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MALARIA; CAUSAL PARASITE AND CLINICAL FEATURES IN PEDIATRIC PATIENTS

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ABSTRACT

Objective: To find the common malarial parasite involved in causing malaria, and its clinical features.

Material and Methods: It was a descriptive Cross Sectional Study done at Pediatric Clinic at Bara, Khyber Agency, Pakistan; from July 2009 to January 2014. One hundred and nineteen patients were selected from patients who attended this clinic during the study period. Data was collected using pre-designed questionnaire. The information on child's age, gender, history of high grade fever, chills, vomiting, abdominal pain, and low grade fever was obtained. The examination included assessing the body weight, axillary's temperature, palmar and conjunctival pallor and spleen for splenomegaly. Heamoglobin of each patient was obtained and analyzed according to WHO criteria.

Results: Among 119 Malarial Parasite (MP) positive patients, the most common symptom was high grade fever (98.3%) followed by chills (9.2%), abdominal Pain (1.7%), vomiting (0.8%) and low grade fever (0.8%). On examination palmar and conjunctival pallor was present in 96.6% children and splenomegaly in 12.6% children. The frequencies of symptoms and signs did not vary significantly across the gender, $P>0.05$. Across Malaria parasite, only splenomegaly vary significantly, $P=0.001$.

Conclusion: Plasmodium vivax was the most common parasite, presented with high grade fever. Chills, with pallor and splenomegaly was common in Plasmodium falciparum Malaria.

Key Words: Malaria, Fever, Pallor, Splenomegaly.

INTRODUCTION

Malaria is a life threatening infectious disease and in severe cases it is associated with calamitous complications and far-reaching consequences within a community. It is estimated that annual global incidence of malaria is 300-500 million cases and each year about 1-3 million deaths occur worldwide[1]. In Pakistan, annual incidence of malaria is half a million cases and each year an estimated fifty thousand deaths occur mostly in infants, children and pregnant women. The disease is caused by infection with a parasitic unicellular organism of genus Plasmodium, that gets injected into the human bloodstream through a bite of female Anopheles mosquito[3]. Traditionally four species of Plasmodium including P. falciparum, P. vivax, P. ovale and P. malariae have known to be causing infections in humans[4]. However another species, P. knowlesi that causes malaria in macaques[5], have been reported to cause malaria in humans[6] and since 2004, increasing data is being published with regards to an increase in its incidence in various Southeast-Asian countries[7]. Plasmodium vivax is the most common human malaria parasite of the four species affecting humans. Currently, this infection is endemic in many countries of Asia, South Pacific, North Africa, Middle East and South and Central America[8]. In Pakistan, Plasmodium vivax is the major parasite and accounts for 70% cases, during peak transmission periods; whereas Plasmodium falciparum is responsible for remaining 30% malaria cases[9]. Though Malaria is endemic in this country, with the majority of cases caused by Plasmodium vivax; recently there has been an alarming shift to infection caused by Plasmodium falciparum, especially in the southern Punjab, Baluchistan and Sindh provinces[10-14].

The epidemiology of malaria varies considerably between countries and regions[15]. Many factors influence the pattern of patients' clinical manifestation such as endemicity of infection, geographical location, availability and accessibility to health care facilities, effectiveness of drugs, and age[16-18].

The most commonly described clinical presentation of P. vivax malaria is fever, headache, chills and sweating. However abdominal and osteomuscular pain, vomiting, diarrhea, hepatomegaly and splenomegaly have been reported[19,20]. Symptoms and clinical signs of the infection caused by P. vivax resemble infection caused by other species[15] and it is not possible to predict diagnosis of malaria or the infecting species involved,based on specific symptom[21].

There has been an increase in the number of reports of complications during P. vivax infection, including cerebral malaria and seizures[22,23], pulmonary

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edema[24-26], respiratory distress syndrome[27], kidney failure[28], and death[29].

