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Multidisciplinary Management of Diabetic Ketoacidosis as an Emergent Complication of Diabetes: Integrating Epidemiology, Primary Care, Radiology, Pharmacy, Nursing, Dental Hygiene, and Family Medicine in a Community Health Framework

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Abstract

Background: Diabetic ketoacidosis (DKA) is a life-threatening metabolic emergency characterized by hyperglycemia, ketonemia, and metabolic acidosis. It affects individuals with both type 1 and type 2 diabetes and is precipitated by infection, nonadherence to therapy, acute illness, and medication-related metabolic disturbances. Emerging triggers include SGLT2 inhibitors, GLP-1 receptor agonists, immune checkpoint inhibitors, and substance use.
Aim: To provide an integrated multidisciplinary overview of DKA, encompassing epidemiology, clinical presentation, evaluation, management, and healthcare team collaboration in improving outcomes.
Methods: This article synthesizes evidence from epidemiologic studies, clinical guidelines, and multidisciplinary practice recommendations. It reviews DKA etiology, population-level trends, diagnostic standards, biochemical evaluation, and current therapeutic strategies, including fluid resuscitation, insulin protocols, and electrolyte correction. It further analyzes the roles of nursing, pharmacy, radiology, dentistry, epidemiology, and family medicine in optimizing care delivery.
Results: DKA incidence varies significantly across populations, with higher rates in women, minority groups, the elderly, and patients with insulin-delivery challenges. Early recognition supported by laboratory criteria—elevated glucose, low pH, low bicarbonate, and high anion gap—remains essential for timely intervention. Standardized treatment involving fluid resuscitation, continuous insulin therapy, and careful electrolyte replacement has significantly reduced mortality. Multidisciplinary collaboration enhances diagnostic accuracy, reduces complications, prevents recurrence, and supports long-term glycemic control.
Conclusion: Given its multifactorial triggers and potentially fatal complications, DKA requires coordinated, evidence-based, multidisciplinary management. Comprehensive evaluation, guideline-driven treatment, and interprofessional collaboration are critical to reducing morbidity, preventing recurrence, and improving patient outcomes.
Keywords: Diabetic ketoacidosis, hyperglycemia, metabolic acidosis, multidisciplinary care, insulin therapy, electrolyte replacement, epidemiology, SGLT2 inhibitors.

Introduction

Diabetic ketoacidosis (DKA) constitutes a life-threatening metabolic derangement characterized by profound hyperglycemia, metabolic acidosis, and elevated circulating ketone bodies. While predominantly observed in individuals with type 1 diabetes mellitus, DKA may also manifest in patients with type 2 diabetes under specific precipitating conditions. The clinical urgency of DKA arises from its rapid progression and the substantial risk of morbidity and mortality when recognition or intervention is delayed [1]. The pathophysiology of DKA is typically triggered by either the initial presentation of diabetes or by factors that disrupt metabolic homeostasis in established diabetic patients. Infections represent a common precipitant, exerting systemic stress that exacerbates insulin deficiency and promotes ketogenesis. Nonadherence to prescribed therapeutic regimens, whether due to patient-related or systemic factors, similarly increases the likelihood of DKA onset. Furthermore, additional stressors such as acute medical illnesses, physical trauma, or pharmacologic agents can act synergistically to precipitate this metabolic crisis. The multifactorial

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etiology underscores the need for comprehensive patient assessment to identify potential risk factors and early warning signs [1]. Prompt recognition and rapid initiation of appropriate therapeutic measures are fundamental to mitigating the adverse outcomes associated with DKA. Effective management strategies, including fluid resuscitation, insulin administration, and correction of electrolyte imbalances, significantly improve survival rates and reduce complication risk. The time-sensitive nature of intervention highlights the necessity for heightened clinical vigilance and structured protocols in both inpatient and outpatient settings to ensure optimal patient outcomes [1].

