References


Address for correspondence: Anup K. Ghosh, Postgraduate Institute of Medical Education and Research, Medical Microbiology, 2nd Fl, Sector-12, Chandigarh, India; email: ak_ghosh3@rediffmail.com, anupkg3@gmail.com

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

Josselin Rigail, Florence Grattard, Sylvain Grange, Fabien Forest, Elie Haddad, Anne Carricajo, Anne Tristan, Frederic Laurent, Elisabeth Botelho-Nevens, Paul O. Verhoeven

Author affiliations: University Hospital of Saint-Etienne, Saint-Etienne, France (J. Rigail, F. Grattard, S. Grange, F. Forest, E. Haddad, A. Carricajo, E. Botelho-Nevens, P.O. Verhoeven); Jean Monnet University, Saint-Etienne (J. Rigail, F. Grattard, A. Carricajo, E. Botelho-Nevens, P.O. Verhoeven); International Centre for Infectiology Research, Lyon, France (A. Tristan, F. Laurent); French National Reference Centre for Staphylococci, Lyon (A. Tristan, F. Laurent)

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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.

Staphylococcus 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
1.87 (corresponding to *S. aureus*, *S. simiae*, or *S. epidermidis*). These scores were lower than those required for reliable identification of species (2.3) or genus (2.1) levels. Antimicrobial drug susceptibility testing (Vitek2; bioMérieux, Marcy l’Etoile, France) identified resistance to penicillin G. Because there was no initial reliable identification of this strain, we performed molecular tests. The strain was negative for *nuc*, *lukS-PV*, *mecA*, *mecC*, *tst-1*, and *spa* genes. The V3 region sequence of the gene coding for 16S rRNA showed 100% identity with that for *S. aureus*.

**Table.** Clinical characteristics and timeline for patient with community-acquired *Staphylococcus argenteus* sequence type 2250 bone and joint infection, France, 2017*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Jul 30</th>
<th>Aug 2</th>
<th>Aug 8</th>
<th>Sep 5</th>
<th>Nov 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital</td>
<td>Local</td>
<td>Tertiary care</td>
<td>Tertiary care</td>
<td>Tertiary care</td>
<td>Tertiary care</td>
</tr>
<tr>
<td>Clinical features</td>
<td>Pain in third finger of right hand</td>
<td>Fever (temperature 38.6°C); pain and functional impotence in flexion of finger</td>
<td>Poor tolerance of antimicrobial drugs</td>
<td>Fever (temperature 38.4°C); pain in finger</td>
<td>Stiffness in finger; no pain</td>
</tr>
<tr>
<td>Signs at physical examination</td>
<td>Inflammatory edema of finger; no inoculation lesion</td>
<td>Phlegmon of finger: inflammatory skin; edema on second phalanx</td>
<td>ND</td>
<td>Misalignment of second phalanx</td>
<td>No signs of infection</td>
</tr>
<tr>
<td>Laboratory findings</td>
<td>Leukocytes, ×10⁹ cells/L†</td>
<td>Culture of infection site not performed (no pus); blood culture not performed</td>
<td>Surgical samples: neutrophils and gram-positive cocci (identified as <em>S. argenteus</em>); blood cultures sterile</td>
<td>ND</td>
<td>11.6</td>
</tr>
<tr>
<td></td>
<td>C-reactive protein, mg/L‡</td>
<td></td>
<td></td>
<td>7.2</td>
<td>7.2</td>
</tr>
<tr>
<td>Microbiological</td>
<td>AMX (1,000 mg/d) and CLA (125 mg/d)</td>
<td>CFZ (2,200 mg/d) and GEN (400 mg/d) for 2 d; AMX (2,000 mg/d), CLA (250 mg/d), RIF (600 mg/d) for 6 wk</td>
<td>Surgical joint lavage and débridement for massive purulent abscess that reached the extensor tendon and joint capsule of second phalanx; no articular cartilage lesion</td>
<td>ND</td>
<td>Surgical lavage and realignment of phalanxes with implantation of external fixator on Sep 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stop AMX and CLA; FUS (1.5 g/d) and RIF (600 mg/d) for 5 wk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sep 8: CLI (900 mg/d) and OFX (400 mg/d) for 6 wk</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>Radiologic findings</td>
<td>Not performed</td>
<td>Radiograph of hand: no signs of osteitis</td>
<td>ND</td>
<td>MRI of hand: osteitis</td>
<td>ND</td>
</tr>
<tr>
<td>Histologic findings</td>
<td>ND</td>
<td>Chronic osteitis</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis considered</td>
<td>Cellulitis of finger</td>
<td>Arthritis of second phalanx; abscess of extensor tendon sheath</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment Antimicrobial drugs</td>
<td>AMX (1,000 mg/d) and CLA (125 mg/d)</td>
<td>CFZ (2,200 mg/d) and GEN (400 mg/d) for 2 d; AMX (2,000 mg/d), CLA (250 mg/d), RIF (600 mg/d) for 6 wk</td>
<td>Surgical joint lavage and débridement for massive purulent abscess that reached the extensor tendon and joint capsule of second phalanx; no articular cartilage lesion</td>
<td>ND</td>
<td>Surgical lavage and realignment of phalanxes with implantation of external fixator on Sep 8</td>
</tr>
<tr>
<td>Surgery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ND</td>
</tr>
<tr>
<td>Outcome</td>
<td>Discharged</td>
<td>Improvement at discharge on Aug 4; patient seen on Aug 6, 8, 10, and 12</td>
<td>Good outcome</td>
<td>Discharged on Sep 11; patient seen on Sep 13, 15, and 22, and Oct 6; external fixator removed on Oct 6</td>
<td>Good outcome and functional rehabilitation; patient seen on Dec 27 and had similar findings</td>
</tr>
</tbody>
</table>

