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# Tropical Splenomegaly Syndrome – The Index for Endemicity in Malaria

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**Abstract**

**Background:** *Malaria is one of the life-threatening diseases across the globe. It is the most common widespread infectious disease in the tropical areas like kurnool district of Andhra Pradesh.*

*Hyper-reactive malarial splenomegaly is an aberrant response to chronic malarial infection that is seen in areas of intense transmission of malaria.*

**Objectives:** *1.To study the clinical presentation and spleen, 2. liver profile changes in children and adults with complicated malaria.*

**Material and Methods***: The present study was carried out on 100cases of falciparum and vivax malaria in the DTR Hospital, Kurnool .All these patients were subjected to blood investigations like Hemoglobin level, total WBC count, random blood sugar, serum Bilirubin and viral screening test.*

*Results: A total 100 patients were diagnosed as positive for malaria by peripheral smear.Of 100cases,69 were p.falciparum,26 were p.vivax and 5 were mixed infection. Out of 100 patients,57 were in children and 43 in adults. Children to adult ratio is 1.3:1 Splenomegaly was leading sign in all forms. Anemia was seen in 79 cases,49from children and 30 from adults. Serum Bilirubin was raised in 93%cases,52%in children and 41% in adults. Liver dysfunction also was observed.Hepatitis B was seen in 14% adults 2%in children, where as Hepatitis C was seen in 8% adults:none in children.*

**Conclusion:** *Splenomegaly is the main clinical marker of endemicity in areas where falciparum transmission is high like kurnool. The spleen is the organ of defence in the human body against malaria. The study also focuses on laboratory findings in p.falciparum and p.vivax infected malaria .In this Endemic area two most reliable hematological parameters for predicting malaria in people were thrombocytopenia and leukopenia. Leucopenia was more predominanty observed in children than in adult population.*

**Keywords***: Malaria, Splenomegaly, hepatitis, anemia.*

## Introduction

Malaria is a major health problem in tropics. Malaria is one of the most common and widespread protozoal parasitic infection in the tropical countries. Malaria in humans is caused by plasmodium parasitic strains namely p. falciparum, P.vivax, P. malariae, P.ovale and P.knowlegi. Among them, Plasmodium falciparum accounts for more than 50% of the cases.

Malarial endemicity is influenced by factors related to the man-host interactions to the parasite, to the vector and to environment. Malarial incidence may fluctuate according to season. Anopheles mosquitoes breed in water and each species has its own breeding preference. Transmission is more intense in places where mosquito life span is longer. Environmental factors play an important role in vector distribution and malaria biodiversity. Climate conditions like rainfall patterns, temperature, humidity, presence of vegetation and available breeding places like stagnated water all are directly related to the malaria transmission cycle. In addition, human activities such as agriculture, irrigation, deforestation, urbanization, population movement, dam/road constructions are also connected to transmission levels and malarial epidemiology. Splenomegaly is one of the cardinal features of clinical malaria. In acute cases, it can be detected as early as 3 to 4 days after the infection and may persist even after the clearance of parasites. In acute malaria with high parasitaemia spontaneous rupture of spleen is a rare but disastrous compication. The spleen is important in removing malaria parasites from the blood. The spleen plays a vital role in policing the circulating RBC population, removing RBC's that are coated with antibody or have reduced deformability and extracting intra cytoplasmic particulate material such as nuclear remnants or oxidized haemoglobin. Splenic clearance is increased in malaria. Spontaneous rupture of spleen is common in vivax malaria and falciparum malaria. Rapid enlargement results in increased capsular tension and increased parenchymal friability. Marked splenomegaly can occur even in law grade parasitemia and it may persist for weeks or months after effective and complete treatment. Patients present with abdominal pain, fever, tachycardia and prostration. Rapidly developing anemia and hypotension are in favour of splenic rupture.

Chronic or repeated malarial infections produce hypergamma-globulinemia, Normochromic-normocytic anemia and splenomagaly. Malarial endemic areas in tropical area exhibit an abnormal immunologic response to repeated infections that is characterized by massive splenomegaly, hepatomegaly, marked elevations in serum titers of IgM and malaria antibody, hepatic sinusoidal lymphocytes is and peripheral B cell lymphocytosis.

