

CASE REPORT

ATYPICAL PRESENTATION OF RICKETTSIAL SPOTTED FEVER

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Acute febrile illness is a common entity in tropics and often is challenging due a host of pathogenic bacteria, viruses and fungi. Extensive work up is required for better management. Rickettsiosis is uncommon and hence comes lower down in the differentials of multiorgan failure being superseded by the more common diseases as malaria, enteric fever and Dengue. We document a case of young male presenting with high grade fever, multiorgan dysfunction (hepatic, renal, neurological and respiratory involvement), conjunctival suffusion, retiform rash and without lymphadenopathy. The diagnosis was further challenging because the rashes appeared late at 8th day in the course of illness, unlike the typical disease where rashes come on early in day 3–6 of the disease. Patient responded to timely treatment with doxycycline. Thus, a high index of suspicion is needed to diagnose Rickettsiosis in geographical areas apparently free of the disease.

Keywords: Rickettsia; Weil-Felix; Purpura fulminans; Multiorgan dysfunction; Rash

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INTRODUCTION

Rickettsial fever is an important differential diagnosis for pyrexia of unknown origin (PUO) since it is mostly treatable and has a good outcome. In India, Rickettsial disease is more commonly reported from South, the North east and the Himalayan belt.¹

The National centre for disease control (NCDC), New Delhi, conducted a sero-survey during a 4 year period (2005–2009), in which only 29 patients were found to be seropositive by Weil Felix test.² Among those that were positive, the most common variety was scrub typhus followed by Indian Tick Typhus (*R. conorii* subspecies indica).² Spotted fever, in itself, is a large subgroup, and most of the diseases in this category are transmitted by Ticks. The classic triad of fever, rash, and history of tick bite is seen in spotted fever, although many of the patients fail to give an account of tick bite. If untreated, mortality rate may be as high as 25%.³

CASE

A 17-year young male presented in the emergency room with total duration of illness was 14 days. Illness began with high grade continuous fever (recorded up to 104° F) that was partially responsive to antipyretics. It was accompanied by headache and body ache. After 4 days of illness, patient developed mild facial and pedal oedema. Two days later, patient had one episode of generalized tonic colonic seizure followed by altered sensorium in form of decreased responsiveness.

He was evaluated by a general practitioner before coming to us who attributed these symptoms to malaria, and started antimalarial (Artesunate) empirically. However, card test and peripheral blood picture for malarial parasite were negative for malaria. On 8th day of illness, the skin lesion started as small purpura which coalesced to form large retiform purpura. One day later, patient also had one episode of malena. There was no history of cough, ear discharge, and jaundice or neck

pain. Meanwhile renal function had started deteriorating with rising creatinine without decreased urine output and patient was managed with two sessions of haemodialysis.

His renal function improved, however there was hardly any improvement in the sensorium of the patient. At this juncture patient developed respiratory distress and was referred to our centre. His past medical history was insignificant. On examination patient was febrile (102.4° F) with a pulse of 94/min regular, blood pressure of 104/60 mmHg, and respiratory rate of 36/min. General exam revealed mild pallor, left conjunctival suffusion, and confluent, retiform rash on both upper and lower limbs sparing palms and soles. (Figure-1). Eschar and lymphadenopathy was absent. Glasgow coma scale (GCS) was 6 (E1V1M4). Neck rigidity and Kernig's sign were absent. Bilateral Pupils were normally reactive to light. Fundus examination failed to reveal any optic disc oedema (Figure-1). Bilateral end inspiratory crepts were present. There was no hepatosplenomegaly. Patient was shifted to intensive care and was put on invasive ventilator owing to poor GCS with respiratory distress. Patient was started on intravenous antibiotics meropenem and teicoplanin while awaiting laboratory results. Laboratory parameters showed mild leucocytosis (TLC=10,700 with 74% neutrophils), a slightly low haemoglobin (10.3 gm/ dl), normal platelet count, deranged transaminases (ALT=272, AST=232), raised LDH (2094), normal renal parameters (Cr=01, urea=80) and a normal blood sugar. Serology for acute viral markers and HIV were negative. CSF picture was normal. Serological testing for other infectious aetiologies including leptospirosis and typhoid fever, was also carried out which were negative. Blood culture and urine culture was sent that later turned out to be normal. Patient was also investigated for a possible vasculitic aetiology, including ANA and Anti ds-DNA which were also normal. INR was normal and APTT was slightly raised [40 (range 23–35)]. D-dimer, procalcitonin and FDP

(fibrin degradation product) were raised giving us sufficient evidence that sepsis was present.

