

CHAPTER 11

HYPERGLYCEMIC EMERGENCIES IN ADULTS

Author:

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Diabetic Ketoacidosis (DKA) and the Hyperosmolar Hyperglycemic Syndrome (HHS) are acute complications of diabetes which are potentially life threatening. Mortality rates are <5% in DKA and 15% in HHS but increase with age. Both syndromes occur as a result of relative lack of insulin action combined with increased counterregulatory hormones. Although DKA generally occurs in individuals with Type 1 DM, it can occur in those with Type 2 DM during extreme stress and counterregulation. Although both syndromes can occur in their pure forms, often there is some overlap in presentation.

11.1 Definition

Diabetic Ketoacidosis (DKA)

- The triad of hyperglycemia, hyperketonemia and metabolic acidosis.
- Potentially life-threatening emergency requiring prompt medical attention.
- Helpful diagnostic criteria: serum glucose > 14, arterial ph < 7.3, serum bicarbonate < 18 mEq/l , anion gap >12, moderate ketonuria or ketonemia.

Hyperosmolar Hyperglycemic Syndrome (HHS)

- Severe hyperglycemia often associated with altered mental status.
- A medical emergency with a high morbidity and mortality.
- Helpful diagnostic criteria: serum glucose > 33 (often extremely high), arterial ph > 7.3, serum bicarbonate > 18, anion gap < 12 (or normal), osmolality > 320 mOsm/kg, mild or no ketonuria or ketonemia.

11.2 Pathogenesis

- The relative or absolute lack of insulin action coupled with high levels of the counterregulatory hormones glucagon, catecholamines, cortisol and growth hormone leads to increased hepatic and renal glucose production and impaired glucose utilization in peripheral tissues.
- Severe hyperglycemia leads to glucosuria, causing an osmotic diuresis with loss of water, sodium, potassium and other electrolytes in both DKA and HHS. The degree of dehydration is generally more severe in HHS.
- In DKA, insulin deficiency results in lipolysis and the release of free fatty acids into the circulation from fatty tissues which are oxidated to ketone bodies (β hydroxybutyrate and acetoacetate) by the liver. This results in ketosis and metabolic acidosis.
- Lactic acidosis.

11.3 Precipitating Factors

- Insulin omission (intentional or accidental), pump malfunction in those on CSII.
- New onset Type 1 DM or previously undiagnosed Type 2 DM.

- Insulin insufficiency during intercurrent illness such as infection (especially pneumonia and urinary tract infections), myocardial infarction, cerebral vascular accidents, pulmonary embolism, perioperative stress.
- Drugs, especially glucocorticoids.

11.4 Clinical Presentation

Diabetic Ketoacidosis

- Usually develops rapidly, in less than 24 hours.
- Nausea and vomiting are common symptoms.
- Abdominal pain, potentially severe and mimicking an acute abdomen.
- Hyperventilation (Kussmaul's breathing) secondary to acidosis.
- Signs of dehydration, tachycardia and hypotension.
- Ketone breath (fruity, smell similar to nail polish remover).
- May be alert, lethargic or have a decreased level of consciousness if hyperosmolar.

Hyperosmolar Hyperglycemic Syndrome

- Symptoms more insidious, developing over days including polyuria, polydipsia, weight loss.
- Altered mental status and coma more common than in DKA because of higher osmolarity.
- Seizures or focal neurologic deficits occur rarely.
- HHS should be considered in any patient who is obtunded and severely dehydrated.
- Signs of significant volume depletion are present and the serum sodium is frequently abnormally high.
- Signs of sepsis may be present.
- Metabolic acidosis is seldom present.
- The calculated or measured serum osmolarity is 330 mosm/kg H₂O or greater.

11.5 Clinical Assessment and Lab Investigations

- Detailed history and physical, especially volume status and level of consciousness, presence of underlying or complicating illness.
- Serum glucose, electrolytes, urea, creatinine, arterial blood gases, serum and urine ketones to establish diagnosis. Calcium, phosphate and magnesium may be abnormal.
- **Establish a flow chart** to monitor glucose, electrolytes, anion gap [$\text{Na} - (\text{Cl} + \text{HCO}_3)$], ABGs, insulin dose, fluid and electrolyte replacement, and mental status. Glucose levels should be done hourly, electrolytes and ABGs at least every 2-4 hours depending on the degree of derangement until the acidosis clears.
- CXR, EKG, body fluid cultures as necessary.

