FALCIPARUM MALARIA IN CHILDREN

Pages with reference to book, From 268 To 271

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Forty three cases of Falciparum Malaria in children are reported to draw attention to the second resurgence of malaria since the start of the malaria eradication (currently control) programme in Pakistan. The clinical features, laboratory findings and response to treatment are discussed. Clinical features are varied but fever with anaemia, splenomegaly, dark coloured urine and meningeal or intracranial irritation signs are highly suggestive of falciparum malaria in children.
The study has shown clinical resistance of plasmodium falciparum to amodiaquine, chloroquine and Fansidar a combination of chloroquine and pyrimethamine(JPMA 38: 268 , 1988).

INTRODUCTION

In Pakistan within ten years of the start of the malaria eradication programme, resurgence duction of newer insecticides, some reduction in prevalence was achieved by the late seventies but a resurgence has again been recorded since the early eighties. Resistance of the malarial parasite of malaria occurred owing to the development of resistance of the vector to DDT. After the intro- to chioroquine[1] has also occurred and reported from many areas including Pakistan.[2]
In Peshawar the influx of millions of Afghan refugees has added to the problem. This report is intended to draw attention to the large number of cases of falciparum malaria being seen here as well as its common clinical features, response to antimalarials and mortality from falciparum malaria.

PATIENTS AND METHODS

Between August 1985 and November 1986 forty three children with fever, without any obvious signs of infection, were investigated for malaria. Thick and thin smears were examined for malarial parasite. Haemoglobin, full blood count and absolute values were also done on venous samples of blood. Liver function tests were done when required. Urine examination for haemoglobin was carried out in cases with dark urine, and CSF examination in cases with meningeal or neurological signs. In two cases with bleeding, coagulation studies were also done. Brain scan was performed in three cases with signs of intracranial lesion.

RESULTS

All 43 cases were positive for malarial parasites. Thirty seven cases showed gametocytes of p. falciparum, with heavy parasitaemia of upto thirty percent in one case (Table I).

TABLE I. Parasitaemia number of Parasites/100 White Cells (Average : 6.3).

S.No	Number of Parasites	S.No.	Number of Parasites	S.No.	Number of Parasites
1	16	16	1	31	4
2	2	17	2	32	3
3	1	18	1	33	12
4	3	19	1	34	3
5	4	20	1	35	3
6	1	21	1	36	4
7	8	22	3	37	1
8	5	23	2	38	13
9	30	24	6	39	7
10	3	25	5	40	1
11	15	26	16	41	3
12	1	27	2	42	1
13	19	28	1	43	14
14	2	29	25		
15	5	30	21		

Peripheral smear of six cases showed trophozoite forms of the parasite.

Twenty seven were male children and sixteen female in the age range of eight months to ten years. The total duration of illness at the time of admission ranged from five to forty five days. History of three-four months illness was given by two patients. Fever was continuous or remittent type in eighty percent of the cases. Twenty percent had intermittent fever. Temperature ranged between 100°F and 105°F. Twenty percent of the patients had history of chills or shivering, but the periodic cold, hot and sweating stage was hardly appreciated in these patients. All the cases were dehydrated, 3 with grade III dehydration. Ten presented with headaches or irritability. Fifteen cases had profuse vomiting. One had frank hematemesis, melaena, epistaxis and bruising all over the body. Seventeen were semiconscious or

drowsy. Twelve were admitted with generalized convulsions, one of them developed complete aphasia, three remained deeply unconscious with generalized spasticity. Five, passing dark-brown urine, were severely anaemic. One was admitted in shock, with profuse watery diarrhoea and high grade fever, there was also a vague history of rnelaena and passing of dark coloured urine. One case presented with dysentery like symptoms lasting over a period of 4 months with mucus and blood in the stools, abdominal distension and fever (Table - II).

TABLE II

Symptoms	No of Patients	Percentage
Fever	43	100
Impaired Consciousness	17	39.5
Vomiting	15	34.8
Convulsions	12	27.9
Pallor	11	25.5
Headache/Irritability	10	23.2
Anorexia	8	18.6
Jaundice	6	13.9
Black urine	5	11.6
Abdominal pain	3	6.9
Diarrhoea	3	6.9
Bleeding	2	4.6

On physical examination 30 patients had enlarged firm spleens I to 5 cm below the costa! margin. Seventeen had enlarged livers, including 6 with clinical jaundice. Twenty nine were anaemic. Three cases had blurring of optic disc margins bilaterally, with fundal haemorrhages in one (Table - III).

