Asian Journal of Medical Sciences 2(5): 214-217, 2010
ISSN: 2040-8773

Submitted Date: July 14, 2010 Accepted Date: August 09, 2010 Published Date: November 10, 2010

Type-2 Diabetes Mellitus and Malaria Parasitaemia: Effect on Liver Function Tests

[1]E.J. Ikekpeazu, [2]E.E. Neboh, [3]I.C. Maduka, [4]I.J. Nwagbara and [5]M.W. Nwobodo
[1]Department of Medical Biochemistry, College of Medicine, University of Nigeria Enugu Campus (UNEC), Enugu State, Nigeria
[2]Department of Chemical Pathology, College of Medicine,
Enugu State University of Science and Technology (ESUT), Enugu State, Nigeria
[3]Department of Chemical Pathology, University of Nigeria Teaching Hospital (UNTH), Enugu, Nigeria
[4]Department of Chemical Pathology, School of Medical Laboratory Sciences,
UNTH, Enugu, Nigeria
[5]Department of Internal Medicine, Enugu State University Teaching Hospital, (ESUTH)
Park Lane, G.R.A, Enugu, Nigeria

Abstract: Diabetes Mellitus and Malaria have continually been shown to be the major killers common in major developing countries in spite of phenomenal progress in medical sciences. The present study is therefore aimed at assessment of liver function in diabetic patients with malaria infection, hence finding the possible contribution of malaria infection of some organs to the pathophysiology of diabetes mellitus. The liver function profile were estimated in a total of 140 subjects, of which 50 were diabetic patients with malaria parasitaemia, 50 diabetics without malaria infection drawn from patients attending the diabetic clinic at the University of Nigeria Teaching Hospital, UNTH, Ituku-Ozalla, Enugu. Fifty (50) non-diabetics without malaria and 50 non-diabetics with malaria were used as controls. All the subjects were aged between 40-70 years old and were involved in the study. The diagnosis for malaria and diabetes mellitus were made according to WHO guidelines. There was no significant difference in the mean blood glucose in the diabetics with and those without malaria. The diabetics with malaria and those without malaria showed normal liver function although alkaline phosphatase and bilirubin were slightly raised but non-significant. Both diabetics and non-diabetics with malaria showed slight elevation in alkaline phosphatase, suggesting that diabetics with severe malaria parasitaemia may be at higher risk of liver dysfunction.

Key words: Assessment, diabetic patients, enzymes, liver dysfunction, parasitaemia

INTRODUCTION

Diabetes mellitus, one of the common non-communicable diseases is caused by absolute or relative insulin deficiency (Crook, 2006). Diabetes is a syndrome characterized by disordered metabolism and abnormally high blood sugar (hyperglycemia) resulting from insufficient levels of the hormone insulin with or without additional resistance of insulin's effect in many body cells (Tierny *et al*., 2002). 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). 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, 2001). There is also evidence that diabetes mellitus is going to be epidemic in many developing and industrialized (Manoj, 2001).

Malaria is the most commonest vector borne parasitic disease of the globe (WHO, 1997) and a major public health problem in more than 100 countries around the globe with more than 2.5 billion people at risk causing 1 to 3 million deaths annually (Park, 1997). Malaria can be transmitted by three known ways; vector transmission (Anderson *et al*., 1981), blood transfusion (Strickland, 1991) and congenital transmission (Ezechukwu *et al*., 2004). Malaria parasite interferes with 3 organs in the body, namely, brain, kidney and liver (Edington, 1967) and the invasion of malaria parasite can cause organ congestion, sinusoidal blockage and cellular inflammation (Jarike *et al*., 2002), leading to the leakage of the parenchymal and membranous enzymes into the

Corresponding Author: Ebele J. Ikekpeazu, Department of Medical Biochemistry, University of Nigeria, Enugu Campus, (UNEC), Enugu State, Nigeria

Table 1: The mean (±SD) of the assayed biochemical parameters in diabetics and non-diabetics with malaria infection

