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Thirty-Days Supplementation of CO Enzyme Q10 In Diabetic Rats on Anti-Retroviral Therapy Modulates Serum Electrolyte

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Abstract **Original Research Article**

Background: Coenzyme Q10 (CoQ10), also known as ubiquinone, is a vitamin-like antioxidant essential for mitochondrial energy production and cellular health. It plays a key role in the electron transport chain, helping transfer electrons between complexes. While the introduction of highly active antiretroviral therapy (HAART) has significantly improved survival rates in individuals living with HIV, it has also been linked to long-term side effects, particularly affecting kidney function. ***Objective:*** This study aimed to evaluate the impact of CoQ10 supplementation—both on its own and in combination with combination antiretroviral therapy (cART)—on kidney health, electrolyte balance, and acid-base regulation in both healthy and diabetic male Wistar rats. ***Methods*:** A total of 102 adult male Wistar rats, weighing 170–200 g, were divided into several groups and treated over two phases. The rats received varying doses of CoQ10 (10, 30, and 50 mg/kg), cART, or a combination of both. Key biochemical markers including serum sodium, potassium, and bicarbonate levels were analyzed to assess renal function and systemic electrolyte homeostasis. ***Results*:** Diabetic rats treated with cART alone showed marked imbalances in electrolytes, notably low sodium and high potassium levels—classic signs of diabetic kidney dysfunction. Interestingly, CoQ10 supplementation at 10 and 50 mg/kg helped normalize these values, suggesting a dose-related protective effect on kidney function. However, the 30 mg/kg dose showed less favorable outcomes. The group receiving 50 mg/kg CoQ10 alongside cART displayed the most consistent improvement in electrolyte and acid-base parameters, indicating enhanced renal tubular function, improved sodium and bicarbonate reabsorption, and better potassium handling. ***Conclusion*:** CoQ10 appears to offer a protective benefit against kidney dysfunction, particularly in diabetic models undergoing antiretroviral treatment. Higher doses may be especially effective in restoring electrolyte balance and preserving renal integrity. These findings highlight CoQ10's potential as an adjunct therapy in managing HAART-induced nephrotoxicity.

Keywords: Coenzyme Q10, kidney function, HIV, antiretroviral therapy, diabetic nephropathy, oxidative stress, electrolyte imbalance, mitochondrial health.

INTRODUCTION

Coenzyme Q10 (CoQ10) or ubiquinone is a fat-soluble, vitamin-like compound with potent antioxidant activity. Its function is so crucial for mitochondrial energy production that it transfers electrons from complexes I and II to complex III within the electron transport chain (Hashim *et al.,* 2025). CoQ10 has been demonstrated to reduce production oxidants in endothelial cells and improve cardiac function in patients with heart failure. Long-term supplementation is safe, well-tolerated, and may reduce risk of the combined category of major cardiovascular events (Mantle, *et al.,* 2024)

CoQ10 plasma levels are lower in CKD (both pre-dialysis and dialysis patient) patients. Moreover, this depletion may diminish electron flow, thus enhancing the production of reactive oxygen species (ROS) and oxidative stress. CoQ10 supplementation could potentially ameliorate mitochondrial dysfunction, attenuate oxidative destruction, and safeguard cardiovascular health in chronic kidney disease (Ahmadi *et al.,* 2024)). New data indicate that CoQ10 may improve glucose metabolism, blood pressure, lipid composition, and inflammation, making it an attractive treatment for patients with kidney disease, including those on dialysis (Ahmadi *et al.,* 2024).

In 1984 Rao *et al.,* described a usual pattern of progressive renal failure and proteinuria in HIV-1 seropositive patients that was the first to establish the relationship between HIV infection and renal disease. However, the introduction of Highly Active Antiretroviral Therapy (HAART) in the mid-1990s — a regimen consisting of at least three antiretroviral drugs of at least two different classes — drastically decreased AIDS-related mortality (Marra *et al.,* 2024). Yet alongside its life-saving effect, HAART came with some adverse effects especially kidney dysfunction. Renal complications among people living with HIV can be due to the virus itself or a complication of antiretroviral therapy (Diana, *et al.,* 2024).

