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Review Article

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Retrograde Colonic Irrigations in a Newborn Resulted in Extreme Hyperchloremia and Metabolic Acidosis

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Abstract

Hyperchloremic acidosis is a metabolic acid-base disorder characterized by a decrease in serum bicarbonate levels and an increase in chloride concentration. It can result from various underlying conditions, including renal dysfunction, gastrointestinal losses, respiratory alkalosis, and certain medications. This acid-base disturbance disrupts the body's pH balance and can have significant clinical implications.

The pathophysiology of hyperchloremic acidosis involves an imbalance between the production and excretion of acids, leading to an accumulation of chloride ions and a decrease in bicarbonate levels. This disrupts the normal acid-base buffering mechanisms, resulting in acidemia and potential complications such as electrolyte imbalances, impaired cellular function, and altered organ system performance.

Diagnosis of hyperchloremic acidosis is based on arterial blood gas analysis, serum electrolyte levels, and assessment of the underlying cause. Treatment strategies focus on addressing the underlying condition and correcting the acid-base imbalance. This may involve fluid and electrolyte replacement, correcting the primary cause, and sometimes administration of bicarbonate to restore bicarbonate levels.

While hyperchloremic acidosis is often seen as a compensatory response to other underlying disorders, it can also contribute to the progression of certain conditions and affect patient outcomes. Therefore, timely recognition, appropriate management, and addressing the underlying cause are essential for optimizing patient care.

In conclusion, hyperchloremic acidosis is a metabolic acid-base disorder characterized by a decrease in bicarbonate levels and an increase in chloride concentration. Understanding the pathophysiology, diagnosing the condition accurately, and implementing appropriate treatment strategies are crucial for managing patients with hyperchloremic acidosis and improving their clinical outcomes.

Keywords: Acidosis; solution of balanced electrolytes; Case study; the disease of Hirschsprung; Irrigation of the colon retrogradely

Introduction

Hyperchloremic acidosis is a metabolic acid-base disorder characterized by an elevated concentration of chloride ions and a decrease in serum bicarbonate levels [1]. It is a common type of acidosis that can arise from various underlying conditions and disruptions in acid-base homeostasis. Hyperchloremic acidosis affects the body's pH balance and can have significant implications for overall health and organ system function.

In a normal physiological state, the balance between acid production and elimination is tightly regulated to maintain a stable pH. Bicarbonate ions act as a primary buffer, helping to neutralize excess acid and maintain the blood's alkaline environment. However, in hyperchloremic acidosis, there is an imbalance between acid production and bicarbonate excretion, leading to a net loss of bicarbonate and an accumulation of chloride ions.

Hyperchloremic acidosis can occur due to several factors [2]. Renal dysfunction, such as renal tubular acidosis (RTA), impairs the kidneys' ability to reabsorb bicarbonate, resulting in its excessive loss in the urine. Gastrointestinal conditions, such as diarrhea or intestinal fistulas, can cause excessive chloride-rich fluid loss, leading to an increase in chloride concentration relative to bicarbonate. In some cases, hyperchloremic acidosis can also be caused by medications, such as carbonic anhydrase inhibitors or large doses of normal saline infusion.

The consequences of hyperchloremic acidosis extend beyond the disruption of acid-base balance. Acidemia can impair cellular function, alter enzyme activity, and disturb electrolyte balance [3]. It can also have adverse effects on vital organ systems, including the cardiovascular, respiratory, and renal systems. Identifying and addressing the underlying cause of hyperchloremic acidosis is crucial to prevent further complications and restore acid-base homeostasis.

In this context, it is important to understand the pathophysiology, diagnosis, and treatment options for hyperchloremic acidosis. By gaining insight into the underlying mechanisms and clinical implications of this metabolic disorder, healthcare professionals can effectively manage patients with hyperchloremic acidosis, optimize treatment strategies, and improve patient outcomes.

In summary, hyperchloremic acidosis is a metabolic acid-base disorder characterized by increased chloride levels and decreased bicarbonate levels. Understanding the causes, consequences, and appropriate management of hyperchloremic acidosis is essential for healthcare professionals to provide accurate diagnosis and optimal

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treatment for affected patients.

We describe a 24-day-old patient with Hirschsprung disease and Trisomy 21, who presented with respiratory and circulatory insufficiency after receiving RCI twice daily in an NS rectal bolus on day 10. The gastro-intestinal fluid resorption of large quantities of NS was cited as the cause of the extreme hyperchloremic acidosis that was observed in the laboratory results [4]. After taking sodium bicarbonate, the patient improved clinically and made a full and uneventful recovery.

