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A CASE STUDY ON RICKETTSIAL FEVER

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ABSTRACT

Introduction: Rickettsial fever is a significant yet frequently underdiagnosed cause of acute febrile illness in endemic regions of India. Nonspecific clinical features and limited diagnostic facilities often delay diagnosis and increase the risk of complications. **Case:** A 36-year-old male presented with fever, chills, generalized body aches and burning micturition for one week. Clinical examination revealed tachycardia and suprapubic tenderness. Laboratory investigations showed anaemia, leucocytosis, thrombocytopenia, hypoalbuminemia, and mildly elevated liver enzymes. Serological testing was positive for Weil-Felix (OX19 and OX2), while dengue and blood cultures were negative. Based on clinical and laboratory findings, rickettsial fever was diagnosed. The patient was treated with doxycycline and supportive therapy, leading to marked clinical improvement. **Discussion:** The nonspecific presentation of rickettsial fever often mimics other febrile illnesses, making early diagnosis challenging. Haematological abnormalities and positive serology were crucial in confirming the diagnosis in this case. Prompt initiation of doxycycline resulted in favourable outcomes. **Conclusion:** Early recognition and timely treatment of rickettsial fever are essential to reduce morbidity. Increased clinical awareness in endemic areas can improve diagnosis and patient outcomes.

KEYWORDS: Rickettsial fever; Weil-Felix test; Doxycycline; Acute febrile illness.

INTRODUCTION

DEFINITION

Rickettsial fever is a febrile illness caused by bacteria in the Rickettsia genus. It's spread by arthropods like ticks, fleas, and mites. Rickettsiae are small gram-negative bacteria with obligate hematophagous arthropod vectors.[1]

ETIOLOGY

Diseases	Rickettsial agent	Insect vectors	Mammalian reservoirs
Typhus group			
a. Epidemic typhus	*R.prowazekki*	Louse	Humans
b. Murine typhus	*R.typhi*	Flea	Rodents
c. Scrub typhus	*O.tsutsugamushi*	Mite	Rodents
Spotted fever group			
a. Indian tick typhus	*R.conorii*	Tick	Rodents, dogs
b. Rocky Mountain spotted fever	*R.rickettsii*	Tick	Rodents, dogs
c. Rickettsial pox	*R.akari*	Mite	Mice
Others			
a. Q fever	*C.brunetti*	Nil	Cattle, sheep, goats
b. Trench fever	*Rochalimaea Quintana*	Louse	Humans

Fig 1: Classification of the Rickettsial diseases.[2]

Among the major groups of rickettsioses, commonly reported diseases in India are scrub typhus, murine flea-borne typhus, Indian Tick Typhus and Q fever.

Scrub typhus is the commonest occurring rickettsial infection in India. The infection is transmitted through the larval mites or "Chiggers" belonging to the family Trombiculidae. Only the larval stages take blood meal. A number of small rodents particularly wild rats of subgenus Rattus are natural hosts for scrub typhus. The field rodent and vector mites act as reservoir and between the two the infection perpetuates in nature. The vector mite is known to be present in diverse ecological niches such as equatorial rain forests, semi deserts and Alpine subarctic terrains in the Himalayan regions. Endemic foci are usually associated with specific habitats such as abandoned plantations, gardens or rice fields, overgrown forest clearings, shrubby fringes of fields and forests, river banks and grassy fields. These ecological patches which attract the natural host of mite vectors are called "mite islands".

Scrub typhus can occur in areas where scrub vegetation- consisting of low-lying trees and bushes is encountered, and also in habitats as diverse as banks of rivers, rice fields, poorly maintained kitchen gardens, grassy lawns which can all be inhabited by chiggers. The chiggers too small to be seen by the naked eye, feed usually on rodents and accidentally on humans, and transmit the infection during the prolonged feeding which can last 1-3 days. Incidence of scrub typhus is higher among rural population.

Cases are more likely to have exposure to rodents at home or at work and to occupational (farming) or recreational activities which expose them to the risk of encountering chiggers sitting in grass blades, bushes, shrubs. The disease is seasonal in many parts of India, which correlates with the appearance and activity of mites.[2]

CLINICAL MANIFESTATION:

Rickettsial infections are generally incapacitating and difficult to diagnose; untreated cases have case fatality rates as high as 30-45% with multiple organ dysfunctions, if not promptly diagnosed and appropriately treated. The vast variability and non-specific presentation of this infection have often made it difficult to diagnose clinically.

