INTRODUCTION

The prevalence of diabetes is on the rise all over the world. Hyperglycemic Emergencies are, therefore, a key component in clinical practice. A high index of suspicion for diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic states (HHS), which are the two of the most serious acute complications of diabetes, is essential for timely intervention and also for prevention of recurrent episodes.

These hyperglycemic emergencies continue to be important causes of morbidity and mortality among patients with diabetes in spite of major advances in the understanding of their pathogenesis and more uniform agreement about their diagnosis and treatment. They can occur in both Type 1 and Type 2 diabetes. The annual incidence rate for DKA estimated from population-based studies ranges from 4.6 to 8 episodes per 1,000 patients with diabetes. In recent epidemiological studies in the U.S., it was estimated that hospitalizations for DKA during the past two decades are increasing. Currently, DKA appears in 4-9% of all hospital discharge summaries among patients with diabetes. The incidence of HHS is difficult to determine because of the lack of population-based studies and the multiple combined illnesses often found in these patients. In general, it is estimated that the rate of hospital admissions due to HHS is lower than the rate due to DKA and accounts for <1% of all primary diabetic admissions.

Mortality rates, which are <5% in DKA and ~15% in HHS, increase substantially with aging and the presence of concomitant life-threatening illness. The prognosis of both conditions is substantially worsened at the extremes of age and in the presence of coma and hypotension.

DEFINITION, CLASSIFICATION, AND CRITERIA FOR DIAGNOSIS

Diabetic Ketoacidosis (DKA) consists of the biochemical triad of hyperglycemia, ketonemia, and academia (Figure 1). As indicated, each of these features by itself can be caused by other metabolic conditions. Hyperglycemic Hyperosmolar State (HHS) has replaced the terms “hyperglycemic hyperosmolar nonketotic coma” and “hyperglycemic hyperosmolar nonketotic state” to highlight that alterations of sensoria may often be present without coma and the HHS may consist of moderate to variable degrees of clinical ketosis. The degree of hyperglycemia in DKA is quite variable and does not determine the severity of DKA. Serum osmolality has been shown to correlate significantly with mental status in DKA and HHS and is the most important determinant of mental status, as demonstrated by several studies.
PRECEPTITATING FACTORS

One of the most common precipitating factors for DKA and HHS is infection in most populations of the world. High temperatures coupled with infections tend to create a stress response and stress hormones (glucagon, catecholamines, cortisol, and growth hormone). The increased production of ketones in DKA is the result of a combination of insulin deficiency and increased concentrations of counterregulatory hormones, particularly epinephrine, which lead to the activation of hormone-sensitive lipase in adipose tissue. The increased activity of tissue lipase causes a breakdown of triglyceride into glycerol and free fatty acids (FFAs). The massive release of FFAs serve as precursors of the ketoacids in DKA. In the liver, FFAs are oxidized to ketone bodies, a process predominantly stimulated by glucagon. In addition to increased production of ketone bodies, there is evidence that clearance of ketones is decreased in patients with DKA.

Disorder of Carbohydrate Metabolism

Insulin deficiency (absolute or relative) results in hyperglycemia which occurs because of (a) increased gluconeogenesis, (b) accelerated glycogenolysis, and (c) impaired glucose utilization by peripheral tissues. Increased glucose production by the liver and kidney represents the major pathogenic disturbance responsible for hyperglycemia in these patients, and gluconeogenesis plays a greater metabolic role than glycogenolysis.

Hyperglycemia causes an osmotic diuresis due to glycosuria, resulting in loss of water and electrolytes, hypovolemia, dehydration, and decreased glomerular filtration rate, which further increase the severity of hyperglycemia. Decreased insulin availability and partial insulin resistance, which exist in DKA and HHS by different mechanisms (see below), also contribute to decreased peripheral glucose utilization and add to the overall hyperglycemic state in both conditions.

