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Review Article

Management of diabetic ketoacidosis in internal medicine: insulin protocols, electrolyte balance, and clinical outcomes

Samar Abed Alharbi[1]*, Amjad Mohammed Meshari[2], Zainab Abdulaziz Henawi[3], Reem Mohammed Alsaadi[4], Shaher Saad Alqarni[5], Ali Fuad Al Hani[6], Ola Hussain Alhamrani[7], Laila Mohammed Saptan[8], Zainab Abduljabbar AL Hamoud[9], Nawaf Khalid Alshanbri[10], Zainab Abdulmonem Jafar[11], Abdelrahman Farag[12]

[1]Department of Internal Medicine, Al Thager Hospital, Jeddah, Saudi Arabia
[2]College of Medicine, Jazan University, Jazan, Saudi Arabia
[3]Al Naeem Primary Healthcare Center, King Fahad General Hospital, Jeddah, Saudi Arabia
[4]Primary Healthcare, King Fahad General Hospital, Jeddah, Saudi Arabia
[5]Nursing Department, General Thriban Hospital, Thriban, Saudi Arabia
[6]College of Medicine, Medical University of Lodz, Lodz, Poland
[7]Al-Faisaliah Health Center, Ministry of Health, Hofuf, Saudi Arabia
[8]College of Medicine, King Khalid University, Abha, Saudi Arabia
[9]Department of Intensive Care Unit, Qatif Health Network, Saihat, Saudi Arabia
[10]College of Medicine, King Abdulaziz Primary University, Jeddah, Saudi Arabia
[11]Department of Nephrology, Al Noor Specialist Hospital, Mecca, Saudi Arabia
[12]Mahsa University, Petaling Jaya, Malaysia

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***Correspondence:**
Dr. Samar Abed Alharbi,
E-mail: Dr_sam1990@hotmail.com

ABSTRACT

Diabetic ketoacidosis (DKA) is a critical complication of diabetes mellitus (DM), characterized by hyper-glycemia, acidosis, and ketosis. It poses a substantial risk of morbidity and mortality, especially in type 1 DM patients. DKA can be triggered by various factors, including insulin deficiency, infections, alcohol abuse, and other medical conditions. Hospital admissions for DKA are increasing, with mortality rates of up to 5-9%, often linked to severe underlying illnesses and complications such as myocardial infarction and stroke. Effective DKA management involves rehydration, correction of electrolyte imbalances, insulin administration, and addressing precipitating factors. Fluid resuscitation with isotonic saline is vital to restore hydration, and continuous intravenous insulin infusion is the preferred method to control blood glucose and suppress ketone production. Electrolyte imbalances, particularly potassium, sodium, phosphate, and magnesium, require careful monitoring and correction. Clinical outcomes in DKA management include resolving acidosis, normalizing blood glucose, and restoring electrolyte balance, all while achieving and maintaining clinical stability. Complications like cerebral edema and acute respiratory distress syndrome can significantly impact the prognosis. Long-term considerations encompass diabetes management, patient education, and follow-up care.