Given below are some of the presenting symptoms and signs of rickettsial infection:

- Acute fever is the most common symptom associated with breathlessness, cough, nausea, vomiting, myalgia, and headache.
- An Eschar (Dead tissue that forms over healthy skin as a scab or crust) that is found on the neck, axillae, chest, abdomen, and groin.[2]

Fig 2: Eschar at neck region.[2]

Fig 3: Eschar in axilla.[2]

Fig 4: Eschar with the typical central black scab suggesting a cigarette burn on the skin.[2]

DIAGNOSTIC TESTS

- Weil Felix.
- IgM and IgG ELISA,
- Polymerase Chain Reaction.
- Immunofluorescent Assay.
- Indirect Immunoperoxidase Assay.
- Hematology- Commonly observed WBC, RBC, Platelets.
- Biochemistry- Commonly observed Raised Transaminase levels.
- Serological Test.[2]

PATHOPHYSIOLOGY

The pathogenic sequence of events that occur in rickettsial infection begins with the entry of organisms inoculated by the feeding tick or mite or scratched into the skin from infected louse or flea feces deposited on the skin. The initial target cells of infection are CD68+ cells (macrophages and/or dendritic cells).

The rickettsiae then spread via lymphatic vessels to the regional lymph nodes as has been observed vividly in the lymphangitis associated with R. sibirica mongolitimoniae infection. Rickettsiae then spread hematogenously throughout the body and infect mainly endothelial cells, but also to a lesser extent, macrophages, in the skin, lungs, brain, liver, gastrointestinal tract, kidneys, heart, and other organs.

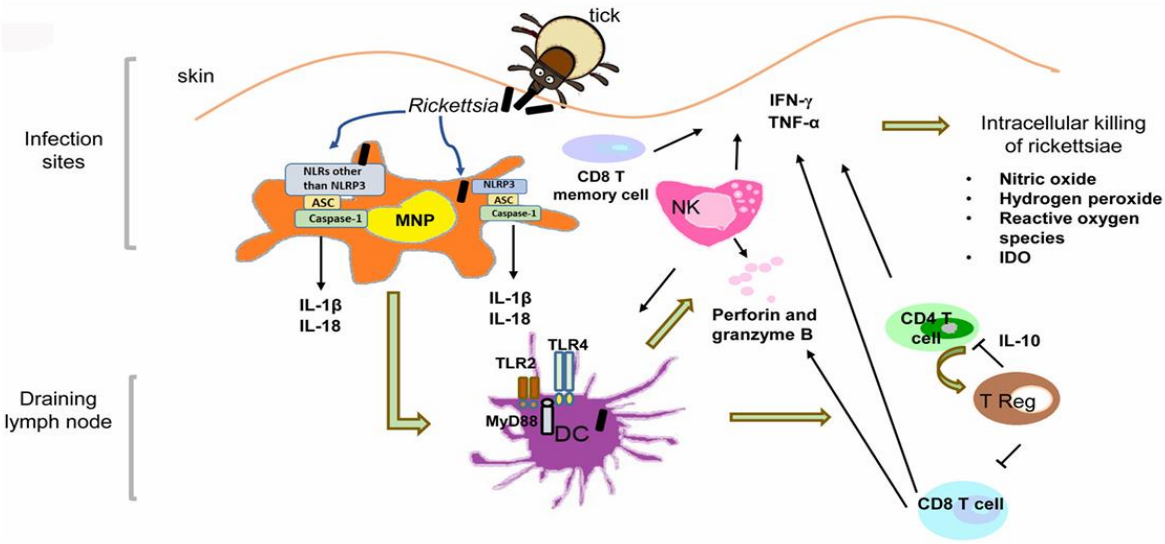

Fig 5: A schematic diagram of interactions of spotted fever group rickettsiae with different components of the host immune system. After tick bite, the inoculated rickettsiae initially target mononuclear phagocytes (MNPs) and then activate dendritic cells (DCs). Rickettsial antigen is presented by DCs and subsequently activates natural killer (NK) cells, CD4 T cells and CD8 T cells. Simultaneously, IL-10-producing inducible CD4+CD25+ T regulatory (T Reg) cells are generated, leading to immunosuppressive T cell response.[3]

EPIDEMIOLOGY

Except Antartica, rickettsial infections are prevalent throughout the world. For India, the reported numbers are an underestimate due to lack of community-based data and the non-availability of confirmatory laboratory tests. Rickettsial disease in India has been documented from Jammu and Kashmir, Himachal Pradesh, Uttaranchal, Rajasthan, Assam, West Bengal, Maharashtra, Kerala and Tamil Nadu (1,6-8). Batra has reported a high magnitude of scrub typhus, spotted fever and Indian tick typhus caused by R. conorii.[4]

Most of the cases in the outbreaks reported have a history of animal contact (dog/cattle) or insect bite. Most of the outbreaks in Karnataka, Tamil Nadu, Andhra Pradesh, and other places have reported patients from rural areas. Although rickettsial diseases are common in rural areas, but its presence has been documented in urban areas such as Delhi and Bengaluru and other towns in India, where mites and other vectors may be present in vegetations near residences. Seasonal trend has been reported in most of the cases. Outbreaks were observed soon after the rainy season (August–November). It is because of the vector breeding habits, especially mites. The egg hatches to larva due to increased humidity post monsoon.

