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Original Research Article

An analysis of clinical presentation and laboratory profile of scrub typhus among pediatric population in a semi urban centre

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ABSTRACT

Background: Scrub typhus is a re-emerging acute infectious disease caused by Orientia (Rickettsia) tsutsugamushi, in India and globally. Scrub typhus goes undiagnosed at early stages of the illness because many factors like of low index of suspicion, nonspecific signs and symptoms. This study was done to analyse the various clinical demographic factors and their significance in making the clinical diagnosis of scrub typhus and also the analysis of other non-specific laboratory parameters present in the serologically confirmed cases of scrub typhus. The objective of this study was to study the clinical presentation and laboratory profile of scrub typhus in pediatric patients admitted in a tertiary care hospital.
Methods: It was prospective and descriptive study conducted in the pediatric ward of a Medical College Hospital. The study population consisted of 50 children aged 5-15 years having fever of more than 5 days duration. Children with persistent fever with known focus and etiology were excluded from the study.
Results: Among the study group of 50 children, majority were between 5 and 15 years age group. Male children constituted 66% (33 out of 50). Out of 50 children, 94% (47 children) had Eschar. 82% of children did not have any history of tick exposure. Less than 25% of children had Vomiting similar to conjunctival congestion. 94% children had lymphadenopathy and 98% children had mild anemia. 54% children had maculopapular rash and 20% had jaundice. 62% children had hepatomegaly and 96% had splenomegaly. Pedal edema was seen in 22% of cases and anasarca in 2% of cases 76% of children had normal WBC counts, 24% had leucocytosis. Platelet counts of 38,000 to 2,87,000 was noted in the study group and 26% of patients had thrombocytopenia. 98% children had hemoglobin less than 10 gms%. C reactive protein was positive in 96% of cases. Hypoalbuminemia was noted in 48% of children. 60% children were positive for urine albumin.
Conclusions: Scrub typhus should be considered in early part of the illness and patient has to be examined for the presence of Eschar. A clinical diagnosis can be made based on the associated rash, splenomegaly, lymphadenopathy etc. and specific treatment with antimicrobials can be initiated for scrub typhus.

Keywords: Children, Clinical and laboratory profile, Scrub typhus

INTRODUCTION

Scrub typhus is the commonest and most widespread zoonotic disease among the diseases caused by Rickettsial organisms both in India and globally. Scrub typhus is an acute, febrile, infectious illness that is caused by Orientia (Rickettsia) tsutsugamushi.[1] It was first described from Japan where it was found to be transmitted by mites.[2] Rickettsiae comprise a group of microorganisms that phylogenetically occupy a position between bacteria and viruses. Orientia (Rickettsia) tsutsugamushi is an obligate intracellular gram-negative coccobacillary forms that multiply within eukaryotic cells

and are transmitted by the bite of infected larvae of trombiculid mite (Chiggers). In humans, they infect vascular endothelium and reticuloendothelial cells. Rickettsial diseases widely vary in severity from self-limited mild illnesses to fulminating life-threatening infections.[3]

Scrub typhus is present in most of the South-East asian countries like India, Indonesia, Maldives, Myanmar, Nepal, Sri Lanka and Thailand. Scrub typhus is prevalent in many parts of India but specific data are not available. There have been outbreaks in areas located in the sub-Himalayan belt, from Jammu to Nagaland. There were reports of scrub typhus outbreaks in Himachal Pradesh, Sikkim and Darjeeling (West Bengal) during 2003-2004 and 2007. Scrub typhus is considered a reemerging infectious disease in India.[4]

The illness often presents with fever without any identifiable focus of infection and has a negative blood or urine culture. Hemogram often shows a normal or increased WBCs with or without thrombocytopenia. C-Reactive protein is often elevated and few studies have shown hypoalbuminemia and elevated liver enzymes being associated with scrub typhus. Many studies have shown the Eschar a pathognomonic sign of scrub typhus is seen in around 30 to 40% of cases. Many times, scrub typhus goes undiagnosed at early stages of illness because of low index of suspicion, nonspecific signs and symptoms, and lack of widely available sensitive and specific diagnostic tests. Failure of timely diagnosis may lead to significant morbidity and mortality. With timely diagnosis, treatment is easier and often successful with dramatic response to antimicrobials.

High index of suspicion, in all persistent fever cases especially those coming from endemic areas, and a thorough examination for the eschar and presence of other common signs of scrub typhus and if feasible confirmation of the illness with specific serological tests is important in the early stage of the disease to prevent significant morbidity and mortality.

METHODS

A Prospective and descriptive study was conducted at Pediatrics department, of a tertiary care centre from Jun 2013 to May 2014. The study was approved by Institutional ethical committee. Informed consent was obtained from the subject's parents or guardians.

Study population consisted of 50 children between 6 months to 15 years of age with fever of more than 5 days duration with or without eschar and other supportive clinical features suggestive of scrub typhus. Children more than 16 year of age and infants less than 6 months and those having signs of other specific infections were excluded from the study.