In view of paucity of data from Federally Administrated Tribal Area (FATA), Khyber Agency, we have attempted to throw some light on the Clinical findings of patients suffering from malaria by clinically examining the patients and comparing them with the available literature worldwide.

MATERIAL AND METHODS

This descriptive cross-sectional study was conducted at a private clinic in Tehsil Bara Khyber Agency, FATA; Pakistan during the four and a half-year period from July 2009 to January 2014.

This is a pediatric clinic which receives patients from across the Bara and suburbs of Khyber Agency cases were only included when age of patients was less than 15 years and malaria was confirmed clinically, with further confirmation through a laboratory test. Children with age more than 15 years, children with history of antimalarial drug intake in last 2 weeks including artem, quinine, amodiaquine, chloroquine and Artemisinin based combination therapy (ACT) and those with Cerebral malaria were excluded from the study.

One hundred and nineteen patients were selected who attended this clinic during the study period. Data was collected using pre-designed questionnaire. All patients included in the study were diagnosed clinically and were confirmed by doing Giemsa stained thick blood smears. Hemoglobin was also obtained for each patient at time of enrollment and was analyzed using WHO criteria. The information on child's age, gender, history of high grade fever, chills, vomiting, abdominal pain, and low grade fever was obtained. Every patient was examined by consultant pediatrician and included assessment of the child's body weight, axillary's temperature, palmar and conjunctival pallor and spleen for splenomegaly. Informed verbal consent was obtained from the parents/guardian before data collection and examination of the patient.

All the data was analyzed using SPSS version 17.0. Non-parametric variables were analyzed using Chi-square test of independence ($\chi 2$), whereas parametric variable was analyzed using Student's T test. Results were considered statistically significant when p-value < 0.05, at 95% level of significance. Extensive literature search was done using PubMed database and Google Scholar, while references were cited using Endnote X1 library.

RESULTS

A total of 119 malaria parasite (MP) positive patients were included in the study of which 79 (52.9%) were males and 40 (47.1%) were females with male to female ratio of 1.97:1. The mean age of patients was 5.62 ± 3.54 years (range 0.2-15 years). The average body weight of children was 17.5±8.4 kg (range 6-58 kg). Out of total 119 cases, 109 (91.6%) cases were positive for P. vivax malaria and 10 (8.4%) cases for P. falciparum malaria.

Main symptoms observed during the clinical disease are shown in Table 1. In females, high grade fever (100% vs. 97.5%) and chills (12.5% vs. 7.6%) were more frequent as compared to males. Whereas spleenomegaly was more frequent in males (15% vs. 7.5%) as compared to females.

In P.falciparum infection, high grade fever (100% vs. 98.2%) and chills (30% vs 7.3%) were more frequent as compared to P.vivax infection ($P>0.05$). Similarly spleenomegaly (50% vs. 9.2%) and pallor (100% vs. 96%) were more common findings in P.falciparum vs. P.vivax infection. However only spleenomegaly vary significantly across the malarial specie, $P=0.001$.

Using WHO criteria, Anemia was defined as Hb<13g/dL in males and Hb<12g/dL in females.100% males and 97.5% females were anemic. Mean Hb in was 10.1 g/dL in males and 9.67g/dL in females. Howerer; Mean Hb did not vary significantly across gender, $P=0.07$, when analyzed using Student T test. Whereas, Mean Hb varied significantly across malaria specie, $P=0.04$, while using Student T test, where in P.falciparum it was lower, 9.2g/dL, as compared to P.Vivax,10.05g/dL.

