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| RESEARCH ARTICLE

Impact of Early Emergency Department Management of Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State on ICU Admission and In-Hospital Mortality: A Retrospective Cohort Study

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| ABSTRACT

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are life-threatening endocrine emergencies frequently encountered in emergency departments (EDs). Delayed recognition and treatment are associated with increased intensive care unit (ICU) admission and mortality [1,2]. To evaluate the impact of early recognition and ED-based management of DKA and HHS on ICU admission and in-hospital mortality. A retrospective cohort study was conducted among adult patients presenting to the ED with confirmed DKA or HHS. Data included time to diagnosis, time to initiation of insulin and intravenous fluids, electrolyte abnormalities, ICU admission, and in-hospital mortality. Early management was defined as diagnosis within ≤60 minutes and initiation of insulin and IV fluids within ≤90 minutes of ED arrival. Logistic regression analysis was used to identify independent predictors of ICU admission and mortality. Among 100 patients, early management was associated with significantly lower ICU admission (24.1% vs. 47.6%, p = 0.01) and mortality (5.2% vs. 14.3%, p = 0.04). Delayed management independently predicted ICU admission (OR 2.82, 95% CI 1.22–6.51) and in-hospital mortality (OR 3.12, 95% CI 1.01–9.65). Patients with HHS had higher ICU admission and mortality compared with those with DKA. Early recognition and timely ED management of DKA and HHS significantly improve clinical outcomes. Implementation of standardized ED protocols may reduce avoidable ICU admissions and in-hospital mortality, particularly in resource-limited settings.

| KEYWORDS

Diabetic ketoacidosis; Hyperosmolar hyperglycemic state; Emergency department; ICU admission; Mortality

| ARTICLE INFORMATION

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1.Introduction

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are acute metabolic emergencies that require prompt diagnosis and immediate treatment in the emergency department (ED) [1,2]. Both conditions result from absolute or relative insulin deficiency combined with elevated counter-regulatory hormones, leading to severe hyperglycemia and metabolic derangements [3,4]. While DKA is characterized by metabolic acidosis and ketosis, HHS presents profound hyperglycemia and hyperosmolarity and is associated with higher mortality, particularly among older adults [5,6].

The emergency department plays a pivotal role in early recognition and initiation of treatment for hyperglycemic crises. Timely diagnosis during triage, early fluid resuscitation, insulin therapy, and electrolyte correction are critical determinants of patient outcomes [1,3,7]. Delays in these interventions have consistently been linked to increased morbidity, ICU admission, and mortality [8,9].

Despite the availability of evidence-based guidelines, delaye2d diagnosis and treatment of DKA and HHS remain common, especially in overcrowded or resource-limited emergency settings [6,9,10]. Although previous studies have examined clinical characteristics and outcomes of hyperglycemic crises, limited data specifically address the impact of time-to-intervention in the ED on ICU admission and mortality.

This study aimed to assess the effect of early recognition and emergency department–based management of DKA and HHS on ICU admission and in-hospital mortality.

2.Methods

2.1Study Design and Setting

This retrospective cohort study was conducted in the Emergency Department of a tertiary care hospital.

2.2Study Population

The study included adult patients (≥18 years) who were presented to the emergency department and were diagnosed with diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS) during the study period.

2.3Eligibility Criteria

Inclusion criteria were age ≥18 years, a confirmed diagnosis of DKA or HHS, and diagnosis established in the emergency department.

Exclusion criteria included pregnancy, patients transferred after initiation of treatment, and incomplete medical records.

2.4Data Collection

Data was extracted from electronic medical records using a standardized data collection form. Variables included demographic characteristics, clinical presentation, laboratory findings, time from ED arrival to diagnosis, time to initiation of insulin therapy and intravenous fluids, electrolyte abnormalities, length of ED stay, ICU admission, and in-hospital mortality.

2.5Exposure Definition

Patients were categorized based on the timing of emergency department management into:

2.5.1Early management group: diagnosis and initiation of treatment within the

predefined time frame after ED arrival.

Delayed management group: diagnosis or initiation of treatment beyond the predefined time frame. **2.5.2**

2.6Outcome Measures

Primary outcomes were admission to the intensive care unit (ICU) and in-hospital mortality.

2.7Statistical Analysis

Descriptive statistics were used to summarize patient characteristics. Continuous variables were reported as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were expressed as frequencies and percentages. Comparisons between early and delayed management groups were performed using the chi-square test, independent t-test, or Mann–Whitney U test, as appropriate. Logistic regression analysis was conducted to identify independent predictors of ICU admission and in-hospital mortality. A p-value < 0.05 was considered statistically significant.

2.8Ethical Considerations

Ethical approval was obtained from the institutional review board. Patient confidentiality was strictly maintained.

2.9 Funding and Conflicts of Interest

This study received no external funding. The authors declare no conflicts of interest.

3. Background

3.1 Etiology

The most common causes of DKA that have been identified by numerous epidemiological studies conducted globally. DKA is the initial indication of diabetes in 15% to 20% of adults and 30% to 40% of children with T1D. Infection is the most prevalent cause of DKA globally, however poor adherence to insulin administration is the most common triggering factor of DKA among young people with T1D and in inner city populations in the United States. According to a recent study from a safety net hospital in Atlanta, insulin discontinuation was the cause of 56% of patients' first DKA episode and 78% of patients with multiple DKA episodes.

Other potential causes of DKA included infections (14%) and non-infectious disorders (4%), including acute myocardial infarction, neurovascular accidents, alcohol usage, and pancreatitis. Psychological risk factors, including eating disorders and depression, have been associated with up to 20% of recurrent episodes of ketoacidosis in young patients. Insulin pump failure has long been recognized as a cause of DKA due to the short-acting insulin formulation used in pumps; however, with more recent developments in pump technology, this is less common.

