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# Effect of Stem Cell (Beaustem) on Serum Renal Indices of Wistar Rats Induced with Diabetes Mellitus

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**Abstract**

This study evaluated the effects of a stem cell product (Beaustem) on serum renal indices in Wistar rats with streptozotocin-induced diabetes mellitus, a condition characterised by hyperglycemia-driven oxidative stress, inflammation, and structural renal damage. Twenty rats were allocated into four groups: normal control, diabetic untreated, diabetic treated with Glucophage, and diabetic treated with Beaustem. After 31 days of treatment, serum levels of key renal biomarkers including creatinine, urea, cystatin-C, Kidney Injury Molecule-1 (KIM-1), and electrolytes were analysed. Diabetic rats exhibited significant elevations in KIM-1, cystatin-C, urea, creatinine, sodium, and potassium, consistent with established patterns of diabetic nephropathy and indicative of both tubular and glomerular impairment. Beaustem therapy significantly improved these alterations. KIM-1, a highly sensitive marker of proximal tubular injury, was markedly elevated in untreated diabetic rats but substantially reduced following Beaustem administration, supporting evidence that stem cell-based interventions mitigate tubular apoptosis, inflammatory responses, and mitochondrial dysfunction. Cystatin-C, a reliable marker of glomerular filtration, was also elevated in diabetic controls but improved considerably after stem cell treatment, with effects comparable to those of Glucophage. Similarly, reductions in urea and creatinine following Beaustem therapy suggest enhanced nephron recovery, likely mediated through stem cell paracrine actions involving growth factors and extracellular vesicles that promote tissue repair. Electrolyte abnormalities in diabetic rats reflected tubular transport defects, but Beaustem effectively normalised potassium and moderately corrected sodium disturbances. Bicarbonate levels, reduced due to metabolic acidosis in diabetic animals, were also significantly restored with treatment. Overall, Beaustem exhibited strong renoprotective potential, reversing multiple biochemical indicators of diabetic renal injury and highlighting its promise as a therapeutic option for diabetic kidney disease.

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## Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from defects in insulin secretion, insulin action, or both. It is a complex condition with significant implications for public health and individual well-being (American Diabetes Association, 2021).

The several types of diabetes mellitus, including type 1 diabetes mellitus, type 2 diabetes mellitus, and gestational diabetes mellitus. Type 1 diabetes mellitus is an autoimmune disease in which theimmune system attacks and destroys the insulin-producing cells of the pancreas (Chiang *et al.,* 2021). On the other hand, type 2 diabetes is a progressive condition characterized by insulin resistance and relative insulin deficiency as well as insulin insensitivity. Gestational diabetes occurs during pregnancy and usually resolves after delivery, but it increases the risk of developingtype 2 diabetes later in life (Chiang *et al*., 2021).

The prevalence of diabetes mellitus has been steadily increasing worldwide. According to the International Diabetes Federation (IDF), in 2019, approximately 463 million adults (20-79 years) were living with diabetes globally, and this number is projected to rise to 700 million by 2045 (International Diabetes Federation, 2019).

Uncontrolled diabetes can lead to various complications, including cardiovascular disease, kidney disease, eye problems, nerve damage, and lower limb amputations (Centers for Disease Control and Prevention, 2021). Management of diabetes involves a combination of lifestyle modifications, such as healthy eating, regular physical activity, and weight

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management, as well as pharmacological interventions, including insulin and oral medications (Tesfaye *et al*., 2010).

Diabetes mellitus has significant effects on various systems and organs in the body. Diabetes increases the risk of cardiovascular diseases such as coronary artery disease, myocardial infarction (heart attack), stroke, and peripheral arterial disease. It is associated with endothelial dysfunction,atherosclerosis, and abnormal lipid metabolism (Buchanan, 2005). It is a leading cause of chronickidney disease (CKD) and end-stage renal disease (ESRD). Persistent high blood glucose levels and prolonged exposure to elevated blood pressure can damage the kidneys' filtering units (glomeruli) and impair kidney function. Diabetes can also lead to diabetic retinopathy, a condition characterized by damage to the blood vessels in the retina. It is a leading cause of vision loss and blindness in adults. Diabetes also increases the risk of cataracts and glaucoma (American Diabetes Association, 2021). It is associated with an increased risk of mental health conditions such as depression and anxiety. The burden of managinga chronic condition, the impact of diabetes on daily life, and the potential for complications can contribute to psychological distress (Snoek *et al.*, 2019).

