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A TALE OF TWO TYPHUS: CONTRASTING PULMONARY AND HLH-LIKE MANIFESTATIONS OF SCRUB TYPHUS ACROSS NORTH AND SOUTH INDIA

Internal Medicine

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ABSTRACT

Background: Scrub typhus, caused by Orientia tsutsugamushi, is an acute febrile illness endemic to South and Southeast Asia. While classical presentations include fever, rash, and eschar, atypical systemic manifestations involving the lungs, liver, and hematologic system are increasingly recognized. These varied presentations can lead to diagnostic delay and mismanagement. **Case Summary:** We report two atypical presentations of scrub typhus: (1) A 60-year-old female with a history of type 2 diabetes mellitus presented with high-grade,fever, headache, and generalized weakness lasting one week, complicated by exudative pleural effusion and hepatocellular dysfunction with a classical eschar, and (2) a 36-year-old male developing severe hyperferritinemia, thrombocytopenia, coagulopathy, and multiorgan dysfunction consistent with hemophagocytic lymphohistiocytosis (HLH)-like syndrome secondary to scrub typhus infection. Both cases tested positive for scrub typhus. The first patient improved with doxycycline and supportive care, while the second required aggressive management with doxycycline, steroids, and critical care support. **Conclusion:** Scrub typhus should be considered in the differential diagnosis of unexplained febrile illness with pleural effusion or pancytopenia, especially in endemic regions. Recognition of HLH-like syndromes and early initiation of doxycycline can be lifesaving.

KEYWORDS

Scrub typhus, Orientia tsutsugamushi, pleural effusion, hemophagocytic lymphohistiocytosis, hyperferritinemia, India.

INTRODUCTION

Scrub typhus is an acute febrile zoonosis caused by Orientia tsutsugamushi, transmitted by larval trombiculid mites ("chiggers"), and remains a significant cause of acute undifferentiated febrile illness across Asia [1,2]

India lies within the endemic "tsutsugamushi triangle," and outbreaks have been increasingly reported from diverse ecological regions ranging from the Himalayan foothills to southern coastal plains [3].

Classically, scrub typhus presents with fever, myalgia, rash, and an eschar, but atypical manifestations involving the lungs, liver, kidney, and hematopoietic systems are frequently observed [4].

Clinical overlap with dengue, leptospirosis, and malaria often results in delayed diagnosis [5]. Severe complications such as ARDS, myocarditis, meningoencephalitis, and disseminated intravascular coagulation may occur, especially in untreated cases [6].

We describe two atypical cases from a tertiary care centre in Mumbai, India-one presenting as exudative pleural effusion and another as hemophagocytic lymphohistiocytosis (HLH)-like syndrome-both due to scrub typhus. These cases underscore the need for high clinical suspicion in endemic zones, as early diagnosis and prompt doxycycline therapy are crucial for favourable outcomes.

CASE REPORTS

CASE 1 – Scrub Typhus Presenting as Exudative Pleural Effusion in a 60-Year-Old Female

A 60-year-old female with a known history of type 2 diabetes mellitus, well controlled on oral hypoglycemic agents, presented with high-grade intermittent fever accompanied by generalized weakness, malaise, and myalgia for 7 days. She denied cough, chest pain, rash, or urinary or gastrointestinal complaints.

Initial Evaluation And Treatment

At presentation to a local practitioner, preliminary investigations were interpreted as consistent with a tropical febrile illness, and the patient was empirically started on intravenous ceftriaxone (2 g/day) and oral azithromycin (500 mg/day) for presumed enteric or respiratory infection. Despite therapy, fever persisted with worsening fatigue.

Routine work-up at that time showed borderline Widal positivity (O and H antigens 1 : 40), negative malaria and dengue serology, and sterile blood cultures. Other parameters were as follows: hemoglobin 10.2 g/dL, WBC 3270/µL (with eosinopenia), platelets 1.61×10^5/µL, CRP 72 mg/L, Na^+ 129 mmol/L, K^+ 4.1 mmol/L, Cl^- 100.2 mmol/L, and serum creatinine 0.77 mg/dL.

She remained hospitalized for intravenous antibiotics and supportive care.