The details of the patient were recorded in a pre-designed proforma with details of name, age, sex, address, duration of fever, other relevant history, clinical examination of all systems and positive findings. Details regarding History of tick exposure, vomiting, eschar, rash, conjucntival congestion, anemia, lymphadenopathy, hepatomegaly, splenomegaly, pedal edema, anasarca, and laboratory investigations like complete blood count, C-reactive protein, urine albumin, serum albumin were recorded. A detailed history was obtained including age, sex, socioeconomic status, duration of fever and other parameters as mentioned above. Complete physical examination of the child was performed with weight, height, head circumference and mid arm circumference to emphasis that there is no evidence of malnutrition.

Statistical analysis of data was done using Pearson chi square test using SPSS software version 15.36.

RESULTS

Out 50 children enrolled in this study, majority were between 5 and 15 years age group. Male children constituted 66% (33 out of 50).

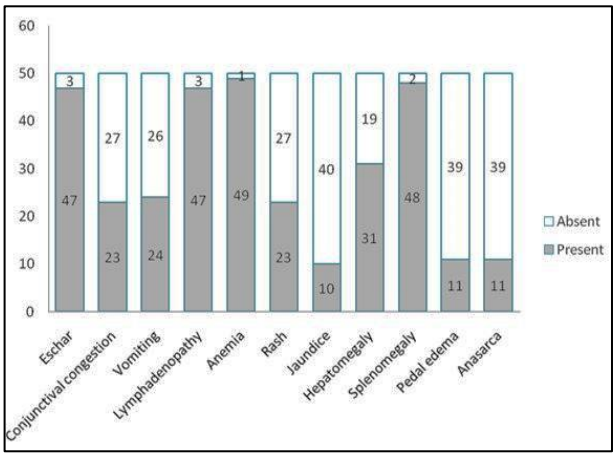

Figure 1: Clinical demography - distribution.

Out of 50 children, 94% (47 children) had Eschar. 82% of children did not have any history of tick exposure. Less than 25% of children had vomiting similar to conjunctival congestion. 94% children had lymphadenopathy and 98% children had mild anemia. 54% children had maculopapular rash and 20% had jaundice. 62% children had hepatomegaly and 96% had splenomegaly. Pedal edema was seen in 22% of cases and anasarca in 2% of cases 76% of children had normal WBC counts, 24% had leucocytosis. Platelet count value from 38,000 to 2,87,000 was noted in our study group and 26% of patients had thrombocytopenia. 98% children had hemoglobin less than 10 gms%. C reactive protein was positive in 96% of cases. Hypoalbuminemia was noted in 48% of children. 60% children were positive for urine albumin.

Eschar was compared with other clinical parameters.

Table 1: Eschar with clinical demography.

Variable	Status	Eschar +ve	Eschar -ve	P value
Conjunctival congestion	Yes	18	5	0.225
	No	25	2	
Lymphadenopathy	Yes	45	2	0.050
	No	2	1	
Anemia	Yes	47	2	0.173
	No	0	1	
Rash	Yes	23	4	1.000
	No	20	3	
Anasarca	Yes	10	1	1.000
	No	33	6	

Eschar was present in 18 patients with conjuctival congestion and absent in 5 of them with conjuctival congestion (Table 1). Conjuctival congestion was not present in 2 patients who also had no Eschar. 25 patients with Eschar had conjuctival congestion. There was no significant p-value.

Lymphadenopathy was seen in 45 patients with Eschar and present in 2 without Eschar. Lymphadenopathy was absent in 2 with Eschar and 1 patient had no eschar and lympadenopathy. The comparison had a significant p value (0.050). Anemia was present in 47 with Eschar and 2 without Eschar. No anemia and Eschar was seen in 1 patient.

Table 2: Eschar with clinical demography.

Clinical variables	Status	Eschar +ve	Eschar -ve	P Value
Rash	Yes	23	4	0.857
	No	20	3	
Hepatomegaly	Yes	28	3	0.281
	No	15	4	
Splenomegaly	Yes	46	2	0.001
	No	1	1	
Jaundice	Yes	8	2	0.541
	No	35	5	

Eschar was seen in 23 patients with rash and 20 without rash. 4 patients had Eschar but had rash. 3 patients had no Eschar and no rash. There was no significant p value. Anasaraca was seen in 10 patients with Eschar and 1 patient who had no Eschar had anasarca. 33 patient who had no anasaraca had Eschar and 6 did not have both. The comparison did not have a significant p value.

Rash was present in 23 patients with Eschar and absent in 20 patients. Eschar was present 15 patients without rash. 3 patients had no rash and Eschar. Hepatomegaly was present in 28 with Eschar and 3 without Eschar. 15 with Eschar had no hepatomegaly. 4 patients did not have both. There was no significant P value (Table 2).

Comparison between splenomegaly and Eschar

Splenomegaly was seen in 46 patients with Eschar and 2 people without Eschar. Splenomegaly was absent in 1 person with Eschar and both were absent in one person. The P value was significant.

