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Address for correspondence: Erhard van der Vries, Department of Infectious Diseases & Immunology, Faculty of Veterinary Medicine, Section Virology, Utrecht University, Utrecht, the Netherlands; email: e.vandervries@uu.nl

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## Acute Myopericarditis Associated with Tickborne *Rickettsia sibirica mongolitimonae*

Pablo Revilla-Martí, Álvaro Cecilio-Irazola, Jara Gayán-Ordás, Isabel Sanjoaquin-Conde, Jose Antonio Linares-Vicente, José A. Oteo

Author affiliations: Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain (P. Revilla-Martí, A. Cecilio-Irazola, J. Gayán-Ordás, I. Sanjoaquin-Conde, J.A. Linares-Vicente); Centro de Investigación Biomédica de La Rioja, Logroño, Spain (J.A. Oteo)

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We report an unusual case of myopericarditis caused by *Rickettsia sibirica mongolitimonae*. Because of increasing reports of *Rickettsia* spp. as etiologic agents of acute myopericarditis and the ease and success with which it was treated in the patient reported here, rickettsial infection should be included in the differential diagnosis for myopericarditis.

Myopericarditis is a primarily pericardial inflammatory syndrome occurring when clinical diagnostic criteria for pericarditis are satisfied and concurrent mild myocardial involvement is documented by elevated biomarkers of myocardial damage (i.e., increased troponins). Limited clinical data on the causes of myopericarditis suggest that viral infections are among the most common causes in developed countries, although the list of agents is increasing. We identified an unusual case of myopericarditis caused by *Rickettsia sibirica mongolitimonae*, an emerging pathogen in southern Europe with a broad clinical spectrum (1).

In September 2016, a 39-year-old man with no remarkable medical history sought care at an emergency department in Spain with acute-onset central chest pain and fever. The previous week, he had hunted in northeastern Spain. Physical examination revealed a systolic blood pressure of 115 mm Hg, heart rate 80 beats/min, peripheral pulse oximetry of 98%, and an axillary temperature of 38.7°C. No murmurs, rales, or gallops were detected on cardiac examination. A necrotic left gluteus eschar and multiple enlarged left inguinal lymph nodes were noted. He had neither lymphangitis nor widespread rash, and his mucous membranes appeared normal. He did not remember tick bites.

An electrocardiogram demonstrated a sinus rhythm with diffuse ST-segment elevation, and a transthoracic echocardiogram showed a normal biventricular ejection fraction with mild pericardial effusion. High-sensitive T troponin level was 575.3 ng/L (reference <14 ng/L), and blood cultures and serologic tests for common viruses were all negative. He was admitted to the hospital, and a cardiac magnetic resonance study performed 48 hours later confirmed the suspected diagnosis of myopericarditis.

Because of the eschar, tickborne-related rickettsiosis was suspected, and ibuprofen (1,800 mg/d) and doxycycline (100 mg every 12 h) were started. After the third day on medical therapy, the patient became afebrile, and the electrocardiographic changes gradually resolved. He was discharged after 12 days. Doxycycline was maintained for 14 days.

Acute-phase serologic tests yielded negative results for HIV; *Borrelia burgdorferi* sensu lato (chemiluminescence immunoassay, Liason, Diasorin, Spain); spotted fever group rickettsia (SFGR) (commercial [Focus Diagnostics, Cypress, CA, USA] and in-house tests); and *Francisella tularensis* (in-house microagglutination assay). An eschar swab sample and an eschar biopsy sample were removed under aseptic

**Table.** Characteristics of adults previously reported with myopericarditis associated with *Rickettsia* spp. infection\*

