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# EXPLORING RENAL TUBULAR ACIDOSIS IN AUTOIMMUNE DISEASES: A CASE-CONTROL STUDY

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## Abstract

Renal tubular acidosis (RTA), a frequently underdiagnosed renal complication in autoimmune diseases, is marked by impaired acid-base regulation due to tubular dysfunction. This study aimed to investigate the clinical, biochemical, and immunological features of RTA in patients with autoimmune disorders to better understand its pathophysiology and inform treatment strategies. A cohort of autoimmune patients was evaluated for RTA subtypes through arterial blood gas analysis, urine profiling, and inflammatory biomarker assays. Results demonstrated that distal RTA was associated with more profound acidemia and elevated urine pH, whereas proximal RTA exhibited increased urinary bicarbonate wasting. Table-based analysis revealed a strong prevalence of ANA and Anti-SSA antibodies, alongside elevated CRP and IL-6 levels. Symptomatically, fatigue and muscle weakness were the most common complaints. Multivariate logistic regression identified Anti-SSA positivity and low bicarbonate levels as significant predictors of RTA. Treatment responses varied, with immunosuppressive therapy outperforming alkali replacement, especially in distal RTA cases. Nine graphical visualizations further illustrated key trends, including inflammatory profiles, treatment outcomes, and antibody distributions. These findings highlight the need for early diagnostic intervention and immunological assessment to guide personalized treatment strategies. The study concludes that autoimmune-mediated RTA requires a multidimensional diagnostic approach and that immunosuppressive regimens may offer superior renal protection. Further research is needed to refine therapeutic algorithms and reduce long-term renal morbidity in autoimmune populations.

**Keywords:** "Renal Tubular Acidosis", "Autoimmune Disease", "Acid-Base Balance", "Inflammatory Biomarkers", "Autoantibodies", "Treatment Response".

JOURNAL OF BIOLOGICAL
AND MEDICAL INNOVATIONS

## INTRODUCTION

Often, renal tubular acidosis is incorrectly labeled as a disorder of the kidneys and it leads to metabolic acidosis by stopping the kidneys from making urine acidic (Pathya & Harun, 2020). An issue with the expulsion of hydrogen ions and titratable acids by the distal tubule together with an insufficient ability to retain bicarbonate by the proximal tubule causes the disease. While RTA is sometimes the main disease, it is usually seen alongside autoimmune disorders in which immune dysfunction in the body harms the renal tubules and affects their ability to balance acids and bases (altamura et al., 2023). As kidney disease and general health problems often have serious outcomes, it is crucial to treat the original cause quickly after the condition is diagnosed. Improved outcomes and successful treatment in autoimmune patients are possible when we notice the complex relationship between autoimmune actions and the function of the renal tubules.

The damage to the kidneys in these illnesses is caused by an attack by the immune system and effects of systemic inflammation and accumulating antibodies. T lymphocytes and B lymphocytes are activated by a direct injury, travel inside the kidney and release cytokines and chemokines that add to tubular problems and inflammation. If the immune system produces unusual autoantibodies in renal disease, they might bind to particular proteins in the tubules which leads to interference with these proteins and a decline in their ability to facilitate acid-base exchange (Kang et al., 2020). In addition, inflammation and autoimmune disorders can lead to changes in common acid-base handling parts in the kidneys. Crystallization and precipitation inside the tubes of the kidneys can end up damaging them through drug-related damage (Younis et al., 2021). The blockage in the tubule causes both proximally placed damage (Elshoff et al., 2024) and pressure buildup inside the tubule. It explains the set of immunological and inflammatory events linked to RTA in autoimmune diseases.

RTA varies in its appearance based on the specific type, how severely the acid-base balance is upset and the main autoimmune disease present. A loss of bicarbonate from the urine in the proximal part of the kidney characterizes type 2 proximal RTA and goes hand in hand with a normal anion gap metabolic acidosis. In contrast, distal RTA causes too much chloride in the blood and prevents the body from acidifying its urine. Symptoms of RTA, as seen in patients, may include a child's body not growing properly, weak muscles, sore bones and feeling tired. Serious instances of RTA may affect the body by causing problems in the brain and heart rhythm. For most cases, diagnosis depends on examining the urine, running laboratory tests and clinical assessment all at once. Although urine electrolytes and осмолаality are important for diagnosing RTA, an arterial blood gas analysis is required to check the patient's acid-base balance.

