Hematological and biochemical evaluation in malaria patients with clinical correlation

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ABSTRACT

Background: Malaria is a major health problem in India. It is one of the biggest burdens in terms of morbidity and mortality. Malaria pathogenesis is based mainly on extensive changes in hematological and biochemical parameters.

Aim: To investigate the effects of severe malaria in infected patients in the Aurangabad on some biochemical and hematological parameters that could provide a credential clues in understanding malaria pathogenesis, diagnosis and management.

Method: A total of 54 (28 males and 26 females) blood samples and 1 healthy man and 1 healthy woman blood sample (as a control) were screened for presence of plasmodium. Level of Hemoglobin in blood and serum level of SGOT, SGPT, ALP, bilirubin, creatinine and urea were estimated.

Result: A significant decrease in the level of Hemoglobin and significant increase in the level of SGOT, SGPT, ALP, bilirubin, creatinine and urea were observed.

Conclusion: This study indicates alternations in these parameters and therefore recommends proper monitoring during treatment in order to reverse them to normal levels.

Keywords: malaria, liver, kidney, hemoglobin

INTRODUCTION

Malaria has emerged as one of the top 10 killer diseases around the globe. It is the major cause of mortality in various tropical and subtropical regions. More than 500 million people reported positive cases of malaria and leading to death in 2 to 3.0 million cases. Four species of malaria parasite cause this disease (p. falciparum, p. vivax. P. malaria, p. ovale) but p. falciparum is the foremost cause of malaria and death. Malaria is not a uniform disease, it encompasses many manifestations and its impact varies on epidemiological setting. It continues to poses a major public health threat in India due to p. falciparum. About 88% of malaria cases and 97% of death due to malaria is reported from North- Eastern states, Chhattisgarh, Jharkhand, Madhya- Pradesh, Orissa, Andhra Pradesh, Maharashtra, Gujarat, Rajasthan, West Bengal and Karnataka.[1,2] Cases of malarial infection associated renal and hepatic impairment have been reported from different parts of malaria endemic countries.[3,4,5] It is more common in adults than children. The severity of malaria infection associated renal impairment in a particular region is largely a function of the disease spread in the region and other an etiological factors prevailing in the area.[6]

Malaria can affect single or multiple organs with different levels of severity which can be determined as neurologic and renal dysfunction, haematologic, cardiovascular, and respiratory dysfunction, as well as hepatic and metabolic dysfunction.[7] This study is an attempt to investigate the effects of severe malaria in infected patients in the Aurangabad region on few biochemical and hematological parameters that could provide a credential clues in understanding malaria pathogenesis, diagnosis and management.

MATERIALS AND METHODS

Collection of Samples: A total of 54 (28 males and 26 females) blood samples were collected for estimation of Hemoglobin from Government Hospital, Aurangabad in the EDTA coated vial and stored at –20^{0}C while the 54 non-haemolysed blood samples of serum for Liver Function and Kidney Function Test were collected in the plain vial. 1 healthy man and 1 healthy woman blood sample (as a control) was also collected.

Staining Method for detection of malaria pathogen: Giemsa stained, peripheral blood smear was used for detection and diagnosis of malaria pathogen.

Estimation of Hemoglobin: Hemoglobin estimation was carried out using Mythic cell counter from the EDTA coated vials.

Biochemical Analysis: Prior to the start of treatment, all serum samples were separated from 2ml of blood and stored at -20^{0}C. SGOT or AST, SGPT or ALT, ALP, Bilirubin, blood urea and creatinine were estimated by using Roche Cobas chemistry analyzer.

Biostatistical Analysis: The data was analyzed using the online statistical mean± standard deviation (SD) tools available on the internet.

RESULTS

The diagnosis of malaria parasite in the blood samples was confirmed by observing the various stages of malaria parasite in the stained blood film under compound microscope. The average hemoglobin level in the male blood samples were found to be 10.91 mg/ml The average serum levels of SGPT and SGOT in plasmodium affected male patients were found to be 38.07 ± 7.44 iu/l and 29.60 ± 10.42 iu/l (Table 1) respectively which were significantly high as compared to the upper limit of the normal range.