Lipid and Ketone Metabolism

The basic underlying mechanism for both HHS and DKA is a reduction in the net effective concentration of circulating insulin, coupled with a concomitant elevation of counterregulatory stress hormones (glucagon, catecholamines, cortisol, and growth hormone). Thus, DKA and HHS are extreme manifestations of impaired carbohydrate regulation. In DKA, the insulin deficiency can be absolute, or it can be insufficient relative to an excess of counterregulatory hormones. In HHS, there is a residual amount of insulin secretion that minimizes ketosis but does not control hyperglycemia. This leads to severe dehydration and impaired renal function, leading to decreased excretion of glucose. Presence of stress in the form of infections and other similar situations results in more severe hyperglycemia than that seen in DKA. Also inadequate fluid intake contributes to hyperosmolarity without ketosis, which is the hallmark of HHS.

Table 1: Causes of Diabetic Ketoacidosis

<table>
<thead>
<tr>
<th>Common causes by frequency</th>
<th>Other causes</th>
<th>Selected drugs that may contribute to diabetic ketoacidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection, particularly pneumonia, urinary tract infection, and sepsis</td>
<td>Acanthosis nigricans</td>
<td>Asymptomatic antipsychotic agents</td>
</tr>
<tr>
<td>New-onset diabetes</td>
<td>Acromegaly</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td>Cardiovascular disease, particularly myocardial infarction</td>
<td>Arterial thrombosis, including mesenteric and iliac</td>
<td>FK506</td>
</tr>
<tr>
<td></td>
<td>Cerebrovascular accident</td>
<td>Glucagon</td>
</tr>
<tr>
<td></td>
<td>Hemochromatosis</td>
<td>Interferon</td>
</tr>
<tr>
<td></td>
<td>Hyperthyroidism</td>
<td>Symptomimetic agents including albuterol (Ventolin), dopamine (Intropin), dobutamine (Dobutrex), terbutaline (Bricanyl), and ritodrine (Yutopar)</td>
</tr>
</tbody>
</table>

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The Role of Counterreulatory Hormones

Growth hormone may also play a prominent role in ketogenesis. Adrenergic stimulation can also increase lipolysis and hepatic ketogenesis. Epinephrine secretion by the adrenal medulla is markedly enhanced in DKA. In addition, epinephrine facilitates hepatic ketogenesis directly. Spontaneous DKA is characterized by simultaneous elevations of multiple insulin-antagonizing (counterregulatory) hormones.
Less ketosis in HHS is probably due to lower growth hormones and higher insulin levels. The higher insulin levels (demonstrated by high basal and stimulated C-peptide) in HHS provide enough insulin to inhibit lipolysis in HHS but not enough for optimal carbohydrate metabolism. Patients with DKA and HHS present with an overlapping clinical picture varying only in degree, but there is no fundamental pathogenetic difference. The hyperosmolality of severe DKA, which occurs in about one-third of DKA patients, is secondary to fluid losses due to osmotic diuresis and to variable degrees of impaired fluid intake due to nausea and vomiting; whereas the hyperosmolality in HHS patients is due to more prolonged osmotic diuresis and to inability to take fluid.

**Water and Electrolyte Metabolism**

The severe derangement of water and electrolytes in DKA and HHS is the result of insulin deficiency, hyperglycemia, and hyperketonemia (in DKA). The development of dehydration and sodium depletion in DKA and HHS is the result of increased urinary output and electrolyte losses. Hyperglycemia leads to osmotic diuresis in both DKA and HHS. The extent of dehydration, however, is typically greater in HHS than in DKA. At first, this seems paradoxical because patients with DKA experience the dual osmotic load of ketones and glucose. The more severe dehydration in HHS, despite the lack of severe ketonuria, may be attributable to the more gradual onset and longer duration of metabolic decompensation, and partially to the fact that patients presenting with HHS typically have an impaired fluid intake. Other factors that may contribute to excessive volume losses include fever, diarrhea, diuretic use, nausea and vomiting. The more severe dehydration, together with the older average age of patients with HHS and the presence of other comorbidities, almost certainly accounts for the higher mortality of HHS.

Estimates of body deficits of water and electrolytes seen in both DKA and HHS are summarised in Table 2.

**Insulin Resistance in Hyperglycemic Crises**

Several studies, however, have demonstrated that when insulin’s action on glucose disposal in diabetic subjects is compared with that in healthy control subjects, both DKA and HHS are associated with a significant amount of insulin resistance.

