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Diabetic ketoacidosis: A Common Complication of Diabetes Mellitus-Diagnosis, Treatment, and Interventions-An Updated Review Article

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Abstract:
Background: Diabetic ketoacidosis (DKA) is a severe and potentially fatal complication of diabetes mellitus (DM), arising from insulin deficiency and characterized by hyperglycemia, hyperketonemia, and metabolic acidosis. Despite advancements in diabetes care, DKA remains a leading cause of hospitalization and mortality among diabetic patients.
Aim: This review aims to consolidate recent findings on the epidemiology, pathophysiology, diagnosis, treatment, and challenges in managing DKA to optimize clinical outcomes. Also, focus on the contribution role of pharmacists, clinical pathologists, and nursing in management and controlling of the DKA.
Methods: A systematic literature search was conducted using PubMed, targeting articles published between 2012 and 2023 in peer-reviewed journals. Boolean operators integrated terms such as "epidemiology," "pathophysiology," "diagnosis," and "therapy for diabetic ketoacidosis." Six relevant articles were selected for detailed analysis.
Results: DKA accounts for approximately 14% of hospital admissions and 16% of diabetes-related deaths. It is more prevalent in type 1 diabetes but also occurs in type 2 cases. Pathophysiologically, insulin deficiency, increased counter-regulatory hormones, and oxidative stress contribute to ketosis and acidosis. Diagnosis relies on clinical presentation and laboratory findings such as elevated plasma glucose, arterial pH, serum ketones, and anion gap. Management involves correcting fluid and electrolyte imbalances, initiating insulin therapy, and addressing precipitating factors like infections. Advances in monitoring and therapeutic strategies have improved outcomes, though challenges such as hypoglycemia, fluid overload, and euglycemic DKA persist.
Conclusion: DKA is a critical metabolic emergency requiring prompt, multidisciplinary intervention. Despite progress in treatment protocols, patient outcomes can be improved by addressing adherence barriers, refining

diagnostic methods, and mitigating treatment complications. Future research should prioritize strategies for early detection and targeted interventions.
Keywords: Diabetic ketoacidosis, insulin deficiency, hyperglycemia, metabolic acidosis, treatment strategies, complications

Introduction:

Diabetic ketoacidosis is a severe and potentially fatal complication of diabetes that arises due to an absolute or relative deficiency of insulin, leading to significant hyperglycemia and the accumulation of ketones in the bloodstream. This condition constitutes a medical emergency that necessitates prompt intervention to avert life-threatening outcomes, including cerebral edema, acute respiratory distress syndrome, and sepsis [1]. The global incidence of diabetic ketoacidosis is rising, particularly among children and adolescents with type 1 diabetes [1]. Despite advancements in insulin therapy and heightened patient awareness, diabetic ketoacidosis continues to represent a frequent cause of hospitalization and is associated with considerable morbidity and mortality rates [2,3].Immediate treatment of severe diabetic ketoacidosis requires a multidisciplinary approach aimed at rectifying fluid and electrolyte imbalances, reinstating insulin sensitivity, and mitigating the risk of complications. One of the primary objectives in initial management is the prevention of progression to critical complications such as cerebral edema, a rare yet fatal consequence [4,5]. Recent studies have elucidated the intricate pathophysiology of diabetic ketoacidosis, highlighting the interplay of insulin deficiency, dysregulated glucose metabolism, and acid-base derangements [3]. Advances in monitoring technologies and therapeutic techniques have enhanced the precision of managing fluid resuscitation, electrolyte correction, and insulin therapy [5].However, the management of diabetic ketoacidosis presents several challenges. For instance, insulin therapy, while essential, can lead to rapid reductions in blood glucose levels, raising the risk of hypoglycemia. Similarly, fluid replacement therapy, though critical for addressing dehydration, carries the risk of fluid overload, particularly in patients with preexisting comorbidities [2]. Addressing these complexities necessitates a comprehensive analysis of current strategies for the immediate management of severe diabetic ketoacidosis [4]. Such an evaluation would integrate contemporary research, offer evidence-based guidelines, and delineate priorities for future investigation to optimize patient outcomes.