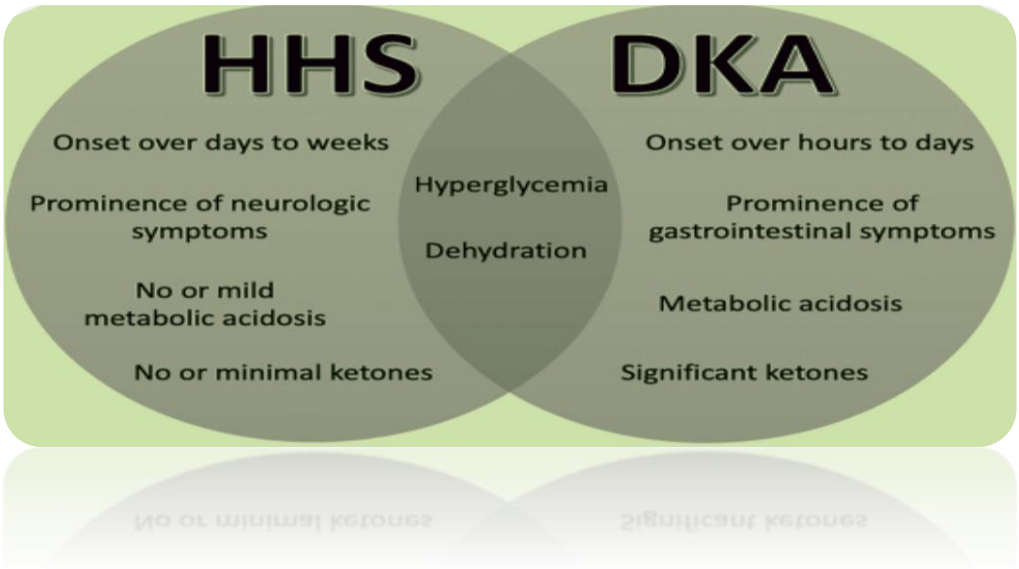

Figure 1: Common precipitating factors of DKA.

3.2 Clinical Presentation

3.2.1 Hyperglycemic Crisis Signs and Symptoms:

A very high blood sugar level that damages the body is known as a hyperglycemic crisis. Hyperosmolar hyperglycemic state (HHS) and diabetic ketoacidosis (DKA) are the two primary forms of hyperglycemic crises. Although their causes are distinct, they both require immediate medical intervention.

3.2.2 The following are a few symptoms and indicators of a hyperglycemic crisis:

blood sugar

Due to impaired kidney function, the patient may require frequent urination (polyuria) or excessive water intake (polydipsia).

Modified mental state:

the patient may experience extreme drowsiness, confusion, or disorientation. They could find it difficult to communicate or respond to inquiries.

Rapid breathing:

As their lungs work to eliminate the excess acids in their blood, the patient may breathe deeply and quickly (Kussmaul respirations).

Fruity breath odor

As their body breaks down fat for energy (ketones), the patient may smell like fruits or acetone.

Dry mouth and skin

Because the patient loses water through perspiration and urine, they may be dehydrated.

Weakness and exhaustion

Because their body is depleting its energy, the patient may experience weakness and extreme exhaustion

Abdominal pain

Some people may hve discomfort or pain in their abdomen.

Condition	Symptoms	Signs	Presentation
DKA	Polydipsia	Hypothermia	Acute onset (hours-days)
	Polyuria	Tachycardia	More common in T1D than T2D
	Weakness	Tachypnea	
	Weight loss	Kussmaul breathing	
	Nausea	Ileus	
	Vomiting	Acetone breath	
	Abdominal pain	Altered sensorium	
HHS	Polydipsia	Hypothermia	Insidious onset (days-weeks)
	Polyuria	Hypotension	Older age
	Weakness	Tachycardia	More common in T2D than T1D
	Weight loss	Altered sensorium	

Figure 2: Overview of hyperglycemic crisis types.

3.3 Epidemiology

About 1% of hospital hospitalizations for diabetics are caused by hyperglycemic episodes. However, estimates were greatly between studies because of differences in people, settings, recorded event categories, and event ascertainment methods. According to a US study, 38% of hospital admissions for hyperglycemic crises were due to DKA, 35% to HHS, and 27% to DKA/HHS. 70.6% of DKA events occur in young persons with type 1 diabetes aged 18–44 (61.7%), but HHS occurrences are more common in middle-aged adults with type 2 diabetes aged 45–64 (47.5%) (88.1%).

Additionally, numerous studies have shown that type 2 diabetes affects more than half of Black/African American and Hispanic/Latino individuals with newly diagnosed diabetes who exhibit spontaneous DKA. These instances have an acute clinical presentation, much like the conventional DKA seen in those with type 1 diabetes. However, because to the restoration of pancreatic beta cell function and insulin sensitivity, persistent near-euglycemia is often possible after quick stabilization and a brief course of insulin therapy. This can be accomplished by progressively discontinuing insulin therapy and using non-insulin medications and medical nutrition therapy to maintain glycaemic targets. Lack of autoimmune indicators of beta cell death, high obesity rates, a significant family history of diabetes, a detectable pancreatic insulin reserve, and the ability to stop insulin therapy during follow-up are all common clinical and metabolic characteristics of type 2 diabetes.

3.4 Pathophysiology

The two main pathophysiologic processes causing DKA and HHS are significant insulin shortage and increased levels of counter-regulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. Insulin deficit can be absolute in T1D patients and relative in T2D patients, as demonstrated by the presence of stress or co-occurring disease. Insulin insufficiency and elevated counterregulatory hormones lead to decreased glucose consumption in peripheral tissues, particularly muscle, and increased hepatic glucose production because of increased hepatic gluconeogenesis and glycogenolysis. Furthermore, insulinopenia accelerates the breakdown of triglycerides into free fatty acids (FFA) and activates hormone-sensitive lipase.