**DIAGNOSIS OF DKA AND HHS**

**History and Physical Examination**

DKA and HHS are both medical emergencies that deserve urgent recognition and treatment. The most urgent laboratory tests after a prompt history and physical examination are determination of blood glucose by finger stick and urinalysis with reagent strips to assess qualitative amounts of glucose, ketones, nitrite, and leukocyte esterase in the urine.

Attention needs to be paid to 1) patency of airway, 2) mental status, 3) cardiovascular and renal status, 4) sources of infection, and 5) state of hydration. Assessment if these factors helps in deciding the urgency with which various laboratory results should be obtained so that treatment can start without delay. DKA usually develops rapidly, over a time span of <24 h, whereas HHS symptoms may occur more insidiously, with polyuria, polydipsia, and weight loss persisting for several days before admission. In patients with DKA, nausea and vomiting is a common symptom. Abdominal pain is occasionally seen in adults (and is commonly seen in children), sometimes mimicking an acute abdomen.

Acidosis, which can stimulate the medullar respiratory center, can cause rapid and deep respiration (Kussmaul breathing).

Physical examination elicits findings, such as a fruity breath odour (similar to the odour of nail polish remover) as the result of volatile acetone and signs of dehydration, tachycardia, including loss of skin turgor, dry mucous membranes, and hypotension. Mental status can vary from full alertness to profound lethargy; however, <20% of patients with DKA or HHS are hospitalized with loss of consciousness. In HHS, mental obtundation and coma are more frequent because the majority of patients, by definition, are hyperosmolar. In some patients with HHS, focal neurological signs (hemiparesis or hemianopsia) and seizures may be the dominant clinical features.

**Laboratory Evaluation**

The initial laboratory evaluation of a patient with suspected DKA or HHS should include immediate determination of blood glucose, blood urea nitrogen (BUN), arterial blood gases, determination of serum electrolytes, osmolality, creatinine, and ketones; urinalysis; and a complete blood count with differential. Bacterial cultures of urine, blood, and other tissues should be obtained, and appropriate antibiotics should be administered if infection is suspected. In children without heart, lung, or kidney disease, the initial evaluation may be modified, at the discretion of the physician, to include a venous pH in lieu of an arterial pH. The workup for sepsis may be omitted in children, unless warranted by initial evaluation, because the most common precipitating factor of DKA in this age-group is insulin omission. A summary of the biochemical criteria for diagnosis and empirical subclassification of DKA and HHS is presented in Table 3.

**Laboratory Diagnosis of DKA**

The most widely used diagnostic criteria for DKA are blood glucose >250 mg/dl, arterial pH <7.3, serum bicarbonate <15 mEq/l, and moderate degree of ketonemia and/or ketonuria. Accumulation
Diagnostic Criteria for Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State

<table>
<thead>
<tr>
<th></th>
<th>Mild DKA</th>
<th>Moderate DKA</th>
<th>Severe DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose (mg per dL [mmol per L])</td>
<td>&gt; 250 (13.9)</td>
<td>&gt; 250</td>
<td>&gt; 250</td>
<td>&gt; 600 (33.3)</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>7.25 to 7.30</td>
<td>7.00 to 7.24</td>
<td>&lt; 7.00</td>
<td>&gt; 7.30</td>
</tr>
<tr>
<td>Serum bicarbonate (meq per L)</td>
<td>15 to 18</td>
<td>10 to &lt; 15</td>
<td>&lt; 10</td>
<td>&gt; 15</td>
</tr>
<tr>
<td>Urine ketones</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Small</td>
</tr>
<tr>
<td>Serum ketones</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Small</td>
</tr>
<tr>
<td>Beta-hydroxybutyrate</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>Normal or elevated&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Effective serum osmolality (mOsm per kg)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Variable</td>
<td>Variable</td>
<td>Variable</td>
<td>&gt; 320</td>
</tr>
<tr>
<td>Anion gap†</td>
<td>&gt; 10</td>
<td>&gt; 12</td>
<td>&gt; 12</td>
<td>Variable</td>
</tr>
<tr>
<td>Alteration in sensoria or mental obtundation</td>
<td>Alert</td>
<td>Alert/drowsy</td>
<td>Stupor/coma</td>
<td>Stupor/coma</td>
</tr>
</tbody>
</table>

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state.

<sup>a</sup>-Effective serum osmolality = 2.3 measured Na (meq per L) + (glucose [mg per dL] ÷ 18).

