Original Article

Study on Presentations and Treatment Outcome of Plasmodium Falciparum Malaria

Treatment of Plasmodium Falciparum Malaria

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ABSTRACT

Objective: To determine the presentation and treatment response to anti malarial drugs of Plasmodium Falciparum malaria.

Study Design: Descriptive / prospective study.

Place and Duration of Study: This study was conducted at the Department of Medicine, PMCH Nawabshah from January 2016 to April 2017.

Materials and Methods: 100 patients were selected for this study, statical analysis was done using SPSS 15 version. Inclusion Criteria is ICT Malaria test positive for Plasmodium Falciparum. Thick and Thin flim positive MP Plasmodium Falciparum. Exclusion Criteria is ICT Malaria test, and thick thin film negative for Plasmodium Falciparum, tuberculosis and typhoid fever.

Results: 100 patients were selected for this study, 53 were males, 47 were females. Age ranged 13-70 years. All patients presented with fever, temperature ranged 100^0F-105^0F.Anemia was present in 40 patients, 20 patients were jaundiced clinically. Altered consciousness was present in 30 patients. 18 patients had raised blood urea, bilirubin was raised in 20 patients, hemoglobin ranged 4-14 gm/dl, TLC ranged 6320-24209/mm^3, random blood sugar ranged 85-199 mg/dl, platelets ranged 40500-488245,PT was raised in 20 patients, LFT deranged in patients. All patients treated by inj. Artesunate2.5 mg/kg i/v BD for 1 day then daily . Out of 100 patients 13 died due to severity of disease.

Conclusion: Plasmodium Falciparum Malaria with complications is a major illness in our country especially patients from rural areas. Patients reach very late in hospital, Cerebral Malaria can be treated with artesunate atemether and quinine. Prevention and awareness is is necessary, mortality can be reduced.

Key Words: Malaria Plasmodium Falciparum

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INTRODUCTION

Plasmodium Falciparum Malaria is common disease in the developing world. According to WHO commonly affected country is sub-Saharan Africa .[1] Where death ratio in children under the age of five years is about 80%. Plasmodium Falciparum Malaria is transmitted by the bite of infected anopheles mosquito of genus plasmodium in human. These are plasmodium ovale, plasmodium malaria, plasmodium vivax, plasmodium falciparum and plasmodium knowlesi. More severe malaria occurs due to plasmodium falciparum and mortality is increased[2]. Clinical features include fever, malaise, headache and vomiting. Jaundice is due to liver dysfunction and hemolysis. Anemia is commonly present. Patient may present with tender hepatomegaly and spleenomegaly. Patients with Plasmodium Falciparum malaria may develop serious complications[3]. Plasmodium Falciparum malaria presented with seizures, ataxia,hemiplegia, coma and death. Neurological damage is common in cerebral malaria. 20% of children who survive after illness, develop cognitive deficits, learning and language impairments, memory impairment, psychiatric disorders, visuospatial and motor deficits.[4] In Europe malaria is common in travelers with morbidity and mortality[5]. Transmission of malaria from human to mosquito depends upon the presence of sexual stages in blood, after the cycle hundred to thousands of sporozoites in the salivary gland of mosquito and infect the host[6]. Malaria is transmitted by 60 species of anopheles mosquito[7]. Life cycle change in mosquito before it becomes infectious to other healthy individuals. The time period is required for the life cycle change increases as the temperature declines, life span of mosquito, transmission is decreased when temperature falls below 18^0C. Malaria parasite cease development when temperature is below 16^0C and malaria is reduced in temperate regions[8]. Malaria transmission increased during rain fall and humudity[9]. Temperature variation due to change in

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weather is the main factor for the geographical distribution of the disease. In the tropical regions exposure to mosquitoes is increased. Several contacts with infected mosquitoes at night, due to such inoculation rates, increased duration of parasite survival in the host, saturate local human populations rapidly and superinfection universal prevalence occur. Due to stable pattern of transmission, even vector control repeatedly failed to eradicate the parasite from tropical and sub tropical regions even control is possible[10]. Diagnostic methods, drugs and control measures has been renewed over the past decade[11]. In 2006 malaria treatment outcome was improved with use with use of artiminism based combination therapy to address resistance of plasmodium falciparum to monotherapy recommended by WHO[12]. Recovery rate with artiminism was 90% and well tolerated[13]. WHO introduced rapid diagnostic test (RPT) in all cases of malaria[14]. Severe Falcparum malaria treatment recommendation is Artesunate 2.4 mg/kg i/v 12 hours for 1 day then daily. Alternate Quinine dihydrochloride 20 mg/kg i/v over 4 hours then 10 mg /kg i/v every 8hours or Artemether 3.2 mg/kg i/m then 1.6 mg/kg/d intravenously. Drugs for prevention of Falciparum Malaria are, chloroquine, malarone, mefloquine and doxycycline.