*AMX, amoxicillin; CFZ, cefazolin; CLA, clavulanic acid; CLI, clindamycin; FUS, fusidic acid; GEN, gentamicin; MRI, magnetic resonance imaging; ND, not determined; OFX, ofloxacin; RIF, rifampin.
†Reference range 4.5–13.5 × 10⁹ cells/L.
‡Reference value <5 mg/L.
Microarray analysis (S. aureus Genotyping Kit 2.0; Alere Technologies GmbH, Jena, Germany) assigned this strain to the clonal complex 2250/2277 of one of the main clusters of S. argenteus (2,4). The patient was discharged and received an oral antimicrobial regimen for 6 weeks; healing was closely monitored (Table).

Five weeks later, the patient was hospitalized because of recurrent signs of infection (Table). Magnetic resonance imaging of the right hand showed osteitis (online Technical Appendix Figure, panel A, https://wwwnc.cdc.gov/EID/article/23/10/18-0727-Techapp1.pdf). A second surgical procedure was performed (Table). Cultures of surgical samples remained sterile after 10 days and a 16S rDNA PCR result was negative. Histologic analysis showed chronic osteitis (online Technical Appendix Figure, panel B). An oral drug regimen, including clindamycin and ofloxacin for 6 weeks, was prescribed. Long-term outcome was good (Table), despite persistence of stiffness in the finger.

This rare case of osteomyelitis caused by S. argenteus highlights the difficulties in correctly identifying this species. As with S. swtzeiteri, S. argenteus is an emergent species that has been described as part of the S. aureus species complex (5). S. argenteus was first described in 2002 as a CC75/sequence type T1223 clone of S. aureus in Aboriginal communities in Australia (6). This species was named S. argenteus in 2011 because of its lack of staphyloxanthin production (1). Most studies reported prevalence rates for S. argenteus among strain collections of 0.16%–18.6% according to geographic distribution, with a clear predominance in Asia and the West Pacific region (1,5–7) and a rare description in Europe (3).