†-Anion gap = Na<sup>+</sup> - (Cl<sup>-</sup> + HCO<sub>3</sub><sup>-</sup>) [meq per L]).


... of ketoacids usually results in an increased anion gap metabolic acidosis. The plasma anion gap is calculated by subtracting the major measured anions (chloride and bicarbonate) from the major measured cation (sodium). The normal anion gap has been historically reported to be 12 meq/L, and values >14-15 meq/L have been considered to indicate the presence of an increased anion gap metabolic acidosis. Most laboratories, however, currently measure sodium and chloride concentrations using ion-specific electrodes. The majority of patients admitted with the diagnosis of DKA present with mild metabolic acidosis; however, they show elevations of both serum glucose and hydroxybutyrate concentration. Most of these patients with mild ketoacidosis are alert and could be managed in a general hospital ward. Milder cases of DKA in which the patient is alert and able to tolerate oral intake may be treated and observed in the emergency room for a few hours and then discharged when stable. Patients with severe ketoacidosis typically present with a bicarbonate level <10 mEq/L and/or a pH <7.0, have total serum osmolality >330 mOsm/kg, usually present with mental obtundation, and are more likely to develop complications than those patients with moderate forms of ketoacidosis. Therefore, a classification of the severity of DKA appears to be more clinically appropriate because it may help with patient disposition and choice of therapy.

Assessment of ketonuria and ketonemia, the key diagnostic features of ketoacidosis, is possible by direct measurement of hydroxybutyrate, which is now available in many hospital settings, is preferable in establishing the diagnosis of ketoacidosis.

Laboratory Diagnosis of HHS:

Diagnostic criteria for HHS include plasma glucose concentration >600 mg/dl, serum total osmolality >330 mOsm/kg, and absence of severe ketoacidosis. By definition, patients with HHS have a serum pH 7.3, a serum bicarbonate >18 mEq/L, and mild ketonemia and ketonuria. Approximately 50% of the patients with HHS have an increased anion gap metabolic acidosis as the result of concomitant ketoacidosis and/or an increase in serum lactate levels.<sup>27</sup>

The majority of patients with hyperglycemic emergencies present with leukocytosis. Admission serum sodium concentration is usually low in DKA because of the osmotic flux of water from the intracellular to the extracellular space in the presence of hyperglycemia. To assess the severity of sodium and water deficits, serum sodium may be corrected by adding 1.6 meq/L to the measured serum sodium for each 100 mg/dl of glucose above 100 mg/dl. Admission serum potassium concentration is usually elevated because of a shift of potassium from the intracellular to the extracellular space caused by acidemia, insulin deficiency, and hypertonicity. Conversely in HHS, the measured serum sodium concentration is usually normal or elevated because of severe dehydration. In this setting, the corrected serum sodium concentration would be very high. Admission serum phosphate level in DKA may be elevated despite total-body phosphate depletion.

Differential Diagnosis

Ketoacidosis could be present in other clinical conditions other than DKA like patients with chronic ethanol abuse with a recent binge culminating in nausea, vomiting, and acute starvation may present with alcoholic ketoacidosis (AKA). Whereas DKA is characterized by hyperglycemia (plasma glucose >250 mg/dl), the presence of ketoacidosis without hyperglycemia in an alcoholic patient is virtually diagnostic of AKA. Additionally, AKA patients frequently have hypomagnesemia, hypokalemia, and hypophosphatemia, as well as hypocalcemia, due to decreased PTH as a result of hypomagnesemia.<sup>28</sup>

Starvation Ketosis is present in patients with decreased food intake (<500 kcal/day) for several days and may present with mild ketoacidosis. A healthy subject is, though, able to adapt to prolonged fasting by increasing the clearance of ketone bodies in peripheral tissues (brain and muscle) and by enhancing the kidneys’ ability to excrete ammonium to compensate for the
increased ketoacid production. Thus, patients with starvation ketosis rarely present with a serum bicarbonate concentration <18 mEq/L and do not exhibit hyperglycemia.

Lactic acidosis, advanced chronic renal failure, and ingestion of such drugs as salicylate, methanol, ethylene glycol, and paraldehyde are clinical conditions which produce high anion gap metabolic acidosis, and thus, need to be differentiated from DKA. Measuring blood lactate concentration easily establishes the diagnosis of lactic acidosis (>5 mmol/L) because DKA patients seldom demonstrate this level of serum lactate.