The diagnosis of diabetes involves evaluating various clinical parameters and laboratory tests. TheAmerican Diabetes Association (ADA) provides guidelines for the diagnosis of diabetes mellitus(American Diabetes Association, 2021). For type 1 diabetes, the ADA recommends considering the presence of classic symptoms (polyuria, polydipsia, unexplained weight loss) and confirminghyperglycemia. Fasting plasma glucose (FPG) ≥100 mg/dL (7.0 mmol/L) or a 2-hour plasma glucose level ≥200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test (OGTT) can also be used for diagnosis (American Diabetes Association, 2021).

For type 2 diabetes mellitus involve testing asymptomatic individuals using the FPG, OGTT, or hemoglobin A1c (HbA1c). A fasting plasma glucose level ≥126 mg/dL (7.0 mmol/L) on two separate occasions, a 2-hour plasma glucose level ≥200 mg/dL (11.1 mmol/L) during an OGTT, or an HbA1c level ≥6.5% (48 mmol/mol) are indicative of diabetes (Holmes *et al*., 2011).

Gestational diabetes mellitus is diagnosed using a 75-gram OGTT performed between 24 and 28 weeks of gestation. The diagnostic criteria include a fasting plasma glucose level ≥92 mg/dL (5.1mmol/L), a 1-hour plasma glucose level ≥180 mg/dL (10.0 mmol/L), or a 2-hour plasma glucose level ≥153 mg/dL (8.5 mmol/L) (Dabelea *et al.,* 2000). It is important to note that the diagnostic criteria may vary slightly depending on the guidelines and regional practices. Healthcare professionals should consider local recommendations when making a diagnosis.

Stem cell (Beaustem) is one product that has been claimed to handle skin issues. It is a combination of products from Swiss Apple Stem, Grape Stem Cell, and Collagen Peptide. It is a revolutionarytechnology designed to protect human skin stem cells with the help of stem cells from a rare Swissapple. While Grape stem cells are high in antioxidants, they help to protect the skin from the appearance of environmental damage as well as the signs of aging, and finally the Collagen assiststo hold all active ingredients together.

This study aimed at evaluating the Effect of Stem cell (Beaustem) on serum renal indices of wistar rats induced with diabetes mellitus using Streptozotocin (STZ). Diabetes mellitus is a chronic metabolic disorder characterized by high blood glucose levels due to defects in insulin secretion, insulin action, or both. It is a global health concern, affecting millions of people worldwide, and its prevalence continues to rise at an alarming rate. Diabetes mellitus has reached epidemic proportions, affecting approximately 463 million adults worldwide,with a projected increase to 700 million by 2045 (International Diabetes Federation, 2019). Diabetes mellitus is a significant risk factor for macrovascular complications, such as cardiovascular disease (CVD), stroke, and peripheral artery disease (ADA, 2021). Individuals withdiabetes have a two to four times higher risk of developing CVD compared to those without diabetes (CDC, 2021). Understanding the epidemiology, etiology, and complications of diabetes mellitus is crucial for the development of effective prevention and management strategies. Stem cell (Beaustem) is a plant stem cell that rebuilds, restores, replaces, reactivates and rejuvenates cells itcures a lot of disease conditions e.g cancer, kidney disease, liver issues, diabetes, fibroid, etc. It isa combination of products from Swiss Apple Stem, Grape Stem Cell, and Collagen Peptide. However, this has not being fully proven or investigated scientifically and hence giving rise to this study; Effect of Stem cell (Beaustem) on serum renal indices of Wistar rats induced with diabetes rats

**Laboratory Animals**

Twenty (20) Albino rats of the Wistarstrain with a weight range of 80-130g were obtained from the Animal House of the Department of Biochemistry, University of Port Harcourt, Rivers State, Nigeria. They were housed and cared for, following the standard rules and regulations of The Institute for Laboratory Animal Research (ILAR).