This review consolidates and critically examines prevalent themes in the literature regarding the epidemiology, pathophysiology, and emergent therapeutic interventions for diabetic ketoacidosis (DKA), a significant complication predominantly associated with type 1 diabetes mellitus (T1DM) and less frequently with type 2 diabetes mellitus (T2DM). The authors employed an advanced and systematic search methodology using PubMed, a robust electronic database offering access to over 35 million articles indexed in MEDLINE across disciplines such as medicine, biomedicine, nursing, and life sciences. Boolean operators were utilized to integrate search terms like "epidemiology," "pathophysiology," "diagnosis," and "emergent therapy for diabetic ketoacidosis." To ensure inclusivity, alternative relevant terms were incorporated into the search strategy.The scope of the review was limited to English-language articles published between 2012 and 2022 in peer-reviewed academic journals. Additionally, the selection criteria mandated that articles contain both abstracts and full texts to facilitate comprehensive screening. The initial search yielded 37 articles, from which six were ultimately included based on their relevance and their discussion of recent advancements in the field.

Definition and Epidemiology

Diabetic ketoacidosis is a critical acute metabolic complication of diabetes mellitus characterized by hyperglycemia, hyperketonemia, and metabolic acidosis. It results from severe insulin deficiency, which heightens the demand for insulin and depletes endogenous reserves [5]. This deficiency triggers excessive fat breakdown, leading to an overproduction of ketone bodies. Despite advancements in diabetic care and patient education, DKA accounts for approximately 14% of hospital admissions among diabetic patients and 16% of diabetes-related mortalities [4].Although DKA is most commonly observed in individuals with type 1 diabetes, it occurs at an incidence rate of two episodes per 100 patient-years. Approximately 3% of individuals with type 1 diabetes present with DKA during their initial diagnosis. While DKA is less frequently observed in type 2 diabetes patients, it remains a potential complication [6,7]. In developing nations, the exact incidence of DKA is unclear, though it may exceed that of developed countries [8]. Among racial groups, a higher prevalence of type 1 diabetes and DKA is observed in White populations. Females are marginally more predisposed to DKA than males, with recurrent cases often attributed to non-adherence to insulin therapy among young females with type 1 diabetes [6,9].DKA is notably more prevalent among children and adolescents with type 1 diabetes compared to adults, although it can occur at any age. Interventions between the onset of symptoms and the progression of DKA are possible, yet factors such as ethnic minority status, absence of health insurance, low BMI, preceding infections, and delays in treatment heighten the risk among children and youth [9].

Pathophysiology

The pathophysiological abnormalities in diabetic ketoacidosis arise from absolute or relative insulin deficiency, coupled with an increase in counter-regulatory hormones that induce a catabolic state and exacerbate insulin resistance. These hormones include glucagon, growth hormone, and catecholamines (epinephrine and norepinephrine) [10]. The most frequent precipitating factor of DKA is a decline in insulin activity or a rise in insulin demand, which can result from missed insulin doses, improper insulin administration, or infection in diabetic patients [11].Insulin deficiency impairs cellular glucose uptake, leading to intracellular energy deprivation. Consequently, cells transition to free fatty acids (FFA) as an alternative energy source [12,13]. In the absence of insulin, lipolysis in adipocytes produces excess FFAs, which are transported to the liver. Within hepatic mitochondria, these FFAs undergo oxidation, producing ketone bodies such as beta-hydroxybutyrate, acetone, and acetoacetate. Insulin typically regulates this biochemical pathway, but insufficient insulin results in unregulated ketone production [14].Under normal conditions, triglycerides predominate over ketones, and the body efficiently disposes of ketones to maintain metabolic balance. However, in the absence of sufficient insulin, excessive ketone accumulation results in ketosis, progressing to DKA [15]. Concurrently, glucagon, catecholamines, cortisol, and growth hormone exacerbate hyperglycemia through gluconeogenesis and glycogenolysis [15].Stressors such as infections (e.g., urinary or lower respiratory tract infections), trauma, myocardial infarction, acute pancreatitis, burns, surgeries, strokes, and substance abuse can further escalate DKA risk [16]. These stressors stimulate the release of inflammatory cytokines, amplifying counter-regulatory hormones like glucagon, catecholamines, cortisol, and growth hormone [17]. The resulting catabolic state drives increased lipolysis and proteolysis to synthesize glucose, exacerbating hyperglycemia [18].