11.6 Treatment

Goals of Therapy

- Provide appropriate monitoring
- Improve circulatory function and tissue perfusion
- Decrease glucose and osmolality towards normal
- Clear serum and urine of ketones
- Correct electrolyte abnormalities

- Identify and treat precipitating factors

Diabetic Ketoacidosis

- Admission to intensive care unit if hemodynamically unstable, severe acidosis (i.e. pH <7.0) or to provide adequate monitoring.
- **Hydration** with normal saline (0.9%) initially, usually 1 - 2 litres given rapidly to achieve hemodynamic stability. Thereafter fluid replacement should be based on the clinical status, estimates of total body fluid depletion and electrolytes. Usually half-normal saline or half-normal saline and a glucose infusion are required.
- **Insulin** by intravenous infusion, 0.1 to 0.2 units/kg body weight bolus followed by 0.1 to 0.2 units/kg/hour infusion of Regular insulin. Titrate insulin infusion rate based on the anion gap. Aim to normalize anion gap or normalize the pH. Do not stop insulin infusion until ketosis cleared.
- Add **glucose** intravenously if serum glucose falls below 13 mmol/L. Give adequate intravenous glucose to avoid hypoglycemia.
- **Potassium** is given to replete potassium salts. The average amount of loss is 5 meq/kg. Give potassium chloride unless patient is hyperkalemic (K >5.5) or anuric. Rates of 10-30 meq/hour of KCl are usually required but higher rates are required if hypokalemia occurs. Aim to keep the K between 3.5 and 5. In the setting of marked hyperkalemia, EKG monitoring of T waves is suggested.
- At presentation the patient may be hyperphosphatemic, but becomes hypophosphatemic as treatment ensues. **Phosphate** is not routinely given unless there is severe hypophosphatemia. Potassium phosphate 30-60 mmol over 24 hours may be given in this case.
- **Bicarbonate** does not improve outcome and is not routinely used unless there is severe acidemia, pH <6.9 or there cardiovascular instability and severe acidemia. NaHCO₃ 44-88 mmol infused over 0.5 - 1 hours may given in this case.
- The usual subcutaneous insulin regimen can be resumed once the DKA is cleared and the patient is well and ready to eat. The insulin infusion should be maintained for an overlap period of 2-3 hours to allow for adequate insulinization with subcutaneous insulin.

Hyperosmolar Hyperglycemic Syndrome

- Treatment and monitoring is similar to DKA
- Hydration with isotonic (0.9% NaCl) or half normal saline (0.45%NaCl) 1 -1.5 litres (15 -20 ml/kg body weight/h) during the first hour should begin to restore fluid status.
- If the corrected serum sodium is normal or low, isotonic saline should be used, if serum sodium is high, half-normal saline should be used.
- This is followed by slower infusion of half normal saline 4-14 ml/kg/h taking cardiopulmonary status into consideration. Iatrogenic volume overload should be prevented.
- Blood pressure, clinical volume assessments must be monitoring frequently.
- A central venous line may be required to monitor volume and pressure.
- Too rapid a correction of electrolytes/osmolality may be associated with mental deterioration. Correction of osmolality should not exceed 3 mOsm/kg/h.
- Rehydration alone can produce a significant drop in blood glucose by improving renal plasma flow and glomerular filtration.
- However, in most cases, insulin is required to treat the hyperglycemia, especially given the relative deficiency of insulin and the limitations to aggressive fluid resuscitation in those with compromised cardiac function.
- An insulin bolus of .05 to .1 units/kg iv bolus followed by .05 to .1 units/kg/h depending on the response is advised.
- If the patient has severe metabolic acidosis, measurements of serum lactate is indicated, patients should be treated as in lactic acidosis.

- Careful attention must be given to potassium replacement and treatment of the underlying illness.
- Following acute treatment, oral hypoglycemics can be started or reinitiated. In those not well controlled on oral medications, insulin therapy should be instituted.

11.7 Complications of Therapy

- **Hypokalemia** is common, and severe hypokalemia should be prevented.
- **Hyperkalemia** may occur especially if there is an element of renal failure.
- **Hyperchloremia** often develops following treatment, but is transient and does not require correction.
- **Hypoglycemia** should be prevented with frequent monitoring.
- **Recurrent or persistent DKA** can occur if insulin is stopped in the face of hypoglycemia but persistent acidosis. Insulin should be maintained and glucose given if hypoglycemia occurs but the acidosis has not yet resolved.
- **Cerebral edema** is rare but carries a high mortality.

11.8 Education

Clients need to be educated as to the etiology and precipitating factors of DKA to prevent recurrence. Trouble shooting and sick day guidelines should be reviewed. If the episode occurred as a result of insulin omission, appropriate psychosocial discussions and referrals should be made. Insulin omission and eating disorders need to be addressed.

References

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