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Acute Complications of Diabetes Mellitus-A Clinicians Experience

Dr Samir Sahu[1],Dr.Ruchi Bhuyan[2*], Dr.Dattatrey Kar[3]
[1]Professor & HOD DEPARTMENT OF GENERAL MEDICINE IMS,SUM HOSPITAL SIKSHA O ANUSANDHAN UNIVERSITY ,BHUBANESWAR
[2] Professor, Department of Oral pathology and Microbiology, IMS, SUM HOSPITAL SIKSHA O ANUSANDHAN UNIVERSITY BHUBANESWAR
[3] Research Associate, Medical Research Health Sciences, IMS, SUM HOSPITAL SIKSHA O ANUSANDHAN UNIVERSITY BHUBANESWAR

ABSTRACT

Acute complication of diabetes pose a serious threat to life in patients of Diabetes Mellitus. There are two types, Diabetic Ketoacidosis (DKA) which is more prevalent in Type 1 Diabetes Mellitus and Hyperglycemic Hyperosmolar State (HHS) which is more common in Type 2 Diabetes Mellitus. The usual Precipitants of these are non compliance, infection, stroke. Clinical features supported by laboratory investigations usually makes the diagnosis. The management is by fluids, Insulin and electrolyte correction.
There are two main types of acute complications of Diabetes Mellitus
1.Diabetic ketoacidosis (DKA)-Usually occurs in Type 1 DM but also occurs in Type 2.
2.Hyperglycemic hyperosmolar state (HHS)-primarily seen in individuals with type 2 DM.
Absolute or relative insulin deficiency, volume depletion and acid-base abnormalities are present in both.

Diabetic Ketoacidosis

Manifestations of Diabetic Ketoacidosis

Usually develop over 24 hours. And type 1DM may be an early introduction, but it's more common with type 1 diabetes.
The most common symptoms of DKA are polydipsia and insidious increase in polyuria (3)
Symptoms may include nausea / vomiting, thirst / polyuria, abdominal pain and shortness of breath.
Occurrences can include inadequate insulin administration, infection, infectious drug (cocaine) and pregnancy. Persistent subcutaneous insulin infusion (CSII) devices (2) malfunction.
Physical discoloration, tachycardia, dehydration / hypotension, tachypnea, shortness of breath, shortness of breath, tenderness in the abdomen, lethargy or bloating or brain swelling and possibly coma.

Pathophysiology

↓ insulin ,counterregulatory hormone (glucagon, catecholamines, cortisol, and growth hormone).
↓ ratio of insulin to glucagon promotes gluconeogenesis, glycogenolysis, and ketone body

Laboratory Values in DKA & HHS

	DKA	HHS
Glucose(mg/dL.)	250–600	600–1200

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Sodium, meq./L	125-135	135-145
Potassium	N to ↑	N
Osmolality (mOsm/mL)	300–320	330–380
Plasma ketones	++++	+/-
Serum bicarbonate, meq./L	<15 meq./L	Normal to slightly ↓
Arterial ph	6.8-7.3	>7.3
Anion gap[Na – (Cl + HCO_3)]	↑	N

Management of Diabetic Ketoacidosis

First we have to confirm the diagnosis and then the patient needs to be admitted. First check the serum electrolytes, acids, base status and kidney function.

Replacement of fluids: 2–3 L of 0.9% NS first 1–3 h → 0.45% saline→ 5% glucose + 0.45% saline when plasma glucose reaches 200 mg/dL

Regular insulin should be given by IV (0.1 units/kg) bolus followed by 0.1 units/kg/ hr by IV infusion

Replace Potassium by 10meq/h when K^+<5.2 meq/L and 40–80 meq/h when K^+< 3.5 meq/L

Measurement Glucose should be done every 1–2 hrly and Electrolytes (K^+, bicarbonate, phosphate) every 4 hrs in the first 24 hrs

The cause of the advance reaction should be found such as non-compliance, infection, trauma, affliction.

Hyperglycemic Hyperosmolar coma (HHS)

The Prototypical patient is an elderly, type 2 DM, presenting polyuria, decreased oral intake, mental confusion, or coma. It's associated with dehydration, hyperosmolality, hypotension.

Nausea, vomiting, abdominal pain &Kussmaul respirations are absent unlike in Diabetic Ketoacidosis

It's precipitated by Acute Myocardial Infarction, stroke, Sepsis and pneumonia

Pathophysiology of HHS

It is mainly caused by insufficient fluid osmotic diuresis .

Striking feature is absence of ketosis because of Insulin deficiency is only relative, Lower levels of counter regulatory hormones & FFA, Liver is less capable of ketone body synthesis and insulin/glucagon ratio does not favour ketogenesis.

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Treatment ofHyperglycemic Hyperosmolar coma HHS
Fluid losses and dehydration in HHS is more than in DKA.
Higher mortality rate than DKA (up to15%).

Initial treatment should be with fluid replacement with 1–3 L Normal Saline in 2–3 hrs. 0.45% saline if serum Na >150mmol/L, water deficit (which averages 9–10 L) displayed over the next 1–2 days. Potassium deficit & Hypophosphatemia should be corrected
Intravenous insulin should be administered more or less in the line of Diabetic Ketoacidosis.

Conclusion
Prompt diagnosis and quick institution of therapy saves life in these conditions. Fluid deficit is more in HHS than in DKA . Fluid and intravenous insulin are the first line of treatment. Potassium correction should be done before administration of insulin if the potassium level is less than 3.2mEq/L. Effective treatment of DKA results in less than 1% mortality, but it is somewhat higher in HHS.

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