The kidneys play a crucial role in drug metabolism and excretion, making them especially vulnerable to damage from antiretroviral and other nephrotoxic drugs. The proximal tubule, which filters large amounts of blood and processes toxins, is particularly susceptible to drug-induced injury (Hoenig, *et al.,* 2025). As HAART often involves multiple medications, the risk of kidney damage may increase due to either direct drug toxicity or complex drug-drug interactions (La Via *et al.,* 2025). Protecting the Kidney while been on this medication is essential for optimizing treatment strategies and minimizing renal complications in patients receiving long-term antiretroviral therapy.

In 2023, approximately 39.9 million people worldwide were living with HIV, these included 38.6 million adults and 1.4 million children under 15, notably, 53% of those affected were women and girls (Mbita *et al.,* 2024). By the end of the year, 77% of people living with HIV (30.7 million) were receiving antiretroviral therapy (ART), a crucial step in the global effort to end AIDS and reduce the public health burden. consistent compliance with HAART can help maintain undetectable viral load, lead long, healthy lives and prevent sexual transmission to HIV-negative partners (Rafaqat *et al.,* 2024).

However, as life expectancy increases with widespread HAART use, the prevalence of non–AIDS-defining conditions is also rising. Individuals on long-term HAART face a higher risk of developing conditions like diabetes, often at a younger age than their HIV-negative peers (Mulindwa *et al.,* 2024)

This study examined the possible defensive role of coenzyme Q10 (CQ10) against the electrolyte imbalance in diabetic rats exposed to Highly Active Anti-Retroviral Therapy (HAART).

METHODOLOGY

Drugs and Chemicals

The drugs administered to the experimental animals included Coenzyme Q10 (100 mg) and a combination antiretroviral therapy (cART) regimen containing Dolutegravir/Lamivudine/Tenofovir disoproxil (50 mg/300 mg/300 mg), these were graciously donated by the University of Port Harcourt Teaching Hospital.

The following chemicals and reagents were used throughout the study:

- **Biochemical Reagent Kits:** Fortress Diagnostics
- **Anesthetic Agent:** Diethyl ether (Sigma)
- **Fixatives and Preservatives:** Formalin 10% (BDH Chemicals Ltd)
- **Solvents:** Ethanol 95% (Sigma), Chloroform (BDH Chemicals Ltd)

All chemicals were of analytical grade and used as received, without further purification. Reagents were handled according to standard safety protocols, and proper disposal methods were observed throughout the experimental procedures.

Animals and Ethical Considerations

A total of 102 adult male Wistar rats, weighing between 170–200 g, were procured from the Animal House of the Department of Experimental Pharmacology and Toxicology, University of Port Harcourt. The animals were housed in cages with plastic bottoms and wire-mesh tops, and fed with standard finisher feeds (Top Feed Ltd) alongside access to tap water ad libitum throughout the experimental period.

The animals were allowed to acclimate to laboratory conditions and handling for three weeks prior to the commencement of the experiment. The housing environment was well-ventilated, maintained at an ambient temperature of 28 ± 2ºC, with natural lighting cycles.

Ethical approval for the study was obtained from the University of Port Harcourt Research Ethics Committee, and all experimental procedures were conducted in compliance with institutional guidelines for the care and use of laboratory animals.

Study Design

The research was conducted in two distinct phases to investigate the effects of Coenzyme Q10 (CoQ10) supplementation, both independently and in combination with combined antiretroviral therapy (cART), in normal and diabetic rats.

Phase I: Effects of CoQ10 and cART in Healthy Rats

This phase explored the impact of CoQ10 supplementation at different doses, either alone or in combination with cART.

The experimental groups were as follows:

Normal Control Group (NC): Rats received standard rat chow with no treatment.

cART Group (CT): Rats were administered cART at a dose of 70 mg/kg body weight daily.

