



Successfully Managed Alcoholic Ketoacidosis with Sepsis Leading to Multiple Organ Dysfunction Syndrome: A Case Report

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Received: 11 October 2023 ♦ **Accepted:** 7 February 2024 ♦ **Published:** 30 June 2024

Citation: Roy N, Roy I. Successfully managed alcoholic ketoacidosis with sepsis leading to multiple organ dysfunction syndrome: a case report. *Folia Med (Plovdiv)* 2024;66(3):409-414. doi: 10.3897/folmed.66.e114010.

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, hypoglycemia, low platelet count, and acute kidney injury. Alcoholic ketoacidosis was suspected due to ketonuria, metabolic acidosis, and ketonemia, compounded by electrolyte abnormalities and radiographic findings of pneumonia.

Prompt intervention included intravenous fluids, 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, with normal renal and hepatic function at follow-up.

This case emphasizes the need for early recognition and comprehensive management in alcoholic ketoacidosis, highlighting thiamine's crucial role in treatment success.

Keywords

alcoholic ketoacidosis, HAKA, metabolic acidosis, multiple organ dysfunction syndrome, sepsis, thiamine

INTRODUCTION

Hypoglycemic alcoholic ketoacidosis, though rare, poses a significant emergent challenge, particularly when compared to the more prevalent hyperglycemic ketoacidosis in individuals with diabetes mellitus.^[1] Currently, diagnostic criteria for hypoglycemic alcoholic ketoacidosis are not well-defined, relying heavily on a detailed medical history that includes inquiries about alcohol consumption and dietary habits. This lack of clear criteria sometimes leads to its oversight, earning it the designation of a "forgotten medical emergency."^[2] Here, we present a case of alcoholic ketoacidosis coupled with severe hypoglycemia in an indi-

vidual who consumed excessive alcohol without adequate food intake over an extended period. 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.^[3] Alcoholic ketoacidosis 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.^[4] 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.^[5] However, when AKA is properly managed, it tends to resolve quickly and fully without any lingering consequences. It is 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 with a lean build who arrived in an emergency with an altered sensorium and fruity breath, reporting chest pain, shortness of breath, and yellowish discoloration of mucous membranes (**Fig. 1**). He exhibited restlessness, disorientation, and a history of fever lasting three days. This patient had a decade-long history of consuming up to one bottle of alcohol daily with one or two packets of bidi regularly, he had previously undergone admissions to a psychiatric ward for alcohol addiction treatment. Over a three-week period of heavy drinking with poor dietary intake, he experienced nausea and general malaise, prompting him to cease alcohol consumption 36 hours before admission. He presented with a urine output of less than 30 ml/hr with serum Cr=3.72 mg/dl and Ur=93.89 mg/dl

ultimately developing kidney failure based on RIFLE classification of acute kidney injury (AKI).^[6] Urine analysis showed reddish color and hazy transparency with positive result of ketone body (+) by dipstick test, protein (+++) hematuria (+) and 10–15 pus cell/hpf. Other blood investigations showed hypoglycemia (RBS=31 mg/dl), low platelet count, leukopenia, and abnormal PT and APTT values. Dyselectrolytemia was noted, with a potassium level of 2.9 mEq/L.

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, 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 (**Fig. 2**). Diuretics were included in the treatment regimen. Over the course of the first day (**Fig. 4**), 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, the patient's creatinine level was 3.72 mg/dl, but by the third day, it had returned to normal levels. The patient continued to receive thiamine and antibiotics. An abdominal ultrasound showed hepatomegaly with grade 2 fatty liver (**Fig. 3**). 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 un-



Figure 1. A 29-year-old patient presenting with altered sensorium and fruity breath, chest pain, shortness of breath, and yellowish discoloration of mucous membranes.

