Scrub Typhus with Acute Respiratory Distress Syndrome: A Case Report with Narrative Review of Current Management Guidelines

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ABSTRACT

Scrub typhus, also known as tsutsugamushi disease, is an acute rickettsial zoonotic infectious disease caused by mite-borne bacterium Orientia tsutsugamushi. The disease spectrum can range from mild, nonspecific, self-limiting febrile illness to rapidly progressive, potentially lethal multiorgan dysfunction involving the lungs, heart, kidneys, liver and brain. Acute respiratory distress syndrome (ARDS) is a serious complication of scrub typhus.

A 51-year-old male patient from Amini Island, Lakshadweep, was admitted to our hospital with a history of five days of fever, headache, bilateral inguinal lymphadenopathy and acute onset of breathing difficulty for one day. A physical examination revealed the presence of an eschar on the right thigh. A serological and imaging diagnostic evaluation identified scrub typhus with thrombocytopenia, which subsequently progressed to ARDS, despite early initiation of oral doxycycline and adjunctive steroids. At FiO_2 of 35% with a PaO_2 of 75 mmHg and SpO_2 of <90%, PaO_2/FiO_2 and SpO_2/FiO_2 ratios were 214 and <257, respectively, suggestive of mild ARDS. He recovered completely after 2 weeks of appropriate treatment and supportive care.

Scrub typhus is a critical differential diagnosis for acute undifferentiated fever with thrombocytopenia or haemorrhage, even in areas not strictly endemic. Patients with clinically suspected scrub typhus should be instituted empirical antibiotic treatment promptly—even before laboratory confirmation, to prevent progression to ARDS. Compared to the 2012 Berlin definition, the 2024 revised diagnostic criteria for ARDS - "New Global Definition" - improve early detection, enhance inclusivity for non-intubated patients, and allow for diagnosis in resource-limited settings.

Keywords: Scrub Typhus, Acute Respiratory Distress Syndrome, Thrombocytopenia, Diagnosis, Management, Guidelines.

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Introduction

Scrub typhus is an acute rickettsial zoonotic infectious disease transmitted by chiggers (larval stage of mites in the family Trombiculidae).[1] and caused by Orientia tsutsugamushi (an obligate intracellular, Gram-negative bacterium in the family Rickettsiaceae).[2] The original name of scrub typhus, as given by Hashimoto in 1810, is "tsutsugamushi disease" meaning tsutsuga ("illness" or "harm") and mushi ("bug" or "insect") in Japanese.[3]

Scrub typhus is an emerging global public health problem that has traditionally affected mainly the "tsutsugamushi triangle" in the Asia-Pacific region, albeit with cases now being increasingly reported in Africa, South America, and the Middle

East.[4] Scrub typhus threatens one billion people and causes an illness incidence of one million worldwide each year.[5] Although the median mortality rate of untreated scrub typhus patients is 6% and that of treated patients is 1.4%, the mortality rate remains high and can reach up to 70% without proper treatment.[6]

Although the tiny, six-legged chiggers (reservoir and vector of Orientia tsutsugamushi) attach, inject digestive enzymes and cause subsequent intense itch[1] that often peaks 24–48 hours after the bite, the initial 'bite' is often painless and unnoticed.[7] In humans, following the incubation period, typically between 10 and 12 days following the bite, nonspecific flu-like symptoms develop- such as fever and rash. A distinctive, if not pathognomonic, painless, non-itchy eschar, not unlike a 'cigarette burn' can additionally develop at the bite site, varying in detection and distribution rates, by sex, race and skin colour.