This syndrome has been associated with the production of cytotoxic IgM antibodies to CD8+T lymphocytes, antibodies to CD5+T lymphocytes and an increase in the ratio of CD4+T cells to CD8+T cells. These events may lead to uninhibited B cell production of IgM and the formation of cryoglobulins. This immunologic process stimulates reticuloendothelial hyperplasia and clearance activity and produces eventually splenomegaly tropical splenomegaly syndrome (TSS) or Hyper reactive malaria splenomegaly (HMS) occurs where transmission of malaria is intense. It has been reported in this tropical area. It usually occurs in young and adults.

**Materials and Methods**

The present prospective study included 100 patients diagnosed as malaria by peripheral smear study admitted to DTR Hospital, Kurnool, Andhra Pradesh, India from April 2016 to March 2017.

Of the 100 cases, 69 were P. falciparum, 26 were P.vivax and 5 were mixed infection. Out of 100 patients 57 were children and 43 were adults. P.falciparum and P.vivax status were diagnosed by rapid diagnostic test.

All the subjects were examined clinically in detail and biochemical evaluation done. All patients were evaluated clinically for history of fever with chills and sweating, headache, abdominal pain, vomiting, pallor, splenomegaly, hepatomegaly, convulsions and coma. Both thick and thin smears were prepared and examined for malarial parasite.

Quantitative buffy coat study was done to confirm the presence of malarial parasite.

Blood investigations like Haemoglobin level, total WBC count, random blood sugar were done in all the cases of adults and children. The laboratory investigations for assessment of liver function included serum bilirubin, serum proteins, Hepatitis B and C infection were also tested to rule out concomitant viral hepatitis.

**Data Analysis:**

Analysis was done by using package for social sciences SPSS version 17. Data was represented in the form of frequencies and percentage with the help of tables and bar diagrams.

**Table 1: Age wise distribution**

| Children Number | Adults Number | Total Number |
|---|---|---|
| 57 (1-13year) | 43 (42-78 years) | 100 |

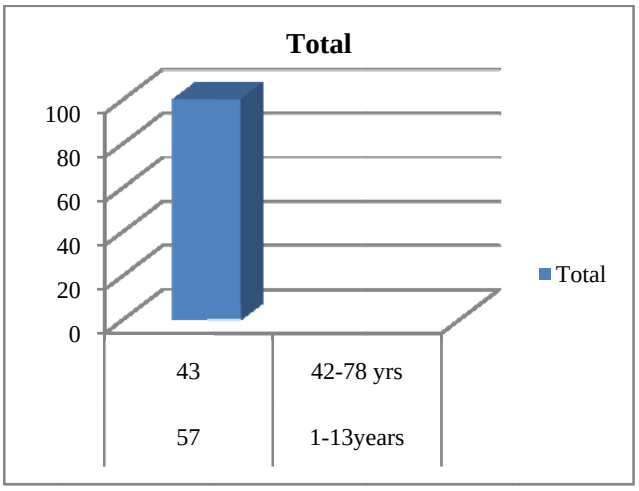


**Fig. 1: Agewise distribution**

**Table 2: Species wise distribution**

| P.falciparum Number | P.Vivax number | Mixed Number |
|---|---|---|
| 69 | 26 | 05 |