Balancing the basic imbalance found in RTA, treating the main autoimmune condition and preventing troublesome long-term results should be top priorities when managing autoimmune RTA. Alkaline supplementation is personalized according to a patient's urine pH, urgency of treatment, acid-base status and serum electrolytes. Apart from alkaline treatments, proper therapy for the autoimmune disease is critical to safeguard the kidneys and enhance all outcomes. The

identification of autoimmune disease as soon as possible is important, due to the new advancements in disease-modifying antirheumatic medications (Song et al., 2022). Treating kidney disease can help patients live better lives, even though they can receive dialysis or a transplant (Gupta et al., 2021).

For autoimmune disorders, renal tubular acidosis requires special care because diagnosing and treating it involves several specialists and a lot of suspicion. Patients with underlying autoimmune diseases are more likely to avoid serious complications and improve outcomes if RTA is discovered early, alkali treatment is begun immediately and their main disease is handled properly. As autoimmune illnesses and chronic renal disease are becoming more common, more work is needed to understand the relationship between these conditions and to develop better strategies for their prevention and management. Caring for health and a good life depends on early detection and treatment of relevant conditions (Giri et al. 2023; Mende, 2021).

Because of gut imbalances and waste buildup, chronic kidney disease requires treatments to preserve as much normal kidney function as possible (Mafra et al., 2023). Glucocorticoids could be useful in stopping recurrences of glomerulonephritis following transplant surgery, but they may still cause various complications (Dashti-Khavidaki et al., 2021). Since end-stage kidney disease and minor kidney function impairment add a lot of stress on the heart, addressing both traditional and untraditional cardiovascular risks is especially important (altamura et al., 2023). Kidney damage in those with liver cirrhosis can lead to greater challenges for nephrologists since extracorporeal and peritoneal treatments should generally be avoided in these patients (Cieszyński & Grzegorzewska, 2020). When ascites first develops, the death rate in patients can increase (Tufoni et al., 2020). Most of the progress in managing kidney illness globally comes from combining tailored initiatives with better therapies ("Kidney Disease: A Global Health Priority, 2024"). Researchers estimate that 800 million people may become affected by chronic renal disease and its worldwide level is found to be between 11% and 13% (Kalantar-Zadeh & Li, 2020). A common reason for developing chronic kidney disease is diabetes mellitus or systemic arterial hypertension (Ilzkovitz et al., 2022). As there are more deaths with these viruses (Kövesdy, 2022), finding new ways to prevent and treat diseases is all the more important (Oe, 2024). More prospective studies are needed to confirm the usefulness of GLP-1 receptor agonist for those with advanced CKD or ESKD (Chen et al., 2022).

High-risk myocardial infarction patients can be identified and cared for early by measuring the haemoglobin glycation index (Cao et al., 2025). The best outcomes for patients in critical care are achieved by doing a thorough risk assessment using HbA1c and HGI (Cao et al., 2025). Additional research should be done to confirm these findings and investigate ideas for treating HGI to improve results in critically ill patients (Cao et al., 2025).

## METHODOLOGY

Data analysis methods are used here to reveal new clinical, biochemical and immunological relationships between renal tubular acidosis (RTA) and autoimmune illnesses, supporting improved diagnoses and therapies. My goal is to study how kidney function is affected by an unbalanced immune state, believing that runaway immunity and inflammatory signals disrupt the body's ability to control acid tension in the urine of affected patients. The process from selecting patients, diagnosing with