The classic case description includes an eschar where the chigger mite bites, regional lymphadenopathy and maculopapular rashes.[8] The typical clinical constellation of scrub typhus is abrupt high fever, severe headache, lymphadenopathy, generalized myalgia, eschar, and rash. Overt hepatosplenomegaly, present in half to three-fourths of children, may be present only in a third of adult cases. However, laboratory signs of liver dysfunction (elevated liver transaminases) and renal impairment are much more common, often in over half to most adult cases. Yet, liver dysfunction and its increased severity in scrub typhus do not correlate very well with increased morbidity and/or poor outcomes.[9]

A fundamental pathologic characteristic of scrub typhus is disseminated vasculitis, which can cause damage to one or more organs, such as the lung, kidney, liver, brain, meninges, heart, and skin.[10] The disseminated vasculitis and endothelial cell infection lead to perivascular T cell and monocyte or macrophage infiltration, followed by inflammatory responses producing a wide range of cytokines.

Notwithstanding the beneficial effects of cytokines in microbial destruction, the immune response's dual nature can lead to intense, overwhelming pro-inflammatory cytokine production with dysregulated self-antigen tolerance that produces severe complications such as respiratory failure, including acute respiratory distress syndrome (ARDS) renal failure, hepatitis, meningoencephalitis, and myocarditis.[11]

Lung infection, as self-resolving or promptly treatable mild interstitial pneumonitis, is common in scrub typhus. In contrast, ARDS that occurs when fluid leaks into the alveoli as a result of sepsis that follows vascular damage, cellular infiltration, pulmonary edema and haemorrhage is a serious and potentially fatal complication of scrub typhus.[12]

While scrub typhus is underdiagnosed because its non-specific symptoms—fever, headache, and rash—mimic other illnesses, including co-endemic diseases like typhoid, dengue, malaria and leptospirosis;[13] its complications, although rare, are difficult to treat. In this manuscript, we report a case of scrub typhus from a non-endemic region that progressed to ARDS and discuss its diagnosis, pathological process, and treatment. Besides, it provides a reference for the diagnosis and treatment of complex tsutsugamushi disease.

Case Report (Results)

A 51-year-old male patient from Amini Island, Lakshadweep, was admitted with fever and headache for 5 days. He was apparently alright without any specific medical history. 4 days before coming to our hospital, He was admitted to a nearby hospital exhibiting signs of fever and bilateral inguinal lymphadenopathy. He was started on intravenous (IV) cephalosporin, paracetamol for fever control and other supportive treatments. He did not have a significant past medical history, except for essential hypertension for which he was on a calcium channel blocker. When he developed respiratory symptoms, he was referred to our hospital.

On admission, he was conscious and oriented; his body temperature was 38.8°C, blood pressure was 100/60 mmHg, pulse rate was 84 beats per minute, and SpO_2 was 96% with minimal oxygen support. A physical examination revealed the presence of an eschar on his right thigh of approximately 2×1 cm (Figure 1). A respiratory system examination by auscultation demonstrated fine, crepitations at the base of both lungs, left more than right.

