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Adult Diabetic Ketoacidosis: An Overview, Diagnosis, Treatment, Emergency, And Nursing Interventions

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Abstract:

Background: Diabetic ketoacidosis (DKA) is a life-threatening complication commonly seen in individuals with diabetes, particularly those with type 1 diabetes mellitus (T1DM), although it also occurs in individuals with type 2 diabetes mellitus (T2DM). DKA is precipitated by factors such as infections, trauma, surgery, and non-compliance with insulin therapy. The condition is marked by hyperglycemia, metabolic acidosis, and ketonemia. Immediate management is crucial to prevent severe complications, including coma and death.

Aim: This article aims to provide an overview of DKA, including its etiology, pathophysiology, diagnostic approaches, treatment strategies, and nursing interventions.

Methods:
A comprehensive review of the existing literature on DKA was conducted, focusing on the epidemiology, pathophysiology, diagnosis, and treatment modalities. The article examines clinical management approaches, including insulin therapy, fluid resuscitation, and electrolyte correction, alongside the role of nursing interventions in the acute care setting.

Results: The incidence of DKA varies across regions, with a higher prevalence observed among individuals with T1DM and in urban populations with poor adherence to insulin therapy. Diagnostic criteria include elevated blood glucose levels, metabolic acidosis, and the presence of ketones. The management of DKA involves aggressive rehydration, insulin administration, and correction of electrolytes. Nursing interventions focus on monitoring vital signs, assessing for complications, and providing patient education to prevent recurrence.

Conclusion: DKA is a preventable but serious complication of diabetes that requires prompt recognition and treatment. Healthcare professionals, particularly nurses, play a critical role in managing the acute phase of DKA and in educating patients to prevent future episodes. Enhanced awareness, early intervention, and improved adherence to treatment can significantly reduce morbidity and mortality associated with DKA.

Keywords: Diabetic ketoacidosis, type 1 diabetes, type 2 diabetes, insulin therapy, metabolic acidosis, nursing interventions, management, complications.

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Introduction:

Diabetic ketoacidosis (DKA) represents a severe, potentially life-threatening complication of diabetes, characterized by a combination of hyperglycemia, acidosis, and ketonemia. It primarily affects individuals with type 1 diabetes mellitus (T1DM), but it is also observed in patients with type 2 diabetes mellitus (T2DM), albeit less frequently. Typically, DKA is precipitated by a range of factors such as new-onset diabetes, infections, and non-compliance with prescribed therapeutic regimens. The condition is marked

by a progressive metabolic derangement that includes elevated blood glucose levels, acidosis, and the production of ketone bodies due to a shortage of insulin. This results in increased lipolysis and ketogenesis, which further exacerbates the clinical scenario. While DKA can occur at any age, it is particularly common in younger individuals with T1DM and is often linked to episodes of acute illness or stress. Immediate intervention is critical to prevent severe complications, including coma and death, and to mitigate the risk of future episodes. This review seeks to provide a comprehensive overview of the etiology, epidemiology, and management of DKA, while examining pharmacological therapies, diagnostic strategies, and the ongoing developments in its treatment.

Etiology:

Diabetic ketoacidosis predominantly affects individuals with type 1 diabetes, although individuals with type 2 diabetes are also susceptible. In both populations, acute medical conditions, such as infections, trauma, or surgery, often serve as precipitants of DKA. Non-compliance with insulin therapy remains one of the most common triggers, especially in patients with established diabetes. The infections most commonly associated with DKA include pneumonia and urinary tract infections. However, other conditions such as alcohol abuse, myocardial infarction, pulmonary embolism, and trauma can also contribute to the onset of DKA. Medications that influence carbohydrate metabolism, such as corticosteroids, thiazide diuretics, sympathomimetic agents, and pentamidine, have been identified as potential precipitants of DKA. Additionally, certain antipsychotic medications, both conventional and atypical, may induce hyperglycemia and, in rare cases, precipitate DKA [1].

Sodium-glucose cotransporter 2 (SGLT2) inhibitors have been recognized as a contributing factor in the development of DKA, especially when used in conjunction with insulin. The use of SGLT2 inhibitors often leads to a reduction in insulin dosages to prevent hypoglycemia, which may, however, be insufficient to inhibit lipolysis and ketogenesis, promoting ketone body production. Moreover, SGLT2 inhibitors stimulate glucagon secretion and potentially reduce the urinary excretion of ketones, resulting in elevated plasma ketone levels and hyperglycemia, both of which are conducive to the onset of DKA. Interestingly, a small subset of patients may develop euglycemic DKA, characterized by a high anion gap metabolic acidosis with positive serum and urine ketones despite serum glucose levels being less than 250 mg/dL. The use of SGLT2 inhibitors has been implicated in the induction of this rare form of DKA [2][3]. Furthermore, non-compliance with insulin therapy remains a major cause of recurrent DKA, particularly within the inner-city populations in the United States. Socioeconomic and educational factors significantly contribute to poor adherence to diabetes management, including insulin therapy. Cocaine abuse has been identified as an independent risk factor for the recurrence of DKA, highlighting the complex interplay of substance use and non-compliance in exacerbating diabetic crises [4].