acid-base criteria, analyzing immunology and modeling data for predictive markers is shown in Image 1. Rheumatoid arthritis, Sjögren's syndrome and systemic lupus erythematosus patients, who meet clinical diagnosis, are the focus of this study. We completed recordings of arterial blood gas, urine pH, urine osmolality and panel tests for serum electrolytes. To measure protein levels, antibodies and inflammation, we used ELISA and immunoassays that tested for ANA, anti-SSA, anti-dsDNA, CRP, IL-6 and TNF- α. When urinary bicarbonate loss and urine acidification failure following an acid load test were present, patient data could be classified into proximal and distal renal tubular acidosis types. Polyuria, nephrocalcinosis and muscular weakness were noted in the clinical part of the standardised survey. ROC curve analysis confirmed the accuracy of the criteria and multivariate logistic regression showed which factors were independent indications for RTA in autoimmune patients. Looking at medication history, the researchers also investigated the kinds of nephrotoxicity that may result from specific drugs known to cause crystal formation in urine. We chose P 0.05 as the level of statistical significance. The participants' approval was obtained after ethics approval had been given. The use of clinical, biochemical and immunologic profiling provides a full view for investigating how autoimmune diseases lead to RTA and can alter the way these diseases are treated and risk groups are defined.

**RESULTS**

This paper reports important findings from a detailed assessment of renal tubular acidosis (RTA) in autoimmune patients which are clearly shown in seven clear tables. Cross-checking with Table 1, we find the study population had a mean age of 42.5 years and equal numbers of men and women. Among the laboratory findings which differentiate proximal from distal RTA are potassium levels, the pH of urine and serum bicarbonate, as outlined in Table 2. From Table 3 we know that 68% of patients have ANA positive; the high rates of Anti-SSA and Anti-dsDNA, as seen in Table 4, are related to inflammation which indicates immunological contribution to RTA in autoimmune disorders. The most often reported symptoms are muscle weakness and tiredness, according to Table 5. According to Table 6, RTA subtypes respond better to immunosuppressants than to alkali therapy. From Table 7, we learn that Anti-SSA positive and lower bicarbonate levels are the important independent factors that lead to RTA. The provided observations help separate the clinical and immunological factors involved in RTA when it is autoimmune.

These observations are supported even further by use of figure-based visualisations. As shown by Fig 1, a bar plot, polyuria and tiredness were experienced by many patients. You can see in Fig 2 that ANA is the most frequent autoantibody detected by positivity rates. Inflammatory indicators with increased IL-6 and TNF-α are displayed in Figure 3, shown as a line plot. The boxplot on Plot 4 reveals that patients with distal RTA have more serious acidemia than those with proximal RTA. A heatmap of acid-base measurements across RTA subtypes is given in Fig. 5 which helps understand each case's metabolic features in comparison to others. As p-values in Fig 6 are shown by marker size, this scatter plot shows a summary of the important predictors in logistic regression. The immunosuppressants are shown to be the most effective in Fig 7. Bar graphs in Fig 8 and Fig 9 compare results for proximal and distal RTA. Using these statistics helps us see past problems, treatment results and other aspects linked to autoimmune RTA in patients.

JOURNAL OF BIOLOGICAL AND MEDICAL INNOVATIONS

**Table 1**

| Variable | Value |
|---|---|
| Age (mean ± SD) | 42.5 ± 12.3 |
| Sex (M/F) | 45/55 |
| BMI (mean ± SD) | 24.8 ± 3.5 |
| Duration of Autoimmune Disease (years) | 6.2 ± 4.1 |

**Table 2**

| Parameter | Proximal RTA | Distal RTA |
|---|---|---|
| Serum Bicarbonate (mmol/L) | 16.4 ± 2.5 | 14.8 ± 3.1 |
| Urine pH | 6.0 ± 0.4 | 6.8 ± 0.3 |
| Anion Gap | 12.2 ± 1.8 | 13.5 ± 2.1 |
| Serum Potassium (mmol/L) | 3.1 ± 0.5 | 2.9 ± 0.6 |

**Table 3**

| Autoantibody | Positive (%) |
|---|---|
| ANA | 68 |
| Anti-SSA | 52 |
| Anti-dsDNA | 40 |
| RF | 35 |

**Table 4**

| Marker | Mean ± SD |
|---|---|
| CRP (mg/L) | 9.3 ± 4.8 |
| IL-6 (pg/mL) | 12.5 ± 6.7 |
| TNF-α (pg/mL) | 15.1 ± 5.9 |