Clinical Deterioration

Parameter	Normal Range	Admission	Discharge
HEMATOLOGY			
Hemoglobin (g/dL)	12–15	10.2 ↓	10.8 * ↑
Platelets (×10³/µL)	150–410	161	210 * ↑
WBC count (×10³/µL)	4–11	3.27 ↓	6.8 * ↑
Neutrophils (%)	40–80	74	68 * ↓
Lymphocytes (%)	20–40	20.5	25 * ↑
INFLAMMATORY MARKERS			
CRP (mg/L)	< 5	72.2 ↑	10 * ↓
WIDAL / TYPHIFAST			
S. typhi O Ag titre	–	1 : 40	–
S. typhi H Ag titre	–	1 : 40	–
S. paratyphi AH Ag	–	No agglutination	–
S. paratyphi BH Ag titre	–	1 : 20	–
S. typhi IgM	–	Negative	–
RENAL / ELECTROLYTES			
Creatinine (mg/dL)	0.51–0.95	0.77	0.75 →
Sodium (mmol/L)	136–148	129.6 ↓	136 ↑
Potassium (mmol/L)	3.5–5.1	4.11	4.3 * →
LIVER FUNCTION	Normal		-
SUMMARY TREND		Admission: microcytic anemia, hyponatremia, high CRP	Discharge: normalized electrolytes & LFTs, CRP fall, Hb ↑

On the seventh hospital day, while being prepared for discharge after transient defervescence, the patient developed acute breathlessness

and oxygen desaturation (SpO_2 86 % on room air). Physical examination revealed reduced air entry and stony dullness at bilateral lung bases. There was no peripheral edema or jugular venous distension.

Radiologic And Pleural Evaluation

A chest X-ray demonstrated left-basal consolidation and blunting of both costophrenic angles, raising suspicion of pleural effusion. She was given intravenous diuretics for presumed fluid overload, with minimal improvement.

Subsequent ultrasound thorax confirmed moderate bilateral pleural effusion, and 700 mL of straw-colored fluid was aspirated from the left hemithorax.

Pleural fluid analysis fulfilled Light's criteria for exudative effusion (fluid protein 2.18 g/dL vs serum protein 6.28 g/dL; fluid LDH 179 IU/L vs serum LDH 280 U/L). Cytology was negative for malignant cells, and ADA 24 IU/L effectively ruled out tuberculosis.

(Insert PLeural effusion photo)

Table 2: Pleural Fluid Analysis

Parameter	Observed Value	Reference Range / Interpretation Basis
Appearance	Clear, straw-coloured	–
Protein (g/dL)	2.5	< 3 → Transudative range
Glucose (mg/dL)	85	> 60 = Normal
LDH (U/L)	150	< 200 = Transudative pattern
Cell count (/µL)	120 (80 % lymphocytes)	< 500 = Low cellularity
ADA (U/L)	20	< 40 = Non-tubercular
Culture / Gram stain	No growth / Negative	Sterile

Definitive Diagnosis

A focused re-evaluation was undertaken in view of persistent fever and new-onset effusion. Detailed systemic and skin examination identified a necrotic black eschar with an erythematous halo in the right posterior axillary fold, which had been overlooked earlier. The patient had recently traveled to Pondicherry, a recognized endemic region for rickettsial infections.

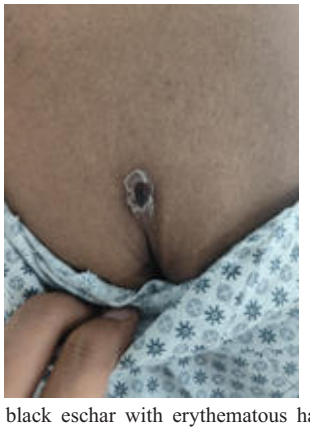

Fig 1: Necrotic black eschar with erythematous halo in the right posterior axillary fold

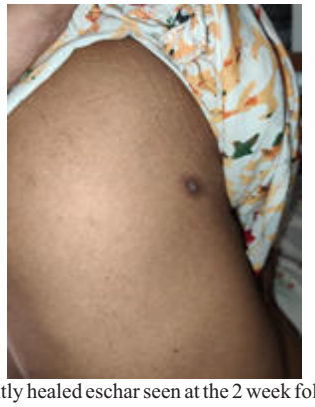

Fig 2: Significantly healed eschar seen at the 2 week follow up

Given the clinical presentation, exudative effusion, and the presence of an eschar, scrub typhus was suspected. IgM ELISA for Orientia tsutsugamushi was strongly positive, confirming the diagnosis.

