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Monitoring of malarial condition in adult patients with respect to haematological parameters

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Abstract
The morbidity and occasionally mortality related with malaria is high and these haematological factors show a significant part in it. Some current studies have shown that P. vivax can also be a source of severe disease and the haematological constraints are usually exaggerated in such cases. Hence the current study was planned to assess the haematological changes in the malarial patients.
The 50 patients identified with the malarial condition in the Department of Medicine in Narayan Medical College & Hospital were enrolled in to the present study. All the patients are informed consents. The entire patient's clinical history was collected. The approval of the institutional ethical committee was received before conduct of the present study. Inclusion Criteria: The patients showing positive malarial signs in pathological diagnosis. Exclusion Criteria: Patients without any other complications were excluded into the study.
The P. Falciparum is the major causative organism responsible for the malaria. Along with it the P. Vivax is also found to cause the malaria. Haematological abnormalities are encountered in vivax malaria. Derangements in haematological and biochemical parameters were more frequently seen in patients with malaria. Hence the precautions should be taken to monitor the changes in the haematological parameters in the malarial patients.

Keywords: malaria, haematological changes, plasmodium falciparum, plasmodium vivax

Introduction
Malaria is a potentially life threatening disease caused by parasites (Plasmodium vivax, Plasmodium falciparum, Plasmodium malariae and Plasmodium ovale) that are transmitted through the bite of infected Anopheles mosquitoes.
In 2014, 91 countries and territories had on-going malaria transmission and an estimated 3.2 billion people – nearly half the world's population – were at risk of malaria. The year saw an estimated 216 million cases and 445000 deaths due to malaria worldwide. Malaria is an acute febrile illness. In a non-immune individual, symptoms usually appear 10–15 days after the infective mosquito bite. The first symptoms – fever, headache, and chills– may be mild and difficult to recognize as malaria. If not treated within 24 hours, P. falciparum malaria can progress to severe illness, often leading to death.
Children with severe malaria frequently develop one or more of the following symptoms: severe anaemia, respiratory distress in relation to metabolic acidosis, or cerebral malaria. In adults, multi-organ involvement is also frequent. In malaria endemic areas, people may develop partial immunity, allowing asymptomatic infections to occur [1].
Malaria infection develops via two phases: one that involves the liver (exoerythrocytic phase), and one that involves red blood cells, or erythrocytes (erythrocytic phase). When an infected mosquito pierces a person's skin to take a blood meal, sporozoites in the mosquito's saliva enter the bloodstream and migrate to the liver where they infect hepatocytes, multiplying asexually and asymptomatically for a period of 8–30 days [2]. After a potential dormant period in the liver, these organisms differentiate to yield thousands of merozoites, which, following rupture of their host cells, escape into the blood and infect red blood cells to begin the erythrocytic stage of the life cycle. The parasite escapes from the liver undetected by wrapping itself in the cell membrane of the infected host liver cell [3].
Within the red blood cells, the parasites multiply further, again asexually, periodically breaking out of their host cells to invade fresh red blood cells. Several such amplification cycles occur. Thus, classical descriptions of waves of fever arise from simultaneous waves of merozoites escaping and infecting red blood cells. Some P. vivax sporozoites do not immediately develop into exoerythrocytic-phase merozoites, but instead, produce hypnozoites that remain dormant for periods ranging from several months (7–10 months is typical) to several years. After a period of dormancy, they reactivate and produce merozoites. Hypnozoites are responsible for long incubation and late relapses in P. vivax infections, although their existence in P. ovale is uncertain [4].
The parasite is relatively protected from attack by the body's immune system because for most of its human life cycle it resides within the liver and blood cells and is relatively invisible to immune surveillance. However, circulating infected blood cells are destroyed in the spleen. To avoid this fate, the P. falciparum parasite displays adhesive proteins on the surface of the infected blood cells, causing the blood cells to stick to the walls of small blood vessels, thereby sequestering the parasite from passage through the general circulation and the spleen. The blockage of the microvasculature causes symptoms such as in placental malaria. Sequestered red blood cells can breach the blood–brain barrier and cause cerebral malaria [5].
According to the World Malaria Report 2014, 22% (275.5m) of India's population live in high transmission (> 1 case per 1000 population) areas, 67% (838.9m) live in low transmission (0–1 cases per 1000 population) areas and 11% (137.7m) live in malaria-free (0 cases) areas. In 2013, 0.88 million cases have

been recorded, with 128 million tests being conducted on the suspected cases, with P. falciparum causing 53% and P. vivax causing 47% of the infections. The incidence of malaria in India accounted for 58% of cases in the South East Asia Region of WHO [6].