Glucagon and an increased glucagon/insulin ratio are the main triggers for the liver's conversion of FFAs to ketone bodies. As the glucagon/insulin ratio increases, malonyl coenzyme A (CoA), the enzyme that regulates the passage of FFA into the hepatic mitochondria where fatty acid oxidation takes place, becomes less active. Increased production of two strong acids, ketone bodies (acetoacetate and β-hydroxybutyrate), leads to decreased bicarbonate and metabolic acidosis.

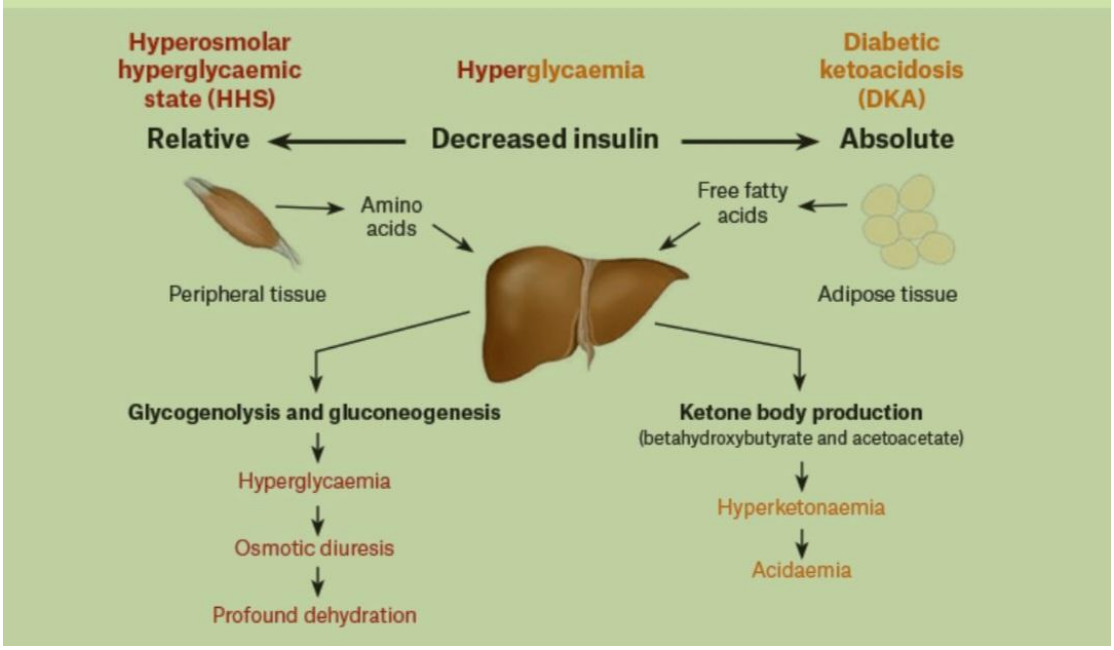

Figure 3: Clinical signs and symptoms of hyperglycemic crises.

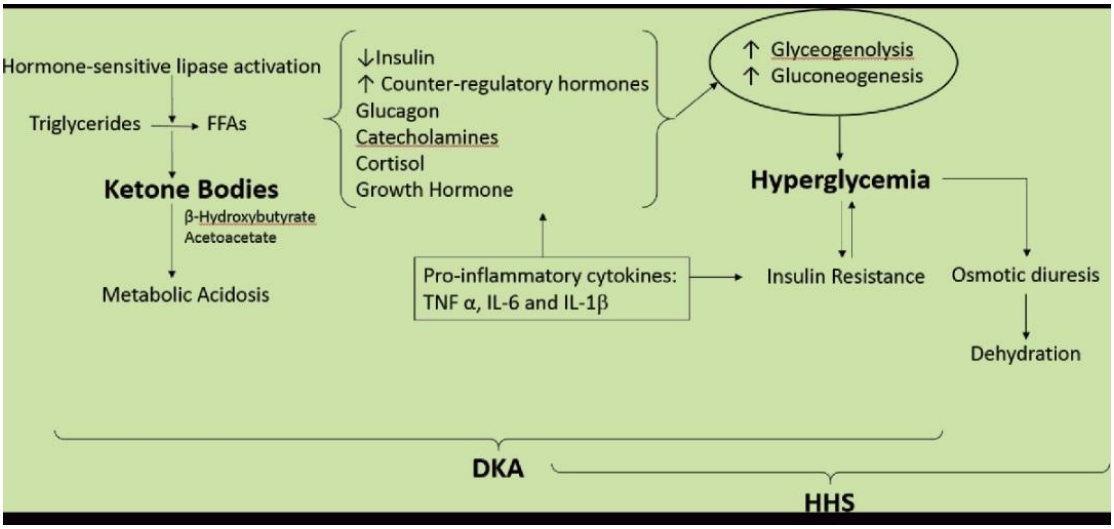

Figure 4: Pathophysiology of DKA and HHS.