MATERIALS AND METHODS

This descriptive and prospective study was conducted in the department of medicine at PMCH Nawabshah from January 2016 to April 2017. 100 patients were enrolled for this study, admitted in the medicine department with the diagnosis of acute Falciparum Malaria, by history general physical examination and ICT Malaria positive for Plasmodium Falciparum. Informed consent was taken from all the patients. The purpose of this study was to study the clinical features and response to anti malarial drugs.

Inclusion Criteria: Age above 12 years. ICT positive for Plasmodium Falciparum. Thick and Thin flim Positive MP Plasmodium Falciparum.

Exclusion Criteria: Afebrile. ICT and Thick and Thin film negative for Plasmodium Falciparum. Age below 12 years

RESULTS

Out of 100 patients 53 were males and 47 were females. Age range from 13 to 70 years. All the patients presented with fever, duration of fever ranged 1 day to 15 days. Fever was low grade in 10 patients, moderate in 41 patients and high grade in 49 patients. Fever range from 100^0F to 105^0F. fever was continuous in 40 patients and intermittent in 60 patients. Fever was associated with rigors and chills in 42 patients, headache 78 patients, other symptoms were abdominal pain 28 patients, vomiting 23 patients, altered consciousness was present in 32 patients, respiratory symptoms in 15 patients. On physical examination BP ranged 60-180 mmHg. All patients were positive for Falciparum Malaria on ICT and thin and thick film (microscopy) Trophozoites in in 83 cases, combined trophozoites and gametocytes in 7 cases were seen. Anemia was present in 41 patients, jaundice was positive in 21 patients Hb range 4 to 14gm/dl. Bilirubin range 0.9 to 11mg/d ,leukocyte count 6320 to 24209, platelets count 40500 to 488245, random blood sugar was between 85 to 199, blood urea 25 to 210 mg/dl, serum creatinine ranged 0.8 to 12 mg/dl, 31 patients had raised urea and 15 patients had raised creatinine level. PT prolonged in 18 patients and dehydration was positive in 23 patients. All patients were given inj Artesunate 2.4 mg/kg i/v BD for 1day then daily, few patients received antibiotic treatment for chest infection and septicemia. Fluids were given according to electrolyte disturbance and dehyderation. Out of 100 patients 13 patients died. Death was due to cerebral malaria with septicemia and renal failure.

Table No.1: Descriptive Statistics:

	N	Minimum	Maximum	Mean	Std.Deviation
Age	100	13.00	70.00	40.5900	13.82071
Sex	100	1.00	2.00	1.4700	0.50161
Hemoglobin	100	4.00	14.00	9.1550	2.71425
L. count	100	6320.00	24209.00	21231.22	21227.42452
Pl.Count	100	40500	488245.00	206044.5	110532.53914
P.T	100	12.00	26.00	15.5600	4.20274
RBS	100	85.00	199.00	145.8500	31.25567
Urea	100	3.00	210.00	63.1100	55.50264
Creatinine	100	0.80	11.00	2.1270	2.70857
Biluribin	100	0.90	11.00	2.2970	2.67833
SGPT	100	29.00	187.00	55.5600	38.56683
Valid N (listwise)	100				

Table No.2: ANOVA

		Sum of squares	Df	Mean Square	F	Sig
Sex	Between Groups	10.698	45	0.238	0.903	0.635
	Within Groups	14.212	54	0.263		
	Total	24.910	99			
Hemoglobin	Between Groups	370.249	45	8.228	1.237	0.226
	Within Groups	359.099	54	6.650		
	Total	729.348	99			
L.Count	Between Groups	1.6E+010	45	364219722.2	0.697	0.892
	Within Groups	2.8E+010	54	522590076.7		
	Total	4.5E+010	99			
Pl.Count	Between Groups	6.0E+011	45	1.339E+010	1.191	0.268
	Within Groups	6.1E+011	54	1124E+010		
	Total	1.2E+012	99			
P.T	Between Groups	938.000	45	20.844	1.389	0.268
	Within Groups	810.640	54	15.012		
	Total	1748.640	99			
RBS	Between Groups	53777.476	45	1195.055	1.503	0.076
	Within Groups	42937.274	54	795.135		
	Total	96714.750	99			
Urea	Between Groups	212909.0	45	4731.312	2.775	0.000
	Within Groups	92064.740	54	1704.903		
	Total	304973.8	99			
Creatinine	Between Groups	531.961	45	4731.312	2.775	0.000
	Within Groups	194.336	54	3.599		
	Total	726.297	99			
Biluribin	Between Groups	476.360	45	10.586	2.445	0.001
	Within Groups	233.809	54	4.330		
	Total	710.169	99			
SGPT	Between Groups	98574.107	45	2190.536	2.430	0.001
	Within Groups	48678.533	54	901.454		
	Total	147252.6	99			

