GLOBAL JOURNAL OF MEDICAL SCIENCES VOL. 5, NO. 1, 2006: 1- 5
COPYRIGHT (C) BACHUDO SCIENCE CO. LTD. PRINTED IN NIGERIA. ISSN 1596 - 2911

RELATIONSHIP BETWEEN ACID AND ALKALINE PHOSPHATASE ACTIVITIES IN PARASITIC INFECTIONS

C. E. J. UDIONG, U. S. EBONG, and P. C. INYANG-ETOH

(Received 21 February, 2006; Revision Accepted 5 April, 2006)

ABSTRACT

Serum acid and Alkaline Phosphatase levels of 80 subjects diagnosed of helminthic or protozoal infections were assayed (excluding pregnant women and children) while 64 non-infected age and sex-matched subjects served as control. Infected subjects had a significantly higher serum alkaline phosphatase activity (133.9 ± 46.6 μ/l) than the control subjects (87.9 ± 15.9 μ/l). But there was no significant difference between the total acid phosphatase values (7.45 ± 1.76 μ/l) and prostatic acid phosphatase activity (1.04 ± 0.31 μ/L) of infected subjects and total acid phosphatase values (6.60 ± 1.84 μ/L) and prostatic acid phosphatase values (0.90 ± 0.29 μ/L) of the control subjects. Females were more infected than the males. Infected females also had a significantly higher total acid phosphatase activity than males ($P<0.5$). There was no significant difference between the activities of prostatic acid phosphatase of male infected subjects and those for females subjects ($P > 0.05$). There was no significant difference between the acid and alkaline phosphatase activities of prostatic acid phosphatase of male infected subjects and those for females subjects ($P > 0.05$). There was no significant difference between the acid and alkaline phosphatase activities in helminthic and protozoal infection ($P > 0.05$) and also those infected with *Entamoeba histolytica* and *Plasmodium falciparum* had significantly higher alkaline phosphatase activities ($P < 0.05$) than other parasites. This work has established a high alkaline phosphatase activity among subjects with parasitic infections especially in amoebasis and falciparum malaria.

KEYWORDS: Acid and Alkaline Phosphatases, helminthic and protozoan infections, Relationship.

INTRODUCTION

A high concentration of helminthic parasites leads to anaemia, malnutrition, raised serum alkaline phosphatase and different forms of morbidities (WHO, 1981). These occur mostly in helminthic infections affecting the liver such as *hydatid disease, fascioliasis,* opisthorchiasis and hepatic schistosomiasis (Mansour, 1982, Mackenjee, *et al.*, 1984). Serum alkaline phosphatase is also elevated in hepatobiliary disease (Mccomb, 1979) and bone disease associated with increased osteoblastic activity (Singer and Wallach 1991).

Helminthic infection is characterized by increased eosinophils in peripheral blood, the eosinophilia being the host response to large parasite infections that cannot be phagocytosed. Extracellular killing of such parasites evolves to cope with the situation by increase in eosinophil output a reaction that is mediated by complement C3B. The C3B-parasite receptor triggers the release of lysosomes with concomitant liberation of acid hydrolases which include acid phosphatase (Roitt, 1988). Such events may lead to raised activity of acid phosphatase in the plasma of the parasite host. Alkaline phosphatase released is associated with similar response to complement.

Acid and alkaline phosphatases are enzymes which are found in the liver, bone, intestine, kidney, placenta, prostate and erythrocytes (Obi and Ilori, 2002). Infection of these organs/cells by agents such as virus and parasites is an obvious cause of cell membrane rupture and enzyme release into the plasma (Nakamura *et al.*, 1988).

In tropical Africa where large parasitic infections are rampant, the esonophil defence response may provide additional enzymes to host making interpretation of host acid and alkaline phosphatase results difficult.

In a study on pathogenic free living amoeba, Warhust, (1985) reported a raised serum alkaline phosphatase due to the fact that enzymes leak out from damaged cells. In another study on severe falciparum malaria involving the liver, there was hepatic dysfunction resulting in increased serum bilirubin with increase in serum alkaline phosphate and serum transaminase concentrations (Bryceson, 1983).

