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Evaluation of Kidney Function Parameters in Diabetic Rats Following Virgin Coconut Oil Diet

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Background: Diabetes mellitus (DM) leads to disruption of kidney function parameters (KFPs) which are markers of kidney diseases, especially nephropathy. Virgin coconut oil (VCO) has been implicated in playing a significant role in DM management. However, its role on KFPs in DM is scarce.

Aim: To evaluate the kidney function parameters following VCO diet in diabetic rats.

Materials and methods: : Twenty-five (25) male rats of 150 – 200 g were divided into 5 groups (n=5): Non-diabetic control (Group 1), diabetes control (Group 2), diabetes + metformin (Group 3), diabetes + 10% VCO (Group 4) and diabetes + 20% VCO (Group 5). Apart from Group 1, other groups were given intraperitoneally 50 mg/kg of streptozotocin to induce diabetes mellitus. After 72 hours, fasting hyperglycaemia was confirmed by glucose oxidase method. All the rats were fed normal rat chow for 8 weeks. At 8th week, serum and urine samples were analysed for biochemical analysis. After 8 weeks, Group 1 and Group 2 continued to be fed on normal rat chow while other groups were treated with diets (VCO) or drug (metformin) for 4 weeks. At 12th week, urine samples were collected for biochemical analysis, the rats were sacrificed, and blood samples were collected by cardiac puncture.

Results: There were significant differences in some KFPs in diabetes control (Group 2) compared to other experimental groups. However, there was no significant difference in glomerular filtration rate (GFR) and serum sodium in all the groups.

Conclusion: VCO supplementary diet improved the altered KFPs and could be a therapy for kidney problems.

INTRODUCTION

Diabetes mellitus is a chronic, metabolic disorder that affects almost every system of the body due to a defect in peripheral insulin action, insulin secretion, or both.[1] Diabetes mellitus has been known with a number of different long-term macrovascular complications (cardiovascular problems) and microvascular complications such as kidney dysfunction, neuropathy and retinopathy.[2] Among these complications, kidney dysfunction is a prevalent and serious complication of both type 1 and type 2 diabetes mellitus.[3] However, the abnormal kidney functions are characterized by elevation of metabolic products such as creatinine, blood urea nitrogen and uric acid in the blood as well as abnormal loss of electrolytes and proteins (especially albumin in the urine) due to kidney impairment.

Consequently, the kidney dysfunction can progress to end-stage renal disease due to persistent hyperglycaemia in untreated diabetic patients.[4] Therefore, hyperglycaemia is the main initial factor in the development of kidney structural changes that eventually cause abnormal functions of the kidney in diabetic individuals. Thus, glycaemic control through diet is the major target for therapy in diabetic patients with a potential of developing kidney disease.[5] Adequate glycaemic control through diet after diagnosis of diabetes will reduce the risk of developing kidney dysfunction and delay the progression of renal disease.

Apart from traditional and modern medicines, considerable interests of consuming certain diet to fight against several diseases have appeared. For instance, virgin coconut oil (VCO) consumption in tropical countries for thousands of years and studies done on native diets have shown that this

population are healthy.[6] The Tokelauans that live in the South Pacific are the best example of this population.[6] They consume more than 60% of their calories from coconuts and are one of the biggest consumers of saturated fat in the world.[6,7]

In previous studies, it was reported that coconut oil, a source of tocotrienols, capric acid, caproic acid, and lauric acid, has anti-diabetic and anti-oxidant properties but its effects on renal function parameters in streptozotocin-induced diabetic rats has not been clarified.[1,8] In the past, study has been done on effect of virgin coconut oil diet on renal function in alloxan-induced diabetic rat for a short period of 3 weeks.[8] Therefore, due to a short duration of the previous study, more information is needed to be provided by examining the effect of virgin coconut oil diet on renal function in a long duration of 8th to 12th week with a comparison to a positive control group. Thus, the aim of this study was to evaluate the renal function parameters in diabetic rats following virgin coconut oil diet and metformin treatment for a longer duration.