At present, official figures for malaria in India, available at NVBDCP [7]. Indicate 0.7–1.6 million confirmed cases and 400-1,000 deaths annually. The malaria parasites, entering the blood after an infective mosquito bite, infect red blood cells. At the end of that infection cycle, red blood cell ruptures. This process lowers the amount of red blood cells and can in a severe stage cause severe anaemia.

Haematological changes, which are the most common systemic complications, play a significant role in these serious complications. The haematological abnormalities that have been reported to consistently companion which comprise anaemia, thrombocytopenia, and atypical lymphocytosis and infrequently disseminated intravascular coagulation. Leukopenia, leukocytosis, Neutropenia, Neutrophilia, Eosinophilia and monocytosis also have been reported. In tropical countries like India, the majorities of the shared complications commencing due to malarial consequences is from hyperparasiteamia. Mortality is very high (10- 30%) in complicated P. falciparum infection.

The morbidity and occasionally mortality related with malaria is high and these haematological factors show a significant part in it. Some current studies have shown that P. vivax can also source of severe disease and the haematological constraints are usually exaggerated in such cases. Hence the current study was planned to assess the haematological changes in the malarial patients.

Materials & Methodology

The 50 patients identified with the malarial condition in the Department of Medicine in Narayan Medical College & Hospital were enrolled in to the present study. The study was conducted from March 2014to December 2014. All the patients are informed consents. The entire patient's clinical history was collected. The approval of the institutional ethical committee was received before conduct of the present study.

Inclusion Criteria: The patients showing positive malarial signs in pathological diagnosis.

Exclusion Criteria: Patients negative for malaria in pathological diagnosis.

Results & Discussion

The 50 patients having positive malarial signs were enrolled into the present study. The data regarding there haematological changes were studied and presented as below. The patient's age group and the number of cases data were presented in the table 1.

Table 1: Age group Vs number of cases

Age Group	Number of Cases	Percentage of Cases
20-30	10	20
31-40	18	36
41-50	8	16
51-60	8	16
61 and above	6	36
Total	50	100

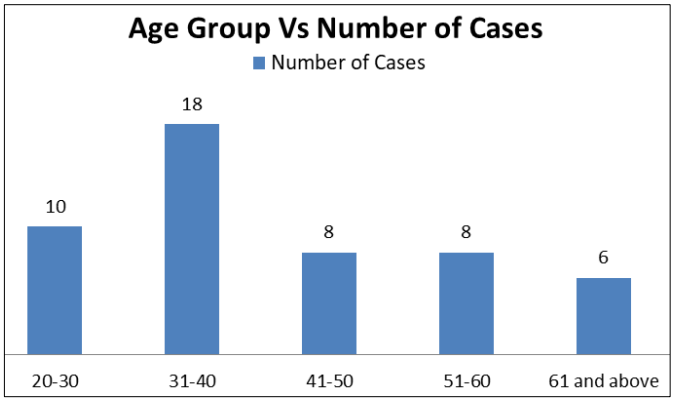

Fig 1

The table 1 suggest that there are 10 cases in age 20-30 years group. The 18 patients were identified in the 31-40 years of age group. The age group 41-50 had about 8 patients of the malaria. The higher age patients of 61 years and above had 6 patients. In Layla A.M. *et al.* [8] study mean age of patients was 25.43years. Khaled Taha *et al.* [9] study mean age of patients was 33.2 years.

Out of the 50 cases 15 patients were females and 35 cases are males. The male to female ratio is 354: 1. In Layla A.M. [8] *et al.* study, there was a male predominance, with a male to female ratio of 3.15:1. In Khaled Taha *et al.* [9] study 77 were male (74.8%) and 26 were female (25.2%) and male to female ratio was 2.96:1. Males are more to malaria due to more daily exposures and working conditions.

Table 2: Infection type with number of cases

Infection type	Number of Cases	Percentage of Cases
P. Falciparum infection	32	64
P. Vivax infection	12	24
Mixed	6	12

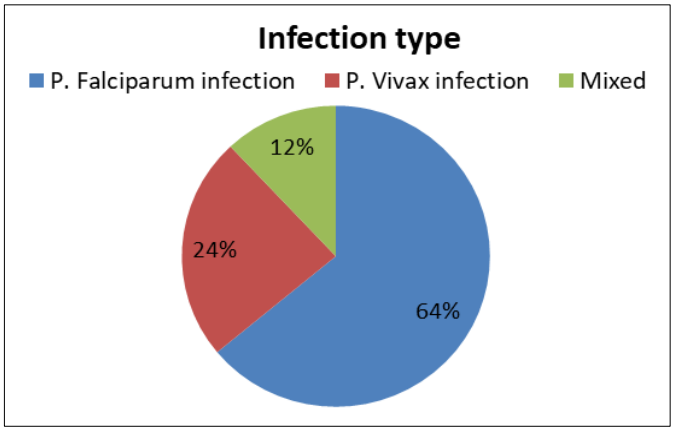

Fig 2

In the 50 patients, 32 patients were observed with P. Falciparum infection. 12 patients were observed with P. Vivax infection. 6 cases are observed with mixed type of infections. In Dr Shamim Akhtar *et al.* [10] P. Falciparum malaria was commoner than P. Vivax having 39 cases (52.7%) versus 27 cases (36.48%) respectively, while mixed infection represented only 8 cases (10.81%). In Khaled Taha *et al.* [9] the most

common type of malaria was P. Falciparum (54.2%), followed by P. Vivax (39%), then (mixed) infections in 17(2.4%). So there has been change in the epidemiological pattern of malaria.

