DM Emergencies

Dr. Zohair Alaseri, MD
FRCPc, Emergency Medicine
FRCPc, Critical Care Medicine
Intensivest and Emergency Medicine Consultant
Chairman, Department of Emergency Medicine
King Saud University Hospitals, Riyadh, KSA

The National Diabetes Data Group (NDDG) defines four major types of diabetes mellitus:

- type 1 diabetes mellitus
- type 2 diabetes mellitus
- gestational diabetes
- Impaired glucose tolerance (IGT) and its analogue, *impaired fasting glucose* (IFG).

The arabic numerals 1 and 2 be used to replace roman numerals I and II in the designation of types “one” and “two.”

DM Emergencies

Acute complications

- Hypoglycemia
- diabetic ketoacidosis
- hyperglycemic hyperosmolar nonketotic coma

DM Emergencies

The CNS cannot

- synthesize glucose
- store more than a few minutes' supply
- concentrate glucose from the circulation

Glucose is the predominant metabolic fuel used by the central nervous system (CNS).

Brief hypoglycemia can cause profound brain dysfunction

Prolonged severe hypoglycemia may cause cellular death

DM Emergencies

Glucose is derived from three sources:

- Intestinal absorption from the diet;
- *Glycogenolysis*, the breakdown of glycogen
- *Gluconeogenesis*, the formation of glucose from precursors, including lactate, pyruvate, amino acids, and glycerol

DM Emergencies

- Sulfonylurea oral hypoglycemic agents work, in part, by stimulating the release of insulin from the pancreas.

DM Emergencies

- Insulin receptors on the beta cells of the pancreas sense elevated blood glucose and trigger insulin release into the blood

DM Emergencies

- Under normal circumstances, insulin is rapidly degraded through the liver and kidney.
- The half-life of insulin is 3 to 10 minutes in the circulation.
- insulin is the major anabolic hormone pertinent to the diabetic disorder
- glucagon plays the role of the major catabolic hormone in disordered glucose homeostasis.

DM Emergencies

- The liver is essentially the sole source of endogenous glucose production.
- Renal gluconeogenesis contribute substantially to the systemic glucose pool only during prolonged starvation.
- Insulin inhibits hepatic gluconeogenesis and glycogenolysis

Glucose Regulatory Mechanisms

Glucoregulatory hormones include

- Insulin
- Glucagon
- Epinephrine
- Cortisol
- Growth hormone.

- Insulin is the main glucose-lowering hormone.
- Insulin suppresses endogenous glucose production and stimulates glucose use

Glucose Regulatory Mechanisms

- The body perceives a “fasting state” and releases *glucagon*, attempting to provide the glucose necessary for brain function

Glucose Regulatory Mechanisms

Epinephrine

- stimulates hepatic glucose production and limits glucose use
- acts directly to increase hepatic glycogenolysis and gluconeogenesis

Type 2 Diabetes Mellitus

- May remain asymptomatic for long periods and show low, normal, or elevated levels of insulin because of insulin resistance

Gestational Diabetes

- abnormal oral glucose tolerance test (OGTT) that occurs during pregnancy
- reverts to normal during the postpartum period or remains abnormal.

Impaired Glucose Tolerance

Impaired glucose tolerance (IGT) and its analogue, *impaired fasting glucose* (IFG).

This group is composed of persons whose plasma glucose levels are between normal and diabetic and who are at increased risk for the development of diabetes and cardiovascular disease

Pathophysiology and Etiology

- Type 1 diabetes results from a chronic autoimmune process that usually exists in a preclinical state for years.

- The classical manifestations of type 1, hyperglycemia and ketosis, occur late in the course of the disease, an overt sign of beta cell destruction.

CLINICAL FEATURES
Type 2

- The diagnosis of type 2 is often made because of an elevated blood glucose found on routine laboratory examination
- Decompensation of disease usually leads to hyperosmolar nonketotic coma rather than ketosis.

Maturity-onset diabetes

They have an autosomal dominant inheritance of their disease

are usually not obese

have a relatively mild course of disease.

