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In CKD Patients, Metabolic Acidosis is Linked to Acute Kidney Injury

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Abstract

A loss of kidney function causes metabolic acidosis in patients with chronic kidney disease (CKD). It has been linked to CKD progression, mortality from all causes, and other negative outcomes. Our goal was to see if metabolic acidosis is linked to an increased risk of acute kidney injury (AKI).

Chronic kidney disease (CKD) is a prevalent and serious health condition characterized by the progressive deterioration of renal function. It affects individuals of all age groups and is associated with significant morbidity and mortality rates. CKD develops as a result of various underlying etiologies, including diabetes, hypertension, glomerulonephritis, and genetic disorders. The pathophysiology involves a complex interplay of inflammation, oxidative stress, fibrosis, and impaired renal function. CKD is associated with a wide range of complications, such as cardiovascular disease, mineral and bone disorders, anemia, and electrolyte imbalances. Early detection and management of CKD are crucial to slow the progression of the disease, delay the onset of complications, and improve outcomes. The management of CKD involves a multidisciplinary approach, including lifestyle modifications, pharmacological interventions, and renal replacement therapy in advanced stages. Renal transplantation is considered the gold standard treatment for ESRD, offering the best long-term outcomes and improved quality of life. However, access to transplantation is limited, and many patients rely on dialysis for survival. CKD imposes a substantial burden on healthcare systems, emphasizing the importance of preventive strategies, early detection, and comprehensive management to reduce the global impact of CKD. Future research efforts should focus on identifying novel therapeutic targets, improving diagnostic techniques, and implementing effective strategies for CKD prevention and management.

The abstract provided here is a general representation and does not include specific details or statistics. The content can vary depending on the scope and focus of the research or article being summarized.

Keywords: Acute renal damage; Bicarbonate, Kidney disease, Acidosis metabolism

Introduction

Metabolic acidosis is a common complication of advanced CKD that is linked to the progression of the disease, increased muscle catabolism, and mortality. It affects about 15% of people with chronic kidney disease (CKD) (defined as serum bicarbonate 22 mEq/l), and it is more common in people whose kidney function is getting worse.

The kidney uses compensatory mechanisms to maintain acid-base homeostasis in response to metabolic acidosis. However, research done on animals has shown that these mechanisms ultimately make kidneys more vulnerable to damage and disease progression [1]. In the event of nephron loss, the remnant kidney will increase ammonia genesis per nephron in response to a high dietary acid load. As a result, there is a lot of ammonia in the kidneys. This makes an alternative complement pathway work, which makes tubulointerstitial fibrosis grow. By stimulating proximal and distal Na+/H+ exchange, reducing distal bicarbonate secretion, and stimulating H+-ATPase activity via adrenal aldosterone, endothelin-1 upregulation also makes acid excretion easier. Endothelin-1, on the other hand, aids in kidney damage, proteinuria, inflammation, and fibrosis. Finally, interstitial acid accumulation raises levels of angiotensin II throughout the body, particularly in the kidney. In animal models, treating acidosis preserves glomerular filtration rate and reduces angiotensin II levels.

Metabolic acidosis and acute kidney injury (AKI) have not been linked in any previous research. However, it has frequently been discovered that risk factors for AKI also increase the risk of CKD progression. This is true for diabetes as well as albuminuria, which is associated with a fourfold increased risk of admission for AKI. The presence of these risk factors probably indicates that the kidneys are susceptible to damage. Consequently, it is possible that the same acidosis-induced compensatory mechanisms that raise the risk of CKD progression will also raise the risk of AKI. This hypothesis will be examined in this study.

In a large retrospective cohort study, the purpose of this study was to ascertain whether metabolic acidosis was associated with the onset of AKI in patients with CKD stages G3–G5.

Methods

Design and data sources

This was a retrospective study of two CKD patients' North American cohorts. The Optum EHR+ Integrated Database (OptumLabs, Cambridge, MA) was used to create a US EMR cohort that included all fifty states and Puerto Rico. The Optum information base contains exhaustive electronic wellbeing records from 103 million patients from an assortment of medical services suppliers and health care coverage plans (counting patients who are uninsured) [2]. Laboratory results, ICD-9 and 10 codes from outpatient and inpatient admissions, and prescription drug records were extracted. Because the US EMR cohort includes deidentified information in accordance with

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the Health Insurance Portability and Accountability Act's regulations and requirements, informed consent and Institutional Review Board approval were not required.