**TREATMENT**

**Therapeutic Goals**

The primary aim of treatment of hyperglycemic crises in diabetes consists of 1) improving circulatory volume and tissue perfusion, 2) decreasing serum glucose and plasma osmolality toward normal levels, 3) clearing the serum and urine of ketones at a steady rate, 4) correcting electrolyte imbalances, and 5) identifying and treating precipitating events (as shown in Table 2 and 3).

**Monitoring**

Monitoring of serum glucose values must be done every 1–2 h during treatment. Serum electrolytes, phosphate, and venous pH...
must be assessed every 2-6 h, depending on the clinical response of the patient. The precipitating factor must be identified urgently and treated. A flow sheet must be maintained to record vital signs, volume and rate of fluid administration, insulin dosage, and urine output and for assessing the efficacy of medical therapy. Figure 2 shows a flow sheet of the management of diabetic ketoacidosis.

Replacement of Fluid and Electrolytes

This is one of the most important aspects of the management of hypoglycemic crises. The severity of fluid and sodium deficits depends upon the duration of hyperglycemia, level of renal function, and patient's oral intake of solute and water. The severity of dehydration and volume depletion can be estimated by clinical examination using the guidelines mentioned below, but these criteria are less reliable in patients with neuropathy and impaired cardiovascular reflexes:

1. An orthostatic increase in pulse without change in blood pressure indicates ~10% decrease in extracellular volume (i.e., ~2 liters isotonic saline).

2. An orthostatic drop in blood pressure (>15/10 mmHg) indicates a 15-20% decrease in extracellular volume (i.e., 3-4 liters).

3. Supine hypotension indicates a decrease of >20% in extracellular fluid volume (i.e., >4 liters).

The initial rate of hydration therapy is repletion of extracellular fluid volume by intravenous administration of isotonic saline to restore intravascular volume; this will decrease counterregulatory hormones and lower blood glucose. The initial fluid of choice is isotonic saline (0.9% NaCl), even in HHS patients or DKA patients with marked hyperglycemia, particularly in patients with evidence of severe sodium deficits manifested by hypotension, tachycardia, and oliguria. Isotonic saline is hypotonic relative to the patient's extracellular fluid and remains restricted to the extracellular fluid compartment. The choice of replacement fluid and the rate of administration in HHS remain Dextrose should be added to replacement fluids when blood glucose concentrations are <250 mg/dl in DKA or <300 mg/dl in HHS. This can usually be accomplished with the administration of 5% dextrose; however, in rare cases, a 10% dextrose solution may be needed to maintain plasma glucose levels and clear ketonemia. This allows continued insulin administration until ketogenesis is controlled in DKA and avoids too rapid correction of hyperglycemia, which may be associated with development of cerebral edema (especially in children). Attention needs to be paid to rapid replacement of fluid in both DKA and HHS for the ongoing urinary losses. Failure to adjust fluid replacement for urinary losses leads to a delay in repair of sodium, potassium, and water deficits.

Insulin Therapy

The important point to emphasize in insulin treatment of patients with DKA and HHS is that insulin should be used after initial serum electrolyte values are obtained while the patient is being hydrated with 1 liter of 0.9% saline. Insulin therapy is then initiated with an intravenous bolus of 0.15 U/kg or 10 U regular insulin, followed by either intravenous infusion of insulin at a rate of 0.1 U · kg⁻¹ · h⁻¹ or subcutaneous or intramuscular injection of 7-10 U/h. However, in children, the initial dose may be 0.1 U/kg continuous infusion with or without an insulin bolus. Some pediatric endocrinologists do not use >3 U/h in children.

In the present proposed protocol, we have used essentially the same insulin regimen for both DKA and HHS. It is advised that only the intravenous route is insulin administration be used because of a greater level of mental obtundation.

