Management of a DKA patient with severe metabolic and ketoacidosis with chronic renal insufficiency

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Diabetic ketoacidosis (DKA) serves as one of the leading causes of mortality in diabetic patients [1]. 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 [2]. Despite a decline in mortality rates over the past twenty years from 7.8% to 0.67%, errors in management of the disease state are associated with significant mortality and morbidity [2]. Utilization of DKA protocols in the acute care setting has slowed the progression of care in and delivery of effective biochemical 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.

Introduction

Case Study

A 55 year old female patient with type 2 diabetes and end-stage renal disease presents to the intensive care unit with diabetic ketoacidosis. The patient had a lab following labs:

- Glucose: 1,107 mg/dL
- Beta hydroxybutyrate: 7.6 mmol/L
- Lactate: 36 mg/dL
- BUN: 80 mg/dL
- GFR: <5 ml/min
- Creatinine: 4.1 mg/dL

ARG
- pH: 4.85
- Co2: 11 mmol/L
- HCO3: 1.9 mmol/L
- PO2: 197 mmHg

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

The patient arrives to the intensive care unit with an intact neurocognitive status, stable vital signs, and open airway. The nurse initiated the insulin drip per hospital’s protocol. This policy states that the nurse is able to double the rate of insulin if the blood glucose level has not decreased after starting the drip. The patient’s blood glucose had not improved despite multiple attempts to increase the rate of insulin. The nurse initiated the drip up to 100 units/hr. At the same time, the patient’s labs started to trend in the right direction with a drop in ketones and decrease in the glouce level, increase in pH and decrease in ketone levels. The insulin infusion rate was at a rate of 100 units/hr for a total of 5 hours before it was changed to a constant rate of 20 units/hr.

During day shift, the patient’s blood glucose levels did not cross 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 of deficient levels of insulin. Insulin is responsible for channeling throughout the body the causing a virtual of hyperglycemia, hyperketonemia, and acidosis [10,14]. Counterregulatory hormones consisting of catecholamines, glucagon, and corticosteroids 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. Serum dehydration and electrolyte abnormalities occur as the result of osmotic diuresis associated with the hyperosmotic state [10].

Patients with chronic kidney disease often develop a common complication of metabolic acidosis [3]. A state of metabolic acidosis is usually maintained by the functional units of the kidneys with the exception through the excretion of ammota with a normal glomerular filtration rate (GFR). When the GFR reaches a functional state of below 60ml/min, there is a decrease in ammonium excretion and an increase in hydrogen ion retention causing a state of acidosis [4]. 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 [1].

Significance of Pathophysiology

Figure 2: Three elements of DKA ([8])

Healthcare providers are able to tailor case-specific treatment through an in-depth analysis and discussion on the pathophysiologic processes associated with a patient in DKA with a comorbidty of chronic kidney disease. The goal of treatment for DKA is to reduce serum glucose levels and ketoads through the administration of parental insulin. This will ultimately result in the improvement of acidosis and will produce a state of acid-base homeostasis. The patient may not be an effective solution to follow in patients who present with severe metabolic and blindness. 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 [2]. Despite a decline in mortality rates over the past twenty years from 7.8% to 0.67%, errors in management of the disease state are associated with significant mortality and morbidity [2]. Utilization of DKA protocols in the acute care setting has slowed the progression of care in and delivery of effective biochemical 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.

Implications for nursing care

- Identify if the patient with DKA has renal insufficiency
- Draw urine samples: Clais 7, Beta hydroxybutyrate, and ABGs q 6 hours
- Consult with physician to obtain order for bicarbonate if pH <6.9
- Follow DKA protocol for fluid management
- Decrease bicarbonate level
- Follow DKA protocol for fluid management
- Bicarbonate administration
- Increase ventilatory rate
- Maintain electrolyte balance
- Increase urine output
- Administer fluids and insulin

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 levels (pH <6.9) before the administration of parental insulin. This approach will increase insulin sensitivity and utilization to decrease blood glucose levels and shorten the process of lipolysis, ultimately resolving DKA.

References