NISEB JOURNAL Vol. 12, No. 2, April 30, 2012
Printed in Nigeria

1595-6938/2012 $5.00 + 0.00

http://www.nisebjournal.org

NISEB 2012042/12203

Kidney function status in Nigerian patients with co-morbidity of diabetes mellitus and malaria

J. C. Anionye[1], R. O. Edosa[1], E. F. Omorowa[1], E. C. Onyeneke*[2], N. U. Okwadje[1], O. E. Onovughakpo-Sakpa[3] and A. I. Anekwe[2]

[1]Department of Medical Biochemistry, College of Medical Sciences, University of Benin, Benin City, Nigeria.
[2]Department of Biochemistry, Faculty of Life Sciences, University of Benin, Benin City, Nigeria.
[3]Department of Chemical Pathology, College of Medical Sciences, University of Benin, Benin City, Nigeria

(Received March 26, 2012; Accepted April 22, 2012)

ABSTRACT: This study was carried out to investigate the kidney function status of patients with co-morbidity of diabetes mellitus and malaria in Benin metropolis, Southern Nigeria, to ascertain if there is any significant renal dysfunction in them. Serum levels of urea and creatinine were assayed from a total of 80 subjects (40 test subjects and 40 control subjects) of both sexes with their age ranging from 40-76 years. The 40 test subjects were known diabetics that presented in the clinic with symptoms of malaria infection. Results obtained reveal a statistically significant increase ($P< 0.05$) in serum creatinine and urea levels in the test subjects (482.06±40.54μmol/L; 12.03±0.32mmol/L, respectively) when compared to that of the control (84.23±7.95 μmol/L; 4.60±0.34 mmol/L, respectively), irrespective of sex, age, degree of parasitaemia and fasting blood sugar. The increase in serum creatinine level was however significantly higher ($P< 0.05$) in the males when compared to the females. The urea:creatinine ratio of the test subjects (0.02±0.01 : 1) apart from being significantly less than ($P<0.05$) that of the control subjects (0.05±0.04 : 1) also fell within the range in keeping with patients with intrinsic renal failure (<0.04 : 1) as opposed to those with post-renal/normal (0.04 - 0.1 : 1) and pre-renal (>0.1 : 1) renal failure. Analyses of the results indicate that there is significant renal dysfunction in patients in Southern Nigeria with co-morbidity of diabetes mellitus and malaria.

Keywords: Diabetes mellitus, malaria, renal dysfunction, co-morbidity.

Introduction

Diabetes and malaria remain devastating global health problems and when they coexist in a patient, they may contribute negative effects against the patient's wellbeing in a synergistic way (Charles, 1998; Cavanaugh, 2007). In tropical countries like Nigeria, *P. falciparum* is the species of *plasmodium* mostly implicated in the causation of severe malaria. Diabetes mellitus on the other hand is one of the common non-communicable diseases rapidly increasing in prevalence in Sub-Saharan Africa (Muller *et al.*, 2005).

*Author to whom all correspondence should be addressed.
E-mail: johnchux@yahoo.com

The metabolic disorder, diabetes mellitus is characterized by chronic hyperglycemia caused by persistent derangement in carbohydrate, fat and protein metabolism that is associated with absolute or relative deficiencies in insulin secretion, insulin action or both (Alberti and Zimmet, 1998 and Charles, 1998).

Type-2 diabetes, also known as non-insulin-dependent diabetes mellitus (NIDDM) is the commonest variety worldwide accounting for about 70-90% of the diabetic cases (Manoj, 2001) and usually affects people of over the age of 40 years (Knowler *et al*., 2002). Type 2 diabetes mellitus increases susceptibility to common infections (Muller *et al*., 2005). The increase in incidence of diabetes in developing countries equally follows the trend of urbanization and life style changes, most importantly, western style "diet" (Gautier *et al*., 2001). There is also evidence that diabetes mellitus is going to be epidemic in many developing and industrialized countries (Manoj, 2001).

Diabetic patients are at an increased risk of developing specific complications including: nephropathy, retinopathy, neuropathy and atherosclerosis; with diabetic nephropathy occurring in approximately one – third type 2 diabetics (Rehman, *et al.*, 2005). In diabetic nephropathy, a number of serum markers are known to be deranged with significant morbidity and mortality (Tanigushi *et al*., 2003; Puepet *et al*., 2003).

Malaria, a mosquito – borne infectious disease of humans is caused by a eukaryotic protist of the genus *Plasmodium.* Malaria is the most common vector borne parasitic disease of the globe (WHO, 2000) and a major public health problem in more than 100 countries around the globe with more than 2.5 billion people at risk causing about 3 million deaths annually (Snow, *et al*., 2005). Almost all complications and deaths from malaria are caused by *Plasmodium falciparum* (Trampuz *et al*., 2003). *Plasmodium falciparum* malaria alone causes an estimated 1 million deaths annually (Lopez *et al*., 2006).