Epidemiology

The incidence of diabetic ketoacidosis (DKA) varies significantly, ranging from 0 to 56 cases per 1000 person-years, as reported across various geographic regions. DKA is more prevalent among women and non-White populations. Individuals using injectable insulin appear to have a higher incidence of DKA compared to those using subcutaneous insulin infusion pumps [5]. The prevalence of DKA in children also shows considerable geographic variation, with the lowest incidence found in Nigeria (2.9 cases per 100,000 population), while Sweden and Finland report much higher rates of 41.0 and 37.4 cases per 100,000 population, respectively [6]. In the United States, nursing home residents accounted for 0.7% of DKA cases, and increased mortality was observed in this group of patients. Mortality rates exceeding 5% have been reported among the elderly and those with concurrent life-threatening conditions. However, death in these cases is rarely attributed solely to the metabolic complications of hyperglycemia or ketoacidosis [7].

The prognosis for individuals with DKA worsens significantly when they are at the extremes of age or when comorbidities such as coma and hypotension are present. Among urban Black populations, poor adherence to insulin therapy remains the leading precipitating factor for DKA. Substance abuse is a critical contributor to non-compliance, and obesity, which is prevalent in individuals with newly diagnosed diabetes, is commonly observed in these patients. Enhanced patient education and improved access to healthcare are

essential strategies to reduce the incidence of these hyperglycemic emergencies [8]. Diabetic ketoacidosis is a preventable, yet life-threatening, complication of diabetes. Data from the Centers for Disease Control and Prevention (CDC) United States Diabetes Surveillance System (USDSS) indicate an upward trend in hospitalization rates for DKA from 2009 to 2014, particularly among individuals aged less than 45 years. However, the overall mortality associated with hyperglycemic crises among adults with diabetes has shown a decline. Despite this, there is still significant room for improvement, particularly in reducing mortality rates among Black men and preventing fatalities in non-hospital settings [9][10]. The geriatric population is particularly vulnerable to developing hyperglycemic crises, including DKA, due to factors such as increased insulin resistance and a diminished thirst response. Older adults are more prone to both hyperglycemia and dehydration, two critical elements that exacerbate hyperglycemic emergencies. With increased surveillance of diabetes and prompt, aggressive treatment of hyperglycemia and its complications, morbidity and mortality from acute diabetic crises in the elderly can be significantly reduced [11].

Pathophysiology

Diabetes mellitus is distinguished by a deficiency in insulin and elevated plasma glucagon levels, both of which can be normalized through insulin therapy [12]. Under normal circumstances, an increase in serum glucose concentration triggers its entry into pancreatic beta cells, stimulating the production of insulin. Insulin then reduces hepatic glucose output by inhibiting both glycogenolysis and gluconeogenesis. Additionally, insulin promotes glucose uptake by skeletal muscle and adipose tissue, leading to a reduction in blood glucose levels. In diabetic ketoacidosis (DKA), however, insulin deficiency, coupled with elevated counter-regulatory hormones, can result in augmented gluconeogenesis, accelerated glycogenolysis, and hindered glucose utilization, thereby exacerbating hyperglycemia. Furthermore, insulin deficiency and elevated counter-regulatory hormones induce the release of free fatty acids from adipose tissue (lipolysis), which are subsequently metabolized in the liver into ketone bodies, such as beta-hydroxybutyrate and acetoacetate, leading to ketonemia and metabolic acidosis [1]. Although glucagon is not essential for the development of ketoacidosis in diabetes mellitus, it may contribute to the acceleration of ketonemia and hyperglycemia during episodes of insulin deficiency [13]. Additionally, patients undergoing treatment with SGLT2 inhibitors face an increased risk of developing euglycemic DKA.

Hyperglycemia-induced diuresis, dehydration, hyperosmolarity, and electrolyte imbalances result in a decline in glomerular filtration. The deterioration of renal function due to these factors further exacerbates hyperglycemia and hyperosmolarity. Potassium utilization by skeletal muscle is also compromised due to hyperosmolarity and impaired insulin action, leading to intracellular potassium depletion. Osmotic diuresis further exacerbates potassium loss, resulting in reduced total body potassium. It is important to note that the potassium levels observed in patients with DKA may not reflect their total body potassium status, as a normal plasma potassium level may still indicate significant depletion of body potassium [4]. Hyperosmolarity is believed to be the primary factor contributing to altered consciousness in DKA patients [14]. Recent studies have indicated that hyperglycemia triggers a significant inflammatory response, characterized by an elevation in proinflammatory cytokines (including tumor necrosis factor-alpha, interleukin-1 beta, interleukin-6, and interleukin-8), C-reactive protein, lipid peroxidation, reactive oxygen species, cardiovascular risk factors, plasminogen activator inhibitor-1, and free fatty acids, even in the absence of infection or cardiovascular pathology. After administering insulin therapy and intravenous fluid hydration, these proinflammatory markers generally return to baseline levels within 24 hours [1].

