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 negative. Physical examination revealed a left ventricular ejection fraction 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 (chemiluminiscence immunoassay, Liaison, 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
To our knowledge, there are few
is an intracellular bacte-
the best methods to identify
culture on a skin biopsy specimen from an eschar is one of
serologic testing, although use of molecular tools or cell
culture on a skin biopsy specimen from an eschar is one of
one third of patients (10). Negative test results for other agents and the
clinical response to doxycycline strongly supported the di-
agnosis 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 ad-
quate epidemiology context.

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

**References**

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   http://dx.doi.org/10.1111/imj.12184

   http://dx.doi.org/10.1086/589868

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   http://dx.doi.org/10.1186/1471-2334-5-90


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

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<td>Age, y/sex</td>
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<td>52/F</td>
<td>74/F</td>
<td>35/M</td>
<td>25/F</td>
<td>54/M</td>
<td>Unk/unk</td>
<td>26/M</td>
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<td>Yes; abdomen</td>
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<td>Yes; abdomen</td>
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<td>Unk</td>
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<td>Yes</td>
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<td>Tigecycline</td>
<td>Doxycycline</td>
<td>Doxycycline</td>
<td>Unk</td>
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<td>Doxycycline</td>
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<tr>
<td><em>LVD</em>, left ventricular dysfunction; PE, pericardial effusion; unk, unknown.</td>
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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**

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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*~H2~ genes, similar to the O80 attaching-effacing Shigatoxigenic *E. coli* isolates found in humans in France. This strain might be emerging.

E nteropathogenic and attaching-effacing Shigatoxigenic *Escherichia coli* (EPEC and AE-STEC) cause bloody diarrhea in humans and young calves. For clarity, we use the term AE-STEC instead of enterohemorrhagic *E. coli*, similar to a previous publication (1), to refer to STEC isolates from animals that produce attaching-effacing lesions. EPEC and AE-STEC that infect humans are diverse and comprise scores of serotypes (2); in contrast, most calf AE-STEC 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-STEC 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-STEC 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-STEC 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-STEC isolates or negative controls tested positive.

We further tested the calf EPEC isolates and 3 human Shiga toxin 2–encoding gene (*stx2*)–positive AE-STEC O80 isolates from the STEC National Reference Center (Brussels, Belgium) by PCR for *flIC*~H2~ and *eae*-ξ genes found in human AE-STEC O80 strains. For amplifying *eae*-ξ, we used previously published PCR conditions (6), and for amplifying *flIC*~H2~, we used primers H2_F (5’-TGATCCGACATCTCTGAGT-3’) and H2_R (5’-CCGTATCCACCATAAAGC-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-STEC 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