Table 1: Frequencies of Clinical features and Anemia in Pediatric Malaria patients

Clinical Features & Anemia	Frequencies	%
High grade Fever Present Absent	 117 1	98.3%
Chill Present Absent	 11 108	9.2%
Vomiting Present Absent	 1 118	0.8%
Abdominal Pain Present Absent	 2 117	1.7%
Low grade fever Present Absent	 1 118	0.8%
Spleenomegaly Present Absent	 15 104	12.6%
Pallor Abnormal Normal	 115 4	96.6%
Hemoglobin(g/dL) Abnormal Normal	 118 1	99.1%

DISCUSSION

In Our study majority of malaria positive patients were suffering from P.vivax malaria (91.6%) and only 8.6% were suffering from P.falciparum malaria infection. This high incidence of P.vivax was in accordance with other studies, reporting that, in Pakistan the major parasites are Plasmodium vivax, which accounts for 70% cases followed by Plasmodium falciparum which accounts for 30% cases[9]. Similarly J Li et al reported that Plasmodium vivax is currently endemic in many countries of Asia, South Pacific, North Africa, Middle East and South and Central America[8]. However, Khan MA et al and other researchers reported that although majority of cases are caused by Plasmodium vivax, there has recently been an alarming shift to infection caused by Plasmodium falciparum, especially in the southern Punjab, Baluchistan and Sindh provinces[10-14].

The most common symptom was high grade fever (98.3%) followed by chills (9.2%), Abdominal Pain (1.7%), Vomiting (0.8%) and low grade fever (0.8%). Finding of hi grade fever was comparable to other studies where it was present in 91% patients[31,32] and in 97% patients[33].

On examination palmar and conjunctival pallor was present in 96.6% children and splenomegaly in 12.6% children. It was comparable to study done in Columbia where splenomegaly was present in 10% of malaria patients[31]. While in Brazil, splenomegaly was detected in 46% of the children[34] and in Surat-India 13% were having splenomegaly[35].

In this study, splenomegaly was present in 50% patients with P.falciparum malaria as compared to P.vivax (9.2%) with P=0.001. This finding is comparable to a study in Shoklo-Thailand, where 25% of P. falciparum 8% and of P. vivax infected children had splenomegaly[36]. Researcher from Karachi reported that splenomegaly were seen more frequently among patients with falciparum malaria compared to those infected with P. vivax (22.3% vs 12.2%)[37,38].

Anemia in malaria is due to the destruction of infected erythrocytes and to bone marrow suppression[37,38]. In our study 99.1% patients were anemic, with anemia being more common in males (100%), as compared to females (97.5%). Mean Hb in females (9.67 ± 1.38 g/dL) was lower than that in males (10.1 ± 1.17 g/dL) with p = 0.07. These results were in good agreement with studies from Columbia and Uganda[39,40], showing mean Hb in females was lower than that males in malaria patients. Also we found that Mean Hb in P.falciparum (9.2 ± 1.91 g/dL) was lower than that in P.vivax (10.05 ± 1.17g/dL) and varied significantly across malaria specie p = 0.04. Again this finding was in agreement with study from Karachi showing that low hemoglobin levels were common and were significantly lower with P. falciparum as compared to P.vivax[39,40].

CONCLUSION

The most common symptoms of malaria regardless of the species involved are high grade fever and chills; and most common signs are pallor and spleenomegaly. We conclude that anemia and spleenomegaly are more common in P.falciparum malaria than P.vivax malaria.