CoQ10 Supplementation Groups: Rats received daily doses of CoQ10 as follows:
Low dose (10 mg/kg body weight)
Medium dose (30 mg/kg body weight)
High dose (50 mg/kg body weight)

Combination Therapy Groups: Rats received cART alongside CoQ10 at varying doses:
cART + CoQ10 Low Dose (10 mg/kg)
cART + CoQ10 Medium Dose (30 mg/kg)
cART + CoQ10 High Dose (50 mg/kg)

Phase II: Effects of CoQ10 and cART in Diabetic Rats
The second phase assessed the combined effects of CoQ10 and cART in diabetic rats to understand the potential interactions in a metabolic disorder setting. The groups were structured as follows:
Normal Control Group (NC): Rats received standard rat chow with no treatment.

cART Group (CT): Rats were administered cART at 70 mg/kg body weight daily.

Diabetes Mellitus Group (DM): Diabetes was induced via intraperitoneal injection of alloxan (50 mg/kg body weight), which was sustained throughout the study.

Combination Therapy in Diabetic Rats: Diabetic rats received cART along with CoQ10 supplementation at different doses:
cART + CoQ10 Low Dose + DM (10 mg/kg)
cART + CoQ10 Medium Dose + DM (30 mg/kg)
cART + CoQ10 High Dose + DM (50 mg/kg)

This design enabled the evaluation of CoQ10's potential protective effects against cART-induced toxicity in both normal and diabetic states, providing insights into its role in mitigating oxidative stress and improving metabolic outcomes.

Induction of Diabetes

Diabetes mellitus was induced in the experimental rats by a single intraperitoneal (IP) injection of alloxan monohydrate, at a dose of 50 mg/kg body weight. Alloxan was freshly prepared in normal saline prior to administration. Alloxan selectively targets and destroys insulin-producing beta cells in the pancreas, leading to impaired insulin secretion and the development of hyperglycemia. Following the injection, the animals were allowed to recover and monitored for any immediate adverse effects.

Forty-eight hours post-alloxan administration, blood glucose levels were measured using a portable glucometer. Rats with blood glucose levels greater than 200 mg/dL were considered to have developed diabetes and were included in the study. Before the injection of alloxan, the animals were fasted for 8 to 12 hours to ensure accurate glucose readings and to standardize the experimental conditions. Water was provided ad libitum during the fasting period and throughout the remainder of the study. This induction method ensured consistent development of diabetes in the rats, enabling the evaluation of therapeutic interventions in the subsequent phases of the experiment.

Materials Required
Mind Ray BS-240 multi-functional benchtop Automated chemistry analyzer (BS-
Blood samples (serum)
Electrolyte reagent kits specific for Na+, K+, Cl-, and HCO_3^-
Sample tubes or serum separator tubes
Pipettes
Distilled water

Preparation

Blood samples was collected from anaesthetized rats and appropriately labeled serum, Centrifuge the samples at 3000 rpm for 10 minutes to separate the serum or plasma from blood cells. Reagents were prepared according to the manufacturer's instructions.

Analyzer Setup: The Mindray BS-240 multi-functional benchtop chemistry analyzer was powered on, and the system was verified to ensure all necessary components, including the optical and ion-selective electrodes (ISE), were functioning correctly. The software interface was checked, and the analyzer was confirmed to be ready for sample analysis.

Sample Loading: Prepared plasma or serum samples were placed into the designated sample racks on the analyzer. Each sample was carefully labeled, and proper alignment was ensured for automatic analysis. The system utilized sample barcode detection to ensure correct identification and tracking of the samples.

Electrolytes Measure: Sodium (Na+), Potassium (K+), Chloride (Cl-) and Bicarbonate (HCO_3^-) Measurement:

DATA ANALYSIS

A comprehensive coding guide was developed alongside the data collection tool to organize and facilitate the efficient analysis of the collected data. The data were then subjected to statistical analysis using one-way analysis of variance (ANOVA) to assess the overall differences among the experimental groups. The statistical analysis was performed using SPSS software and the results were presented as the mean ± standard error of the mean (SEM), ensuring a clear and concise depiction of the central tendency and variability within each group. The results were interpreted with careful attention to the p-values, where a p-value less than 0.05

indicated a statistically significant difference between the groups under study.