DIAGNOSTIC STRATEGIES

Serum Glucose

- any random plasma glucose level greater than 200 mg/dL

- a fasting plasma glucose concentration greater than 140 mg/dL, or a 2-hour postload OGTT is sufficient to establish the diagnosis of diabetes

- Formal OGTTs are unnecessary

DIAGNOSTIC STRATEGIES

Glycosylated Hemoglobin HbA1

- provides insight into the quality of glycemic control over time
- Given the long half-life of red blood cells, the percentage of HbA1c is an index of glucose concentration of the preceding 6 to 8 weeks,
- normal values approximately **4% to 6%** of total hemoglobin

DIAGNOSTIC STRATEGIES

Urine Glucose

- Two types: reagent tests and dipstick tests

Dipsticks

- Both falsely high and falsely low urine glucose readings can also occur.

DIAGNOSTIC STRATEGIES

Urine Ketones

Urine ketone dipsticks use the nitroprusside reaction

good test for acetoacetate

but does not measure β-hydroxybutyrate.

usual acetoacetate/β-hydroxybutyrate ratio in DKA is 1:2.8,

it may be as high as 1:30, in which case the urine dipstick does not reflect the true level of ketosis.

DIAGNOSTIC STRATEGIES

Dipstick Blood Glucose

Hematocrits

- below 30% cause false high readings
- above 55% cause false low readings

HYPOGLYCEMIA

- the most dangerous acute complication
- Hypoglycemia may be associated with significant morbidity and mortality.
- Severe hypoglycemia is usually associated with a blood sugar level below 40 to 50 mg/dL

HYPOGLYCEMIA

- Diabetic patients using insulin are vulnerable to hypoglycemia because of insulin excess and failure of the counterregulatory system

Hypoglycemia may be caused by

- missing a meal
- increasing energy output
- increasing insulin dosage.

Precipitants of Hypoglycemia in DM Patients

- Addison's disease
- Antimalarials
- Decrease in usual food intake
- Ethanol
- Factitious hypoglycemia
- Hepatic impairment
- Increase in usual exercise
- Insulin
- Malnutrition Old age
- Oral hypoglycemics
- Pentamidine
- Propranolol
- Recent change of dose or type of insulin or oral hypoglycemic
- Salicylates
- Sepsis

HYPOGLYCEMIA

Hypoglycemia unawareness

is a dangerous complication of type 1 diabetes probably caused by previous exposure to low blood glucose concentrations

HYPOGLYCEMIA

- single hypoglycemic episode can reduce neurohumoral counterregulatory responses to subsequent episodes.

Factors associated with recurrent hypoglycemic attacks include

- overaggressive or intensified insulin therapy
- longer history of diabetes
- autonomic neuropathy
- decreased epinephrine secretion or sensitivity

HYPOGLYCEMIA

Somogyi phenomenon

- common problem associated with iatrogenic hypoglycemia in the type 1 diabetic patient.

- *excessive insulin dosage, which results in an unrecognized hypoglycemic episode that usually occurs in the early morning while the patient is sleeping.*
- *The counterregulatory hormone response produces rebound hyperglycemia, evident when the patient awakens.*
- *Often, both the patient and the physician interpret this hyperglycemia as an indication to increase the insulin dosage, which exacerbates the problem*

HYPOGLYCEMIA

Clinical Features

- Symptomatic hypoglycemia occurs in most adults at a blood glucose level of 40 to 50 mg/dL.

Signs and symptoms of hypoglycemia are caused by excessive secretion of epinephrine and CNS dysfunction and include

- Sweating
- Nervousness
- Tremor
- Tachycardia
- Hunger
- neurologic symptoms ranging from bizarre behavior and confusion to seizures and coma

HYPOGLYCEMIA

Management

- In alert patients with mild symptoms, consumption of sugar-containing food or beverage orally is often adequate

HYPOGLYCEMIA

Management

- ABC
- one to three ampules of 50% dextrose in water (D50W)

- Augmentation of the blood glucose ampule of D50W may range from less than 40 to more than 350 mg/dL

HYPOGLYCEMIA

Management

- All patients with severe hypoglycemic reactions require aspiration and seizure precautions

HYPOGLYCEMIA

Management

- D50W should not be used in infants or young children because venous sclerosis can lead to rebound hypoglycemia.