Table 2: Haematological profile in the malarial patients

Haematological Parameter	Number of Cases
Blood Urea %	
▪ 41-100	45
▪ 101-200	5
Serum Bilirubin (per cu mm)	
▪ Less than 1.2 mg/dl	32
▪ Between 1.2-1.5 mg/dl	16
▪ More than 5-10 mg/dl	2
Haemoglobin	
▪ Less than 5 mg	5
▪ Between 5-10 mg	15
▪ More than 10 mg	30
Serum Creatinine mg	
▪ 1.4-3	36
▪ 3-10	10
▪ More than 10	4
Platelet Count (per cu mm)	
▪ Less than 50,000	12
▪ Between 50,000-1,50,000	18
▪ More than 1,50,000	20
SGPT	
▪ Less than 10-40 IU/L	30
▪ Between 1.2-1.5 IU/L	15
▪ More than 5-10 IU/L	5

Table 2 showed Haematological profile in the malarial patients. The haemoglobin level showed that 40% of the patients showed the anaemia. 5 patients are having significant lower level of Hb. As the per platelets count data 12 patients showed severe thrombocytopenia whereas 18 patients showed moderate thrombocytopenia. The 32 patients are having bilirubin level less than 1.2 mg/dl. 16 patients had Serum Bilirubin Between 1.2-1.5 mg/dl. And only 2 patient had bilirubin level More than 5-10 mg/dl. The SGPT level of less than 10-40 IU/L was found in 30 patients. Between 1.2-1.5 IU/L SGPT was seen in 15 patients. 56 patients showed the SGPT level more than 5-10 IU/L. 9 patients showed the blood urea level of above normal level. 45 patients were having normal blood urea levels. 5 patients showed higher level of the Serum Creatinine.

Conclusion

The P. Falciparum is the major causative organism responsible for the complications in malaria. Along with it the P. Vivax is also found to cause the malarial complications. Haematological abnormalities are encountered in vivax & Falciparum malaria. Derangements in haematological and biochemical parameters were more frequently seen in patients with Falciparum malaria. Hence the precautions should be taken to monitor the changes in the haematological parameters in the malarial patients.

References

1. http://www.who.int/en/news-room/fact-sheets/detail/malaria
2. Bledsoe GH. Malaria primer for clinicians in the United States. Southern Medical Journal. 2005; 98(12):1197-204. Quiz 1205, 1230. Doi: 10.1097/01. smj. 0000 189904.50838.eb. PMID 16440920. Archived from the original on 2011-05-14.
3. Vaughan AM, Aly AS, Kappe SH. Malaria parasite pre-erythrocytic stage infection: Gliding and hiding. Cell Host & Microbe. 2008; 4(3):209-18.
4. Richter J, Franken G, Mehlhorn H, Labisch A, Häussinger D. What is the evidence for the existence of Plasmodium ovale hypnozoites?. Parasitology Research. 2010; 107(6):1285-90. Doi: 10.1007/s00436-010-2071-z. PMID 20922429.
5. Rénia L, Wu Howland S, Claser C, Charlotte Gruner A, Suwanarusk R, Hui Teo T, *et al*. Cerebral malaria: mysteries at the blood-brain barrier. Virulence. 2012; 3(2):193-201. doi:10.4161/viru.19013. PMC 3396698. PMID 22460644.
6. www.who.int/mediacentre/factsheets/fs094/en/
7. nvbdcp.gov.in/malaria-new.html
8. Layla AM, Bashawri FCP, Ahmed A, Mandil PH, Mirghani A. Ahmed MD. Malaria hematological aspects. Annals of Saudi Medicine. 2002; 22:5-6.
9. Khaled Taha, Soheir Zein El-Dein, Majid Idrees, *et al*. Hematological Changes in Malaria: Relation to Plasmodium Species Kuwait Medical Journal. 2007; 39(3):262-267.
10. Shamim Akhtar, Raghvendra Gumashta, Sadhana Mahore, *et al*. Hematological changes in malaria: A comparative study. IOSR Journal of Pharmacy and Biological Sciences (IOSRJPBS).