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Diabetic Ketoacidosis: Management and Complications - A Brief Review

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ABSTRACT

Diabetic Ketoacidosis (DKA) is a serious acute complication of diabetes mellitus characterized by hyperglycemia, metabolic acidosis and ketonemia. Although more frequent in patients with type 1 diabetes, it can also occur in individuals with type 2 diabetes under metabolic stress conditions. Main triggers include inadequate insulin use, infections and events that increase insulin demand or counterregulatory hormone production. Management requires a multidisciplinary approach, encompassing fluid replacement, electrolyte correction, insulin administration and treatment of the precipitating factor. Restoration of intravascular volume, inhibition of lipolysis and reversal of ketogenesis are fundamental for clinical stabilization. In this context, continuous monitoring of blood glucose and electrolytes particularly potassium is essential to avoid serious complications such as fatal hypokalemia. The most common iatrogenic complications are hypoglycemia and electrolyte disturbances (hypokalemia and hypophosphatemia), as well as cerebral edema, more prevalent in pediatric patients. Early identification and control of severity signs, together with effective management of the precipitating factor, are crucial to reduce DKA associated morbidity and mortality.

Keywords:

Diabetic ketoacidosis, Hyperglycemia, Insulin, Complications, Clinical management.

Introduction

Diabetic Ketoacidosis (DKA) is a metabolic emergency that occurs primarily in people with type 1 diabetes but may also present in other forms of diabetes under conditions of metabolic stress. Before the advent of exogenous insulin, DKA was almost invariably fatal in type 1 diabetes; despite therapeutic advances, it remains a significant cause of morbidity and mortality especially in settings with limited healthcare access or poor treatment adherence. The pathophysiology of DKA stems from absolute or relative insulin deficiency coupled with elevated counterregulatory hormones (glucagon, cortisol, catecholamines, growth hormone). Insulin deficiency promotes lipolysis, proteolysis and glycogenolysis. Impaired insulin-mediated glucose uptake leads to hyperglycemia, while intense lipolysis and ketone body production (acetoacetate and β hydroxybutyrate) result in metabolic acidosis. Consequently, hyperglycemia in DKA is driven by reduced peripheral glucose utilization, increased hepatic glucose production and impaired renal glucose excretion. Osmotic diuresis causes cellular dehydration and glucosuria, further exacerbating fluid loss. DKA is defined by serum glucose >200 mg/dL, ketosis and metabolic acidosis (serum bicarbonate <15 mmol/L with elevated anion gap). Various precipitating factors include infections, insulin omission, myocardial infarction, stroke, pancreatitis and medications that elevate blood glucose (corticosteroids, diuretics, beta blockers, etc.). Clinically, patients present with polydipsia, polyuria, fatigue, nausea, vomiting, Kussmaul respiration and, in severe cases, altered consciousness. Prompt initiation of therapy is essential once DKA is diagnosed.

Objectives

To review the pathophysiological, diagnostic and therapeutic aspects of Diabetic Ketoacidosis (DKA), and to discuss its main associated complications.

Discussion

DKA remains relevant in both developing and developed countries, reflecting challenges in glycemic control and treatment adherence. Vulnerable populations face issues such as lack of supplies, inadequate glucose monitoring and delays in addressing precipitating factors [1]. The main triggers are infections (especially respiratory and urinary), poor adherence to insulin therapy, cardiovascular events (myocardial infarction, stroke), pancreatitis and certain medications. Correct identification of the precipitant is crucial to prevent recurrence [2].

Management is based on correcting metabolic disturbances, reversing clinical signs and treating the underlying cause [3]. Initial volume resuscitation uses isotonic saline (15-20 mL/kg), followed by continuous intravenous infusion of regular insulin at

0.1 U/kg/h provided serum potassium is ≥ 3.3 mEq/L. This rate typically lowers blood glucose by 50-70 mg/dL per hour. When glucose reaches 200-250 mg/dL, 5% dextrose is added to avoid hypoglycemia, adjusting infusion rates as needed. Electrolyte repletion particularly potassium, phosphate and magnesium is essential to prevent life threatening arrhythmias or muscle weakness. Treatment of the precipitating factor (e.g., infections, hemodynamic support in myocardial infarction or stroke, adjustment of hyperglycemic drugs) is equally important. Sodium bicarbonate is reserved for severe acidemia (pH <6.9) and used cautiously due to risks of hypokalemia and rebound alkalosis [4]. Iatrogenic complications include hypoglycemia from overly rapid glucose correction or insufficient dextrose infusion; hypokalemia and hypophosphatemia with risk of arrhythmias and muscle weakness; and cerebral edema particularly in childrenlinked to rapid glucose shifts and inflammatory mechanisms. Rigorous monitoring throughout therapy is therefore essential [5].

Conclusion

Diabetic ketoacidosis remains a major cause of morbidity and mortality in people with diabetes, reflecting both resource limitations and treatment non adherence. The cornerstone of therapy is careful correction of hyperglycemia, dehydration and electrolyte imbalances, with continuous monitoring and identification of precipitants to prevent recurrence. Therapeutic success also depends on a multidisciplinary approach including nutritional education, psychological support and primary care follow up. Clear discharge planning and outpatient follow up strategies are vital to reducing recurrent DKA. Transition from hospital to home requires efficient coordination across healthcare levels, ensuring ongoing therapeutic adjustments and education.

Globally, persistent DKA rates signal the need for healthcare system improvements both in provider training and public policies that broaden supply and monitoring technology access. Strengthened health education interventions must address not only insulin and monitoring techniques but also socioeconomic and psychological support, bridging the gap between theoretical knowledge and daily self care. Adoption of innovative tools such as telemedicine and artificial intelligence is increasingly essential for early detection of glycemic imbalances and individualized treatment adaptation. Thus, DKA should be viewed not merely as an acute emergency but as an indicator of the necessity for continuous, personalized, comprehensive diabetic care. Integration of early diagnosis, effective management and recurrence prevention constitutes the most promising strategy for reducing mortality and improving quality of life in people with diabetes.

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