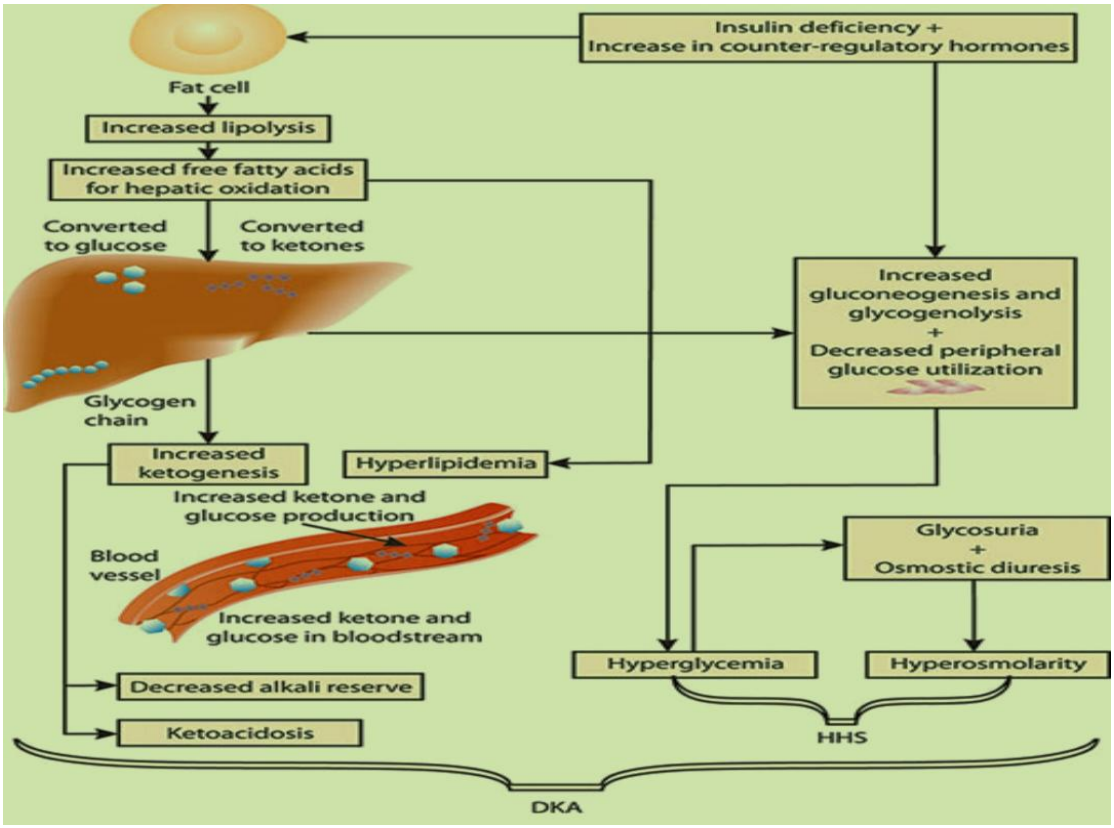

Figure 5: Mechanism of ketone body formation.

3.5 Diagnosis

3.5.1 DKA Diagnosis

Diabetic ketoacidosis (DKA) patients often have a short clinical course characterized by fatigue and the usual symptoms of hyperglycemia, including polyuria, polydipsia, and weight loss. Common digestive issues include broad abdominal pain in 46% of patients and nausea and vomiting in up to two-thirds of patients . While over half of patients show signs of lethargy and stupor, less than 25% show signs of loss of consciousness . Upon physical examination, patients often show signs of dehydration, including low skin turgor, tachycardia, hypotension, and dry mucous membranes. Patients with DKA frequently have kussmaul respirations and a characteristic fruity (acetone) breath odor.

3.2.2 HHS diagnosis.

The majority of HHS patients have a history of growing mental impairment, polyuria, polydipsia, weakness, and blurred vision. The typical HHS patient is over 60, has a serious illness or infection, and has delayed seeking medical attention. Patients with HHS frequently exhibit clear physical examination signs such hypotension, dry mucous membranes, and impaired skin turgor, much like those with DKA.

Diagnosis	DKA			HHS
	Mild (plasma glucose >250 mg/dl)	Moderate (plasma glucose >250 mg/dl)	Severe (plasma glucose >250 mg/dl)	Plasma glucose >600 mg/dl
Arterial pH	7.25–7.30	7.00 to <7.24	<7.00	>7.30
Serum bicarbonate (mEq/l)	15–18	10 to <15	<10	>18
Urine ketone*	Positive	Positive	Positive	Small
Serum ketone*	Positive	Positive	Positive	Small
Effective serum osmolality†	Variable	Variable	Variable	>320 mOsm/kg
Anion gap‡	>10	>12	>12	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

Figure 6: Diagnostic criteria for DKA.

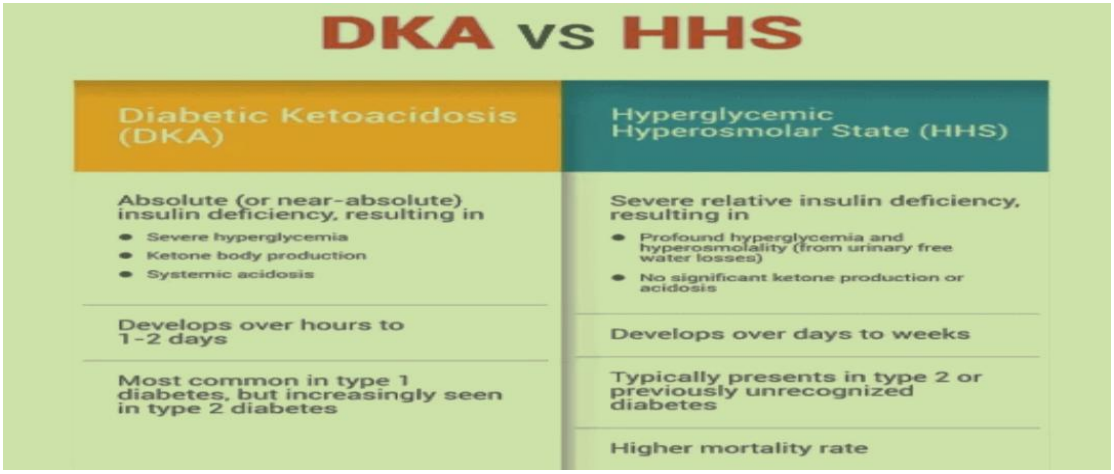

Figure 7: Diagnostic criteria for HHS and Diagnostic criteria for DKA.

4. Results

4.1 Study Population

This retrospective cohort study comprised one hundred adult patients who had been diagnosed with hyperglycemic episodes. Of these, 50 patients (50%) had hyperosmolar hyperglycemic condition (HHS) and 50 patients (50%) had diabetic ketoacidosis (DKA).