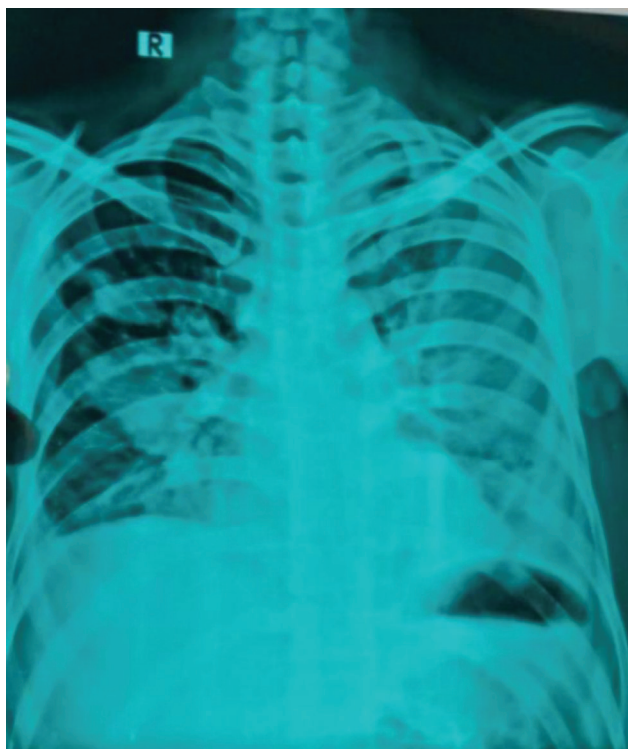


Figure 2. Chest X-ray showing bilateral consolidation with obliteration of the costophrenic angle.

eventful, and a follow-up after four weeks demonstrated normal liver and kidney function tests, with no further sequelae observed.

DISCUSSION

Alcoholic ketoacidosis 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. studied a group of nine people who developed severe ketoacidosis despite not having diabetes mellitus, and they all showed signs of prolonged and excessive alcohol consumption.^[7] AKA is characterized by severe metabolic acidosis, elevated anion gap, and electrolyte imbalances. Individuals experiencing alcoholic ketoacidosis commonly exhibit a background of prolonged, unhealthy alcohol consumption, nutritional deficiencies, and a recent episode of excessive binge drinking.^[8] 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.^[9] The process of ethanol oxidation to acetaldehyde plays a key role in sig-

nificantly decreasing NAD to NADH. To restore NADH back to NAD, pyruvate is converted into lactate.^[10] 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.^[11]

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 30 mg/dL). Furthermore, the subject's lactate levels reached 6.27 mmol/L. These findings collectively align with the diagnosis of hypoglycemic alcoholic ketoacidosis.^[4]

Various case reports were published addressing alcoholic ketoacidosis, but in our study, early recognition and diagnosis are crucial for effective intervention, as it may lead to irreversible encephalopathy.^[12] Patients with a chronic alcohol-use disorder frequently experience electrolyte imbalances, with the clinical relevance of these disturbances often contingent upon the quantity and duration of the individual's alcohol consumption.^[13] In our case, the presence of ketonuria, metabolic acidosis, ketonemia, electrolyte abnormalities, and a history of prolonged alcohol use with poor dietary intake strongly indicated AKA. The crucial aspect of our study lies in the prompt commencement of treatment, incorporating essential measures such as electrolyte correction, thiamine supplementation, and administration of broad-spectrum antibiotics to address potential infections. This approach proved instrumental in facilitating the patient's recovery within an impressive 72-hour timeframe, distinguishing our findings from those reported in other case reports.^[4,14] Thiamine supplementation deserves special attention as it significantly improved the patient's consciousness within an hour of admission comparable to other studies.^[5] 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 which otherwise can be fatal.^[15,16] The rapid progression from admission to discharge, marked by early positive outcomes, adds a unique dimension to our findings, suggesting a potentially more efficient and targeted management strategy for cases of hypoglycemic alcoholic ketoacidosis with sepsis.

