Case Report

Successfully Managed Alcoholic Ketoacidosis With Sepsis Leading to Mods: A Case Report

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Successfully Managed Alcoholic Ketoacidosis with Sepsis Leading to Mods: A Case Report

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Abstract: We present the case of a 28-year-old male with a history of alcohol dependency and smoking, who presented with chest pain, shortness of breath, and altered sensorium. He exhibited severe metabolic acidosis, low platelet count, and acute kidney injury. Alcoholic ketoacidosis (AKA) was suspected due to ketonuria, metabolic acidosis, and ketonemia, compounded by electrolyte abnormalities and radiographic findings of pneumonia. Prompt intervention included electrolyte correction, thiamine supplementation, broad-spectrum antibiotics, and diuretics. Thiamine played a pivotal role in the patient's recovery, with significant improvement in consciousness observed within a day. After six days, the patient was discharged in stable condition, showing normal renal and hepatic function during follow-up. This case emphasizes the need for early recognition and comprehensive management in AKA, highlighting thiamine's crucial role in treatment success.

Keywords: alcoholic ketoacidosis; sepsis; multiple organ dysfunction syndrome; thiamine; metabolic acidosis

INTRODUCTION

Alcoholic ketoacidosis (AKA) is a condition characterized by severe metabolic acidosis, typically observed in individuals who are malnourished and have recently engaged in heavy alcohol consumption. This condition frequently occurs in individuals with a history of alcohol dependency [1]. Alcoholic ketoacidosis (AKA) usually manifests as a severe metabolic acidosis with elevated anion gap and imbalances in electrolytes. If promptly identified and managed correctly, these abnormalities can be treated. Hypoglycemic alcoholic ketoacidosis is a critical medical condition that can lead to sudden death. However, it lacks clear diagnostic criteria and is occasionally overlooked as a medical emergency [2]. The symptoms of alcoholic ketoacidosis, such as nausea, uncontrollable vomiting, and abdominal pain, can be similar to other acute crises experienced by individuals with alcohol dependence [3]. However, when AKA is properly managed, it tends to resolve quickly and fully without any lingering consequences. It's important to note that these symptoms can be misleading as they resemble those of various conditions in alcohol-dependent patients. Nonetheless, with prompt and effective treatment, AKA typically resolves without causing any long-term issues or complications.

CASE PRESENTATION

We present the case of a 29-year-old male who arrived in an altered sensorium, reporting chest pain, shortness of breath, and yellowish discoloration of mucous membranes. He exhibited restlessness, disorientation, and a history of fever lasting three days. This patient had a ten-year history of alcohol dependence and was a known smoker. He presented with a urine output of less than 30ml/hr, ultimately developing Acute Kidney Injury (AKI). Urine ketone testing yielded a positive result, and arterial blood gas (ABG) analysis revealed metabolic acidosis. Blood parameters were significantly deranged, including low platelet count, leukopenia, and abnormal PT and APTT values. Dyselectrolytemia was noted, with a potassium level of 2.8 mEq/L.

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Considering the clinical presentation, background history, and the presence of dyselectrolytemia, metabolic acidosis, and ketonemia, a diagnosis of alcoholic ketoacidosis (AKA) was suspected. Emergency treatment commenced promptly, focusing on electrolyte correction through intravenous fluids (IVF), and thiamine supplementation was initiated. Broad-spectrum antibiotics were administered due to sepsis concerns. Chest X-ray revealed bilateral consolidation with obliteration of the costophrenic angle. Diuretics were included in the treatment regimen. Over the course of the first day, thrice-daily dosing of thiamine resulted in the patient regaining consciousness. Three doses of vitamin K were administered, and urine output remained stable. Electrolyte imbalances were corrected, and blood parameters improved. On admission, his creatinine level was 4 mg/dL, but by the third day, it had returned to within the normal range. The patient continued to receive thiamine and antibiotics. An abdominal ultrasound showed hepatomegaly with grade 2 fatty liver. By the sixth day, the patient was mobilizing well, and chest auscultation revealed no abnormalities. Consequently, the patient was discharged. The post-discharge period was uneventful, and follow-up after four weeks demonstrated normal liver and kidney function tests, with no further sequelae observed.