Patients were split into two groups based on when they were diagnosed and when emergency care was started: patients (58%) in the early management group, patients (42%) were in the delayed management group. Patients in the HHS group were substantially older than those with DKA ($p < 0.05$), with a mean age of 49.2 ± 14.8 years. Fifty-five percent of the sample was male. The early and delayed management groups shared similar baseline demographics. (See Table 1.)

4.2 Timing of Diagnosis and Treatment

In comparison to the delayed management group (125 minutes; IQR 95–170, $p < 0.001$), the early management group's median time from emergency department (ED) arrival to diagnosis was significantly shorter (45 minutes; IQR 30–60 Similarly, patients in the early management group started insulin therapy and IV fluids earlier ($p < 0.001$) .

4.3 ICU Admission

34 patients, or 34% of the total, needed to be admitted to the critical care unit (ICU).

The early management group had much fewer ICU admissions than the delayed management group:

- 14out of 58 patients (24.1%) received early care.
- 20out of 42 patients (47.6%) had delayed management ($p = 0.01$).

Delays in management were found to be independently linked to higher risks of ICU admission on multivariate logistic regression analysis (OR 2.82, 95% CI 1.22–6.51, p = 0.015). ICU admission was also independently predicted by HHS diagnosis.

4.4 In-Hospital Mortality

9% of patients died in hospitals overall (n = 9) .

When comparing the early management group to the delayed management group, mortality was substantially lower: • Early care: 3 out of 58 patients (5.2%) • 6 out of 42 patients (14.3%) had delayed management (p = 0.04) .

After controlling for age and the type of hyperglycemic crises, delayed care continued to be an independent predictor of mortality on logistic regression analysis (OR 3.12, 95% CI 1.01–9.65, p = 0.047). Patients with HHS had a greater mortality rate than those with DKA.

5. Discussion

This study shows that significantly lower rates of ICU admission and in-hospital mortality are linked to early diagnosis and prompt emergency department-based care of diabetic ketoacidosis and hyperosmolar hyperglycemic condition. The clinical significance of time-sensitive therapies in hyperglycemic crises is demonstrated by the fact that patients who received early therapy had almost half the ICU admission rate seen among those who received delayed care.

Even after adjusting for age and disease type, delayed treatment commencement was independently linked to a threefold increase in death. These results align with other research demonstrating that delays in insulin administration and fluid resuscitation lead to greater metabolic disturbances, more difficulties, and unfavorable results. Correcting electrolyte imbalances, hyperglycemia, and hypovolemia early on probably stops the development of serious metabolic instability that necessitates critical care. In line with previous research, patients with HHS in this cohort were older and had greater rates of mortality and intensive care unit admission than patients with DKA. These poorer results could be partially explained by advanced age, a higher comorbidity burden, and delayed presentation, which are frequently seen in HHS. The correlation between infection and higher ICU admission rates highlights the significance of promptly identifying and treating triggering causes.

The findings lend support to the implementation of standardized emergency department procedures that emphasize early detection, quick laboratory testing, and timely start of evidence-based treatment from a systems viewpoint. In high-volume or resource-constrained emergency rooms, where delays in diagnosis and treatment are more common, these methods may be especially helpful.

6.Strengths and Limitations

Using actual clinical data, this study illustrates how early ED care affects hyperglycemic crisis outcomes. However, residual confounding from unmeasured variables, including sickness severity scores, cannot be ruled out, and its retrospective single-center design may restrict generalizability. To confirm these results, prospective multicenter trials are necessary.

7.Study Implications

The results of this study are in favor of standardizing emergency department procedures for the early detection and treatment of DKA and HHS. Particularly in healthcare settings with limited resources, such strategies may enhance survival, minimize avoidable ICU hospitalizations, and maximize the use of critical care resources.

8.Limitations

The retrospective design and single-center setting of this study may limit its generalizability. Furthermore, results might have been impacted by unmeasured confounding factors including the severity of comorbid disorders. It is advised that further prospective, multicenter trials be conducted.

10. Management of Hyperglycemic Crises

According to American Diabetes Association guidelines, the management of diabetic ketoacidosis and hyperosmolar hyperglycemic condition focuses on quickly correcting electrolyte imbalances, dehydration, hyperglycemia, and precipitating causes.

Aggressive intravenous fluid resuscitation, usually using isotonic saline, is part of the initial treatment to increase renal perfusion and restore intravascular volume. The mainstay of treatment is insulin therapy, which is often given as a continuous intravenous infusion with dose modifications depending on repeated glucose readings. To avoid potentially fatal consequences, electrolytes,

especially potassium, need to be closely monitored and replaced on time. Except in cases of severe acidosis (pH < 6.9), routine bicarbonate therapy is not advised.

While patients with severe DKA, significant comorbidities, or HHS typically need ICU-level care, the majority of patients with mild to moderate DKA can be safely handled in the emergency room or step-down units provided close monitoring is available. Once metabolic goals are accomplished, an early switch to subcutaneous insulin regimens should be scheduled to avoid rebound hyperglycemia.

