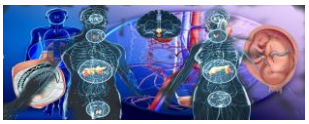
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Short Review **International Journal of Endocrinology and Disorders** **Open Access**

A Retrospective Study of Diabetic Ketoacidosis and Initial Management

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Abstract

Diabetic ketoacidosis (DKA) is characterized by uncontrolled hyperglycemia, metabolic acidosis, and increased body ketone concentration. It is a life-threatening complication of diabetes and is usually seen in patients with type-1 diabetes mellitus. Rarely it may also occur in patients with type-2 diabetes mellitus. DKA is a state of a relative or absolute insulin deficiency that is worsened by hyperglycemia, dehydration, and acidosis. In most cases, the trigger is an infection, new-onset diabetes, or lack of compliance with treatment.

Keywords: Abnormal laboratory parameters, type-1 diabetes mellitus, serum glycemic levels

Introduction

Diabetic ketoacidosis (DKA) is not a rare presentation to hospital, despite being an entirely preventable condition. DKA is caused by a decrease in effective circulating insulin associated with elevations in counter-regulatory hormones. This potentially life-threatening complication of type 1 diabetes mellitus (T1DM) is frequently mismanaged, leading to morbidity and increased length of stay. Mortality rates have fallen significantly in the past 20 years to <1%. Advances in near-patient testing technology have improved patient care, by facilitating rapid diagnosis and closer monitoring of treatment response.

Causes of DKA

- New diagnosis of T1DM.
- Poor concordance with insulin treatment.
- Inadequate insulin therapy in hospital.

One of the major causes of recurrent DKA in the inner-city population in the United States is non-compliance with insulin. Socioeconomic and educational factors play a significant role in poor adhesion to medications, including insulin. A recent report suggests that cocaine abuse is an independent risk factor associated with DKA recurrence.

Epidemiology

Diabetic ketoacidosis incidence ranges from 0 to 56 per 1000 person-years, shown in different studies from different geographic areas. DKA has a higher prevalence rate among women and non-Whites. Incidence is higher among patients using injectable insulin compared to the subcutaneous insulin infusion pumps.

The geriatric population is at particular risk for developing hyperglycemic crises with the development of diabetes. Some of the causes are increased insulin resistance and a decrease in the thirst mechanism. The elderly are particularly vulnerable to hyperglycemia and dehydration, the critical components of hyperglycemic emergencies. With increased diabetes surveillance and aggressive early treatment of hyperglycemia and its complications, morbidity, and mortality from acute diabetic crises in the geriatric population can be significantly reduced.

Pathophysiology: Diabetes mellitus is characterized by insulin deficiency and increased plasma glucagon levels, which can be normalized by insulin replacement. Normally, once serum glucose concentration increases, it enters pancreatic beta cells and leads to insulin production. Insulin decreases hepatic glucose production by inhibiting glycogenolysis and gluconeogenesis. Glucose uptake by skeletal muscle and adipose tissue is increased by insulin. Both of these mechanisms result in the reduction of blood sugar. In diabetic ketoacidosis, insulin deficiency and increased counter-regulatory hormones can lead to increased gluconeogenesis, accelerated glycogenolysis, and impaired glucose utilization.

New data suggests that hyperglycemia leads to a severe inflammatory state and an increase in proinflammatory cytokines (tumor necrosis factor-alpha and interleukin-beta, -6, and -8), C-reactive protein, lipid peroxidation, and reactive oxygen species, as well as cardiovascular risk factors, plasminogen activator inhibitor-1 and free fatty acids in the absence of apparent infection or cardiovascular pathology.

DKA Management

The most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by insulin administration. The main aims for **fluid replacement** are:

- restoration of circulatory volume
- clearance of ketones
- correction of electrolyte imbalance.

Insulin should be administered intravenously and given at a fixed rate using a weight-based formula: 0.1 units per kilogram body weight per hour. It may be necessary to estimate the patient's weight; treatment should not be delayed waiting for an accurate weight.

Metabolic treatment targets

- Reduction of the blood ketone concentration by 0.5 mmol/L/hour.
- Increase the venous bicarbonate by 3.0 mmol/L/hour.
- Reduce capillary blood glucose by 3.0 mmol/L/hour.

f the patient was on twice daily fixed-mix insulin (eg NovoMix 30), restart

their usual SC insulin either before breakfast or before the evening meal. Continue IV insulin infusion until 30–60 minutes after the SC insulin was given. Decisions regarding SC insulin treatment (what regimen/which insulin/what doses) to use in the newly diagnosed patient should be on the advice of the diabetes specialist team.

DKA in association with sodium-glucose cotransporter-2 inhibitors

Sodium-glucose cotransporter-2 inhibitors (SGLT2is) such as empagliflozin are the newest class of oral therapies used for the treatment of T2DM, licensed in the UK since 2013. SGLT2is reversibly inhibit sodium-glucose cotransporter 2 in the renal proximal convoluted tubule, which reduces glucose reabsorption and thus increases urinary glucose excretion.

Conclusion

DKA is the most common hyperglycemic emergency and causes the greatest risk for mortality in patients with diabetes mellitus. Epidemiological studies showed more prevalence of DKA among patients with type-1 diabetes, yet almost a third of the cases occur among those with type 2 diabetes. DKA is characterized by hyperglycemia, metabolic acidosis and ketosis. Patients with diabetes in general, and those with type-1 diabetes would benefit from being educated to recognize and reduce early signs of DKA. Standard management of DKA requires hospitalization and involves aggressive intravenous fluids, insulin therapy, electrolyte replacement.

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