History and Physical

The clinical presentation of diabetic ketoacidosis (DKA) may encompass a range of symptoms and physical findings. Patients typically experience signs of hyperglycemia, such as polyphagia, polyuria, or polydipsia. As volume depletion progresses, symptoms of dehydration, including decreased urine output, dry mouth, and reduced sweating, may become apparent. Additionally, patients may report anorexia, nausea, vomiting, abdominal pain, and weight loss. In cases where an infection serves as a precipitating factor for DKA, the patient may present with symptoms indicative of infection, such as fever, cough, or urinary complaints. If

cerebral edema is developing, patients may exhibit headache or confusion. A comprehensive medication history should be taken to determine any prescribed treatments and their proper usage. Furthermore, the use of substances such as drugs or alcohol should be assessed [15]. Upon physical examination, vital signs commonly reveal tachycardia and tachypnea. If an infectious trigger for DKA is suspected, the patient may present with either fever or hypothermia. Blood pressure may also fluctuate, with hypotension often indicating a more severe disease state. Patients generally appear ill, and Kussmaul breathing, characterized by labored, deep, and rapid breaths, may be observed. A fruity odor, indicative of acetone, may be detectable on the patient's breath. Signs of dehydration, including poor capillary refill, diminished skin turgor, and dry mucous membranes, may be evident. Abdominal tenderness may also be present. In severe cases, altered mental status, general drowsiness, and focal neurologic deficits may suggest cerebral edema, which requires prompt treatment [16].

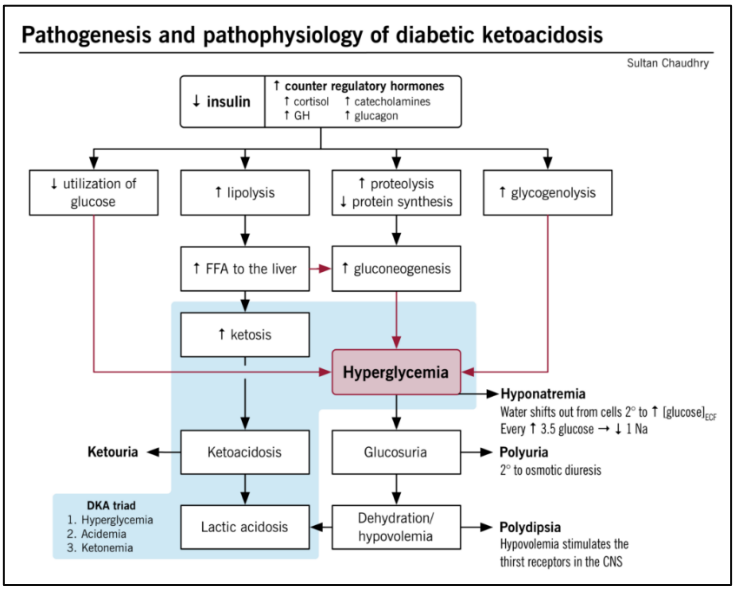

Figure 1: Pathogenesis and Pathophysiology of DKA.

Evaluation

The diagnostic criteria for diabetic ketoacidosis typically include a blood glucose level exceeding 250 mg/dL, arterial pH below 7.3, serum bicarbonate concentration below 15 mEq/L, and the presence of ketonemia or ketonuria. A normal anion gap is approximately 12 mEq/L, and an anion gap greater than 14-15 mEq/L is indicative of an elevated anion gap metabolic acidosis [17]. In some instances, arterial pH may be normal or even elevated if coexisting metabolic or respiratory alkalosis is present, as may occur with vomiting or diuretic use [18]. Blood glucose levels may be within normal or mildly elevated ranges (<300 mg/dL) in DKA patients who are at risk for hypoglycemia, such as those with alcohol use disorder or those receiving insulin or SGLT2 inhibitors. Leukocytosis is commonly observed in DKA patients upon hospital presentation. The serum sodium concentration may appear falsely low in DKA, a discrepancy that can be corrected by adding 1.6 mEq/L to the measured sodium level for every 100 mg/dL increase in glucose above 100 mg/dL. Serum potassium levels are typically elevated due to the shift of potassium from the intracellular to the extracellular space caused by acidosis and insulin deficiency. However, total body potassium may be depleted or become depleted rapidly with insulin treatment. Magnesium levels are frequently low and require supplementation. Serum phosphate may be elevated despite total body phosphate depletion [19]. Additional diagnostic tests, including urine, sputum, and blood cultures, as well as serum lipase measurements and chest radiographs, may be required depending on the case. Pneumonia and urinary tract infections are the most common infections that precipitate DKA. Measuring glycated hemoglobin (A1C) provides useful information on long-term glucose trends.