The rates of absorption of regular insulin administered intramuscularly and subcutaneously are comparable, with the subcutaneous route being less painful. However, an intravenous route should be used exclusively in the case of hypovolemic shock due to poor tissue perfusion. The insulin rate may be decreased to 0.05-0.1 U · kg⁻¹ · h⁻¹ when blood glucose reaches 250-300 mg/dl. A dextrose solution 5% or, rarely, a 10% solution is added to the hydrating solution at this time to keep blood glucose at its respective level (by adjusting the insulin rate) until the patient has recovered from DKA. Blood glucose monitoring every hour will guide the treating physician whether this is sufficient to produce a consistent reduction in blood glucose. If blood glucose fails to decrease at a rate of 50-70 mg · dl⁻¹ · h⁻¹, the patient's volume status should be reassessed to ensure adequate volume repletion.

Potassium

Serum potassium levels deficits are present in both DKA and HHS and represent intracellular losses. Extracellular hyperosmolarity, secondary to hyperglycemia, causes a shift of water and potassium from the intracellular to the extracellular space, resulting in normal or elevated serum potassium concentrations despite total-body potassium deficits of 500-700 mEq/l. This potassium shift is further enhanced by insulin deficiency and the presence of acidosis and accelerated breakdown of intracellular protein.

Excessive urinary potassium losses, which occur as a result of osmotic diuresis with increased delivery of fluid and sodium to potassium secretory sites in the distal nephron, are ultimately responsible for the development of potassium depletion. During treatment of DKA and HHS with hydration and insulin, there is typically a rapid decline in plasma potassium concentration as potassium reenters the intracellular compartment. However, potassium replacement should not be initiated until the serum potassium concentration is <5.5 mEq/l.

Bicarbonate

The routine use of alkali therapy in DKA is not recommended because DKA tends to correct with insulin therapy. Insulin
Table 4: Strategies to Prevent Diabetic Ketoacidosis

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Diabetic education</td>
<td>Blood glucose monitoring</td>
</tr>
<tr>
<td>Sick-day management</td>
<td>Home monitoring of ketones or beta-hydroxybutyrate</td>
</tr>
<tr>
<td>Supplemental short-acting insulin regimens</td>
<td>Easily digestible liquid diets when sick</td>
</tr>
<tr>
<td>Reducing, rather than eliminating, insulin when patients are not eating</td>
<td>Guidelines for when patients should seek medical attention</td>
</tr>
<tr>
<td>Case monitoring of high-risk patients</td>
<td>Special education for patients on pump management</td>
</tr>
</tbody>
</table>

administration inhibits ongoing lipolysis and ketoad production and promotes ketoanion metabolism. The only indication for the use of bicarbonate is life-threatening hyperkalemia.35 It was, therefore, concluded that administration of bicarbonate in DKA patients (with pH of 6.9-7.14) provided no measurable advantage either biochemically or clinically. Bicarbonate should be administered as an isotonic solution, which can be prepared by addition of one ampoule of 7.5% NaHCO₃ solution (50 mmol HCO₃⁻) to 250 ml sterile H₂O. One needs to add 15 mEq of KCl for each ampoule of bicarbonate administration (if serum potassium is <5.5 mEq/l).

Phosphate Therapy

There is a shift of phosphate, along with potassium, from the intracellular to the extracellular compartment in response to hyperglycemia and hyperosmolarity. Osmotic diuresis also leads to enhanced urinary phosphate losses. Because of the shift of phosphate from the intracellular to the extracellular compartment, serum levels of phosphate at presentation with DKA or HHS are typically normal or increased. As insulin therapy is initiated, phosphate reenters the intracellular compartment, leading to mild to moderate reductions in serum phosphate concentrations. Replacement of phosphate is essential in with serum phosphate concentrations <1.0 mg/dl and to patients with moderate hypophosphatemia and concomitant hypoxia, anemia, or cardiorespiratory compromise. If phosphate replacement is needed, 20-30 mEq/l potassium phosphate can be added to replacement fluids and given over several hours. In such patients, because of the risk of hypocalcemia, serum calcium and phosphate levels must be monitored during phosphate infusion.

Table 4 summaries strategies to prevent diabetic ketoacidosis.

Immediate Posthyperglycemic Care

Low-dose insulin therapy provides a circulating insulin concentration of ~60-100 µU/ml. Sudden interruption of insulin infusion can lead to rapid lowering of insulin concentration, resulting in a relapse into DKA or HHS. Thus, there is a need for frequent monitoring during the posthyperglycemic period.14

REFERENCES

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