Table 1: Effect of malaria infection on hematological and biochemical parameters as with control or normal person

Sr. No.	Parameter and Normal Values	Quality Control		Patients	
		Male	Female	Male Mean ± SD	Female Mean ± SD
1	Hemoglobin	13.4 mg/ml	11.94 mg/ml	10.91± 1.29	10.33 ± 1.085
2	SGPT (ALT)	11.86 iu/l	14.57 iu/l	38.07 ± 7.44	51.05 ± 15.74
3	SGOT (AST)	10.45 iu/l	12.56iu/l	29.60 ± 10.42	25.52 ± 4.62
4	ALP	45.63 iu/l	37.65 iu/l	68.11 ± 13.68	67.20 ± 0.28
5	Bilirubin	0.54 mg/dl	0.47 mg/dl	0.9 ± 0.2	0.78 ± 0.38
6	Creatinine	0.69 mg/dl	0.68 mg/dl	0.95 ± 0.30	1.01 ± 0.22
7.	Urea	14.25 mg/dl	12.47 mg/ld	17.12± 6.91	22.73± 8.94

Out of 28 male patients 5 patients showed significantly higher values of SGPT and SGOT. While in female patients it is significantly high as compared to the upper limit of the normal range. Out of 26 female patients 6 patients showed significantly higher values of SGPT and SGOT.

The average serum level of ALP in plasmodium affected male patients was found to be 68.11 ± 13.68 iu/l (Table 1) which is significantly high. Seven patients' shows significantly higher values of ALP and also in female patients the average serum levels of ALP were found to be 67.20 ± 0.28 iu/l (Table I) which is significantly high and out of 26 female patients 6 patients showed higher values than the normal range.

The average serum levels of Bilirubin in plasmodium affected male patients were found to be 0.9 ± 0.2 mg/dl (Table 1) be significantly high.

Out of 28 male patients the 5 patients shows significantly higher values of bilirubin and also in female patients the average serum levels of bilirubin were found to be 0.78 ± 0.38 mg/dl.

The average serum levels of creatinine and urea in plasmodium affected male patients were found to be 0.95 ± 0.30 mg/dl and 17.12 ± 6.91 mg/dl respectively which is significantly high. In male group 7 patients showed significantly higher values of creatinine whereas in females it was only in 2 patients. In female patients the average serum levels of creatinine and urea found to be 1.01 ± 0.22 mg/dl and 22.73 ± 8.94 mg/dl.

DISCUSSION

Malaria may be associated with life threatening complications such as cerebral malaria, severe anemia, acidosis, respiratory distress and acute renal failure (ARF).[8] Plasmodium falciparum infection is commonly associated with clinically significant renal and renal related disorders which are thought to be mediated by a complex interaction of mechanical, immunologic, cytokine, humoral, acute phase response, nonspecific factors, and hemodynamic factors.[9]

Alternations in hematological and biochemical parameters have been investigated and reported in the malaria infection. It is vital to include hematological and biochemical investigation in the diagnosis of malaria infection so as to detect early complication associated with acute malaria infection. This helps to intensively care for the patients and prevent death that may results from such complications.[10] The result of this study shows that the malaria infection resulted in the alternations of few parameters. Hemoglobin was significantly reduced in malaria which can be due to increase breakdown of red blood cells by the parasites .[11]

As SGOT, SGPT and ALP is synthesized in the liver hence, it is possible that initial inflammation of the liver may increase its production due to infection of plasmodium to the liver.[12] Also, symptoms of these infections associated with vomiting could have caused increased hemo-concentration and lead to initial increase in serum SGOT, SGPT and ALP due to the breakdown of liver cells after the infection.

