LMG_4337t (92.3% identity). On the basis of Clinical Laboratory Standards Institute guidelines (2), this similarity indicated a possible novel species.

The DPHL Microbiology Department’s laboratory manager sent the organism to CDC’s Special Bacteriology Reference Laboratory. Results identified the organism as *H. jordaniae*. Upon review of MicrobeNet at a later date, the organism was found to match *H. jordaniae* H5569–con t by 98.9%.

*H. jordaniae* is a common environmental microbe, but it was implicated in this clinical case in a man in Delaware. The patient had symptoms characteristic of other pathogenic bacterial illnesses. Concern exists that slow-growing, gram-negative rods identified in blood culture could be potential bioterrorism agents. Humrighouse et al. (1) described how *Francisella tularensis* infection was suspected in 2 clinical cases that were actually *H. jordaniae* infections.

Humrighouse et al. (1) proposed the name *H. jordaniae* on the basis of an isolate received in 2010. Previously, 14 organisms identified at CDC were isolated from blood taken from men 39–78 years of age with symptoms including swelling of the lower extremities (2 patients), septicemia (3 patients), and bacteremia (1 patient). The symptoms of the patient we report mirrored those symptoms.

This discovery is important because it demonstrates that organisms conceived to be environmental in nature and suspected to have limited clinical implications are emerging as human pathogens. The ability to identify bacteria by sequencing (in this case, sequencing of the 16S rRNA gene) was necessary to identify *H. jordaniae* because clinical information on this pathogen is still limited.

**Acknowledgments**

We would like to thank Daniel Bolon for technical support in this study and Gary Richards, Andrea Wojcik, Donna Sharp, and Delaware’s Public Health Privacy Board for revising and editing this article.

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**References**


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**Molecular Typing and Antifungal Susceptibility of Candida viswanathii, India**

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We report invasive candidiasis caused by *Candida viswanathii* over 2 time periods during 2013–2015 in a tertiary care hospital in Chandigarh, India. Molecular typing revealed multiple clusters of the isolates. We detected high MICs for fluconazole in the second time period.

Invasive candidiasis is a life-threatening infection caused by various *Candida* species. Although *C. albicans* has been the predominant species causing invasive candidiasis, non-albicans *Candida* (NAC) species have emerged globally (1). *C. viswanathii*, a pathogen first isolated from the cerebrospinal fluid of a patient in 1959 (2), is rarely encountered, and only 17 cases have been reported worldwide (3). This agent has been isolated sporadically from animal and environmental sources (4–6).

We report on 23 cases of invasive candidiasis caused by *C. viswanathii* at a tertiary care center in Chandigarh, India, involving 7 case-patients during December 2013–April 2014.
and 16 case-patients during December 2014–April 2015. In the first time period, all isolates were from blood, whereas in the second time period, the agent was isolated from pus (n = 5), blood (n = 5), cerebrospinal fluid (n = 3), and lung nodule, lung aspirate, and ieliac fluid (n = 1 each).

Of the 23 patients, 16 were men and 7 were women. Six (26%) patients had neutropenia, and 18 (90%) had tuberculosis, pancreatitis, or chronic kidney disease. Eight (34.7%) patients acquired the infection after surgery. Twelve patients used indwelling devices: 3 (15%) had a central venous catheter, 4 (20%) an endotracheal tube, 3 (15%) a drainage catheter, and 2 (10%) a urinary catheter.

We screened the hospital environment and the hands of healthcare workers for a possible source of

C. viswanathii

infection during the second time period. We could not isolate

C. viswanathii

from any of those samples from a total of

46 workers and 57 different environmental sites.

Conventional methods failed to differentiate

C. viswanathii

and

C. tropicalis.

C. viswanathii

assimilated sucrose and cellobiose but failed to assimilate trehalose and raffinose. C. tropicalis has a variable assimilation pattern for these sugars.