- In a child younger than 8 years it is advisable to use 25% (D25W) or even 10% dextrose (D10W).

- The dose is 0.5 to 1 g/kg body weight or, using D25W, 2 to 4 mL/kg.

HYPOGLYCEMIA

Management

- 25-75 g glucose as D50W (1-3 ampules) IV
- Children: 0.5-1 g/kg glucose as D25W IV (2-4 mL/kg)
- Neonates: 0.5-1 g/kg glucose (1-2 mL/kg) as D10W

HYPOGLYCEMIA

Management

If unable to obtain IV access:

1-2 mg glucagon IM or SC; may repeat q20min

Children: 0.025-0.1 mg/kg SC or IM; may repeat q20min

- The onset of action is 10 to 20 minutes
- peak response occurs in 30 to 60 minutes.
- It may be repeated as needed.

Glucagon is ineffective in causes of hypoglycemia in which glycogen is absent, notably alcohol-induced hypoglycemia.

Nondiabetic Hypoglycemia

Postprandial & Fasting

Postprandial hypoglycemia

The most common cause of is alimentary hyperinsulinism, such as

gastrectomy, gastrojejunostomy, pyloroplasty, or vagotomy.

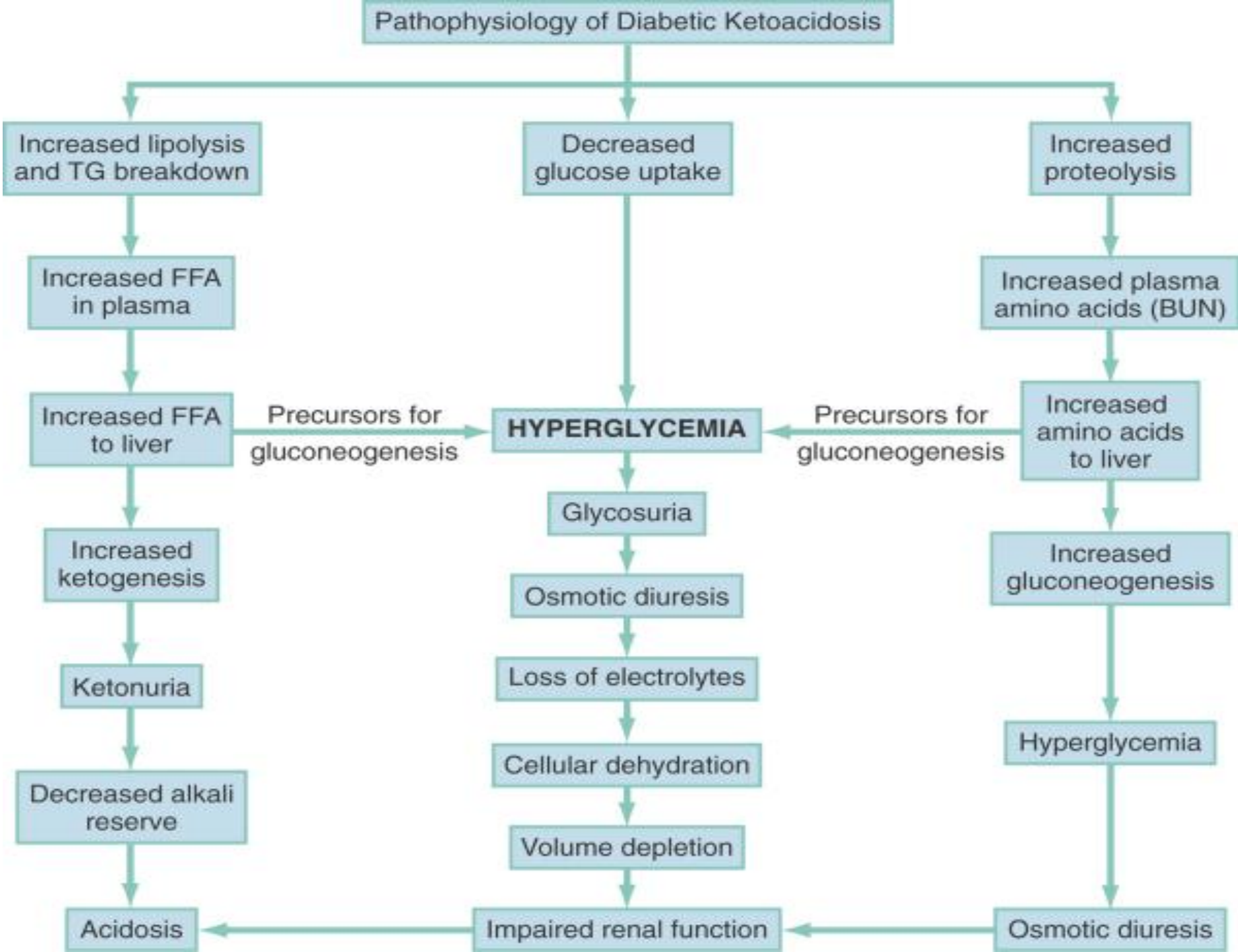
Fasting hypoglycemia is caused when there is an imbalance between glucose production and use.

