Diabetic Ketoacidosis Lecture Notes

Diabetic ketoacidosis is a life-threatening complication of diabetes associated with hyperglycemia, ketosis, and metabolic acidosis. It is characterized by elevated serum glucose level (greater than 250 mg/dL), decreased pH (less than 7.3), decreased serum bicarbonate level (less than 18 mEq/L), elevated ketone level, and dehydration.

DKA occurs mostly in people with type 1 diabetes mellitus (T1DM) but can also occur in those with type 2 diabetes mellitus (T2DM), especially those with new-onset diabetes

It may be precipitated by such events as an infection (such as pneumonia or urinary tract infection), discontinuation of or inadequate insulin therapy, myocardial infarction, stroke (cerebrovascular accident), sepsis, pancreatitis, cocaine use, and malfunctioning of continuous subcutaneous insulin infusion devices

Pathophysiology

- A hyperglycemic crisis occurs as a result of insulin deficiency (absolute or relative) and is accompanied by a subsequent increase in or imbalance of counterregulatory hormones (such as cortisol, epinephrine, glucagon, and growth hormone), enhancing hepatic gluconeogenesis, glycogenolysis, and lipolysis.
- As a result of insulin deficiency, glucose accumulates in the blood, and hyperglycemia results. As the cells starve, the liver converts glycogen to glucose; as levels exceed the kidney threshold, glucose spills over into the urine.
- Because cells can't use glucose, they metabolize protein, releasing potassium and phosphorus in the process. Amino acids convert to urea and glucose. Serum osmolarity increases, leading to glycosuria. Osmotic diuresis results, causing fluid and electrolyte imbalances and dehydration from massive fluid loss. Dehydration decreases the glomerular filtration rate, further raising the blood glucose level because glucose can't be excreted.
- The cells also convert fats into glycerol and fatty acids for energy. The fatty acids accumulate in the liver because they can't be quickly metabolized and convert into ketones (ketoacids). As ketones accumulate in the blood (ketonemia) and urine (ketonuria), acidosis occurs, leading to more tissue breakdown, more ketosis, and acidosis. The combined effects of serum hyperosmolarity, dehydration, and acidosis increase osmolarity in brain cells, producing an altered level of consciousness (LOC). Shock and mental status changes ensue and can progress to coma and death.
- In DKA, the serum concentration of ketones is commonly greater than 5 mEq/L, and hyperglycemia commonly results in blood glucose levels greater than 250 mg/dL. The disorder also results in acidosis, with a pH that can fall below 7.3. Serum bicarbonate levels can drop to 18 mEq/L or less, with levels below 5 mEq/L in severe DKA.
- For a patient with T1DM that's not well controlled or a patient who has an underlying disease process, a blood glucose level of 250 mg/dL isn't typically high enough to induce DKA; such a patient may have a higher blood glucose level, ranging from 300 to 400 mg/dL.

Causes

- Acute insulin deficiency with T1DM
- Medications (such as corticosteroids and thiazides), especially with T2DM
- Poor adherence to insulin injections, blockage of insulin infusion catheter, or insulin infusion pump failure
- Uncontrolled T1DM
- Illness with T1DM or T2DM
- Idiopathic

Risk Factors

- History of diabetes mellitus, especially T1DM
- Illness (such as myocardial infarction [MI] or stroke)
- Age over 40
- Infection, including coronavirus disease 2019 (COVID-19)
- Use of illicit drugs (such as, cocaine)
- Pregnancy
- Stress (medical, surgical, or emotional)
- Trauma
- Nonadherence to diabetes treatment plan
- Socioeconomic factors, such as inability to obtain medication or medical care

Incidence

- Females have a slightly higher incidence.
- DKA accounts for 50% of diabetes-related admissions in young people and 1% to 2% of all primary diabetes-related admissions.
- DKA commonly coincides with an initial diagnosis of T1DM.
- Although the exact incidence is unknown, DKA is estimated to occur in 1 of every 2,000 people with T1DM.