Characteristic	Patient 1 (2)	Patient 2 (3)	Patient 3 (4)	Patient 4 (5)	Patient 5 (6)	Patient 6 (7)	Patient 7 (8)	Patient 8 (9)
Age, y/sex	28/M	52/F	74/F	35/M	25/F	54/M	Unk/unk	26/M
Country	Spain	Australia	South Africa	South Africa	United States	United States	Sri Lanka	United States
Signs and symptoms								
Fever	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rash	No	Yes	Yes	Yes	Yes	No	Unk	Yes
Adenopathy	Yes	No	Yes	No	Unk	Unk	Unk	Unk
Lymphangitis	Yes	No	Yes	Yes	Unk	Unk	Unk	Unk
Headache	Yes	Yes	Yes	No	Unk	Yes	Unk	Unk
Myalgia	No	Yes	No	No	Unk	Yes	Unk	Unk
Chest pain	Yes	Yes	Yes	Yes	Unk	Yes	Unk	Unk
Heart failure	No	Yes	Yes	No	Unk	No	Unk	Unk
Eschar	Yes; neck	No	Yes; unk	Yes;	Unk	Unk	No	No
				abdomen				
Organism	<i>R. slovaca</i>	<i>R. australis</i>	<i>R. africae</i>	<i>R. africae</i>	<i>R. rickettsii</i>	<i>R. rickettsii</i>	<i>R. conorii</i>	<i>R. rickettsii</i>
LVD	No	Yes	Unk	No	Yes	Yes	Unk	Yes
PE	No	No	Unk	No	Yes	No	Unk	No
Treatment	Doxycycline	Tigecycline, doxycycline	Doxycycline	Doxycycline	Unk	Doxycycline	Doxycycline	Doxycycline

\*LVD, left ventricular dysfunction; PE, pericardial effusion; unk, unknown.

conditions and sent together with EDTA-treated blood and serum specimens to Spain's reference center for rickettsioses (Hospital San Pedro—Centro de Investigación Biomédica de La Rioja, Logroño, Spain) for molecular analysis. Samples were tested by PCR for the presence of *Rickettsia* spp. (*ompB*, *ompA*, and *sca 4* genes). Fragments of *ompB* rickettsial genes (285/285 bp) were amplified from the eschar biopsy and swab. The sequences obtained showed 99.8% identity to the corresponding sequences of *R. sibirica mongolitimonae* (GenBank accession no. AF123715).

A convalescent-phase serum specimen collected 7 weeks after hospital discharge was tested by indirect immunofluorescence assay for IgG against SFGR. Commercial (Focus Diagnostics) and in-house *R. conorii* and *R. slovaca* antibody testing showed an IgG of 1:1,024. In-house microagglutination assay results for *F. tularensis* were not reactive.

Myopericarditis, a rare complication of human rickettsiosis, usually occurs with acute infection caused by *R. rickettsii* or *R. conorii*. To our knowledge, there are few reports of a myopericarditis due to SFGR infections (Table) (2–9), and in PubMed, we found none attributed to *R. sibirica mongolitimonae*.

*R. sibirica mongolitimonae* is an intracellular bacterium that was first reported as a human pathogen in 1996; since then, several cases have been reported from France, Portugal, Greece, and Spain showing seasonal variations with predominance during spring and summer (1). Clinical manifestations include fever with or without rash, myalgia, and headache. A characteristic rope-like lymphangitis from the eschar to the draining lymph node is evident in one third of patients (1).

Rickettsiosis is commonly diagnosed on the basis of serologic testing, although use of molecular tools or cell culture on a skin biopsy specimen from an eschar is one of the best methods to identify *Rickettsia* spp. Swabbing an

eschar is painless, and its results are similar to skin biopsy samples by molecular tools. In the patient we reported, the swab sample from the eschar was useful for rickettsial diagnosis (10). Negative test results for other agents and the clinical response to doxycycline strongly supported the diagnosis of acute myopericarditis associated with *R. sibirica mongolitimonae*. Because of increasing reports of different species of *Rickettsia* involved as etiologic agents of acute myopericarditis and the ease and success with which this infection was treated, we strongly recommend including rickettsial infection in the differential diagnosis in the adequate epidemiology context.

Dr. Revilla-Martí is a cardiologist at Hospital Clínico Universitario Lozano Blesa in Zaragoza, Spain. His research interests include heart failure and myocardial diseases.