In acute DKA, the ketone body ratio of 3-beta-hydroxybutyrate to acetoacetate typically increases from a normal ratio of 1:1 to as high as 10:1. In response to insulin therapy, 3-beta-hydroxybutyrate (3-HB) levels

typically decline before acetoacetate (AcAc) levels. The nitroprusside test, often used for detecting ketones, only detects acetoacetate in blood and urine and provides a semiquantitative measure, which may yield false positives. Recently, inexpensive quantitative tests for 3-HB levels have become available, offering better options for monitoring and managing conditions characterized by abnormal ketone metabolism [20]. Serum levels of pancreatic enzymes may be elevated in DKA due to disruptions in carbohydrate metabolism [21]. In DKA patients presenting with abdominal pain and elevated pancreatic enzymes, a diagnosis of acute pancreatitis should not be made immediately [22]. If diagnostic uncertainty persists, imaging, such as a CT scan, may help differentiate between mild to moderate enzyme elevations from acute pancreatitis. Lipid abnormalities are frequently observed in DKA patients. In one study, mean plasma triglyceride and cholesterol levels before insulin therapy were 574 mg/dL (range 53-2355) and 212 mg/dL (range 118-416), respectively. Following insulin treatment, plasma triglyceride levels rapidly decreased to below 150 mg/dL within 24 hours. Plasma apoprotein B levels were initially within the normal upper range (101 mg/dL) and decreased with therapy due to significant reductions in VLDL, but not in IDL or LDL apoprotein B [23]. An electrocardiogram (ECG) is valuable for detecting ischemic changes or signs of potassium imbalances, such as hyperkalemia or hypokalemia. Peaked T waves indicate hyperkalemia, while low T waves with U waves are indicative of hypokalemia. For imaging, a chest X-ray may be necessary to exclude consolidation. Magnetic resonance imaging (MRI), and to a lesser extent, CT imaging of the head, can identify cerebral edema; however, imaging should not delay treatment if cerebral edema is suspected.

Treatment / Management

Fluid resuscitation, insulin therapy, electrolyte replacement, and supportive care constitute the principal components in the management of diabetic ketoacidosis (DKA).

Hydration

In patients diagnosed with DKA, fluid loss can amount to 10-15% of body weight [1]. Immediate fluid resuscitation is critical for correcting hypovolemia, restoring tissue perfusion, and facilitating the elimination of ketones. Hydration plays a pivotal role in improving glycemic control, independent of insulin administration.

Choice of Fluids

Isotonic fluids have been established as the preferred fluid for over 50 years. A 2013 meta-analysis comparing colloids and crystalloids in critically ill patients concluded that crystalloid fluids were non-inferior to colloids [24]. Traditionally, 0.9% normal saline has been used in DKA management. Although concerns about normal saline contributing to hyperchloremia and hyperchloremic metabolic acidosis exist, particularly when used in large volumes, studies comparing normal saline with other solutions like Ringer's lactate have shown no significant differences in clinical outcomes [25][26][27]. Therefore, normal saline remains the fluid of choice for initial hydration.

Infusion Rate

Initial:

The recommended infusion rate is typically 15-20 ml per kilogram of body weight during the first hour. A study comparing aggressive hydration (1 liter per hour for 4 hours) to slower infusion rates found that the latter was equally effective [28]. However, in critically ill patients, including those with hypotension, aggressive fluid therapy is preferred. The risk of cerebral edema associated with early aggressive volume resuscitation remains a point of debate. Studies have indicated an increased incidence of cerebral edema in pediatric populations with aggressive fluid therapy, though other studies report no difference in outcomes, suggesting that patients at highest risk for cerebral edema typically present later and exhibit more severe volume depletion [29].

Maintenance:

The subsequent fluid replacement strategy is determined by hemodynamics, hydration status, serum electrolyte concentrations, and urinary output [1]. For patients with elevated serum sodium levels, the infusion of 0.45% NaCl at 4-14 ml/kg/hour or 250-500 mL/hour is appropriate. For those with hyponatremia, 0.9% NaCl at the same infusion rate is preferred [30]. If hyperchloremic metabolic acidosis becomes a concern, the maintenance fluid can be switched to Ringer's lactate.