MATERIALS AND METHODS

Preparation of Virgin coconut oil (VCO)

Dried and matured coconut fruits were purchased from Okuku, and Bekwarra markets in the northern part of Cross River State, Nigeria. The VCO was extracted by wet extraction method.[8,9] The preparation of the 10% and 20% virgin coconut oil meal was done on regular demand.

Animals

Male albino rats with body weight of 170–200 g were used for this study. The rats were obtained from animal holding unit of Physiology Department, Cross River University of Technology, Okuku Campus, Cross River State. They were kept in a standard animal facility under controlled environmental conditions at room temperature (27°C), humidity (55±5%) and 12-hour light-dark cycle. The rats received a standard pellet diet (Vital feed) and water *ad libitum* for 2 weeks to acclimatize. The animals were divided into 5 groups: Group 1 – non-diabetic control group (negative control), Group II – diabetes control group, Group III – metformin treated group (positive control), Group IV - 10% VCO treated group, Group V - 20% VCO treated group. This study was carried out in absolute compliance with the guidelines of the ethical committee of the Faculty of Basic Medical Sciences, Cross River University of Technology.

Induction of diabetes mellitus

The animals in all the groups fasted for 12 hours. The control group was given 0.2 mL of 0.01M citrate buffer via intraperitoneal route while the rats in the other groups were rendered diabetic by intraperitoneal injection of 50 mg/kg of streptozotocin (Santa Cruz Biotechnology, USA) dissolved in 0.01M citrate buffer, pH 4.5.[10] In order to control the hypoglycemic shock after the streptozotocin administration, 5% glucose solution was administered orally to the diabetic rats for two days. After 3 days of streptozotocin injection, blood glucose concentrations were determined via AccuChek Active glucometer to confirm hyperglycaemia (the major feature of diabetes mellitus). Animals with blood glucose concentration of 250 mg/dl and above were considered for this study.[10] Apart from confirmation of the diabetes mellitus, the blood glucose was also measured in all the groups at 8 and 12 weeks.

Experimental Procedure

After diabetes induction, all rats in each group were fed normal rat chow for 8 weeks. At 8 weeks, kidney function was assessed in all the animals by estimation of the albumin and creatinine concentrations in the urine to confirm kidney dysfunction before any treatment. In addition, the non-diabetic control and diabetes control groups continued to be fed normal rat chow while the other groups were treated with different diets (10% and 20% VCO diet) or reference drug (metformin, 100 mg/kg) for another 4 weeks, i.e. till 12th week

At the 12th week, the animals were placed in individual metabolic cages for 24 hours to collect urine samples for glomerular filtration rate (GFR), albumin and other kidney function parameters analyses. The urine samples volumes were measured, centrifuged and aliquots of the samples were stored in 5-mL sample bottles in the refrigerator (2°C – 8°C). Thereafter, all the rats fasted for 12 hours, blood samples were collected via cardiac puncture into 5-mL EDTA sample bottles after anaesthetising the animals with diethyl ether. The blood samples were centrifuged at 3000 g for 10 min to obtain clear sera for biochemical analysis of electrolytes, serum creatinine, serum albumin and blood urea nitrogen. The kidneys from each animal were excised, washed in cold saline and weighed on weighing balance.

Determination of GFR

The GFR was calculated from the estimation of

creatinine in the serum and urine as follows:

$$\text{Creatinine Clearance or GFR} = \frac{\text{Mg creatinine/dl urine} \times \text{mL urine 24 hrs}}{\text{Mg creatinine/dl serum} \times 60 \text{ min} \times 24 \text{ hrs}} [\text{mL/min}]$$

Biochemical Analysis

The kidney function parameters and electrolytes were evaluated with specific analytical kits from Fortress Laboratories Limited (United Kingdom) by spectrophotometry method via microplate spectrophotometer (Spectra Max Plus, Molecular device, USA).