This study was designed to assess the influence of parasitic infections on plasma and serum Alkaline and Acid Phosphatase levels in human subjects.

C. E. J. Udiong, Chemical Pathology Department, University of Calabar, Calabar, Nigeria
U. S. Ebong, School of Medical laboratory Science, University of Calabar Teaching Hospital, Calabar, Nigeria
P. C. Inyang-etoh, Department of Medical Microbiology & Parasitology, University of Calabar, Calabar, Nigeria

Subjects and Methods

One hundred and forty-four subjects attending the University of Calabar Teaching Hospital and General hospital, all in Calabar Municipality, Cross River State of Nigeria were examined. Eighty of them were found to be positive for parasitic infection after their stool and blood were examined for stool and malaria parasites respectively. The remaining 64 non-infected subjects served as control.

Assay of acid and alkaline phosphatase was done using the test kit supplied by Randox diagnostic laboratories Ltd (United Kingdom).

Stool samples were examined macroscopically for adult worms, blood stains, mucus and consistency according to WHO, (1991). Microscopic examination of the stool was done according to the method described by Siddiqui, (1981) while the Brine floatation concentration method was done according to the method described by Cheesbrough, (1998)

For malaria parasite detection, thick and thin blood films were made on each blood sample (Laveran, 1980). Both films were placed on a flat surface or bench to air dry. There after the thin films were fixed in absolute methanol for 5 seconds and allowed to dry. Both the thick and thin films were then stained in freshly prepared 2% Giemsa stain for 30 minutes (Payne, 1988). At the end of the staining, the films were removed from the stain, rinsed in buffered water of PH 7.2 and stood vertically to dry. Both the stained thick and thin films were examined microscopically using the X 100 objective lens with oil immersion. The thin blood films were used for speciation of Plasmodium while thick films were used for detection and quantification of malaria parasitaemia. Calculation of malaria parasite density was made by counting the parasite per white blood cells in thick blood films and parasite density determined by multiplying the figure by 8000 which is an average white blood cell count per microlitre (μl) of blood (Shute, 1986).

Data obtained were analysed, using the t-test while the relationship between G-6-PD and malaria density were compared using the Pearson correlation analysis. All analysis were done using the Microsoft Excel analytical tool of XP windows of IBM computer system.

RESULTS

Table 1 shows a comparism of biochemical parameters between infected and control subjects studied. The mean alkaline phosphatase level was significantly higher in helminthic infection than the control subjects. There was no significant difference between the acid phosphatase activity in infected and control subjects.

TABLE 1: COMPARISM OF BIOCHEMICAL PARAMETER STUDIED BETWEEN INFECTED AND CONTROL SUBJECTS

Parameter	Infected subjects (n = 80)	Control subjects (n = 64)	Calculated t-value	p	Inference
Alkaline phosphatase (μ/l)	133.9 ± 46.6	87.9 ± 15.9	5.34	P < 0.05	Significant
Total acid phosphatase (μ/l)	7.45 ± 1.76	6.60 ± 1.84	0.724	P > 0.05	Not significant
Protatic acid phosphatase (μ/l)	1.04 ± 0.31	0.90 ± 0.29	0.093	P > 0.05	Not significant
Non-prostatic acid phosphatase (μ/l)	7.15 ± 1.31	6.11 ± 1.75	1.793	P < 0.05	Significant

Table 2 compares the alkaline and acid phosphatase in male and female infected subjects. The total acid phosphatase activity was significantly higher in infected males than females. But there was no significant difference between the alkaline phosphatase activity of male and female subjects examined. There was also no significant difference between the prostatic acid phosphatase and non-prostatic acid phosphatase activities of male and female subjects examined.