TABLE – III

Signs	No.of Patients	Percentage
Dehydration	43	100
(a) Mild to Moderate	40	93.1
(b) Severe	3	6.9
Splenomegaly	30	69.7
Anaemia	29	67.4
Haemolytic	28	65.1
Aplastic	1	2.3
Hepatomegaly	17	39.5
Nuchal Rigidity	4	9.3
Generalized Spasticity	3	6.9
Papilloedema	3	6.9
Bleeding	2	4.6
Shock	1	2.3
Aphasia	1	2.3
Pulmonary Oedema	1	2.3

Laboratory data showed hemoglobin in the range of 4-11 g%. Twenty four cases had Hb below 8 g% and were given blood transfusions. M.C.V. was reduced in all cases with severe anaemia while other absolute values were normal. Total and differential leucocyte count was within the normal range. There was mild to moderate reduction in the platelet count with sixty percent having counts below 150,000.

Two had counts of less than 10,000/cumm, and presented with bleeding. Bone marrow aspiration was done in three cases. In one it was loaded with gametocytes of p. falciparum, and showed evidence of bone marrow suppression. Three cases had reduced G.6 P D. levels. Seven had mild to moderate rise of blood urea, ranging from 45mg to 76mg%. Liver function tests in six patients showed mild to moderate elevation of S.G.P.T. upto 80 units/ nil. In one case presenting with black water fever and G. 6 P.D. deficiency, the liver functions were grossly abnormal. In five cases urine was positive for haemoglobin. C.S.F. examination was completely normal in the 6 cases in whom. meningeal or intracranial pathology was suspected. Two children died. Both were brought unconscious to hospital. Cardiorespiratory arrest and hypostatic pneumonia respectively were the immediate causes of death.
The response to various antimalarials is shown in Table-IV.

TABLE – IV. Response of Falciparum Malaria to different Drugs.

Drugs used	Route of Adminis-tration	Cases with clinical response	Cases with no clinical response
* Amodiaquine	Oral	25	5
* Chloroquine	Oral	3	–
* Chloroquine	I.M.	1	–
with Steroids	I.M.	3	
* Fansidar	Oral	5	1
* Septran and Chloroquine + Pyrimethamine	Oral	3	–
	Oral	3	–

* One case showed no response to any of the above antimalarials.

Amodiaquine was used in 30 patients. Five did not show clinical response. Chioroquine was given to seven. One did not show improvement to intramuscular chloroquine.
Fansidar (chioroquine + pyrimethamine) was administered to six. One case showed clinical resis. tance. One child failed to respond to all the above drugs.

DISCUSSION

The forty three diagnosed cases of falciparum malaria seen over a period of fifteen months confirm our clinical impression that another resurgence of malaria has occurred. After the 1969 WHO global

programme for eradication of malaria was started in the early sixties, a resurgence was recorded in South East Asia, Indo Pak subcontinent and El-Salvador[1-5] in the mid seventies. The eradication programme was then modified to a more realistic and flexible strategy of malaria control with emphasis on collaboration with basic health services in urban and rural area[5]. Some success was achieved and the incidence dropped to the 1973 level in the early eighties. However, this appeared to be shortlived and another resurgence is being recorded now.

Currently the malaria control programme is depending on case finding and treatment along with selective spraying of endemic areas. Medical practitioners should therefore, be familiar with the common clinical features of malaria especially in children and maintain a high index of suspicion. Our study shows that falciparum malaria in children has varied symptomatology. However fever with anaemia, hepatosplenomegaly, and convulsions are highly suggestive. A positive blood smear confirms the diagnosis but unfortunately the laboratories are not always helpful in looking diligently for the scanty parasites present in the blood of semi immune populations of endemic areas.

The resistance of the malarial parasite to chioroquine has become widespread[1,2,3,5]. Our study also shows clinical resistance to amodiaquine and Fansidar (chloroquine + pyrimethamine). The situation is becoming rather alarming since the development of new drugs is not keeping pace with the development of parasite resistance.[5] The development of vaccine against malaria is the future hope of control of the disease. Intense research is continuing in this field and much of the preliminary work of identifying and purifying of antigens has been done. Three types of vaccines against p. falciparum are under investigation; an asexual erythrocytic vaccine, a sporozoite vaccine and a gamete reactive vaccine. The hope is that before long effective control of the disease would become possible.

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