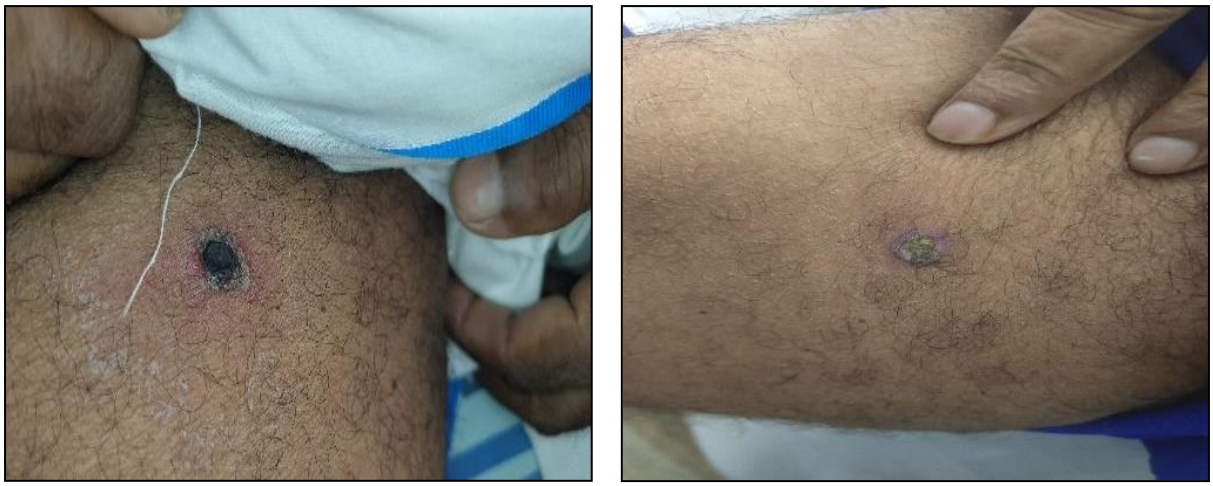

Figure 1: **Eschar: a clinical photograph showing erythematous papule on the right thigh with scab and resolution of eschar in the second photograph.**

Laboratory findings upon admission revealed that his haemoglobin level was 11.7 g/ dL, haematocrit was 38%, white blood cell count was 14600/µl, and platelet count was 100,000/µL. Serum blood urea and creatinine levels were 67mg/dl and 1.4 mg/dL, respectively. The total bilirubin level was 3.2 mg/dL; aspartate aminotransferase, 75 IU/L; alanine aminotransferase, 99IU/L; and alkaline phosphatase, 359 IU/L. Other laboratory tests revealed glucose, 128 mg/dL; There were also electrolyte disturbances, including hyponatremia (Na+ 123 mmol/L) and hypokalaemia (K+ 3.7 mmol/L). C-reactive protein, 133.7 mg/dL; Procalcitonin, 1.96, activated partial thromboplastin time, 41.2 seconds; prothrombin time, 13.7 seconds.

Table 1: Clinical and laboratory parameters on admission, progression to ARDS and at recovery.

Parameter	Normal range/value	On admission	ARDS	At recovery
Body temperature	36.1 to 37.2°C	38.8°C		
Blood pressure	120/80 mmHg	100/60 mmHg		
Pulse rate	60 to 100 beats/min	84 beats/min		
SpO_2	95% to 100%	96%		
Haemoglobin	13.8 to 17.2 g/dL	11.7 g/ dL		
Hematocrit	41% to 50%	38%		
White Blood Cell count	4,000 to 11,000 µL	14,600/µL		
Platelet count	150000 to 450000	100,000/µL	86000/µL	97000/µL
Serum blood urea	7 to 20 mg/dL	67mg/dl		
Creatinine levels	0.7 to 1.3 mg/dL	1.4 mg/dL		
Bilirubin level	0.1 to 1.2 mg/dL	3.2mg/dL		
Aspartate Aminotransferase	8 to 48 IU/L	75 IU/L	154 IU/L	

Alanine Aminotransferase	7 to 56 IU/L	99 IU/L	182 IU/L	88 IU/L
Alkaline Phosphatase	44 to 147 IU/L	359 IU/L	476 IU/L	227 IU/L
Glucose	70 to 99 mg/dL	128 mg/dL		
Serum Sodium	135 to 145 mmol/L	123 mmol/L	134 mmol/L	138 mmol/L
Serum Potassium	3.5 to 5.0 mmol/L	3.7 mmol/L	3.8 mmol/L	4.1 mmol/L
C-reactive protein	<0.3 mg/dL	133.7 mg/dL		
Procalcitonin	<0.05 µg/L	1.96		
Activated partial thromboplastin time	21 to 40 seconds	41.2 seconds	43 seconds	38 seconds
Prothrombin time	10 to 13.5 seconds	13.7 seconds	15 seconds	13.2 seconds
Arterial Blood Gas Analysis (ABGA)				
pH	7.35 to 7.45	7.44	7.47	7.38
$PaCO_2$	35 to 45 mmHg	37 mmHg	38 mmHg	35 mmHg
PaO_2	75 to 100 mmHg	106 mmHg	75 mmHg	101 mmHg
HCO_3^-	22 to 26 mmol/L	25.7 mmol/L	27.9 mmol/L	24.2 mmol/L
SpO_2	95 to 100%	98%	90%	98%

The initial arterial blood gas analysis (ABGA) revealed the following: pH, 7.44; $PaCO_2$, 37 mmHg; PaO_2, 106 mmHg; HCO_3^-, 25.7 mmol/L; and oxygen saturation, 98%. (Table 1) Chest radiographs and computed tomography (CT) indicated bilateral pulmonary exudation. (Figures 2 and 3).