Insulin Therapy

The advent of insulin, alongside antibiotics, has significantly reduced the mortality associated with DKA, which now stands at approximately 1%. Intravenous insulin infusion remains the standard treatment. Traditional protocols recommended an initial insulin bolus of 0.1 U/kg, followed by continuous infusion at 0.1 U/kg/h. However, a recent randomized prospective trial demonstrated that an initial bolus may be unnecessary when patients receive hourly insulin infusion at 0.14 U/kg/h [31]. Upon plasma glucose levels reaching 200-250 mg/dl, with an ongoing anion gap, the introduction of dextrose-containing fluids is necessary, and the insulin infusion rate may need adjustment. In adult patients with uncomplicated and mild DKA, subcutaneous insulin lispro administered hourly in a non-intensive care setting has proven to be safe and cost-effective, as opposed to intravenous regular insulin in an intensive care setting, as shown in numerous studies [32]. One such study involved patients receiving subcutaneous insulin lispro at an initial dose of 0.3 U/kg, followed by 0.1 U/kg every hour until blood glucose fell below 250 mg/dl. The insulin dose was then reduced to 0.05 or 0.1 U/kg every hour until DKA resolution [32]. Similarly, insulin aspart has been shown to have similar efficacy [33]. Insulin administration should continue until the resolution of DKA. The criteria for resolution include blood glucose below 200 mg/dl and two of the following: a serum bicarbonate level ≥15 mEq/l, a venous pH >7.3, or a calculated anion gap ≤12 mEq/l. Once DKA is resolved and the patient can tolerate oral intake, transition to subcutaneous insulin is feasible. Patients who had previously been on insulin may resume their home dosage if their condition was well-controlled. For insulin-naive patients, a multi-dose insulin regimen starting at 0.5 to 0.8 U/kg/day should be administered. To prevent DKA recurrence during the transition period, intravenous insulin should continue for 2 hours after the initiation of subcutaneous insulin, and blood glucose and metabolic profiles should be reassessed before discontinuing the insulin drip. In cases where the patient cannot tolerate oral intake, intravenous insulin and fluids may be continued. The use of long-acting insulin analogs may also facilitate the transition from intravenous to subcutaneous insulin therapy [34].

Electrolyte Replacement

Potassium

Initial potassium levels in DKA patients are often elevated (mild to moderate hyperkalemia), despite a total body potassium deficit. The introduction of insulin causes a shift of potassium intracellularly, potentially leading to severe hypokalemia [35][36]. Thus, patients with serum potassium levels below 3.3 mmol/L should first undergo fluid resuscitation and potassium replacement before starting insulin treatment to prevent the risk of cardiac arrhythmias, respiratory muscle weakness, and cardiac arrest. For other patients, potassium replacement is required when serum concentrations fall below 5.2 mEq/L, aiming for a level between 4 and 5 mEq/L. A typical dose of 20-30 mEq of potassium per liter of fluids is sufficient for most patients, though lower doses are necessary for those with renal failure, either acute or chronic [37].

Magnesium

Hypokalemia often coexists with hypomagnesemia. Replenishing both potassium and magnesium may be necessary, as it can be challenging to correct potassium levels without addressing magnesium deficiency.

Bicarbonate

The efficacy of bicarbonate replacement in DKA has been questioned. One study found no significant difference in the time to resolution of acidosis (8 hours vs. 8 hours, p = 0.7) or time to hospital discharge (68 hours vs. 61 hours, p = 0.3) between patients who received intravenous bicarbonate and those who did

not [38]. In a pediatric study, children with DKA and low PaCO2 and high BUN concentrations at presentation were at higher risk for cerebral edema if treated with bicarbonate [39]. Potential pitfalls of bicarbonate therapy include paradoxical cerebrospinal fluid acidosis, hypokalemia, large sodium bolus, and cerebral edema. Nevertheless, bicarbonate may be considered in patients with severe acidemia. The most recent guidelines from the American Diabetes Association (ADA) recommend bicarbonate therapy for patients with a pH <7.1 [38].

Phosphate

Phosphate replacement in DKA has been investigated, though studies have not shown any substantial impact on the duration of DKA, the insulin dosage needed to correct acidosis, glucose disappearance, or morbidity and mortality. Despite its theoretical appeal, phosphate therapy is generally not essential in treating DKA, except in rare cases, such as severe hypophosphatemia (e.g., 1.0 mg/dl) leading to seizure [40].

Laboratory Monitoring

Hourly point-of-care testing (POCT) of glucose levels is essential. Serum glucose and electrolyte levels should be monitored every 2 hours until the patient stabilizes, after which monitoring can occur every 4 hours. Initial blood urea nitrogen (BUN), venous blood gas (VBG), or arterial blood gas (ABG) monitoring should also be conducted, with additional tests based on precipitating events.

Intubation

Intubation carries multiple risks in DKA patients and should be avoided unless absolutely necessary. Fluid and insulin administration typically result in the improvement of acidosis and overall clinical status. Patients compensate for severe acidosis with compensatory respiratory alkalosis, manifested by tachypnea and Kussmaul breathing. If patients can no longer generate respiratory alkalosis due to comatose states or severe fatigue, intubation may be warranted. However, intubation increases the risk of elevated PaCO2 during sedation or paralysis, exacerbating acidosis and increasing the risk of aspiration from gastroparesis. Furthermore, intubation makes it challenging to match respiratory compensation with metabolic acidosis. When intubation is necessary, it is crucial to adjust minute ventilation to create respiratory alkalosis, thereby compensating for the metabolic acidosis of DKA. Without proper ventilation, acidosis may worsen, potentially leading to cardiac arrest. Ventilator settings should start with a tidal volume of 8 ml/kg based on ideal body weight and a respiratory rate that mimics the patient's compensating respiratory rate, with careful attention to avoid auto-positive end-expiratory pressure (PEEP) from rapid respiratory rates [41].

