



Fulminant Myocarditis from Murine Typhus: A Case Misled by Positive *Mycoplasma* Serology

Narut Chancharussin

Division of Infectious Diseases and Tropical Medicine,
Department of Medicine, Faculty of Medicine Siriraj Hospital,
Mahidol University, Bangkok, Thailand

ABSTRACT

Background: Acute myocarditis is a life-threatening condition caused by various infectious agents. Reliance on single serologic test results without considering the clinical course may lead to diagnostic errors.

Methods: We report the clinical course, diagnostic evaluation, and management of a young woman presenting with myocarditis at a super tertiary care hospital in Thailand.

Results: A previously healthy 20-year-old woman presented with a 1-day history of acute febrile illness followed by syncope. Initial manifestations included fever, rhinorrhea, and diarrhea, with progression to syncope. Electrocardiography demonstrated conduction abnormalities evolving from sinus bradycardia to complete heart block with hypotension, consistent with fulminant myocarditis. Cardiac biomarkers were markedly elevated, while transthoracic echocardiography revealed mildly reduced left ventricular systolic function without pericardial effusion. Initial evaluation revealed a positive *Mycoplasma pneumoniae* antibody titer of 1:80 early in the illness, leading to a preliminary diagnosis of *Mycoplasma*-associated myocarditis and initiation of macrolide therapy. However, the absence of respiratory manifestations and a subsequent decline in *Mycoplasma* antibody titer to 1:40 during convalescence suggested a non-causative association. Paired serologic testing by indirect immunofluorescence assay demonstrated seroconversion for *Rickettsia typhi*, with titers rising from <1:50 in the early phase to 1:200 during convalescence, confirming murine typhus-associated myocarditis. The patient required temporary transvenous pacing and high-dose intravenous methylprednisolone, with gradual recovery of atrioventricular conduction.

Conclusion: Murine typhus should be recognized as a cause of fulminant myocarditis. Accurate interpretation of serologic testing must be anchored to the illness timeline and paired sera, as isolated positive *Mycoplasma* antibodies may be misleading and result in inappropriate attribution of disease etiology in endemic areas.

PATIENT HISTORY

A 20-year-old Thai woman living in Salaya Subdistrict, Nakhon Pathom Province, Thailand.



Day 0

Fever of 38.5°C, with myalgia, rhinorrhea, and sore throat

Day 1

She developed three episodes of watery diarrhea. After getting up from bed to go to the bathroom, she experienced dizziness followed by a syncopal episode. She then went to a nearby hospital.

Physical examination:

- Vital signs: T 36.5°C, PR 65 BPM, BP 95/55 mmHg, RR 20/min, SpO2 98% on room air
- GA: alert and oriented
- CVS: normal S1, S2, no murmur

Investigations: Troponin-T = 1,697 ng/L

EKG:

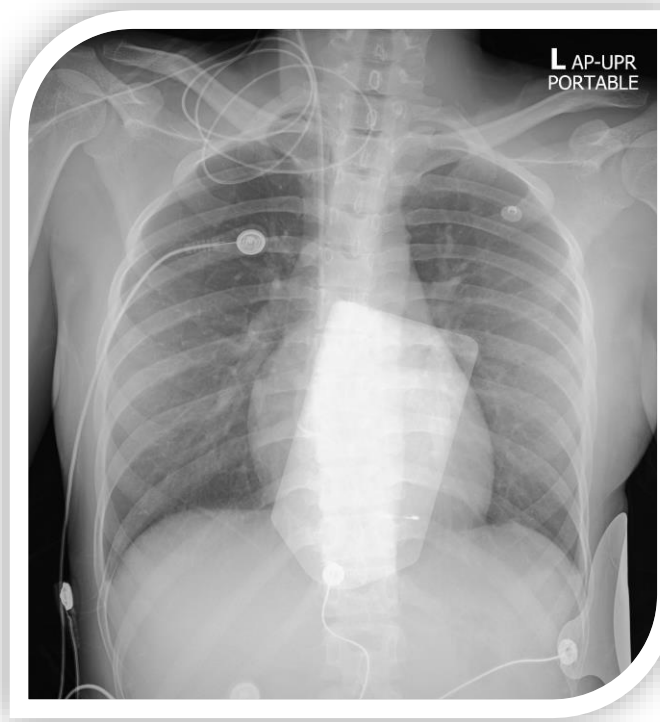
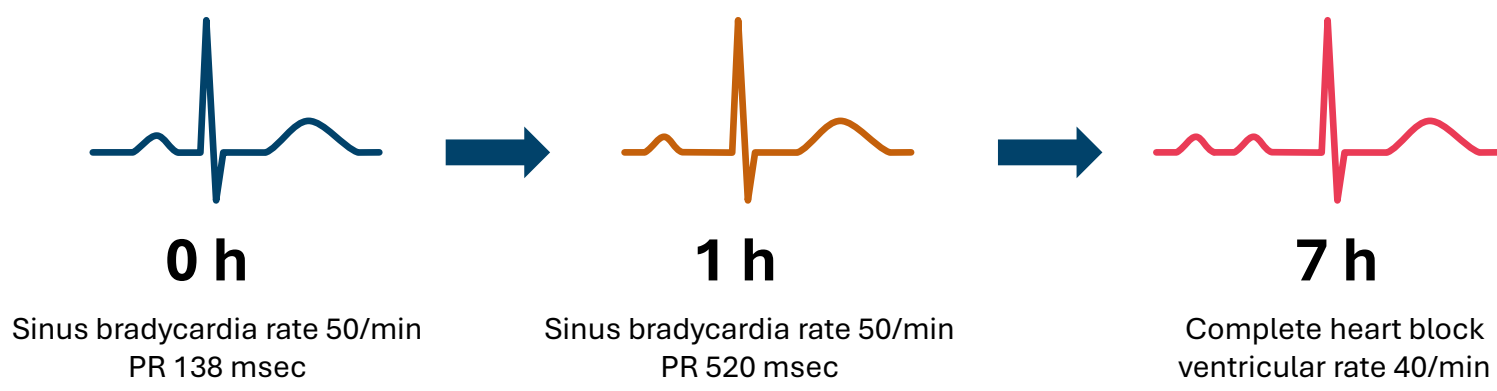


Figure 1. The patient's chest x-ray at the first day of admission

Transthoracic echocardiography: LVEF 50%, and global left ventricular hypokinesia

Provisional diagnosis:

Fulminant myocarditis with complete heart block

MANAGEMENT



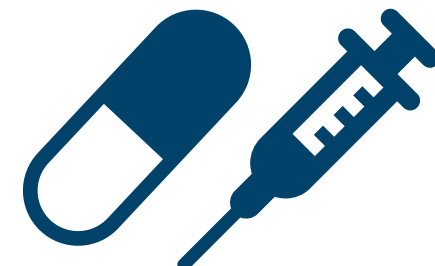
Hemodynamic management

- Transcutaneous pace maker
- Atropine 0.6 mg IV
- Continuous monitoring in the cardiac care unit (CCU)



Immunotherapy

- Methylprednisolone IV 1 g/day x 3 days



Antibiotics

- Azithromycin 500 mg IV
- Ceftriaxone 2 g IV
- Ivermectin 12 mg oral

Figure 2. Summary of the patient's management during admission.

INVESTIGATIONS

Serology test

Table 1. Serologic results for *Mycoplasma pneumoniae*, *Rickettsia typhi* and *Orientia tsutsugamushi* during admission (day 4) and after 14 days (day 18).