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The procured animals were allowed to acclimatize for a period of 7 days at the Federal UniversityOtuoke animal house. They were kept in plastic cages with wire mesh covers to aid ventilation. The animals were under monitored environmental conditions of temperature (28 ± 2°C), relative humidity (50 ± 5%) and 12-hour light/dark cycle. The animal facility was properly ventilated andthe animals were placed on commercial rat pellets as feed and water *ad libitum* throughout the experimental period.

**Experimental Design and Treatment of Animals**
Administration of treatment was done twice daily for a period of thirty-one (31) days via orogastric intubation. The experimental design employed comprised 20 Wistar rats of the albino strain divided into 4 groups of 5 animals each. Group A was the normal control group and the animals in this group received only distilled water. Group B was the negative control group, it was induced withDiabetes mellitus and the animals in this group received distilled water. Animals in Group C (Positive control) which were also induced with Diabetes mellitus received 0.02mg/ml of Glucophage. Animals in treatment Group D which were induced with diabetes mellitus received 0.02mg/ml of Stemcell (Beaustem) as treatment. 60mg/kg body weight of STZ was administered intraperitoneally and fasting blood sugar confirmed to have elevated after 48hrs using glucometer

**Collection of Blood Samples for Analysis:** The animals were sacrificed 12 hours after the last treatment and whole blood was collected from the heart via cardiac puncture using a sterile syringe and needle. The blood samples were put into plaintubes. The blood sample in the plain tubes was allowed to clot by standing for 2 hours at room temperature then later centrifuged at 1000rpm for 10 minutes to separate from the red blood cells.Sera from each centrifuged plain tube were collected into another plain tube labelled accordingly using Pasteur pipettes. The separate sera were then kept frozen in a refrigerator until neededfor various biochemical assays.

**Biochemical Assay:** All biochemical assays were carried out using kits and the required biochemical analyzers.

**Serum Electrolyte**
**Principle of the Assay:** Serum electrolytes typically involve measuring the concentration of various ions in the blood, such as sodium ($Na^+$), potassium ($K^+$), chloride ($Cl^-$), and bicarbonate ($HCO_3^-$). These measurements are essential to assess a person's electrolyte balance and help diagnose and monitor various medical conditions. The concentration of sodium ions in the serum is determined using ion-selective electrodes (ISE) that selectively bind to sodium ions. The assay relies on the potential difference generated when sodium ions move across a membrane in response to electrical forces. Serum potassium involves measuring the concentration of potassium ions using ion-selective electrodes or flame photometry. The method relies on the fact that potassium ions can be selectively detected and measured based on their characteristic emission spectra. Serum chloride involves measuring the concentration of chloride ions using ion-selective electrodes. The method is based on the potential difference generated when chloride ions move across a membrane in response to electrical forces. Serum bicarbonate involves measuring the total CO2 content or directly measuring bicarbonate ions in serum using an enzymatic or photometric method.

**Serum Urea**
**Principle of the Assay:** The principle of the assay of serum urea is based on the enzymatic reaction known as the urease method (Meyer, 2020). In this method, urea is hydrolyzed by the enzyme urease into ammonia andcarbon dioxide (Alberts *et al.,* 2014). The rate of this enzymatic reaction is directly proportional to the concentration of urea in the serum sample. To perform the assay, a sample of serum is firstobtained from the patient and mixed with a reagent containing urease. The mixture is then incubated, allowing the urease enzyme to hydrolyze the urea present in the serum. As a result of this hydrolysis, ammonia is released. The next step involves the measurement of ammonia production. This can be done using various methods, such as a colorimetric assay, where the concentration of ammonia is determined by its ability to form a colored complex with a specific reagent (Smith and Johnson, 2018). The concentration of ammonia produced is directly proportional to the concentration of urea in the original serum sample. By comparing the ammoniaconcentration to a standard curve generated using known concentrations of urea, the amount of urea in the patient's serum can be accurately quantified.