Etiology

Diabetic ketoacidosis (DKA) primarily affects patients with type 1 diabetes mellitus, although it is increasingly recognized in individuals with type 2 diabetes under certain stress conditions. The pathogenesis of DKA involves a relative or absolute insulin deficiency coupled with increased counter-regulatory hormones such as glucagon, cortisol, catecholamines, and growth hormone. These hormonal changes promote hyperglycemia, ketogenesis, and metabolic acidosis, establishing the clinical triad that defines the syndrome [1]. Common triggers for DKA include newly diagnosed diabetes, inadequate insulin therapy, or poor adherence to prescribed treatment regimens. Acute medical conditions, infections, and physical stressors frequently precipitate metabolic decompensation. Urinary tract infections, pneumonia, sepsis, trauma, myocardial infarction, and pulmonary embolism are among the most frequently documented precipitating events, each contributing to catabolic stress and an increased risk of DKA [1]. Pharmacological agents that alter carbohydrate metabolism, including corticosteroids, thiazide diuretics, sympathomimetics, and pentamidine, can also precipitate DKA episodes. Notably, both conventional and atypical antipsychotics have been associated with hyperglycemia and, in rare cases, can trigger full-blown ketoacidosis [2]. These precipitating factors underscore the multifactorial nature of DKA and the necessity for clinicians to assess underlying metabolic, pharmacologic, and systemic contributors in each case. Although hyperglycemia is a hallmark feature of DKA, a subset of patients develops euglycemic DKA, wherein glucose levels remain below the conventional threshold of 250 mg/dL, yet ketonemia and high anion gap metabolic acidosis persist. This phenomenon often occurs in patients receiving insulin therapy who are underdosed or experiencing acute metabolic decompensation [3]. Euglycemic DKA has become increasingly relevant in the context of novel antidiabetic therapies, particularly sodium-glucose cotransporter 2 (SGLT2) inhibitors and glucagon-like peptide 1 (GLP-1) receptor agonists. SGLT2 inhibitors reduce renal glucose reabsorption, thereby lowering circulating glucose and reducing insulin requirements. However, these agents also enhance lipolysis, ketogenesis, and glucagon secretion while decreasing renal ketone clearance, cumulatively raising plasma ketone concentrations and predisposing patients to DKA [4][5]. GLP-1 receptor agonists exert multiple effects that may precipitate ketosis, including delayed gastric emptying, suppression of glucagon, and augmentation of insulin secretion. In certain clinical scenarios, these pharmacologic actions can induce hypoglycemia or a relative starvation state, thereby accelerating lipolysis and ketone production. Adverse effects such as nausea and vomiting may exacerbate dehydration and ketone accumulation, further contributing to the risk of DKA [6][7]. Euglycemic DKA may also arise in pregnancy, a state characterized by altered insulin sensitivity, increased lipolysis, and enhanced ketogenesis, highlighting the importance of heightened clinical vigilance in this population [8].

The expanding use of immune checkpoint inhibitors (ICIs) in oncology has introduced a novel etiology of DKA. These agents, which enhance antitumor immune responses, are associated with immune-related endocrinopathies, including immune checkpoint inhibitor-induced diabetes mellitus (ICI-DM). Patients with ICI-DM frequently lack a prior history of diabetes and often present acutely with DKA. The pathophysiology involves autoimmune destruction of pancreatic β-cells triggered by the immune-modulating effects of ICIs, resulting in absolute insulin deficiency and metabolic decompensation. This subset of patients underscores the importance of recognizing drug-induced and immune-mediated mechanisms as emerging contributors to DKA in modern clinical practice. Substance use represents another important and underrecognized precipitant of DKA. Chronic cocaine use has been identified as an independent risk factor for recurrent DKA episodes. The mechanisms likely involve catecholamine-mediated insulin resistance, increased gluconeogenesis, and behavioral factors affecting adherence to therapy [9]. Cannabis use also increases the risk of DKA, particularly among chronic users who develop cannabinoid hyperemesis syndrome. Persistent vomiting and dehydration in this context can precipitate recurrent metabolic crises in individuals with diabetes, highlighting the interaction between behavioral factors, substance exposure, and metabolic vulnerability [10]. These findings indicate that comprehensive assessment of DKA etiology must extend beyond traditional medical triggers to include lifestyle, behavioral, and pharmacologic influences. Collectively, the etiologies of DKA reflect a complex interplay of metabolic, infectious, pharmacologic, immunologic, and behavioral factors. Type 1 diabetes remains the predominant underlying condition, but type 2 diabetes patients are increasingly susceptible under stress or medication-induced metabolic perturbations. Classic triggers such as infections, trauma, and medication nonadherence remain relevant, while newer etiologies, including SGLT2 inhibitor therapy, GLP-1 receptor agonist use, immune checkpoint inhibitor-related diabetes, and substance abuse, have expanded the clinical spectrum. Recognition of these diverse etiologies is essential for accurate diagnosis, prompt intervention, and tailored preventive strategies. A nuanced understanding of the underlying mechanisms, patient-specific risk factors, and emerging triggers allows clinicians to anticipate DKA, implement early interventions, and optimize outcomes across a broad spectrum of patient populations [1][2][3][4][5][6][7][8][9][10].

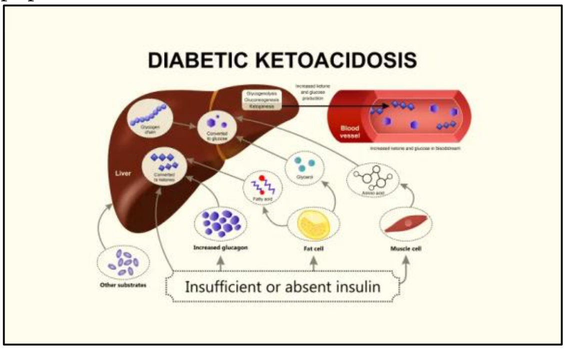

Fig. 1: Diabetic ketoacidosis.