- The causes of inadequate glucose production include hormone deficiencies, enzyme defects, substrate deficiencies, severe liver disease, and drugs.

- Causes of overuse of glucose include the presence of an insulinoma, exogenous insulin, sulfonylureas, drugs, endotoxic shock, extrapancreatic tumors, and a variety of enzyme deficiencies.

DIABETIC KETOACIDOSIS

- Insulin deficiency and Glucagon excess


Pathophysiology of Diabetic Ketoacidosis
Increased lipolysis and TG breakdown
Decreased glucose uptake
Increased proteolysis
Increased FFA in plasma
Increased plasma amino acids (BUN)
Increased FFA to liver
Precursors for gluconeogenesis
HYPERGLYCEMIA
Precursors for gluconeogenesis
Increased amino acids to liver
Glycosuria
Increased ketogenesis
Osmotic diuresis
Increased gluconeogenesis
Loss of electrolytes
Ketonuria
Hyperglycemia
Cellular dehydration
Decreased alkali reserve
Volume depletion
Acidosis
Impaired renal function
Osmotic diuresis

DIABETIC KETOACIDOSIS

- The hyperosmolarity produced by hyperglycemia and dehydration is the most important determinant of the patient's mental status

DIABETIC KETOACIDOSIS

- Glucose in the renal tubules draws water, sodium, potassium, magnesium, calcium, phosphorus, and other ions from the circulation into the urine.
- This osmotic diuresis combined with poor intake and vomiting produces the profound dehydration and electrolyte imbalance associated with DKA

DIABETIC KETOACIDOSIS

- Sodium level is normal or low.

Potassium, magnesium, and phosphorus deficits are usually marked.

- As a result of acidosis and dehydration, however, the initial reported values for these electrolytes may be high.
- Hypokalemia may further inhibit insulin release.

DIABETIC KETOACIDOSIS

The cells

- unable to receive fuel substances from the circulation.

- They accelerate proteolysis such that large amounts of amino acids are released to the liver and converted to two-carbon fragments.

DIABETIC KETOACIDOSIS

Insulin deficiency results in

1---activation of a hormone-sensitive lipase that increases circulating (FFA) levels and converted in the liver to acetoacetate and β-hydroxybutyrate

2---there is decrease in the peripheral tissue's use of ketones as fuel.

The combination of increased ketone production with decreased ketone use leads to ketoacidosis.

DIABETIC KETOACIDOSIS

Ketoalkalosis

- vomiting for several days and in some with severe dehydration and hyperventilation.

- The finding of alkalemia, however, should prompt the consideration of alcoholic ketoacidosis

DIABETIC KETOACIDOSIS

DKA can also occur in

- type 2 patients
- may be associated sepsis or gastrointestinal (GI) bleeding.

Approximately 25% of all episodes of DKA occur in patients whose diabetes was previously undiagnosed

DIABETIC KETOACIDOSIS

Clinicaly

Elevated temperature

- is rarely caused by DKA itself and suggests the presence of sepsis.