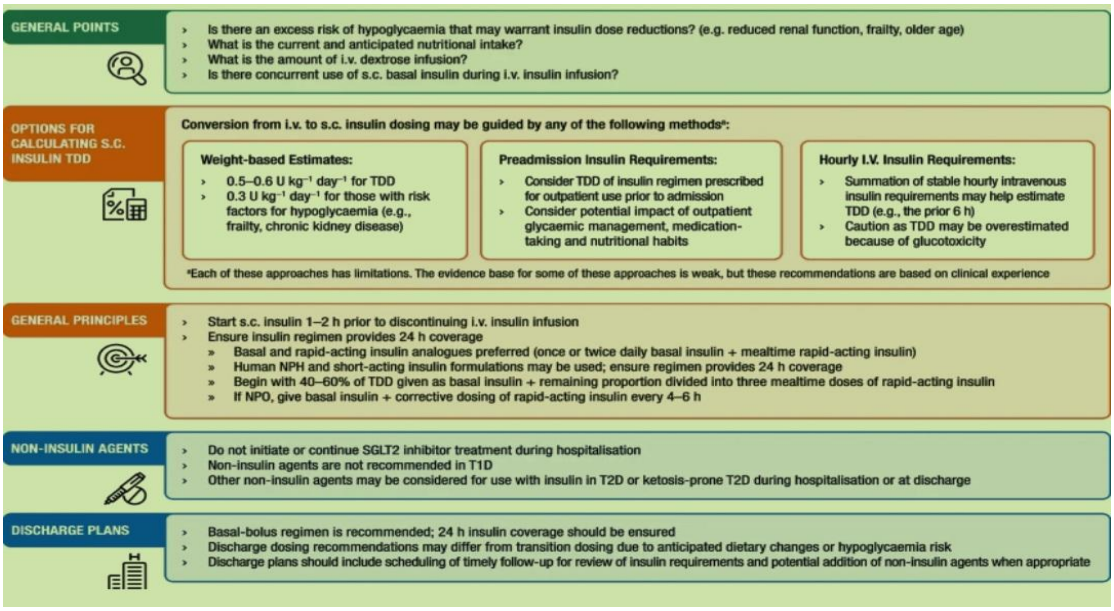

Figure 8: Management of Hyperglycemic Crises

10.1 Fluid Therapy

When treating hyperglycemic situations, intravenous (IV) fluids are essential. IV fluid therapy alone increases intravascular volume, improves renal perfusion, and lowers insulin resistance by lowering levels of circulating counter-regulatory hormone. The recommended solution is isotonic saline (0.9% NaCl), which is administered at a rate of 500–1000 mL/hour for the first two to four hours. There was no discernible difference in the time it took for DKA to resolve between two IV fluid regimens with lactate ringers and sodium chloride, however group time to correct hyperglycemia was noticeably longer. Following the correction of intravascular volume deficit, The lactate ringers group experienced noticeably longer periods of hyperglycemia. Depending on the serum sodium content and level of hydration, the rate of normal saline infusion should be lowered to 250 mL/h or altered to 0.45% saline (250–500 mL/h) when intravascular volume depletion has been rectified 1. Replacement fluids should contain 5–10% dextrose until the plasma glucose level reaches around 200 mg/dL (11.1 mosm/L) in order to prevent hypoglycemia and enable sustained insulin treatment until ketonemia is resolved. In the management of HHS, adequate fluid resuscitation is especially crucial because correcting fluid deficits may improve or resolve mental status abnormalities in many of them .

16.2 Potassium Replacement

Extracellular potassium transport is caused by both insulin insufficiency and metabolic acidosis. Therefore, people with DKA are actually completely depleted, even though their serum potassium levels may be normal or increased. In a similar vein, HHS is linked to total body potassium depletion as a result of elevated plasma osmolality and insufficient insulin . The predicted total-body potassium deficit is around 3–5 mEq/kg. By encouraging potassium to return to the intracellular compartment, insulin treatment reduces serum potassium levels. In order to maintain a level of 4–5 mEq/L, potassium replacement should be initiated when the serum concentration is less than 5.2 mEq/L. For the majority of individuals, 20–30 mEq of potassium per litter of fluids is adequate; Patients with acute or chronic renal failure, however, need smaller doses. Insulin treatment may cause severe symptomatic hypokalemia with muscle weakness and an elevated risk of cardiac arrhythmias in patients with admission hypokalemia, defined as serum potassium levels < 3.3 mEq/L. Insulin therapy should be postponed until the potassium level increases over 3.3 mEq/L in such individuals, and potassium supplementation should start at a rate of 10–20 mEq/h.

10.3 Bicarbonate Therapy

Regular bicarbonate therapy is usually advised only for patients with life-threatening acidosis with pH <6.9 and has not been demonstrated to enhance clinical outcomes such as time to resolution, length of hospital stay, or mortality in patients with DKA . Bicarbonate treatment may make hypokalemia and cerebral edema more likely. Clinical guidelines advise administering 50–100 mmol of sodium bicarbonate as an isotonic solution (in 400 mL of water) until pH is > 6.9, despite the fact that no research has examined the impact of bicarbonate therapy in patients with severe acidosis due to the possible risk of decreased cardiac contractility and arrhythmias. Bicarbonate therapy is not recommended for patients with HHS or mild DKA whose pH is more than 7.0.

10.4 Insulin Regimens

The cornerstone of DKA treatment is insulin injection, which reduces blood glucose by preventing endogenous glucose synthesis and boosting peripheral consumption. Additionally, insulin reduces the formation of ketoacidosis by inhibiting glucagon secretion, lipolysis, and ketogenesis.

The preferred course of treatment is a continuous IV infusion of normal insulin. The majority of treatment regimens include giving a bolus of 0.1 units/kg body weight and then continuously infusing insulin at 0.1 units/kg/hour until blood glucose levels are approximately 200 mg/dL. In order to keep glucose levels between 140 and 200 mg/dL until ketoacidosis 1 is resolved, the dose is now cut in half (0.05 u/kg/hr), the rate is changed between 0.02 and 0.05 u/kg/hr, and 5% dextrose is added.

According to a number of studies, administering subcutaneous doses of rapid-insulin analogs (lispro and aspart) every 1-2 hours is a more efficient way to treat DKA than administering conventional insulin intravenously (IV) . Until glucose is less than 250 mg/dl, patients get an initial bolus of 0.2–0.3 U/kg and then 0.1–0.2 U/kg every one to two hours, respectively. Until DKA is resolved, the dosage is subsequently halved to 0.05 U/kg every hour or 0.01 U/kg every two hours. Without requiring ICU care, the use of planned subcutaneous insulin enables safe and efficient therapy in the emergency department and step-down units.