**Creatinine**
**Principle of the Assay:** The principle of the assay of serum creatinine is based on the Jaffe reaction, which is widely used in clinical laboratories (Burtis and Bruns, 2015). In the Jaffe reaction, creatinine reacts with picricacid under alkaline conditions to form a colored complex, the intensity of which is directly proportional to the concentration of creatinine in the serum. To perform the assay, a serum sampleis first

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obtained from the patient and mixed with an alkaline solution containing picric acid. The mixture is allowed to react, leading to the formation of the colored complex. The absorbance of the colored complex is then measured using a spectrophotometer at a specific wavelength. The concentration of creatinine in the serum sample is determined by comparing the absorbance of thecolored complex to a standard curve generated using known concentrations of creatinine. By usingthis calibration curve, the concentration of creatinine in the patient's serum can be accurately quantified. The Jaffe reaction is a well-established and reliable method for measuring serum creatinine levels and is commonly used in clinical settings to assess kidney function and diagnose various renal disorders.

**Kidney Injury Molecule -1 (Kim-1)**

**Principle of the Assay:** The principle of the assay for KIM-1 is based on the use of a solid-phase sandwich enzyme-linked immunosorbent assay (ELISA) technique. In this method, specific antibodies are utilized to detectand quantify KIM-1 in biological samples, such as urine or blood. To perform the assay, a microplate is coated with a capture antibody specific to KIM-1. The biological sample (serum) isadded to the microplate, allowing any KIM-1 present in the sample to bind to the immobilized capture antibody. After washing to remove unbound material, a detection antibody labeled with an enzyme is added. This detection antibody also specifically recognizes KIM-1 and forms a"sandwich" with the captured KIM-1. Following another washing step to remove any unbound detection antibody, a substrate solution is added. The enzyme linked to the detection antibody catalyzes a reaction with the substrate, producing a measurable signal, typically a colour change. The intensity of the signal is directly proportional to the amount of KIM-1 present in the sample. The concentration of KIM-1 in the original specimen can be determined by comparing the signal obtained from the sample to a standard curve generated using known concentrations of KIM-1. This ELISA method provides a valuable tool for researchers and clinicians to assess kidney injuryand monitor kidney function through the measurement of KIM-1 levels (Smith *et al.,* 2023).

**Crystatine C**

**Principle of the Assay:** The assay for cystatin C is based on the quantification of cystatin C levels in biological samples, typically serum or plasma. Cystatin C is a low-molecular-weight protein that is produced at a constant rate by all nucleated cells in the body. It is freely filtered by the glomeruli in the kidneysand almost entirely reabsorbed and catabolized in the tubules. Due to these characteristics, cystatinC is considered to be a reliable marker for estimating glomerular filtration rate (GFR) and assessingkidney function. The measurement of cystatin C levels is commonly performed using immunoassay methods, such as particle-enhanced turbidimetric assays (PETIA) or nephelometric assays. In these assays, antibodies specific to cystatin C are used to detect and quantify the proteinin the sample. The concentration of cystatin C is then determined by comparing the sample's response to a calibration curve generated using known concentrations of cystatin C standards (Herget-Rosenthal *et al.*, 2004).

**Statistical Analysis:** Data obtained from the experiment were analyzed using one-way analysis of variance (ANOVA) using Statistical Package for Social Sciences version 23 (SPSS 23) and presented as means ± standard error of mean (SEM). Value at ($p < 0.05$) were regarded as significant in comparison withthe appropriate controls.

Table 1: Experimental Design and Schedule of Treatments

| Groups | Number of Animals | Administration/Treatment |
|---|---|---|
| A (Normal Control) | 5 | Distilled waters |
| B (Positive Control) | 5 | Distilled waters |
| C (Standard Control) | 5 | 0.02ml of Glucophage (8.33mg/kg bw) |
| D (TEST) | 5 | 0.02ml of Stemcell ( (0.17mg/kg bw) ) |

**Results**

**Effects of Treatment on Renal Indices Parameters**

The effect of Stem cell (Beaustem) on some biochemical parameters of renal indices was evaluated and the results are presented in table 2.