Complications

- Acute gastric dilation
- Cardiac arrhythmia
- Cerebral edema
- Stroke
- Complicated pregnancy
- Deep vein thrombosis
- Erosive gastritis
- Hypokalemia
- Hypophosphatemia
- Infection
- Late hypoglycemia
- MI
- Pulmonary edema

- Respiratory distress or failure

Assessment

History

- Possibly a recent illness, changes in diet or medications, or missed insulin dose
- Abdominal pain
- Anorexia or increased appetite
- Fatigue
- Confusion
- Decreased perspiration
- Generalized weakness
- Nocturia
- Polydipsia
- Polyuria
- Lethargy
- Malaise
- Nausea
- Vomiting
- Rapid weight loss (with new diagnosis of T1DM)
- Low socioeconomic status
- Nonadherence to diabetes treatment plan

Physical Findings

- Abdominal tenderness
- Altered mental status (confusion to coma)
- Decreased bowel sounds
- Decreased reflexes
- Dry mucus membranes, poor skin turgor
- Fruity odor to breath (acetone smell)
- Hypotension
- Hypothermia or fever (if infection is present)
- Increased capillary refill time
- Tachycardia
- Labored respirations, tachypnea, and Kussmaul respirations
- Signs and symptoms of precipitating cause (that is, source of infection or signs and symptoms of a possible concurrent illness, such as MI)

Diagnostic Test Results

Laboratory

- A fingerstick blood glucose or serum blood glucose level test shows hyperglycemia (usually 250 to 800 mg/dL).
- Potassium level (serum) is initially high and then drops rapidly with treatment. However,

severe acidosis can produce an artificially high potassium level.
- Sodium level (serum) may be low because of the dilutional effect of hyperglycemia. Sodium is suppressed at a rate of 1.6 mg/dL for every 100 mg/dL of glucose over normal, and hypertriglyceridemia may cause an artificially low sodium concentration.
  When glucose levels fall, serum sodium level rises accordingly.
- Chloride level (serum) and phosphate level (serum) are low.
- Blood urea nitrogen level test results and creatinine level (serum) are typically increased, although a markedly high serum ketone level may cause a falsely high serum creatinine level.
- Hyperamylasemia, hyperlipidemia, hypertriglyceridemia, or hypercholesterolemia may be present.
- Osmolality (serum) may be increased (greater than 290 mOsm/L), as may urine osmolality. Osmolalities greater than 330 mOsm/kg H2O are common in comatose patients; a lesser osmolality in a comatose patient may indicate the cause of altered LOC to be something other than DKA.
- An anion gap (AG) test shows an increased level (greater than 12 mEq/L).
- A complete blood count (CBC) with differential shows an increased white blood cell (WBC) count, even without signs of infection. However, a high WBC count (greater than 15,000/mL) suggests underlying infection.
- Urine, blood, or lumbar puncture cultures are positive if an infection is present.
- Serum ketone reagent strips are positive for ketosis. Nitroprusside reaction, which measures only acetoacetate, may not be strongly positive.
- Urinalysis reveals urine ketosis (which may initially be falsely negative), ketonuria, and glycosuria. (Urinalysis may identify only acetoacetate and not beta-hydroxybutyrate.)
- Arterial blood gas analysis findings reflect metabolic acidosis (pH less than 7.30) and a low bicarbonate level (less than 15 mEq/L); the more severe the DKA, the lower these levels are.
- A beta-hydroxybutyrate assay shows levels greater than 0.5 mmol/L; levels of 3 mmol/L or greater indicate the need for treatment.

Imaging

- Chest radiography is performed to rule out infection, such as pneumonia.
- Computed tomography scanning (brain) or magnetic resonance imaging of the head is performed to evaluate the possible source of the patient's altered LOC, such as cerebral edema, cerebral ischemia, or malignancy.

Diagnostic Procedures

- Electrocardiography (ECG) performed every 6 hours during the first day may reveal signs of acute MI, which can go unnoticed in patients with diabetes—particularly in those with autonomic neuropathy. In addition, T-wave changes can produce the first warning sign of altered serum potassium levels. Peaked T waves are present in hyperkalemia, whereas low T waves and an apparent U wave are present in hypokalemia.