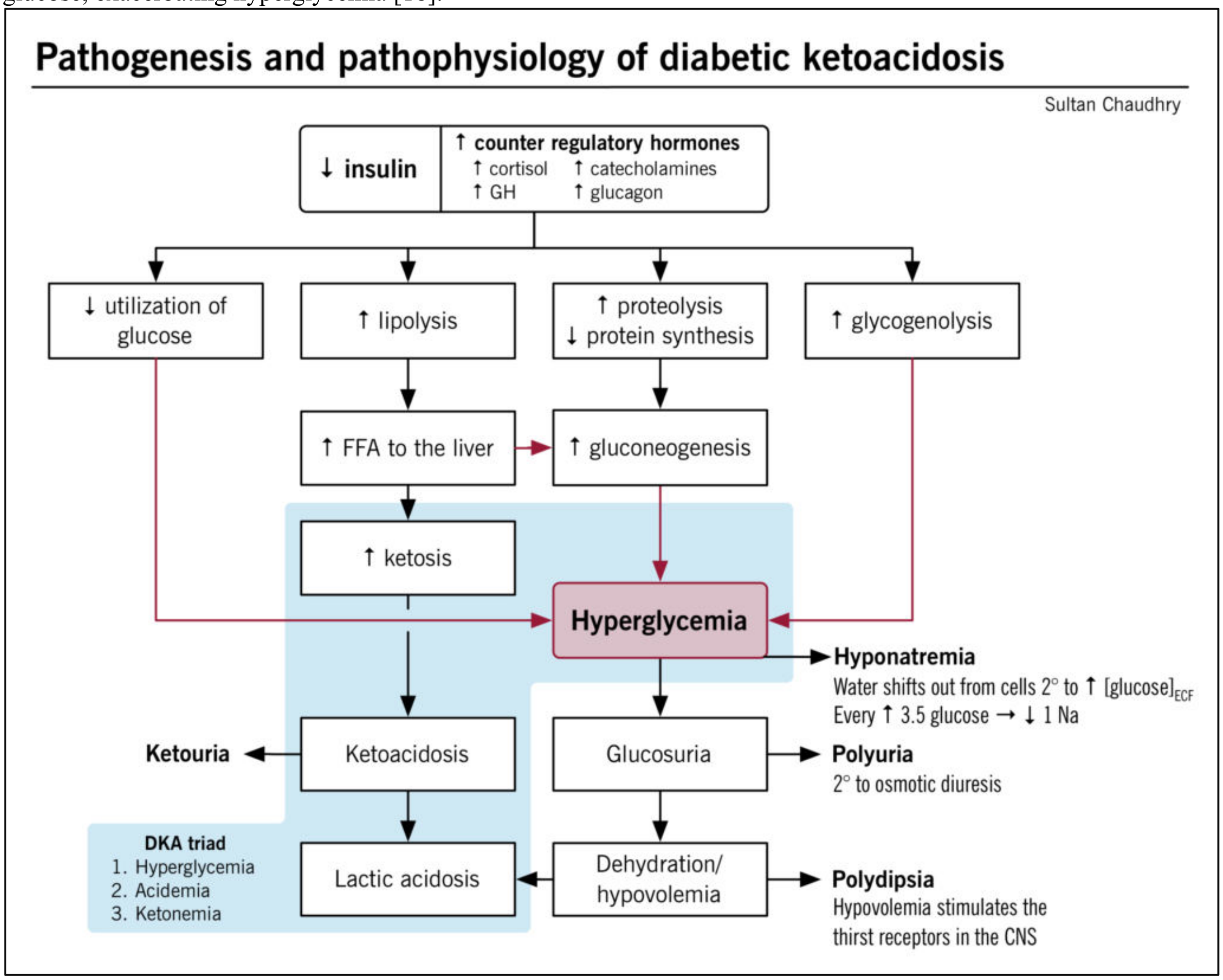

Figure 1: Pathophysiology of DKA.

Causes

Diabetic ketoacidosis (DKA) commonly arises from poor adherence to insulin therapy, infections, or an initial diagnosis of diabetes [19]. Among patients with type 1 diabetes, the primary precipitating factor is noncompliance with prescribed treatment protocols. Conversely, infections are the predominant trigger in patients with type 2 diabetes [20]. Additional causative factors include vascular events such as acute coronary syndrome, cerebrovascular accidents, critical limb ischemia, bowel ischemia, and various forms of shock. Excessive alcohol consumption and the use of illicit drugs like cocaine and methamphetamine are also recognized as potential causes. Moreover, certain medications, including antipsychotics such as clozapine, risperidone, and olanzapine, have been implicated in the onset of DKA [19].