Parameters	Diabetics with malaria	Non-diabetics with malaria	p-value
Parasite density(Count/mL)	103.9±41.2	164.4±98.0	p<0.05
Blood glucose(mmol/L)	8.94±2.27	4.93±0.35	p<0.05
Total bilirubin(mmol/L)	11.1±3.8	9.55±4.7	p>0.05
Conjugated bilirubin (mmol/L)	5.33±2.1	4.74±2.6	p>0.05
Alanine transaminase (iu/L)	8.0±4.4	8.4±2.5	p>0.05
Aspartate transaminase (iu/L)	10.8±3.5	9.8±3.4	p>0.05
Alkaline phosphatase (iu/L)	59.3±28.8	59.1±19.5	p>0.05

circulation (Burtis *et al.*, 2001). Rapid and unprecedented urbanization, in addition to often declining economies might have profound implication for the epidemiology and control of malaria, as the relative disease burden increase among urban dwellers (WHO, 1997). In tropical Africa, malaria poses a dangerous health threat for centuries more than any other disease, with 80% of malaria cases and mortality occurring in Africa, affecting both young and old (Afolabi, 2001).

In spite of phenomenal progress in medical sciences, malaria and diabetes mellitus continue to be the major killers (Park, 1997). As both diseases are common in developing countries, such as Nigeria, there is need to evaluate the biochemical profile of type-2 diabetic patients with malaria parasitemia to know the possible contribution of malaria infection of the hepatic organ to the pathophysiology of diabetes mellitus. This will help in better management of diabetic patients in the tropics, since it is not unlikely to come across diabetic patients with malaria.

MATERIALS AND METHOD

Subjects: A total of Fifty (50) type -2 diabetic patients with malaria parasitaemia and 50 without malaria parasitaemia and attending the diabetic clinic of the University of Nigeria Teaching Hospital UNTH Ituku Ozalla, Enugu State, Nigeria were enrolled as the test subjects. The test subjects were randomly selected from those attending the diabetic clinic of the hospital between June and August of 2009. The control subjects were made up of twenty (20) non-diabetic subjects with malaria parasitaemia and 20 without malaria parasitaemia. Only type-2 diabetic patients with and without evidence of malaria parasite infection and without any previous record of renal failure, hepatic involvement and infections like pneumonia and urinary tract infection were enlisted for the study.

The study was conducted at the University of Nigeria Teaching Hospital, UNTH, Ituku-Ozalla, between the months of June and August 2009 and approval was given by the ethics committee of the institution while informed consent was given by all the subjects before the commencement of the study.

Sample collection and preparation: Fasting venous blood was collected aseptically from the subjects using 5 mls disposable syringes. The blood samples were collected with minimum stasis from the ante-cubital fossa while the subjects were in sitting position and 4 mls were transferred into plain bottles for the biochemical assays whereas the remaining 1ml was transferred into EDTA bottles for malaria parasite tests.

The blood samples in the plain bottles were allowed to clot and retract after which they were centrifuged at 3000 rpm for 10mins and the serum transferred into sterilized plain bottles for the biochemical analysis.

Analytical methods:

Malaria parasite density test: The parasite density was calculated from Giemsa stained peripheral blood smear. The films were examined microscopically using×100 objective under oil immersion (Cheesbrough, 1998).

Biochemical analysis: The blood glucose was estimated using glucose oxidase method of Trinder (1969) where as alkaline phosphatase was assayed using Kind and King method (1954). The estimation of transaminases (ALT and AST) was done by Reitman and Frankel (1957) and the serum total and direct bilirubin was estimated using the method of Powell (1994) and Burtis *et al.* (2001). The optical densities were measured using spectronic-20 spectrophotometer.

Statistical analysis: The data obtained was analyzed using students t-test and the results were given as mean ± standard deviation (±SD).