DISCUSSION

Alcoholic ketoacidosis (AKA) is a medical emergency typically seen in individuals with a history of heavy alcohol consumption. It arises due to prolonged drinking leading to malnutrition and acute cessation of alcohol intake. Dillon et al. reported on a group of nine individuals who experienced severe ketoacidosis, despite not having diabetes mellitus, and all of them exhibited signs of prolonged and excessive alcohol consumption [4]. AKA is characterized by severe metabolic acidosis, elevated anion gap, and electrolyte imbalances. Symptoms often include nausea, vomiting, abdominal pain, and confusion. Timely intervention involves correcting electrolyte abnormalities, administering thiamine, and addressing underlying alcohol withdrawal or infections.

Broadly, alcoholic ketoacidosis leads to an elevated ratio of nicotinamide adenine dinucleotide + hydrogen (NADH) to nicotinamide adenine dinucleotide (NAD) by metabolizing ethanol. This increase inhibits liver gluconeogenesis and disrupts the oxidation of fatty acids [5]. The process of ethanol oxidation to acetaldehyde plays a key role in significantly decreasing NAD to NADH. To restore NADH back to NAD, pyruvate is converted into lactate [6]. This process ultimately results in metabolic acidosis characterized by an elevated anion gap and the presence of ketone bodies, particularly 3-hydroxybutyrate. It has been documented that the ratio of 3-hydroxybutyrate to acetoacetate is notably higher in individuals with alcoholic ketoacidosis when compared to those with diabetic ketoacidosis [7].

In cases of alcoholic ketoacidosis, insufficient food intake results in decreased glycogen storage in the liver, leading to hypoglycemia. Additionally, alcoholic ketoacidosis can lead to elevated lactate levels, with reports indicating higher lactate levels in alcoholic ketoacidosis compared to diabetic ketoacidosis. The subject in question exhibited an increased anion gap, elevated ketone bodies, particularly 3-hydroxybutyrate, and severe hypoglycemia (plasma glucose level of 25 mg/dL). Furthermore, the subject's lactate levels reached 6.27 mmol/L [2]. These findings collectively align with the diagnosis of hypoglycemic alcoholic ketoacidosis.

Various case reports were published addressing alcoholic ketoacidosis, in our study early recognition and diagnosis were critical for effective intervention. In our case, the presence of ketonuria, metabolic acidosis, ketonemia, electrolyte abnormalities, and a history of prolonged alcohol use strongly indicated AKA. Timely initiation of treatment, including electrolyte correction, thiamine supplementation, and broad-spectrum antibiotics to address potential infections, played a pivotal role in the patient's recovery. Thiamine supplementation deserves special attention as it significantly improved the patient's consciousness within a day. This underscores the importance of recognizing and addressing potential vitamin deficiencies in alcohol-dependent individuals. Furthermore, the successful management of AKI in this case underscores the resilience of the kidneys when appropriate care is provided promptly. Renal function was restored within days, indicating the potential for recovery even in severe cases.

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CONCLUSION

This case emphasizes the importance of considering AKA in alcohol-dependent patients with metabolic disturbances. Early diagnosis and a comprehensive treatment approach, including electrolyte correction, thiamine supplementation, and addressing underlying infections, can lead to favorable outcomes, preventing life-threatening complications. It also highlights the significance of follow-up care to ensure the patient's continued well-being and recovery.

Human subjects: Consent was obtained or waived by all participants in this study.

Conflicts of Interest: In compliance with the journal's uniform disclosure form, all authors declare the following:

Payment/Services Info: All authors have declared that no financial support was received from any organization for the submitted work.

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Conflict of Interest: All authors have declared that there are no conflict of interest that could appear to have influenced the submitted work.

Abbreviations

AKA: Alcoholic Ketoacidosis

MODS: Multiple Organ Dysfunction Syndrome

IND: India

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