Moreover, it is the larval stage that transmits the disease. Outbreaks in India have reported cases that are more prevalent in male gender.[5]

Table 1: Outbreaks reported in India before the year 2000.[5]

Disease	Years	Cases	Place
Scrub typhus	1934	Firs case reported	Himachal Pradesh
Scrub typhus	1946	616	Assam
Scrub typhus	1947-1950	912	Jamshedpur, Bihar
Scrub typhus	1965	Few cases	Indo-Pak war in Jammu-Sialkot sectors
Indian tick typhus	1984		
Scrub typhus	1990	11	Pakistan border of India
Scrub typhus	1992	Few cases	IMA Dehradun
Scrub typhus	1993	6	Eastern sector of Pakistan border of India

Table 2: Outbreaks reported in India between the year 2000 and 2018.[5]

Disease	Years	Cases	Place
Scrub typhus	2001-2002	28	Tamil Nadu
Rickettsial infection	2003-2004	150	Himachal Pradesh
Indian Tick typhus	2007	357	Himachal Pradesh
Rickettsial infection	2007	38	Manipur
Spotted fever	2006-2008	58	Tamil Nadu
Rickettsial infections	2005-2009	87	Delhi
Scrub typhus	2009	9	Uttarakhand
Rickettsial infections	2009	27	Jammu and Kashmir
Scrub typhus	2009-2010	75	Central India
Rickettsial infections	2010-2012	62	Bengaluru
Scrub typhus	2010	80	Meghalaya
Scrub typhus	2011	200	Himachal Pradesh
Scrub typhus	2011	9	Nagaland
Scrub typhus	2011-2012	176	Andhra Pradesh
Spotted fever	2013	4	Uttar Pradesh
Scrub typhus	2013-2015	5	West Bengal
Scrub typhus	2013-2015	115	Uttarkhand
Flea-borne rickettsial disease	2013	300	Sub-himalayan region of Himachal Pradesh
Malignant Mediterranean spotted fever	2015	1	Karnataka
Scrub typhus	2014-2016	283	Mizoram
Typhus fever	2016	25	Maharashtra
Indian tick typhus	2017	1	Karnataka

Table 3: Geographical distribution of Rickettsial infections in India.[5]

Biogroup	Disease	Vector	Rickettsial species	Geographical distribution
Typhus group	Endemic typhus	Ratflea (Xenopsylla cheopis) Rarely a cat flea (Ctenocephalides felis)	Rickettsia typhi Rickettsia felis	Shimla, Kashmir, Jabalpur, Mumbai, Lucknow, Pune.
Spotted fever group	Indian tick typhus Flea-borne spotted fever	Tick (Rhipicephalus sanguineus) Rat flea (Ceratophyllus fasciatus)	Rickettsia conorii Rickettsia species (R14 strain)	Nagpur, Jabalpur, Sagar, Pune, Lucknow, Bengaluru, Secunderabad. Western Himalayan region- Himachal Pradesh
Scrub typhus (Most common)	Scrub Typhus	Mite (Trombiculide)	Orientia tsutsugamuhi	Sub-Himalayan belt from Jammu to Nagaland, Sikkim, Darjeeling, Himachal Pradesh, Bihar, Rajasthan, Maharashtra, South India- Puducherry, Tamil Nadu, Kerala, Karnataka.

CASE PRESENTATION

A 36-year-old male patient was admitted to Ballari Medical College and Research Centre, Ballari, Karnataka, with chief complaints of fever with chills, burning micturition, and generalized body aches for the past week and patient was also presented with purple pin point spots(PETECHIAE) which looked like rash.

PAST HISTORY

No history of similar complaints in the past.

No history of Hypertension, Diabetes Mellitus, or ischemic heart disease.

PERSONAL HISTORY

Diet: Mixed

Sleep: Normal

Bowel & Bladder: Regular

Habits: Nil.

