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ORIGINAL ARTICLE

USEFULNESS OF ACID PHOSPHATASE LEVEL IN MALARIAL PATIENTS- A CLINICAL STUDY

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ABSTRACT:
Background: Acid phosphatases are a family of enzymes that are widespread in nature and can be found in many animal and plant species. The greatest concentration of acid phosphatase (ACP) activity occurs in liver, spleen, milk, erythrocytes, platelets, bone marrow, and the prostate gland. The present study was conducted to evaluate the levels of acid phosphatase in patients with malaria. **Materials & Methods:** In present study subjects were divided into 3 groups. Group I- included 20 patients suffering from (7 P. falciparum malaria), 7 with P. vivax malaria and 6 with mixed malaria. Group II- included 20 non malarial fever patients. Group III- included 20 healthy subjects. For detection of malarial parasite, a finger prick sample was taken. The hemoglobin (Hb) content of erythrocytes was determined by the Cyanmethaemoglobin method. **Results:** Out of 60 examined subjects, 30 were males and 30 were females. The difference was statistical non significant (P-1). Subjects were divided into 3 groups. Group I included 20 malarial patients, group II (non malarial fever) had 20 patients and group III had normal healthy subjects. The mean age of 7 patients suffering from P. malaria was 30.24 years, P. vivax 34.65 years, mixed malaria 35.11 years. The mean age of patients in group II was 36.27 years and in group III was 32.29 years. The difference was statistical non significant ($p > 0.05$). ACP level in P. falciparum is 7.21, in P.vivax is 6.78, in mixed malarial is 7.82, in group II patients 3.46 and in group III 2.12. The difference was significant ($P < 0.05$). Hemoglobin (Hb) level in patients suffering from P. falciparum was 10.11 gm%, in P. vivax was 10.69 gm%, in mixed malarial was 10.01 gm%, in group II patients was 11.86 gm% and in group III subjects 13.42 gm%. The difference in Hb level was significant ($P < 0.05$). **Conclusion:** There is increase in acid phospahatase level in patient with malaria. This is used as a diagnostic marker. The level of Hb was decreased in all malaria patients which indicates that malarial parasite uses host erythrocytes Hb as major nutrient source.
Key words: Malaria, P. falciparum, P. vivax

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INTRODUCTION

Acid phosphatases are a family of enzymes that are widespread in nature and can be found in many animal and plant species. The greatest concentration of acid phosphatase (ACP) activity occurs in liver, spleen, milk, erythrocytes, platelets, bone marrow, and the prostate gland. The last is the richest source, and it contributes a small proportion of the enzyme present in sera from healthy males. Increasing levels of ACP are consistent with prostatic cancer.[1]

Human acid phosphatase (ACP) has a considerable impact as tools of clinical investigation and intervention. Its levels are increased in various diseases like prostate cancer that has spread to the prostate gland and to the bone, Paget's disease, hemolytic anemia, prostatitis, thrombophlebitis, Gaucher's disease, hyperparathyroidism etc. which helps in their diagnosis.[2]

This enzyme is a phosphatase of low specificity. It hydrolyses phosphoric acid esters. The optimum pH is between 4 and 5.5. This enzyme occurs in the prostate and a variety of tissues like the liver, spleen, erythrocyte etc. isoenzymes of ACP are described.[3]

The optimal pH for the individual ACPs varies depending on the tissues from which they are obtained. The observed pH optimum also varies with the substrate on which the enzyme acts; the more acidic the substrate, the lower the

pH at which maximum activity is obtained. The ACPs are unstable, especially at temperatures above 37°C and at pH levels above 7.0. Some of the enzyme forms in serum are particularly labile and more than 50% of the ACP activity may be lost in 1 hour at room temperature. Acidification of the serum specimen to a pH below 6.5 aids in stabilizing the enzyme.[4] Erythrocytic ACP gene is located on chromosome 2, osteoclast ACP is located on chromosome 19 and prostatic ACP gene is located on chromosome 13. The prostatic isoenzyme is inactivated by tartaric acid and cupric ions inhibit the erythrocytic ACP. The total value of ACP is increased in prostatic carcinoma and bone metastasis.[5]

The level of ACP in infectious diseases like malaria is not well known. Malaria is caused by the protozoan plasmodium species such as Plasmodium falciparum, P. vivax, P. ovale and P. malariae. Of these P. Vivax and P. falciparum account for more than 95% of the cases of malaria. Human infection begins when a female anopheline mosquito inoculates the sporozoites from its salivary glands during a blood meal. Hence the parasite completes its life cycle in two hosts- man and female anapheline mosquito. Malaria is a major cause of mortality in developing countries.[6] The present study was conducted to evaluate the levels of acid phosphatase in patients with malaria.