Pathogen	Day 4	Day 18	Interpretation
<i>M. pneumoniae</i> Ab titer	1:80	1:40	False positive
<i>R. typhi</i> IgG titer (IFA)	1:50	1:200	Seroconversion
<i>R. typhi</i> IgM titer (IFA)	<1:50	<1:50	Negative
<i>O. tsutsugamushi</i> IgG titer (IFA)	<1:50	<1:50	Negative
<i>O. tsutsugamushi</i> IgM titer (IFA)	<1:50	<1:50	Negative

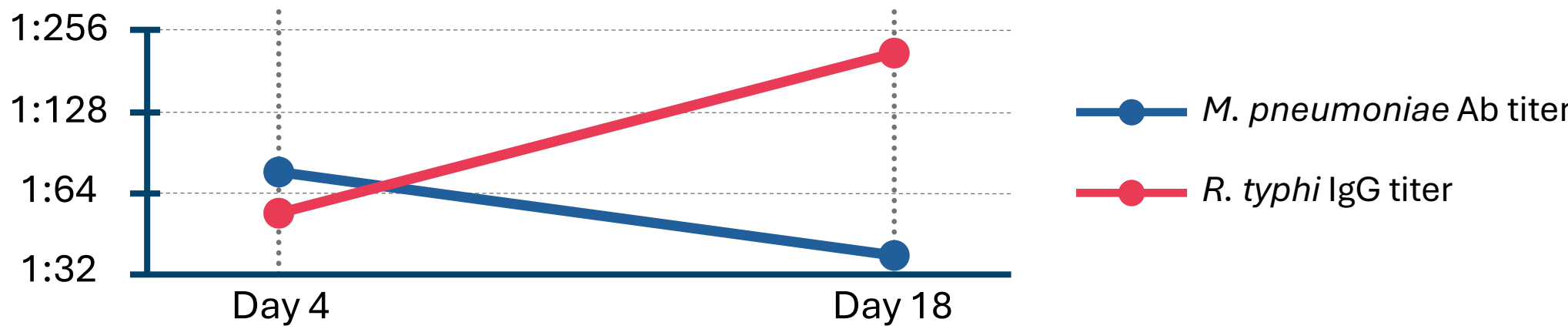


Figure 3. A linear graph demonstrating a dynamic changes in *M. pneumoniae* antibody titer (blue line) and *R. typhi* IgG titer (red line) during admission (day 4) and after 14 days (day 18).

Other tests



Dengue NS1Ag/IgG/IgM

Negative



Anti-HIV Ab, HBsAg, Anti-HCV Ab

Negative



Blood for bacterial culture

Negative



Blood for Enterovirus 71 RNA PCR

Negative



Respiratory viral panel

Negative



Next-generation sequencing

Negative

OUTCOMES

- Serial EKG showed recovery of intrinsic rhythm → temporary pacemaker removed by day 4
- Echocardiography: LVEF ↑50%, no structural or pericardial abnormalities
- Stable rhythm during admission with only occasional premature ventricular conduction (PVCs), no recurrent high-grade AV block
- Discharged in stable condition

DISCUSSION & KEY LEARNING POINTS

- Murine typhus (*Rickettsia typhi*) can present with fulminant myocarditis and complete heart block.
- Positive *Mycoplasma pneumoniae* serology early in the illness may be incidental; paired sera are essential for accurate interpretation
- Clinical context, exposure history, and illness timeline are critical to avoid being misled by laboratory results.

CONCLUSION

- Murine typhus should be recognized as a cause of fulminant myocarditis.
- Accurate interpretation of serologic testing must be anchored to the illness timeline and paired sera, as isolated positive *Mycoplasma* antibodies may be misleading and result in inappropriate attribution of disease etiology in endemic areas.

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- Toy D, Vo C, Kwan WC, Shavelle DM. Fulminant myocarditis secondary to murine typhus mimicking acute coronary syndrome. *J Cardiol Cases*. 2021 Mar 4;24(3):99-101.
- Madan R, Muthukumar V, Premji S, Khan R, Schmidt RM. Murine Typhus-Induced Myocarditis. *Am J Med*. 2022 Oct;135(10):e397-e398.