Treatment And Outcome

She was commenced on oral doxycycline 100 mg twice daily for 10 days. Fever subsided within 48 hours, respiratory distress improved, and oxygen saturation normalized. Repeat ultrasonography after 7 days showed near-complete resolution of pleural fluid. She was discharged afebrile and asymptomatic, with the eschar healing significantly by the two-week follow-up.

CASE 2 – Scrub Typhus Presenting with HLH-like Syndrome

A 36-year-old previously healthy male presented with a 5-day history of high-grade continuous fever, accompanied by generalized weakness, body ache, and progressive confusion for the last two days. The patient had returned from Delhi one week prior to admission. There was no history of chronic illness, recent blood transfusion, jaundice, rash, cough, or urinary complaints.

Initial Presentation And Prior Management

Two days after the onset of fever, he consulted a local practitioner, where the illness was attributed to a viral upper respiratory tract infection (URTI). He was empirically prescribed antipyretics, antihistamines, and etizolam (0.5 mg nightly) for presumed anxiety-induced tachycardia, as his pulse was recorded at 130–140/min with a normal ECG. Within 24 hours of starting etizolam, the patient developed marked drowsiness, irritability, and disorientation to time and place, though he remained oriented to person and responded to commands. Family members noted fluctuating alertness, reduced oral intake, and worsening fatigue, prompting hospital admission.

Examination On Admission

On arrival to the emergency department, the patient was febrile (39.4°C), tachycardic (150 bpm, regular), blood pressure 130/80 mmHg, respiratory rate 28/min, and SpO_2 90% on room air. He appeared icteric, mildly dehydrated, and drowsy but arousable. Neurological examination revealed no neck stiffness or focal deficits, but he was disoriented to time and place, with an altered sleep–wake pattern consistent with grade I hepatic encephalopathy. Abdominal examination revealed diffuse tenderness, mild distension, and shifting dullness, suggesting ascites; however, there was no palpable hepatosplenomegaly. Cardiovascular and respiratory examinations were otherwise unremarkable, with no rash, eschar, or lymphadenopathy.

Initial Clinical Impression

The constellation of fever, jaundice, altered sensorium, and tachycardia raised initial suspicion of acute viral hepatitis with hepatic encephalopathy, leptospirosis, or dengue-associated hepatic injury. Differential diagnoses also included sepsis-associated coagulopathy and tropical fever–related multiorgan dysfunction.

Comprehensive investigations were ordered, including complete blood count, liver and renal function tests, coagulation profile, ferritin, triglycerides, and a tropical fever panel (dengue, malaria, leptospira, scrub typhus, and typhoid).

Parameter	Units	Normal Range	Admission	Discharge
HEMATOLOGY				
Hemoglobin	g/dL	13–17	11.2	12.4*
Platelet count	×10³/µL	150–410	47	215*
WBC count	×10³/µL	4–11	10.8	7.5*
COAGULATION / HLH PROFILE				
INR	—	<1.1	1.29	1.10*
APTT	sec	24.6–28.9	45.5	28.0*
Fibrinogen (plasma)	mg/dL	180–350	112	210*
Ferritin	ng/mL	30–400	8,578	520*
Triglycerides	mg/dL	<150	622	140*
LIVER FUNCTION				
Total Bilirubin	mg/dL	0.3–1.2	6.36	0.90*
Direct Bilirubin	mg/dL	<0.3	6.04	0.40*
Indirect Bilirubin	mg/dL	0.1–0.9	0.32*	0.50*
AST (SGOT)	U/L	10–50	115	42*
ALT (SGPT)	U/L	10–50	84	68*
ALP	U/L	40–129	194	86*