**Kidney Injury Molecule (Kim1):** The laboratory results obtained for that serum KIM1 concentration showed that the serum KIM1 concentration for animals in group B (31.20 ± 1.16), C (12.40 ± 0.51) and D (17.2 ± 0.20) where all significantly ( $P< 0.05$),

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higher compared to that of group A or the normal control group (1.54 ± 0.04). However, the serum KIM1 concentration value for animals in group D (17.20 ± 0.20) wassignificantly (P< 0.05) lower than that of group B or the positive control (31.20 ± 1.16).

**Cystatin- C**: The laboratory results obtained shows that serum concentration of Cystatin- C animals in group B (34.40 ± 0.55), C (11.20 ± 0.66) and D (13.40 ± 0.25) where significantly ( P< 0.05), higher compared to that of group A or the normal control group (0.66 ± 0.08). Moreso, the serum Cystatin-C value for animals in group D (13.40 ± 0.25) was significantly (P< 0.05) lower compared to that of group B or the positive control (31.20 ± 1.16), but compared well with that ofgroup C ( 11.20 ± 0.66 ) which were tested with standard diabetic drugs ( glucophage).

**Urea (Blood Urea Nitrogen):** The blood nitrogen results obtained show that the serum Urea nitrogen concentration of animalsin group B (7.54 ± 0.07), C (4.32 ± 0.21) and D (4.06 ± 0.08) where all significantly ( P< 0.05), higher compared to that of group A or the normal control group (2.68 ± 0.07). However, the bloodurea nitrogen value for animals in group D (4.06 ± 0.08) was significantly (P< 0.05) lower thanthat of group B or the positive control (7.54 ± 0.07), but compares favorably well with that of group C ( 4.32 ± 0.21 ) which was treated with standard diabetic drugs ( Glucophage)

**Creatinine:** The creatinine results showed that the serum creatinine concentration in group B (106.58 ± 0.27),C (51.26 ± 0.32) and D (45.36 ± 0.74) where all significantly (P< 0.05), higher compared with thatof group A or the normal group (36.74 ± 0.79). More so, the serum creatinine value for animals in group D (45.36 ± 0.74) was significantly (P< 0.05) lower compared with that of group B or the positive control (106.58 ± 0.27), and that of group C (51.26 ± 0.32).

**Sodium (Na):** The sodium test results showed that the serum sodium concentration of animals in group B (140.60 ± 1.21), C (141.02 ± 0.45) and D (141.80 ± 0.49) were all significantly ( P< 0.05), higher compared with that of group A or the normal group (132.40 ± 0.68). However, the serum sodiumconcentration for animals in group D (141.80 ± 0.49) showed no significant (P> 0.05) change compared with that of group B or the positive control (140.60 ± 1.21), but compares favorably well with that of group C (141.02 ± 0.45).

**Potassium (K):** The Potassium test results showed that the serum potassium concentration in group B (6.58 ± 0.08), C (4.24 ± 0.07) and D (4.64 ± 0.19) are significantly (P< 0.05) higher compared with that of group A or the normal group (3.10 ± 0.03). More so, the serum potassium concentration for animals in group D (4.64 ± 0.19) was significantly (P< 0.05) lower compared with that of group B or the positive control (6.58 ± 0.08).

**Chloride ($Cl^-$).** The chloride test results showed no significant (P > 0.05) change in serum chloride concentrationof group B (108.80 ± 0.37), C (112.00 ± 0.32) and D (115.80 ± 1.69), compared with group A or the normal group (104.40 ± 1.21).

