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Management of a DKA Patient with Severe Metabolic and Ketoacidosis with Chronic Renal Insufficiency

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Introduction

Diabetic ketoacidosis (DKA) serves as one the leading causes of mortality in diabetic patients [14]. The mortality has decreased over the past several decades due to the rapid recognition of the disease state and the improvement of management of DKA [14]. Despite a decline in mortality rates over the past twenty years from 7.96% to 0.67%, errors in management of the disease state are associated with significant morbidity and mortality [2]. Utilization of DKA protocols in the acute care setting have allowed congruency in care and delivery of effective lifesaving treatment. Despite advances in standardized DKA protocols, there still remains a gap in how to manage specific patient populations with end stage renal disease. Understanding the pathophysiology behind these patient populations will yield better outcomes with the ultimate goal of decreasing the mortality rate.

Case Study

A 55 year old female patient with a past medical history of type one diabetes and end-stage renal disease presents to the intensive care unit with diabetic ketoacidosis. The patient had the following lab values:

- **Glucose-1,107 mg/dL**
- **Beta hydroxybutyrate-7.6 mg/dL**
- **Anion Gap-36 mEq/l**
- **Creatinine-4.54 mg/dL**
- **BUN-80 mg/dL**
- **GFR-<5ml/min**
- **Lactate-4.1 mg/dL**

ABG:

- **pH-6.85**
- **Co2-12 mmHg**
- **HCO3-1.9 mmol/L**
- **PO2-197 mmHg**

These lab values indicate the combination of severe ketoacidosis from DKA and metabolic acidosis related to chronic renal insufficiency.

The patient arrives to the intensive care unit with an intact neurological status, stable vital signs, and on room air. The nurse titrated the insulin drip per hospital's DKA policy. This policy states that the nurse is able to double the insulin infusion rate if the blood glucose level has not decreased after initiating the drip. The patient's blood glucose had not improved despite intravenous IV insulin; therefore, the nurse titrated the drip up to 100 units/hr. At the same time, the patient received 2 amps of sodium bicarbonate IV push and was started on a sodium bicarbonate infusion. After some time, the patient's labs started to trend in the right direction with a closing anion gap, decrease in glucose level, increase in pH and decrease in ketone levels. The insulin infusion rate ran at a rate of 100units/hr for a total of 5 hours then it was weaned off based on the closure of the anion gap and decrease in the patient's glucose level.

During day shift, the patients' blood glucose plummeted into the low 40s on three different occasions. Each time, the nurse gave 1 amp of Dextrose 50%.

Underlying Pathophysiology

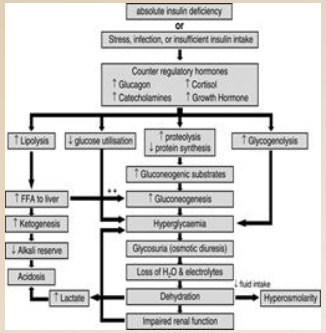

Figure 1: Pathological process of DKA [15]

DKA develops as a result deficient levels of insulin circulating throughout the body causing a triad of hyperglycemia, hyperketonemia, and acidosis [10,14]. Counterregulatory hormones consisted of catecholamines, glucagon, and cortisol are activated in a state of insulin deficiency ultimately causing an increased blood glucose level. The glucagon activates gluconeogenesis in the liver to form more glucose and the body starts to break down adipose tissue through lipolysis which yields free fatty acids and a state of hyperketonemia. The circulating ketones induces a state of acidosis with a decreased bicarbonate reserve. Severe dehydration and electrolyte abnormalities occur as the result of osmotic diuresis associated with the hyperglycemic state [10].

Patients with chronic kidney disease often develop a common complication of metabolic acidosis [3,7]. A state of ph homeostasis is usually maintained by the functional units of the kidneys or nephrons through the excretion of ammonia with a normal glomerular filtration rate (GFR). When the GFR reaches a dysfunctional state of below 40ml/min, there is a decrease in ammonium excretion and an increase in hydrogen ion retention causing a state of acidosis [3]. Other key factors that contribute to the acidosis are the decreased synthesis of the acid buffer bicarbonate and the increased excretion of the bicarbonate through the gastrointestinal and urinary tract [7].