Data Presentation

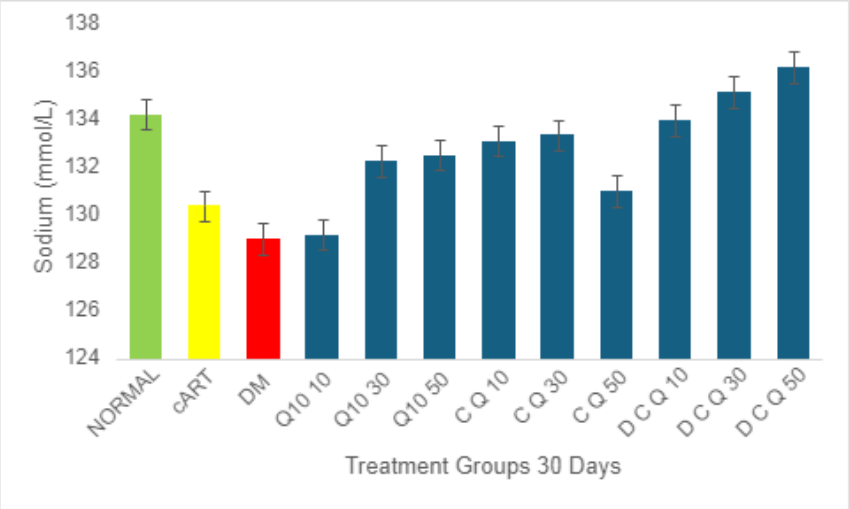

Figure 1: Sodium levels Associated with different Treatment Groups

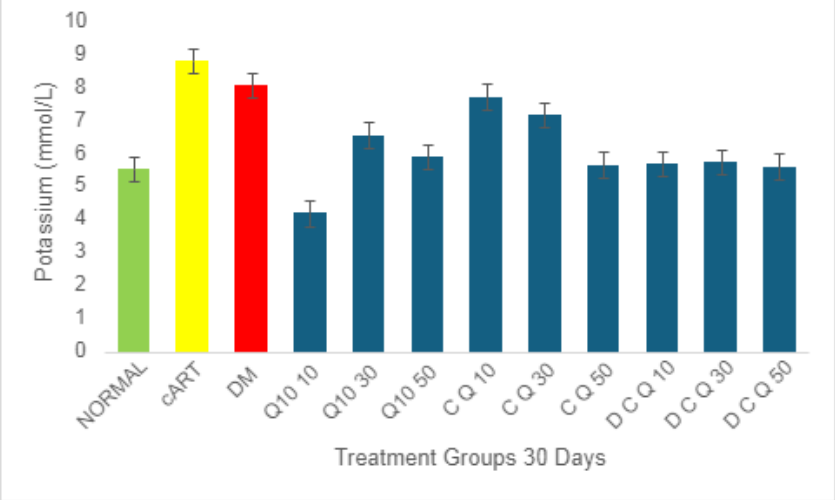

Figure 2: Potassium levels Associated with different Treatment Groups

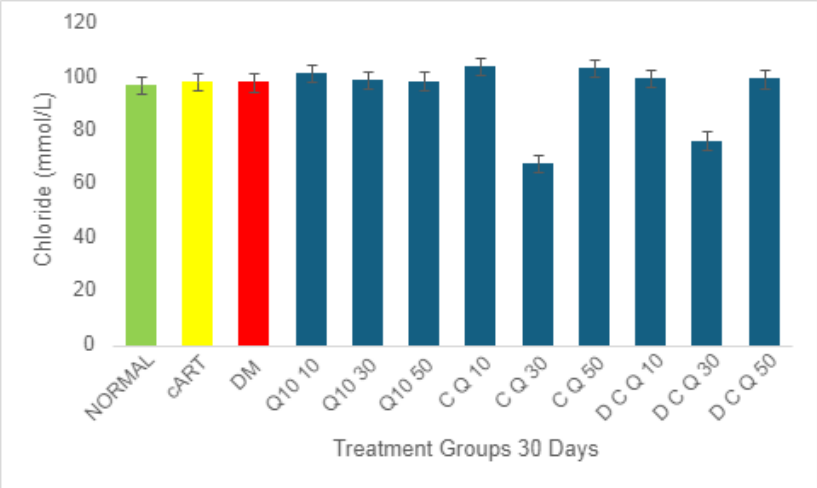

Figure 3: Chloride levels Associated with different Treatment Groups

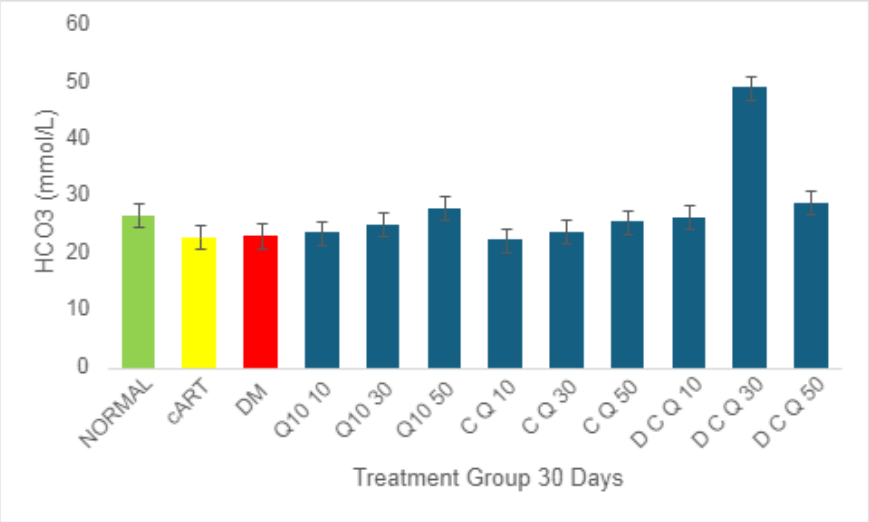

Figure 4: Bicarbonate levels Associated with different Treatment Groups

RESULTS AND DISCUSSION

Sodium Levels Across Experimental Groups

The sodium concentration in the normal control group was 134.29 ± 1.49 mmol/L, serving as the baseline reference. In the cART group, sodium levels decreased to 130.43 ± 4.61 mmol/L, while the diabetes mellitus (DM) group exhibited a slightly lower concentration of 129.07 ± 2.29 mmol/L when compared to the normal group.

In the CoQ10-supplemented groups, sodium levels showed varying responses depending on the dosage. The Q10 10 mg/kg group recorded 129.23 ± 4.99 mmol/L, whereas the Q10 30 mg/kg group exhibited a modest increase to 132.30 ± 1.67 mmol/L. The highest sodium concentration within this set was observed in the Q10 50 mg/kg group, at 132.57 ± 2.70 mmol/L, indicating a potential dose-dependent effect of CoQ10 supplementation on sodium homeostasis.

Combination therapy with cART and CoQ10 yielded mixed results. The C Q 10 group (cART + 10 mg/kg CoQ10) had sodium levels of 133.17 ± 6.98 mmol/L, while the C Q 30 group (cART + 30 mg/kg CoQ10) showed a slight increase to 133.40 ± 1.13 mmol/L. Conversely, the C Q 50 group (cART + 50 mg/kg CoQ10) exhibited a small decline, with sodium levels measuring 131.07 ± 3.31 mmol/L.

In diabetic groups receiving combination therapy, sodium levels were generally elevated relative to both the DM and cART-only groups. The D C Q 10 group (DM + cART + 10 mg/kg CoQ10) recorded 134.03 ± 0.15 mmol/L, closely aligning with the normal control group. A further increase was observed in the D C Q 30 group (DM + cART + 30 mg/kg CoQ10) at 135.23 ± 0.06 mmol/L. Notably, the D C Q 50 group (DM + cART + 50 mg/kg CoQ10) demonstrated the highest sodium concentration among all groups, reaching 136.27 ± 1.69 mmol/L, suggesting a pronounced sodium-regulatory effect with higher doses of CoQ10 in diabetic conditions.