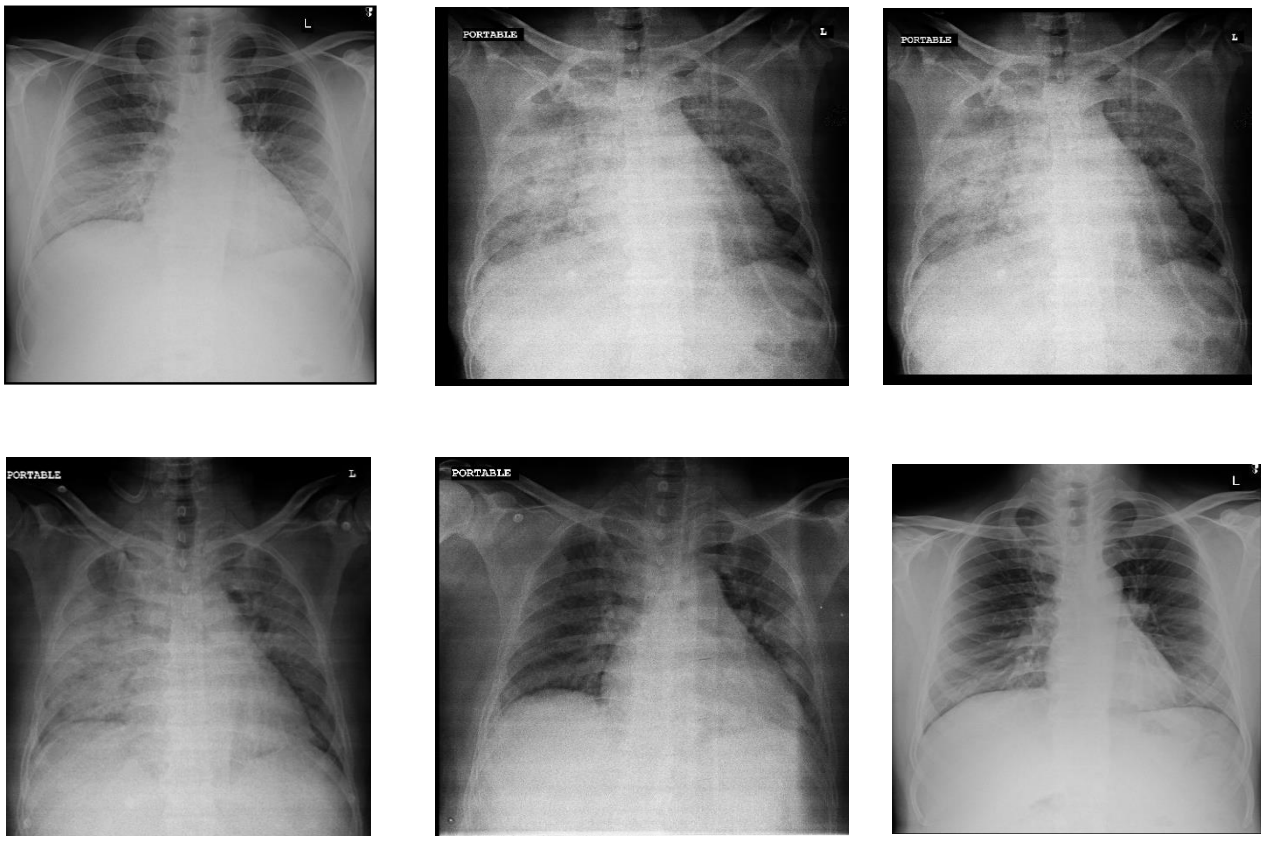

Figure 2: Initial Chest radiographs indicate intensifying bilateral pulmonary exudation (2a to 2c). Subsequent radiographs show progressive and significant resolution of the exudates following intensive treatment (2d to 2f).