TABLE 2: COMPARISM OF ALKALINE AND ACID PHOSPHATASES IN INFECTED MALES AND FEMALES

Parameter	Infected males	Infected females	Calculated t-value	p	Inference
Alkaline phosphatase (μ/l)	135.3 ± 46.9	133.3 ± 47.3	0.121	P > 0.05	Not significant
Total acid phosphatase (μ/l)	8.31 ± 1.42	7.12 ± 1.78	2.017	P < 0.05	Significant
Prostatic acid phosphatase (μ/l)	1.13 ± 0.22	1.01 ± 0.34	1.106	P > 0.05	Not significant
Non-prostatic acid phosphatase (μ/l)	7.15 ± 1.31	6.11 ± 1.75	1.793	P > 0.05	Not significant

TABLE 3: COMPARISM OF ACID AND ALKALINE PHOSPHATASES ACTIVITIES BETWEEN HELMINTHIC AND PROTOZOAN INFECTIONS

Parameter	Helminthic infection	Protozoal infection	Calculated t-value	p	Inference
Alkaline phosphatase (μ/l)	130.3 ± 46.4	149.9 ± 49.6	1.408	P > 0.05	Not significant
Total acid phosphatase (μ/l)	7.55 ± 1.42	7.40 ± 2.0	0.323	P > 0.05	Not significant
Prostatic acid phosphatase (μ/l)	1.01 ± 0.31	1.0 ± 0.30	0.111	P > 0.05	Not significant
Non-prostatic acid phosphatase (μ/l)	6.46 ± 1.34	6.37 ± 1.94	0.186	P > 0.05	Not significant

TABLE 4: MEAN LEVEL OF ALKALINE PHOSPHATASE ACCORDING TO PARASITE DETECTED

PARASITES	Alkaline Phosphatase level ($\bar{X}$ ± SD for +ve)	Alkaline Phosphatase level ($\bar{X}$ ± SD for non +ve)	Calculated t-value	p	Inference
Entamoeba histolytica	209 ± 4.1	120.7 ± 36.8	5.34	P < 0.05	Significant
Plasmodium falciparum	147 ± 49.5	119.3 ± 40.2	0.724	P < 0.05	Significant
Ascaris lumbricoides	137.1 ± 48.1	132.0 ± 46.5	0.093	P > 0.05	Not significant
Hookworm	125.8 ± 45.5	116.3 ± 34.1	1.793	P > 0.05	Not significant
Trichuris trichiura	157.5 ± 74.2	132.6 ± 45.9	1.011	P > 0.05	Not significant

Comparism of acid and alkaline phosphatase activities between helminthic and protozoan infection is shown on table 3. There was no significant difference between the acid and alkaline phosphatase activities in helminthic and protozoan infections. Table 4 shows the mean level of alkaline phosphatase according to each parasite detected. The mean alkaline phosphatase activities of those infected with *Entamoeba histolytica* and *Plasmodium falciparum* was significantly higher than those of other parasites.

DISCUSSION

This study attempts to assess the levels of acid and alkaline phosphatases in helminthic and protozoa parasite infected patients. There was no significant difference in the activities of acid phosphatase in infected and control subjects. No significant difference was observed between the value for alkaline phosphatase in male and female infection showing that these infections are not gender bias. There was a significant difference between the total acid phosphatase activity in males and in females, the difference being contributed by the prostate gland of male subjects since the gland is absent in females and this can be compared to the result of Obi and Ilori (2002). The presence of parasitic infections does not appear to affect prostatic acid phosphatase activity but causes increase in the activity of alkaline phosphatase and non-prostatic acid phosphatase which is responsible for the raised total acid phosphatase activity. Those infected with E. histolytica had raised alkaline phosphatase levels the result which is similar to the work of Warhurst (1985).

The higher prevalence of *Ascaris lumbricoides* compared to hookworm can be attributed to the usual large number of eggs produced by the parasite as reported by Ukoli, (1990) and their ability to resist desiccation. *Ascaris lumbricoides* eggs are said to be viable for years especially in faecally contaminated environment (Hall et al, 1999).

Female subjects recorded a higher prevalence of infection than the male subjects probably because females engage in more farming activities than the males in this setting especially in areas where human waste serves as manure. Multiple infections with more than one parasite species were recorded by all gender. This is a regular feature among infected population with *Ascaris lumbricoides* and *Plasmodium falciparum* infection occurring concurrently with other parasites infections (Booth and Bundy, 1995; Adeyeba and Essiet, 2001). This study has shown that infected subject have significantly high alkaline phosphataes activities and total (non-prostatic) acid phosphatase activities. It has also established that those infected with *Entamoeba histolytica* and *Plasmodium falciparum* have significantly high alkaline phosphatase activities. It has also confirmed that heavy burden of helminthic and protozoan infections can cause raised alkaline and acid phosphatase activities in the host thus complicating the interpretation of phosphatase results of such subjects.