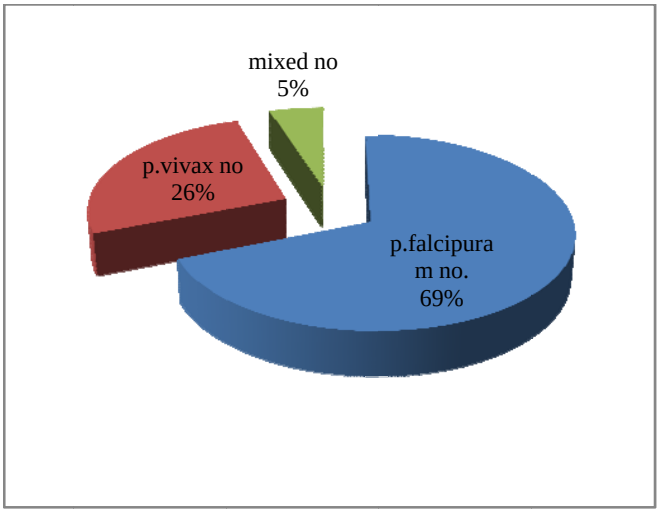


**Fig. 2: Species wise distribution**

**Table: 3 Clinical Profile of Children and Adults.**

| Sign | Children N=57 | | Adults N=43 | | Total N =100 | |
|---|---|---|---|---|---|---|
| | N | % | N | % | 100 | % |
| Fever | 57 | 100% | 43 | 100% | 100 | 100% |
| Chills | 51 | 89% | 38 | 88% | 89 | 89% |
| Nausea & Vomiting | 43 | 75% | 22 | 51% | 65 | 65% |
| Headache | 56 | 98% | 42 | 98% | 98 | 98% |
| Convulsion | 39 | 68% | 23 | 53% | 62 | 62% |
| Splenomegaly | 53 | 93% | 41 | 95% | 94 | 94% |
| Anemia | 49 | 86% | 30 | 70% | 79 | 79% |

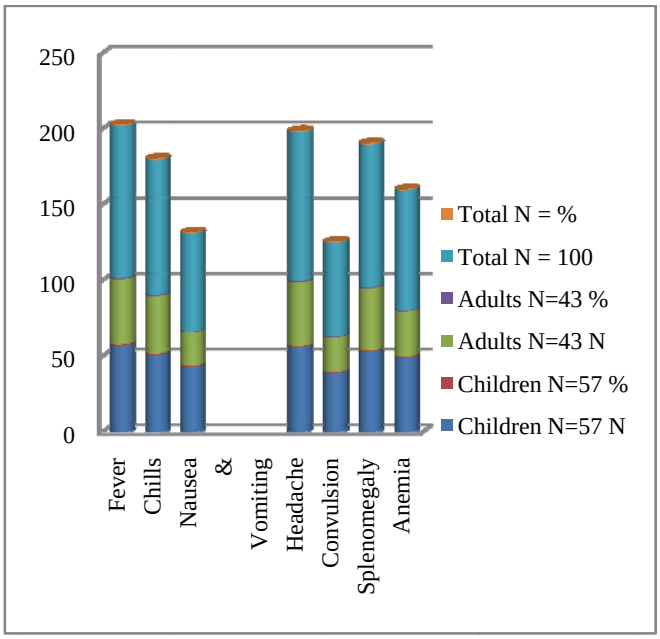


**Fig. 3: Clinical Profile of Children and Adults**

**Table 4: Laboratory findings in children and adults**

| Para meters | Children | | Adults | | Total | |
|---|---|---|---|---|---|---|
| | N=57 | Per | N=43 | Per | N=100 | Per |
| Low Hg | 47 | 82% | 29 | 67% | 76 | 76% |
| Low RBC Count | 39 | 68% | 29 | 67% | 68 | 68% |
| Low Wbc Count | 18 | 32% | 16 | 37% | 34 | 34% |
| Thrombo cyto penia | 33 | 58% | 29 | 43% | 62 | 62% |
| Low blood sugar | 35 | 61% | 32 | 74% | 67 | 67% |
| Serum bilirubin | 52 | 91% | 41 | 95% | 93 | 93% |
| Hepatitis B | 02 | 0.04% | 14 | 33% | 16 | 16 |
| Hepatitis C | - | - | 08 | 19% | 08 | 8% |