Keywords: DM, DKA, Insulin therapy, Fluid resuscitation, Electrolyte imbalances

INTRODUCTION

Diabetic ketoacidosis (DKA) is a serious complication of DM, commonly encountered in clinical practice which poses a substantial risk of morbidity and mortality.[1-3] According to Al-Bunyan et al it is distinguished by the triadic symptoms of hyperglycemia, acidosis, and ketosis.[4] Plasma glucose concentrations >250 mg/dl (>13.8 mmol/l), venous pH<7, bicarbonate (HCO3) <18 mEq/l, and ketonemia and/or ketonuria are definitive laboratory diagnosis of DKA.[3] DKA results from a complex interplay of factors, including insulin deficiency, increased counterregulatory hormones, and elevated blood glucose levels, leading to metabolic derangements and the production of ketones. Compared to type 2 DM, type 1 DM patients are more commonly affected by DKA. The most frequent cause of DKA development is missing insulin dosages. Infections, cerebrovascular accidents, alcohol abuse, pancreatitis, myocardial infarction, trauma, and medications like corticosteroids, thiazides, and sympathomimetic agents are additional factors that can cause DKA. In addition, DKA can develop in patients with type 1 DM who have recently developed the disease, have discontinued insulin, or are not using it sufficiently. Twenty percent of recurrent ketoacidosis cases in young patients with type 1 DM may be related to psychological issues with eating disorders.[5] Hospital admissions for DKA and hyperosmolar hyperglycemia state (HHS) are on the rise, despite changing practices.[3,6] In patients with severe comorbidities and the elderly, DKA is linked to mortality rates as high as 5-9%.[3,7] While severe underlying illnesses and comorbidities are frequently the cause of death from DKA, DKA itself induces a hypercoagulable state that can lead to possibly lethal outcomes such as myocardial infarction, stroke, and disseminated intravascular coagulation.[3,8,9] Rehydrating, correcting electrolyte imbalances-especially hypokalaemia-administering insulin, correcting metabolic acidosis, and treating precipitants like sepsis, pancreatitis, trauma, and myocardial infarction are all part of the management process.[10-12] Severe electrolyte derangement and pulmonary venous congestion are challenges associated with the management of DKA. A significant potential complicating factor is cerebral oedema, though most cases of this have been seen in pediatric patients.

This paper provides a review of the management strategies for DKA, encompassing fluid resuscitation, insulin therapy, and electrolyte replacement. It also discusses clinical outcomes and potential complications and emphasizes the significance of regular monitoring and follow-up care in achieving favorable outcomes.

METHODS

This study is based on a comprehensive literature search conducted on November 7, 2023, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the management of DKA in internal medicine, with a focus on insulin protocols, electrolyte balance, and clinical outcomes. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

The pathophysiological basis of DKA primarily involves insulin deficiency, which leads to increased lipolysis and subsequent ketone production. Additionally, hyperglycemia and dehydration exacerbate the acidosis and electrolyte imbalances observed in DKA.[3] DKA typically presents with symptoms such as polyuria, polydipsia, nausea, vomiting, and altered mental status. Diagnostic criteria include hyperglycemia (blood glucose >250 mg/dL), arterial pH <7.30, bicarbonate < 18 mEq/L, and the presence of ketonemia or ketonuria.[13]

Fluid resuscitation in DKA management

Fluid resuscitation is a pivotal component in the management of DKA. DKA often leads to significant dehydration due to the excessive urination driven by hyperglycemia. This dehydration can result in symptoms such as intense thirst, dry mucous membranes, tachycardia, and diminished tissue perfusion, potentially progressing to hypotension and shock if left unaddressed.[14] The primary objective of fluid resuscitation in DKA is to restore intravascular volume and correct dehydration. Isotonic saline, typically 0.9% sodium chloride, is the preferred fluid choice for initial resuscitation, as it helps replenish intravascular volume and provides necessary sodium.[15] An initial bolus of isotonic saline is often administered at a rate of 1 to 2 litters over the first hour, with subsequent maintenance fluids used to address ongoing fluid losses while preventing rapid drops in blood glucose levels.[16] Close monitoring, including clinical assessment, fluid balance tracking, and blood glucose monitoring, guides the adjustments in fluid administration rates and volumes, ensuring the patient's hydration is corrected appropriately. Pediatric patients and those with severe hypotension may require special considerations.[17] Overall, fluid resuscitation serves as the foundation for subsequent insulin administration and the correction of metabolic imbalances in DKA management.

Insulin protocol in DKA management

DKA is primarily characterized by insulin deficiency and the presence of excess counterregulatory hormones. Therefore, the central goal of DKA treatment is to reverse these abnormalities. Intravenous insulin infusion is the cornerstone of DKA management.[18] It helps suppress ketone production, normalize blood glucose levels, and

correct acidosis.[19] Two common insulin protocols for DKA management are continuous intravenous insulin infusions and subcutaneous insulin regimens.