Epidemiology

The epidemiology of diabetic ketoacidosis (DKA) demonstrates considerable variability across populations, age groups, and geographic regions. Reported incidence rates range broadly from 0 to 56 cases per 1000 person-years, reflecting differences in study design, population characteristics, and healthcare infrastructure [11]. Epidemiologic evidence indicates a higher prevalence of DKA among women and individuals from non-White ethnic backgrounds, suggesting the influence of genetic, socioeconomic, and healthcare access factors on disease risk. Furthermore, the method of insulin delivery appears to affect DKA susceptibility; patients using injectable insulin are more frequently affected than those utilizing continuous subcutaneous insulin infusion pumps, highlighting the role of treatment modality in disease prevention and management [11]. In pediatric populations, the incidence of DKA exhibits marked international variation. In Nigeria, the reported incidence is among the lowest globally at 2.9 cases per 100,000 children, whereas Northern European countries such as Sweden and Finland report significantly higher rates, at 41.0 and 37.4 per 100,000, respectively [12]. These disparities likely reflect differences in early diabetes detection, healthcare system capacity, cultural attitudes toward disease management, and public health infrastructure. In the United States, certain vulnerable subgroups, such as nursing home residents, are particularly affected. A study demonstrated that this population accounted for 0.7% of DKA cases, with an associated increase in mortality, emphasizing the compounded risk in older adults with comorbid conditions [13]. Mortality rates in DKA exceed 5% among older adults and those with concurrent life-threatening illnesses, although death is rarely attributable solely to hyperglycemia or ketoacidosis, instead resulting from the interplay of multiple comorbidities [2].

Prognosis is strongly influenced by patient age, clinical presentation, and comorbidities. Extreme age, the presence of coma, hypotension, or significant underlying disease substantially worsens outcomes [2]. In urban Black populations, poor adherence to insulin therapy has been identified as a primary precipitating factor for DKA, with substance abuse exacerbating the risk of non-adherence [14]. These findings underscore the importance of patient education, tailored support, and improved access to medical services as essential strategies to prevent DKA and reduce associated morbidity. Interventions aimed at reinforcing adherence to insulin therapy and addressing behavioral determinants are critical to mitigating the risk of hyperglycemic emergencies. Despite its potential lethality, DKA remains largely preventable with appropriate monitoring, education, and early intervention. Surveillance data from the United States, including the CDC's United States Diabetes Surveillance System (USDSS), indicate a rising trend in hospitalization rates for DKA between 2009 and 2014, particularly among individuals younger than 45 years [15]. This trend may reflect increasing prevalence of diabetes, lifestyle factors, and gaps in disease self-management among younger populations. Notably, overall mortality associated with hyperglycemic crises among adults in the United States has declined over recent decades, reflecting improvements in clinical management, early recognition, and access to emergency care. Nevertheless, disparities persist, particularly among Black men and individuals who die outside of healthcare settings, highlighting the continued need for targeted interventions to reduce preventable mortality [16]. The geriatric population represents a particularly vulnerable group for DKA and other hyperglycemic emergencies. Age-related physiological changes, including increased insulin resistance, reduced renal function, and impaired thirst perception, increase susceptibility to hyperglycemia and dehydration, both critical determinants of DKA [17]. Older adults are also more likely to have multiple comorbidities, which complicates the clinical picture and may delay recognition and treatment. With comprehensive diabetes surveillance, timely intervention, and aggressive management of hyperglycemia, morbidity and mortality in the elderly population can be substantially reduced. Early recognition of precipitating factors, individualized treatment plans, and education regarding fluid intake and medication adherence are essential components of effective prevention strategies in this age group. In summary, the epidemiology of DKA reflects wide variations influenced by age, ethnicity, comorbidities, treatment modalities, and geographic region. Pediatric populations demonstrate significant international variability, while older adults, particularly those in institutional settings, remain at elevated risk for severe outcomes. Adherence to therapy, behavioral factors, and access to timely medical care significantly influence the incidence and prognosis of DKA. Public health measures, patient education, and early clinical intervention have contributed to reductions in mortality, yet disparities persist, indicating ongoing opportunities to improve outcomes. Focused strategies addressing high-risk populations, particularly younger adults at risk of hospitalization and older adults with comorbidities, are essential to mitigate the burden of DKA and optimize clinical outcomes across diverse populations [11][12][13][14][15][16][17].