Abdominal pain

In children

- usually idiopathic
- probably caused by gastric distention or stretching of the liver capsule
- resolves as the metabolic abnormalities are corrected.

In adults

- more often signifies true abdominal disease.

Typical Laboratory Values in Diabetic Ketoacidosis (DKA) and Hyperglycemic Hyperosmolar Nonketotic Coma (HHNC)

	DKA	**HHNC**
Glucose (mg/dl)	>350	>700
Sodium (mEq)	low 130s	140s
Potassium (mEq)	~4.5–6.0	~5
Bicarbonate (mEq)	<10	>15
BUN (mg/dL)	25–50	>50
Serum ketones	Present	Absent

DIABETIC KETOACIDOSIS
Investigations

- Euglycemic DKA (blood glucose <300 mg/dL) in 18% of patients

- Venous pH is not significantly different from arterial pH in patients with DKA

DIABETIC KETOACIDOSIS
Investigations

Metabolic acidosis with anion gap is secondary to

- **Elevated plasma levels of acetoacetate and β-hydroxybutyrate**
- Lactate
- FFAs
- Phosphates
- Volume depletion

a well-hydrated patient with DKA may have a pure hyperchloremic acidosis and no anion gap

DIABETIC KETOACIDOSIS
Investigations

- Despite initial potassium levels that are normal to high, a total potassium deficit of several hundred milliequivalents results from potassium and hydrogen shifts.

DIABETIC KETOACIDOSIS
Investigations

The serum *sodium* value

- is often misleading in DKA.
- When *hyperglycemia* is marked, water flows from the cells into the vessels to decrease the osmolar gradient, thereby creating dilutional hyponatremia.

- Lipids also dilute the blood, thereby further lowering the value of sodium.

- Newer autoanalyzers remove triglycerides before assay, thus eliminating this artifact.

- the true value of sodium may be approximated by adding 1.3 to 1.6 mEq/L to the sodium value on the laboratory report for every 100 mg/dL glucose over the norm

DIABETIC KETOACIDOSIS
Investigations

Acidosis and the hyperosmolarity

shift *potassium, magnesium*, and *phosphorus* from the intracellular to the extracellular space.

Dehydration produces hemoconcentration, contributes to

- normal or high initial serum potassium, magnesium, and phosphorus readings in DKA, even with profound total deficits
- True K is by subtracting 0.6 mEq/L from the laboratory potassium value for every 0.1 decrease in pH noted in the ABG analysis

DIABETIC KETOACIDOSIS
Investigations

- Leukocytosis more closely reflects ketosis than the presence of infection.

- Only the elevation of band neutrophils has been demonstrated to indicate the presence of infection

with a sensitivity of 100% and a specificity of 80%.

DIABETIC KETOACIDOSIS
Investigations

Amylase

- The diagnosis of pancreatitis is confounded by the usually elevated urine and serum amylase levels in DKA.

- Typically, this is salivary amylase, but most laboratories are not equipped to make this distinction.

- A serum lipase determination helps to distinguish pancreatitis from elevated salivary amylase levels.

DIABETIC KETOACIDOSIS
Management

- ABC
- Once the patient is intubated, hyperventilat to prevent worsening acidosis.
- Shock requires aggressive fluid resuscitation with 0.9% saline solution rather than pressors.
- Search for other possible causes of shock

DIABETIC KETOACIDOSIS
Management

- Supplement insulin.
  NOOOOOOOOOO Bolus

- Maintenance: 0.1 U/kg/hr regular insulin IV

- Change IV solution to D5W 0.45% normal saline when glucose ≤300 mg/dL

DIABETIC KETOACIDOSIS
Management

Rehydrate.

- 1-2 L normal saline IV over 1-3 hours
- Children: 20 mL/kg normal saline over first hour
  Follow with 0.45% normal saline

DIABETIC KETOACIDOSIS
Management

Correct electrolyte abnormalities.

Sodium

- Correct with administration of normal saline and 0.45% normal saline.

Potassium

- Ensure adequate renal function.
- Add 20-40 mEq KCl to each liter of fluid.