Potassium Levels Across Experimental Groups

Potassium levels were assessed across all experimental groups, revealing significant variations associated with disease states and treatment regimens. The cART group exhibited elevated potassium levels (8.87 ± 2.85 mmol/L), while the diabetes mellitus (DM) group showed similarly high levels (8.11 ± 1.79 mmol/L). These elevated values suggest electrolyte imbalance and potential metabolic disturbances, particularly in the context of diabetes and antiretroviral therapy. In contrast, the Normal/Control group maintained potassium levels at 5.58 ± 1.03 mmol/L, indicative of stable electrolyte homeostasis.

In the CoQ10-supplemented groups, potassium concentrations varied with dose. The Q10 10 mg/kg group exhibited the lowest potassium level (4.22 ± 2.91 mmol/L), while the Q10 30 mg/kg group demonstrated a moderate increase to 6.60 ± 0.60 mmol/L. The Q10 50 mg/kg group showed potassium levels of 5.93 ± 0.32 mmol/L, suggesting that higher doses of CoQ10 may promote potassium stability. Notably, potassium levels in all CoQ10-treated groups were lower than those observed in the cART and DM groups, suggesting a potential protective effect of CoQ10 against potassium dysregulation.

Chloride (Cl^-) Levels Across Experimental Groups

Chloride (Cl^-) levels were evaluated to monitor electrolyte balance and assess their contribution to fluid regulation and acid-base homeostasis across various experimental groups. Maintaining appropriate chloride concentrations is essential for cellular function and overall metabolic stability.

In the normal control group, chloride levels were 97.58 ± 1.10 mmol/L, aligning with reference

values observed in healthy individuals. The cART group demonstrated slightly higher chloride levels (98.60 ± 0.75 mmol/L), while the diabetes mellitus (DM) group presented similar levels (98.43 ± 2.31 mmol/L). This similarity suggests that diabetes alone did not induce substantial chloride dysregulation, and cART administration had a minimal impact on chloride homeostasis.

In the CoQ10-supplemented groups, chloride levels exhibited dose-dependent variations. The Q10 10 mg/kg group displayed the highest levels (101.90 ± 4.59 mmol/L), potentially indicating increased chloride retention with low-dose supplementation. Conversely, the Q10 30 mg/kg and Q10 50 mg/kg groups showed more moderate chloride concentrations (99.33 ± 1.80 mmol/L and 99.03 ± 2.64 mmol/L, respectively), suggesting a stabilizing effect with higher CoQ10 doses.

Combination therapy groups displayed broader chloride fluctuations. The C Q 10 group had the highest chloride concentration (104.37 ± 3.89 mmol/L), while the C Q 30 group showed a markedly lower and more variable level (68.02 ± 58.24 mmol/L). The C Q 50 group recorded elevated chloride levels (103.63 ± 5.65 mmol/L), highlighting potential dose-related influences of combined cART and CoQ10 therapy on electrolyte balance.

In the diabetic combination therapy groups, chloride levels varied but remained within physiologically acceptable ranges. The D C Q 10 group recorded 100.07 ± 1.00 mmol/L, while the D C Q 30 group displayed highly variable levels (76.70 ± 39.77 mmol/L). The D C Q 50 group exhibited relatively stable chloride concentrations (99.73 ± 1.22 mmol/L).

Bicarbonate (HCO_3^-) Levels Across Experimental Groups

Bicarbonate (HCO_3^-) levels were evaluated as an indicator of acid-base balance and the blood's buffering capacity, which are critical for maintaining physiological pH homeostasis. Changes in bicarbonate concentration can reflect underlying metabolic disturbances, particularly in conditions like diabetes and with drug interventions.

At day 30, the normal control group exhibited bicarbonate levels of 26.78 ± 1.55 mmol/L, consistent with the typical reference range for healthy individuals. In contrast, the cART group and DM group displayed reduced bicarbonate levels (23.01 ± 4.36 mmol/L and 23.21 ± 2.27 mmol/L, respectively), suggesting a possible metabolic acidosis associated with chronic drug exposure and diabetes-induced metabolic dysregulation.