Cerebral Edema

Monitoring mental status and neurologic function is essential in all DKA patients. For patients who are severely obtunded, comatose, or exhibit a decline in mental status despite treatment, or present with focal neurologic deficits, cerebral edema should be suspected. Mannitol is typically the first-line agent used in these cases, although there are studies examining the use of 3% saline in both traumatic brain injury (TBI) and DKA literature.

Precipitating Events

Infection is a common trigger for DKA in patients with new-onset or established diabetes. If an infection is suspected, prompt administration of antibiotics is necessary. Other events that may trigger DKA should also be addressed in conjunction with the treatment of the underlying causes.

Differential Diagnosis

Diabetic ketoacidosis (DKA) has a wide-ranging presentation, and its symptoms can overlap with those of other common conditions. It is essential for healthcare providers to consider several potential differential diagnoses when DKA is suspected. These include hyperosmolar hyperglycemic nonketotic syndrome, starvation ketosis, myocardial infarction, pancreatitis, alcoholic ketoacidosis, lactic acidosis, sepsis,

toxicological exposures such as ethylene glycol, methanol, paraldehyde, and salicylates, overdose of diabetic medications, and uremia.

Prognosis

DKA still has a mortality rate between 0.2% and 2.5% in developing countries. Patients who are present with comatose states, hypothermia, and oliguria are at higher risk for adverse outcomes. For the majority of patients who receive timely intervention, the prognosis is positive, especially if the underlying trigger is not an infection. However, elderly individuals with concurrent conditions, such as myocardial infarction, pneumonia, or sepsis, tend to experience extended hospital stays and have a higher risk of mortality. The leading cause of death in young patients is cerebral edema, which occurs due to intracellular fluid shifts. Renal dysfunction is another contributing factor to morbidity in DKA. Recent studies have shown that patients with type-2 diabetes who experience DKA are more likely to suffer from a stroke within the first six months following the event.

Complications

Hypoglycemia is a frequent complication during the treatment of diabetic ketoacidosis, occurring in approximately 5% to 25% of patients with DKA. Acute outcomes of hypoglycemia, such as seizures, arrhythmias, and cardiovascular events, require close monitoring of blood glucose levels during the acute treatment phase. Another common issue is hypokalemia, which can lead to muscle weakness, cardiac arrhythmias, and even cardiac arrest in severe cases. Additional electrolyte disturbances that may occur include hyperchloremia, which affects up to one-third of patients, as well as hypomagnesemia and hyponatremia. Cerebral edema, although less common in adults, remains a significant risk, with contributing factors including younger age, new-onset diabetes, prolonged symptoms, lower carbon dioxide levels, severe acidosis, and low initial bicarbonate, sodium, and high glucose levels at presentation. Rapid hydration and fluid retention can also increase the risk. Rhabdomyolysis, more frequently seen in hyperosmolar hyperglycemic state (HHS), may also occur in DKA, potentially leading to acute kidney failure. Severe hypophosphatemia, related to DKA, can exacerbate this condition. Acute respiratory failure may also develop, stemming from pneumonia, acute respiratory distress syndrome (ARDS), or pulmonary edema. There are two recognized types of pulmonary edema associated with DKA: one linked to elevated pulmonary venous pressure and the other resulting from increased pulmonary capillary permeability. Other complications, such as thrombotic thrombocytopenic purpura (TTP) and myocarditis, have also been reported in DKA patients.

Patient Education

It is crucial to provide patients with education about diabetes, focusing on both short- and long-term complications. Patients should be instructed on how and when to monitor their blood glucose levels and the correct use of oral hypoglycemic agents or insulin, including their potential side effects and the importance of adherence to prescribed treatment regimens. Involvement from dietitians, nurses, and multidisciplinary home health teams plays an important role in facilitating this education, ensuring that patients understand their condition and manage it effectively.

Enhancing Healthcare Team Outcomes

Diabetic ketoacidosis is a life-threatening complication of diabetes, and any delay in treatment can result in fatal outcomes. The condition can manifest with a variety of symptoms that affect multiple organ systems, underscoring the need for management by an interdisciplinary team focused on the care of diabetes mellitus patients. Most individuals initially present to the emergency department, where treatment typically begins. The triage nurse must be well-versed in recognizing the signs and symptoms of DKA and must immediately admit the patient, notifying the emergency department physician. During resuscitation, as the patient is monitored and blood is drawn, consultation with intensivists and endocrinologists is essential. Prompt blood work is necessary to assess the severity of ketoacidosis, and imaging may be needed to rule out pneumonia. If the patient's mental status is altered, a CT scan should be performed, and the radiologist must be informed of the patient's hemodynamic status. No DKA patient should be sent

unmonitored to the radiology department. Infectious disease specialists and cardiologists should be consulted if there is suspicion that infection or myocardial infarction is the triggering factor. Additionally, pharmacists and nurses should verify whether the patient has adhered to their insulin regimen. Social workers should be involved in post-discharge care, particularly because recurrent DKA admissions are common in inner-city hospitals, often influenced by socioeconomic factors, education, access to insulin, healthcare coverage, and mental health issues. An interdisciplinary approach, including the involvement of social workers, is often necessary to address these unique challenges. Detailed discharge planning, involving social workers for patients with social needs and hospital-initiated follow-up clinics, is vital for reducing recurrent DKA episodes. Patient education is also crucial, as non-compliance with treatment is frequently the underlying cause of DKA episodes.

