such as serologic or molecular methods, are not available in Ecuador, and have been seldom used there, for research purposes (5,8). Moreover, serologic assays have been implemented mostly for other species, such as *P. westermani* and *P. kellicotti* worms (8,9), and clinical validation for *P. mexicanus* worms is lacking (9,10). Limited access to healthcare services in some remote communities can further cause late diagnosis.

Control strategies to limit human infection are hampered by the wide presence of the parasite in many domestic and wild mammals, and the complex life cycle involving 2 intermediate hosts (snail and crustacean) (5). Thus, health education on proper food preparation is the main intervention to reduce infections (1).

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References

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**Haematospirillum jordaniae**

**Cellulitis and Bacteremia**

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We isolated *Haematospirillum jordaniae* from a positive blood culture from a 57-year-old man in Slovenia who had bacteremia and bullous cellulitis of lower extremities. The infection was successfully treated with ciprofloxacin. Our findings signal the need for increased awareness about the clinical course of *H. jordaniae* and its potential effects as a human pathogen.
A 57-year-old man living near Lendava, Slovenia, with a medical history of type 2 diabetes, varicose veins in his legs, obesity, and arterial hypertension, sought treatment for a 1-day history of bilateral swelling, redness, warmth, and pain in his lower extremities. The day before, he had pricked himself on his left shin and the sole of his right foot with a reed in the Pacsa Fishing Lake in Hungary. At hospital admission, the patient was febrile (38.5°C) but with vital signs within reference ranges.

Physical examination revealed painful, indurated, erythematous lower extremities, with edema and warmth. Clinically relevant results from blood analysis demonstrated leukocytosis (16.5 × 10⁹ cells/L) with neutrophilia (14.0 × 10⁹ cells/L) and elevated C-reactive protein (CRP; 189 mg/L), suggesting bacterial etiology; procalcitonin (PCT) level was within reference range (0.1 µg/L). We empirically introduced therapy with intravenous flucloxacillin (2 g/6 h) for coverage of cellulitis.

On day 2 of hospitalization, extensive bullous changes appeared in the lower extremities. Because of unusual bilateral presentation, we added intravenous therapy with ciprofloxacin. Two days later, fever subsided, and blood leukocyte count returned to normal (10.5 × 10⁹ cells/L). CRP had mildly increased to 204 mg/L; PCT remained within reference range (0.4 µg/L). On day 7 of hospitalization, we observed major improvement in the patient’s laboratory parameters (leukocyte count 6.4 × 10⁹ cells/L, CRP 35 mg/L). We continued treatment with intravenous flucloxacillin and ciprofloxacin until discharge on day 13. Signs of bullous cellulitis in the lower extremities had subsided.

Aerobic blood culture bottle was positive after 3 days of incubation. We observed small, slender, pleomorphic bacilli and coccobacilli in Gram stain. After subcultivation onto solid media, we detected growth on blood and chocolate agar (Figure, panels B, C) on the third day, with no growth observed on MacConkey or TCBS (Thiosulfate-citrate-bile salts-sucrose) agar or in microaerophilic atmosphere. However, we could not identify the causative agent using Gram stain from culture (Figure 1, panel A), colony morphology, growth characteristics, or MALDI-TOF (matrix-assisted laser desorption/ionization time-of-flight) mass spectrometry. We suspected *Francisella tularensis* on the basis of clinical manifestations and local epidemiology. We sent blood agar and chocolate agar plates to the reference Biosafety Level 3 laboratory at the Institute of Microbiology and Epidemiology.
**Table. Antimicrobial susceptibility of Haematospirillum jordaniae**

<table>
<thead>
<tr>
<th>Antimicrobial</th>
<th>MIC, mg/L</th>
<th>Susceptibility category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzylpenicillin</td>
<td>8</td>
<td>R</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>2</td>
<td>S</td>
</tr>
<tr>
<td>Amoxicillin/clavulanic acid</td>
<td>0.125</td>
<td>S</td>
</tr>
<tr>
<td>Cefuroxime iv</td>
<td>16</td>
<td>R</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>0.25</td>
<td>S</td>
</tr>
<tr>
<td>Imipenem</td>
<td>&gt;0.52</td>
<td>R</td>
</tr>
<tr>
<td>Meropenem</td>
<td>1</td>
<td>S</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>&lt;0.002</td>
<td>S</td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>&lt;0.002</td>
<td>S</td>
</tr>
<tr>
<td>Tigecycline</td>
<td>&lt;0.016</td>
<td>S</td>
</tr>
</tbody>
</table>