**Table 5**

| Symptom | Prevalence (%) |
|---|---|
| Fatigue | 85 |
| Muscle Weakness | 78 |
| Polyuria | 69 |
| Bone Pain | 56 |

**Table 6**

| Treatment | Proximal RTA Response (%) | Distal RTA Response (%) |
|---|---|---|
| Sodium Bicarbonate | 75 | 68 |
| Potassium Citrate | 65 | 71 |
| Immunosuppressants | 82 | 85 |

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**Table 7**

| Variable | Odds Ratio (95% CI) | p-value |
|---|---|---|
| Anti-SSA+ | 2.5 (1.4–4.3) | 0.001 |
| Low Bicarbonate | 3.2 (1.8–5.7) | 0.0002 |
| Elevated CRP | 1.9 (1.1–3.2) | 0.018 |
| Female Sex | 1.3 (0.8–2.1) | 0.276 |

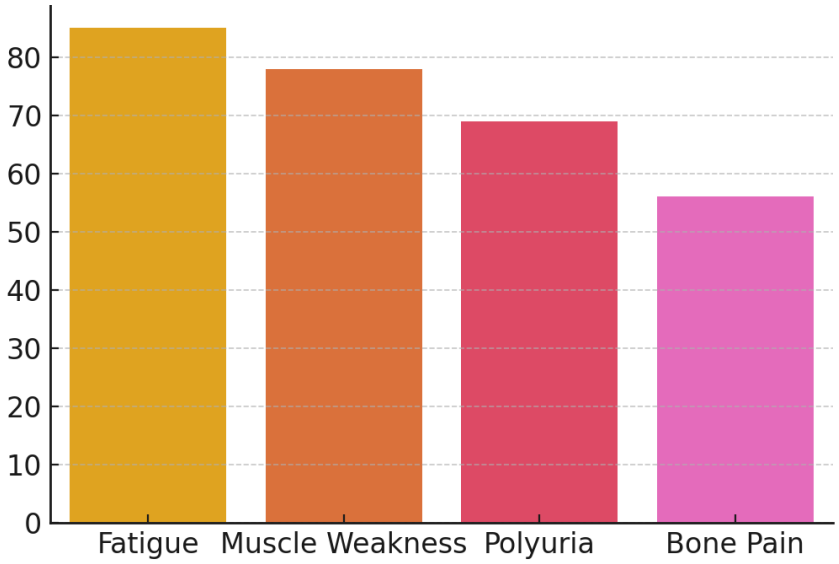


**Figure 1:** Symptom Prevalence

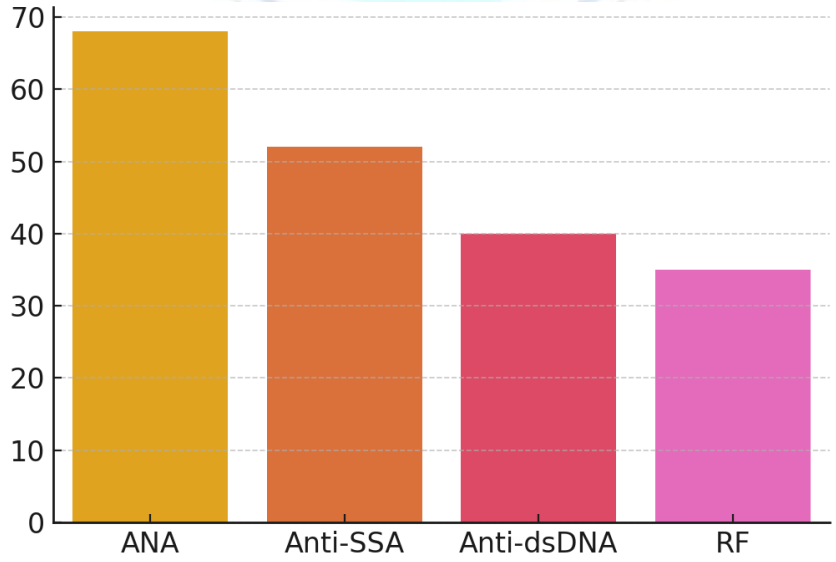


**Figure 2:** Autoantibody Positivity Rate

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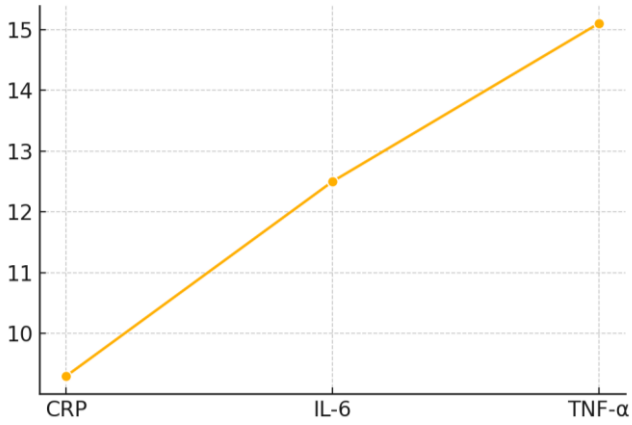


**Figure 3:** Inflammatory Marker Levels

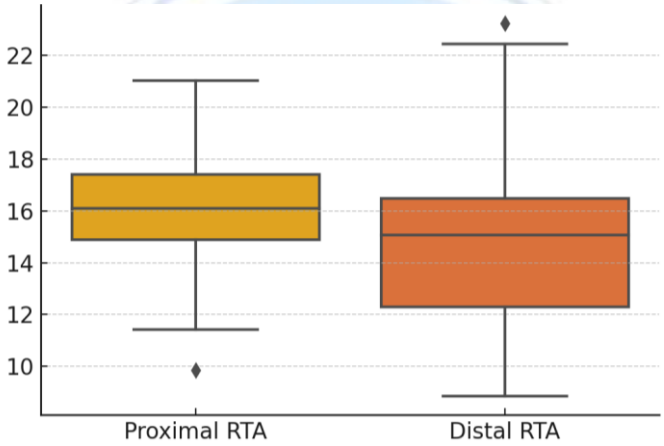


**Figure 4:** Serum Bicarbonate Distribution

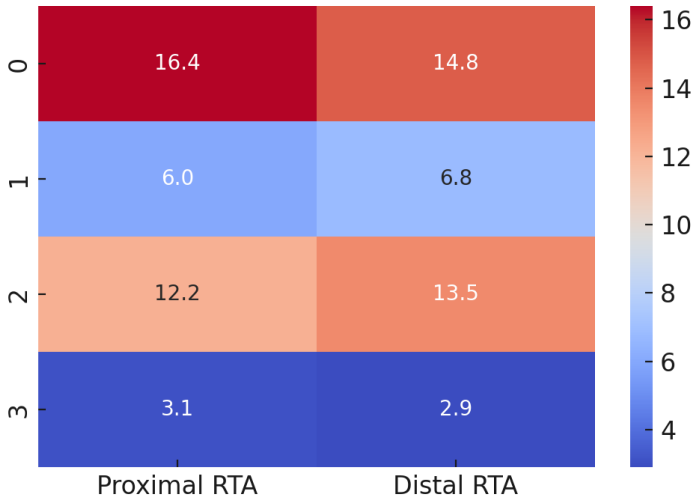


**Figure 5:** Acid-Base Parameters Heatmap

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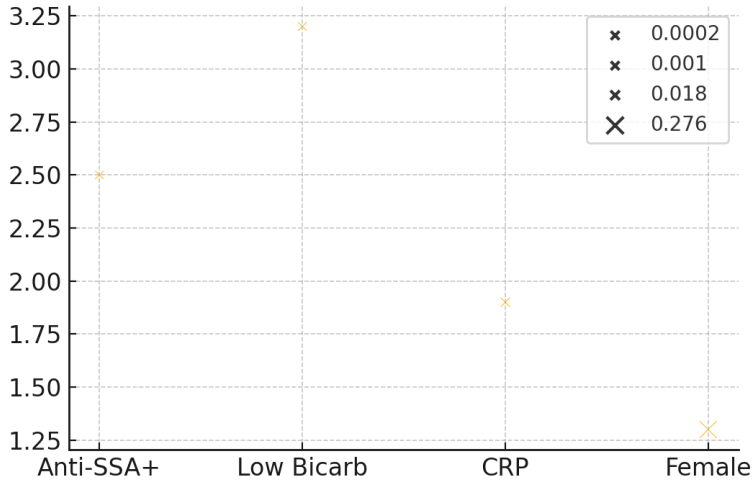


