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Case Report

Scrub typhus: Atypical presentation in subhimalayan region

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Introduction

Scrub typhus is caused by *Orientia tsutsugamushi*, which is an obligate, intracellular gram-negative coccobacilli and is distributed throughout the Asia Pacific. Scrub typhus is an acute febrile disease characterised by a eschar, lymphadenopathy, rash, fever, headache, myalgia and cough. Severe complications include encephalitis, interstitial pneumonia, acute respiratory distress syndrome (ARDS), myocarditis and pericarditis, cardiac arrhythmia, acute renal failure, acute hepatic failure and acute hearing loss.¹

Indian army is deployed at various field locations, which are considered pockets of scrub typhus. Scrub typhus has been documented in Indian troops, as early as World war II and during 1965 Indo–Pak war. In 1990, an army unit, deployed at

the Pakistan border of India, documented resurgence of this disease.² Presence of eschar, which is so often documented in western studies, and is considered pathognomic of scrub typhus, is uncommonly seen in Indian patients. Because of non-specific presentation of the disease, absence of eschar and paucity of confirmatory diagnostic tests, scrub typhus is grossly under-diagnosed in India.³ We report two soldiers, posted in hilly terrain of Himachal, who presented with gastrointestinal symptoms progressing to ARDS and eventually diagnosed as scrub typhus sans eschar or rash.

Case 1

A 28-year-old male patient, with no comorbidities, reported from a operational area with 2 days history of pain abdomen

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localised to paraumbilical area. Pain was mild, continuous, associated with small bowel diarrhoea of about 4–5 episodes per day for the past 2 days and recurrent vomiting also of 2 days duration. He had developed dry cough and dyspnoea on exertion since last 12 h, which had progressed to dyspnoea at rest.

On examination, he was febrile, pulse of 100/min regular, respiratory rate of 30/min, no lymphadenopathy and blood pressure of 120/70 mmHg. His tongue was dry and chest examination revealed bibasilar crackles with SpO_2 of 85% in room air. His abdomen was soft with mild tenderness in umbilical area. He had no rash or any eschar.

Investigations revealed haemoglobin of 14.2 gm/dl and leucocyte count of 8700/cu mm. He had transaminitis (AST 81 U/L, ALT 66 U/L); the rest of his biochemical and haematological profiles were normal. Chest radiograph showed bilateral fluffy opacities in all lung fields and $\text{PaO}_2/\text{FiO}_2$ of 140 (Fig. 1). Ultrasound abdomen showed hepatomegaly of 16 cm.

With these findings, a diagnosis of fever with ARDS was made and patient was worked up for various tropical infections. Dengue IgM/IgG were negative. His peripheral blood smear for malarial parasite was negative. Serum Typhi DOT(IgM) was also negative. He had rising weil felix titres and positive IgM scrub typhus. He was started on betalactam antibiotics, doxycycline, continuous positive pressure ventilation (CPAP) and central venous pressure (CVP) guided fluids. He showed good response to treatment and was discharged after 7 days.

Case 2

A 36-year-old male patient was admitted with history of loose watery stools, nausea and diffuse pain in abdomen of 3 days duration. On examination, he had tachycardia (pulse 110/min),

Fig. 1 – Bilateral fluffy opacities.

Fig. 2 – Dilated upper lobar vessels (likely pulmonary veins).

blood pressure 108/70 mmHg and temperature of 100F. His tongue was dry; the rest of the systemic examination was normal. Two days later, he developed rapidly progressing dyspnoea. On examination, he had a pulse of 120/min, respiratory rate of 36/min, blood pressure of 130/90 mmHg and temperature of 101F. Chest examination showed bibasilar crackles. His CVP was 6 cm of water.

Chest radiograph showed dilated apical pulmonary veins (Fig. 2). ECG showed sinus tachycardia, cardiac enzymes (CKMB, Troponin) and brain natriuretic peptide; 2D ECHO was normal.

Investigations showed haemoglobin of 13 gm/dl and leucocyte count of 8000/cumm. His peripheral blood smear for malarial parasite was negative. He had mild transaminitis. Serum lactate dehydrogenase was slightly raised to 470.00 U/L (Normal <248.00). Serum Typhi DOT(IgM) was nonreactive. Dengue IgM/IgG were negative. He had rising weil felix titres and positive IgM scrub typhus. Patient was started on doxycycline and betalactam antibiotics, furosemide infusion and CPAP. He started improving after 24 h of treatment and was discharged after a week.

Discussion

Scrub typhus affects around one million people annually around the world. It is a potentially fatal, mite-borne acute febrile illness caused by *O. tsutsugamushi*.⁴ During world war II, scrub typhus took its toll on the troops fighting in marshy and heavily vegetated areas and caused epidemics in military platoons posted in India and Burma. Trombiculid mite (chigger) of the *Leptotrombidium* genus, which feeds on wild rats, transmits infection to humans and is found in grassy fields and marshy areas around lakes and streams. Infection presents around 7–10 days after the bite.

An eschar is considered the most important clinical finding for the diagnosis of scrub typhus and is formed at the site bitten by trombiculid mites. Prevalence of eschar is highly variable in different regions of the world, ranging from 10 to 92%.⁵

In Indian studies, eschar has been documented variably, ranging from as high as 46% by Vivekanandan et al.⁶ from South India to as less as 9.5% by Mahajan et al.⁷ from North India.

In a study conducted from October to December 2012 in heavy vegetation districts of Dausa and Karauli in Rajasthan, a total of 170 clinically suspected cases were examined and investigated for scrub typhus. 24.7% (42/170) patients were eventually diagnosed to have scrub typhus. Eschar was not present even in a single patient.⁸ Another prospective study performed at SGRR Institute of Medical & Health Sciences, Dehradun, from December 2012 to November 2013, diagnosed a total of 47 patients of scrub typhus. Not even a single patient had a eschar.⁹ Thus in subhimalayan areas, scrub typhus patients rarely have a eschar. Indian troops, because of operational exigencies, are deployed in various subhimalayan field areas, which are considered pockets of scrub typhus; clinicians should not abrogate scrub typhus merely by absence of eschar.

Acute fever is the most common presenting symptom, often associated with breathlessness, cough, myalgia and headache.⁷ About one-third of patients with scrub typhus present with GI symptoms, such as nausea, vomiting, diarrhoea and abdominal pain.¹⁰ Mild transaminitis is the most consistent laboratory finding in scrub typhus, being reported in 90% of cases.¹¹

Our both patients had thrombocytopenia and transaminitis. Severe complications, including ARDS, hepatitis, renal failure, meningoencephalitis and myocarditis with shock, may occur in varying proportions of patients. Approximately, 11% of scrub typhus patients progress to ARDS and mortality rate among these ARDS cases is 25%.¹² Renal dysfunction has been reported in 10–20% of scrub typhus cases.

Recommended treatment of scrub typhus is doxycycline (100 mg twice a day for 7–15 days), azithromycin (500 mg orally for 3 days) or chloramphenicol. The disease can be prevented by use of repellents and by wearing protective clothing during travel to endemic areas.

Scrub vegetations are known to have a strong association with acquisition of the infection. Both our patients were posted in subhimalayan forest area. They presented with gastrointestinal symptoms, did not have an eschar or rash and progressed to respiratory distress. The chances of a missed

diagnosis increase in the absence of any rash or a typical eschar. Hence, while handling cases of pyrexia of unknown origin in subhimalayan areas, clinicians should work-up for scrub typhus even in absence of eschar or rash, as delay may be disastrous.

Conflicts of interest

The authors have none to declare.

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