REFERENCES

1. Guinovart C, Navia MM, Tanner M, Alonso PL. Malaria: burden of disease. Current molecular medicine. 2006; 6(2): 137-40.
2. Yasinzai MI, Kakarsulemankhel JK. Incidence of human malaria infection in Barkhan and Kohlu, bordering areas of East Balochistan. Pakistan Journal Of Medical Sciences. 2008; 24(2): 306-10.
3. Schlagenhauf-Lawlor P. Travelers Malaria. PMPH-USA; 2008.
4. Davidson SS, Haslett C, Chilvers ER, Boon NA, Colledge NR. Davidson's principles and practice of medicine. Churchill Livingstone; 2002.
5. Collins WE. Plasmodium knowlesi: A Malaria Parasite of Monkeys and Humans*. Annual review of entomology. 2012; (57): 107-21.
6. Chin W, Contacos PG, Coatney GR, Kimball HR. A naturally acquired quotidian-type malaria in man transferable to monkeys. Science. 1965; 149: 865-70.
7. McCutchan TF, Piper RC, Makler MT. Use of malaria rapid diagnostic test to identify Plasmodium knowlesi infection. Emerging infectious diseases. 2008; 14(11): 1750-59.
8. Li J, Collins WE, Wirtz RA, Rathore D, Lal A, McCutchan TF. Geographic subdivision of the range of the malaria parasite Plasmodium vivax. Emerging infectious diseases. 2001; 7(1): 35-41.
9. Newman RD. World Malaria Report 2011.
10. Memon IA, Kanth N, Murtaza G. Chloroquine Resistant Malaria in Children. Journal-Pakistan Medical Association. 1998; 48: 98-99.
11. Khan MA, Smego Jr RA, Razi ST, Beg MA. Emerging drug--resistance and guidelines for treatment of malaria. Journal of the College of Physicians and Surgeons–Pakistan: JCPSP. 2004; 14(5): 319-24.
12. Bhalli MA. Falciparum Malaria-A Review of 120 cases. Journal-College of Physicians and Surgeons of Pakistan. 2001; 11: 300-03.
13. Rana MS, Tanveer A. Chloroquine resistance and Plasmodium falciparum in Punjab, Pakistan during 2000-2001. 2004. Southeast Asian J Trop Med Public Health. 2004; 35: 288-291.
14. Hozhabri S, Akhtar S, Rahbar MH, Luby SP. Prevalence of plasmodium slide positivity among the children treated for malaria, Jhangara, Sindh. Prevalence.J Pak Med Assoc. 2000; 50: 401-405.

15. Breman JG, Egan Aa, Keusch GT, Remme JHF, Binka F, Nabarro D. Toward a framework and indicators for monitoring Roll Back Malaria. Am J Trop Med Hyg. 2001; 64(suppl 1): 76-84.

16. Mockenhaupt FP, Ehrhardt S, Burkhardt J, Bosomtwe SY, Laryea S, Anemana SD, et al. Manifestation and outcome of severe malaria in children in northern Ghana. The American journal of tropical medicine and hygiene. 2004; 71(2): 167-72.

17. Snow RW, Bastos de Azevedo I, Lowe BS, Kabiru EW, Nevill CG, Mwankusye S, et al. Severe childhood malaria in two areas of markedly different falciparum transmission in East Africa. Acta tropica. 1994; 57(4): 289-300.

18. World Health O. Severe falciparum malaria. Transactions of the Royal Society of Tropical Medicine and Hygiene. 2000; 94(Supplement 1): 1-90.

19. Malik GM, Seidi O, El-Taher A, Mohammed AS. Clinical aspects of malaria in the Asir region, Saudi Arabia. Annals of Saudi medicine. 1998; 18: 15-17.

20. Isselbacher K, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL. Principios de Medicina Interna de Harrison. Mc Graw-Hill, Interamericana Madrid. 1994; 13: 1860-1.

21. Luxemburger C, Nosten F, Kyle DE, Kiricharoen L, Chongsuphajaisiddhi T, White NJ. Clinical features cannot predict a diagnosis of malaria or differentiate the infecting species in children living in an area of low transmission. Trans R Soc Trop Med Hyg. 1998 Jan-Feb; 92(1): 45-49.

22. Valecha N, Bagga A, Chandra J, Sharma D. Cerebral symptoms with P. vivax malaria. Indian Pediatr. 1992; 29: 1176-78.

23. Wattanagoon Y, Srivilairit S, Looareesuwan S, White NJ. Convulsions in childhood malaria. Transactions of the Royal Society of Tropical Medicine and Hygiene. 1994; 88(4): 426-28.

24. Curlin ME, Barat LM, Walsh DK, Granger DL. Noncardiogenic pulmonary edema during vivax malaria. Clinical infectious diseases. 1999; 28(5): 1166-67.

25. Munteis E, Mellibovsky L, Márquez MA, Mínguez S, Vázquez E, Díez A. Pulmonary involvement in a case of Plasmodium vivax malaria. CHEST Journal. 1997; 111(3): 834-35.