Further inquest into history led us to the conclusion that there may possibly have been a contact/exposure to rats and dogs. Doxycycline was added empirically and Weil-Felix test was requested. OX 19 and OX 2 were present in high titers (1:320 and 1:640 respectively). Patient's sensorium as well as respiratory distress improved gradually within 3 days of starting doxycycline therapy and patient was subsequently extubated. Doxycycline was continued for a total of 14 days. A diagnosis of Rickettsial spotted fever with sepsis with multiorgan dysfunction was kept finally.



Figure-1: Conjunctival suffusion, optic disc showing no signs of papilledema, retiform rash involving both lower and upper limbs

DISCUSSION

Rickettsiosis is an uncommon disease in India. Most of the cases that have been documented, used clinical suspicion, non-specific serological testing (such as Weil-Felix), and good response to doxycycline as the means to identify rickettsiosis.⁴ For sub species identification Polymerase chain reaction(PCR) is required.

Rickettsia spp. is classically divided into the typhus group and spotted fever group. Spotted fever group is less common among the two in this part of subcontinent.¹ The disease is more common in rural locations and is characterized by sudden onset of moderate to high grade fever, malaise, deep muscle pain, headache, and conjunctival suffusion.³ Lymphadenopathy and Eschar are usually absent in spotted fever; while they are more commonly present in typhus group. The disease may follow a milder course or may prove to be fatal if timely therapy is not instituted. Laboratory abnormalities

may include mild leucocytosis, anaemia, and thrombocytopenia, Disseminated intravascular coagulation (DIC) in severe ill patients, elevated aminotransferases, azotaemia and elevated prothrombin time (PT) and Activated Partial Thromboplastin Time (APTT).³ Weil-Felix test has got a low sensitivity and specificity for diagnosing Rickettsiosis. Either a four-fold rise in agglutinin titer in paired sera or single titer of more than equal to 1:320 is considered diagnostic for infection.⁵ Serologic tests are usually negative at the time of presentation for medical care. The only test that is useful during acute illness is immune-histologic examination of a cutaneous biopsy sample from a rash lesion for *Rickettsia rickettsii* with a sensitivity of 70% and specificity of 100%.³ Indirect immunofluorescence, indirect hemagglutination, complement fixation and PCR are some of the more sensitive and specific tests for the identification of rickettsia but are not widely available in resource poor setting. In our patient, who presented with fever multiorgan dysfunction and rash, we initially suspected Thrombotic thrombocytopenic purpura (TTP) clinically, however platelets were normal and patient improved without plasmapheresis as TTP has a mortality rate of around 95% without plasmapheresis. After ruling out other infectious causes which may cause fever with rash, rickettsiosis was suspected. Such high titer of OX2 (1:640) was certainly diagnostic. Unfortunately, we could not delineate the subspecies in our case due to lack of PCR in our setting. Patient had an uneventful recovery and was discharged.

A high index of suspicion is needed to diagnose rickettsiosis particularly with atypical rash and multiorgan dysfunction. Finally, treatment must be instituted timely to prevent poor outcome.

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REFERENCES

1. Dasari V, Kaur P, Murhekar MV. Rickettsial disease outbreaks in India: A review. *Ann Trop Med Public Health* 2014;7(6):249–54.
2. Mittal V, Gupta N, Bhattacharya D, Kumar K, Ichhpujani RL, Singh S, *et al.* Serological evidence of rickettsial infections in Delhi. *Indian J Med Res* 2012;135(4):538–41.
3. Kasper D, Fauci A, Hauser S, Longo D, Jameson J, Loscalzo J. Harrison's principles of internal medicine, 19e. USA 2015. 2015.
4. Murali N, Pillai S, Cherian T, Raghupathy P, Padmini V, Mathai E. Rickettsial infection in South India-How to spot the spotted fever. *Indian Pediatr* 2001;38(12):1393–6.
5. Kularatne SA, Gawarammana IB. Validity of the Weil-Felix test in the diagnosis of acute rickettsial infections in Sri Lanka. *Trans R Soc Trop Med Hyg* 2009;103(4):423–4.

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