Pathophysiology

Diabetic ketoacidosis (DKA) arises from the complex interplay between insulin deficiency and elevated counter-regulatory hormones, leading to profound metabolic derangements. Diabetes mellitus, the underlying condition predisposing to DKA, is defined by inadequate insulin secretion and relative hyperglucagonemia, which can be corrected with exogenous insulin therapy [18][19]. Under physiological conditions, rising plasma glucose levels stimulate pancreatic beta cells to produce insulin, which in turn reduces hepatic glucose output by suppressing glycogenolysis and gluconeogenesis, while promoting peripheral glucose uptake in skeletal muscle and adipose tissue. These mechanisms maintain glucose homeostasis and prevent hyperglycemia. In DKA, however, insulin deficiency combined with elevated counter-regulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone, disrupts these regulatory pathways. This results in excessive hepatic glucose production, impaired glucose utilization, and accelerated glycogenolysis, culminating in significant hyperglycemia [20]. The metabolic consequences extend beyond hyperglycemia. Insulin deficiency and elevated counter-regulatory hormones stimulate lipolysis, releasing free fatty acids from adipose tissue into circulation. These free fatty acids undergo hepatic oxidation, producing ketone bodies, primarily beta-hydroxybutyrate and acetoacetate, which accumulate in plasma and urine, causing metabolic acidosis [2]. Although glucagon is not absolutely required for ketogenesis, its presence exacerbates both hyperglycemia and ketone production in the context of insulin deficiency [21]. The emergence of euglycemic DKA, particularly in patients treated with sodium-glucose cotransporter 2 (SGLT2) inhibitors, highlights the variable relationship between glucose levels and ketogenesis, with ketone accumulation occurring despite near-normal serum glucose concentrations.

Hyperglycemia and ketonemia precipitate osmotic diuresis, leading to significant fluid loss, dehydration, and hyperosmolarity. This impairs renal perfusion and glomerular filtration, further exacerbating hyperglycemia and hyperosmolarity. The deranged renal function also contributes to disturbances in potassium homeostasis. Hyperosmolality and impaired insulin-mediated cellular uptake reduce intracellular potassium, while osmotic diuresis promotes urinary potassium loss, resulting in total body potassium depletion even if plasma potassium levels appear normal [9]. Hyperosmolarity, in particular, is a critical factor in the neurologic manifestations of DKA, including altered consciousness and coma [22]. Recent research has elucidated the inflammatory component of DKA, demonstrating that hyperglycemia triggers a systemic proinflammatory state. Elevated levels of cytokines, including tumor necrosis factor-alpha, interleukin-1 beta, interleukin-6, and interleukin-8, occur alongside increased C-reactive protein, lipid peroxidation, reactive oxygen species, plasminogen activator inhibitor-1, and circulating free fatty acids. Notably, these changes arise even in the absence of overt infection or cardiovascular disease, suggesting that DKA itself induces a transient but profound inflammatory response. Timely insulin therapy combined with intravenous fluid resuscitation normalizes these proinflammatory mediators within 24 hours, highlighting the reversibility of the inflammatory and oxidative stress responses when metabolic control is restored [2]. Overall, the pathophysiology of DKA reflects a multifactorial cascade where insulin deficiency, counter-regulatory hormone excess, ketogenesis, osmotic diuresis, electrolyte disturbances, and systemic inflammation converge to create a life-threatening metabolic crisis. Understanding these mechanisms is essential for targeted clinical interventions that address not only hyperglycemia and acidosis but also the fluid, electrolyte, and inflammatory derangements that underlie morbidity and mortality in affected patients.

History and Physical

Patients presenting with diabetic ketoacidosis (DKA) often exhibit a broad spectrum of clinical symptoms that reflect the underlying metabolic disturbance and precipitating factors. Commonly, patients report classic manifestations of hyperglycemia, including polyphagia, polyuria, and polydipsia. These symptoms may progress to signs of significant volume depletion as the osmotic diuresis advances. Decreased urine output, dry mucous membranes, reduced sweating, and general signs of dehydration are frequently observed. Gastrointestinal complaints are also common, with patients reporting anorexia, nausea, vomiting, abdominal pain, and unintentional weight loss. When DKA is precipitated by a superimposed infection, additional infectious symptoms may be present, such as fever, productive cough, dysuria, or other localized signs of infection. Neurologic symptoms, including headache, confusion, or altered mental status, may indicate developing cerebral edema, particularly in pediatric patients or severe cases. A comprehensive history should include detailed medication and social history to identify potential drug-induced or substance-related triggers, including corticosteroids, SGLT2 inhibitors, antipsychotics, alcohol, and recreational drugs [23]. The physical examination in DKA frequently demonstrates abnormalities consistent with the metabolic derangements. Vital signs often reveal tachycardia and tachypnea, with the latter sometimes manifesting as Kussmaul respirations—deep, rapid, and labored breathing aimed at compensating for metabolic acidosis. Blood pressure can range from normotension to hypotension, with hypotension indicative of severe dehydration and volume depletion. Temperature assessment may reveal febrile responses when infection is present or hypothermia in more advanced illness. The general appearance is typically ill, reflecting the systemic impact of DKA. Clinicians may note a characteristic fruity odor on the patient's breath, attributable to acetone accumulation. Signs of dehydration, such as poor skin turgor, delayed capillary refill, and dry mucous membranes, are commonly observed. Abdominal examination may reveal tenderness without peritoneal signs, often secondary to gastrointestinal effects of acidosis and dehydration. In severe cases, neurologic evaluation may uncover drowsiness, lethargy, confusion, or focal deficits. These findings necessitate immediate assessment for cerebral edema, which requires prompt intervention to prevent further neurologic compromise [24]. Accurate and thorough clinical evaluation is critical for early recognition and management of DKA. Eliciting the patient's history, including both metabolic and precipitating factors, allows clinicians to identify potential triggers and tailor treatment appropriately. Physical examination findings not only confirm the presence of metabolic derangements but also help stratify severity and guide urgent interventions. Early identification of dehydration, electrolyte disturbances, and neurologic complications facilitates rapid correction of metabolic abnormalities, reduces morbidity, and improves overall patient outcomes. Close attention to both historical and physical findings ensures that treatment is targeted, timely, and comprehensive, addressing the multifactorial nature of this life-threatening diabetic emergency.