Comparison between lymphadenopathy and Eschar

Lymphadenopathy was seen in 45 patients with eschar and was present in 2 patients without eschar. Lymphadenopathy was absent in 2 patients with eschar and one patient had no eschar and lymphadenopathy. The comparison had a significant P value.

Table 3: Eschar versus laboratory profile.

Variable	Status	Eschar +ve	Eschar -ve	P value
WBC status	Normal	28	4	0.684
	Abnormal	15	3	
Platelet status	Normal	11	2	0.867
	Abnormal	32	5	
C-reactive protein	Normal	45	3	0.715
	Abnormal	0	1	
Urine albumin	Present	26	4	0.868
	absent	17	3	
Serum albumin	Normal	21	4	0.684
	Abnormal	22	3	
Hypona-tremia	Present	16	4	0.318
	absent	27	3	

DISCUSSION

Present study showed a male predominace of 66% out of the total enrolled. Mahajan SK et al study in himalyas region showed a male predominance of 60%.[5] We had 62% of patients with hepatomegaly and 96% with splenomegaly. Mahajan SK et al. Study had 40% of patients with each hepatomegaly and splenomegaly. 76.19% patients presented with hepatomegaly and 76.19% with splenomegaly in study done by Digra SK.[6]

Rathi NB et al study showed 97% patients each with hepatomegaly and splenomegaly.[7] Bhat NK et al study showed 93% with hepatomegaly and 87% with splenomegaly. 95% patients had hepatomegaly and 87% had splenomegaly in Sankhyan N study.[8,9]

Considering international studies Silva ND 95% of patients had hepatomegaly and 35% had splenomegaly Huang CT study showed 35.7% patients with hepatomegaly and 17.9% with splenomegaly.[10,11]

We compared the WBC status of our study with Rathi N study.[7] Our study had a minimum WBC count of

4,000/mm3 compared to Rathi N, 1,800/mm3. The maximum WBC count in our study was 24,000/mm3 comparing to Rathi N study of 38,000/mm3. The mean for us was 10,706/mm3 against their studies mean of 9,800/mm3.[12]

The minimum platelet value in or study was 38,000 cubic mm and in Rathi N was 12,000 and maximum in or study was 2,87,000 cubic mm compared to their studies 5,99,000 cubic mm.[12] The mean values where, 1,14,000 cubic mm in our study and 1,03,000 cubic mm in Rathi N study.

The hemoglobin value in our study was a minimum of 8 gm/dl to a maximum of 10 gm/dl. In Rathi N study the minimum hemoglobin value was 4.2 gm/dl to maximum of 12.2 gm/dl.[7] The mean in our study was 9.13 gm/dl where as in their study was 8.8 gm/dl.

The minimum C-reactive protein value in our study was Omg/dl to a maximum of 192 mg/dl. In Rathi N study the minimum C-reactive protein value was 6 mg/dl to a maximum of 169 mg/dl.[7] The mean value in our study was 45.92 mg/dl and in Rathi N study was 55 mg/dl.

In our study there a 60% cases with albuminuria, 48% with hypo albuminemia and 40% low serum sodium levels. Rathi N et al study showed 40% with albuminuria, 51% with hypo-albuminemia and 64% low serum sodium levels.[7] Sankhyan N study showed 100% with albuminuria, 100% with hypo-albuminemia and 53% low serum sodium levels.[9] Other studies did not document any.

Limitations

Limitation of study duration was limited over a period of one year and less number of patients were studied as compared to some of the other studies. Due to resource limitations, another laboratorial parameter like transaminase could not be studied.

CONCLUSION

A significant co-relation of Eschar with splenomegaly and lymphadenopathy.

Out of 50 patients with positive ELISA IgM, 47 children (94%) had eschar. Hence absence of Eschar does not rule out scrub typhus. A combination of prolonged fever without any other focus of infection, from endemic area with splenomegaly, lymphadenopathy should arise suspicion of scrub typhus in the clinician's mind.

Presence of Eschar had a positive correlation with splenomegaly and lymphadenopathy in IgM ELISA positive cases. Hence a conglomerate of these symptoms should evoke suspicion of scrub typhus by the treating physician.

There was no significant correlation between IgM ELISA positive scrub typhus cases with clinical features like hepatomegaly, edema and rash. Hence, this could not conclude a strong clinical sign from our study. However, in other Indian and international studies these clinical signs were useful. This discrepancy could be due to low number of patients studied compared to these studies.

Though a major number of cases are from rural areas (42 out of 50, 84%) there is no positive correlation between area of residence and eschar as a presentation. The cause of this lack of association could not be ascertained but the percentage of patients from rural areas matched with other studies. Scrub typhus is now emerging in semi urban areas and no more a rural disease.

From the correlation one can conclude that a strong suspicion of scrub typhus can be made in those patients who have prolonged fever, Eschar with splenomegaly and lymphadenopathy. Absence of eschar in these eases does not rule out scrub typhus. In the absence of eschar, they can even be treated empirically if specific laboratory tests are not available and the patient is coming from the endemic area.

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