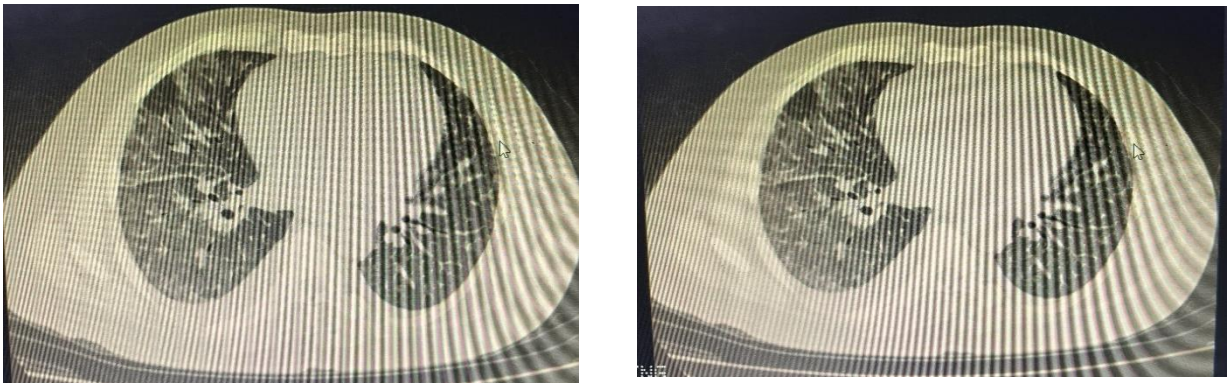

Figure 3: Computed tomography (CT) indicated bilateral pulmonary exudation with multiple peripheral ground-glass opacities predominantly in the middle and lower lobes.

Based on his clinical history, findings of eschar, as well as the laboratory and imaging examinations, a preliminary diagnosis of scrub typhus with evidence of deranged liver and renal function tests was arrived at.

Meropenem and Clindamycin was used as initial empirical antibiotics, and doxycycline orally (100 mg twice a day) was used to treat scrub typhus. In addition, he received symptomatic treatment, such as, liver protection – Silymarin and Glycyrrhizin Acid Preparations; and gastric acid inhibition with proton pump inhibitors. Steroids (Methyl prednisolone 125mg/day) were also administered as an adjunctive, short-term therapy.

However, liver and renal function parameters continued to be mildly elevated and platelet count decreased to 86000/µL. On ICU Day 2, ABGA revealed the following: pH, 7.47; $PaCO_2$, 38 mmHg; PaO_2, 75 mmHg; HCO_3^-, 27.9 mmol/L; even with oxygen therapy, saturation was not maintained above 90% and lead to the development of ARDS. (Table 1) Thereafter, an end-expiratory pressure (EEP) of 8 cm H_2O for non-invasive ventilation (NIV/CPAP) was used to manage hypoxemia.

At that time, detection of IgM antibodies in human serum for Orientia tsutsugamushi was observed to be reactive and the diagnosis of scrub typhus was confirmed serologically. Blood, sputum and urine culture showed no growth as well. The patient also tested negative for Leptospira antibody, Dengue non-structural protein 1 (NS1) and IgM antibody, hepatitis viral panel, and Malaria rapid antigen diagnostic test and blood smear microscopy.

After 5 days of intensive treatment, the platelet count was elevated, and other blood parameters showed improvement. (Table 1) There was a significant improvement in the chest signs as well as a reduction in exudates, radiologically. (Figure 2) We were able to taper oxygen support. In addition, the eschar had nearly cleared. On day 10 of admission, the patient was taken off oxygen support, and the antibiotics were tapered; doxycycline was continued; the patient was then shifted to the ward.