In our case, no epidemiologic link to Asia or the West Pacific region was observed. The clinical spectrum of infections with S. argenteus remains poorly described, but varies from asymptomatic nasal carriage (8) to community-acquired infections, including skin and soft tissue infections (1,6), bacteremia (1,9), and foodborne illness (8). Apart from 2 cases of bacteremia reported by Dupieux et al. (9), clinical data for S. argenteus infections have been poorly detailed (online Technical Appendix Figure). To the best of our knowledge, only 1 case of osteomyelitis has been reported (7).

Previous cases and the case we report indicate that S. argenteus could be responsible for invasive infections that are difficult to manage. S. argenteus was initially considered to be less virulent than S. aureus on the basis of a study in Australia, which reported that this species was associated mainly with skin and soft tissue infections and rarely with bacteremia (3/220 cases) (1). However, a comparative study of 311 cases of S. argenteus and S. aureus sepsis in Thailand showed a similar outcome after 28 days (10). Moreover, virulence factors, such as Panton-Valentine leucocidin and enterotoxins, have been described in S. argenteus isolates (4,8–10). In contrast to reports from Aboriginal communities in Australia (6) and remote populations in the West Pacific region (online Technical Appendix Table), resistance to methicillin was not detected in strains from the case-patients in this study.

In contrast to S. aureus, the effect of carriage of S. argenteus has not been studied. For our case-patient, screening for S. argenteus nasal carriage was not performed. However, a recent study of foodborne illness outbreaks reported the ability of this bacterium to spread in the environment and colonize food handlers (8).

S. argenteus is an emerging species for which its clinical spectrum remains poorly described. Further studies are needed to better address the global prevalence and clinical role of this bacterium, including its potential effects in chronic human infections.

Acknowledgments
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J.R., S.G., and F.F. performed clinical imaging and collected histologic data; E.H. performed surgery; F.G., A.C., and A.T. analyzed the bacterial strain; and F.L., E.B.-N., and P.O.V. critically revised the manuscript and made final corrections. All authors approved the final manuscript.

About the Author
Dr. Rigaill is a microbiology fellow at the University Hospital of Saint-Etienne, Saint-Etienne, France. His primary research interest is identifying the determinants of S. aureus carriage.

References


Address for correspondence: Elisabeth Botelho-Nevers, Infectious Diseases Department, University Hospital of Saint-Etienne, Av Albert Raimond, 42055 Saint-Etienne CEDEX 02, France; email: elisabeth.botelho-nevers@univ-st-etienne.fr

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**Circulation of Influenza A(H5N8) Virus, Saudi Arabia**

**Hussain Al-Ghadeer,**1 Daniel K.W. Chu,**1**


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Highly pathogenic avian influenza A(H5N8) viruses have been detected in several continents. However, limited viral sequence data are available from countries in the Middle East. We report full-genome analyses of highly pathogenic H5N8 viruses recently detected in different provinces in Saudi Arabia.

On December 19, 2017, a high number of dead birds from various species was reported in a live bird market in Riyadh, Saudi Arabia, by the Department of Animal Resources Services, Ministry of Environment, Water, and Agriculture. Oropharyngeal and cloacal swab samples were collected from affected birds and investigated for highly pathogenic avian influenza (HPAI) viruses in Riyadh Veterinary Diagnostic Laboratory using reverse transcription PCR (RT-PCR) (1). These tests detected HPAI A(H5N8) virus. After this index outbreak, HPAI was reported in adjacent provinces. Surveillance studies were performed in all provinces (>1 major poultry market and 10 backyard farms per province) to estimate disease prevalence. As of May 2018, a total of 7,273 birds had been investigated; 805 were positive for H5N8, which was detected in 7 provinces (Riyadh, Eastern, Al-Qasim, Makkah, Al-Madinah, Asir, and Jizan). The highest number of positive results was reported in Riyadh (693 samples), in which different commercial poultry farms (22 farms for laying hens, 2 for broiler breeders, and 1 for quail) were affected. A contingency plan, based on a stamping-out policy, was implemented to control the disease. More than 8.8 million birds were depopulated.

1These authors contributed equally to this article.