Outcomes

In developed nations, the morbidity and mortality associated with DKA are relatively low due to the coordinated efforts of interdisciplinary teams in managing the condition. However, in developing countries, mortality rates of 0.3% to 2.5% continue to be reported. The leading cause of death among younger patients remains cerebral edema, underscoring the importance of early intervention and comprehensive care.

Nursing and Emergency Interventions:

Nursing interventions in the management of Diabetic Ketoacidosis (DKA) are critical in stabilizing patients and preventing further complications. DKA is a severe, life-threatening condition that occurs primarily in individuals with diabetes mellitus, typically resulting from insulin deficiency and the inability of cells to access glucose for energy. This leads to hyperglycemia, ketosis, and acidosis, which can rapidly progress to more serious complications without prompt intervention. Nurses are often the first line of defense in the early recognition and management of DKA, and their interventions are essential for ensuring patient safety and improving outcomes. The first and most critical step in nursing care for patients with DKA is the rapid assessment of the patient's condition using a systematic approach. Nurses should assess vital signs, including blood pressure, heart rate, respiratory rate, and oxygen saturation, while also performing a thorough physical examination to identify signs of dehydration, altered mental status, and other symptoms associated with DKA. Nurses should prioritize the management of airway, breathing, and circulation (ABCs), following the established guidelines for emergency care. In particular, the assessment of fluid and electrolyte imbalances is crucial, as patients with DKA often experience significant dehydration and disturbances in electrolytes, particularly potassium, sodium, and chloride.

Once the initial assessment is completed, nursing interventions should focus on the correction of hyperglycemia and the restoration of fluid balance. Intravenous (IV) fluids, usually starting with isotonic saline, are administered to rehydrate the patient and dilute excess glucose in the blood. Nurses must monitor fluid input and output meticulously, adjusting the infusion rate as needed based on the patient's response. Additionally, insulin therapy is initiated to lower blood glucose levels. Insulin administration should be carefully managed, typically beginning with an IV bolus followed by a continuous infusion. Nurses need to frequently monitor blood glucose levels and adjust insulin doses, accordingly, ensuring that the reduction in glucose is gradual to avoid complications such as cerebral edema. Electrolyte imbalances are common in DKA and require prompt attention. Potassium levels, in particular, must be closely monitored since insulin therapy can cause potassium to shift into cells, potentially leading to hypokalemia. Nurses must assess potassium levels regularly and administer potassium replacements as needed, ensuring that these interventions are done cautiously to avoid further complications. In cases where acidosis is severe, sodium bicarbonate may be used to correct the pH imbalance, but this decision should be made with careful monitoring and in consultation with a healthcare provider.

As part of comprehensive care, nursing interventions also include providing patient education and psychological support. Nurses play a vital role in educating patients about the importance of diabetes management, including consistent insulin use, blood glucose monitoring, and recognizing early signs of DKA. This education is crucial in preventing future episodes of DKA and improving long-term diabetes management. Nurses should also address any emotional or psychological needs of the patient, offering

reassurance and guidance throughout the treatment process, as DKA can be a distressing experience for both patients and their families. In summary, nursing interventions in the management of DKA are essential for stabilizing the patient and preventing complications. Prompt and accurate assessments, fluid and electrolyte management, insulin administration, and patient education all play a pivotal role in improving outcomes. The ability of nurses to act quickly and effectively in emergency situations can significantly reduce mortality and morbidity in patients with DKA. Effective teamwork and collaboration with other healthcare professionals, including physicians, endocrinologists, and pharmacists, are also critical in ensuring optimal care [45].

Conclusion:

Diabetic ketoacidosis (DKA) is a serious, often life-threatening complication of diabetes that requires immediate medical intervention. The primary factors contributing to the development of DKA include insulin deficiency, infection, non-compliance with insulin therapy, and acute illnesses such as trauma or surgery. Although the condition is most commonly observed in individuals with type 1 diabetes mellitus (T1DM), it can also affect individuals with type 2 diabetes mellitus (T2DM), particularly under certain conditions like infections or the use of medications such as SGLT2 inhibitors. The pathophysiology of DKA involves a combination of hyperglycemia, acidosis, and ketonemia, triggered by a shortage of insulin and an increase in counter-regulatory hormones like glucagon. This metabolic imbalance leads to increased lipolysis, ketogenesis, and the production of ketone bodies, resulting in metabolic acidosis. Complications such as dehydration, hyperosmolarity, and electrolyte imbalances further contribute to the severity of the condition, increasing the risk of complications like cerebral edema and cardiovascular instability. Effective diagnosis is based on clinical criteria, including elevated blood glucose levels, anion gap metabolic acidosis, and the presence of ketones in the blood or urine. Once diagnosed, DKA is managed through a combination of fluid resuscitation, insulin therapy, and correction of electrolyte imbalances, particularly potassium. Close monitoring of vital signs, urine output, and laboratory values is essential during the acute phase to prevent complications. Nursing interventions play a key role in ensuring that patients receive timely care, manage their fluid and electrolyte balance, and are educated on the importance of consistent insulin therapy to prevent future episodes. The epidemiology of DKA indicates a higher incidence in specific populations, particularly among younger individuals with T1DM and those with poor adherence to treatment. Studies show that socioeconomic and educational factors, including substance abuse, contribute to the non-compliance seen in at-risk populations. Therefore, addressing these factors through patient education and improving access to healthcare can reduce the incidence of DKA. Moreover, the increasing use of SGLT2 inhibitors in diabetic patients requires attention due to their potential role in inducing euglycemic DKA, a rare form of the condition characterized by normal or low blood glucose levels despite severe acidosis. In conclusion, DKA remains a preventable yet critical complication that requires timely intervention, effective treatment protocols, and patient education. Healthcare professionals, especially nurses, have a vital role in managing DKA and ensuring that individuals with diabetes are adequately educated on managing their condition to prevent recurrent episodes. While advances in medical care have improved outcomes, there is still significant work to be done in reducing the incidence of DKA through better disease management and prevention strategies.

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الحُماض الكيتوني السكري لدى البالغين: نظرة عامة، التشخيص، العلاج، التدخلات الطارئة، والتدخلات التمريضية

الملخص:

الخلفية :الحُماض الكيتوني السكري (DKA) هو مضاعف مهدد للحياة يُلاحظ بشكل شائع لدى الأفراد المصابين بداء السكري، خصوصًا أولئك المصابين بداء السكري من النوع 1(T1DM) ، رغم أنه يحدث أيضًا لدى الأشخاص المصابين بداء السكري من النوع 2 .(T2DM) يُحفّز الحُماض الكيتوني السكري بواسطة عوامل مثل العدوى، الصدمات، الجراحة، وعدم الالتزام بالعلاج بالأنسولين. تتميز هذه الحالة بارتفاع سكر الدم، الحُماض الأيضي، ووجود الكيتونات في الدم. إن الإدارة الفورية أمر بالغ الأهمية لمنع حدوث مضاعفات شديدة، مثل الغيبوبة والموت.

الهدف :يهدف هذا المقال إلى تقديم نظرة عامة حول الحُماض الكيتوني السكري، بما في ذلك أسبابه، الفيزيولوجيا المرضية، أساليب التشخيص، استراتيجيات العلاج، والتدخلات التمريضية.

الطرق :تم إجراء مراجعة شاملة للأدبيات الحالية المتعلقة بالحُماض الكيتوني السكري، مع التركيز على الوبائيات، الفيزيولوجيا المرضية، التشخيص، وأساليب العلاج. يناقش المقال أساليب الإدارة السريرية، بما في ذلك العلاج بالأنسولين، الإنعاش السوائي، وتصحيح الإلكتروليتات، بالإضافة إلى دور التدخلات التمريضية في البيئة الطارئة.

النتائج :تختلف معدلات الإصابة بالحُماض الكيتوني السكري حسب المناطق، حيث يُلاحظ انتشار أعلى بين الأفراد المصابين بداء السكري من النوع 1 وفي السكان الحضريين الذين يعانون من قلة الالتزام بالعلاج بالأنسولين. تشمل معايير التشخيص ارتفاع مستويات الجلوكوز في الدم، الحُماض الأيضي، ووجود الكيتونات. يشمل علاج الحُماض الكيتوني السكري إعادة الترطيب بشكل مكثف، إعطاء الأنسولين، وتصحيح مستويات الإلكتروليتات. تركز التدخلات التمريضية على مراقبة العلامات الحيوية، تقييم المضاعفات، وتقديم التثقيف الصحي للمريض للوقاية من التكرار.

الخلاصة :الحُماض الكيتوني السكري هو مضاعف قابل للوقاية، ولكنه خطير ويتطلب التعرف المبكر والعلاج الفوري. يلعب المتخصصون في الرعاية الصحية، وخاصة الممرضات، دورًا حاسمًا في إدارة المرحلة الحادة للحُماض الكيتوني السكري وفي تثقيف المرضى للوقاية من الحلقات المستقبلية. يمكن أن يقلل الوعي المحسن، والتدخل المبكر، والالتزام الأفضل بالعلاج بشكل كبير من معدلات المرض والوفاة المرتبطة بالحُماض الكيتوني السكري.

الكلمات المفتاحية :الحُماض الكيتوني السكري، داء السكري من النوع 1، داء السكري من النوع 2، العلاج بالأنسولين، الحُماض الأيضي، التدخلات التمريضية، الإدارة، المضاعفات.