Diabetes mellitus increases susceptibility to common infections, malaria being one of them (Muller *et al*., 2005). Diabetes mellitus and malaria infection from indications can independently lead to renal impairment (Woodrow *et al.,* 1994; Nand *et al*,. 2001).

This study was carried out to investigate the kidney function status of patients with co-morbidity of diabetes mellitus and malaria in Benin metropolis, Southern Nigeria, to ascertain if there is any significant renal dysfunction in them.

Materials and Methods

Study subjects: A total of 40 known diabetic subjects who reported ill with signs and symptoms suggestive of malaria and had not been placed on any anti – malaria drug served as test subjects for the study. They were drawn from both sexes aged between 40 – 76 years, attending Time Hospital, Cicigide Clinic, Mount Gilead Clinic and God's Care Hospital – all in Benin metropolis, Edo State. Forty (40) apparently healthy age – matched individuals, who did not test positive to having the malaria parasite or diabetes were used as control subjects. Informed consent was obtained from all the volunteers and adequate approval was given by the personnel in charge of ethics in the health institutions before the study commenced. The subjects are all Nigerians, resident in and around Benin City. Those with confounding ailments and those on drugs that may affect the results of this study were excluded from the study. They were divided into four different age groups of: 40-49, 50-59, 60-69, and 70-79 years.

Sample collection and preparation: The subjects were first tested for malaria parasite and diabetes. Malaria parasite density was determined by making Giemsa stained thick films of blood samples obtained from the patients. Giemsa stained thin films confirmed the malaria parasite as *Plasmodium falciparum.* The malaria parasite density was determined and graded according to the method described by Cheesbrough (1998). The subjects' blood glucose level was determined using the glucometer.

Fasting venous blood sample was then collected from each subject (both test and control) into plain sample tubes using 5ml syringes. The samples in the plain tubes were spun at 3000rpm for 5 minutes to derive serum which was stored at -4^0C if not analyzed immediately.

Assay Methodology: Creatinine was assayed by Bartels and Bohmer (1972) method using the Teco diagnostic Kit while urea was analysed by Berthelots reaction using Randox kit (Weatherburn, 1967).

Statistical analysis: All results were expressed as mean ± standard error of mean (SEM). The Data was analyzed using the student's t – test from the statistical package for social sciences (SPSS). Means of the same row or column with different superscript letters differ significantly at 95% level of confidence ($P<0.05$).

Results and Discussion

Table 1: Urea and Creatinine levels in diabetes patients with malaria infection

	Control Subjects	Test Subjects
Creatinine (μmol/l)	84.23 ± 7.95^a	482.06 ± 40.54^b
Urea (mmol/l)	4.60 ± 0.34^a	12.03 ± 0.32^b
Urea : Creatinine ratio (mmol/l : μmol/l)	$0.05 \pm 0.04 : 1^a$	$0.02 \pm 0.01 : 1^b$

Values are expressed as Mean ± SEM. Means of the same row with different superscript letters differ significantly ($P<0.05$).

Table 2: Urea and Creatinine levels according to sex of diabetes patients with malaria infection

Sex	Creatinine (μmol/l)		Urea (mmol/l)	
	Control Subjects	Test Subjects	Control Subjects	Test Subjects
M	78.31 ± 11.09^a	497.08 ± 49.99^b	3.53 ± 0.31^a	11.85 ± 0.36^b
F	91.47 ± 11.43^a	461.73 ± 68.79^b	5.91 ± 0.52^a	12.28 ± 0.5^b

Values are expressed as Mean ± SEM. Means of the same row in the same column with different superscript letters differ significantly ($P<0.05$).

Table 3: Comparison of Urea and Creatinine levels according to sex of diabetes patients with malaria infection

Creatinine (μmol/l)		Urea (mmol/l)	
M	F	M	F
497.08 ± 49.99^a	461.73 ± 68.79^b	11.85 ± 0.36^a	12.28 ± 0.5^a

Values are expressed as Mean ± SEM. Means of the same row in the same column with different superscript letters differ significantly ($P<0.05$).