To identify the isolates, we performed matrix-assisted laser desorption/ionization time-of-flight mass spectrometry using MALDI-TOF MS, version 3 (Bruker Daltonik GmbH, Bremen, Germany) and sequenced the D1/D2 region of a large subunit of ribosomal DNA (7,8). Because we could not identify

C. viswanathii

using the MALDI-TOF MS version 3 database, we updated the database in-house by adding sequence-proved isolates of

C. viswanathii.

We identified the isolates with a log score of >1.8 by using the modified database. The rDNA sequence of the isolates showed 100% similarity with the type strain of

C. viswanathii

ATCC 22981 (GenBank accession no. NG_054835.1) except for 1 isolate (99% similarity with type strain, accession no. MF682371). The molecular phylogenetic analysis revealed that 1 isolate (B-30815) had 1 nucleotide substitution (T to C), which was 1 of the 5 substitutions we observed in

C. pseudoviswanathii

while comparing it with

C. viswanathii

(9).

Amplified fragment-length polymorphism revealed a similarity coefficient of ≥90% of the isolates (online Technical Appendix Figure, https://wwwnc.cdc.gov/EID/article/24/10/18-0801-Techapp1.pdf) (7). The isolates from the first time period formed 2 clusters (clusters A and B); 1 isolate from the second period was also in cluster B. Isolates of the second time period had 3 major clusters (clusters C, D, and E) and had higher MICs for fluconazole.

We performed antifungal susceptibility testing for amphotericin B, fluconazole, itraconazole, voriconazole, caspofungin, anidulafungin, and micafungin by the microbroth dilution method recommended by the Clinical and Laboratory Standards Institute (10). After incubating the plates for 24 h at 37°C, we took a visual reading to determine the MICs. The isolates of the second time period had higher MICs (MIC<sub>90</sub> 64 μg/mL, MIC<sub>90</sub> 0.125 μg/mL) compared with the isolates of the first period (MIC<sub>90</sub> 1 μg/mL, MIC<sub>90</sub> 0.125 μg/mL). We also recorded higher MICs (MIC<sub>90</sub> 1 μg/mL, MIC<sub>90</sub> 2 μg/mL) for voriconazole for the isolates of the second period (Table).

In conclusion, our study showed multiple clusters of

C. viswanathii

causing invasive infections in patients with neutropenia and chronic diseases at a single healthcare center in India. We could not trace the source of the agent. Conventional identification methods could not differentiate the isolates from those of

C. tropicalis.

The high MICs for fluconazole among the isolates from the second time period also raise concerns about possible antifungal resistance.