Statistical Analysis

The statistical data were presented in bar charts with mean ± SEM and analysed using two-way analysis of variance (ANOVA) with Bonferroni's test (posthoc test) via GraphPad Prism 5 software. Also, paired t-test was conducted to analyse the significant difference between the parameters at 8 and 12 weeks. The results were considered significant at $p<0.05$.

RESULTS

Kidney weight and urine volume

The kidney weights, body weights, and kidney weight to body weight ratio are shown in **Table 1**. The kidney weights of the diabetic rat treated with metformin, 10% VCO diets and diabetes control except that of diabetic rats treated with 20%VCO were significantly increased ($p<0.05$) from that of the non-diabetic control group. There was a significant decrease between kidney weights of diabetic rat treated with metformin, 10% VCO diets and 20% VCO diet compared to that of the diabetes control group. However, there was a significant difference in the body weight but no significant difference in the kidney weight to body weight ratio.

The urine volumes are shown in **Table 2**. The urine volumes of all the test groups at 8 weeks of the experiment were significantly increased from that of the non-diabetic control group. In addition, the urine volumes of the diabetic groups treated with metformin and VCO at the 12th week of the experiment were also significantly different from that of the non-diabetic control and diabetic control groups. The urine volume of diabetes control group was also significantly increased ($p<0.001$) compared to that of the non-diabetic control group at 12 weeks.

Blood glucose

The blood glucose levels in all the test groups were significantly increased compared to non-diabetic control group at 8 weeks ($p<0.001$). The blood glucose of diabetic groups treated with metformin and VCO were significantly decreased compared to diabetes control group but significantly increased compared to non-diabetic control group at 12 weeks. The blood glucose of diabetes control group was significantly increased ($p<0.001$) when compared to non-diabetic control group at the 12th week as shown in **Table 2**.

Creatinine and GFR

The serum creatinine in diabetic groups treated with metformin and VCO were significantly increased at the 8th week but not significantly increased at the 12th week when compared to non-diabetic control group. The serum creatinine of diabetic control group was significantly increased at both 8 and 12 weeks when compared to the non-diabetic control group. However, the serum creatinine in the diabetic groups treated with metformin and VCO were significantly decreased ($p<0.05$) when compared to diabetic control group at the 12th week as shown in **Table 2**. The urine creatinine in the diabetic groups treated with metformin and VCO were significantly decreased compared to the non-diabetic control group at 8 and 12 weeks. Conversely, there was no significant difference between the urine creatinine of the diabetic groups treated with metformin and VCO compared to the diabetic control group at 8 weeks and 12 weeks. The GFRs of the diabetic groups treated with metformin and VCO were not significantly different when compared to the non-diabetic control group and diabetic control group at 8 and 12 weeks. At the 12th week, the GFR of the diabetic control group slightly reduced but not significant as shown in **Table 2**.

Albumin and BUN (Blood Urea Nitrogen)

The serum albumin of the diabetic rats treated with VCO diets significantly decreased from that of the non-diabetic control group, diabetic control group and diabetic rats treated with metformin at 8 weeks.

At 12 weeks, the serum albumin of the diabetic rats treated with VCO diets was significantly increased than the diabetic control group and diabetic rats treated with metformin but not significant with non-diabetic control group. As shown in **Table 2**, the urine albumin level of all the diabetic groups (test groups) significantly increased compared to the non-diabetic control group at 8 weeks. Also, the urine albumin level was only significant ($p<0.01$) in the diabetic control group when compared to the control group at 12 weeks.