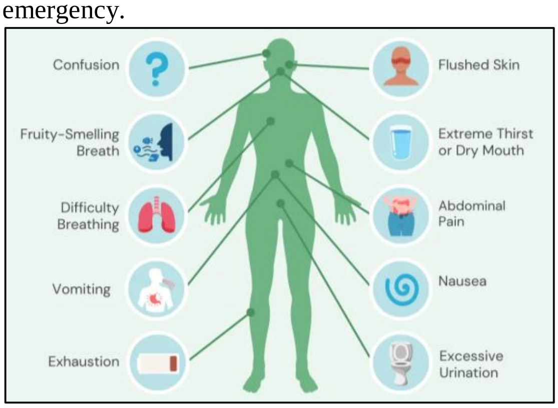

Fig. 2: Signs and symptoms of diabetic ketoacidosis.

Evaluation

The evaluation of adult patients with diabetic ketoacidosis (DKA) requires a comprehensive approach that integrates laboratory diagnostics, clinical assessment, and identification of precipitating factors. Adult DKA is commonly defined by specific laboratory criteria, including a blood glucose level exceeding 250 mg/dL, an arterial pH below 7.3, serum bicarbonate levels less than 15 mEq/L, and evidence of ketonemia or ketonuria. The anion gap serves as a critical marker for metabolic acidosis, with normal reference values around 12 mEq/L. An anion gap exceeding 14–15 mEq/L signifies the presence of increased anion gap metabolic acidosis [25]. It is important to recognize that arterial pH may appear normal or elevated in the presence of concurrent metabolic or respiratory alkalosis, as occurs in patients with persistent vomiting or diuretic use [26]. Blood glucose levels may also be deceptively normal or only moderately elevated (<300 mg/dL) in patients at risk for hypoglycemia, such as those with alcohol use disorder or patients receiving insulin or SGLT2 inhibitors [27]. Laboratory evaluation in DKA frequently demonstrates leukocytosis, even in the absence of infection, reflecting the stress response associated with hyperglycemia and acidosis. Sodium levels are often falsely low due to hyperglycemia-induced osmotic shifts and should be corrected by adding 1.6 mEq/L for each 100 mg/dL of glucose above 100 mg/dL. Serum potassium is typically elevated as a result of intracellular-to-extracellular shifts caused by insulin deficiency and acidosis. However, total body potassium is often depleted and may rapidly decline with insulin therapy. Magnesium and phosphate levels are frequently abnormal, necessitating careful monitoring and supplementation during treatment [28]. Evaluation must also include assessment of precipitating factors. Cultures of blood, urine, or sputum should be obtained when infection is suspected, as pneumonia and urinary tract infections are among the most common triggers of DKA. Measurement of glycated hemoglobin (A1C) provides insight into long-term glycemic control, helping clinicians understand the chronic contribution to acute metabolic decompensation. In acute DKA, the ratio of 3-beta-hydroxybutyrate (3-HB) to acetoacetate may increase from a normal 1:1 to as high as 10:1. Insulin therapy preferentially reduces 3-HB levels before acetoacetate, highlighting the importance of quantitative ketone monitoring, as nitroprusside tests detect only acetoacetate and provide semiquantitative results that are prone to false positives. Recent low-cost assays for 3-HB facilitate precise monitoring and therapeutic adjustments [29].

Pancreatic enzyme elevations are commonly observed in DKA, reflecting metabolic stress rather than primary pancreatic pathology. Patients presenting with abdominal pain and elevated pancreatic enzymes require careful differentiation from acute pancreatitis, which may necessitate imaging studies such as computed tomography (CT) to accurately identify structural pancreatic abnormalities [30][31]. Similarly, lipid disturbances are prevalent, with markedly elevated triglyceride and cholesterol levels observed prior to insulin therapy. Insulin rapidly reduces triglycerides to below 150 mg/dL within 24 hours, accompanied by decreases in very low-density lipoprotein (VLDL) and apolipoprotein B levels, while intermediate-density lipoprotein (IDL) and low-density lipoprotein (LDL) apolipoprotein B remain relatively unchanged [32]. Additional diagnostic studies enhance evaluation and risk stratification. Electrocardiography (ECG) detects cardiac effects of electrolyte imbalances, with peaked T waves indicating hyperkalemia and flattened T waves with U waves suggestive of hypokalemia. Imaging studies play a complementary role; chest x-rays can identify pulmonary infections or consolidation, while brain imaging with CT or magnetic resonance imaging (MRI) can detect cerebral edema. MRI is more sensitive but less readily available, and CT is often employed for urgent assessment. Importantly, imaging should never delay immediate treatment when cerebral edema is suspected, as timely intervention is critical to prevent neurologic deterioration. Comprehensive evaluation in DKA, encompassing laboratory assessment, identification of precipitating factors, and adjunctive diagnostic studies, is essential for guiding therapy, monitoring response, and preventing complications. The integration of quantitative ketone monitoring, electrolyte management, and early imaging allows clinicians to deliver targeted, individualized care while mitigating risks associated with severe metabolic derangements and associated comorbidities.