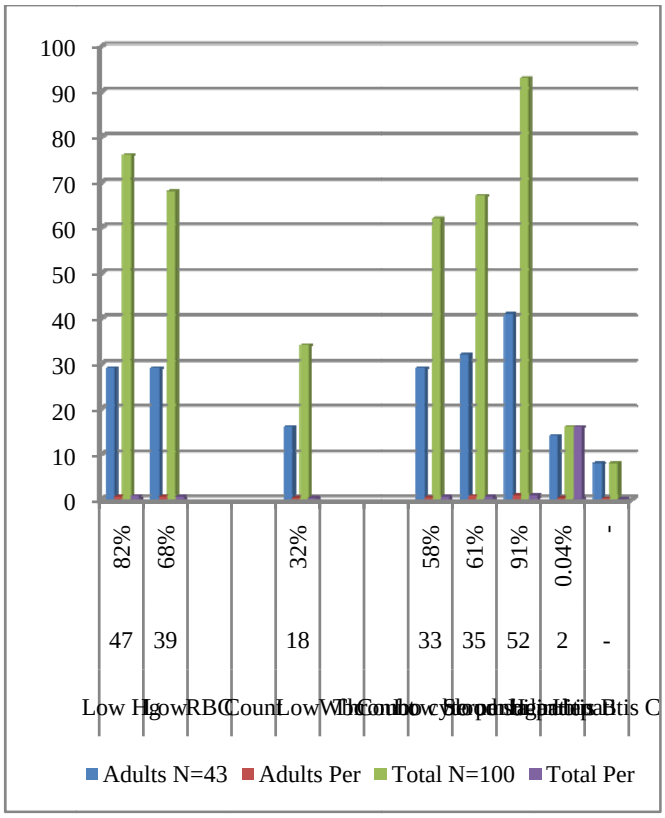


**Fig. 4: Laboratory findings inchildren and adults**

## Results

In the present study 100 cases were enrolled. There were 57 children and 43 adults. The ratio between children to adult is 1.3:1. The age group of children 1-13 years and adults were in the age group of 42-78years (Table:1) in the present study splenomegaly was leading sign in all forms of the 100 patients, 69 were Plasmodium falciparum 26 were Plasmodium vivax while 5 showed mixed infection (Table:2)

Fever was the predominant complaint in all the patients 100%, other main symptoms were chills. Headache, nausea, vomiting particularly in P.falciparum and mixed infections. Chills were seen in 51 cases (89%) of children and 38 (88%) cases of adults. Headache was seen in 98% of the total patients. Nausea & vomiting were seen in 43(75%) and 22(51%) of children and adults respectively, convulsions were seen in 39(68%) of children and 23(53%) of adults (Table:3).

Among 100 patients of cerebral malaria 79% had severe anemia, of these 49(86%) children and 30 (70%) adults respectively (Table :3) splenomegaly was seen in 94% of patients, it was found more frequently in P.vivax and mixed

infection (100%) than P.falciparum (91%) splenomegaly was present in 53 (93%) cases of children and 41(95%) of adults cases (Table:3). Hepatomegaly was also seen in 61% of patients. Icterus and high serum bilirubin was found were in P.falciparum and mixed infections as compared to P.vivax infections. Serum Bilirubin was increased in 93% of total cases, where as in children 52(91%) and 41(95%) of adults.

Hemoglobin of lessthan 9.2gms/d1 was seen in 76 cases of total, in children 47(82%) where as in adults 29 (67%) RBC level was decreased ($3.2x10^6$ml) in 39 (68%) children and 29(67%) in adults. WBC count was low in 18(32%) of children and in 16 (37%) adults. Thrombocytopenia was seen (<1,16,000ml) in 33(58%) children and in 29(43%) adults and in 62 cases of total (Table:4) Low Blood sugar (randomly) was seen 35(61%) in children and 32 (74%) in adults. Liver dysfunction was seen in the patients. Hepatitis-B was observed in 2 (0.04) children only where as in 14(33%) adults. Hepatitis was seen only in 8(19%) of adults (Table:4).

**Discussion**

Splenomegaly is the main clinical marker of the endemicity in P.falciparum and P.vivax transmission in known endemic areas like Kurnool. Splenomegaly was recorded in 53 cases of children and 41 cases of adults and 94% of total population. It has appeared after treatment of malaria.

The reason for the splenomagaly is due to the destruction of the infected RBCs. Malarial parasites sequester in the pulp capillaries to avoid splenic clearance and active cytoadherence of infected reticulocytes to the blood barriers of fibroblastic origin. The spleen plays a pivotal role in policing the circulating RBC population. These sequestered red cells exchange slowly with the circulating red cells. The number of red cells that may be trapped varies greatly not only with the size of the spleen, but also with the underlying disease process and may exceed red cell mass in massively enlarged spleens. Pain due to splenic infarction was noticed. Liver is also enlarged.

Jaundice is a common clinical presentation in severe malaria in patients with falciparum infection. Jaundice in falciparum malaria has been more often associated with haemolysis than hepatocellular damage. Our data indicates that it is not an uncommon entity and in endemic area it is seen in patients with falciparum malaria. High serum bilirubin was found in 93% of total cases, in children 52 (91%) and in 41 (95%) of adults. The causes of hyperbilirubinema in malaria included intravascular haemolysis from parasitized and non-parasitized red blood cells and also hepatic dysfunction during acute malaria illness. In this study raised bilirubin was mainly due to hepatic dysfunction and hemolysis. The incidence of hepatitis B in patients with falciparum malaria, only 14(33%) adult cases were noted and only 02(0.04%) children had evidence of malaria hepatitis. In adults, the reported incidence of hepatitis C was only 08(19%) but no cases was seen in children.