Table 1. The kidney weight, body weight and the ratio of kidney to body weight in the control, diabetes control and diabetic groups treated with metformin and VCO (mean±SEM, n=5)

Groups Parameters	Non-diabetic Control	Diabetes Control	Diabetes + Metformin	Diabetes + 10% VCO	Diabetes + 20% VCO
Kidney weight (g)	1.18±0.02	1.59±0.06***	1.38±0.04***###	1.41±0.02***###	1.30±0.03***###
Body weight (g)	181.40±3.67	205.70±5.50*	217.40±3.01**	219.50±2.66**	202.40±2.97*
Kidney weight/ Body weight ratio	0.007±6.63(x10^{-5})	0.008±9.27(x10^{-5}) ns	0.006±1.21(x10^{-4}) ns	0.006±6.00(10^{-5}) ns	0.006±1.34(x10^{-5}) ns

***p<0.001, **p<0.01, *p<0.05 (significantly different from non-diabetic control group)
###p<0.001 (significantly different from diabetic control group)
ns: not significant

The results of the BUN are shown in **Fig. 1**. The BUN of diabetic groups treated with metformin and VCO were significantly decreased (p<0.001) compared to that of the diabetic control group but not significant when compared to the non-diabetic control group. On the other hand, the BUN of the diabetic control group were significantly increased (p<0.001) compared to that of the non-diabetic control group.

Electrolytes (potassium and sodium ions)

The serum potassium of the diabetic control and diabetic rats treated with metformin groups were significantly decreased (p<0.001) compared to non-diabetic control group. In addition, the serum potassium of the diabetic rats treated with either 10% or 20% VCO diet was significantly increased compared to that of the diabetic control group but not significant from non-diabetic control group as shown in **Fig. 2**.

As shown in **Fig. 3**, the serum sodium level in the diabetic groups treated with metformin and VCO was not significantly different from that of the non-diabetic control and diabetes control groups.

DISCUSSION

Kidney function parameters are indices of assessing the functionality of the kidney in health and diseases. Consequent alterations of these parameters in diabetes mellitus indicated that diabetes mellitus causes abnormal functions of the kidney and kidney impairment in diabetics. In this study, there were different alterations in the kidney function parameters at the 8th week before treatment and the 12th week after treatment.

The histomorphological structural changes such as glomerular basement membrane thickening and mesangial matrix expansion due to sustained hyperglycaemia probably contributed to the increased kidney weights. The kidney weight was significantly increased in the diabetic control group compared to non-diabetic control group. The increase in kidney weight was in accordance with the findings of earlier research studies.[11,12] It has been described that kidney enlargement leads to increased kidney weight in diabetes mellitus due to certain factors like glucose over-utilization, glycogen accumulation, lipogenesis and protein synthesis in the kidney tissue.[13] Actually, it may also be as a result of glomerular hypertrophy.[14] The VCO diets administered to the diabetic rats successfully prevented the enlargement of the kidney.

Table 2. The urine volume, blood glucose, GFR, serum and urine creatinine and albumin in non-diabetic control, diabetes control and diabetic groups treated with metformin and VCO at 8 weeks and 12 weeks of the experiment (mean±SEM, n=5)

Parameters	Weeks	Non-diabetic control	Diabetes control	Diabetes + metformin (100 mg/kg)	Diabetes + 10%VCO	Diabetes + 20% VCO
Urine volume (ml)	8	5.00±0.97	25.70±2.50***	24.10±2.27***	24.80±2.79***	25.30±4.98***
	12	5.00±1.51	35.80±4.97***	18.20±4.29*#	17.60±2.03*#	16.30±1.01*#
Blood glucose (mmol/l)	8	5.11±0.15	17.58±0.85***	18.08±1.88***	17.82±1.11***	17.57±1.27***
	12	5.12±0.33	24.62±1.07***&	11.98±1.29***###&	11.39±1.43***###&	11.49±1.32***###&
Serum creatinine (mg/dl)	8	1.00±0.07	4.78±0.58**	3.89±0.51*	3.75±0.50*	3.93±0.11*
	12	1.09±0.07	5.42±0.71***	2.30±0.18#&	2.28±0.17#&	2.13±0.20#&
Urine creatinine (mg/dl)	8	430.06±63.65	336.23±52.75*	238.94±26.04**	229.92±16.17**	275.18±22.15*
	12	483.59±57.73	215.64±48.56***	281.49±59.70**	243.15±11.45**#	251.12±6.93**#
Glomerular filtration rate (GFR) (ml/min)	8	1.21±0.16	1.16±0.13 ns	1.00±0.10 ns	1.00±0.12 ns	1.16±0.18 ns
	12	1.14±0.17	0.89±0.16 ns	1.17±0.09 ns	1.15±0.01 ns	1.14±0.11 ns
Serum albumin (mg/l)	8	35.07±2.75	31.58±5.10	35.69±2.62	21.34±4.87*#	18.00±2.04*#
	12	30.34±2.93	25.53±1.99	23.18±3.73&	32.70±3.33##&	32.97±2.98##&
Urine albumin (mg/l)	8	0.20±0.20	34.40±12.30*	40.00±11.51**	44.80±11.76**	40.60±12.20**
	12	0.20±0.20	46.80±10.46**	22.40±4.02	27.20±3.87	28.80±8.00

