International Journal of Clinical and Diagnostic Pathology

ISSN (P): 2617-7226
ISSN (E): 2617-7234
www.patholjournal.com
2021; 4(4): 160-162
Received: 23-11-2021
Accepted: 10-12-2021

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A Study of status of liver enzymes in malaria

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DOI: https://doi.org/10.33545/pathol.2021.v4.i4c.437

Abstract
Mildly elevated liver enzymes have been reported in dengue infection. Malaria destroys the liver tissue and the enzymes are elevated in blood that is absorbed partially or fully in the blood stream. The liver enzymes in malaria are elevated in early malaria infection and it's a rule and natural history of the disease. The enzymes can be used as a predictor for assessing the disease severity and higher the levels of liver enzymes poorer is the prognosis of the disease. Most of the studies showed that unlike other viral infections, in dengue the rise of SGOT is usually more than SGPT and is believed to be due to release from the damaged myocytes. In view of this biochemical pattern, it is possible to confuse liver involvement in malaria infection with typical acute viral hepatitis, especially in countries where outbreaks of hepatitis A and E are common. So an attempt has been made to study the hepatic enzyme values and whether it reflects the prognosis is checked in this study.

Keywords: LFT, SGPT, SGOT, Malaria, Infection

Introduction
Surveys of falciparum malaria have reported lower rates of jaundice at presentation in endemic populations (2.6–5.3% of cases) compared with epidemic malaria (11.5–62% of cases) [1-7]. Other *Plasmodium* species have been associated with a relatively lower rate of jaundice compared with falciparum [8, 9]. Mild elevations of transaminases are common, although more significant elevations associated with multiorgan dysfunction have also been reported [10-14]. The syndrome of "malarial hepatopathy" has been recently proposed, being defined as a bilirubin level >2.5 times upper limit of normal (ULN) with associated transaminase elevation >3×ULN (where ALT is considered the more liver-specific enzyme) [1, 2, 15-19]. Although the clinical significance of malarial hepatopathy has not been fully elucidated, it represents an attempt to further describe malaria-associated liver injury and has been associated with more severe disease and other organ dysfunction [1]. The relationship of malarial hepatopathy to antimalarial treatment needs to be evaluated to differentiate it from clinically significant drug-induced liver injury meeting Hay's Law, which has a similar definition [20]. Malarial hepatopathy has been reported in 2.6–45% of all malaria cases and up to 87.5% of cases presenting with clinical jaundice [1, 2, 4, 13, 18, 21, 22]. Kupffer cell hyperplasia, hemozoin loading, and monocyte infiltration are the most frequently reported histological findings in malaria-associated liver injury; all seem to resolve after antimalarial treatment [2, 4, 12, 23-27]. So an attempt has been made to study the hepatic enzyme values and whether it reflects the prognosis is checked in this study.

Aims and Objectives: Hepatic Enzyme as a prognostic evaluator in malaria.

Materials and methods
Methodology: The present study was conducted in the Department of Medicine, in Srinivas Institute of Medical Sciences, Mangalore.
45 patients were chosen for the study.
The study was done in 45 patients who were admitted with Dengue Positive.

Inclusion Criteria
1. Cases confirmed peripheral smear.
2. Cases with elevated liver enzymes.

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1. Cases confirmed peripheral smear
2. Cases with elevated liver enzymes.

Exclusion Criteria

1. Alcoholics.
2. Patients on Hepatotoxic drugs.

All the statistical analysis is done using the ANNOVA.