	Mild DKA	Moderate DKA	Severe DKA	HHS
Plasma glucose mmol/L	> 14	> 14	> 14	> 33.3
Arterial pH	7.25-7.30	7.00-7.24	< 7.00	> 7.30
Sodium Bicarbonate (mEq/L)	15 – 18	10 - <15	< 10	> 15
Urine Ketones	Positive	Positive	Positive	Small
Serum Ketones	Positive	Positive	Positive	Small
Serum Osmolality (mOsm/kg)	Variable	Variable	Variable	> 320
Anion Gap	> 10	> 12	> 12	variable
Mental Status	Alert	Alert/Drowsy	Stupor/Coma	Stupor/Coma

Fig. 3: Diagnostic criteria of DKA.

Treatment / Management

The management of adult diabetic ketoacidosis (DKA) is a complex, multifaceted process that requires prompt recognition and systematic intervention to correct fluid deficits, restore metabolic balance, replace electrolytes, and provide supportive care [33]. Effective management reduces morbidity and mortality, improves patient outcomes, and prevents long-term complications associated with severe hyperglycemia and metabolic acidosis. Fluid resuscitation constitutes the cornerstone of initial DKA management. Patients presenting with DKA often exhibit fluid deficits ranging from 10% to 15% of total body weight, necessitating rapid correction of hypovolemia to restore tissue perfusion, improve renal function, and facilitate clearance of ketone bodies [2]. Fluid replacement alone can also aid glycemic control independent of insulin therapy by reducing hyperosmolality and improving circulatory dynamics. The choice of fluids has traditionally favored isotonic crystalloids, particularly 0.9% normal saline, which remains the standard for initial resuscitation. Although concerns have been raised regarding hyperchloremic metabolic acidosis from large-volume saline administration, studies comparing normal saline to other crystalloids, including Ringer's lactate, demonstrate no significant difference in clinical outcomes, making saline the preferred initial fluid [34][35][36][37]. Colloids have not shown superior efficacy in correcting DKA-related hypovolemia and are generally reserved for specific circumstances. Initial infusion rates typically involve 15 to 20 mL/kg over the first hour, with adjustments made based on hemodynamic stability and patient comorbidities. Comparative studies between rapid and slower infusion rates suggest that gradual hydration can be equally effective in non-critically ill patients, although aggressive fluid resuscitation remains essential in hypotensive or severely volume-depleted individuals [38]. Pediatric populations demonstrate an increased risk of cerebral edema with aggressive early volume replacement, but adult studies indicate that the most significant risk arises in patients with advanced dehydration and severe metabolic derangements [39]. Subsequent maintenance fluid therapy is individualized according to serum electrolytes, hemodynamic status, and urine output, with 0.45% NaCl recommended for hypernatremic patients and 0.9% NaCl for hyponatremia, adjusted further if hyperchloremic metabolic acidosis develops [40].

Insulin therapy is central to reversing the catabolic state of DKA. Continuous intravenous infusion of regular insulin remains the standard of care, and therapy should only commence when serum potassium is ≥3.5 mmol/L to prevent hypokalemia-induced arrhythmias, cardiac arrest, or respiratory failure. Traditional protocols include a 0.1 U/kg bolus followed by a 0.1 U/kg/h infusion. More recent studies suggest that a bolus may be omitted if the infusion is delivered at 0.14 U/kg/h [41]. When plasma glucose falls to 200–250 mg/dL, dextrose-containing fluids are added to prevent hypoglycemia, and the insulin infusion rate is typically reduced to 0.05 U/kg/h. Euglycemic DKA cases, including those induced by SGLT2 inhibitors, require lower insulin infusion rates from initiation and concurrent dextrose supplementation to avoid precipitous hypoglycemia. Subcutaneous insulin regimens provide a safe and effective alternative to intravenous insulin in mild, uncomplicated DKA. Insulin lispro administered hourly has demonstrated equivalent efficacy in non-intensive care settings, offering a cost-effective approach while maintaining glycemic control [42].