Table 4: Urea and Creatinine levels according to age of the diabetes patients with malaria infection

Age (yrs)	Creatinine (μmol/l)		Urea (mmol/l)	
	Control Subjects	Test Subjects	Control Subjects	Test Subjects
40-49	112.41 ±9.01[a]	517.93± 94.60[b]	4.47±0.59[a]	12.10±0.63[b]
50-59	87.74 ±13.19[a]	452.46± 75.13[b]	5.44± 0.60[a]	11.76 ± 0.6[b]
60-69	75.54± 17.40[a]	462.29± 86.78[b]	4.26 ±0.80[a]	13.10±0.76[b]
70-79	46.33±21.63[a]	494.15 ±73.34[b]	3.87±0.79[a]	11.23±0.47[b]

Values are expressed as Mean ± SEM. Means of the same row in the same column with different superscript letters differ significantly (P<0.05).

Table 5: Urea and Creatinine levels according to the degree of parasitaemia in the diabetes patients with malaria infection

Degree of parasitaemia	Creatinine (μmol/l) of test subjects	Urea (mmol/l) of test subjects
+	241.36 ± 18.15[a]	12.64 ± 0.69[a]
++	419.58 ± 45.20[b]	11.92 ± 0.45[a]
+++	687.17 ± 59.68[c]	11.78 ± 0.56[a]

Values are expressed as Mean ± SEM. Means of the same column with different superscript letters differ significantly (P<0.05).

Table 6: Urea and Creatinine levels according to the Fasting Blood Sugar (FBS) of the diabetes patients with malaria infection

FBS (mg/dl)	Creatinine (μmol/l) of test subjects	Urea (mmol/l) of test subjects
100-199	463.27 ± 62 .58[a]	11.28 ± 0.41[a]
200-299	512 .90 ± 76.30[b]	12.37 ± 0.57[b]
300 and above	496.57 ± 76.19[c]	12.94 ± 0.69[c]

Values are expressed as Mean ± SEM. Means of the same column with different superscript letters differ significantly (P<0.05).

The results of this study show that the urea and creatinine levels of the diabetes patients with malaria infection (482.06±40.54μmol/L; 12.03±0.32mmol/L, respectively) is significantly increased (P<0.05) when compared with their controls (84.23±7.95 μmol/L; 4.60±0.34 mmol/L, respectively), irrespective of the sex, age, degree of

parasitaemia and fasting blood sugar of the patients (Tables 1,2,4,5 and 6). The increase is however significantly higher ($P<0.05$) in the males (497.08±49.99 µmol/l) when compared to their female counterparts (461.73±68.79 µmol/l) (Tables 2 and 3). The urea:creatinine ratio (in mmol/l : µmol/l) of the test subjects (0.02±0.01 : 1) was significantly less than ($P<0.05$) that of the control subjects (0.05±0.04 : 1) (Table 1). The elevated creatinine is highest in the 40-49 age group, while urea is highest in the 60-69 age group (Table 4). The level of creatinine significantly increases ($P<0.05$) while that of urea slightly decreases but not significantly ($P>0.05$) as the degree of parasitaemia increases (Table 5). The levels of creatinine and urea however relatively increase with increase in the severity of the uncontrolled diabetes as they increase significantly ($P<0.05$) with increase in the fasting blood sugar (FBS) (Table 6).

This study revealed an elevation in serum urea and creatinine in the test subjects and from the findings of Wilairatana *et al*. (1999), Emian-Ong (2002), and Ekeanyanwu and Ogu (2010), an increase in serum urea concentration with concomitant increase in serum creatinine concentration in diseased conditions like in malaria and diabetes, suggests that the normal functioning of the kidneys has been compromised. They opined further that though blood urea increases more rapidly than serum creatinine concentration at the onset of kidney failure, blood urea levels however do not reflect the performance of the kidneys as compared to serum creatinine. This is because urea produced is also altered by dehydration, food intake and tissue catabolism while creatinine is synthesized in the body at a fairly constant rate from creatine, which is produced during muscle contraction from creatine phosphate. This fact had been earlier buttressed by Morgan, *et. al.* (1977) who opined that elevated urea alone should not be used to judge renal failure as the plasma creatinine concentration was a more useful discriminant between pre-renal ureamia and intrinsic renal failure than was the plasma urea concentration or the urea:creatinine ratio, especially as plasma creatinine concentration of >250 µmol/l indicated intrinsic renal failure with a 90% probability.

In the light of the findings of the studies mentioned above and the fact that the results of this study not only reveal a concomitant rise in both urea and creatinine concentrations but also reveals a creatinine concentration of well over 250 µmol/l, it therefore is likely that a form of renal impairment/dysfunction might be the cause of the results of this study. This insinuation is further strengthened by the fact that the urea:creatinine ratio of the test subjects (0.02±0.01 : 1) apart from being significantly ($P<0.05$) less than that of the control subjects (0.05±0.04 : 1) also fell within the range in keeping with patients with intrinsic renal failure (<0.04 : 1) as opposed to those with post-renal/normal (0.04 - 0.1 : 1) and pre-renal (>0.1 : 1) renal failure when the urea:creatinine ratio is measured in mmol/l : µmol/l (Morgan, *et. al*., 1977; Urashima, *et.al*., 1992 and Feinfeld, *et. al*., 2002). The higher increase in serum creatinine levels in the males compared to the females can be attributed to the higher muscle mass in the former (Harita *et al*., 2008; Wagle, 2010). The slight decrease in urea level from mild to moderate parasitaemia may be as a result of the moderate malaria parasitaemic (++) patients being more hydrated than the mild malaria parasitaemic (+) patients at the time of the study.

