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ELEVATED SERUM ACID PHOSPHATASE: PROSPECTIVE MALARIAL MARKER

GULAB KANWAR, MAMTA YADAV, LEEZUM LEPCHA & SURENDER KUMAR

Department of Biochemistry, Government of Medical College, Kota, Rajasthan, India

ABSTRACT

Present study was conducted to investigate serum acid phosphatase (ACP) and hemoglobin (Hb) level in malaria and non-malarial fever patients. Total 90 samples were analysed (39 malarial, 20 non-malarial and 31 healthy controls). Serum ACP level was significantly raised in malarial patients with mean serum ACP level 5.40±0.395 U/dl (p value <0.003). The level of Hb was decreased in all malaria patients which indicates that malarial parasite uses host erythrocytes Hb as major nutrient source. These results suggest that measurement of ACP could be used as a marker of malaria.

KEYWORDS: Acid Phosphatase, Hemoglobin, Malaria

INTRODUCTION

Under the name of acid phosphatase (ec 3.1.3.2, ACP) are included all the phosphatases with optimal activity below the ph of 7.0. ACP is present in lysosomes in all cells and exist extralysosomally in erythrocytes. Greatest concentration of ACP activity occurs in prostate, bone (osteoclast), spleen, platelets and erythrocytes. Lysosomal and prostatic enzymes are inhibited by tartarate ions whereas erythrocytic and bone isoenzymes are not. Tartarate resistant acid phosphatase (TR-ACP) level increases in various diseases like paget's diseas, haemolytic anaemia, hyper-parathyroidism and bone metastasis etc. Which helps in their diagnosis[(1)]. Little is known about level of ACP in infectious diseases like malaria. Malaria is caused by protozoan plasmodium species. Four species of genus plasmodium infect humans i.e plasmodium falciparum, P. vivax, P. malariae and P. ovale. The WHO (World Health Organisation) estimates that in 2010 there were 219 million cases of malaria resulting in 660000 deaths world-wide[(2),(3)]. In india malaria continues to cause a major public health threat. About 88% of malaria cases and 97% of deaths due to malaria reported from highly endemic states including Rajasthan[(4)]. When infected female anopheline mosquitoe bites a person, sporozoites in mosquito's saliva enter the bloodstream and migrate to liver to infect hepatocytes. After dormancy of 8-30 days hepatocytes rupture, yielding thousands of merozoites which enter in blood-stream to infect erythrocytes. In erythrocytes parasite multiplies asexually and periodically ruptures erythrocytes (i.e haemo-lysis) to infect new erythrocytes[(5)]. Considering the high ex-tralysosomal TR-ACP in erythrocytes and hemo-lysis in malaria, the aim of our study was to evaluate the level of ACP in malaria and to check its possible use as a marker enzyme in its detection and prognosis.

Material and method: the study group consisted of 90 subjects of 20-50 years age group including both males and females. Group 1 consisted of 39 malarial fever patients (18 P. falciparum, 18 P.vivax, 3 both P. vavax and P. falciparum). These patients attended the OPD of new hospital and medical college Kota (Rajasthan) from september 2013 to june 2014. These patients presented with symptoms of intermittent fever, chills, rigor, vomitting and headache. They also presented with hepatomegaly and splenomegaly. Group 2 included 20 non-malarial fever patients and group 3

included 31 healthy controls of same age group. These selected subjects had no prostate problems, previous anaemia, or any other kind of bone disorder.

A finger prick sample was taken to prepare thick and thin blood films to assess for presence or absence of malaial parasite. 5 ml of venous blood was collected randomly; of which 2 ml in EDTA for hemoglobin and 3 ml in plain vial was used for ACP estimation. Serum was seperated taking care to avoid haemolysis for ACP ectimation.

ACP estimation was done by kit method using coral clinical systems ACP reagent set [6],[7]. α-napthol released from substrate α-napthyl phosphate by ACP is coupled with fast red tr to form a diazo dye complex, which absorbs light at 405 nanometer proportional to ACP activity in sample. Testing in presence of tartarate is done to find nonpro-static ACP. The Hb estimation of RBC done by cyanmethemoglobin method[7].

Statistical comparison among the groups was carried out by standard error of difference between two means, p-value <0.05 is considered as significant. Correlation between parameters were estimated by coefficient of correlation.

RESULTS

Serum level of ACP and Hb content in malarial patients, non-malarial fever patients and healthy controls is given in table 1. Serum ACP level are highly increased in malarial patients as compared to non-malarial fever patients and controls. Increase in ACP is greatest in P. falciparum and mixed malaria as compared to P.vivax malaria. The hemoglobin content is decreased in malarial patients as compared to controls. There is no significant difference in rise in ACP levels between males and females suffering from malaria. There is negative correlation between ACP and Hb in malarial patients (r= -0.707) which is statistically significant.

DISCUSSIONS

The present study shows that increased ACP activity in malaria patients was statistically significant. The cell membrane of erythrocytes plays central role in the growth and propagation of malarial parasite in the human body [7],[8]. Human erythrocytes are invaded by malarial parasite during erythrocytic scizogony phase the RBCs are attacked by pre erythrocytic cryp-tomerozoites or the later exoerythrocytic micro-meta cryptomerozoites. Each merozoite grows in RBC to produce 6 signet ring stages during which stages hemozoin pigments are seen. After sometime totally exhausted corpuscle bursts and the merozoites, toxic products and enzymes like ACP are released in blood plasma[9]. After invasion of erythrocytes, in order to survive in human erythrocyte, plasmodium parasite brings about considerable metabolic changes in host cell. The host cells become more vulnerable to damage due to toxic metabolites derived from both; host and the parasite[10-12].

A number of reactive oxygen species are generated during this host parasite interaction. Increase in ros and decrease in antioxidants are reported in malaria patients[13-15]. Alteration in major antioxidants and peroxidelysis of erythrocytes may result in release of enzymes like ACP[16]. In our study ACP levels are increased and hemoglobin levels are decreased in malaria patients. This could be due to erythrolysis caused by host-parasite interaction and oxidative stress. Hemoglobin is thought to be broken down to provide amino acids for growth and maturation of parasite[11],[14].

Thus serum ACP levels in malaria patient could serve as a marker for hemolysis indicating the active stage of the disease; which may be used as an additional marker enzyme of malaria.

Finally to conclude, there is a significant increase in serum ACP levels in malaria patients. And significant negative correlation between serum ACP and hemoglobin. There is need for further study to use this enzyme as a diagnostic and prognostic marker in malaria in addition to other routine tests involved.

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APPENDICES

Table 1: Levels of Serum ACP and Hb in Malaria Patients, Non-Malarial Fever Patients and Control Subjects

	P.falciparum (18)	P.vivax (18)	Mixed Malaria (3)	Malaria (Total) (39)	Control (31)	Non-Malarial Fever (20)
Hb	9.17±0.508*	10.0±0.539*	8.67±0.410*	9.51±0.692	11.41±0.831	10.73±0.523
ACP	5.57±0.335*#	5.16±0.320*#	5.80±0.173*#	5.40±0.395	2.72±0.480	3.51±0.263

Statistical comparison among the groups was carried out by Standard error of difference between two means.
*P<0.003 hs (compared with control group).
# P<0.003 hs (compared with non-malarial fever group).