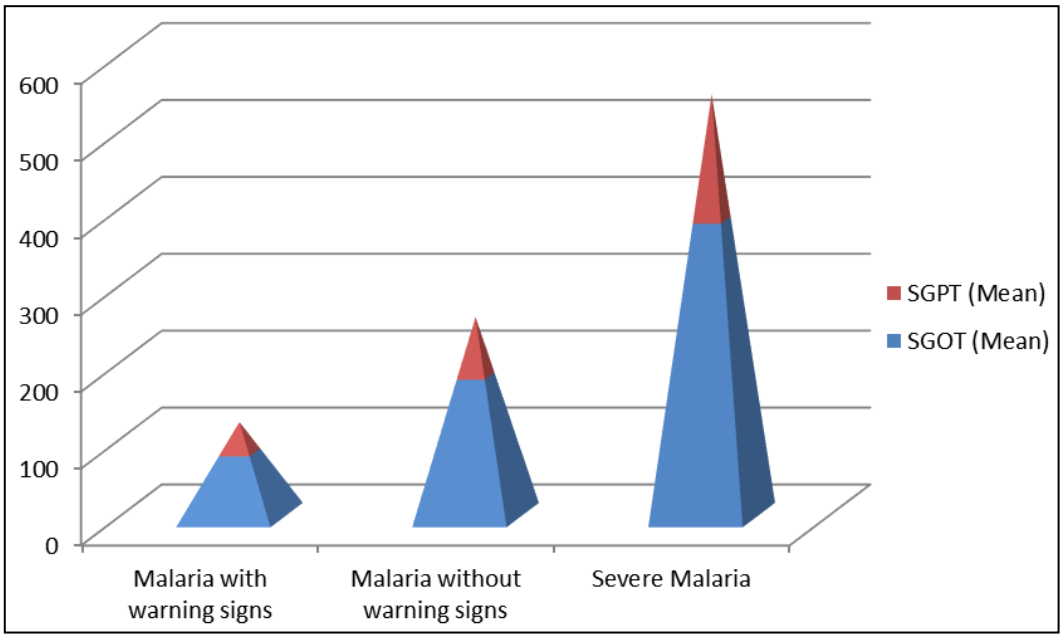
Results

Table 1: Age

Total	Mean Age	SD
45	41.39 years	± 5.39 years

Table 2: Sex Distribution

Total	Male	Female
45	31	14



Graph 1: Enzyme

Table 5: Significance of rise in enzymes

Value of SGOT	X-Value	Significance
375.29	9.38	0.0018

Discussion

Peaks in transaminase levels after treatment have also been recently reported; these could represent a distinct type of malaria-associated liver injury [28-31]. In a small retrospective comparative European study, asymptomatic transaminase elevation was observed in returned travellers hospitalized for malaria after treatment with the Artemisinin-based combination therapy artemether–lumefantrine. The authors postulated that the rapid parasiticidal effect of the antimalarial treatment may promote accelerated hepatic heme loading and oxidative stress [28]. Liver injury through this proposed mechanism is not exactly an adverse drug reaction but rather may be influenced by factors, such as parasite burden, rate of clearance, and host heme metabolism. In prospective trials using the induced blood stage malaria (IBSM) model in healthy malaria-naive participants, asymptomatic moderate (2.5–5.0×ULN) to more rarely severe (>5.1×ULN) elevations of transaminase enzymes have been reported in a subset of participants. These isolated transaminase elevations are associated with an ALT/AST ratio >1 and a normal bilirubin level, peaking 4–12 days after treatment, and have been reported following multiple antimalarial compounds in both falciparum and vivax malaria models [29, 30]. Similar elevations have been observed after sporozoite malaria challenge [31, 32]. Delayed transaminase elevation may be attributed to the antimalarial compound, the use of acetaminophen for symptom relief [33] the malaria parasite, or host factors associated with parasite clearance. Although the relative contribution of each of these factors is uncertain, it is important to understand their relative contribution, given the implications that unexplained transaminase elevation may have on antimalarial drug development.

Conclusion

Both SGOT and SGPT are significantly elevated in severe dengue cases than SGPT. But the increase of SGOT is significantly higher.

References

1. Jain A, Kaushik R, Kaushik RM. Malarial hepatopathy: clinical profile and association with other malarial complications. *Acta Trop*. 2016;159: 95–105.
2. Anand AC, Ramji C, Narula AS, Singh W. Malarial hepatitis: a heterogeneous syndrome? *Natl Med J India*. 1992;5:59–62.
3. Mehta SR, Naidu G, Chandar V, Singh IP, Johri S, Ahuja RC. Falciparum malaria—present day problems. An experience with 425 cases. *J Assoc Physicians India*. 1989;37:264–267.
4. Murthy GL, Sahay RK, Sreenivas DV, Sundaram C, Shantaram V. Hepatitis in falciparum malaria. *Trop Gastroenterol*. 1998;19:152–154.
5. Mazumder R, Mishra RK, Mazumder H, Mukherjee P. Jaundice in falciparum malaria—some prospective observations. *J Indian Med Assoc*. 2002;100:312–314.
6. Diriba Taye, Monenus Etefa. Review on improving nutritive value of forage by applying exogenous enzymes. Int J Vet Sci Anim Husbandry 2020;5(6):72-

