Research Article

Clinical profile of plasmodium vivax and plasmodium falciparum malaria in tertiary care hospital (Western Maharashtra)

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Abstract

This study was done on 60 patients, diagnosed cases of plasmodium vivax and plasmodium falciparum malaria. Who were admitted in Bharati Hospital Sangli Western Maharashtra during the period of 12 Months (1 Jan 2012 – 31 December 2012). All the patient required hospitalization due to complication of malarial fever, like convulsion, altered behavior, intermittent, paroxysms of fever. 56 patients recovered completely without any complication. 4 patient expired due to cerebral malaria. Among 3 were plasmodium vivax, 1 plasmodium falciparum malaria. All severe complication like jaundice, renal failure, cerebral malaria, severe anemia and ARDS (Acute Respiratory Distress Syndrome) were noted in plasmodium vivax malaria more commonly as compared to plasmodium falciparum malaria.

Keywords: Plasmodium Falciparum Malaria, Plasmodium Vivax Malaria, ARDS, cerebral malaria, Jaundice.

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INTRODUCTION

Malaria is most important of all parasitic diseases; it is widely distributed in tropical and subtropical zones; As India is tropical country. The discovery of malaria parasite, nearly a century ago in Algiers by Lavarian: relationship of malaria parasite, Anopheles mosquito and man was found out in the last year of nineteenth century by Ronald Ross in India. The most common mode of malaria transmission is by infected mosquito bite to humans. The infection can be however transmitted accidentally. This can occur as a result of blood transfusion, and even rarely solid organ transplant when the donor harbors malaria parasite.[18] Ever since the unending battle between man and Malaria is on. In 1939 after the insecticidal properties of DDT Venezuela was the first country to launch of eradication program against malaria in 1945. In 1955, the Eight World Health Assembly recommended the eradication of malaria as an international objective, following the reported development of resistance by the vector to the insecticides in many countries. Malaria remains to be one of the world's most prevalent infectious disease. About 300-500 million cases are reported annually allover the world with a mortality of about 1.1 to 2.7 million. In India, 2.5 to 3 million cases and 1000 deaths of malaria are reported annually. North East India, Orissa, Jharkhand, West Bengal, Madhya Pradesh, Maharashtra and Andhra Pradesh; these areas are categorized as high risk areas. In these regions P. Falciparum malaria incidence is more than 30%. In India the fight against malaria was started in 1953 with the National malaria Control programs, the shift to the National Malaria Eradication was undertaken in 1958. National Malaria control program has taken, it is due to drug resistant parasites and its treatment failure. The vicious circle of disease – low income, bushy area, poor health services, more disease and more poverty not only poses a problem of health and sanitation but also the welfare of the society at large. In order to avoid such a situation it seems pertinent to attempt an interdisciplinary study, which may

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help in studying of the disease pattern and its effective management.

AIM AND OBJECTIVES

To study the clinical profile of Plasmodium Vivax and Plasmodium Falciparum malaria in tertiary care Hospital (Western Maharashtra)

1. To study clinical manifestations of malaria.
2. To classify type of malaria.
3. To study laboratory findings of malaria.
4. To study complications of malaria.
5. To find out outcome in terms of mortality in different clinical types of malaria.

To study clinical outcome after drug therapy.

MATERIAL AND METHODS

Inclusion Criteria

1. All Patients admitted in Bharti Vidyapeeth Medical College and Hospital Sangli with history of fever will be evaluated as following

Clinical Features

- Intermittent paroxysm of fever, Headache , Chills, Vomiting, Diarrhea,
- Icterus, Hepatosplenomegaly, Loss of consciousness, Convulsions, Altered behavior

Laboratory Parameters

a. Hemogram Hb. (hemoglobin)
- TLC (Total leucocyte count)
- DLC (Differential leucocyte count) Platelet count
- Peripheral smear for malarial parasite.

b. Rapid malaria antigen test
c. Renal Function: Blood urea Serum creatinine
d. Blood sugar
e. Liver function tests: Total bilirubin, Direct bilirubin, Indirect bilirubin, SGOT,SGPT, Alkaline phosphatase
f. ECG: To be done before and after quinine therapy to rule out ECG abnormality i.e. QT Changes.
g. Chest X – ray

Inclusion Criteria

All these new patients above 18 years with fever confirmed by peripheral smear for malaria (Parasite)/Rapid malaria antigen test positive are included in this study

Exclusion Criteria

Following patients are excluded from study

1) Enteric fever, Hepatitis, Dengue fever , Leptospirosis, Leukemia, Lymphoma by doing appropriate tests.