Laboratory Abnormalities and Diagnosis

In 2003, the American Diabetes Association (ADA) updated the diagnostic criteria for DKA to include severity classifications of mild, moderate, and severe. These classifications are determined based on laboratory parameters such as plasma glucose, arterial pH, serum bicarbonate, ketone levels, anion gap, and sensorium status [2,21].Patients with mild DKA exhibit plasma glucose levels exceeding 13.9 mmol/L, arterial pH between 7.25 and 7.30, serum bicarbonate levels of 15–18 mmol/L, and positive urine and serum ketones. The anion gap is typically greater than 10, and serum sodium and potassium levels remain within the normal range.Moderate DKA is characterized by plasma glucose levels above 13.9 mmol/L, arterial pH ranging from 7.00 to 7.24, and serum bicarbonate levels of 10–14.9 mmol/L. Ketone levels are elevated (+++), and the anion gap exceeds 12. Sensorium changes may include drowsiness, and serum sodium levels tend to be low, while serum potassium and phosphate levels are elevated.In severe cases, plasma glucose remains above 13.9 mmol/L, arterial pH falls below 7.00, and serum bicarbonate levels drop below 10 mmol/L. Ketones are significantly elevated (+++), and the anion gap exceeds 12. Severe DKA often presents with stupor or coma, accompanied by low serum sodium and elevated potassium and phosphate levels [21–23].The diagnosis of diabetic ketoacidosis is confirmed by the triad of hyperglycemia, ketonemia, and metabolic acidosis, which are supported by the observed laboratory abnormalities [2,21–23].

An evaluation of electrolytes and blood gases should be performed to detect anion gap metabolic acidosis in patients who report with increased blood glucose levels greater than 13.9 mmol/l (250 mg/dl), constitutional symptoms, or suspected diabetic ketoacidosis (DKA) [2]. If insulin therapy is stopped suddenly or if the insulin pump malfunctions, diabetic ketoacidosis (DKA) in people with type 1 diabetes can appear quickly—within hours [21]. A blood glucose threshold of 13.9 mmol/l (250 mg/dl) is part of the diagnostic criteria for DKA, using the American Diabetes Association's revised definition [2]. However, unless certain factors, such pregnancy or poor oral intake, are present, DKA rarely happens at lower glucose levels [22]. Even in people who have never been diagnosed with diabetes, clinicians should check serum glucose levels and consider DKA as a differential diagnosis in patients with anion gap metabolic acidosis [15]. Although DKA is strongly suggested by a blood glucose level exceeding 13.9 mmol/l (250 mg/dl), an increased glucose level by itself is not enough to make the diagnosis. Patients with diabetes who also have concurrent infections, myocardial infarction, stroke, or other serious illnesses should be suspected of having DKA by healthcare professionals because these disorders frequently cause DKA and require intensive treatment [13]. Moreover, diabetic individuals who experience nausea and vomiting should be evaluated for DKA, even if their blood glucose levels are less than 13.9 mmol/l (250 mg/dl) [10].

Patients with hepatic dysfunction or prolonged fasting who continue insulin therapy are more likely to experience euglycemic DKA, which is defined by normal glucose levels and occurs in 1–7% of documented cases [12]. Atypical antipsychotic medicines including risperidone and clozapine, as well as glucocorticoids and thiazides, might worsen hyperglycemia and possibly cause DKA [23]. Because atypical antipsychotic medications are known to raise the risk of diabetes, glucose intolerance, and diabetic ketoacidosis (DKA), patients on these prescriptions must have their anion gap and ketone levels closely monitored. These issues might be resolved by switching to different antipsychotic medications [24, 25].

Depending on how severe the incident was, DKA can appear in a variety of ways. Mild to moderately ill patients may have headaches, lethargy, exhaustion, or poor appetite. While polyuria and polydipsia may have emerged abruptly in type 1 diabetes, they may have developed over weeks or months in type 2 diabetes [8]. Dehydration, hypokalemia, ketonemia, and delayed stomach emptying all contribute to the common occurrence of nausea, vomiting, and abdominal pain [24]. Dehydration symptoms include poor skin turgor, decreased axillary perspiration, and postural hypotension are frequently found during physical examinations [2]. Another compensatory deep breathing pattern that may be seen in response to metabolic acidosis is Kussmaul respirations [5]. While its absence does not rule out DKA, a fruity smell on the breath brought on by high acetone levels may be a clinical sign. Because it can either go away with DKA treatment or point to an underlying acute abdominal disease that caused the episode, abdominal discomfort can be difficult to diagnose [19]. The degree of acidity is frequently correlated with the intensity of stomach pain [15]. In order to detect any potential triggering causes, such as infections or cardiovascular events, clinicians should thoroughly assess the physical examination. There may also be noticeable changes in mental status, from mild lethargy to coma; in severe cases, tachycardia, hypotension, or coma may be present [20].