GGT	U/L	8–61	213	110*
Total Protein	g/dL	6.6–8.7	5.08	6.8*
A/G Ratio	—	1.0–2.1	—	1.3*
RENAL & ELECTROLYTES				
Creatinine	mg/dL	0.67–1.17	0.9–1.1	0.60*
Sodium	mmol/L	136–148	128	137*
Potassium	mmol/L	3.5–5.1	4.4	4.5
Bicarbonate	mmol/L	22–29	13.7	24*
INFLAMMATORY / CARDIAC				
CRP	mg/L	<5	197	8*
LDH	U/L	135–225	570	300*
NT-proBNP	pg/mL	<125	327	120*
SEROLOGY / MICROBIOLOGY				
Scrub Typhus PCR	—	—	Positive	—
Dengue/Leptospira/Malaria	—	—	Negative	—

Renal function was mildly impaired (eGFR 69 mL/min), and NT-proBNP 327 pg/mL suggested inflammatory myocardial stress. Peripheral smear showed normocytic anemia and thrombocytopenia without malarial parasites.

Diagnostic Reasoning

The initial differential diagnosis included acute viral hepatitis, leptospirosis, enteric fever, or sepsis with DIC. However, the tropical fever PCR panel returned positive for Orientia tsutsugamushi, while tests for dengue, malaria, and leptospira were negative.

Given the combination of fever, cytopenia, hyperferritinemia, hypertriglyceridemia, low fibrinogen and splenomegaly the patient fulfilled ≥5 HLH-2004 criteria, confirming a scrub typhus–associated HLH-like syndrome.

DISCUSSION

Scrub typhus remains an underrecognized cause of acute febrile illness in India, despite rising incidence [7]. Caused by Orientia tsutsugamushi, it infects endothelial cells and macrophages, leading to widespread vasculitis, vascular leakage, and immune dysregulation [8]. This mechanism underlies the multisystemic presentations and the atypical cases described herein.

Epidemiological Perspective

India lies within the endemic "tsutsugamushi triangle" extending from Japan to northern Australia and Pakistan [3]. Seasonal spikes occur post-monsoon, such as in our case. However, absence of eschar (seen in <50% of Indian patients) often delays diagnosis . Presence of eschar is considered as a marker of poor prognosis[9].

An important observation from our report is the wide geographic spread of scrub typhus across India. The first patient had recently traveled to Pondicherry (South India), while the second had returned from Delhi (North India)-demonstrating that Orientia tsutsugamushi infection is not confined to a single climatic region..

Large multicentric studies have confirmed its north–south distribution, with prevalence ranging from 9–47% of acute febrile illnesses and seropositivity as high as 40% in Tamil Nadu and 30% in Himachal Pradesh [7,10,11].

These findings-and our two cases from opposite ends of the country-underscore the pan-Indian emergence of scrub typhus and the need for clinicians nationwide to maintain a uniform index of suspicion.

Pathophysiology

Vascular endothelial injury by Orientia causes increased capillary permeability, perivascular infiltration, and microthrombi . Systemic vasculitis results in pulmonary, hepatic, renal, and CNS involvement . Cytokine storms involving TNF-α, IFN-γ, and IL-6 exacerbate tissue damage [12-14]

Pleural Effusion and Pulmonary Manifestations

Pulmonary involvement occurs in 20–70% of hospitalized scrub typhus cases. Pleural effusion develops due to endothelial vasculitis of pleural microcirculation, producing exudative effusions. The effusion typically has lymphocytic predominance and low ADA (<40 IU/L), as seen in Case 1[8, 15].

This along with its acute presentation as apposed to that of tuberculsis distinguishes it from tuberculous effusions and highlights the importance of early serological testing. Timely doxycycline therapy rapidly reverses pleural inflammation [16].

Scrub Typhus–Associated HLH

Secondary HLH is a hyperinflammatory syndrome characterized by uncontrolled activation of macrophages and T cells, leading to multiorgan dysfunction [17]. Infection-triggered HLH, particularly with O. tsutsugamushi, has been increasingly reported. As per Diagnostic criteria (HLH-2004) our patient had fever, cytopenia,, hypertriglyceridemia / hypofibrinogenemia, splenomegaly and ferritin >500 ng/mL. Our patient met five of these, supporting a diagnosis of scrub typhus–associated HLH [17].

Varghese et al. and Rathi et al. demonstrated elevated cytokines (IL-8, MCP-1) and incidence of HLH in up to 6% of severe scrub typhus [18]. Ferritin >5,000 ng/mL is both sensitive and specific for HLH [19].