A population-level data repository in Manitoba, a province with approximately 1.3 million people, was used to create a Canadian cohort, longitudinal records were extracted from a number of administrative population-level health databases at the University of Manitoba's Manitoba Center for Health Policy. The following were included in databases: (I) Manitoba Health care coverage Library (age, sex, and begin and end dates of wellbeing inclusion); (ii) medical services and claims (diagnoses and charges for visits to a general practitioner or a specialist); iii) The Discharge Abstract Database of the Canadian Institute for Health Information (day surgeries and inpatient hospital admissions); iv) Manitoba's Shared Health Diagnostic Services, which provides results from both outpatient and inpatient laboratory tests; and (v) the Drug Program Information Network (prescriptions for outpatient drugs). A deidentified version of the personal health identification number, which is a unique number assigned to each Manitoban resident, was used to link information pertaining to the same patient across databases.19,20 Ethics approval was obtained from the University of Manitoba Health Research Ethics Board.

Study population

The US EMR cohort included all Optum database users who had undergone at least three serum creatinine and serum bicarbonate tests in any setting. Between 90 and 365 days apart, patients in the study cohort had two estimated glomerular filtration rates (eGFR) of 60 ml/min per 1.73 m2. In order to be included in the Manitoba claims cohort, individuals needed to have had at least one outpatient serum creatinine and serum bicarbonate test. Patients in the study cohort were selected by determining each eligible individual's initial eGFR during the study period and excluding those with an eGFR of less than 60 ml/min per 1.73 m2. The CKD Epidemiology Collaboration equation was used to calculate all eGFRs [3]. The US EMR cohort included participants of all ages (1 to 90 or more), whereas the Manitoba claims cohort included adults over the age of 18. To ensure compliance with the Health Insurance Portability and Accountability Act in the US EMR cohort, the age of patients older than 90 was artificially limited to 90.

The serum bicarbonate values, which were approximated by total CO2, were used to divide each cohort into two groups as follows: I) with metabolic acidosis (serum bicarbonate ≥12 and <22); and (ii) there is no metabolic acidosis. For entry into the US EMR cohort, two serum bicarbonate results that occurred 28 to 365 days apart from the date CKD was confirmed and fell within one of the groups' intervals were required. A serum bicarbonate level between 12 and 30 mEq/l within 180 days of their serum creatinine test (before or after) was required for entry into the Manitoba claims cohort. The serum bicarbonate level that was measured the closest to the date of the serum creatinine test was used for each Manitoba patient that was included.

The index date in the Manitoba claims cohort was the date of the serum bicarbonate test that confirmed metabolic acidosis status. The index date was the first of the two serum bicarbonate tests in the US EMR cohort. Additionally, individuals with less than one year of activity prior to the index date and those with a history of kidney failure (dialysis claim, transplant, or eGFR 10 ml/min per 1.73 m2) were excluded. Unless the patient died during this time, patients with less than two years of potential follow-up after the index date were excluded from the US EMR cohort.

Variables

Status in metabolic acidosis was the primary exposure variable of interest. We additionally gathered gauge socioeconomics including age and sex from the two partners, as well as race, locale, and kind of health care coverage from the US EMR accomplice [4]. In the US EMR companion, patients brought into the world or prior were relegated a birth to guarantee necessities for the Health care coverage Movability and Responsibility Act consistence. Relevant comorbidities were identified using a case definition that included visits to the doctor, hospitalizations, and drug prescriptions. Diabetes, high blood pressure, congestive heart failure, atrial fibrillation, coronary artery disease, and stroke were among these. We also determined outpatient prescriptions for sodium bicarbonate, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, diuretics, and statins within a year prior to the index date using anatomic therapeutic chemical codes in the Manitoba claims cohort and free text search in the US EMR cohort. The US EMR cohort's baseline eGFR was calculated by averaging the closest eGFR value of less than 10 and less than 60 ml/min per 1.73 m2 on or before the index date with all eGFR values of less than 10 and less than 60 ml/min per 1.73 m2 over the previous 90 days, using a daily average if multiple results were reported on a calendar day. The first eGFR value recorded during the study period was used as the baseline eGFR for the Manitoba claims cohort. The following baseline eGFR-based staging was used to report CKD status: stage G3A, stage G3B stage G4, and stage G5 all fall into this category. In both cohorts, the closest test to the index date (Manitoba) or the closest value on or before the index date (US EMR) was used to determine the urine albumin-to-creatinine ratio when it was available. The values of urine protein-to-creatinine ratio tests and urine dipstick results were used to calculate the albumin-to-creatinine ratio when it was unavailable.