Continuous Intravenous Insulin Infusion

Continuous intravenous insulin infusion is the preferred and most effective method for treating DKA due to its precise control over insulin delivery.[20] It enables rapid reductions in blood glucose levels and suppression of ketone production. The use of intravenous insulin allows for frequent adjustments according to blood glucose levels and prevents the risk of hypoglycemia associated with intermittent dosing.[21] Typically, an initial bolus of regular insulin is administered, followed by a continuous infusion at an established rate (e.g., 0.1 units/kg/hour). The insulin infusion rate can be titrated based on frequent blood glucose measurements (e.g., every hour) to achieve a gradual reduction in blood glucose levels. Continuous monitoring of blood glucose and ketone levels is essential during intravenous insulin therapy. The aim is to maintain blood glucose within the target range (e.g., 150-200 mg/dL) while correcting acidosis.[22] The frequent monitoring allows for prompt adjustments to the insulin infusion rate.

Subcutaneous insulin regimens

Subcutaneous insulin regimens, such as the use of basal and rapid-acting insulins, may be considered in select stable patients with mild to moderate DKA who can tolerate oral intake and have good compliance.[20] However, this approach is not suitable for patients with severe acidosis, hemodynamic instability, or those requiring intensive care. Patients receiving subcutaneous insulin regimens often require multiple daily injections of basal and rapid-acting insulin analogs. The total daily insulin dose is divided into basal and bolus components to maintain glycemic control. Subcutaneous insulin regimens require frequent monitoring of blood glucose and ketone levels is essential, based on which insulin doses are adjusted. Patients must be able to consume and tolerate oral fluids and nutrition. This approach may not achieve rapid correction of DKA seen with intravenous insulin infusion. Choice of insulin protocol in DKA management depends on the severity of condition, patient stability, and healthcare setting. Continuous intravenous insulin infusion offers advantage of precise control and rapid correction of hyperglycemia and ketosis, making it preferred choice for severe cases.[23] Subcutaneous insulin regimens may be considered for less severe cases with good patient compliance and stability.

Electrolyte balance in DKA management

Electrolyte balance is a critical consideration when managing DKA because the condition is associated with profound disturbances in electrolyte levels, particularly potassium, sodium, phosphate, and magnesium.[24] Proper assessment, monitoring, and correction of these electrolyte imbalances are essential to prevent complications and ensure the patient's safety and recovery.

Potassium (K+)

DKA is often characterized by total body potassium deficits, even when serum potassium levels appear normal or elevated.[25] This paradoxical situation arises due to intracellular potassium shifts driven by insulin deficiency, acidosis, and osmotic diuresis. Serum potassium levels should be closely monitored, preferably hourly during the initial phase of treatment, and then at regular intervals. Continuous electrocardiographic monitoring is essential to detect any changes in cardiac rhythm.[26] Potassium replacement should be initiated early, especially if the initial serum potassium level is <5.3 mEq/l.[25] Replacement can be done orally or intravenously, with careful consideration of the patient's renal function and urine output. Potassium replacement should be started only after ensuring adequate urine output to minimize the risk of hyperkalemia. As insulin therapy corrects acidosis and drives potassium back into cells, serum potassium levels may decrease rapidly, requiring continued monitoring and adjustment of replacement rates.[18]

Sodium (Na+)

Hyponatremia may occur in DKA due to hyperglycemia-related osmotic diuresis.[24] As glucose is excreted in urine, it draws water along with it, potentially diluting serum sodium concentrations. Serum sodium levels should be monitored regularly. Correcting hyper-glycemia and acidosis should gradually correct hyponatremia, but it is important not to overcorrect sodium levels, which can lead to central pontine myelinolysis.[27] Primary focus in treating hyponatremia in DKA is to address underlying cause (hyperglycemia and acidosis). In most cases, direct sodium replacement is not required.

Phosphate (PO4-)

DKA often leads to hypophosphatemia (low serum phosphate levels) due to urinary losses and intracellular shifts of phosphate during acidosis correction.[28] Serum phosphate levels should be assessed regularly, especially in severe DKA cases. Hypophosphatemia can lead to muscle weakness, respiratory failure, and cardiac dysfunction.[29] Phosphate replacement is typically reserved for patients with severe hypophosphatemia (<1.0 mg/dL)/those exhibiting clinical symptoms.[30] Replacement can be done orally or intravenously, and rate of replacement should be adjusted based on serum phosphate levels.