CONCLUSION

This case emphasizes the importance of considering AKA in alcohol-dependent patients with metabolic disturbances. Early diagnosis and a comprehensive treatment ap-

ID ABG/

Sex M

USG OF WHOLE ABDOMEN

LIVER: Liver is enlarged in size with increased echogenicity. No focal SOL is seen. Intra hepatic biliary radicles are not dilated. Portal vein measures 10 mm in diameter at porta. Liver span measures 187 mm

GALLBLADDER: Gallbladder is normally distended with normal wall thickness. Mild sludge is seen.

C B D: Common bile duct is not dilated and measures 4 mm in diameter. No calculus is seen (visualized part).

PANCREAS : Pancreas is normal in size and echotexture. Pancreatic duct is not dilated. No focal SOL is seen. No peripancreatic collection is seen.

SPLEEN: Spleen is normal in size with homogeneous echopattern. No focal sol is seen. Splenic span is 102 mm.

RIGHT KIDNEY: Right kidney is normal in size, shape, position. Cortical echogenicity and corticomedullary differentiation are normal. No calculus or hydronephrosis is seen. Right kidney measures 118 mm.

LEFT KIDNEY: Left kidney is normal in size, shape, position. Cortical echogenicity and corticomedullary differentiation are normal. No calculus or hydronephrosis is seen. Left kidney measures 103 mm.

URETERS: Ureters are not visualized, hence not dilated.

URINARY BLADDER: Urinary bladder is normally distended normal wall thickness. No SOL or calculus is seen in the lumen.

PROSTATE : It is normal in for age.

- No retroperitoneal lymph node enlargement is seen.
- No ascites is seen.

IMPRESSION:

- 1) **Hepatomegaly with grade II fatty changes.**
- 2) **Mild sludge in gallbladder.**

Suggested – Other investigations and clinical correlation.

Figure 3. Abdominal ultrasound: diagnosis – hepatomegaly with grade 2 fatty liver.

proach, 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.

Disclosures

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

All authors have declared that no financial support was received from any organization for the submitted work.

Financial relationships

All authors have declared that they have no financial rela-

tionships at present or within the previous three years with any organizations that might have an interest in the submitted work.

Conflict of Interest

All authors have declared that there are no conflicts of interest that could appear to have influenced the submitted work.

Acknowledgements

We gratefully acknowledge the Lions Club Mother and Child Nursing Home team for their outstanding patient care and efforts in managing such a critical patient.