Significance of Pathophysiology

Healthcare providers are able to tailor case-specific treatment through an in-depth analysis and dissection on the pathophysiological processes associated with a patient in DKA with a comorbidity of chronic kidney disease. The goal of treatment for DKA is to reduce serum glucose levels and ketoacidosis through the administration of parental insulin. This will ultimately result in the improvement of acidosis and will produce a state of acid-base homeostasis. This may not be an effective solution to follow in patients who present with severe metabolic and ketoacidosis.

Insulin resistance is defined as "a clinical condition where there is a reduced biological effect for any given blood concentration of insulin," [8]. It is characterized as reduced tissue responses to the action of insulin [1]. One study concluded that there is a direct relationship between a lower serum bicarbonate and high anion gap which are both independently associated with reduced insulin sensitivity [4]. The data from this study supports the hypothesis that the production of organic acid serves as a central feature of insulin resistance [4]. The use of alkali therapy in DKA is controversial because the acidosis should correct itself through the administration of insulin and fluids; however, many studies show that insulin sensitivity is decreased in uremic patients; therefore, treatment of metabolic acidosis through alkali therapy is necessary in achieving a decrease in insulin resistance with patients that also present with end stage renal disease [1,9,11,13]. Data suggests that acidosis should be corrected as soon as possible which will result in increased insulin sensitivity to lower blood glucose levels and decrease lipolysis [6]. The exogenous insulin given to treat patients in DKA is insensitive and ineffective without neutralizing the blood pH through bicarbonate therapy in such extreme circumstances. The American Diabetes Association states how severe acidosis in hyperglycemic crises can lead to more adverse effects and that adult patients with a pH < 6.9 should receive sodium bicarbonate until pH>7.0 is achieved [5].

The kidneys play a major role in metabolism of insulin [12]. Approximately 25% of daily insulin production is degraded by the kidneys [12]. Renal metabolism is augmented in diabetic patient's receiving exogenous insulin because the insulin bypasses filtration through the liver when injected into the systemic circulation [12]. Since the renal clearance of insulin is 200ml/min, patients with an impaired GFR will have unfiltered insulin freely circulating in the body [12]. This becomes significant when treating a DKA patient with renal insufficiency. Administering exogenous IV insulin to this patient without correcting the patient's acidosis can result in serious adverse outcomes and even death by inducing a state of hypoglycemia. The patient's extreme metabolic acidosis will result in a decreased insulin sensitivity and a decreased ability to filter insulin out of the body; therefore, the patient is at risk for developing hypoglycemia when the acidosis is corrected. This concept was evident in the case study when the patient's blood glucose levels had dramatically declined the following day due to increased levels of circulating insulin along with the correction of the patient's acidosis

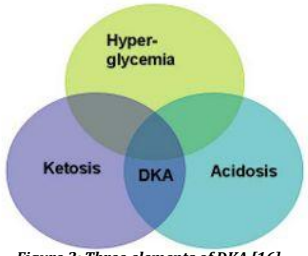

Figure 2: Three elements of DKA [16]

Implications for nursing care

- Identify if the patient with DKA has renal insufficiency
- Draw serial labs: Chem 7, Beta hydroxybutyrate, and ABGs q 6 hours
- Consult with physician to obtain order to administer sodium bicarbonate if pH<6.9
- Monitor hourly blood glucose level
- Follow DKA protocol for fluid management strategy
- Provide electrolyte supplementation as blood glucose levels normalize
- Call physician to transition patient onto subcutaneous insulin when anion gap closes

Conclusion

The current standardized DKA protocols do not address patients with renal insufficiency. Treatment for diabetic patients in DKA with chronic kidney disease should revolve around correcting extreme metabolic acidosis states (pH<6.9) before the administration of parental insulin. This approach will increase insulin sensitivity and utilization to decrease blood glucose levels and halt the process of lipolysis, ultimately resolving DKA.

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