MATERIALS & METHODS

The present study was conducted in year 2014. It included 60 subjects (males- 30, females- 30). All subjects were informed regarding the study and written consent was taken. Subject information regarding name, age, sex etc. was taken. They were divided into 3 groups.

Group I- included 20 patients suffering from (7 P. falciparum malaria), 7 with P. vivax malaria and 6 with mixed malaria.

Group II- included 20 non malarial fever patients.

Group III- included 20 healthy subjects.

For detection of malarial parasite, a finger prick sample was taken. 5 ml of venous blood was collected randomly in EDTA bottles from malaria 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 Teco Diagnostics kit-Acid phosphatase reagent set. The a-naphthol released from the substrate a-naphthyl 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-tartarate inhibits prostatic acid phosphatase but does not interfere with the reaction mechanism. The hemoglobin (Hb) content of erythrocytes was determined by the Cyanmethaemoglobin method. Results were tabulated and subjected for correct inferences. P value < 0.05 was considered significant.

RESULTS

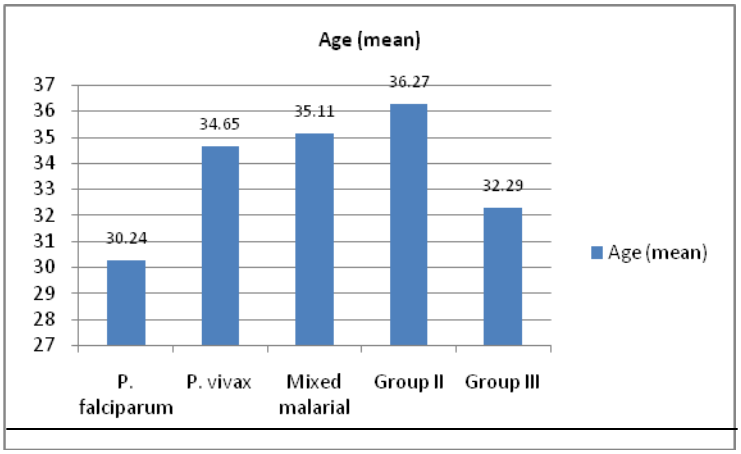
Out of 60 examined subjects, 30 were males and 30 were females. The difference was statistical non significant (P-1) (Table I). Table II shows that subjects were divided into 3 groups. Group I included 20 malarial patients, group II (nonmalarial fever) had 20 patients and group III had normal healthy subjects. Graph I shows that mean age of 7 patients suffering from P. malaria was 30.24 years, P. vivax 34.65 years, mixed malaria 35.11 years. The mean age of patients in group II was 36.27 years and in group III was 32.29 years. The difference was statistical non significant ($p > 0.05$). Graph II shows that ACP level in P. falciparum is 7.21, in P.vivax is 6.78, in mixed malarial is 7.82, in group II patients 3.46 and in group III 2.12. The difference was significant ($P < 0.05$). Graph III shows that hemoglobin (Hb) level in patients suffering from P. falciparum was 10.11 gm%, in P. vivax was 10.69 gm%, in mixed malarial was 10.01 gm%, in group II patients was 11.86 gm% and in group III subjects 13.42 gm%. The difference in Hb level was significant ($P < 0.05$).

Table I Distribution of patients

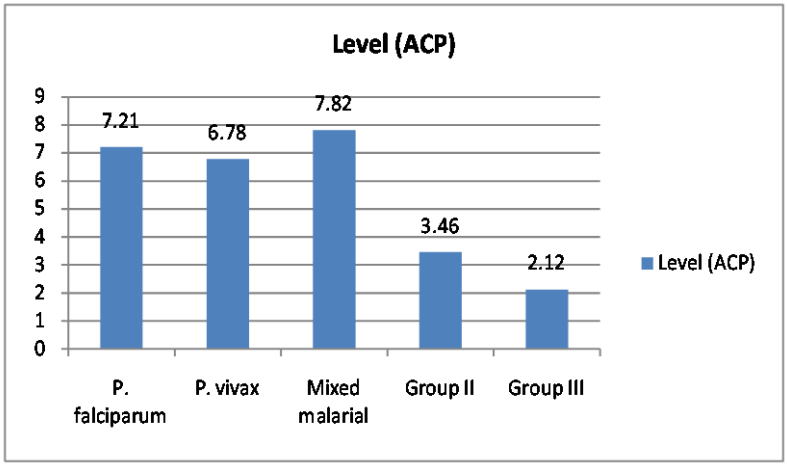
Total examined - 60			
Gender	**Male**	**Female**	**P value**
Number	30	30	1