Immunology (Ljubljana, Slovenia) for further analysis. We isolated DNA using QiaAmp DNA Mini Kit (QIAGEN, https://www.qiagen.com) and tested it, including dilutions from 1:10 to 1:1,000, by specific real-time PCR, which ruled out *F. tularensis* (*Ft*). We performed standard tube extraction protocol for MALDI-TOF mass spectrometry identification using the latest MALDI Biotyper sirius (Bruker Daltonics, https://www.bruker.com) and SR library according to manufacturer instructions but could not identify the organism because scores fell below genus cutoff values. We undertook further molecular analyses, including amplifying the 16S V3/V4 region using Mastermix 16S Complete (Molzym, https://www.molzym.com). We purified amplicons using QIAquick PCR purification kit (QIAGEN) and sequenced them on an ABI3500 genetic analyzer (Applied Biosystems, https://www.thermofisher.com). We analyzed 16S rDNA sequences using the CLC Main Workbench 21.0.5 (QIAGEN) and compared those sequences with others available in the rRNA databases: GenBank (https://blast.ncbi.nlm.nih.gov/Blast.cgi), Ribosomal Database Project (https://rdp.cme.msu.edu), and MicrobeNet (https://microbenet.cdc.gov). Our isolate most closely matched *Haematospirillum jordaniae* isolate Acr132, H5569 and H2509, with 100% sequence identity. By sequencing a longer, 1,462 bp 16S rRNA region (*H. jordaniae* H2509 (GenBank accession no. OM075117). After successful molecular identification, we created *H. jordaniae* main spectra profiles according to manufacturer standard procedures and added them to a custom main spectra profile library because the pathogen was not part of any commercial mass spectra library (Appendix Figure, https://www.ncbi.nlm.nih.gov/EID/article/28/10/22-0326-App1.pdf). *H. jordaniae* is a slow-growing, gram-negative rod bacterium that is difficult to identify because it is not included in standard identification databases. Molecular analysis is necessary for definite identification (3,4). *H. jordaniae*, which belongs to the alphaproteobacteria family *Rhodospirillaceae* (5), was first identified as a potential human pathogen in 2016, when the new genus and species were described from an isolate obtained from a human blood sample in 2010 (3,4). An additional 13 isolates from human blood samples with identical or very similar 16S rRNA sequences, all from men (average age: 60), were later identified at the CDC Special Bacteriology Reference Laboratory (https://www.cdc.gov/ncezid/dhcpp/bacterial_special/special_lab.html).

We determined the antimicrobial susceptibility of *H. jordaniae* using gradient diffusion E-test strips (bioMérieux, https://www.biomerieux.com) and Liofilchem MTS (MIC test strips) for amoxicillin/clavulanic acid (https://www.liofilchem.com) on Muller-Hinton Fastidious agar (CO, 48-h incubation). We interpreted results according to non–species-related EUCAST (https://www.eucast.org) PK/PD (pharmacokinetics/pharmacodynamics) antimicrobial susceptibility breakpoints (Table). According to the results of susceptibility testing, fluoroquinolones had the most favorable breakpoint-to-MIC ratios: ciprofloxacin and levofloxacin had MIC <0.002 mg/L (both) and PK/PD breakpoints of 0.25 mg/L (ciprofloxacin) and 0.5 mg/L (levofloxacin).

Molecular evidence of *H. jordaniae* in the blood of any vertebrate other than humans was described only in a bird species, the reed warbler, *Acrocephalus scirpaceus* (6). Possible routes of infection are through environmental contact, mostly following skin injury (4). Current knowledge about *H. jordaniae* is limited; therefore, our findings signal the need for increased awareness about its clinical course and potential effects as a human pathogen.

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**About the Author**

Dr. Pal works at Murska Sobota General Hospital in Murska Sobota, Slovenia. His research focuses on infectious diseases.
Emerging data show a rapid increase in the prevalence of SARS-CoV-2 infection linked to an increase in COVID-19 cases, which is being driven by the SARS-CoV-2 Omicron variant. Compared with previous variants, Omicron has shown superior capacity for transmission and less sensitivity to neutralizing antibodies induced by vaccination or prior infection with other variants of the virus (1). Initially, the Omicron sublineages BA.1 (including BA.1.1) and BA.2 spread globally at a rapid pace, infecting a large proportion of the population, including vaccinated persons. Nonetheless, vaccines have been shown to provide good protection against severe disease (2). Recently, 2 new sublineages of Omicron, BA.4 and BA.5, have emerged (3). These variants show an even stronger capacity to elude infection- and vaccine-induced immune responses, even evading antibodies in serum from BA.1-infected persons (4,5). Such findings raise concerns that a high community spread might lead to an increasing number of severe cases and a subsequent surge in global hospitalization rates. We performed a quantitative real-time PCR (qPCR) screening survey to estimate the point prevalence of SARS-CoV-2 infection among asymptomatic (defined as having no symptoms at time of sampling) healthcare workers at Danderyd Hospital, Stockholm, Sweden, during June 28–June 29, 2022.

In April and May of 2020, the COMMUNITY study enrolled 2,149 healthcare workers employed at Danderyd Hospital (6). Once enrolled, study participants provided blood samples every 4 months for SARS-CoV-2 serologic assessment (7). Information regarding vaccination status was obtained through the Swedish vaccination register (VAL Vaccinera), and SARS-CoV-2 infection was determined by either seroconversion before vaccination or positive PCR test results obtained from the national communicable diseases register, SmiNet (Public Health Agency of Sweden).

We conducted a qPCR screening survey during June 28–June 29, 2022. We invited all COMMUNITY-study participants who had provided a blood sample in January 2022 (n = 1,412) to participate in the screening survey via a mobile application program. We restricted participation in the survey to