***p<0.001, **p<0.01, *p<0.05 (significantly different from non-diabetic control group)
###p<0.001, ##p<0.01, #p<0.05 (Significantly different from diabetic control group)
&Significant difference between 8th and 12th week (paired t-test)
ns: not significant

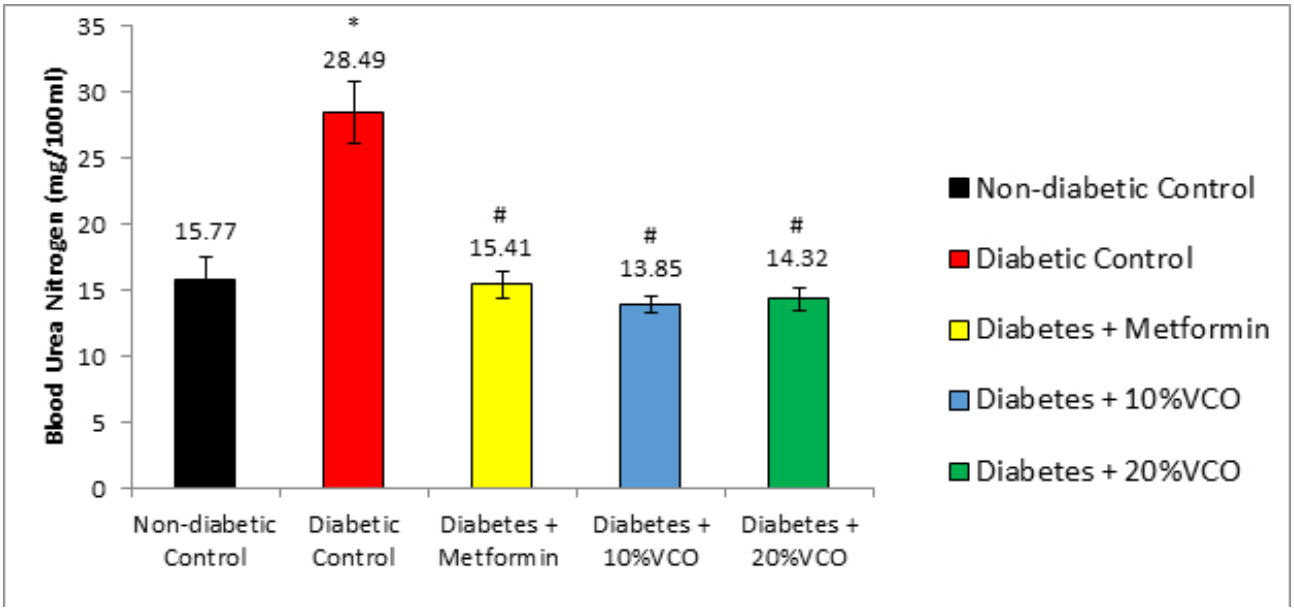

Figure 1. The blood urea nitrogen in the non-diabetic control, diabetic controls, and other diabetic groups following VCO diet or metformin.
*Significantly different from control group at $p<0.05$
#Significantly different from diabetic control group at $p<0.0$