79.
7. Ramachandran S, Perera MV. Jaundice and hepatomegaly in primary malaria. *J Trop Med Hyg*. 1976;79:207–210.
8. Hazra BR, Chowdhury RS, Saha SK, Ghosh MB, Mazumder AK. Changing scenario of malaria: a study at Calcutta. *Indian J Malariol*. 1998;35:111–116.
9. Tangpukdee N, Thanachartwet V, Krudsood S, Luplertlop N, Pornpininworakij K, Chalermrut K *et al*. Minor liver profile dysfunctions in *Plasmodium vivax*, *P. malariae* and *P. ovale* patients and normalization after treatment. *Korean J Parasitol*. 2006;44:295–302.
10. Mishra SK, Mohanty S, Das BS, Patnaik JK, Satpathy SK, Mohanty D *et al.* Hepatic changes in *P. falciparum* malaria. *Indian J Malariol*. 1992;29:167–171.
11. Gupta UC, Kataria ML. *Plasmodium falciparum* hepatitis during malaria epidemic. *J Assoc Physicians India*. 1993;41:292.
12. Kochar DK, Agarwal P, Kochar SK, Jain R, Rawat N, Pokharna RK *et al.* Hepatocyte dysfunction and hepatic encephalopathy in *Plasmodium falciparum* malaria. *QJM*. 2003;96:505–512.
13. Abro AH, Ustadi AM, Abro HA, Abdou AS, Younis NJ, Akaila SI. Jaundice with hepatic dysfunction in *P. falciparum* malaria. *J Coll Physicians Surg Pak*. 2009;19:363–366.
14. Patwari A, Aneja S, Berry A, Ghosh S. Hepatic dysfunction in childhood malaria. *Arch Dis Child*. 1979;54:139–141.
15. Anand AC, Puri P. Jaundice in malaria. *J Gastroenterol Hepatol*. 2005;20:1322–1332.
16. Kochar DK, Kaswan K, Kochar SK, Sirohi P, Pal M, Kochar A *et al.* A comparative study of regression of jaundice in patients of malaria and acute viral hepatitis. *J Vector Borne Dis.* 2006;43:123–129.
17. Fazil A, Vernekar PV, Geriani D, Pant S, Senthilkumaran S, Anwar N *et al.* Clinical profile and complication of malaria hepatopathy. *J Infect Public Health*. 2013;6:383–388.
18. Saya RP, Debabrata G, Saya GK. Malarial hepatopathy and its outcome in India. *N Am J Med Sci*. 2012;4:449.
19. Bhalla A, Suri V, Singh V. Malarial hepatopathy. *J Postgrad Med*. 2006;52:315–320.
20. Temple R. Hay's law: predicting serious hepatotoxicity. *Pharmacoepidemiol Drug Saf*. 2006;15:241–243.
21. Bag S, Samal GC, Deep N, Patra UC, Nayak M, Meher LK. Complicated falciparum malaria. *Indian Pediatr*. 1994;31:821–825.
22. Shah S, Ali L, Sattar RA, Aziz T, Ansari T, Ara J. Malarial hepatopathy in falciparum malaria. *J Coll Physicians Surg Pak*. 2009;19:367–370.
23. Kachawaha S, Pokharana R, Rawat N, Garg P, Badjatiya H, Kochar DK. Ultrasonography in malarial hepatitis. *Indian J Gastroenterol*. 2003;22:110.
24. Chawla LS, Sidhu G, Sabharwal BD, Bhatia KL, Sood A. Jaundice in *Plasmodium falciparum*. *J Assoc Physicians India*. 1989;37:390–391.
25. Joshi YK, Tandon BN, Acharya SK, Babu S, Tandon M. Acute hepatic failure due to *Plasmodium falciparum* liver injury. *Liver*. 1986;6:357–360.
26. McMahon AE, Kelsey JE, Derauf DE. Hepatitis of malarial origin: clinical and pathologic study of fifty-four Korean veterans. *AMA Arch Intern Med*. 1954;93:379–386.
27. Kleeberg J, Birnbaum D. Studies on liver damage in acute malaria. *Trans R Soc Trop Med Hyg*. 1948;41:555–566.
28. Silva-Pinto A, Ruas R, Almeida F, Duro R, Silva A, Abreu C, *et al.* Artemether–lumefantrine and liver enzyme abnormalities in non-severe *Plasmodium falciparum* malaria in returned travellers: A retrospective comparative study with quinine-doxycycline in a Portuguese centre. *Malar J*. 2017;16:43.
29. McCarthy JS. A phase II pilot trial to evaluate safety and efficacy of ferroquine against early *Plasmodium falciparum* in an induced blood-stage malaria infection study. *Malar J*. 2016;15:469.
30. Griffin P. Safety and reproducibility of a clinical trial system using induced blood stage *Plasmodium vivax* infection and its potential as a model to evaluate malaria transmission *PLoS Negl Trop Dis* 2016;10:e0005139.
31. Nyunt MM, Hendrix CW, Bakshi RP, Kumar N, Shapiro TA. Phase I/II evaluation of the prophylactic antimalarial activity of pafuramidine in healthy volunteers challenged with *Plasmodium falciparum* sporozoites. *Am J Trop Med Hyg*. 2009;80:528–535.
32. Epstein JE, Rao S, Williams F, Freilich D, Luke T, Sedegah M, *et al.* Safety and clinical outcome of experimental challenge of human volunteers with *Plasmodium falciparum*-infected mosquitoes: An update. *J Infect Dis.* 2007;196:145–154.
33. Watkins PB, Kaplowitz N, Slattery JT, Colonese CR, Colucci SV, Stewart PW, *et al.* Aminotransferase elevations in healthy adults receiving 4 grams of acetaminophen daily: A randomized controlled trial JAMA. 2006;296:87–93.
34. Wilairatana P, Looareesuwan S, Charoenlarp P. Liver profile changes and complications in jaundiced patients with falciparum malaria. *Trop Med Parasitol*. 1994;45:298–302.