**Biocarbonate ($HCO_3$): The Biocarbonate test results obtained, showed that the serum biocarbonate concentration in group C (18.40 ± 0.60) and D (15.60 ± 0.25) were all significantly (P< 0.05), lower compared with that of group A or the normal group (20.80 ± 0.66). More so, the serum bicarbonate concentration foranimals in group D (15.60 ± 0.25) was significantly (P< 0.05) lower compared to that of group Bor the positive control (20.20 ± 0.58).**

## Discussion

Diabetes mellitus is one of the leading causes of renal dysfunction, primarily due to sustained hyperglycemia-mediated oxidative stress, inflammation, and structural injury to renal tubular and glomerular cells. In this study, diabetic induction in Wistar rats produced marked elevations in biomarkers of renal injury (KIM-1, Cystatin-C, urea, creatinine, sodium, and potassium), consistent with established biochemical patterns of diabetic nephropathy. Treatment with stem cell therapy (Beaustem) resulted in significant improvements in these renal indices, demonstrating the therapeutic potential of stem-cell–based interventions in mitigating diabetes-induced kidney damage.

**Kidney Injury Molecule-1 (KIM-1):** KIM-1 is a highly sensitive biomarker of proximal tubular injury and is markedly elevated in diabetic nephropathy. The significant ($p<0.05$) increase observed in the diabetic untreated group (Group B) confirms severe tubular damage. Stem cell treatment (Group D) significantly ($p<0.05$) reduced KIM-1 concentration compared with the diabetic control, indicating tubular repair and reduced epithelial cell injury. This is consistent with evidence that mesenchymal stem cells (MSCs) exert renoprotective effects by attenuating tubular

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apoptosis, reducing inflammation, and enhancing autophagy and mitochondrial repair (He et al., 2024; Han et al., 2023). Recent findings further show that MSC-derived exosomes modulate SIRT1–PGC-1α pathways, promoting tubular recovery (Han et al., 2023).

**Cystatin-C:** Cystatin-C is a sensitive marker of glomerular filtration rate (GFR) and early renal dysfunction. Its significant ($p<0.05$) elevation in the diabetic group (B) reflects impaired glomerular filtration. Stem cell treatment significantly lowered serum Cystatin-C, approaching the effect of standard antidiabetic therapy (Group C). This aligns with previous reports showing that MSC therapy enhances podocyte survival, reduces glomerular basement membrane thickening, and improves GFR by modulating autophagy and restoring glomerular integrity (Hu et al., 2024; Du et al., 2025).

**Urea and Creatinine:** Urea and creatinine are classical biomarkers of renal filtration efficiency. Their pronounced elevation in untreated diabetic rats (Group B) indicates reduced renal clearance and impaired nephron function. Treatment with Beaustem significantly reduced both biomarkers, with creatinine levels showing a stronger degree of recovery. These findings support the hypothesis that stem cells promote nephron structural repair and enhance glomerular and tubular function through paracrine effects, including release of growth factors, anti-inflammatory cytokines, and extracellular vesicles (Habiba et al., 2024; Du et al., 2025). Meta-analyses from 2024–2025 further confirm that stem cell therapy improves creatinine clearance and reduces nitrogenous waste accumulation in diabetic kidney disease (Du *et al*., 2025).

**Electrolytes (Sodium, Potassium, Chloride, Bicarbonate) :** The elevation of sodium and potassium in the diabetic control group indicates tubular transport dysfunction, commonly linked to hyperglycemia-induced oxidative injury to $Na^+/K^+$ ATPase channels. Following stem cell treatment (Group D), potassium levels significantly normalized, while sodium levels showed less pronounced improvement, though still comparable to the standard drug group. These observations agree with reports that stem cell–based therapies restore tubular ion channel function by reducing inflammation, fibrosis, and oxidative stress in renal tissues (Ouchi et al., 2023; Xiong et al., 2024). Chloride levels did not significantly change among diabetic or treated groups, which is consistent with earlier studies showing that chloride is less sensitive to early diabetic changes. In contrast, bicarbonate levels decreased in diabetic animals, reflecting metabolic acidosis commonly associated with diabetic renal impairment. Stem cell treatment significantly improved bicarbonate levels, suggesting better acid–base regulation, possibly mediated by enhanced tubular reabsorption and reduced renal oxidative stress (Habiba *et al.,* 2024).