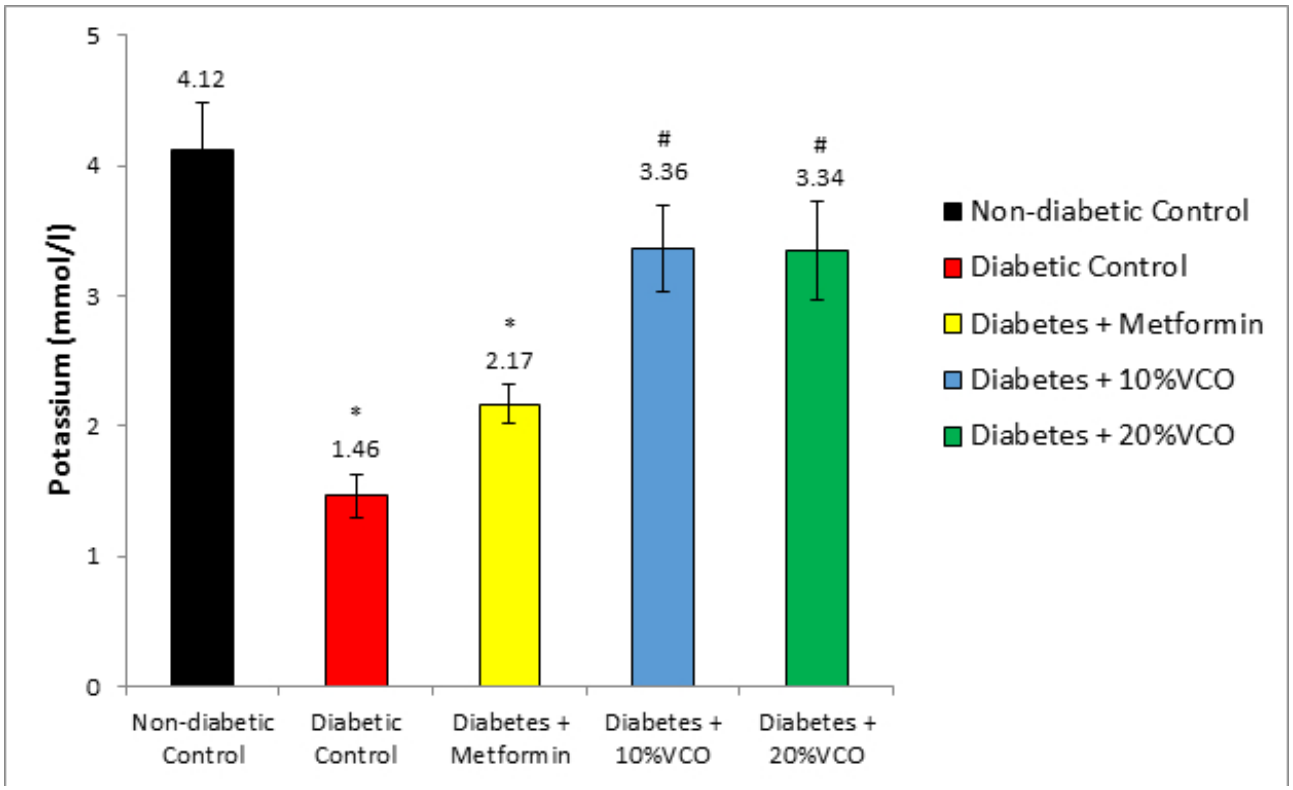

Figure 2. The serum potassium of non-diabetic control, diabetic control, and other diabetic groups treated with VCO diet or metformin.
*Significantly different from control group at $p<0.05$
#Signficantly different from diabetic control group at $p<0.05$

