# CASE REPORT

## **MYOCARDITIS AND RICKETTSIA CONORII INFECTION**

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#### Abstract

Myocarditis and pericarditis are rare complications of human rickettsiosis, usually seen in the setting of an acute disseminated infection of rickettsia rickettsii and conorii. Clinical laboratory findings such as thrombocytopenia (particularly with Indian spotted fever), normal or low white cell counts, mild to moderate serum elevations of hepatic aminotransferases, and hyponatremia suggest some common pathophysiological mechanisms. In endemic areas patients presenting with fever, rash or a skin lesion consisting of a black necrotic area or a crust surrounded by an erythema should be considered to have one of the rickettsial spotted fevers. We from a developing country report a rare case of rickettsia conorii myocarditis, its serological diagnosis and management.

**Key Words:** Rickettsia Conorii, Myocarditis, Pericarditis, Indian Tick Typhus, Inoculation Eschar, Boutonneuse Fever.

### Introduction

Rickettsiosis consists of a spectrum of vector borne diseases caused by small gram negative obligate intracellular bacteria and this includes common illness like epidemic typhus, scrub typhus and spotted fever. Howard Taylor Rickets was the person who successfully observed the organisms in tick tissues in the early 20th century. The important complication of rickettsial fever includes encephalitis, gangrene, pneumonia/ARDS, myocarditis, DIC, hepatitis and renal failure. We here report a serologically documented case of rickettsial myocarditis in a child.

## **Case Report**

One year old child was referred to our hospital with history of abrupt onset of generalised rash, fever and irritability of 5 days duration. General physical examination revealed febrile child with generalised erythematous maculonodular rash. There was no inoculation eschar noted. Pallor was noted. Vital parameters were pulse rate of 150/ minute, respiratory rate was 66/minute, weight-9 kg. Systemic examination of abdomen revealed tender hepatomegaly and splenomegaly. On auscultation child had bilateral basal crepetations. There was no cyanosis noted. On cardiovascular examination child had gallop. Both respiratory and cardiac findings which were suggestive of congestive cardiac failure were further supported by cardiomegaly on chest radiograph. Central nervous system examination in detail did not reveal any deficits. Routine blood testing revealed haemoglobin 7 gm%, total white cell count of 25300/cumm, with neutrophils of 58%, lymphocytes of 32% and platelet count of 96,000/cumm. Blood sent for Widal and Weil-Felix was negative. One blood sample was kept apart for future testing. Child was empirically started on injection ceftriaxone 500 mg intravenously every 12 hourly and doxycycline 10 mg twice on first day followed by 10 mg once a day for seven days along with injection furosemide 1 mg/ kg three times a day. Echocardiography was done, which revealed dilated left ventricle with mild mitral regurgitation and tricuspid regurgitation with ejection fraction of 25%-30%. There was no pericardial effusion noted. Blood samples, one which was kept aside on the day of admission and one on day 10 were sent to CDC (Centre For Disease Control & Prevention USA.) for indirect immunoflourescence assay which showed positive Rickettsia conorii IgG (RCO - IgG [> 512 on Day 1 and > 8192 on Day 10 (Antibody titers > or = 64 suggest exposure to or infection with R Conorii or antigenically related spotted fever group rickettsiae). Rickettsia typhi IgG were negative (<32). There was gradual but dramatic improvement in patient's condition with the above mentioned antibiotics and he was discharged on day 15.

## **Discussion**

Rickettsiosis is acute febrile, arthropod-borne diseases caused by obligate intracellular bacteria, and are classified into three major bio-groups: (1) Scrub typhus -caused by Orientia tsutsugamushi, widely endemic in Asia, (2) Spotted fever- caused by R. rickettsii, R. conorii, R. africae, and R. japonica, and (3) Murine/Epidemic typhus-caused by Rickettsia typhi and R. prowazekii.(1-5)

These infections are endemic, some of them are emerging, and they are important cause of fever in certain populations and travelers. (6-8) The disease spectrum of rickettsiosis is wide. In some patients, the disease is mild; however, other patients develop serious complications and fatalities are not uncommonly reported (up to 35% with scrub typhus). (1-3) High infectivity and severe illness after inhalation makes R. prowazekii, R. typhi, R. rickettsii, R. conorii and Coxiella burnetti bioterriosm threats. Establishing the etiological diagnosis is very difficult during the acute stage of illness & definitive diagnosis usually requires examination of paired serum samples after convalescence and heightened clinical suspicion.(9) Serologic response is usually undetectable during early illness and molecular testing by polymerase chain reaction (PCR) is generally unavailable.(1, 3, 10)

R. conorii is prevalent in southern Europe, Africa and south-western & south-central Asia. Regional name of R conorii prevalent in south-western and south-central Asia is Indian tick typhus, Mediterranean spotted fever. Transmission occurs after bite of brown dog tick R. Sanguineus or other tick species. House hold dog is a potential vehicle for transmission. The disease is characterised by high fever, rash and in most geographic locales - an inoculation eschar at the site of tick bite (9). A severe form of disease with 50% mortality occurs in patients with diabetes, alcoholism, or heart failure.

The presence of Rickettsial diseases in India has been from several parts of India (10) but myocarditis

as presentation is rare. Clinical feature of Indian tick-typhus is usually a history of recent tick-bite and a careful examination reveals a lesion or eschar at the site of bite. After an interval of 2 to 3 weeks, malaise and headache occurs. A maculopapular rash appears on the third day. Unlike the rash in other rickettsial diseases, the rash appears first on extremities-ankles and wrist, moves centripetally and involve the rest of the body. The clinical features may be confused with atypical measles.

It has been demonstrated by multivariate analyses that delayed doxycycline therapy is independently associated with increased risk of major organ dysfunction and protracted illness (prolonged hospitalization) after adjustment for rickettsia biogroup and baseline characteristics.(11) This finding is supported by earlier (univariate analysis) observations that late treatment of rickettsial infection - 5 days from disease onset is associated with higher risk of progressive clinical deterioration and death.(4, 11-12) Death occurs in 1.4% to 5.6% of cases.

Successful therapeutic agents for treatment include doxycycline, tetracycline, ciprofloxacin and chloramphenicol. Pregnant patients should be treated with Josamycin (3 gram/day for 5 days). (9)

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