DISCUSSION

Cerebral Malaria is major health problem in many countries including Pakistan, patient presented with fever, chills, headache[9], anemia, bleeding from nose, delirium, coma and spleenomgaly. Plasmodium Falciparum Malaria causes major complications, hemoglobinurea, jaundice, shock, renal failure, lactic acidosis, abnormal bleeding, pulmonary edema and adult respiratory distress syndrome. Few patients develop cerebral venous or dural sinus thrombosis and cortical infarcts due to coagulation disorders. Patients presented with shock, bacterial infection[10]. Morbidity and mortality is due to irregular treatment and late treatment. Death in Plasmodium Falciparum Malaria is mainly due to respiratory failure and brain stem signs. Death commonly occur within 24 hrs of presentation in clinic or hospital[11]. The diagnosis of Plasmodium Falciparum malaria depend upon the neurological signs and asexual forms of the parasite on peripheral smear. It is necessary to exclude other causes of unconsciousness, e.g. bacterial meningitis, viral encphlitis and hypoglycemia. Malaria is fatal without treatment. In patients mortality was decreased in those patients who were on intravenous artesunate[12]. Some of the patients fully recovered few discharged with neurological deficit. central hypotonia ataxia and blindness occur and recover with time. Faciparum malaria can contribute to the development of epilepsy in later life. In some studies spinal cord lesions and peripheral neuropathy are reported in cerebral malaria, confirmed on nerve conduction studies and CSF examination[13]. In the acute phase of Plasmodium Falciparum Malaria the patients who die, many of the cerebral capillaries and venules are packed with parasitized RBC and other adjacent capillaries and venules are not obstructed. Coma and death associated with degree of packing and congestion of the cerebral micro vessels with infected and un infected RBC[14]. Retinopathy occurs in malaria in a study. Systemic pyruvate and lactate increased with severity of illness. In Plasmodium Falciparum Malaria micro vascular obstruction and impaired perfusion occurs as a pathophysiological process, mild vascular permeability

and increase vascular permeability with a disruption of endothelial intercellular junction[14]. Gross anemia occurs in young age in cerebral malaria, due to red cell removal by spleen and erythrocyte destruction at parasite shizogony. Anemia occurs rapidly and there is increased need of blood transfusion[15], thrombocy-topenia is usual and DIC is unusual. Acidosis results from accumulation of organic acids and death occur due to Plasmodium Falciparum Malaria. Acidotic breathing cause of respiratory distress is a poor prognostic sign of Cerebral Malaria. Combination of anaerobic glycolysis in tissues cause lactic acidosis, lactate production by malarial parasite and failure of real and hepatic clearance of lactate[16], hypovolumia is a contributory factor. Hypoglycemia associated with poor prognosis in Plasmodium Falciparum malaria associated with lactic acidosis, hypoglycemia is due to failure of hepatic gluconeogenesis. Due to treatment of quinine hyperinsulinaemic hypoglycemia occur[17], common in pregnancy. Quinine cause hypoglycemia due to powerful stimulation of pancreas and recurrent[17]. ARDS is complication of Cerebral Malaria. Due to anti malarial treatment pulmonary capillaries permeability is increased. In Cerebral Malaria pulmonary sequestration is increased, care in fluid management, rapid administration of large volume can be lethal[18]. Cerebral Malaria may present with oligouric renal failure, pathogenesis is unclear but inflammation and reduced microcirculatory flow. Early hem dialysis improve the condition of the patient[19]. Severe jaundice occurs in Plasmodium Falciparum Malaria more in adults than children, it is due to hemolysis, hepatic injury and cholestasis. In severe malaria liver blood flow is reduced, impaired gluconeogenesis, impaired drug metabolism, hypoglycemia and metabolic acidosis are due to hepatic dysfunction[20].

CONCLUSION

Uncomplicated Falciparum Malaria responds well to treatment, complicated Falciparum Malaria is major problem in our country especially in rural areas come late in teaching hospital, initially they receive treatment from GP or non qualified doctors. In our country mortality is common in adults and extended illness with complications. Mortality can be reduced by awareness about treatment prevention of malaria mosquito nets, spray and lotion. Poor prognosis for pregnant woman with prematurity, low birth weight baby and mortality.

Author's Contribution:

Concept & Design of Study:	Jeando Khan Daidano
Drafting:	Akbar Hussain Yousfani
Data Analysis:	Akbar Hussain Yousfani
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