Phosphorus

- Usually unnecessary to replenish

Magnesium

- Correct with 1-2 g $MgSO_4$ (in first 2 L if magnesium is low).

DIABETIC KETOACIDOSIS
Management

- Search and correct underlying precipitant.

Monitor progress and keep meticulous flow sheets.

- Vital signs
- Fluid intake
- urine output
- Serum glucose
- K+, Cl-, HCO3+, CO2, pH
- Amount of insulin administered

DIABETIC KETOACIDOSIS
Management

- Admit to hospital or intensive care unit.

Consider outpatient therapy in children with reliable caretaker *and*

- Initial pH > 7.35
- Initial HCO3- ≥ 20 mEq/L
- Can tolerate PO fluids
- Resolution of symptoms after treatment in emergency department
- No underlying precipitant requiring hospitalization

DIABETIC KETOACIDOSIS
Management

Insulin

- DKA cannot be reversed without insulin
- low-dosage insulin therapy has proved as effective as high-dosage therapy
- High dosages of insulin have potentially harmful effects, including a greater incidence of iatrogenic hypoglycemia and hypokalemia
- Because the half-life of regular insulin is 3 to 10 minutes, IV insulin should be administered by constant infusion rather than by repeated bolus

DIABETIC KETOACIDOSIS
Management

Insulin

Reduction of glucose levels in children should be gradual

Children are more likely than adults to develop cerebral edema in response to a rapid lowering of plasma osmolarity.

DIABETIC KETOACIDOSIS
Management

Insulin

- resistance occurs rarely in diabetic patients and requires an increase in dosage to obtain a satisfactory response esp in obese

DIABETIC KETOACIDOSIS
Management

Fluid resuscitation

The severely dehydrated patient is likely to have a fluid deficit of 3 to 5 L.

- No uniformly accepted formula
- alone help to lower hyperglycemia
- Acidosis also decreases after fluid infusion alone.
  - diminishing the formation of lactate.
  - Increased renal perfusion promotes renal H+ loss, and the improved action of insulin in the better-hydrated patient inhibits ketogenesis.

DIABETIC KETOACIDOSIS
Management

Potassium

- Should be administered while the laboratory value is in the upper half of the normal range.
- Renal function should be monitored.

- In patients with low serum potassium at presentation, hypokalemia may become life threatening when insulin therapy is administered. IV potassium should be aggressively administered in concentrations of 20 to 40 mEq/L as required.

DIABETIC KETOACIDOSIS
Management

Phosphorus

- no clinical benefit from the routine administration of in DKA has been shown

DIABETIC KETOACIDOSIS
MagnesiumManagement

Magnesium

- Deficiency may exacerbate vomiting and mental changes, promote hypokalemia and hypocalcemia, or induce fatal cardiac dysrhythmia.
- it is reasonable to include 0.35 mEq/kg of magnesium in the fluids of the first 3 to 4 hours, with further replacement dependent on blood levels and the clinical picture.

DIABETIC KETOACIDOSIS
Management

Morbidity

largely iatrogenic:

(1) hypokalemia from inadequate potassium replacement

(2) hypoglycemia from inadequate glucose monitoring and failure to replenish glucose in IV solutions when serum glucose drops below 250 to 300 mg/dL

(3) alkalosis from overaggressive bicarbonate replacement

(4) congestive heart failure from overaggressive hydration

(5) cerebral edema probably caused by too rapid osmolal shifts.

Poor prognostic signs include

- hypotension, azotemia, coma, and underlying illness

DIABETIC KETOACIDOSIS
Management

Morbidity

The primary causes of death

infection (especially pneumonia)

arterial thromboses

shock.

The decrease in mortality demonstrates that appropriate therapy can make a difference.

DIABETIC KETOACIDOSIS
Management

Complication

Cerebral edema

- should be suspected when the patient remains comatose or lapses into coma after the reversal of acidosis.
- It generally occurs 6 to 10 hours after the initiation of therapy.
- There are no warning signs, and the mortality is currently 90%.
- associated with
  - low PCO2
  - high BUN concentration
  - use of bicarbonate.