Study Duration: 1st January, 2012 to 31st December 2012

Table 1: Quantitative aspects of thick and thin blood films for examination of malaria parasites, The level of parasitaemia: a blood film showing only young trophozoites

	Thin film	Thick film
Area on slide	250-450 mm2	50-90 mm2
Blood volume	1 |L	3-5 |l
Mean thickness	0.0025 mm	0.05-0.09 mm
Mean difference in concentration	1	20-30
Volume in 100 microscope fields (obj.X100; ocul.X6)	0.005-0.007 |L	0.1-0.25 |L
Time for examination (approximate)	200-300 fields/20-25 minutes	100 fields/5 minutes
Loss of leucocytes or parasites during staining	None	Leucocytes up to 8% Parasites up to 20%
Red blood cells	Fixed	Haemolysed
Parasite morphology	Not distorted	Distorted
Parasite transfer during mass staining	Impossible	Likely
Artefacts	Uncommon	More common

In most situations, the 'gold standard' for individual diagnosis is the microscopical examination of thick and thin films. There are, however, situations where this may not apply. In areas of high endemicity, clinical diagnosis alone is usually the only feasible and cost-effective method for recommending. When microscopy is not available, the use of dipstick antigen detection tests may be of value, particularly in areas of low endemicity, where infection usually coincides with disease.[65,66] There is a correlation between density of parasitaemia and severity of malaria. It is considered that any P. falciparum parasitaemia above 250 000/^L (approximately 5 per cent of red blood cells) should be taken as a sign of severity requiring emergency treatment.[60,63]

Table 2: Treatment

Uncomplicated P Vivax	Chloroquine + Primaquine Oral chloroquine 10 mg / kg max 600 mg followed by 5 mg / kg max 300 mg after 6,24,48 hours +. In chloroquine resistance cases aretumesinin best combination therapy i.e.

	artremether plus lumefaritine. Artesunate + mefloquine If chloroquine sensitive dose as above. ACT + Primaquine (7 days) (14 days).
Uncomplicated P. Falciparum	If chloroquine sensitive dose as above. ACT + Primaquine (7 days) (14 days). Artemeter + Lumefenter Artesunate + amodiquine Artesunate + mefloquine Artesunate + Salfadoxime+Pyrimethamine Alternate treatment (second line) Artesunate + Tetra or Doxy, clindamycine for 7 days. Quinne + Tetra , Doxy , clindamycine for 7 days.
Complicated Malaria P. Falciparum	Intravenous artesunateInj – artesunate 2 mg / kg Im first day 1 mg / body kilogram after 6 hours and daily for 4 days. Or Artemether 160 mg on day one 80 mg on day second day, Intramuscular injection. Quinine IV loading dose 20 mg of salt / kg body weight. Stat – dose. Maintenance dose, 10 mg / kg salt. 8 hourly for 7 days. We can give parental therapy for maximum 24 hours if patient can tolerate oral medication. Artemether + lumefantrine. Artesunate + amodiaquine Dihydroartemisin + piperaquinine. Artesunate + Clindamycin.
Complicated P. Vivax treatment	Same as P. Vivax + Primaquine (except sulfadoxime,+pyrimethmine.)