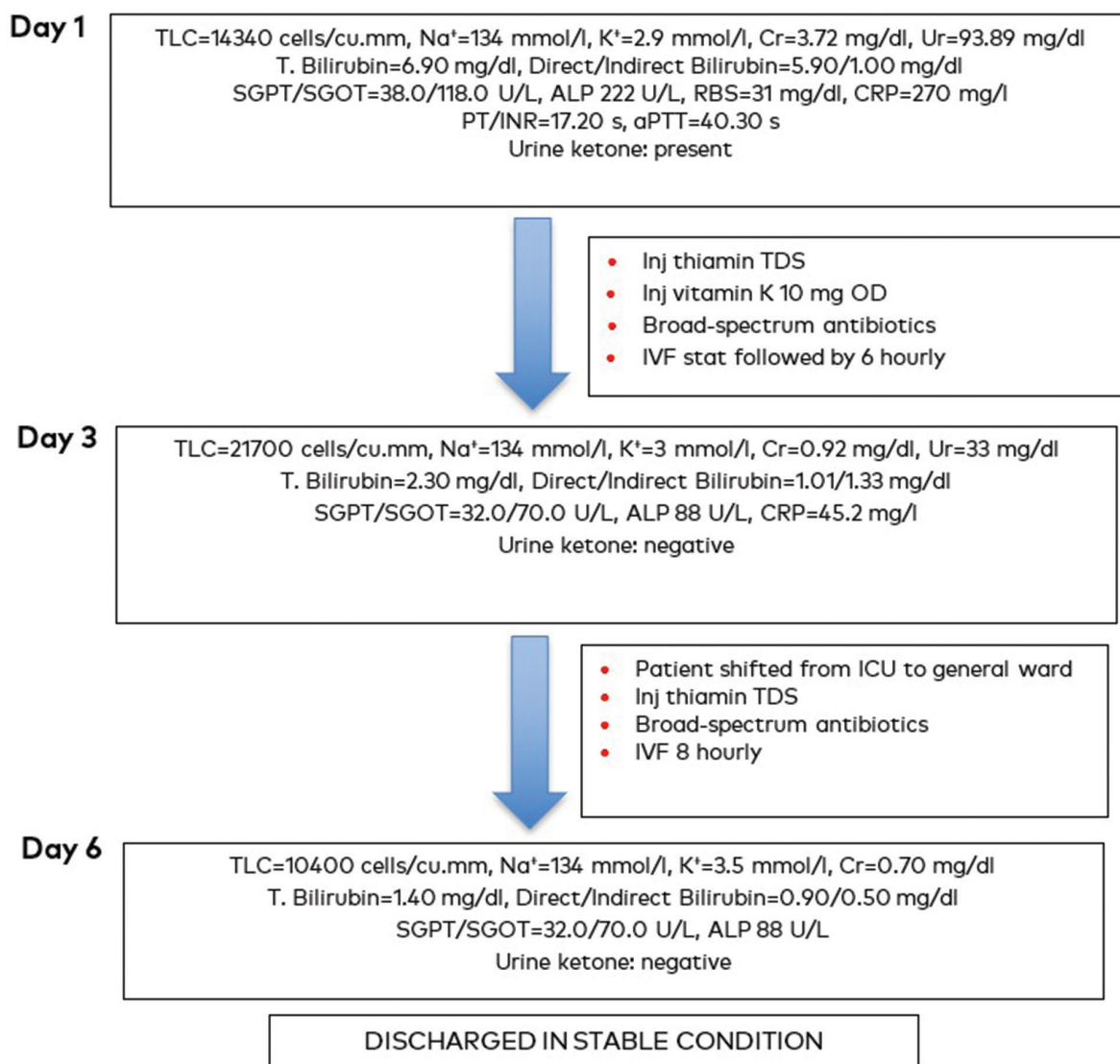


Figure 4. Treatment flowchart.

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Успешное лечение алкогольного кетоацидоза с сепсисом, приведшим к синдрому полиорганной недостаточности: отчёт о клиническом случае

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Дата получения: 11 октября 2023 ♦ **Дата приемки:** 7 февраля 2024 ♦ **Дата публикации:** 30 июня 2024

Образец цитирования: Roy N, Roy I. Successfully managed alcoholic ketoacidosis with sepsis leading to multiple organ dysfunction syndrome: a case report. *Folia Med (Plovdiv)* 2024;66(3):409-414. doi: 10.3897/folmed.66.e114010.

Резюме

Мы представляем случай 28-летнего мужчины с историей алкогольной зависимости и курения, у которого были боли в груди, одышка и нарушения работы нервной системы. У него наблюдался тяжёлый метаболический ацидоз, гипогликемия, низкий уровень тромбоцитов и острое повреждение почек. Алкогольный кетоацидоз был заподозрен из-за кетонурии, метаболического ацидоза и кетонемии, усугублённых нарушениями электролитного баланса и рентгенологическими признаками пневмонии.

Немедленное вмешательство включало внутривенное введение жидкостей, коррекцию электролитного баланса, добавление тиамин, антибиотики широкого спектра действия и диуретики. Тиамин сыграл ключевую роль в выздоровлении пациента, при этом значительное улучшение сознания наблюдалось в течение дня. Через шесть дней пациент был выписан в стабильном состоянии с нормальной функцией почек и печени при последующем наблюдении.

Этот случай подчёркивает необходимость раннего распознавания и комплексного лечения алкогольного кетоацидоза, подчёркивая решающую роль тиамин в успехе лечения.

Ключевые слова

алкогольный кетоацидоз, метаболический ацидоз, синдром полиорганной недостаточности, сепсис, тиамин