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- Marked hyperglycemia, hyperosmolarity and dehydration, and decreased mental functioning that may progress to frank coma.
- Ketosis and acidosis are generally minimal or absent.
- Focal neurologic signs are common.
- DKA and HHNC may occur together

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

HHNC)

- May occur in patients who are not diabetic,
- Burns
- Parenteral hyperalimentation
- Peritoneal dialysis
- Hemodialysis

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- The urine is extremely hypotonic
  - urine sodium concentration between 50 and 70 mEq/L, compared with 140 mEq/L in extracellular fluid.

- This hypotonic diuresis produces
  - profound dehydration, leading to hyperglycemia, hypernatremia, and associated hypertonicity

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- The reason for the absence of ketoacidosis in HHNC is unknown.
    - FFA levels are lower than in DKA, thus limiting substrates needed to form ketones

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

Clinically

- extreme dehydration
- Hyperosmolarity
- CNS findings predominate

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- 20% of patients have no known history of type 2 diabetes.

The most common associated diseases are CRF, gram-negative pneumonia, GI bleeding, and gram-negative sepsis.

- Of these patients, 85% have underlying renal or cardiac impairment as a predisposing factor.

- Arterial and venous thromboses often complicate the picture.

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- On average, the HHNC patient has 9 L in the 70-kg patient.
- The depression of the sensorium correlates directly with the degree and rate of development of hyperosmolarity.
- Some patients have normal mental status.
- Seizures are usually associated with neurologic findings, especially epilepsia partialis continua (continuous focal seizures) and intermittent focal motor seizures.
- Stroke and hemiplegia are also common.

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

Laboratory findings

- Glucose greater than 600 mg/dL
- Serum osmolarity greater than 350 mOsm/L.
- The BUN concentration is invariably elevated

- May have a metabolic acidosis secondary to some combination of lactic acidosis, starvation ketosis, and retention of inorganic acids attributable to renal hypoperfusion.

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)

- Initial serum sodium readings are inaccurate because of hyperglycemia

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)
Management

- Same as in DKA
- Overly rapid correction of serum osmolarity may predispose to the development of cerebral edema in children

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)
Management

- *Phenytoin* (Dilantin) is contraindicated for the seizures of HHNC because it is often ineffective and may impair endogenous insulin release.

- Phenytoin-induced HHNC even occurs in nondiabetic patients.

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)
Management

- Low-dosage subcutaneous *heparin* may be indicated to lessen the risk of thrombosis, which is increased by
  - Volume depletion
  - Hyperviscosity
  - Hypotension
  - Inactivity

HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA (HHNC)
Management

- All patients with HHNC must be hospitalized

DM Medications

Sulfonylureas

- increase insulin secretion by binding to specific beta cell receptors
- works best in patients with early onset of type 2 diabetes and fasting glucose less than 300 mg/dL.
- contraindicated in patients with known allergy to sulfa agents.

DM Medications

Metformin

- works by decreasing hepatic glucose output, leading to decreased insulin resistance and lower blood glucose.
- does not cause hypoglycemia
- contraindicated in patients with renal insufficiency and metabolic acidosis.
- should be withheld for 48 hours before or after administration of iodinated contrast media because of the risk of acidosis.
- must be used with caution in patients with hypoxemia, liver compromise, and alcohol abuse.

These patients are at increased risk for developing lactic acidosis, which has a 50% mortality rate.

NEW-ONSET HYPERGLYCEMIA

- Glucose greater than 200 mg/dL but are not ketotic.

- These patients with normal electrolytes may be treated with IV hydration alone or with insulin, often reducing the glucose to 150 mg/dL.

NEW-ONSET HYPERGLYCEMIA

- In reliable patients whose initial glucose is greater than 400 mg/dL
- An HbA1c value should be obtained
- Start with

  sulfonylureas is appropriate; glyburide (2.5 to 5 mg once daily) or glipizide (5 mg once daily)
- In obese patients or those in whom sulfonylureas are contraindicated, metformin may be an alternative.
- Follow-up should be stressed and warning signs of hypoglycemia discussed.