Considering the fact that the subjects of this study did not have any other ailments apart from diabetes and malaria, the higher values of serum creatinine and urea observed from this study may therefore be attributed to impairment in renal function associated with *P. falciparum* infection and uncontrolled diabetes mellitus; as renal impairment is characterized by multiple derangements in blood constituents, resulting from the inability of the kidney to properly clear waste products such as creatinine and urea from the blood (Cavanaugh, 2007). Serum creatinine and urea concentration change inversely with changes in glomerular filtration and are therefore useful in gauging the degree of renal dysfunction. Changes in serum creatinine concentration more reliably reflect changes in glomerular filtration rate (GFR) than do serum urea but when assayed together they both give a relatively good idea about the level of renal dysfunction (Emian- Ong, 2002).

The findings of this study corroborate the findings of Onyeneke, *et al*., (2003), Ogbadoyi and Gabi (2007), Idonije, *et al*., (2011a) and Wagle (2010). Onyeneke, *et al.*, (2003) revealed that serum creatinine levels were found to increase significantly in moderate and severe parasitaemia when compared to the control, with this increase being more in the males when compared to the females. Ogbadoyi and Gabi (2007) and Idonije, *et al*., (2011a) while carrying out studies on malaria patients in middle-belt Nigeria and southern Nigeria respectively, observed a significant ($P<0.05$) elevation in blood urea and creatinine in these patients with the values obtained for females being lower than that of their male counterparts. Idonije, *et al.*, (2011a) also found an excellent positive correlation between hyperparasitaemia and increase in levels of plasma creatinine. The research by Wagle (2010) showed a progressive decrease in renal function in male and female diabetic patients as from the age of 40 years and beyond as evidenced by increased serum creatinine levels with the male diabetic patients having a slightly higher serum creatinine level than the females.

These findings of elevated urea and creatinine in this study also aligns with the result of Ekeanyanwu and Akpoilih (2010) in relation to renal dysfunction from *Plasmodium* parasitaemia and that of Aldler, *et al.*, (2003),

Tanigushi *et al*., (2003), Puepet *et al*., (2003), Judykay, *et al*., (2007), Harita *et al*., (2008) and Idonije, *et al*., (2011b), in relation to diabetic nephropathy. The findings of Ekeanyanwu and Akpoilih (2010) revealed that malaria patients in eastern Nigeria had an elevated level of serum urea and creatinine when compared with their respective control values with the urea level not really increasing with increasing parasitaemia.

Aldler *et al*. (2003) found a raised plasma creatinine and urea levels in diabetic patients which they ascribed to renal problems. Tanigushi *et al*., 2003 and Puepet *et al*., 2003 ascribed to diabetic nephropathy the derangement of urea and creatinine and a number of other serum markers which they said contributed to significant morbidity and mortality in such patients. Judykay (2007) in his submission suggested that high creatinine levels observed in diabetic patients may be due to impaired function of the nephrons and that high urea levels in diabetes mellitus patients could be attributed to a fall in the filtering capacity of the kidney thus leading to accumulation of such waste products within the system (Idonije, *et al*., 2011b). Harita *et al*. (2008), where of the opinion that the lower serum creatinine levels in diabetic females was a reflection of their lower volume of skeletal muscle. Results obtained by Idonije, *et al*., (2011b) showed that in addition to elevated blood sugar level in type 2 diabetes mellitus, plasma creatinine and urea concentration are also significantly increased in male and female diabetics compared with their levels in apparently healthy non-diabetic male and female controls with the male diabetic patients having a significantly higher serum creatinine level than their female counterparts. They ascribed these findings to intrinsic renal damage solely from the diabetic ailment.

Diabetes mellitus increases susceptibility to common infections, malaria being one of them (Muller *et al*., 2005). As was indicated in the studies mentioned above, diabetes mellitus and malaria infection can independently lead to renal impairment and so when they coexist in a patient, they can therefore both contribute to renal dysfunction in a synergistic way. The results of this study indicate that there is significant renal dysfunction in patients in southern Nigeria with co-morbidity of diabetes mellitus and malaria.

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