Insulin aspart has similarly shown effectiveness for subcutaneous administration [43]. Transitioning from intravenous to subcutaneous therapy is appropriate once DKA resolves, defined by blood glucose <200 mg/dL in addition to normalization of two of the following parameters: serum bicarbonate ≥15 mEq/L, venous pH >7.3, or an anion gap ≤12 mEq/L. Insulin-naive patients should begin a multidose regimen at 0.5–0.8 U/kg/day, continuing the intravenous infusion for at least two hours post-transition to prevent recurrence [44]. Blood glucose and metabolic panels should be reassessed prior to discontinuation of intravenous therapy. Overall, DKA management requires a coordinated strategy that integrates fluid resuscitation, electrolyte correction, insulin therapy, and ongoing monitoring. Individualization of therapy based on patient age, comorbidities, hemodynamic status, and precipitating factors is essential to ensure safe and effective resolution of the acute metabolic derangements while minimizing complications and optimizing long-term glycemic control. Timely intervention, close monitoring, and appropriate transition to subcutaneous insulin therapy remain the pillars of successful management of adult DKA.

Electrolyte Replacement

Management of electrolyte disturbances in adult diabetic ketoacidosis (DKA) is essential to prevent life-threatening complications, optimize metabolic recovery, and ensure safe administration of insulin therapy. Potassium homeostasis is particularly critical, as patients with DKA often present with mild to moderate hyperkalemia despite significant total body potassium deficits [45][46]. The initiation of insulin therapy drives potassium into the intracellular space, rapidly lowering serum potassium levels and predisposing patients to severe hypokalemia, which can precipitate cardiac arrhythmias, arrest, and respiratory muscle weakness. Consequently, serum potassium should be closely monitored, and insulin therapy should be withheld if potassium is below 3.5 mmol/L. In patients with potassium concentrations between 3.5 and 5.2 mEq/L, replacement therapy is indicated to maintain serum potassium within the optimal range of 4–5 mEq/L. Administration of 20–30 mEq of potassium per liter of intravenous fluids is typically sufficient for most patients, with lower doses indicated for individuals with renal impairment [44][47]. Magnesium replacement is often required concurrently, as hypomagnesemia frequently coexists with hypokalemia. Correction of magnesium deficiency is necessary to achieve effective potassium repletion, as potassium levels may remain refractory until magnesium stores are restored. Magnesium supplementation therefore constitutes an integral component of electrolyte management in DKA, particularly in patients with prolonged or recurrent hypokalemia. The role of bicarbonate replacement in DKA remains limited and is generally reserved for severe acidemia. Clinical trials have shown no significant difference in the time to acidosis resolution or length of hospital stay between patients receiving intravenous bicarbonate and those who did not [48]. In pediatric populations, bicarbonate therapy has been associated with increased risk of cerebral edema, particularly in patients with low $PaCO_2$ and elevated blood urea nitrogen at presentation [49]. Potential risks of bicarbonate administration include paradoxical cerebrospinal fluid acidosis, hypokalemia, a sodium load, and exacerbation of cerebral edema. Current American Diabetes Association (ADA) guidelines recommend sodium bicarbonate administration only for patients with arterial pH below 7.1 [48].

Phosphate replacement in DKA is generally not required except in cases of severe hypophosphatemia. Randomized trials have demonstrated that phosphate supplementation does not significantly alter DKA resolution, insulin requirements, glucose metabolism, or clinical outcomes in most patients [50][51]. Rare complications, such as seizures in patients with critically low phosphate levels, underscore the need for individualized assessment and supplementation in select cases. Laboratory monitoring forms the backbone of safe and effective electrolyte replacement. Hourly point-of-care glucose testing guides insulin therapy and fluid adjustments, while serum electrolytes should be assessed every two hours initially, decreasing to every four hours once clinical stability is achieved. Baseline renal function should be evaluated using blood urea nitrogen, and venous or arterial blood gases should be measured at presentation and repeated as clinically indicated to monitor acid-base status and guide therapy. Supportive care is another critical component of DKA management. Intubation should be avoided whenever possible due to associated risks, including elevation of $PaCO_2$ during sedation, reduced compensatory respiratory alkalosis, aspiration, and difficulty in maintaining adequate ventilation. Intubation may be necessary when patients lose the ability to compensate for metabolic acidosis, often in cases of coma or profound fatigue. Ventilator settings should replicate the patient's compensatory minute ventilation to prevent further acid-base derangements, with initial tidal volumes of approximately 8 mL/kg of ideal body weight and a respiratory rate matching the patient's pre-intubation rate [52]. Cerebral edema is a rare but serious complication of DKA, necessitating close neurological monitoring. Patients presenting with altered mental status, focal deficits, or coma require immediate intervention. Therapeutic options include mannitol and hypertonic saline, both of which increase serum osmolarity and reduce brain volume. Hypertonic saline may offer advantages, including lower hypotension risk and improved cerebral perfusion [53]. Addressing precipitating factors is essential for comprehensive management. Infections are common triggers of DKA and require prompt identification and initiation of appropriate antimicrobial therapy. Other contributors, such as medication changes, acute illness, or substance use, must be recognized and managed simultaneously with the metabolic derangements of DKA to prevent recurrence and optimize patient outcomes. Effective electrolyte replacement, vigilant laboratory monitoring, careful supportive care, and targeted treatment of precipitating causes collectively ensure the stabilization and recovery of patients with DKA, minimizing complications and improving short- and long-term prognosis.