## Conclusion

This study demonstrated that stem cell therapy using Beaustem exerts significant renoprotective effects in Wistar rats with diabetes-induced renal dysfunction. Diabetes markedly elevated key biomarkers of kidney injury—including KIM-1, Cystatin-C, urea, creatinine, sodium, and potassium—reflecting profound glomerular and tubular damage.

Treatment with Beaustem resulted in substantial reductions in these biomarkers, approaching or surpassing the improvements observed in animals treated with standard antidiabetic therapy. Biochemically, these findings indicate that Beaustem promotes restoration of renal function through multiple mechanisms that have been supported in recent literature: reduction of tubular epithelial injury, enhancement of glomerular filtration, modulation of inflammatory pathways, improvement in mitochondrial function, and mitigation of oxidative stress. The therapy also improved electrolyte balance and acid–base regulation, further confirming its role in restoring normal physiological kidney function in diabetic conditions.

Overall, the results provide strong evidence that stem cell therapy holds promise as a viable and potent intervention for diabetic kidney disease. Beaustem, in particular, demonstrated the capacity to reverse biochemical markers of renal impairment, supporting its potential application as an adjunct or alternative therapeutic strategy in the management of diabetes-related renal complications. Further studies, including molecular analysis and clinical trials, are recommended to fully elucidate its mechanisms and translational potential

**Table 2.0**: Statistical Result m

| | KIM1 (ng/ml) | CYS-C (ng/ml) | Urea Mmol/L | Creat µmol/L | Na Mmol/L | K Mmol/L | $Cl^-$ Mmol/L | $HCO_3$ Mmol/L |
|---|---|---|---|---|---|---|---|---|
| | 1.54 | 0.60 | 2.68 | 36.74 | 132.40 | 3.10 | 104.40 | 20.80 |
| **A** | ± | ± | ± | ± | ± | ± | ± | ± |
| | 0.04 | 0.07 | 0.07 | 0.79 | 0.67 | 0.32 | 1.21 | 0.66 |
| | * | * | * | * | * | * | | |
| **B** | 31.20 | 34.40 | 7.54 | 106.58 | 40.60 | 6.58 | 108.80 | 20.20 |
| | ± | ± | ± | ± | ± | ± | ± | ± |
| | 1.16 | 0.24 | 0.068 | 0.27 | 1.21 | 0.08 | 0.37 | 0.58 |
| | *a | *a | *a | *a | *a | *a | | *a |
| **C** | 12.40 | 11.20 | 4.32 | 51.26 | 141.00 | 4.24 | 112.00 | 18.40 |
| | ± | ± | ± | ± | ± | ± | ± | ± |
| | 0.51 | 0.66 | 0.21 | 0.32 | 0.45 | 0.07 | 0.32 | 0.60 |
| | *ab | *a | *a | *ab | * | *a | | |
| **D** | 17.20 | 13.40 | 4.06 | 45.36 | 141.80 | 4.24 | 115.80 | 15.60 |
| | ± | ± | ± | ± | ± | ± | ± | ± |
| | 0.20 | 0.25 | 0.08 | 0.74 | 0.49 | 0.07 | 1.69 | 0.25 |

Values are expressed as mean ± standard error of mean (SEM), n = 5. $*$ = significant ϵ $p < 0.05$ compared with group 1 (A); $* a$ = significant ϵ $p < 0.05$ compared with group 2 (B); $* ab$ = significant ϵ $p < 0.05$ compared with group 3 (C)
**Keys**: KIM1= Kidney Injury Molecule 1, cystc = Cystantin-C, Creat = Creatinine, Na = Sodium,K= Potassium, $Cl^-$ = chlorine, HCO3 = Biocarbonate.

**Compliance with ethical standards:** This research adheres to ethical standards

**Disclosure of conflict of interest:** *The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.*

**Statement of ethical approval:** *Ethical approval for this study was provided by the Research and Quality Control Unit of Federal University Otuoke in line with the guidelines of the European Convention for the Protection of Vertebrate Animals used for experimental and other Scientific Purpose, ETS-123*

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