According to this study, a significant reduction of Hb level below 9.2g/dl. is reported. Reduced haemoglobin in malaria may be attributed to the increase of breakdown red blood cells by the parasites. It was observed that anemia, is common amongst the patients infected by P.falciparum in this endemic area. RBC's counts and WBC count level in patients infected with malaria were decreased in children and adults. The decrease in lymphocytes counts associated with malaria may be due to reflecting redistribution of lymphocytes with sequestration in the spleen. Low platelet count is a finding of malarial infection and thrombocytopenia is common in both children and adults. In present study, platelet count in malaria shows that thrombocytopenia is directly to the severity of splenomegaly.

Hypoglycemia was observed in children and adult patients. 61% of children and 74% of adults population have developed hypoglycemia result was shown in Table:4. In case of severe falciparum infection, increased consumption of glucose by the host and growing patients and failure of hepatic gluconeogenesis and glycogenolysis as a result of impaired liver function.

**Conclusion**

The present study suggests that hepatomegaly, like splenomegaly may be assessed as a possible malariometric index of the intensity of transmission in children & adults in an endemic area.

Malaria is a potential cause of morbidity and mortality in the tropical countries. Jaundice is one of the common presentation of falciparum malaria. The raised serumbilirubin could be diagnosed early and treatment will help in reducing further complications.

Thrombocytopenia and hepatic dysfunction are commonly seen and are early indicators for the severity of the disease which can open a new door in therapeutic development to cure this disease.

This information in turn should help in the design of noval approaches in malaria vaccine development.

**Acknowledgement**

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**References**

1) Guerra CA, Gikandi PW, Tatem AJ, Noor AM, Smith DJ., Hay SI, Snow RW. The limits and intensity of Plasmodium falciparum transmission; implications for malaria control and elimination world wide. PLOS Medicine 2008; 5(2): e38 http://dx.doi.org/10.1371/ . Journal Dmed.0050038 [PubMed].
2) Sinke ME, Bangi MJ, Manguins, Chareonvirinapha PT, Patil AP. Temperely WH, Gething PW, Elyazar IR, Kabaria CW, Harbach RE, Hay SI. The dominant Anopheles vectors of human malaria in the Asia- Pacific region; occurrence data distribution maps and bionomic précis. Parasite vectors. 2011;4:89. http://dx.doi.org/10, 1186/1756-3305-4-89.[PMC free article] [PubMed].

3) Machault V, Vignolles C, Borchi F, Vounatrou P, pages F, Briolant S, Lacaux JP, Rogier C. The use of remotely sensed environmental data in the study of malaria. Geospatial Health. 2011;5 (2): 151-168 [PubMed].
4) Kesinee chotirenich, Nicholas J.White, central Role of the spleen in malaria parasite clearance,.J. infect. Dis.2004, 186(6) 1052-1055.
5) Crane GG. Tropical splenomegaly, part 2: Oceania. Clin Haemetol 1981:10:976-982
6) Murthy GJ, Sahey RK, Sreenivas DV, Sundaram C, Shantaram V. Hepatitis in falciparum malaria. Trop Gastroenterol 1998:19:152-4.
7) Bryceson A, Fakunle YM, Fleming AF, et al Malaria and splenomegaly [letter]. Trans R Soc Trop Med Hyg 1983; 77:879.
8) Choudhary J.Uttam K.G, and Mukhopadhyey.M (2008) spontaneous rupture of malarial spleen. Indian pediatr 45:327-328.
9) Imbert P.Rapp. C…., and Buffet P.A. (2009) Pathological rupture of the spleen in malaria, analysis of 55 cases (1958-2008). Travel med infect Dis. 7:147-159.
10) Moulin F, Lesage F, Legros AH, maroge C, et al Thrombocytopenia and Plasmodium falciparum malaria in children with different exposures. Arch Dis Child. 2003:88:540-541: doi:10-1136/adc 88.6.546 PubMed.