteinuria, hypoglycemia
- Diagnostic Tests: 24 hr. urine, increased serum BUN & creatinine
- Management: prevention, low protein diet, control HTN

Diabetic Retinopathy

- S/S: microaneurysms in retinal capillaries, retinal edema, vision changes (red/black lines or spots), retinal detachment
- Dx.Tests: opthalmic examination
- Management: prevention, control BG, control HTN, laser photocoagulation, ASA

Diabetic Neuropathy

- S/S: affects every system, paresthesia, hyperesthesia, joint deformities, muscle atrophy, decreased reflexes, progressive tissue destruction
- Diagnostic tests: physical assessment specific to system
- Management : prevent control BG

Autonomic Neuropathies

Affect multiple systems

S/S: hypoglycemic unawareness

- GU: neurogenic bladder, incontinence
- GI: gastroparesis, diarrhea, constipation
- CV: postural hypotension, tachycardia, MI
- Reproductive: sexual dysfunction

DM Foot Care

Prevention:

Inspection:

Daily visual inspection

Daily foot hygiene

Protection:

Shoes-correct size & fit

Socks-always wear

Bare foot-never

Intervention

Podiatry-careful toe nail maintenance

Routine MD visits-include foot eval.

DM: Increased Infections

Rationale: **Complications**

Decreased oxygenation due to ASCVD & PVD

Vascular insufficiency limits inflammatory response

Decreased phagocytosis

Hyperglycemia slows wound healing

Nursing Diagnosis

- Imbalanced Nutrition: less / more
- Ineffective tissue Perfusion
- Risk for Infection
- Risk for impaired Skin integrity
- Risk for Injury
- Risk for unstable BG

Deficient Knowledge: Diabetes Management

NIC

- Teaching: Individual
- Teaching: Disease Process
- Teaching: Foot Care
- Teaching: Prescribed Diet

NOC

- Knowledge: DM Management
- Knowledge: Disease Process
- Knowledge: Treatment Regimen

Goals of DM Care

Optimal control of BG
Improve nutritional intake
Achieve & maintain desired weight
Improve diabetes self-care activities
Maintain absence of complications

Expected Outcomes

Avoids extremes of glucose levels
Exhibits BG levels within target range
Recognizes & treats hypoglycemia
Identifies food choices in prescribed diet
Demonstrates proper method of BG monitoring
Verbalizes importance of carrying Medical ID
Verbalizes rules for sick days
Describes individual treatment interventions: nutrition, exercise, BG monitoring, meds.

Interventions in acute care

Hospitalized patients with DM:
Hyperglycemia & Hypoglycemia during hospitalization
Alterations in diet
Hygiene
Stress