In the CoQ10-supplemented groups, bicarbonate levels varied with dosage. The Q10 10 mg/kg group maintained levels within the normal range (23.82 ± 1.24 mmol/L). Meanwhile, higher doses of CoQ10 resulted in increased bicarbonate concentrations, with the Q10 30 mg/kg group reaching 25.25 ± 3.58 mmol/L and the Q10 50 mg/kg group rising to 28.09 ± 1.99 mmol/L, indicating a potential dose-dependent enhancement of acid-base stability.

Among the combination therapy groups, bicarbonate levels fluctuated. The C Q 10 group had the lowest bicarbonate concentration (22.51 ± 3.47 mmol/L), suggesting a slight acidotic shift, possibly due to drug interactions or cumulative metabolic stress. However, the C Q 30 and C Q 50 groups showed more balanced levels (23.92 ± 2.65 mmol/L and 25.69 ± 2.75 mmol/L, respectively), implying a more stabilized acid-base status with higher CoQ10 doses in combination with cART.

In the diabetic combination therapy groups, bicarbonate levels were generally well-maintained. The D C Q 10 group recorded 26.50 ± 1.11 mmol/L, which falls within the normal range. Interestingly, the D C Q 30 group exhibited an elevated value of 29.28 ± 3.58 mmol/L, potentially reflecting biological variability or outlier effects. The D C Q 50 group showed similarly elevated levels (29.08 ± 0.92 mmol/L), suggesting that higher doses of CoQ10 in combination with cART may contribute to improved buffering capacity and acid-base regulation in diabetic conditions.

Electrolyte and Acid-Base Balance

Electrolyte disturbances were most notable in the DM group, characterized by significant sodium depletion and potassium elevation. The elevated potassium levels suggest impaired renal potassium excretion, which, along with reduced sodium, is indicative of diabetic nephropathy and similarly observed by Lengeiya, *et al.,* (2024). The cART group, on the other hand, exhibited relatively stable sodium levels but showed increased potassium, potentially due to drug-induced tubular dysfunction, Giri, *et al.,* (2024) also observed similar changes.

Q10-based treatments demonstrated varying effects on electrolyte balance. The Q10 10 and Q10 50 groups exhibited sodium and potassium levels similar to normal, suggesting improved homeostasis at these doses. However, Q10 30 resulted in elevated potassium, indicating a dose-dependent effect. Among the combination groups, C Q 30 and D C Q 50 had more balanced sodium and potassium levels, suggesting that these regimens may help stabilize electrolyte disturbances. Bicarbonate levels, which were significantly depleted in DM, showed improvement in most Q10-treated groups, particularly D C Q 50, which had the highest bicarbonate concentration, indicating a positive effect on acid-base balance, these results are in agreement with those from Abd-Elhakim, *et al., (*2024).

Potential Mechanism

Renal impairments in DM can be attributed to chronic hyperglycemia-induced damage to the nephrons which leads to reduced glomerular filtration and impaired electrolyte handling Młynarska, *et al.,* (2024). The elevated creatinine and urea levels reflect declining renal clearance, while the potassium retention suggests tubular dysfunction. cART-related changes may stem from drug-induced nephrotoxicity, where certain antiretroviral agents alter renal tubular secretion and electrolyte balance (Ogedengbe *et al.,* (2024).

Some mechanisms have been documented for the observed renal changes. Coenzyme Q10 is a major player in mitochondrial bioenergetics, it enhances cellular energy metabolism and reduces oxidative stress, all contributors to renal dysfunction in DM and cART patients. It plays a role as an antioxidant mitigating oxidative damage to renal cells, improving filtration and electrolyte regulation. The more balanced electrolyte and acid-base parameters in certain Q10-treated groups, particularly D C Q 50, could be linked to its ability to modulate renal tubular transport processes, thereby enhancing sodium and bicarbonate reabsorption while facilitating potassium excretion (Kara *et al.,* 2024).

Conclusively, Q10 may have modulated electrolyte imbalance brought on by Diabetes we recommend Routine electrolyte balance check in DM and cART patients to assess early signs of kidney dysfunction.

Q10 should be considered as a supportive therapy to help preserve kidney function and maintain electrolyte homeostasis.

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