Enhancing Healthcare Team Outcomes in Diabetic Ketoacidosis: Integrating Nursing, Pharmacy, Dentistry, Radiology, Epidemiology, and Family Medicine

Diabetic ketoacidosis (DKA) remains a critical and potentially fatal metabolic complication of diabetes, manifesting as hyperglycemia, ketonemia, and metabolic acidosis. Although it is most frequently observed in type 1 diabetes, patients with type 2 diabetes are also susceptible, particularly under stressors such as acute illness, infections, medication nonadherence, or new-onset diabetes. Recognition of these precipitating factors and prompt intervention are essential to prevent complications such as cerebral edema, cardiovascular instability, electrolyte imbalance, and multi-organ dysfunction. Effective management is complex and relies on the coordinated efforts of a multidisciplinary healthcare team encompassing nursing, pharmacy, dental hygiene, radiology, epidemiology, and family medicine. Physicians and family medicine practitioners play a pivotal role in the early identification and diagnosis of DKA. They assess clinical presentation, order laboratory investigations including serum glucose, electrolytes, ketone levels, and arterial blood gases, and initiate critical interventions such as fluid resuscitation and intravenous insulin therapy. Family medicine specialists additionally ensure continuity of care, integrating preventive strategies and education into outpatient follow-up to reduce recurrence and support long-term diabetes management. Nurses are integral to continuous monitoring and bedside management. They assess vital signs, neurologic status, hydration, and signs of infection, facilitating rapid intervention when deterioration occurs. Nurses also administer prescribed therapies, monitor response to fluid and insulin replacement, and provide patient education regarding self-monitoring of blood glucose, adherence to therapy, and early recognition of recurrent symptoms. Their consistent observation and timely reporting allow for adjustments in therapy that optimize patient outcomes. Pharmacists contribute by ensuring safe and effective medication management. They evaluate potential drug interactions, recommend insulin titration strategies, and guide the use of adjunctive therapies such as SGLT2 inhibitors or GLP-1 receptor agonists, which may influence ketone production. Pharmacists also counsel patients on the safe administration of medications and provide guidance on maintaining adherence, particularly in complex polypharmacy scenarios [52][53].

Dental hygienists contribute indirectly by promoting oral health, which can reduce systemic infections that may precipitate DKA. They identify oral infections, recommend preventive strategies, and collaborate with the broader healthcare team to mitigate infection-related triggers. Radiologists support diagnosis and complication monitoring through imaging, including chest radiographs to identify pneumonia, computed tomography for abdominal complications, and brain imaging to detect cerebral edema. Their input informs clinical decision-making and guides targeted interventions. Epidemiology technicians contribute to population-level understanding and prevention of DKA episodes. They track incidence, identify high-risk populations, analyze trends in hospital admissions, and monitor outcomes. Their data supports evidence-based interventions, quality improvement initiatives, and public health strategies that reduce DKA-related morbidity and mortality. Collaboration among these specialties enhances patient-centered care by ensuring that each aspect of DKA management is addressed comprehensively. Clear communication, shared protocols, and coordinated interventions minimize delays, optimize fluid and insulin therapy, manage electrolyte disturbances, and address underlying infections or comorbidities. Interprofessional education and teamwork improve adherence to treatment plans, reduce hospital stay durations, and lower the risk of recurrent episodes. In summary, managing DKA effectively requires a robust, coordinated healthcare team. Physicians and family medicine practitioners provide early diagnosis and treatment, nurses deliver continuous monitoring and education, pharmacists optimize medication safety, dental hygienists mitigate infection risks, radiologists support diagnostic accuracy, and epidemiology technicians guide preventive strategies. The integration of these specialties ensures rapid stabilization, prevents complications, and promotes long-term patient outcomes, illustrating the critical value of interprofessional collaboration in the care of patients with DKA [53].

Conclusion:

Diabetic ketoacidosis remains a major acute complication of diabetes, but the article demonstrates that it is highly preventable and treatable when recognized promptly and managed systematically. DKA results from a convergence of metabolic, infectious, pharmacologic, and behavioral factors, emphasizing the importance of comprehensive assessment and early identification of precipitating causes such as infection, medication nonadherence, and newer pharmacologic agents. Effective treatment hinges on rapid fluid resuscitation, insulin administration, and vigilant electrolyte correction, all of which have been shown to significantly reduce morbidity and mortality when applied using standardized protocols. A key contribution of the article is its emphasis on multidisciplinary collaboration. Physicians guide diagnosis and acute management; nurses provide continuous monitoring and patient education; pharmacists optimize medication safety; radiologists support detection of complications; dental hygienists help reduce infection-related risks; and epidemiology teams monitor population trends to inform prevention strategies. This coordinated approach strengthens every phase of care—from prevention to acute intervention to long-term management. Ultimately, the article reinforces that improved outcomes in DKA rely not only on clinical treatment but also on communication, patient engagement, and system-wide collaboration. When healthcare teams integrate their expertise, they can significantly reduce recurrence, hospitalizations, and the overall burden of this life-threatening condition.

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