Our patient's extreme ferritin elevation (8578 ng/mL), hypofibrinogenemia, and coagulopathy reflect profound macrophage activation.

Therapeutic Approach

Doxycycline (100 mg BID for 7–10 days) remains first-line therapy.Clinical defervescence within 48 hours is both therapeutic and diagnostic. Azithromycin is an effective alternative in pregnancy and doxycycline intolerance [20]

In scrub typhus–associated HLH, antimicrobial therapy alone is insufficient; corticosteroids are recommended to control cytokine-mediated injury [21]

T Zhang et al. demonstrated >80% survival with doxycycline–steroid combination therapy. In refractory cases, IVIG or etoposide may be considered [22,23]

Diagnostic Challenges

The absence of eschar, frequent false-positive Weil felix reactions, and overlapping features with dengue or leptospirosis hinder timely diagnosis. IgM ELISA remains the most accessible, with 95% sensitivity and specificity [24], while IFA and PCR are confirmatory [25]. Delayed doxycycline initiation (>7 days) is associated with a high mortality [26].

Prognostic Indicators

Poor prognostic markers include advanced age, thrombocytopenia, elevated transaminases, hypoalbuminemia, and multiorgan dysfunction. Mortality can exceed 30% in untreated cases but drops below 5% with timely therapy. [27]

Our cases illustrate this contrast: Case 1, treated early, recovered rapidly, while Case 2, with delayed recognition, developed HLH requiring aggressive therapy.

Clinical Challenges And Misdiagnosis

Scrub typhus is often misdiagnosed due to non-specific early symptoms-fever, malaise, and headache-mimicking enteric fever, malaria, or dengue In endemic regions, cross-reactivity of the Widal test is well documented [28], as seen in our case.

The presence of an eschar provides a strong diagnostic clue, but its detection requires careful examination of covered body areas. The eschar prevalence varies widely (7–80%) depending on clinician awareness, patient skin tone, and geography. A north Indian study by Sethi et al. reported an eschar in 45% of confirmed cases. Our case underscores the importance of thorough skin inspection, especially in intertriginous regions.

Public Health And Clinical Implications

In India, environmental changes, deforestation, and agricultural exposure increase mite contact [29]. Despite availability of effective treatment, low index of suspicion remains the key barrier. Routine inclusion of scrub typhus serology in febrile illness workups and early empirical doxycycline use can drastically improve outcomes

Laboratory Diagnosis

Serologic tests remain the mainstay of diagnosis. IgM ELISA is widely used due to its >90% sensitivity and specificity, especially in endemic populations.

The Indirect Immunofluorescence Assay (IFA), though gold standard, is not routinely available. PCR assays for O. tsutsugamushi DNA in blood or eschar tissue offer early diagnosis but remain limited to research or tertiary settings. In our case, IgM ELISA positivity combined with clinical context confirmed the diagnosis.

Public Health And Clinical Implications

The resurgence of scrub typhus in India is associated with environmental changes and inadequate vector control. Delayed diagnosis contributes to high mortality, yet it remains under-recognized in clinical practice [30,31]. In endemic areas, empirical doxycycline therapy should be considered for acute febrile illness with pulmonary findings, even before serological confirmation. Awareness programs and inclusion of scrub typhus in differential algorithms for fever of unknown origin are urgently needed

CONCLUSION

Scrub typhus can manifest with atypical presentations, including bilateral exudative pleural effusion that closely mimics other respiratory infections. A high index of clinical suspicion, combined with a detailed travel history and careful search for an eschar, remains essential for early recognition. Timely initiation of doxycycline typically results in rapid defervescence and prevents progression to severe, potentially fatal complications.

These cases underscore that scrub typhus is a master clinical mimicker, capable of presenting with isolated pleural effusion or hemophagocytic lymphohistiocytosis (HLH)–like syndromes. The absence of classical findings, such as an eschar, should not exclude the diagnosis, especially in endemic regions. Clinicians should consider scrub typhus in any febrile illness with pulmonary or hematologic involvement. Prompt diagnosis and doxycycline therapy are life-saving, while early recognition of immune-mediated complications like HLH warrants adjunct corticosteroid therapy to prevent multi-organ failure and mortality.

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