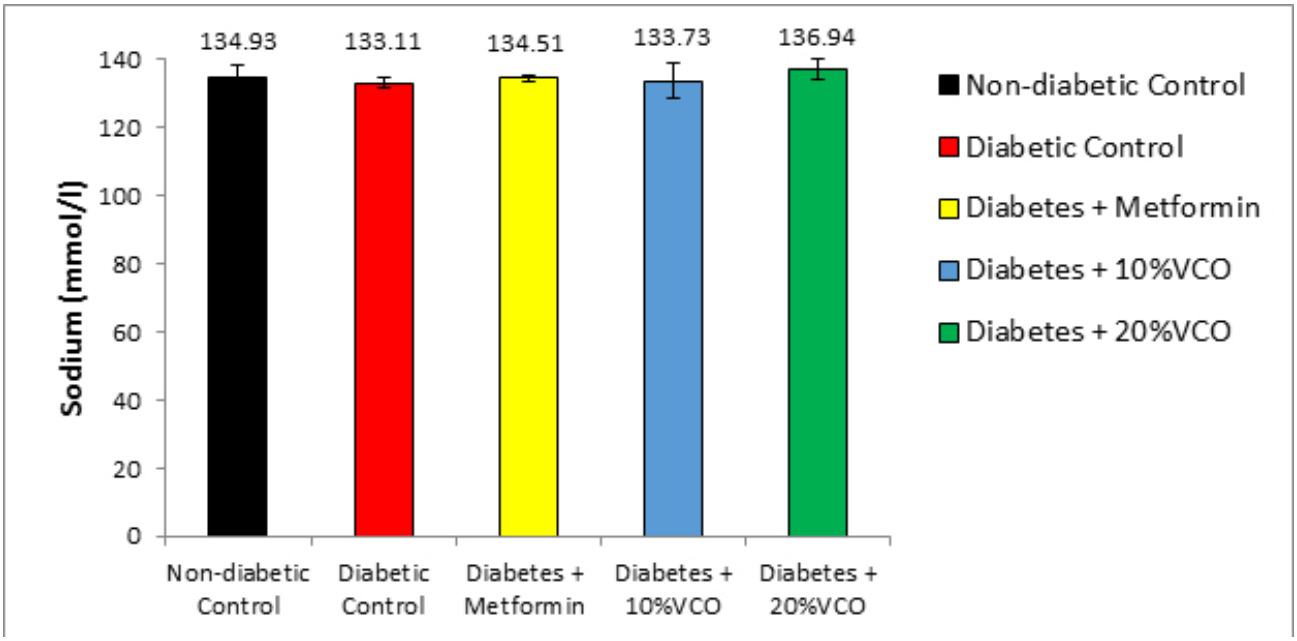

Figure 3. The serum sodium levels in the non-diabetic control , diabetic control and other experimental diabetic groups.

Hyperglycaemia is a major phenomenon in diabetes mellitus as a result of the destruction of beta cells of the pancreas or insulin resistance. Therefore, there was a significant increase in blood glucose level in all the diabetic groups compared to non-diabetic control at 8 weeks and 12 weeks. Apart from hyperglycaemia, polyuria is also a feature in diabetes mellitus due to osmotic diuresis. The increased urine volume observed in this study was in accordance with osmotic diuresis associated with glucosuria.[15]

Moreover, the increased serum creatinine and BUN observed in the diabetic control group compared to diabetic treated groups and non-diabetic control group in this study at 12 weeks, was in accordance with previous studies where the same parameters were measured in diabetic patients.[16,17] This increase can be suggested to be due to reduced excretory and regulatory role of the kidney to maintain a constant homeostasis of these parameters in the untreated diabetic patient compared to treated diabetic patients.[17] Thereby, causing an upsurge of these parameters in the serum of diabetic control group.

Furthermore, GFR was slightly reduced in diabetic control group though not significant in comparison to other groups. In spite of this, it implies that GFR in the diabetic control group may be significantly decreased from other groups with an increase in duration of the experiment. In addition, due to kidney compensatory mechanisms in regulating GFR, a significant reduction in GFR may not be observed in diabetic individuals unless there is a severe damage to the kidney.