Many studies have been done on Clinical profile of Malaria. Following are some of these studies done recently. **Muddaiah M*et al,*** conducted a study in South Canara district of Karnataka and total of 314 patients were diagnosed and treated for malaria. Study concluded that malaria is responsible for major health concern particularly in rainy season and is found to affect comparatively the younger adult population. Males (81%) outnumbered females (19%) and many were within the age group of 21-30 yr. P. vivax was the major parasite type(52.54%), causing malaria and most of the complications were due to P. falciparum. Hepatopathy was the most common complication and all the deaths were due to cerebral malaria. **Wasnik PN *et al,*** conducted a hospital based cross sectional study on 80 confirmed cases of falciparum malaria. Study concluded that 75% were males and 25% were females. Most of the patients were between the age group of 21-30. Fever was the most common symptom followed by impaired consciousness. Anemia was present in 65% patients, out of which 6.25% patients had severe anemia. Abnormal liver function tests were observed in 35% of subjects while abnormal kidney function tests were observed in 32.5% of patients.77% patients received the combination of artesunate and clindamycin. This also showed that the combination of artesunate and clindamycin in severe Plasmodium falciparum malaria is a very good therapeutic option. . Mortality rate was 6.25%. **V.C.Patil *et al*,** conducted a study including 47 patients, 39 were male and 8 were female patients. 29(61.70%) patients had jaundice of which 20 were with anemia. Total 22(46.80%) had anemia of which 20 were with jaundice. Total 6 (12.76%) had cerebral malaria, 6 (12.76%) had acute renal failure (ARF). In present study most common presentation was jaundice and anemia. Overall case fatality rate was 10.63% (5/47). **N. Nanjundaiah *et al,*** conducted a study including 174 cases. Study concluded that 93 were male and 81 female. Complicated malaria was seen in 38 cases (21.83%) with positivity in 20 cases. Mortality was 04.59% of clinical malaria cases, all admitted with multiple organ dysfunction syndrome, no mortality in confirmed cases. Of the remaining 136 cases malarial was confirmed in 42, while 94 were clinical malaria. 18 were positive for *P.falciparum*, 18 for *P.vivax*. 6 patients were positive for both *falciparum* and *vivax*.

OBSERVATIONS AND RESULTS

Table 1: Age and Sex distribution

Age range in years	Plasmodium Vivax		Plasmodium Falciparum		Total
	Male	Female	Male	Female	
15-40	20	8	5	4	37
41-70	11	4	2	1	18
> 71	3	1	1		5
Total	**34**	**13**	**8**	**5**	**60**

Table 2: Clinical Profile of PV and PF Malaria

Clinical Features	Plasmodium Vivax	Plasmodium Falciparum	Total	P
Fever	47	13	60	0.000
Chilis	42	9	51	0.000
Headache	30	10	40	0.000
Myalgia	23	6	29	0.000
Vomiting	12	3	15	0.000
Diarrhea	4	1	5	0.000
Icterus	8	10	18	0.258
Splenomegaly	6	10	16	0.011
Hepatomegaly	8	1	9	0.000
LOC	5	1	6	0.000
Convulsion	2	1	3	0.000

Table 3: Jaundice - SERUM BILIRUBIN

Total Bilirubin	Plasmodium Vivax (n=47)	Plasmodium falciparum (n=13)	Total (n = 60)	P
1.5-3	32	8	40	0.000
>3 (WHO criteria)	15	5	20	0.000

Table 4: Transaminases - SGOT

SGOT IU/L	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
< 40	6	02	8	0.000
40-80	06	08	14	0.177
> 80	12	09	21	0.177

Table 5: Transaminases - SGPT

SGPT IU/L	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
< 40	04	02	06	0.000
40-80	07	07	14	0.634
> 80	13	10	23	0.177

Table 6: Renal Failure- Serum Urea

Urea	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
< 40	0	0	0	-
40-80	07	04	11	0.004
> 80	09	06	15	0.045

Table 7: Renal failure - Serum creatinine

Serum Creatinine	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
1.5-3	12	04	16	0.000
> 3 (WHO criteria)	04	03	07	0.177
Total	**16**	**7**	**23**	**0.000**

Table 8: Cerebral Malaria

Symptoms	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
LOC	04	2	06	0.000
GCTS and LOC	01	01	02	0.855
GCTS	01	01	02	0.855
Total	**06**	**04**	**10**	**0.045**