Scrub typhus diagnosis based on IgM ELISA was confirmed by paired serology done 2 weeks later. The patient was discharged after 15 days on oral doxycycline, and there was no relapse of symptoms on review after 1 month.

Discussion

We report the successful diagnosis and treatment of scrub typhus complicated by ARDS, in Kochi, Kerala, in a patient from Amini Island, Lakshadweep.

Scrub typhus, the commonest occurring rickettsial infection in India, is endemic in several

states in South India - Karnataka, Tamil Nadu, Puducherry and Kerala.[14] In Kerala, the most recent data shows 70 per cent of cases from Thiruvananthapuram and the rest from Kozhikode, Idukki, Kollam and Wayanad districts.[15] Although not endemic per se to Kochi or Lakshadweep, our case highlights the need for clinicians to maintain a high index of suspicion for scrub typhus in endemic adjoining regions, or even non- endemic areas.

Our subject had presented with an eschar on the right thigh and fever with thrombocytopenia. Fever with thrombocytopenia occurs in scrub typhus in about half of cases and is associated with ARDS and a poorer prognosis.[16] Scrub typhus was diagnosed based on IgM ELISA and confirmed by paired serology 2 weeks later. Scrub Typhus IgM antibody is known to remain in circulation above threshold even for 12 months, and a four-fold rise in titre by paired sera (acute and convalescence) is essential to confirm recent infection.[17]

Our subject with scrub typhus confirmed by IgM ELISA, had deranged liver and kidney function tests with thrombocytopenia. Worsening of ABG and respiratory parameters lead to the diagnosis of ARDS. During ARDS management, intubation was not considered as the patient did not develop acidosis (pH<7.25) or a respiratory rate >35 while on NIV, CPAP EEP of 8 cm H_2O.[18]

The PF ratio (PaO_2/FiO_2) is an invasive, critical care metric used to assess hypoxemia and severity of lung injury (e.g., ARDS) by dividing the arterial partial pressure of oxygen (PaO_2, in mmHg) by the fraction of inspired oxygen (FiO_2, as a decimal). A normal ratio is >450. Peripheral oxygen saturation (SpO_2) from a pulse oximeter is a non-invasive clinical surrogate of the gold standard-PaO_2.

According to the 2012 Berlin definition, ARDS must be diagnosed within a week of the development of new or worsening respiratory symptoms or a recognised clinical insult. Additionally, bilateral opacities that cannot be entirely attributed to effusion, lobar or lung collapse, or nodules must be detected on chest computed tomography (CT) or X-ray. When the patient is on continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP), the PaO_2/FiO_2 (PF) ratio must be ≤300.[19]

Although the Berlin definition increases diagnostic accuracy, its application is limited by its reliance on arterial blood gas (ABG) analysis and PEEP settings, potentially underestimating the burden of ARDS in low-and middle-income countries. The Kigali modification was therefore created, in which, lung ultrasound was added as an alternate diagnostic imaging modality, the PEEP requirement was removed, and the oxygenation criterion was changed from the PF ratio to the SpO_2/FiO_2 (SF) ratio ≤315 (with SpO_2 ≤97% as an extra criterion). Several clinical studies have demonstrated a strong positive correlation between the invasive PF ratio and the non-invasive SF ratio as - Mild (200 < PF ratio ≤ 300 or 235 < SF ratio ≤ 315); moderate (100 < PF ratio ≤ 200 or 148 < SF ratio ≤ 235); and severe (PF ratio ≤ 100 or SF ratio ≤ 148).[20]

The diagnostic criteria of ARDS have therefore been updated in 2024 to stratify not only intubated, but also non-intubated settings, together with a revision of the definition to accommodate resource-poor settings as well. In the non-intubated ARDS group, a minimum High-Flow Nasal Oxygen (HFNO) flow rate of 30 L/min or an end-expiratory pressure of at least 5 cm H_2O for NIV/CPAP is required to diagnose ARDS. While the intubated category aligns with the Berlin 2012 criteria, ARDS identification in resource-limited settings is now possible irrespective of oxygen therapy type, provided SpO_2 remains ≤97% and the SF ratio is ≤315 mm Hg.[21] Our patient at FiO_2 of 35% with a PaO_2 of 75 mmHg, and SpO_2 of <90% yielded a PF and SF ratios of 214 and <257, respectively, suggestive of mild ARDS.