Treatment

General

- Oxygen administration and airway management, with possible ventilator support
- Treatment of underlying or concurrent illnesses, infections, and conditions
- Blood glucose monitoring every hour while on continuous insulin infusion

Diet

- Nothing by mouth initially
- Gradual return to previous diet when nausea and vomiting are controlled; avoidance of foods with a high glycemic index

Activity

- Bed rest until hemodynamically stable
- Gradual increase in activity, as tolerated

Medications

- IV fluid administration, initially normal saline solution, with the addition of dextrose as glucose levels decrease
- IV infusion of insulin (regular) in moderate to severe DKA or, in less severe cases, insulin administered by subcutaneous route
- Potassium replacement, usually given IV, which begins when potassium level is 5.0 mg/dL or less and urine output is adequate

When administering potassium replacement by IV infusion, start with 30 to 40 mEq/L of IV fluids, with an increased dose (up to 60 mEq/L) if the potassium level is 3.5 mg/dL or lower. If the potassium level is 2.5 mg/dL or lower, expect to withhold insulin and replace potassium with 1 mEq/kg IV over 1 hour. When monitoring pH, for each 0.1 unit of pH, serum potassium will change by approximately 0.6 mEq in the opposite direction.

- Phosphorus replacement if levels are very low (less than 1.0 mg/dL), avoiding routine replacement of phosphorus because it can lead to hypocalcemia
- Magnesium replacement IV if magnesium level is 1.8 mg/dL or less and the patient is symptomatic
- Antibiotics, as appropriate, if infection is present

Nursing Considerations

Nursing Interventions

- Frequently assess vital signs and LOC, watching for signs and symptoms of increased intracranial pressure.
- Assess oxygen saturation level using pulse oximetry or arterial blood gas (ABG) analysis. Administer oxygen, as ordered, and anticipate the need for ventilator assistance.

- Consult with a specialized diabetes or glucose management team, if available.
- Administer IV fluid resuscitation with normal saline solution to correct extravascular and intravascular fluid losses and electrolyte losses and to dilute glucose levels and levels of circulating counterregulatory hormones.
- Assess for signs and symptoms of dehydration or fluid overload and electrolyte imbalances. Obtain serial ECGs, as indicated and per facility protocol, and assess for an underlying cardiac condition.
- Administer prescribed fluids and medications; assess the IV insertion site and ensure IV patency.
- Institute continuous cardiac monitoring; note and report any cardiac arrhythmias. Anticipate the need for insertion of an arterial catheter, a central venous catheter, or both to evaluate hemodynamic status.
- Frequently obtain specimens for laboratory testing, including blood glucose levels every hour until discontinuation of the insulin infusion and then every 2 to 6 hours thereafter; electrolyte levels (sodium, potassium, and bicarbonate) every 2 hours; phosphate, calcium, and magnesium level every 4 to 6 hours; and arteriovenous pH every 2 to 6 hours, as needed.
- Administer insulin therapy, as ordered, until ketosis subsides and the anion gap closes.
- Organize patient care and activities to provide uninterrupted rest; keep the patient's environment calm and quiet. Encourage the use of energy-conservation measures.
- Provide a diet to meet caloric and nutritional requirements, as tolerated. Enlist the aid of a dietitian to assist in nutritional counseling.
- Provide emotional support, and encourage verbalization of feelings to help reduce stress.
- Explore the reason for DKA development, if not readily apparent, including potential socioeconomic barriers, such as an inability to obtain medications or adequate medical care.