ON EXAMINATION

BP: 120/70mmHg

Pulse: 102bpm

Spo2: 96%

GRBS: 115mg/dl

PICCLE: Negative

CNS: Patient was alert and oriented.

P/A: Tenderness was present in the suprapubic region on deep palpation.

RS: Bilateral air entry present; no added sounds.

CVS: S1 and S2 heard, normal.

Fig 6: Petechial Purpuric Rash.

LABORATORY INVESTIGATIONS

PARAMETER	D1	D2	D3	D4	D5	REFERENCE RANGE
Haemoglobin	11.2	9.2	9.0	9.4	9.3	13-18gm/dl
Total leucocyte count	10260	13780	16400	14150	12547	4000-11000cells/cumm
Deferential count	76/20	80/12	88/6	80/16	74/20	40-70/20-40%
Platelet count	0.68	0.79	0.77	1.060	1.84	1.5-4.5lakh/cumm
Packed cell volume	35.6	28.6	77.7	28.9		45-55%
Mean corpuscular	83.4	81.0	80.8	80.6		80-100fl

volume						
Mean corpuscular haemoglobin	26.3	26.1	26.2	26.3		27-34pg
Erythrocyte sedimentation rate	16					0-20mm/hr
Random blood sugar	138					70-140mg/dl
Blood urea	23		38		20	
Serum creatinine	1.2		0.9		0.8	0.7-1.4mg/dl
Serum sodium	138		136		130	136-145mEq/l
Serum potassium	5.2		5		4.6	3.48-5mEq/l
Total protein	6.4		5.2		5.6	6-8.3g/dl
Albumin	3.5		2.8		2.2	3.2-5.4g/dl
Alanine amino transferase	77		39		46	0-45IU/L
Aspartate amino transferase	28		38		43	0-40IU/L
Alkaline phosphatase	40		160		197	20-140U/L

OTHER TESTS

- Culture Sensitivity: -No growth
- Widal: - Postive
- USG Abdomen: -Normal
- **Weli felix: - OX19, OX2 +ve 1:320 OXK -ve**
- Dengue: - Negative
- ECG: - Normal sinus rhythm
- Leptospira IgM ELISA: - Equivocal

TREATMENT CHART

SL. No	GENERIC NAME	ROUTE	DOSE	FREQUENCY	DURATION
01	Inj.Ceftriaxone	IV	1g	1-0-1	D1-D2
02	Inj.Paracetamol	IV	100ml	1-0-1	D1-D9
03	Inj.Pantoprazole	IV	40mg	1-0-0	D1-D9
04	IVF.2-pint NS	IV	150ml/hr		D1-D9
05	Tab.Doxycycline	PO	100mg	1-0-1	D3-D8
06	Inj.Cefotaxime	IV	1g	1-1-1	D3-D5
07	Tab.Paracetamol	PO	500mg	1-1-1	D5-D9
08	Inj.Piperacilline+tazobactam	IV	4.5g	1-1-1	D5-D9
09	Liquid paraffin+ Betamethasone cream	Locally			D6-D9
10	T. Vitamin.C	PO	90mg	Once daily	D6-D9
11	Calamine lotion	Locally			D6-D8

DISCUSSION

Rickettsial fever is most common undiagnosed cause of acute febrile illness because of its nonspecific signs and symptoms. Symptoms such as fever, headache chills etc are similar to symptoms of dengue and typhoid fever early diagnosis is difficult. Here a 36 years old male patient is presented with fever, chills and body aches and burning micturition and petechial purpuric rash. Objective evidences showed leucocytosis, thrombocytopenia, low albumin levels with slightly elevated liver enzymes suggesting rickettsial infection. To support above evidence, weil-felix was positive for ox19 and ox2. Whereas a negative dengue and blood culture ruled out other causes of fever. The patient showed improvement after starting Doxycycline which is drug of choice for rickettsial infections. Supportive pharmacotherapy of antipyretics, IV fluids and antibiotics stabilized patient. This case emphasizes need of awareness about rickettsial infections among healthcare professionals. Clinical pharmacist can take active role in the identification, treatment chart review and monitoring of patient during hospital stay.

CONCLUSION

Rickettsial fever should be considered in a patient presenting with acute fever and non-specific symptoms in endemic areas. Early diagnosis and treatment with choice of drug selection (such as doxycycline) can improve clinical outcomes. This case highlights importance of timely recognition, appropriate investigations and rational antimicrobial therapy. Increased awareness among clinicians can help in early diagnosis and reduction in reducing complications associated with rickettsial infections. A pharmacist can educate about disease to patient and create awareness even in general public as well.

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