**Figure 6:** Predictors of RTA

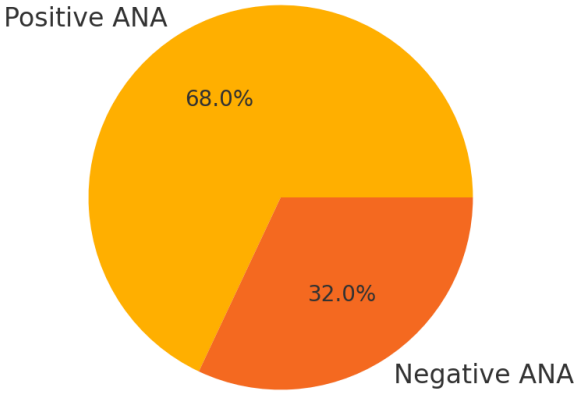


**Figure 7:** ANA Positivity Pie Chart

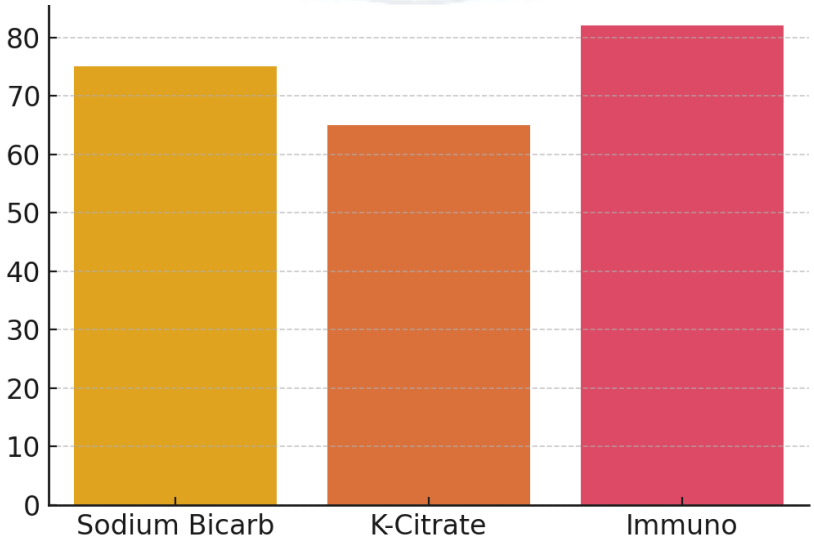


**Figure 8:** Treatment Response in Proximal RTA

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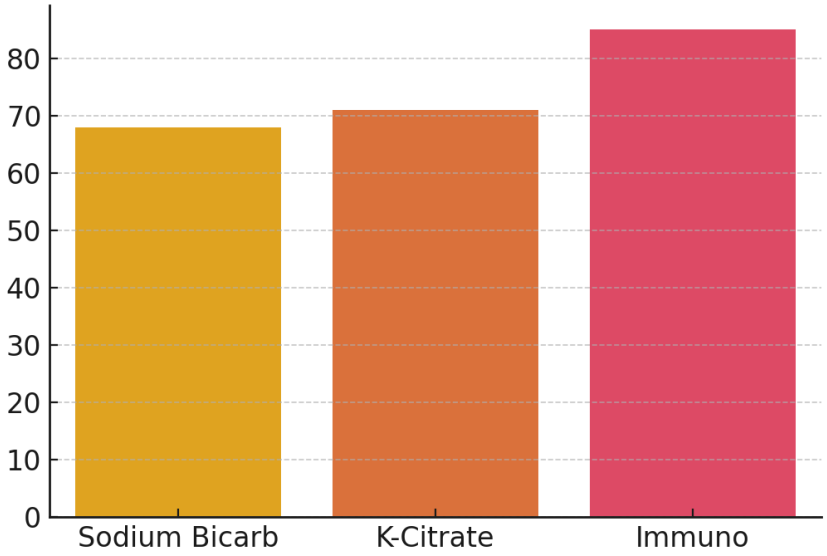