Group	**Group I**	**Group II**	**Group III**	**P Value**
Number	**20**	**20**	**20**	1

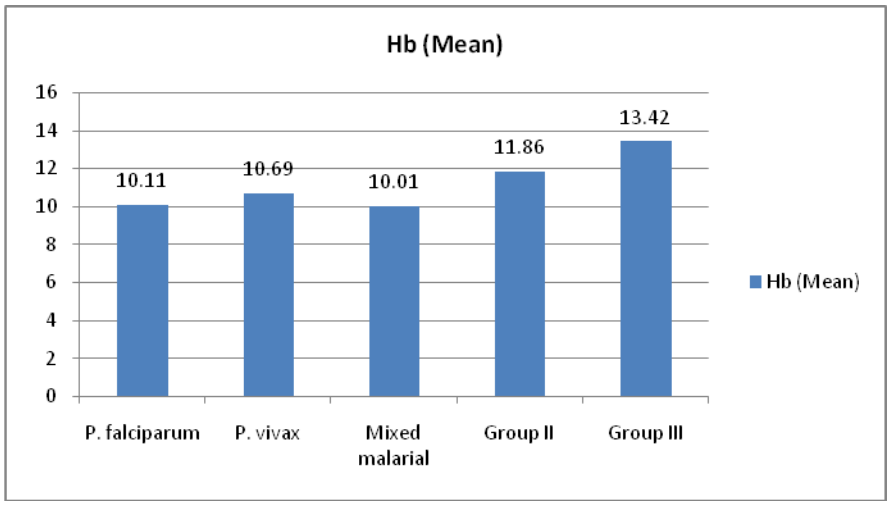
Graph I Age distribution of subjects



Graph II Acid phosphatase (ACP) level



Graph III Hemoglobin level in subjects



DISCUSSION

The WHO (World Health Organisation) estimates that in 2010 there were 219 million cases of malaria resulting in 660000 deaths world-wide. 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. 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 to infect new Erythrocytes. [7] The present study was conducted to evaluate the levels of acid phosphatase in patients with malaria.

In present study, 30 were males and 30 were females. Subjects were divided into 3 groups of 20 subjects each. We found that mean age of 7 patients suffering from P. malaria was 30.24 years, P. vivax 34.65 years, mixed malaria 35.11 years. The mean age of patients in group II was 36.27 years and in group III was 32.29 years. Our results are in accordance to Gupta CM et al.[8]

We evaluated ACP level in all subjects and found that ACP level in P. falciparum is 7.21, in P.vivax is 6.78, in mixed malarial is 7.82, in group II patients 3.46 and in group III 2.12. This is similar to Prassannachandra et al.[9]

We found that hemoglobin (Hb) level in patients suffering from P. falciparum was 10.11 gm%, in P. vivax was 10.69 gm%, in mixed malarial was 10.01 gm%, in group II patients was 11.86 gm% and in group III subjects 13.42 gm%. Our results are in accordance to Goldberg DE et al.[10]

ACP level is abundant in RBCs. The cell membrane plays a central role in the growth and propagation of the malarial parasite in the blood. It also contains parasite specific receptor sites on its surface, on other hand, it allows the

parasite to derive from the host blood plasma the nutrients essential for the intracellular parasite development and growth.[11]

The invasion of the human erythrocytes by the malarial parasite is during the phase of erythrocytic scizogony. The RBC's are attacked by the pre erythrocytic cryptomerozoites or the later exo erythrocytic micro-meta crypto merozoites. Each merozoite buries itself in the RBC's and gradually increase in size to produce at least six signet ring stages. It is during this stage that hemozoin pigments are seen. After sometime the cell membrane of the totally exhausted corpuscle bursts and the merozoites, toxic products and the enzymes like ACP are released into the blood plasma.[12]

CONCLUSION

There is increase in acid phospahatase level in patient with malaria. This is used as a diagnostic marker. The level of Hb was decreased in all malaria patients which indicates that malarial parasite uses host erythrocytes Hb as major nutrient source.

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