Monitoring

- Glucose and ketone levels
- Vital signs
- Neurologic, cardiac, and respiratory status
- Energy level and activity tolerance
- Dietary choices
- Daily weight
- Intake and output
- Blood chemistry findings
- Culture and sensitivity results
- Oxygen saturation
- ABG values

Associated Nursing Procedures

- Arterial puncture for blood gas analysis
- Blood glucose monitoring
- Cardiac monitoring
- Intake and output measurement
- IV bolus injection

- IV catheter insertion
- IV catheter removal
- IV insulin administration
- IV pump use
- IV secondary line drug infusion
- Nutritional screening
- Safe medication administration practices, general
- Subcutaneous injection
- 12-lead electrocardiogram (ECG)
- Urine glucose and ketone tests
- Urine pH measurement
- Urine specific gravity measurement
- Urine specimen collection from an indwelling urinary catheter (Foley)
- Weight measurement

Patient Teaching

General

Include the patient's family or caregiver in your teaching, when appropriate. Provide information according to their individual communication and learning needs. Be sure to cover:

- disease process, diagnostic testing, and treatment plan
- procedures, including the rationale for each procedure and its possible outcome
- possible contributing factors and causes
- prescribed medication therapy, including names, dosages, frequency of administration, expected results, and possible adverse effects
- proper administration of insulin, especially for a new diagnosis of T1DM
- dietary restrictions, including appropriate food choices that have a low glycemic index and the prescribed diet to manage diabetes
- signs and symptoms of hyperglycemia and hypoglycemia as well as measures to address each condition and signs and symptoms to immediately report to the practitioner
- actions to take if blood glucose alterations occur and appropriate responses to signs and symptoms
- medications that may affect blood glucose levels
- resources for diabetes self-management education and support, including mobile apps, simulation tools, digital coaching, and digital self-management interventions, as appropriate
- self-monitoring of blood glucose levels, including how to measure blood glucose levels and when to perform this test, use of a continuous blood glucose monitor, if appropriate
- specific glycemic targets based on individual criteria, such as potential risks, disease duration, life expectancy, comorbidities, vascular complications, patient preference, and support systems
- prevention of infection (such as the need to receive appropriate immunizations and avoid crowds during influenza season)
- foot care measures
- energy-conservation measures, including the need to plan for rest periods
- progressive resumption of activities and the importance of exercise
- importance of adhering to the diabetic treatment regimen to prevent future episodes of DKA

- importance of adhering to the follow-up schedule for evaluation and laboratory testing to determine the effectiveness of therapy.

Discharge Planning

- Participate as part of a multidisciplinary team to coordinate discharge planning efforts. This team may include a primary bedside nurse, social worker, care manager, nutritionist, diabetes educator, endocrinologist, pharmacist, and primary care practitioner.
- Assess the patient's and family's understanding of the diagnosis, treatment, prognosis, follow-up, and warning signs for which to seek medical attention.
- Identify the patient's formal and informal supports.
- Identify the patient's and family's goals, preferences, comprehension, and concerns about discharge.
- Confirm arrangements for transportation to the first few follow-up visits.
- Confirm that the patient has adequate access to appropriate food sources.
- Provide a list of prescribed drugs, including the dosage, prescribed time schedule, and adverse reactions to report to the practitioner. Provide the patient (and family or caregiver, as needed) with written information on the medications that the patient should take after discharge.
- Assess the patient's and family's understanding of prescribed medication, including dosage, administration, expected results, duration, and possible adverse effects.
- Assess the patient's ability to obtain medications; identify the party responsible for obtaining medications.
- Instruct the patient to provide a list of medications to the practitioner who will be caring for the patient after discharge; to update the information when the practitioner discontinues medications, changes doses, or adds new medications (including over-the-counter products); and to carry a medication list that contains all of this information at all times in the event of an emergency.
- Ensure that arrangements for home health care services are in place, as needed.
- Ensure that the patient and caregivers receive medical contact information.
- Ensure that the patient or caregiver receives a copy of the discharge instructions and that a copy is placed in the patient's medical record.
- Provide contact information regarding local support groups and services, including telehealth resources.
- Recommend that the patient receive influenza and pneumococcal vaccines and consider the hepatitis B, COVID-19, and herpes zoster vaccines, if indicated.
- Provide information on smoking cessation, as needed.
- Document the discharge planning evaluation in the patient's clinical record, including who was involved in discharge planning and teaching, their understanding of the teaching provided, and any need for follow-up teaching.