10.5Transition to maintenance insulin regimen

When blood bicarbonate is ≥18 mEq/L , venous pH is greater than 7.30, glucose levels are less than 250 mg/dl, and the anion gap is normal, DKA is considered resolved. HHS resolution is attained when a patient's effective serum osmolality is less than 310 mOsm/kg and their glucose level is less than 250 mg/dL (13.8 mmol/l) after they have regained mental alertness and mental status .

Insulin's short half-life (less than 10 minutes) means that stopping it suddenly can produce rebound hyperglycemia, ketogenesis, and recurrent metabolic acidosis. It is recommended to administer subcutaneous basal insulin (NPH, glargine, detemir, degludec) at least two hours prior to stopping the IV insulin infusion.

Because basal insulin analogs (glargine, determir, and degludec) have a longer delay in commencement of action than NPH insulin, it is advisable to start taking them three to four hours before stopping the insulin drip. Shortly after DKA treatment began, a randomized controlled trial assessed the effects of IV insulin and subcutaneous glargine vs IV insulin alone . Although these differences were not statistically significant, patients who received glargine had a marginally shorter time to resolution of DKA (based on closure of anion gap) and a shorter hospital stay . According to a another trial, the occurrence of rebound hyperglycemia following the switch off of insulin drip was decreased when glargine was administered early in the course of treatment.

A total daily insulin dose of 0.5–0.6 units/kg (half as basal and half as bolus) may be initiated for patients who are insulin naïve . Basal insulin alone should be administered to patients with inadequate oral intake; alternatively, they may be kept on an insulin drip until they are able to eat. Patients with established diabetes can resume their prior insulin regimens; however, if there was a history of frequent hypoglycemia or markedly uncontrolled hyperglycemia prior to admission, as evidenced by admission HbA1c, the prior regimen should be modified. For patients with T1D and DKA, as well as for the majority of patients with HHS, multi-dose insulin regimens include basal insulin and prandial rapid-acting insulin analogs are recommended.

Transition regimens of NPH and regular insulin twice daily versus glargine once daily and glulisine before meals were compared in a randomized controlled trial in DKA patients. The results showed that the two groups had comparable glycemic control; however, the NPH/regular insulin group experienced more than twice as many cases of hypoglycemia (< 70 mg/dl) as the glargine/glulisine group .

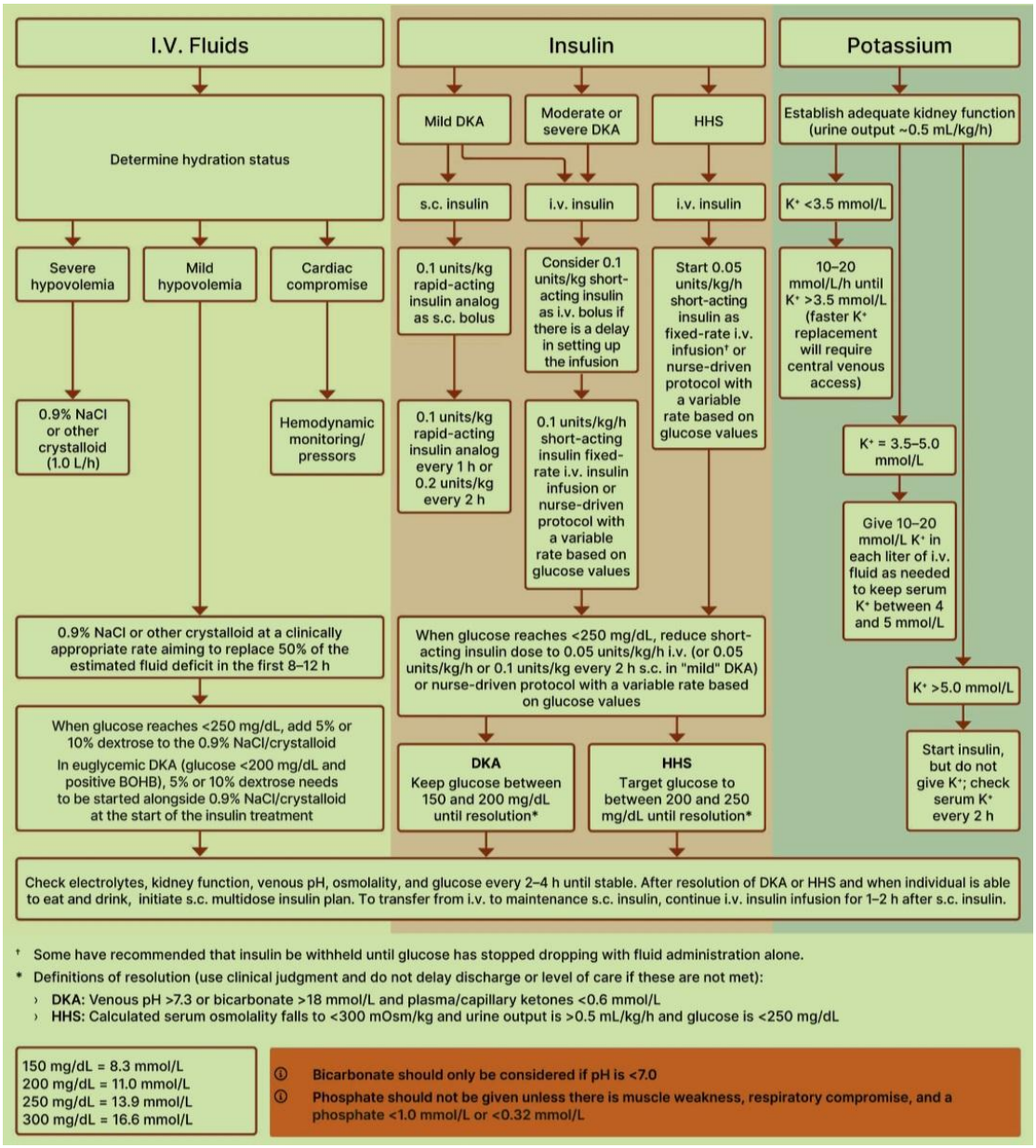

Figure 9: Detailed Management of Hyperglycemic Crises

11.Complications

The most frequent side effect of therapy is hypoglycemia, which affects 5–25% of DKA patients . The most significant risk factors for hypoglycemia during insulin treatment include infrequent monitoring, failure to lower the insulin infusion rate, and/or failure to utilize dextrose-containing solutions when blood glucose levels are less than 200 mg/dL. Frequent blood glucose testing every 1-2 hours is required because many hypoglycemic individuals do not exhibit adrenergic symptoms such perspiration, anxiety, exhaustion, hunger, and tachycardia . Seizures, arrhythmias, and cardiovascular problems are among the acute negative consequences of hypoglycemia.