More than one-third of patients who need hospitalisation have pneumonia that progresses to ARDS, making it one of the most common

complications of scrub typhus. According to several studies, the mortality rate of ARDS in scrub typhus is significant, ranging from 22% to 45%. The risk is considerably increased when ARDS develops, even if the overall fatality rate from untreated scrub typhus is 7–30%.[22,23]

Older age, early onset pneumonitis, development of thrombocytopenia, and delayed diagnosis have been identified as risk factors for scrub typhus progression to ARDS.[24] Recent studies on scrub typhus, particularly in South Asia, indicate that mortality associated with ARDS is largely driven by its presence within a broader spectrum of multiorgan dysfunction syndrome (MOD), rather than ARDS alone. Furthermore, higher mortality rates and longer Intensive Care Unit (ICU) stays are strongly correlated with higher Sequential Organ Failure Assessment (SOFA) scores (>7), as well as encephalopathy, hypoalbuminemia, sepsis and septic shock.[16,25–27] The quick SOFA score, which includes the respiratory rate above 22/min, altered mentation, and systolic BP below 100 mmHg, is a suitable practical tool to predict mortality in scrub typhus with ARDS.[27]

Given the high correlation of ARDS, a leading cause of mortality for scrub typhus patients, with delay in diagnosis and treatment; early diagnosis with timely institution of antibiotics like doxycycline (first-line/drug of choice for all ages, typically 100 mg twice daily for 7 days) or azithromycin (first-line alternative in cases of pregnancy, children under eight years, or when doxycycline is contraindicated), as a single 500 mg dose or 3-day course can prevent progression to serious complications such ARDS, pneumonia, and MOD.[16]

In critically ill scrub typhus patients, particularly those in shock, IV doxycycline should be used since the absorption of orally administered doxycycline may be suboptimal; or where unavailable, IV azithromycin may be used either alone or in combination with oral doxycycline.[28] A recently published Indian randomised controlled trial (RCT) has demonstrated a significantly better primary outcome (a composite of 28-day mortality, day-7 complications, and day-5 fever) from combination therapy with IV doxycycline and IV azithromycin than with either drug alone; each being equally effective for monotherapy. The combination likely provides a dual-targeted, complementary, and more comprehensive inhibition of Orientia tsutsugamushi protein synthesis; with azithromycin binding the 23S rRNA of the 50S ribosomal subunit, and doxycycline preventing aminoacyl-tRNA binding to the 30S ribosomal subunit.[29]

In addition to their antibacterial activity, studies have shown that several antibiotic classes, such as macrolides and tetracyclines, immunomodulate and rectify dysregulated inflammatory pathways in ARDS. Doxycycline, a tetracycline antibiotic, acts as a potent Matrix Metalloproteinase (MMP) inhibitor (specifically MMP-8 and MMP-9). Doxycycline functions as a host-modulating drug by chelating the zinc ions required for enzyme activation, thereby reducing excessive extracellular matrix disintegration, neutrophil recruitment, and tissue inflammation in ARDS.[30]

While steroids were administered early alongside standard antibiotics in our patient, an Indian RCT to clarify the role of adjunctive steroids in scrub typhus with pneumonitis/ARDS is underway.[31]

Conclusion

Given its re-emerging trend across geographies, even in areas not strictly endemic, febrile patients with a high index of clinical suspicion of scrub typhus should be instituted early empirical antibiotic treatment—even before laboratory confirmation, to prevent progression to ARDS. Compared to the 2012 Berlin definition, the 2024 revised diagnostic criteria for ARDS improve early detection, enhance inclusivity for non-intubated patients, and permit diagnosis in resource-limited settings.

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