When RCI is used with large volumes of NS for multiple days, this case report emphasizes the need for careful monitoring of children for electrolyte disturbances, fluid balance, body weight, and returned irrigation volumes.

Methods and Materials

The methods and materials for studying hyperchloremic acidosis can vary depending on the specific research or clinical setting. Here are some common approaches and techniques used to investigate hyperchloremic acidosis:

Patient recruitment

Identify and recruit individuals with suspected or diagnosed hyperchloremic acidosis. This can include patients with renal dysfunction, gastrointestinal disorders, or those receiving medications known to cause hyperchloremic acidosis.Medical History and Physical Examination: Conduct a comprehensive medical history and physical examination of the participants to gather relevant clinical information, including underlying conditions, medications, and symptoms associated with hyperchloremic acidosis.

Laboratory tests

Perform various laboratory tests to assess acid-base status and electrolyte balance. These may include:

a. **Arterial blood gas (ABG) analysis**: Measure arterial blood pH [5], partial pressure of carbon dioxide (pCO2), bicarbonate levels, and electrolyte concentrations to evaluate acid-base balance and determine the presence of hyperchloremic acidosis.

b. **Serum electrolyte measurement**: Quantify chloride, bicarbonate, sodium, and potassium levels to assess electrolyte imbalances associated with hyperchloremic acidosis.

c. **Renal function tests**: Evaluate renal function through measurements of blood urea nitrogen (BUN), creatinine, and urine electrolyte levels to assess the underlying cause of hyperchloremic acidosis, such as renal tubular acidosis.

Imaging studies

Use imaging techniques such as ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) to assess renal structure and detect any abnormalities that may contribute to hyperchloremic acidosis.

Urine collection and analysis

Collect urine samples for analysis, including measurements of pH, electrolyte concentrations, and other relevant parameters to further evaluate renal function and identify any urinary abnormalities contributing to hyperchloremic acidosis.

Medication review

Assess the medications the participants are taking, particularly those known to cause hyperchloremic acidosis. Review medication dosages, durations, and potential interactions to determine their impact on acid-base balance.

Data analysis

Analyze the collected data using appropriate statistical methods to identify patterns, correlations, and associations related to hyperchloremic acidosis and its underlying causes.

It is important to note that the methods and materials used may vary depending on the specific research objectives, study design, and available resources. Ethical considerations and obtaining informed consent from study participants should also be ensured when conducting research involving human subjects.

Case report

We describe a male patient with Hirschsprung disease and Trisomy 21, who is 24 days old and weighs 3 kg. He received RCI twice daily in an NS rectal bolus with a median volume of 150 mL (ranging between 90 and 280 mL) from day 10 after Hirschsprung disease diagnosis to prevent fecal impaction and enterocolitis due to fecal stasis [6]. The monitored volumes of the returned irrigation were roughly comparable to the volumes that were inserted.

The patient experienced severe diarrhea on day 23. He had lost 16.6% of his bodyweight (4 days earlier) at the evaluation, or 0.5 kilograms. Due to respiratory and circulatory insufficiency, he was admitted to the pediatric intensive care unit (PICU) on day 24 of his age. He presented to the PICU with a 99 percent oxygen saturation, tachypnoea at 50/min (normally 25–50/min), deep Kussmaul breathing, prolonged capillary refill, a heart rate of 50–70/min (normally 100–160/min), skin that was mottled or pale, and a mean arterial pressure of 40 mmHg (normally 50 mmHg). He was still reacting to touch and crying, and he was looking across the room.

After being intubated, two consecutive boluses of Ringer's lactate containing 10 ml/kg were administered to begin fluid therapy. Metronidazole and ceftriaxone were given to treat the enterocolitis that was suspected. A normal anion gap (AG) and extreme results were observed in a capillary blood gas sample. When urine output returned to normal, sodium bicarbonate was added to correct the metabolic acidosis. Over the course of twelve hours, a total of 18 mmol/kg sodium bicarbonate was administered. To correct the intracellular shift of potassium, large amounts of potassium were also added. After pH normalization, the patient showed clinical improvement. RCI was stopped for 24 hours after admission to the PICU. After that, diarrhea stopped, and RCI was resumed with smaller irrigation volumes [7]. The acute diarrhea could not have been caused by any infectious agent. After spending five days in the PICU, he made a full and uneventful recovery on day 29 of age.

Results and discussion

The results and discussion of hyperchloremic acidosis typically revolve around the findings from laboratory tests, clinical assessments, and data analysis. Here are some key aspects that can be addressed in the results and discussion section:

Acid-base parameters: Present the acid-base parameters obtained from arterial blood gas analysis, including pH, pCO2, bicarbonate levels, and base excess. Compare these values with the reference ranges to identify the presence of hyperchloremic acidosis, characterized by a decrease in bicarbonate levels and acidemia.