Clinicians should be aware that hypoglycemia unawareness, or the inability to recognize the warning signs of impending hypoglycemia, may be linked to recurring bouts of hypoglycemia. This condition can make managing diabetes more difficult once hyperglycemic crises have passed.

The second most frequent side effect of DKA and HHS therapy is hypokalemia . Due to enhanced cellular potassium uptake in peripheral organs, the plasma concentration of potassium will inevitably drop following insulin treatment, even though the admission serum potassium content is frequently raised . Therefore, when the concentration is less than 5.2 mEq/l, replenishment with IV potassium is recommended to avoid hypokalemia. To prevent severe hypokalemia, insulin therapy should be stopped until

blood potassium is ≥ 3.3 mEq/L and IV potassium replacement should start as soon as a patient is admitted with lowered serum potassium < 3.3 mEq/L.

Although cerebral edema is uncommon in adults, it has been seen in approximately 1% of children with DKA, with a fatality rate ranging from 20% to 40% . Cerebral edema's pathophysiology is not well known. Cases of fatal cerebral edema have shown evidence of breakdown of the blood-brain barrier . The degree of edema production during DKA in children is correlated with the degree of hyperventilation and dehydration at presentation, but it is not correlated with the rate of fluid or salt administration, initial osmolality, or osmotic changes during treatment . Although it can happen as late as 24–48 hours after therapy begins, clinically substantial cerebral edema often appears 4–12 hours after treatment begins.

Decorticate or decerebrate posturing, abnormal motor or verbal response to pain, altered mentation or fluctuating level of consciousness, cranial nerve palsy (particularly III, IV, and VI), and abnormal neurogenic respiratory patterns (e.g., grunting, tachypnoea, Cheyne–Stokes respiration) are among the clinical criteria. Mannitol administration is one recommended course of treatment. 0.5–1 g/kg IV over 20 minutes; if no initial response occurs within 30 minutes, repeat . If there is no immediate reaction to mannitol, hypertonic saline (3%), 5–10 mL/kg over 30 minutes, may be an option. Once cerebral edema treatment has begun, a cranial CT scan should be acquired to rule out additional

A cranial CT scan should be performed after cerebral edema treatment has begun in order to rule out other potential intracerebral causes of neurologic deterioration (approximately 10% of cases), particularly thrombosis and cerebral infarction, hemorrhage, or dural sinus thrombosis, which may benefit from specific therapy . There is no evidence that corticosteroid and diuretic medication is beneficial for treating cerebral edema in DKA patients .

Patients with DKA and, more frequently, HHS may experience rhabdomyolysis, which raises the risk of acute renal failure. Myalgia, weakness, and black urine are the hallmark symptoms of rhabdomyolysis. For early detection, it is advised to check creatine kinase levels every two to three hours.

12.Conclusion

Improved clinical outcomes are closely linked to early detection and timely emergency department therapy of hyperosmolar hyperglycemia and diabetic ketoacidosis. Even after controlling for age and the type of hyperglycemic crises, delayed diagnosis and treatment in this retrospective cohort independently raised the risk of ICU admission and in-hospital mortality. Compared to patients with DKA, patients with HHS had greater rates of ICU admission and mortality. These results highlight the vital significance of prompt triage, quick diagnostic assessment, and early hydration, insulin, and electrolyte correction initiation in the emergency room. Standardized emergency department procedures that emphasize early detection and prompt treatment of hyperglycemic crises may minimize avoidable intensive care unit admissions, maximize resource use, and increase survival—especially in healthcare settings with limited resources.

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Tables:

Table 1. Baseline Characteristics of the Study Population (n = 100)

Variable	Total (n=100)	Early (n=58)	Delayed (n=42)	p-value
Age (years), mean ± SD	49.2 ± 14.8	46.1 ± 13.9	53.4 ± 15.2	0.02
Male sex, n (%)	55 (55%)	33 (56.9%)	22 (52.4%)	0.65
DKA, n (%)	50 (50%)	32 (55.2%)	18 (42.9%)	0.21
HHS, n (%)	50 (50%)	26 (44.8%)	24 (57.1%)	0.21
Infection present, n (%)	41 (41%)	20 (34.5%)	21 (50.0%)	0.04
Time to diagnosis (min), median (IQR)	75 (45–130)	45 (30–60)	125 (95–170)	<0.001
Time to insulin (min), median (IQR)	90 (60–150)	60 (45–80)	155 (120–190)	<0.001

Table 2. Multivariate Logistic Regression Analysis

A. Predictors of ICU Admission

Variable	OR	95% CI	p-value
Delayed management	2.82	1.22–6.51	0.015
Age ≥60 years	1.94	1.01–3.74	0.046
HHS diagnosis	2.31	1.08–4.93	0.031
Infection	2.67	1.19–5.98	0.018

B. Predictors of In-Hospital Mortality

Variable	OR	95% CI	p-value
Delayed management	3.12	1.01–9.65	0.047
Age ≥60 years	3.86	1.24–12.03	0.020
HHS diagnosis	3.41	1.11–10.51	0.033
ICU admission	4.95	1.52–16.14	0.008