This study observed a significant increase in the level of bilirubin in the malaria in Aurangabad region. This finding is comparable with another work.[12] Many authors have reported presence of jaundice in cases of malaria, typhoid and some other disease causing infections.[13,14,15]

The concurrent increase in the serum creatinine level is mostly the result of impaired glomerular filtration of urea and creatinine and that is also an indicator for acute malarial severity.[16] Authors have also reported high levels of creatinine in childrens and adults with malaria infection. [8,12,15]

This study reports hyperuricemia in the malaria patients which can be attributed to increased catabolic rate which characterize the disease. This observation is supported by yet another study.[17] An authors have reported that increased Urea: Creatinine ratio in malaria patients also indicate that the causes of Uraemia in these patients are largely prerenal and may be due to reduced renal blood flow to the glomeruli due to malaria associated hypertension and may be responsible for the reduced glomerular filtration rate and hence decreased renal excretion of the analytes.[16]

CONCLUSION

Malaria has a significant impact on Hematological and biochemical profile therefore ita must be considered as a leading differential diagnosis in

acute febrile patients with more abnormalities like splenomegaly, fall in hemoglobin level, and raised bilirubin levels and serum creatinine-urea level.

AUTHOR NOTE

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REFERENCES

1. Sitprija V. Nephropathy in falciparum malaria. *Kidney Int*. 1988Dec;34(6):867-77.
2. Naqvi R, Ahmad E, Akhtar F, Naqvi A, Rizvi A. Outcome in severe acute renal failure associated with malaria. *Nephrol Dial Transplant*. 2003 Sep;18(9):1820-3.
3. Mishra SK, Mohapatra S, Mohant SY. Jaundice in falciparum malaria. *J Indian Academy Clin Med.* 2003;4:12-13.
4. Sharma SK, Sharma BHK, Shakya K, Khanal B, Khaniya S, Shrestha N, et al. *Journal of Nepal Medical Association.* 2004;43:7-9.
5. Ogbadoyi EO, Gabi B. *African Journal of Infectious Diseases.* 2007;1(1):57-64.
6. WHO. Severe falciparum malaria. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 2000;94(1):1-90.
7. Mohapatra MK, Das SP. The malaria severity score: a method for severity assessment and risk prediction of hospital mortality for falciparum malaria in adults. *J Assoc Physicians India*. 2009;57:119-126.
8. Idonije BO, Nwoke EO, Festus O, Oluba OM. Plasma concentrations of Kidney function indicators in Malaria Patients in Ekpoma, South-South Nigeria. *Int J Trop Med*.2011;6(1):4-7.
9. Eiam-Ong S. Malarial nephropathy. *Semin Nephrol*. 2003;23(1):21-33.
10. Mishra SK, Mohanty S.. Problems in the management of severe malaria. *J Trop Med*. 2003;1:1-10.
11. Goselle ON, Onwuliri COE, Onwuliri VA. Malaria infection in HIV-AIDS patients and its correlation with Packed Cell Volume (PCV). *J Vector Borne Dis*. 2009;46:205-211.
12. Adeosun OG, Oduola T, Akanji BO, Sunday AM, Udoh SJ, Bello IS. Biochemical alternation in Nigerian children with acute Falciparum malaria. *Afr J Biotechnology*. 2007;67:881–885.
13. Mazumder RN, Pietroni MA, Mosabbir N, Salam MA. Typhus fever: An overlooked diagnosis. *J Health Popul Nutr*. 2009;276:419-421.
14. Paula EB, Christopher BB. Prevention and management of malaria infection in people living with HIV. *Arch. Int. Med*. 2007;167:1827–1836.
15. Rajagopala S, Ritesh A, Dheeraj G. Severe sepsis due to severe *falciparum* malaria and leptospirosis co-infection treated with activated protein C. *Malar J*. 2007;6:42-42.
16. Etim OE, Ekaidem IS, Akpan EJ, Usoh IF, Akpan HD,. Effect of quinine treatment on some indices of protein metabolism in plasmodium falciparum infected human subjects. *Acta. Pharm. Sci*., 2009;51: 21–26.
17. Ogbadoyi EO, Tsado RD. Renal and hepatic dysfunction in malaria patients in Minna, North Central Nigeria. Online *J Health Allied Sci*. 2009:8.