Magnesium (Mg2+)

Hypomagnesemia is common in DKA, resulting from urinary losses and intracellular shifts.[31] Low magnesium

levels can contribute to cardiac arrhythmias and neuromuscular complications.[32] Serum magnesium levels should be monitored, especially in patients with severe DKA or cardiac symptoms. Magnesium replacement should be considered in patients with symptomatic hypomagnesemia.[31] Replacement can be administered intravenously, and the rate should be adjusted based on serum magnesium levels.

Proper electrolyte management is crucial in DKA to prevent life-threatening complications such as arrhythmias, seizures, and cardiac arrest.[24] The balance between correcting electrolyte imbalances and avoiding rapid shifts is delicate, necessitating close monitoring and individualized treatment plans.

Clinical outcomes

Clinical outcomes in the management of DKA encompass several essential aspects of patient care. Firstly, the resolution of acidosis, as indicated by normalization of arterial pH and serum bicarbonate levels, signifies the successful correction of metabolic abnormalities, leading to symptom relief.[33] Secondly, the normalization of blood glucose levels, often targeted within the range of 150-200 mg/dL, reduces the risk of hyperglycemia-related complications and alleviates symptoms like polyuria and polydipsia.[34] Concurrently, the clearance of ketones from the bloodstream is critical, as persistent ketosis can perpetuate acidosis and associated symptoms.[35] Additionally, maintaining or restoring electrolyte balance, particularly potassium, sodium, phosphate, and magnesium, is essential for preventing complications such as cardiac arrhythmias and muscle weakness.[24]Achieving and maintaining clinical stability, characterized by normalized vital signs, improved mentation, and resolution of dehydration, further ensures patient well-being. Effective DKA management includes the prevention and early management of complications like cerebral edema and acute respiratory distress syndrome.[36,37] Long-term follow-up and diabetes management are crucial for preventing recurrent DKA episodes, while patient education empowers individuals with the knowledge and skills needed for self-management and improved long-term outcomes in diabetes care.

Complications and prognosis

Complications associated with DKA can have serious implications for patient outcomes. Cerebral edema, albeit rare, poses a life-threatening risk, especially among children and adolescents, with symptoms ranging from altered mental status to seizures or coma.[38] Acute respiratory distress syndrome (ARDS) can develop in severe DKA cases, requiring mechanical ventilation and intensive care.[37] As noted earlier, electrolyte derangements may result in cardiac arrhythmias, muscle weakness, and neuromuscular symptoms.[25] Additionally, DKA heightens the risk of thromboembolic events, such as deep vein thrombosis (DVT) or pulmonary embolism (PE).[39,40] The prognosis in DKA management largely depends on timely and appropriate treatment, with most patients achieving full recovery. Favorable outcomes entail the resolution of acidosis, normalization of blood glucose, ketone levels, and electrolyte balance restoration.[41] Clinical stability, characterized by improved vital signs and mental status, is also indicative of a positive prognosis. However, the development of complications, particularly cerebral edema and ARDS, can significantly impact the prognosis, with long-term considerations involving diabetes management, patient education, and follow-up care being critical factors in achieving optimal outcomes.[42] The variability in prognosis is influenced by the severity of the episode, the presence of complications, the patient's overall health, and their response to treatment.

CONCLUSION

In conclusion, as the pathophysiological basis of DKA revolves around insulin deficiency, leading to increased lipolysis and ketone production, exacerbated by hyperglycemia and dehydration, managing DKA involves key components targeting the same. These include fluid resuscitation, insulin therapy, and electrolyte balance correction. Optimal clinical outcomes in DKA management entail the successful correction of metabolic abnormalities, reflected in normalized arterial pH, serum bicarbonate levels, and blood glucose, clearance of ketones, restoration of electrolyte balance, and the attainment of clinical stability, ultimately ensuring patient safety and well-being. Success in DKA care not only relies on the precision of medical interventions but also on the collaboration between healthcare providers and patients, highlighting the pivotal role of education, follow-up care, and ongoing diabetes management in ensuring the best possible outcomes for individuals grappling with this complex metabolic condition.

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