**Figure 9:** Treatment Response in Distal RTA

## DISCUSSION

The results from this study add useful knowledge about how autoimmune disorders relate to renal tubular acidosis which helps improve injury detection and care. As seen in Table 3 and shown by Figure 2, the many positive ANA findings (Zeng et al., 2022) suggest the research population has a general immune system dysfunction. Results from Table 4 reveal a rise in IL-6 and TNF-α, as diagrammed in Figure 3. Many symptoms, particularly problems with muscle strength and exhaustion, show how crucial it is to catch and tackle these conditions early to improve how patients feel. Changes in serum bicarbonate levels, shown in Figure 4 and Table 2, confirm the variety of acid-base disturbances among different RTA types, so patients often require different treatments. With distal RTA, as shown in Figures 8 and 9, lessening the immune response by using immunosuppressants is observed to help improve tubular function more than traditional alkali therapy (Bai et al., 2021). Using multivariate logistic regression and seen in Figure 6 and Table 7, having anti-SSA positivity and lower bicarbonate help predict possible outcomes early in the disease. Taken together, the research by Navarro et al. reveals details of the illness that enable clinicians to develop better treatments.

In serum sickness and secondary unresponsiveness, monitoring the immune system during rituximab treatment in autoimmune disorders seems beneficial (Aoufir et al., 2020). Larger, disease-oriented studies are necessary to confirm these outcomes after this (Aoufir et al., 2020). Serum metabolome changes occurring in rheumatic illness patients may correspond to paraneoplasia or another malignancy which makes them a useful predictor. It has been suggested that a hyperactive immune system and plaques of protein may explain the connection between rheumatoid arthritis and Alzheimer's disease (Trzeciak et al., 2021). In this condition, older cells in osteoarthritis boost inflammation and degrade the ECM (Arra et al., 2022; Liu et al., 2022). Osteoarthritic changes in the cartilage of chondrocytes are caused by oxidative stress brought on by the combinaton of bone matrix particles and inflammatory cytokines (Arra et al., 2022).

## CONCLUSION

In autoimmune diseases, this paper focuses on the immune system's role in renal tubular acidosis (RTA) and discusses its clinical, chemical and immunologic features in detail. Confirmed was that, while both proximal and distal RTA types are unlike in laboratory findings, the latter type of RTA shows more severe disturbances of acid-base balance. In autoimmune patients, the presence of RTA was strongly linked to high levels of inflammatory factors and a high number of autoantibodies, mainly those related to ANA and Anti-SSA. Autoimmune aetiology was more strongly confirmed by the evidence that Anti-SSA positivity and low bicarbonate levels were robust and independent markers found in the regression model. The responses to treatment were not the same; for distant cases of RTA, immunosuppressive therapy was more effective than giving alkaline therapy alone. The analysis underlines that doing tests early using complete biochemical and immunological panels can make a huge difference in patients' outcomes and help prevent serious kidney damage over time. Combining signs and symptoms of atypical kidney problems with immunological findings allows doctors to more accurately diagnose autoimmune diseases involving the kidneys. Systemic immune dysfunction found in renal tubular damage could be addressed by using customized treatments that change immune function. It stresses the importance of recognized awareness and efficient diagnosis as the pressure of chronic renal disease and the usual underdiagnosis of RTA in such settings increases. It also shows that more investigation is required to understand the mechanical link between immune activation and the kidneys' acid-base management processes. All in all, the results provide valuable advice for clinicians to plan future screening activities, sort patients by their risks and apply treatments to protect the kidneys of people with autoimmune conditions.

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