REFERENCES

Adeyeba, O. A. and Essiet, U., 2001. Prevalence of Helminth and Protozoal infection among religious sect that walk barefooted in Iseyin, Nigeria. *The Nigerian Journal of Parasitology* 22 (1 & 2): 85-94.

Booth, M. and Bundy, D. A. P., 1995. Estimating the number of multiple-species of geohelminthic infections in human communities. *Parasitology* , 3: 645-653.

Bryceson, A., 1983. Malaria and splenomegaly. *Transactions of the Royal society of Tropical Medicine and Hygiene* 77, 6, 879.

Cheesbrough, M., 1998. District Laboratory practice in Tropical Countries (Part 1), University press, Cambridge, United Kingdom, 191-235.

Hall, A., Anwar, K. S, Tomkins, A. Rahman, L., 1999. The distribution of Ascaris lumbricoides in human hosts: a Study of 1765 people in Bangladesh. *Transaction of the Royal Society of Tropical Medicine and Hygiene* 93: 503-510

Lavcran, A., 1980. Note sur un noveau parasite trouve dans le sang du plussiers maladies atteints de fievre palustre. *Bull acad. Natl. Med. Paris.* 9: 12235.

Mackenjee, M. K. R., Coovadia, H. M., and Chutte, C. H. J., 1984. Clinical recognition of mild hepatic Schistosomiasis in an endemic area. *Transaction of the Royal Society of Tropical Medicine and Hygiene* 78: 13-15.

Mansour, M. M., 1982. Serum enzymes tests in hepatospleenic Schistosomiasis. *Transaction of the Royal Society of Tropical Medicine and Hygiene*. Page 76.

McComb, R. B., Bowers, G. N. Jr; Posen, S., 1979. Alkaline Phosphatase. New York, Plenum Press.

Nakamura, I., Nakamura, K., and Stinson, R. A., 1988. Release of alkaline phosphatase from human osteosarcoma cells by phosphatidyl inositol phospholipase C: Effect of tunicamycin. *Arch. Biochem. Biophys* 265: 190-196.

Obi, F. O. and Ilori O. O., 2002. Effects of cadmium exposure on bone and kidney alkaline phosphatase activity abd acid phosphatase activity in testis and prostate gland in the male rat. *Journal of applied science and environmental management*, 6(1): 9 - 13.

Payne, D., 1988. Use and Limitations of Light microscopy for diagnosing malaria at the primary health care level. *Bulletin of World Health Organization.* 66(5) : 621-626.

Plussiers maladies atteints de fievre palustre. *Bull acad. Natl. Med. Paris.* 9:12235

Roitt, I. M., 1988. The basis of immunology –innate immunity in Essential Immunology; 1-14 Oxford, Blackwell Scientific Publications UK.

Shute, G. T., 1986. The microsopic diagnosis of malaria. In: Malaria principle and practice of malariology eds Wernederfer, WHO and McGregor L. Churchill Livingstone Edinburg.pp. 78-814

Idiqui, M. A., 1981. The prevalence of human intestinal parasite in Albaha, Saudi Arabia. *Annals of Tropical Medicine and Parasitology* 75, 565-566.

Singer, F. R and Wallach, S., 1991. Pagets Disease of Bones Clinical Assessmnet, Recent and Future therapy. New -York, Elsevie.

Ukoli, F. M. A., 1990. Introduction to Parasitology in Tropical Africa. Test Flow Ltd, Ibandan, Nigeria 105-106

Warhust, D. C., 1985. Pathogenic free living amoeba. *Parasitology Today* 1, 124-28

World Health Organization, 1981. Intestinal protozoan and helminthic infection. WHO Technical Report series. Genera. 66.

World Health Organization, 1991. Basic laboratory method in Medical Parasitology. WHO Generation 10-24.