A sophisticated diagnostic method for measuring β-hydroxybutyrate (β-OHB), the main ketone body in DKA, is capillary blood ketone testing [13]. Capillary blood ketone measurement yields more accurate findings than urine ketone testing, although it is more expensive. In the early stages of DKA or when β-OHB is being converted to acetoacetate during recovery, traditional urine dipsticks that detect acetoacetate but not β-OHB may provide false positives [23]. Although venous pH measurement may provide a workable substitute in the majority of clinical circumstances, arterial blood gas analysis is still the gold standard for determining the level of acidity. In healthy people, the anion gap usually ranges between 7 and 9 mmol/l; in DKA patients, it rises to about 25 mmol/l [2]. Although they are uncommon, mixed acid-base disturbances with pH levels close to normal shouldn't change how DKA is treated [17]. There are few exceptions to the rule that hyperglycemia of greater than 13.9 mmol/l is necessary for the diagnosis of DKA. For example, persistent alcohol use, prolonged vomiting, malnutrition, liver failure, or pregnancy can all result in DKA without hyperglycemia [9]. In the pathophysiology of DKA, the release of acetyl-CoA due to lipolysis results in the hepatic synthesis of three ketone bodies: acetone, acetoacetate, and β-OHB. Acetone, which gives DKA its fruity smell, has no effect on acidity, whereas β-OHB is the main contributor to metabolic acidosis [18]. In contrast to urine testing, the American Diabetes Association advises blood ketone testing for β-OHB as the best way to diagnose and track DKA [8]. Arterial pH measurement, which is usually less than 7.3 in DKA, is nevertheless essential for diagnosis and determining the severity of the illness. Unless a respiratory examination is necessary, venous pH measurements—which are roughly 0.03 units lower than arterial pH—may be adequate [12, 23]. If unexpected laboratory data, including near-normal pH levels, are seen in DKA cases, clinicians should think about other possible diagnosis or contributory causes.

Differential Diagnosis

Diabetic ketoacidosis (DKA) often presents with a broad spectrum of clinical manifestations that can resemble various other common pathological conditions. This complexity necessitates a thorough differential diagnosis to distinguish DKA from conditions with overlapping presentations. Such conditions include starvation ketoacidosis, pancreatitis, alcoholic ketoacidosis, lactic acidosis, uremia, diabetic medication overdose, hyperosmolar hyperglycemic nonketotic syndrome, and myocardial infarction. Therefore, when DKA is suspected, it is critical to rule out these alternative pathologies to avoid misdiagnosis and ensure accurate treatment [1][2].

Treatment

The primary therapeutic objectives in managing hyperglycemic crises, including DKA, are to restore circulatory volume and tissue perfusion, gradually reduce serum glucose and osmolality, correct electrolyte imbalances, and identify and address any underlying precipitating factors. Achieving these goals requires diligent patient monitoring using both clinical evaluations and laboratory parameters. The following sections elaborate on the specific therapeutic interventions for DKA management [2][26].

Fluid Therapy:

DKA is characterized by significant volume depletion, typically amounting to an estimated 6 liters of total body water deficit. The initial fluid therapy is designed to expand intravascular volume and maintain adequate urine output. The recommended first-line intervention involves isotonic saline, administered at a rate of 15–20 ml/kg of body weight per hour or 1–1.5 liters during the first hour. Subsequent fluid replacement should be tailored based on hydration status, serum electrolyte concentrations, and urine output. Over a period of 12–24 hours, approximately half of the estimated sodium and water deficits should be replenished. Initiating fluid therapy prior to insulin administration allows for the measurement of serum potassium levels and facilitates osmotic diuresis while suppressing counter-regulatory hormone secretion, which collectively contribute to blood glucose reduction.

Insulin Therapy:

Insulin is indispensable for treating DKA. Regular insulin is administered as an intravenous (IV) bolus at a dose of 0.1 U/kg body weight, followed by a continuous infusion at the same rate (0.1 U/kg/hour). When plasma glucose levels decline to 200–250 mg/dl, the infusion rate should be reduced to 0.05 U/kg/hour. The insulin infusion rate must be adjusted to maintain optimal blood glucose levels. Given its short half-life and ease of titration, continuous IV infusion of regular insulin is preferred.

Potassium Therapy:

DKA is frequently associated with mild to moderate hyperkalemia resulting from insulin deficiency and acidosis. Initiating insulin therapy lowers serum potassium levels. Therefore, potassium replacement should be commenced as soon as serum potassium levels fall below 5.3 mmol/L to prevent hypokalemia. Simultaneous correction of acidosis and fluid volume restoration also helps stabilize potassium levels.

Bicarbonate Therapy:

Bicarbonate therapy is not routinely indicated for DKA management, as its benefits are limited. However, in adult patients with a pH of 6.9 or lower, administering 100 mmol of sodium bicarbonate diluted in 400 ml of sterile water, along with 20 mmol of potassium chloride (KCl), at a rate of 200 ml/hour for 2 hours is recommended. This treatment should be repeated every 2 hours until the pH rises to 7.0.

