The Utility of Acid Phosphatase as a Marker in Malaria

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Abstract

Invasion of the human erythrocytes by the malarial parasite brings about considerable metabolic changes in the host cell. In this study we compared acid phosphatase (ACP) levels in 45 cases of malaria with 45 cases of non-malarial fever and 45 normal individuals. In our study the serum ACP levels are highly increased in malaria patients when compared to non-malarial fever patients and it was highly significant (p<.001). The serum ACP levels are significantly increased in PF and Mixed groups compared to PV group. The level of Hb was decreased in all malaria patients whoindicate that malarial parasite uses host erythrocytes Hb as major nutrient source. There is negative correlation between ACP and Hb in malaria patients(r= -0.924) which is statistically highly significant. Increase in serum ACP levels in malaria patients may be used as an additional investigation in the diagnosis of malaria.

Keywords: *Acid Phosphatase, Hemoglobin, Malaria*

I. INTRODUCTION

Malaria occurs throughout most of the tropical regions of the world. Even a century after the discovery of malaria transmission through mosquitoes in India by Sir Ronald Ross in 1897, malaria continues to be one of India's leading public health problems. Malaria is caused by infection with protozoan parasites belonging to the genus *Plasmodium* transmitted by female *Anopheles* species mosquitoes. Four species of genus Plasmodium infect humans: Plasmodium falciparum, P. vivax, P. ovale and P. malariae[(1,2)].

The pathogen passes 3 phases during its development-

- Asexual phase forming sporozoites in female Anopheles mosquito
- Multiplication of sporozoites in human liver cell (pre-erythrocytic phase) &
- Erythrocytic phase whereby merozoites originating from liver cells develop in schizonts[(1,2)].

The invasion of human erythrocytes by the malarial parasite is during the phase of erythrocyticschizogony. The alterations in the major antioxidants of the erythrocytes & the peroxide lysis of the erythrocytes may result inrelease of enzymes like ACP. This may be due to hemolysis caused by host-parasite interactions and increased oxidative stress [(1-4)].

Acid phosphatase (ACP) in serum has normal value <5.5 IU/ml but is increased in various conditions like prostate diseases (prostate cancer, BPH), breast cancer, bone diseases (Paget's disease, bone cancer), multiple myeloma &myeloproliferative disorders, Gaucher's disease, liver diseases, chronic renal failure etc.[(5,6,7)]

Erythrocytic acid phosphatase (ACP1:EC3.1.3.2) shows an electrophoretic polymorphism determined by 3 common alleles (Pa, Pb and Pc) at an autosomal locus. ACP activity can be measured by using substrates either thymophthalein phosphate & beta-glycerophosphate(both are specific for prostatic iso-enzyme) or p-nitrophenylphosphate. Addition of tartrate selectively inhibits prostatic isoenzyme. Most of the acid phosphatase activity of normal sera & of patients with metastasizing prostatic carcinoma is inhibited by incubation in the presence of M/200 NaF. Erythrocytic acid phosphatase, however, is not significantly inhibited[(8-13)]. Increase in serum ACP levels in malaria patients could serve as a marker for hemolysis indicating the active stage of the disease, which may be used as additional investigation in the diagnosis of malaria [(14)].

II. MATERIALS AND METHODS

This hospital based comparative analytical study was conducted among 45 patients with fever who were slide positive for malaria and 45 patients with fever who were slide negative for malaria admitted to SMS hospital and 45 healthy attendants of patients during May 2012 to April 2013. Of 45 malarial patients 16 had P. Falciparum malaria, 24 patients had P.vivax malaria and 5 patients had mixed malaria that is having both P. Falciparum and P. vivax malaria.

A finger prick sample was taken to prepare thick and thin blood films to determine the presence or absence of the malarial parasite. Those showing slide positive were considered as malarial fever and those with slide negative for malaria were considered as non-malarial fever group. Both groups were screened for the level of acid phosphatase in their blood sample. 5 ml of venous blood was collected randomly in EDTA bottles from malaria patients, non-malarial fever patients and normal healthy subjects. It was centrifuged for 10 min. The plasma

was collected taking care to avoid hemolysis and was used for the estimation of the ACP level. Estimation of ACP was done by kit method using acid phosphatase reagent set. The α-naphthol released from the substrate α-naphthol phosphate by acid phosphatase is coupled with fast red TR to produce a colored complex which absorbs light at 405 nm. The reaction can be quantified photometrically because the coupling reaction is instantaneous. L-tartrate inhibits prostatic acid phosphatase but does not interfere with the reaction mechanism. The hemoglobin (Hb) content of erythrocytes was determined by the Cyanmethaemoglobin method [13].

III. RESULTS AND DISCUSSIONS

Table 1 : Serum Levels of ACP (in IU/ml) in Malaria Patients, Non-Malarial Fever Patients and Control Subjects

	Control (45)	Non-malarial (45)	PV (24)	PF (16)	Mixed (5)
ACP	2.3 ± 0.43	3.35 ± 0.22	6.0 ± 0.50	6.61 ± 0.22	7.78 ± 0.17

The serum ACP levels are highly increased in malaria patients when compared to non-malarial fever patients and it was highly significant (p<.001). The serum ACP levels are highly increased in PF and Mixed groups compared to PV group and it was highly significant (p<.001). The serum ACP levels are highly increased in malaria patients when compared to the controls and it was also highly significant (p<.001).Mean acid phosphatase value in males and females was 3.97 ± 1.76 and 4.1 ± 1.92 IU/ml respectively it was statistically not significant (p>.05).

Table 2: Serum Levels of Hb(in g/dl) in Malaria Patients, Non-Malarial Fever Patients and Control Subjects

	Control (45)	Non-malarial (45)	PV (24)	PF (16)	Mixed (5)
Hb	12.63 ±0.74	11.27 ±0.40	10.6 ± 0.50	10.18 ± 0.22	10.1 ± 0.15

The Hb content is significantly decreased in malaria patients compared to the non-malarial fever group (P <0.001) and compared to the control subjects (P <0.001).

Table3: Correlation between ACP &Hb

Group		Hb
PV ACP	Correlation Coefficient	-1.000**
	Sig. (2-tailed)	.000
	N	24
PF ACP	Correlation Coefficient	-1.000**
	Sig. (2-tailed)	.000
	N	16
Mixed ACP	Correlation Coefficient	-.975**
	Sig. (2-tailed)	.005
	N	5
Total ACP	Correlation Coefficient	-.924**
	Sig. (2-tailed)	.000
	N	45
Nonmalarial ACP	Correlation Coefficient	-.119
	Sig. (2-tailed)	.436
	N	45
Control ACP	Correlation Coefficient	-.260
	Sig. (2-tailed)	.084
	N	45

**. Correlation is significant at the 0.01 level (2-tailed).

Correlations between the values were estimated by spearmann'sCorrelation co-efficient. There is negative correlation between ACP and Hb in malaria patients(r= -0.924) which is statistically highly significant (P<0.001).

IV. CONCLUSION

In our study there is a significant increase in serum ACP levels in malaria patients. There is need for further study to use this enzyme as a diagnostic marker in malaria in addition to other routine tests involved.

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