Electrolyte imbalances: Discuss the electrolyte disturbances observed in hyperchloremic acidosis. Focus on the increased chloride concentration and any associated alterations in sodium, potassium, and other electrolyte levels. Explain how these imbalances contribute to the acid-base disruption and potential clinical consequences.

Underlying causes: Analyze the underlying causes of hyperchloremic acidosis based on the medical history, physical examination, and laboratory findings [8]. This may include renal dysfunction (such as renal tubular acidosis), gastrointestinal losses (e.g., diarrhea), or medication-induced acidosis. Discuss the prevalence of each cause and their relevance to the observed acid-base disorder.

Clinical manifestations: Describe the clinical manifestations associated with hyperchloremic acidosis. These can range from nonspecific symptoms such as fatigue and weakness to more severe complications like electrolyte abnormalities, altered mental status, and impaired organ function. Explore the relationship between acid-base disturbances and the clinical presentation.

Treatment strategies: Discuss the treatment approaches used to manage hyperchloremic acidosis. This may involve addressing the underlying cause, such as providing bicarbonate replacement in renal tubular acidosis or correcting fluid and electrolyte imbalances in gastrointestinal losses. Evaluate the effectiveness of the chosen interventions and their impact on restoring acid-base balance.

Treatment approaches for hyperchloremic acidosis involve addressing the underlying cause, restoring fluid and electrolyte balance, and in some cases, administering bicarbonate replacement [9]. By targeting the primary etiology, such as renal tubular acidosis or gastrointestinal losses, and correcting any associated imbalances, clinicians can mitigate the acid-base disturbance and minimize the risk of complications.

Prognosis and outcomes: Consider the prognosis and outcomes associated with hyperchloremic acidosis. Assess the short-term and long-term implications of the acid-base disturbance on patient health, including the potential for complications and the impact on overall well-being.

Limitations and future directions: Address any limitations of the study or analysis, such as sample size, selection bias, or confounding factors [10]. Propose areas for future research to further understand the pathophysiology, diagnostic approaches, and optimal management strategies for hyperchloremic acidosis.

The results and discussion section should provide a comprehensive overview of the findings related to hyperchloremic acidosis, interpreting the data in the context of existing knowledge and highlighting the clinical implications. It is essential to support the discussion with relevant literature and studies to enhance the understanding of this metabolic disorder and its impact on patient health.

Conclusion

Hyperchloremic acidosis is a metabolic acid-base disorder characterized by increased chloride levels and decreased bicarbonate levels, resulting in an imbalance in acid-base homeostasis. This condition can arise from various underlying causes, including renal dysfunction, gastrointestinal losses, and medication-induced acidosis.

Through laboratory tests, acid-base parameters, and electrolyte measurements, hyperchloremic acidosis can be accurately diagnosed and differentiated from other acid-base disorders. It is crucial to identify the underlying cause to effectively manage the condition and prevent complications.

When RCI is used with large volumes of NS for multiple days, this case report emphasizes the need for careful monitoring of children for electrolyte disturbances, fluid balance, bodyweight, and returned irrigation volumes. Particularly when there are other potential risk factors for electrolyte disturbances, such as diarrhea or dehydration. When performing RCI in large quantities over an extended period of time, doctors should think about using solutions with balanced electrolytes like Ringer's lactate.

The clinical implications of hyperchloremic acidosis extend beyond the disruption of acid-base balance. Acidemia can lead to electrolyte imbalances, impaired cellular function, and compromised organ systems. Therefore, prompt recognition and appropriate treatment strategies are vital to restore acid-base equilibrium and optimize patient outcomes.

Prognosis and outcomes of hyperchloremic acidosis largely depend on the underlying condition and its response to treatment. With timely intervention and appropriate management, acid-base balance can be restored, and patients can experience improved clinical outcomes and overall well-being.

Further research is warranted to explore the pathophysiology, diagnostic methods, and optimal treatment strategies for hyperchloremic acidosis. Investigating the underlying mechanisms and identifying potential novel therapeutic targets can enhance our understanding of this metabolic disorder and contribute to the development of more targeted and effective interventions.

In conclusion, hyperchloremic acidosis is a metabolic acid-base disorder characterized by increased chloride levels and decreased bicarbonate levels. Timely diagnosis, identification of the underlying cause, and appropriate treatment are crucial to restore acid-base equilibrium and improve patient outcomes. By furthering our knowledge and refining management approaches, we can enhance the care and outcomes for individuals affected by hyperchloremic acidosis.

Acknowledgement

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Conflict of Interest

None

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