RESULTS

Table 1 shows the malaria parasite density, blood glucose and liver function profile in diabetics with malaria and non-diabetics with malaria. The results show that the mean parasitic count of the diabetics with malaria (103.9 count/mL) was significantly lower than non-diabetics (164.4 count/mL). There was also statistically significant difference ($p<0.05$) in the mean blood glucose of the diabetics and non-diabetics with malaria infection.

When the diabetics and non-diabetics without malaria were compared, there was significant difference in the mean blood glucose whereas the liver function profile were non significant Also the liver function profile of the diabetics with malaria was within the normal range compared to those without malaria with the exception of

Table 2: The mean (±SD) of the assayed biochemical parameters in diabetics and non-diabetics without malaria infection

Parameters	Diabetics without malaria	Non-diabetics without malaria	p-value
Blood glucose(mmol/L)	8.81±3.4	4.8±0.42	p<0.05
Total bilirubin(mmol/L)	9.99±2.1	8.97±2.5	p>0.05
Conjugated bilirubin (mmol/L)	4.78±2.2	4.21±2.0	p>0.05
Alanine transaminase (iu/L)	7.4±3.5	7.95±3.4	p>0.05
Aspartate transaminase (iu/L)	10.3±4.3	10.6±5.4	p>0.05
Alkaline phosphatase (iu/L)	55.7±22.8	55.5±18.5	p>0.05

Table 3: The mean (±SD) of the assayed biochemical parameters in diabetics with and without malaria infection

Parameters	Diabetics with malaria	Diabetics without malaria	p-value
Blood glucose(mmol/L)	8.94±2.27	8.81±3.4	p>0.05
Total bilirubin(mmol/L)	11.1±3.8	9.99±2.1	p>0.05
Conjugated bilirubin (mmol/L)	5.33±2.1	4.78±2.2	p>0.05
Alanine transaminase (iu/L)	8.0±4.4	7.4±3.5	p>0.05
Aspartate transaminase (iu/L)	10.8±3.5	10.3±4.3	p>0.05
Alkaline phosphatase (iu/L)	59.3±28.8	55.7±22.8	p>0.05

serum bilirubin and alkaline phosphatase activity, which showed slight elevation though not statistically significant (Table 2). However, a comparison of diabetics with and without malaria showed no significant difference in all the parameters studied, (Table 3).

DISCUSSION

As seen in clinical practice, diabetes mellitus and malaria parasite infection are common in developing countries such as Nigeria. The study was carried out to assess the liver function profile of diabetic patients with and without malaria parasitaemia.

The mean parasitic count in diabetics with malaria was significantly lower ($p<0.05$) compared to that of non-diabetics with malaria. The low parasitic count in the diabetics was in agreement with the study of Manoj (2001) and could also be linked to the slow parasitic multiplication observed in experimental malaria mice.

However there was no significant difference ($p>0.05$) in the mean blood glucose of the two groups of diabetics (with and without malaria).

The liver enzymes of the test subjects when compared with the control were within normal range except for the alkaline phosphatase, which showed slight elevation though not significant. The non-significant increase in enzymes can be due to improved glycemic control and improvement in insulin resistance which can reduce mild chronic elevation of transaminases found in type -2 diabetics (Lebovitz *et al*., 2002), as the test subjects were on drugs. Increased but non-significant alkaline phosphatase was also previously observed in experimental mice by Adekunle *et al*. (2007), who suggested the elevation to indicate the hepatic stage of the parasite's life cycle in its host, usually accompanied by significant perturbation of the hepatocyte membrane, leading to leakage of the enzyme out of the liver cells.

There was also slight but non-significant increase in bilirubin level of diabetics with malaria when compared with other group of subjects. This was in contrast with the finding of Manoj (2001), who reported significant increase in the bilirubin level of diabetics with severe falciparum malaria. The result from this study might be as a result the reduced severity of malaria parasitaemia in the test subjects, as shown in the malaria parasite density, in comparison with the study of Manoj (2001).