In fact, alteration of albumin concentration in serum and increased urinary albumin in diabetic complications have been associated with faster kidney disease progression if unchecked or managed. However, it was also observed in this study that there was an increased urine albumin in diabetic control group although the increase was not significant compared to diabetic groups treated with metformin and VCO but was significant compared to the non-diabetic control group. On the other hand, there was a significant decrease in serum albumin of diabetic groups treated with metformin and VCO compared to diabetic control and non-diabetic control groups. This probably suggests that untreated diabetes mellitus caused dysfunction of the kidney which led to gradual loss of serum albumin as a result of loss of negatively charged glycosaminoglycans in the cellular basement membrane and the subsequent enlargement of basement membrane pore size, thus leading to albuminuria which was in accordance with the previous reports.[5,17,18] Interestingly, the serum albumin of the diabetic rats treated with VCO and metformin was significantly reduced at 8 weeks, but at 12 weeks of the experiment, the serum albumin had returned to normal level. Thus, this revealed the therapeutic potency of VCO and metformin in

ameliorating kidney function as reported in previous researchers.[19]

Also, it was observed that there was a significant decrease in serum potassium of diabetic control group and diabetic treated group with metformin compared to non-diabetic control and diabetic groups treated with VCO. This significant decrease might be due to loss or reduced cellular uptake of potassium as well as loss of the regulatory role of the kidney in handling potassium absorption. In addition, insulin is a hormone that enhances cellular uptake of potassium ion.[15] Therefore, it is reasonable to suggest that in untreated diabetes, there may be reduced cellular uptake of potassium ion due to insulin resistance, thus leading to hypokaelemia as a result of increased potassium loss in urine. On the other hand, the serum sodium level was constant without any significance in all the experimental groups, and this may probably be due to other peripheral regulation of sodium level in the blood apart from the kidney.[20] In this study, there was evidence that showed alterations in kidney function parameters in the diabetic control group but dietary VCO ameliorated the alterations.

CONCLUSIONS

Diabetes mellitus has degenerative and destructive effects on the kidneys by altering the kidney functions but these effects occur over a longer duration of time and can be prevented, reversed or ameliorated by VCO supplementary diet.

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Оценка параметров функции почек у крыс с диабетом после диеты с приёмом кокосового масла

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Введение: Сахарный диабет (СД) приводит к нарушению параметров функции почек (ПФП), которые являются маркерами заболеваний почек, особенно нефропатии. Считается, что кокосовое масло (КМ) играет значительную роль в контроле СД. Однако его влияние на ПФП при СД незначительно.

Цель: Оценить параметры функции почек после диеты с КМ у крыс с диабетом.

Материалы и методы: Двадцать пять (25) самцов крыс весом 150-200 грамм были разделены на 5 групп (n = 5): недиабетическая контрольная группа (группа 1), диабетическая контрольная группа (группа 2), диабет + группа метформина (группа 3), диабетическая группа + 10 % КМ (группа 4) и диабетическая группа + 20 % КМ (группа 5). В дополнение к группе 1 стрептозотоцин 50 мг / кг вводили внутрибрюшинно другим группам для индукции диабета. Через 72 часа гипергликемия натощак была подтверждена методом глюкозооксидазы. Всех крыс кормили стандартным кормом для крыс в течение 8 недель. По истечении восьмой недели группу 1 и группу 2 продолжали кормить стандартным кормом для крыс, в то время как другие группы были поставлены на диету с КМ или получали лекарственный препарат (метформин) в течение 4 недель. На 12 неделе брали пробы мочи для биохимического анализа, крыс умерщвляли и брали пробы крови путём пункции сердца.

Результаты: В контрольной группе с диабетом (группа 2) были значительные различия в некоторых ПФП по сравнению с другими экспериментальными группами. Тем не менее, не было значительных различий в скорости клубочковой фильтрации (СКФ) и натрия сыворотки во всех группах.

Выводы: Режим приёма КМ улучшил модифицированный ПФП и вполне может быть использован для лечения проблем с почками.