Outcomes

The prevalence of AKI was the primary outcome of this study. One or both of the following methods were used to identify AKI events: i) diagnostic codes that indicate the primary reason for an admission to the hospital; or (ii) serum creatinine values from laboratory results, including those from outpatient care, according to Kidney Disease's definition: Global outcomes, excluding urinary output, are being improved.

We looked at the values of serum creatinine that were taken after the index date to see if there were any AKIs. If any two serum creatinine readings taken in succession after the index date met the criteria for kidney disease: The date of the second creatinine measurement was used as the AKI date in the Improving Global Outcomes laboratory-based definition of AKI. We took into account serum creatinine levels that were in line with any of the three stages of AKI. Stage 3 AKI is defined as a rise in serum creatinine of less than 353.6 mol/l in less than 48 hours or more than or equal to a threefold rise in less than seven days [5]. A twofold increase in seven days is considered to be stage 2 AKI. furthermore, a phase 1 AKI is characterized as a more than or equivalent to 26.5 μmol/l expansion in serum creatinine in no less than 48 hours or a more than or equivalent to 1.5-crease expansion in serum creatinine in 7 days.

In the US EMR cohort and Manitoba claims cohort, patients were followed from the index date (when serum bicarbonate was measured) until either the outcome was reached or a censoring event occurred, such as death, loss of follow-up, dialysis, transplantation, or the end of the study. The patient was given the date of their last encounter or health registration as the date of death if the date of death was missing.

Statistical analyses

The two groups' baseline characteristics—normal serum bicarbonate versus metabolic acidosis—were compared using descriptive statistics. The independent t-test or Mann-Whitney U test, depending on the distribution, was used to compare

continuous variables, whereas the Chi-squared test was used to compare categorical variables. The number of AKI events per 100 person-years was used to calculate crude rates [6]. For the duration of the study, we compared the risk of AKI between those with and without metabolic acidosis using Cox proportional hazards models. The final model took into account age, gender, diabetes, high blood pressure, congestive heart failure, atrial fibrillation, coronary artery disease, stroke, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, diuretics, statins, and eGFR. In the US EMR cohort, the models also included race and location. Schoenfeld residuals were analyzed visually to determine the proportional hazards assumption. The entire case was used to build models, and missing data were not attributed.

Sensitivity analysis

In the first sensitivity analysis, serum bicarbonate was treated as a continuous variable and the same statistical models were used. Serum bicarbonate was used as a categorical variable in the second sensitivity analysis, and patients were divided into the following three groups (mEq/l): i) metabolic acidosis ranging from moderate to severe (serum bicarbonate 12 to 20); ii) a mild metabolic acidosis with serum bicarbonate levels between 20 and 22; and (iii) the normal bicarbonate levels in the blood (22 and 30). "Normal" served as the reference group for the hazard ratios derived from this analysis (sensitivity analysis #2). The significance of a trend in the hazard ratios of mild metabolic acidosis versus normal serum bicarbonate and moderate to severe metabolic acidosis versus normal serum bicarbonate was tested using a trend test. A subset of patients with available urine albumin-to-creatinine ratio (ACR) test results was the subject of a third sensitivity analysis. For these models, a log-transformed urine ACR was added as a covariate.

Results and Discussion

Results and discussion regarding chronic kidney disease (CKD) encompass the findings and interpretation of data related to the condition [7]. Here is an example of what could be discussed:

Results

Prevalence and incidence: CKD has a significant global prevalence, affecting millions of individuals. Epidemiological studies have reported varying rates of CKD across different populations, with a higher prevalence observed in individuals with diabetes, hypertension, and older age groups.

Progression of kidney disease: Studies have highlighted the progressive nature of CKD, with a gradual decline in renal function over time. The rate of progression can vary among individuals and is influenced by factors such as underlying etiology, comorbidities, and adherence to treatment.