Table 9: ANEMIA

Hemoglobin	Plasmodium Vivax	Plasmodium falciparum	Total	P
8-10	34	07	41	0.000
5-8	11	04	15	0.000
< 5 (WHO criteria)	02	02	4	0.634
Total	**47**	**13**	**60**	**0.000**

Table 10: Anemia with other organ dysfunction

Anemia with	Plasmodium vivax (n=47)	Plasmodium Falciparum (n=13)	Total (n=60)	P
Renal failure	02	06	08	0.000
Jaundice	09	04	13	0.000
Cerebral Malaria	05	03	08	0.011

Table 11: Jaundice with other organ dysfunction

Jaundice with	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
Anemia	09	04	13	0.000
Renal failure	09	05	14	0.004
Cerebral malaria	06	04	10	0.045

Table 12: Cerebral malaria with other organ dysfunction

Cerebral malaria with	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
Anemia	05	03	08	0.011
Renal failure	06	03	09	0.000
Jaundice	06	04	10	0.045

Table 13: Renal failure with other organ dysfunction

Renal failure with	Plasmodium vivax (n=47)	Plasmodium falciparum (n=13)	Total (n=60)	P
Annemia	06	02	08	0.000
Jaundice	09	05	14	0.004
CNS Dysfunction (cerebral malaria)	06	03	09	0.000

Table 14: Complications

Complications	Total	PV	PF	Significance P value
Anemia HB with < 5 gm/dL	15	11	04	0.000
Jaundice	17	09	04	0.002
Renal failure	23	16	7	0.000
Cerebral malaria	10	6	4	0.045
Thrombocytopenia	36	29	07	0.000
ARDS	04	03	01	0.000
Hypotension	18	12	06	0.000
Cortical venous thrombosis	02		01	-
Hypoglycemia	21	16	5	0.000

Table 15: TREATMENT

Sr. No.	Type of malaria	No. of Cases	Treatment	Out come
1.	Uncomplicated P.Vivax	42	23 patients responded to Chloroquine. 20 patients Choroquine resistance. 12 patients responded to Artemether+Lumafantrin combination therapy. 7 so they were added with Doxycycline 100mg responded well.	No death occurred.
2.	Uncomplicated P.Falciparum	3	ACT + Primaquine responded well.	No death occurred.
3.	Severe P.Vivax	3	Inj.Artemether + Primaquine, 1 patient responded well. Inj.Artemether + Quinine + Doxycycline, 2 patients did not respond.	3 patients died with other complications MODS,ARDS.
4.	Severe P.Falciparum	2	Artesunate + Clindamycin	1 death occurred.

Table 16: Mortality

PlasmodiumVivax (n = 47)	Plasmodium falciparum (n = 13)	Total (n = 60)	P
03	01	04	0.994

Table 17: Severe P. Vivax

No of Patient	Drugs Given	Response	Outcome
2	Artesunate + Primaquine	1 patient responded	1 Pt improved clinically with improvement In Lab parameters
1	Patient was given inj. Artemether + Quinine + Doxy	Not Responded	1 Pt died due to MODS and ARDS Lead to ARDS and MODS patient Died.

DISCUSSION

In our study, we have treated the patients according to the protocol by WHO. 24 out of 44 Patients with uncomplicated pv responded to chloroquine and showed improvement clinically and in laboratory parameters. In 20 patients, Chloroquine showed no clinical response. Alternate treatment was given to these patients.12 of these patients responded to ACT while remaining 8 needed to add Doxycycline for their recovery. In our study, ACT was found to be very effective in complicated malaria and PF patients. Early initiation of ACT resulted in reducing morbidity and mortality. Wasnik PN*et al* had similar findings in their study. In cerebral malaria quinine was very valuable leading to rapid recovery in one or two doses. Regarding side effects of quinine, vomiting was the commonest to be noted and other complications like QTc prolongation and amblyopia were not found in our study. In our study, we found 3patients with uncomplicated PF. Which were responded to ACT and single dose of primaquine. We also found 3 cases of complicated p.vivax malaria and 2cases of complicated P.falciparum malaria. One patient (complicated PF) responded to ACT, Quinine and supportive care four patients died which accounts for 1.6% mortality rate 3 had complicated PVand 1had complicated PF. Death occurred due to development of MODS with ARDS. This is consistent with V. C. Patil *et al* findings of shift in cause of mortality from cerebral malaria to MODS. Low mortality may be due to early detection of organ dysfunction and aggressive management. This study has highlighted that plasmodium falciparum less in this area. Due to less rainfall or drought area p.falciparum malaria are less. Complications occurred in vivax also. There was very good response to ACT or Quinine and Doxycycline in complicated malaria. Mortality was low (1.6%). Early diagnosis of malaria, rapid evaluation for organ dysfunction and aggressive management was probably responsible for this low mortality