### Table.

In vitro antifungal susceptibility data of

C. viswanathii

isolates from a tertiary care hospital in Chandigarh, India

<table>
<thead>
<tr>
<th>Isolate</th>
<th>Antifungal agent</th>
<th>MIC&lt;sub&gt;Range&lt;/sub&gt;</th>
<th>MIC&lt;sub&gt;90&lt;/sub&gt;</th>
<th>MIC&lt;sub&gt;90&lt;/sub&gt;</th>
<th>Geometric mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. viswanathii 2013–2014, n = 7</td>
<td>Amphotericin B</td>
<td>0.03–0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Itraconazole</td>
<td>0.03–0.5</td>
<td>0.125</td>
<td>0.25</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>Fluconazole</td>
<td>0.125–1</td>
<td>1</td>
<td>1</td>
<td>0.41</td>
</tr>
<tr>
<td></td>
<td>Voriconazole</td>
<td>0.03–0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.14</td>
</tr>
<tr>
<td></td>
<td>Micafungin</td>
<td>0.125</td>
<td>0.125</td>
<td>0.125</td>
<td>0.125</td>
</tr>
<tr>
<td></td>
<td>Caspofungin</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Anidulafungin</td>
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<td>0.5</td>
<td>0.5</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>Posaconazole</td>
<td>0.0312–0.0625</td>
<td>0.0312</td>
<td>0.0625</td>
<td>0.03</td>
</tr>
<tr>
<td>C. viswanathii 2015, n = 16</td>
<td>Amphotericin B</td>
<td>0.06–0.25</td>
<td>0.125</td>
<td>0.25</td>
<td>0.14</td>
</tr>
<tr>
<td></td>
<td>Itraconazole</td>
<td>0.03–1</td>
<td>0.25</td>
<td>0.25</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Fluconazole</td>
<td>0.5–64</td>
<td>64</td>
<td>64</td>
<td>29.34</td>
</tr>
<tr>
<td></td>
<td>Voriconazole</td>
<td>0.03–8</td>
<td>1</td>
<td>2</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>Micafungin</td>
<td>0.125</td>
<td>0.125</td>
<td>0.125</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Caspofungin</td>
<td>0.125–1</td>
<td>0.5</td>
<td>0.5</td>
<td>0.40</td>
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<tr>
<td></td>
<td>Anidulafungin</td>
<td>0.125–1</td>
<td>0.5</td>
<td>1</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>Posaconazole</td>
<td>0.0321–0.5</td>
<td>0.25</td>
<td>0.25</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Mr. Shankarnarayan is a PhD student in the Department of Medical Microbiology, PGIMER, Chandigarh, India. His doctoral focus is on evaluating the dynamics of invasive

Candida

infections among hospitalized patients.
We report a rare case of *Staphylococcus argenteus* bone and joint infection in a 9-year-old boy in France. His finger arthritis was complicated by osteitis 5 weeks later, which resulted in a secondary intervention. This case indicates the virulence of *S. argenteus*, an emerging pathogen whose clinical effects are poorly described.

*S. argenteus* (formerly *S. aureus* clonal complex 75) is an emerging species in the *S. aureus* complex (1). Several studies reported sporadic cases of *S. argenteus* infections mainly in Asia, Oceania, and the Pacific Islands (2) but rarely in Europe (3). We report the clinical characteristics of a community-acquired bone and joint infection with *S. argenteus* in a child living in France.

At the end of July 2017, a 9-year-old boy with no unusual medical history or previous local trauma was hospitalized because of acute signs of infection of the third finger on his right hand. He was first seen in a local hospital and given an initial diagnosis of cellulitis (arthritis). Two days later, he was admitted to the emergency pediatric ward of a tertiary care hospital where a surgical joint exploration was performed and confirmed the diagnosis of arthritis associated with an abscess of the extensor tendon sheath (Table).

Surgical microbiological samples cultured on blood agar plates (aerobic conditions at 37°C for 24 h) grew a strain that was identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (Microflex LT, Bruker, France) as having log scores ranging from 1.39 to 1958

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**Community-Acquired Staphylococcus argenteus Sequence Type 2250 Bone and Joint Infection, France, 2017**

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DOI: https://doi.org/10.3201/eid2410.180727

We report a rare case of *Staphylococcus argenteus* bone and joint infection in a 9-year-old boy in France. His finger arthritis was complicated by osteitis 5 weeks later, which resulted in a secondary intervention. This case indicates the virulence of *S. argenteus*, an emerging pathogen whose clinical effects are poorly described.

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**References**


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Community-Acquired *Staphylococcus argenteus* Sequence Type 2250 Bone and Joint Infection, France 2017