26. Pukrittayakamee S, Chantra A, Vanijanonta S, White NJ. Pulmonary oedema in vivax malaria. Transactions of the Royal Society of Tropical Medicine and Hygiene. 1998; 92(4): 421-22.

27. Carlini ME, White AC, Atmar RL. Vivax malaria complicated by adult respiratory distress syndrome. Clinical infectious diseases. 1999; 28(5): 1182-83.

28. Ahmad SH, Danish T, Faridi MMA, Ahmad AJ, Fakhir S, Khan AS. Renal function in acute malaria in children. Journal of tropical pediatrics. 1989; 35(6): 291-94.

29. Lobovska A, Rubik I, Holub M, Selinger P. The first death from tertian malaria in the Czech Republic]. Casopís lékařů českých. 1999; 138(2): 52.

30. World Health O. Indicators and Strategies for Iron Deficiency and Anemia Programmes; Report of the WHO/UNICEF/UNU Consultation; 6-10 December, 1993; Geneva,Switzerland: WHO. 1994.

31. Echeverri M, Tobón A, Álvarez G, Carmona J, Blair S. Clinical and laboratory findings of Plasmodium vivax malaria in Colombia, 2001. Revista do Instituto de Medicina Tropical de Sao Paulo. 2003; 45(1): 29-34.

32. Gonzalez LM, Guzmán Mn, Carmona J, Lopera T, Blair S. Caracteristicas clinico-epidemiologicas de 291 pacientes hospitalizados por malaria en Medellin (Colombia); Clinical and epidemiologic characteristics of 291 hospitalized patients for malaria in Medellin (Colombia). Acta méd. colomb. 2000; 25(4): 163-70.

33. Beg MA, Sani N, Mehraj V, Jafri W, Khan MA, Malik A, et al. Comparative features and outcomes of malaria at a tertiary care hospital in Karachi, Pakistan. International Journal of Infectious Diseases. 2008; 12(1): 37-42.

34. Ventura AMS, Pinto AYN, Silva RSU, Calvosa VSP, Silva Filho MG, Souza JM. Malaria por Plasmodium vivax em crianças e adolescentes: aspectos epidemiológicos, clínicos e laboratoriais. J Pediatr (Rio J). 1999; 75(3): 187-94.

35. Chandramohan D, Carneiro I, Kavishwar A, Brugha R, Desai V, Greenwood B. A clinical algorithm for the diagnosis of malaria: results of an evaluation in an area of low endemicity. Tropical medicine & international health. 2001; 6(7): 505-10.

36. Luxemburger C, Nosten Fo, Kyle DE, Kiricharoen L, Chongsuphajaisiddhi T, White NJ. Clinical features cannot predict a diagnosis of malaria or differentiate the infecting species in children living in an area of low transmission. Transactions of the Royal Society of Tropical Medicine and Hygiene. 1998; 92(1): 45-49.

37. Kurtzhals JAL, Rodrigues O, Addae M, Commey JOO, Nkrumah FK, Hviid L. Reversible suppression of bone marrow response to erythropoietin in Plasmodium falciparum malaria. British journal of haematology. 1997; 97(1): 169-74.

38. Berhman RE, Kliegman RM, Jenson HB. Nelson tratado de pediatria. McGraw-Hill Interamericana; 2001.

39. Bouyou-Akotet MK, Mboumba DPM, Kendjo E, Mbadinga F, Obiang-Bekale N, Mouidi P, et al. Anaemia and severe malarial anaemia burden in febrile Gabonese children: a nine-year health facility based survey. The Journal of Infection in Developing Countries. 7(12): 983-89.

40. Kiggundu VL, O'Meara WP, Musoke R, Nalugoda FK, Kigozi G, Baghendaghe E, et al. High Prevalence of Malaria Parasitemia and Anemia among Hospitalized Children in Rakai, Uganda. PloS one. 2013; 8(12): 840-55.