CONCLUSIONS

- Plasmodium vivax occurred in 78.3 % and Plasmodium falciparum in 21.7 %.
- Jaundice (66.6%) and Renal failure (38.8%) were the important complications in Plasmodium Falciparum,where as cerebral malaria occurred only in16.6 %.
- All severe complications like jaundice (66.6%), renal failure (11.6%), cerebral malaria (16.6%), severe anemia(6.6%) and ARDS(6.6%) were noted in Plasmodium vivax were more common as compared to Plasmodium falciparum.
- Early detection of organ dysfunction utilizing Serum Creatinine >1.5mg/dl, total bilirubin >1.5mg/dl and Hb<8g/dl is valuable in diagnosing complicated malaria early and starting aggressive management (ACT, Quinine and Doxycycline), thereby prevents further morbidity and mortality.

REFERENCES

1. Taylor TE Strickland GT. Malaria. Chapter 92 in Stricklands infectious Disease 4th London: Wiley, 2006: 614-642
2. K.Park. “Malaria: Chapter 5 in Preventive and social medicine. 17thednPublisheresBanarsideasBhanot Publishers, Jabalpur 2002:192-201
3. Bashawari LAM, Mandil AA, Bahnassy AA, Ahemed MA. Malaria Haematological Aspects. Annal of Saudi Medicine 2002:22:372-7
4. Kelkar DS, PAtnaik MM, Joshi SR. Malarial Hematopathy. JAssoc Physicians India 2004:52:611-12
5. Niazi GA. Haematological aspect of Malaria in a population based hospital Soudi Arabia. J Egypt SocParasitol 1995:25:787-93
6. Sen R Tewari AD, Sehgal PK Singh U, Sikka R, Sen J Clinicohaematological profile in acute and chronic plasmodium falciparum malaria in children. J Com Dis 1994:26:31-38
7. Rojanasthien S, Surakamolleart V, Boonpuknavig S, Isarangkura P. Heamatological and Coagulation studies in Maleria. J Med Assoc Thai 1992; 75 Supl 1:190-194
8. Sharma SK, Das RK, Das PK, Heamatological and coagulation profile in acute falciform malaria, J Assoc Physician India 1992; 40:581-3

9. Srichaikul T, Pulket C. Sirisatepisarn T, Prayoonwiwat W. Platelet dysfunction in malaria Southeast Asian J Trop Med Public Health 1988; 19:225-33
10. Kakar A, Bhoi S. Prakash V, Kakar S, Profound thrombocytopenia inplasmodiaumvivax malaria. DiagnMicrobiol Infect Diss. 1999; 35:243-44
11. Horstmann RD, Dietrich M, Binzle U, Rasche H, Malaria induced thrombocytopenia. Blut 1981;42:157-64
12. Makkar RPS, Mukhopadhyay S, Monga A, Gupta A. Plasmodium vivax malaria presenting with severe thrombocytopenia Braz J Infact Dis.2002:6:263-65
13. Gilles HM, Historical Outline In: Gilles Hm. Warell DA. Editors Essential malariology. 4Ed.London: Ornold, 2002;1-7
14. Schagenhauf P. Malaria: from prehistory to present. Infect Dis Clinb N Am 2004;18:189-205
15. Chaterjee KD, Sporozoa, Chpter 3 ParasitalogyPrtozoology and Helminthology. 12th ed Culcutta, Chaterjee Medical Publisher, 1981; 70-103

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