**Technical Appendix**

**Technical Appendix Table.** Characteristics of reported cases of infection with *Staphylococcus argenteus*.

<table>
<thead>
<tr>
<th>Reference</th>
<th>No. cases</th>
<th>Patients</th>
<th>Signs/symptoms</th>
<th>Diagnostic method</th>
<th>Bacterial strain characteristics</th>
<th>Treatment</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holt et al., 2011 (1)</td>
<td>1</td>
<td>Woman from Australia</td>
<td>Necrotizing fasciitis and bacteremia</td>
<td>WGS</td>
<td>Resistant to methicillin</td>
<td>Not described</td>
<td>Not reported</td>
</tr>
<tr>
<td>Dupieux et al., 2015 (2)</td>
<td>2</td>
<td>25-year-old woman</td>
<td>Pulmonary and bacteremia</td>
<td>Microarray, MLST</td>
<td>Susceptible to methicillin, positive for PVL</td>
<td>Amoxicillin/clavulanic acid, roxithromycin, ceftriaxone, spiramycin, oxacillin, clindamycin, linezolid</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18-month-old child</td>
<td>Knee arthritis, bacteremia, and multiple pulmonary abscesses</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thaipadungpanit et al., 2015 (3)</td>
<td>10</td>
<td>270 in Thailand</td>
<td>2 with bacteremia; 7 with SST; 1 with osteomyelitis (2 healthcare-associated infections)</td>
<td>SNP</td>
<td>All resistant to methicillin; none positive for PVL</td>
<td>Not described</td>
<td>Same prognosis as <em>S. aureus</em> infections for death and illness</td>
</tr>
<tr>
<td>Chantratita et al., 2016 (4)</td>
<td>58</td>
<td>311 in Thailand</td>
<td>23 with bacteremia</td>
<td>PFGE, MLST</td>
<td>No strain resistant to methicillin; 15 strains positive for PVL</td>
<td>Not described</td>
<td>Same death rate as for <em>S. aureus</em> infections</td>
</tr>
<tr>
<td>Suzuki et al., 2017 (5)</td>
<td>2</td>
<td>Intoxicated 3 h after eating lunch</td>
<td>Food poisoning</td>
<td>WGS</td>
<td>Both strains produced SEB; no SCCmec elements</td>
<td>Not described</td>
<td>Not described</td>
</tr>
<tr>
<td>Wakabayashi et al., 2018 (6)</td>
<td>4 in 2014; 13 in 201</td>
<td>3 patients and 1 food handler in 2014; 12 patients and 1 food handler in 2015</td>
<td>Food poisoning</td>
<td>PFGE, MLST</td>
<td>All strains positive for SEB</td>
<td>Not described</td>
<td>Not described</td>
</tr>
<tr>
<td>Reference</td>
<td>No. cases</td>
<td>Patients</td>
<td>Signs/symptoms</td>
<td>Diagnostic method</td>
<td>Bacterial strain characteristics</td>
<td>Treatment</td>
<td>Prognosis</td>
</tr>
<tr>
<td>-----------</td>
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<td>----------------</td>
<td>-------------------</td>
<td>-------------------------------</td>
<td>-----------</td>
<td>----------</td>
</tr>
<tr>
<td>This study</td>
<td>1</td>
<td>9-year-old boy in France</td>
<td>Finger arthritis and osteomyelitis</td>
<td>Microarray</td>
<td>Susceptible to methicillin</td>
<td>Amoxicillin/clavulanic acid, ceftazolin, gentamicin, rifampin, fusidic acid, clindamycin, ofloxacin</td>
<td>Recovered</td>
</tr>
</tbody>
</table>

*MLST, multilocus sequence typing; PFGE, pulsed-field gel electrophoresis; PVL, Panton-Valentine leukocidin; SCCmec, staphylococcal cassette chromosome mec; SEB, staphylococcal enterotoxin B; SNP, single-nucleotide polymorphism; SSTI, skin and soft tissue infection; WGS, whole-genome sequencing.*
References


Technical Appendix Figure. Community-acquired *Staphylococcus argenteus* sequence type 2250 bone and joint infection in a 9-year-old boy, France 2017. A) Fat-saturated, T1-weighted, magnetic resonance image after gadolinium injection in coronal plane of the right hand, showing evidence of osteitis at the base of middle phalanx up to cartilaginous growth plate of third finger with local necrosis at the time of infection. B) Infected bone tissue showing infiltration with numerous plasmocytes and lymphocytes on the left at the time of infection (hematoxylin and eosin stained, original magnification ×100).