Phosphate Therapy:

Phosphate supplementation is not typically necessary but may be considered in patients experiencing adverse effects due to severe hypophosphatemia. Care must be taken, as excessive phosphate administration can result in hypocalcemia.

Nursing Aspects of DKA:

Nursing care plays a critical role in managing DKA, particularly in patients presenting in comatose or pre-comatose states. Nurses are responsible for maintaining infusions, managing nasogastric tubes, central venous pressure (CVP) lines, urinary catheters, and ECG monitors. Vital signs, including temperature, pulse, blood pressure, respiration, and cognitive state, should be assessed hourly to monitor patient progress and guide treatment adjustments.Healthcare professionals must also recognize that patients with DKA may have normal potassium levels before treatment initiation. Close monitoring is crucial to prevent severe hypokalemia during insulin therapy.

Endocrine Society's Clinical Practice Guidelines for Hospitalized Patients with Diabetes

The Endocrine Society has established evidence-based guidelines to enhance glycemic management and overall outcomes for hospitalized patients with diabetes or hyperglycemia. These guidelines emphasize several critical components of care:

- Continuous glucose monitoring devices are recommended for patients with impaired glycemic control to prevent hypoglycemic crises.
- Scheduled insulin therapy is advised for patients on glucocorticoid therapy or enteral nutrition who develop hyperglycemia.
- Psychologically and physically capable outpatients using regular insulin pump therapy may continue self-management under medical supervision.
- Educating hospitalized diabetic patients about disease management improves glycemic control post-discharge and reduces readmission rates.
- Elective surgical patients with diabetes should achieve preoperative HbA1c levels below 8% and immediate preoperative blood glucose levels under 180 mg/dl to optimize postoperative recovery.
- Carbohydrate-containing drinks should not be administered to preoperative diabetic patients.
- Correctional insulin alone can be used for initial management in newly diagnosed hyperglycemia or well-controlled diabetes upon hospital admission.
- Patients with persistent blood glucose levels exceeding 180 mg/dl while on insulin therapy should transition to scheduled insulin regimens.
- For patients with type 2 diabetes and mild hyperglycemia, a combination of Dipeptidyl peptidase inhibitors and correctional insulin may be considered, provided no contraindications exist.

These guidelines provide a comprehensive framework for the inpatient management of non-critically ill adults with diabetes or hyperglycemia, aiming to improve both in-hospital outcomes and post-discharge glycemic control [2][26-31].

Nursing Intervention Plans:

Effective nursing intervention plans are integral to the comprehensive management of diabetic ketoacidosis (DKA). These plans encompass a range of targeted actions designed to stabilize the patient's condition, prevent complications, and promote recovery. The nursing care process requires a multidimensional approach that includes monitoring physiological parameters, administering appropriate treatments, and ensuring patient education for long-term disease management. Below is a detailed exploration of nursing interventions for DKA.

Monitoring and Assessment

The initial focus of nursing care is continuous monitoring and assessment of the patient's vital signs, laboratory values, and clinical status. Frequent monitoring of blood glucose levels is essential to evaluate the patient's response to insulin therapy. Blood glucose levels should ideally be assessed hourly to ensure they are within the therapeutic range. Monitoring serum electrolytes, particularly potassium, sodium, and bicarbonate, is critical due to the electrolyte imbalances commonly associated with DKA. Additionally, arterial blood gas (ABG) analysis should be performed to assess the severity of metabolic acidosis and the effectiveness of treatment interventions. Observing and documenting changes in blood pressure, heart rate, respiratory rate, and cognitive status is essential for detecting complications such as hypovolemic shock, arrhythmias, or cerebral edema.

Fluid Replacement and Electrolyte Management

DKA is characterized by significant fluid deficits, often requiring immediate intervention. The nursing team plays a crucial role in administering intravenous (IV) fluids to restore intravascular volume and prevent hypovolemic shock. Initial fluid therapy typically involves isotonic saline at a rate of 15–20 mL/kg of body weight per hour during the first hour. The rate and type of fluid are subsequently adjusted based on the patient's hydration status, electrolyte levels, and urinary output. Close attention is paid to serum potassium levels as insulin therapy can cause a rapid shift of potassium into the intracellular compartment, leading to hypokalemia. Potassium replacement should be initiated once levels drop below 5.3 mmol/L and continue as required to maintain normal serum concentrations.