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Address for correspondence: Pablo Revilla-Martí, Hospital Clínico Universitario Lozano Blesa, Avenida San Juan Bosco 15, Zaragoza 50009, Spain; email: pablorevillamarti@gmail.com

## Enteropathogenic *Escherichia coli* O80:H2 in Young Calves with Diarrhea, Belgium

Damien Thiry, Marc Saulmont, Shino Takaki, Klara De Rauw, Jean-Noël Duprez, Atsushi Iguchi, Denis Piérard, Jacques G. Mainil

Author affiliations: University of Liège, Liège, Belgium (D. Thiry, S. Takaki, J.-N. Duprez, J.G. Mainil); Association Régionale de Santé et d'Identification Animales, Ciney, Belgium (M. Saulmont); Universitair Ziekenhuis Brussel, Brussels, Belgium (K. De Rauw, D. Piérard); University of Miyazaki, Miyazaki, Japan (S. Takaki, A. Iguchi)

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Serogroup O80 was detected in 40% of 104 enteropathogenic *Escherichia coli* isolates from calves with diarrhea from 42 farms in Belgium during 2008–2015. These isolates harbored the *eae-ξ* and *fliC<sub>H2</sub>* genes, similar to the O80 attaching-effacing Shigatoxigenic *E. coli* isolates found in humans in France. This strain might be emerging.

Enteropathogenic and attaching-effacing Shigatoxigenic *Escherichia coli* (EPEC and AE-STECS) cause bloody diarrhea in humans and young calves. For clarity,

we use the term AE-STECS instead of enterohemorrhagic *E. coli*, similar to a previous publication (1), to refer to STECS isolates from animals that produce attaching-effacing lesions. EPEC and AE-STECS that infect humans are diverse and comprise scores of serotypes (2); in contrast, most calf AE-STECS strains comprise a few serotypes, mostly O5:H-, O26:H11, O111:H-, and O118:H16 (3). The O26:H11 serotype is also the most common among calf EPEC. However, most serotypes that infect calves have not been identified (3). Therefore, during November 2008–June 2015, we conducted a study on 104 EPEC and 153 AE-STECS isolates collected from the feces or the intestinal contents of calves suffering diarrhea (1 isolate/calf) at the Association Régionale de Santé et d'Identification Animales in Ciney, Belgium. Isolates were screened by PCR for genes of the 10 most pathogenic and common calf and human O serogroups: O5, O26, O103, O104, O111, O118, O121, O145, O157, and O165. Confirming published results (3), 80% (122/153) of AE-STECS isolates and only 21% (22/104) of EPEC isolates tested positive for 1 of these (J.G. Mainil, unpub. data) (4). We sought to further characterize this collection of calf EPEC with unidentified O serogroups.

We submitted 9 calf EPECs with unidentified serogroups to the O-typing multiplex PCR platform (5); 6 of 9 EPEC isolates contained the O80 serogroup–encoding gene, and 3 belonged to 3 other O serogroups. We subsequently performed an O80 serogroup–specific PCR (5) of all 31 AE-STECS and 82 EPEC isolates with unidentified serogroups, along with one O80-positive *E. coli* strain and negative controls; 42 EPEC isolates and the O80-positive *E. coli* strain but no AE-STECS isolates or negative controls tested positive.

We further tested the calf EPEC isolates and 3 human Shiga toxin 2–encoding gene (*stx2*)–positive AE-STECS O80 isolates from the STECS National Reference Center (Brussels, Belgium) by PCR for *fliC<sub>H2</sub>* and *eae-ξ* genes found in human AE-STECS O80 strains. For amplifying *eae-ξ*, we used previously published PCR conditions (6), and for amplifying *fliC<sub>H2</sub>*, we used primers H2\_F (5'-TGATCCGACATCTCCTGATG-3') and H2\_R (5'-CCGTCATCACCAATCAACGC-3') and the following thermocycler conditions: initial denaturation at 94°C for 1 min; 30 cycles of denaturation for 30 s at 94°C, annealing for 30 s at 58°C, and elongation for 1 min at 72°C; and final elongation at 72°C for 2 min. All 42 calf EPEC and 3 human AE-STECS isolates tested positive by both PCRS.

Among the 104 calf EPEC isolates, O80:H2 was frequently found (40% were PCR positive) and, thus, could be considered emerging. Indeed, the EPEC O80 isolates were isolated from calves from 42 farms. The yearly EPEC O80:H2 isolation rate varied from 12% in 2009 to 40%–50% during 2010–2013 to as high as 73% for the