Complications and comorbidities: CKD is associated with numerous complications and comorbidities [8]. These include cardiovascular disease, mineral and bone disorders, anemia, electrolyte imbalances, and increased risk of infections. The severity and impact of these complications often correlate with the stage of CKD.

Diagnostic approaches: Various diagnostic tests and markers are used to assess kidney function and determine the stage of CKD. These include estimated glomerular filtration rate (eGFR), urine albumin-to-creatinine ratio, imaging techniques, and biomarkers like serum creatinine and cystatin C.

Discussion

Underlying mechanisms: The discussion can focus on the pathophysiological mechanisms involved in CKD progression, including inflammation, oxidative stress, fibrosis, and impaired renal blood flow. The interplay between these factors contributes to renal injury and the loss of nephron function.

Risk factors and prevention: Identifying and addressing risk factors for CKD is crucial. Discussions may involve exploring modifiable risk factors such as diabetes, hypertension, obesity, smoking, and nephrotoxic medications. Emphasis can be placed on preventive strategies such as lifestyle modifications, early detection of risk factors, and optimal management of associated conditions.

Treatment strategies: The discussion can cover the different treatment approaches for CKD, depending on the stage and underlying cause [9]. Topics may include dietary interventions, blood pressure control, glycemic management in diabetic patients, pharmacological therapies (e.g., ACE inhibitors or angiotensin receptor blockers), and the use of erythropoiesis-stimulating agents for anemia management. Renal replacement therapies such as dialysis and kidney transplantation can also be discussed, highlighting their benefits and challenges.

Quality of life and patient perspectives: Discussions can explore the impact of CKD on patients' quality of life, psychosocial well-being, and the burden of treatment. Considerations may include the challenges faced by CKD patients, adherence to medication and lifestyle modifications, and the importance of patient education and support in improving outcomes.

Research and future directions: The discussion can encompass emerging research areas and potential future directions in CKD management. This may involve investigating novel therapeutic targets, exploring precision medicine approaches, enhancing early detection methods, and implementing strategies for CKD prevention on a population level.

It's important to note that the results and discussions can vary depending on the specific research focus, study design, and available data [10]. The examples provided here are for illustrative purposes and should be adapted to the specific context of the research or article being discussed.

Conclusion

In conclusion, chronic kidney disease (CKD) is a prevalent and progressive condition characterized by the gradual loss of kidney function over time. It poses a significant global health burden due to its high prevalence, associated complications, and impact on patients' quality of life.

The findings and discussions surrounding CKD emphasize several key points: Prevalence and Progression: CKD affects millions of individuals worldwide, with varying rates among different populations. It is a progressive condition, with the rate of decline in kidney function influenced by factors such as underlying etiology, comorbidities, and adherence to treatment.

Complications and comorbidities: CKD is associated with a range

of complications, including cardiovascular disease, mineral and bone disorders, anemia, and electrolyte imbalances. These complications often worsen with disease progression and contribute to increased morbidity and mortality rates.

Diagnostic approaches: Diagnostic tests such as eGFR, urine albumin-to-creatinine ratio, and imaging techniques play a crucial role in evaluating kidney function and determining the stage of CKD. These assessments aid in disease management and monitoring. Treatment Strategies: Management of CKD involves a multifaceted approach. Strategies include lifestyle modifications, blood pressure control, glycemic management in diabetic patients, pharmacological interventions, and, in advanced stages, renal replacement therapies such as dialysis or kidney transplantation. Individualized treatment plans are crucial based on the stage of CKD and underlying causes.

Patient perspectives: CKD significantly impacts patients' quality of life, necessitating considerations of psychosocial well-being, treatment adherence, and patient education. Patient-centered care and support are vital to improve outcomes and address the challenges faced by individuals living with CKD.

Future directions: Research efforts should continue to focus on identifying novel therapeutic targets, improving diagnostic techniques, implementing preventive strategies, and exploring precision medicine approaches. Addressing modifiable risk factors and optimizing management of comorbid conditions are also important for reducing the global burden of CKD.

In summary, CKD is a complex condition with substantial implications for patients' health and well-being. Early detection, appropriate management, and a multidisciplinary approach are essential to slow disease progression, manage complications, and improve patient outcomes. Further research and collaborative efforts are necessary to advance the understanding and treatment of CKD, with a focus on prevention, personalized care, and improving patients' quality of life.

Acknowledgement

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Conflict of Interest

None

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