CONCLUSION

Studies have suggested that improved glycemic control and improvement in insulin resistance can reduce mild elevation of transaminases in diabetic patients. Severe malaria pasitaemia, which is a common occurrence in the developing countries, especially the tropics will most likely be an extra burden to the hepatic organ of a diabetic patient. The raised bilirubin and alkaline phosphatase may suggest that diabetic patients with severe malaria parasitaemia may be at higher risk of liver dysfunction.

REFERENCES

Adekunle, A.S., O.C. Adekunle and B.E. Egbewale, 2007. Serum status of selected biochemical parameters in malaria. Animal Model: Institute of Medical Research and Training, 18(2): 1-9.

Afolabi, B.M., 2001. Malaria: The global scourge. Nig. Clin., 5: 9-12.

Anderson, G.C., C. Morton and I. Green, 1981. Parasites and Human Diseases. In: Community Health. 3rd Edn., Churchill Livingstone, USA, pp: 45-68.

Burtis, C., E. Ashwood and B. Border, 2001. Liver Function. In: Tietz, E. (Ed.), Tietz Fundamentals of Clinical Chemistry. 5th Edn., W.B. Saunders Company, pp: 748-770.

Cheesbrough, M., 1998. Laboratory diagnosis of malaria parasite. District Laboratory Practice in Tropical Countries, Cambridge University Press, pp: 246-250.

Crook, M.A., 2006. Carbohydrate Metabolism. In: Clinical Chemistry and Metabolic Medicine. 7th Edn., Edward Arnold Publishers, pp: 174 -197.

Edington, G.M., 1967. Pathology of malaria in West Africa. Brit. Med. J., 1: 715-721.

Ezechukwu, C., E. Ekejindu, E. Ugochukwu and M. Oguatu, 2004. Congenitally acquired malaria in hyperendemic area. A Cohot study: Trop. J. Med. Res., 8(2): 44-48.

Gautier, J.F., E. Sobngwi, F. Mauvais-Jarvis, P. Vexian and J.C. Mbanya, 2001. Diabetes in Africans epidemiology and clinical specificities. Diabetes Metab., 27: 628-632.

Jarike, A.E., E.E. Emuveyon and S.F. Idogun, 2002. Pitfalls in the interpretation of liver parenchymal and membranous enzyme results. In Pre-clinical *P. falciparum* malaria in the Nigerian Environment. Nig. J. Clin. Med., 10(2): 21-27.

Kind, P.R.N. and E.J. King, 1954. Method of estimating alkaline phosphatase. J. Clin. Path., 7: 322- 327.

Knowler, W., E. Barret-Connor, S. Fowler, R. Hamman, J. Lachin, E. Walker and D. Nathan, 2002. Reduction in incidence of type-2 diabetes with lifestyle intervention or metform. N. Engl. J. Med., 346(6): 393-403.

Lebovitz, H., M. Kreider and M. Freed, 2002. Evaluation of liver function tests in type-2 diabetic patients during clinical trials. Evidence that rosiglitazone does not cause hepatic dysfunction. Diab. Care., 25: 815-821.

Manoj, K.M., 2001. Profile of severe falciparum malaria in diabetes. Int. J. Diab. Dev. Countries, 21: 156-161.

Strickland, G.T., 1991. Life Cycle of Malaria Parasite. In: Tropical Medicine. 7th Edn., W.B. Saunders, USA, pp: 586-601.

Park, E.D., 1997. Parks Test Book for Prevention and Social Medicine. 15th Edn., pp: 115-130.

Powell, W.N., 1994. Method of estimating serum bilirubin. Am. J. Clin. Pathol., 28: 30-36.

Reitman, S. and S. Frankel, 1957. Methods in Clinical Chemistry. Am. J. Clin Chem., 28: 56-59.

Tierny, L.M., S.J. Mcphee and M.A. Papadahis, 2002. Current Medical Diagnosis and Treatment. Int. Edn., Mc Graw Hill, pp: 1203-1215.

Trinder, P., 1969. Method of estimating blood glucose. Ann. Clin. Biochem., 6: 20-24.

WHO, 1997. World malaria situation. Wkly Epidermiol. Res., 72: 269-276.