Administration of Insulin Therapy

Insulin therapy is a cornerstone of DKA management and must be administered with precision to avoid complications such as hypoglycemia or hypokalemia. Nurses are responsible for initiating a continuous intravenous infusion of regular insulin at 0.1 units/kg per hour after an initial IV bolus. The infusion rate is adjusted based on the patient's glucose levels, with the aim of gradually lowering blood glucose to avoid osmotic shifts that could result in cerebral edema. Nurses must frequently reassess blood glucose levels and titrate the insulin dose to maintain a steady decline until target levels are achieved.

Management of Acid-Base Imbalances

The metabolic acidosis associated with DKA often necessitates correction. While bicarbonate therapy is not routinely indicated, it may be required in cases of severe acidosis with a pH below 6.9. Nurses are responsible for administering bicarbonate solutions in accordance with physician orders and monitoring the patient for any adverse effects, such as hypernatremia or hypocalcemia. Regular ABG assessments are essential to evaluate the progress of acid-base correction and the overall effectiveness of the intervention.

Preventing and Managing Complications

Nursing interventions also focus on preventing and managing complications that may arise during DKA treatment. One major complication is cerebral edema, particularly in pediatric and adolescent patients. Nurses must monitor for signs such as altered mental status, changes in pupil size, or abnormal respiratory patterns, and immediately alert the healthcare team if these symptoms occur. Additionally, the risk of hypoglycemia increases as blood glucose levels normalize. Frequent glucose monitoring and timely adjustments to the insulin infusion rate are crucial to mitigate this risk.

Supporting Nutritional Needs

Once the patient's condition stabilizes and they transition from IV to subcutaneous insulin, the nurse's role extends to supporting the patient's nutritional needs. Gradual reintroduction of oral intake is recommended, starting with easily digestible, carbohydrate-rich foods. Nurses should collaborate with dietitians to develop a meal plan that aligns with the patient's dietary preferences, glycemic control goals, and overall health status.

Psychological Support and Education

The psychological impact of a DKA episode can be profound, often leaving patients anxious or fearful about their condition. Nurses provide reassurance and emotional support, addressing the patient's concerns and encouraging open communication. Patient education is a vital component of the nursing care plan, aimed at preventing recurrent episodes of DKA. Education should focus on the importance of regular blood glucose monitoring, adherence to prescribed insulin regimens, recognizing early symptoms of DKA, and understanding the role of lifestyle modifications such as balanced nutrition and physical activity. Nurses should also provide guidance on how to manage blood glucose levels during periods of illness or stress, commonly referred to as "sick-day management."

Coordination with the Multidisciplinary Team

Nurses serve as key coordinators within the multidisciplinary healthcare team, ensuring that all aspects of the patient's care are seamlessly integrated. Collaboration with physicians, dietitians, and endocrinologists is essential to develop and implement an individualized care plan. Additionally, nurses play a critical role in communicating the patient's progress and any emerging concerns to the healthcare team, facilitating timely interventions.

Discharge Planning and Follow-Up

As the patient transitions from acute care to discharge, the nursing team focuses on discharge planning and follow-up care. This involves providing the patient with detailed instructions on insulin administration, glucose monitoring, and emergency procedures for managing hyperglycemic episodes. Scheduling follow-up appointments and ensuring the patient has access to necessary resources, such as glucose monitoring devices and medication, are crucial to maintaining glycemic control post-discharge. Nursing intervention plans for DKA are multifaceted and require a combination of technical expertise, continuous monitoring, and compassionate care. By addressing the physiological, psychological, and educational needs of patients, nurses play a pivotal role in achieving optimal outcomes and preventing future episodes of DKA. Effective collaboration within the healthcare team further enhances the quality and continuity of care provided to these patients.

Conclusion:

Diabetic ketoacidosis (DKA) continues to represent a critical challenge in diabetes care, despite advancements in therapeutic protocols and monitoring technologies. This updated review highlights the persistent burden of DKA as a leading cause of hospitalizations and diabetes-related mortalities, particularly among individuals with type 1 diabetes mellitus (T1DM). Key insights into its pathophysiology underscore the central role of insulin deficiency and the subsequent biochemical cascade involving lipolysis, ketogenesis, and acid-base derangements. These processes result in hyperglycemia, ketosis, and metabolic acidosis, the hallmark features of DKA. Effective management of DKA relies on a timely, multidisciplinary approach that includes fluid resuscitation, electrolyte correction, insulin therapy, and addressing precipitating factors such as infections or missed insulin doses. Advances in diagnostic criteria, such as the American Diabetes Association's classification of DKA severity, have improved the precision of disease recognition and management. Moreover, the use of advanced monitoring technologies has facilitated better control of critical parameters, reducing complications like cerebral edema and hypoglycemia. However, significant challenges remain, including the risk of fluid overload, euglycemic DKA, and complications arising from rapid glucose corrections. Patient-related barriers, such as non-adherence to insulin therapy and lack of access to healthcare resources, exacerbate the incidence and recurrence of DKA, particularly among vulnerable populations such as children, adolescents, and individuals in developing countries. Addressing these disparities requires an integrated approach that combines patient education, regular monitoring, and robust healthcare policies to ensure timely access to medical interventions. Future research should focus on identifying predictive biomarkers for early detection, refining therapeutic algorithms to minimize adverse effects, and exploring innovative treatments that target the underlying metabolic derangements. Collaborative efforts between healthcare providers, policymakers, and researchers are essential to mitigate the global burden of DKA, improve patient outcomes, and ultimately advance diabetes care. Through these measures, DKA management can transition from reactive intervention to proactive prevention.

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الحُماض الكيتوني السكري: من المضاعفات الشائعة لداء السكري ـ التشخيص، العلاج، والتدخلات ـ مراجعة محدثة

الملخص:
الخلفية:يُعتبر الحُماض الكيتوني السكري (DKA) من المضاعفات الخطيرة والمميتة المحتملة لداء السكري(DM) ، وينتج عن نقص الإنسولين ويتميز بفرط سكر الدم، وارتفاع مستويات الكيتونات في الدم، والحماض الأيضي. وعلى الرغم من التقدم في رعاية مرضى السكري، لا يزال الحُماض الكيتوني السكري أحد الأسباب الرئيسية لدخول المستشفيات والوفيات بين مرضى السكري.
الهدف:تهدف هذه المراجعة إلى جمع أحدث النتائج حول وبائيات الحُماض الكيتوني السكري، وأساسياته الفسيولوجية المرضية، وتشخيصه، وعلاجه، والتحديات المرتبطة بإدارته لتحسين النتائج السريرية. كما تركز على دور الصيادلة وأخصائي الباثولوجيا السريرية والتمريض في إدارة ومراقبة الحُماض الكيتوني السكري.
الطرق:تم إجراء بحث منهجي في الأدبيات باستخدام قاعدة بياناتPubMed ، استهدف المقالات المنشورة بين عامي 2012 و2023 في المجلات المُحكّمة. واشتملت استراتيجية البحث على استخدام عوامل منطقية لدمج مصطلحات مثل "الوبائيات"، و"الفسيولوجيا المرضية"، و"التشخيص"، و"علاج الحُماض الكيتوني السكري". وتم اختيار ست مقالات ذات صلة لتحليلها بشكل تفصيلي.
النتائج:يشكل الحُماض الكيتوني السكري حوالي 14% من حالات دخول المستشفيات و16% من الوفيات المرتبطة بالسكري. وهو أكثر شيوعًا بين مرضى السكري من النوع الأول، ولكنه يظهر أيضًا في حالات النوع الثاني. فسيولوجيًا، يؤدي نقص الإنسولين وزيادة الهرمونات المضادة للإنسولين والإجهاد التأكسدي إلى حدوث الكيتونية والحماض. يعتمد التشخيص على الأعراض السريرية والنتائج المخبرية مثل ارتفاع مستوى الجلوكوز في البلازما، ودرجة الحموضة الشريانية، الكيتونات في الدم، والفجوة الأيونية. يتضمن العلاج تصحيح اختلال السوائل والشوارد، وبدء العلاج بالإنسولين، وعلاج العوامل المسببة مثل العدوى. وعلى الرغم من التحسينات في استراتيجيات المراقبة والعلاج، لا تزال هناك تحديات مثل نقص السكر في الدم، وفرط السوائل، والحُماض الكيتوني السكري ذو السكر الطبيعي.
الخاتمة:يُعد الحُماض الكيتوني السكري حالة طارئة تتطلب تدخلاً سريعًا متعدد التخصصات. وعلى الرغم من التقدم في بروتوكولات العلاج، يمكن تحسين نتائج المرضى من خلال معالجة حواجز الامتثال، وتحسين أساليب التشخيص، والتقليل من مضاعفات العلاج. يجب أن تركز الأبحاث المستقبلية على استراتيجيات الكشف المبكر والتدخلات المستهدفة.
الكلمات المفتاحية:الحُماض الكيتوني السكري، نقص الإنسولين، فرط سكر الدم، الحماض الأيضي، استراتيجيات العلاج، المضاعفات.