**Candida auris** Sternal Osteomyelitis in a Man from Kenya Visiting Australia, 2015

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In Australia in 2015, *Candida auris* sternal osteomyelitis was diagnosed in a 65-year-old man with a history of intensive care treatment in Kenya in 2012 and without a history of cardiac surgery. The isolate was South Africa clade III. Clinicians should note that *C. auris* can cause low-grade disease years after colonization.

1These authors contributed equally to this article.
We performed whole-genome sequencing (WGS) on the isolate (FSMC57608) using the NextSeq platform (Illumina, https://www.illumina.com/) and then assembled Illumina paired-end sequencing data using SPAdes, St. Petersburg genome assembler 3.1.1 (http://spades.bioinf.spbau.ru/release3.1.1/manual.html). We identified core genome single-nucleotide polymorphisms (SNPs) using Snippy version 4.0 (http://www.vicbioinformatics.com/software.snippy.shtml), using the C. auris B8441 genome for reference and previously described methods (2), and mapped ≈97.77% of the reads. A maximum-parsimony phylogenetic tree was constructed by using MEGA version 7.0 (https://www.megasoftware.net/) and 10 other C. auris genomes (2). Results showed that FSMC57608 (GenBank accession no. SRP156632) is a South Africa clade III isolate (Appendix Figure 2) with SNPs V125A and F126L and wild-type at amino acid positions 132 and 143 of Erg11 (gene associated with azole class antifungal drug resistance) (Appendix Figure 3).

Extensive nosocomial transmission of C. auris has been documented, and mortality rates of 40%–60% have been reported for patients with candidemia (2–4). C. auris can colonize human skin for months (5,6). Of 620 cases of C. auris infection linked to outbreaks in Europe during 2013–2017, a total of 466 (75.2%) patients became colonized (3). We postulate that our patient became colonized in 2012 in an intensive care unit in Kenya. This case also illustrates that clinical manifestations of C. auris infection can progress slowly for >12 months. C. auris is multidrug resistant and, therefore, poses a risk for all patients, given the limited antifungal options available. Tentative C. auris–specific MIC breakpoints exist, pending further correlation between MICs and clinical outcomes (2). Proposed breakpoints are derived from expert opinion and/or those of closely related Candida species for antimicrobial drugs (e.g., amphotericin B) that do not have breakpoints. Despite breakpoint uncertainty and concerns about emergent multidrug resistance among C. auris isolates, we had prescribed oral posaconazole for our patient because of the in vitro MIC results and his strong preference for oral antifungal therapy.

WGS results show C. auris isolates fall into 4 distinct clades that appear to have emerged almost simultaneously in different geographic regions of the globe (2–4). Isolate FSMC57608 has SNPs V125A and F126L in Erg11, the latter SNP, F126L, having been described in previous investigations (J.F. Muñoz, unpub. data, https://doi.org/10.1101/299917) (2,7). This isolate was also wild type at amino acid positions 132 and 143 of Erg11, as seen in Africa isolates (J.F. Muñoz, unpub. data, https://doi.org/10.1101/299917), further supporting that the infection originated in Africa (7).

In summary, we describe a case of travel-linked C. auris infection manifesting as chronic sternal osteomyelitis, diagnosed in Australia in 2015. The patient had a history of intensive care treatment in Kenya, a country with documented C. auris transmission (2); he required treatment in Australia 3 years later and exhibited clinically significant disease associated with South Africa clade III C. auris infection.

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About the Author
Dr. Heath is an infectious diseases physician in the Department of Infectious Diseases at Fiona Stanley Hospital and Royal Perth Hospital in Perth, Western Australia, Australia, and a clinical microbiologist for PathWest Laboratory Medicine, FSH Network, Perth; he has research interests in laboratory
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References


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Appendix

Appendix Table. Antifungal susceptibility testing results of Candida auris isolate FSMC57608, Australia, 2015

<table>
<thead>
<tr>
<th>Drug</th>
<th>MIC, mg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amphotericin B</td>
<td>1.0</td>
</tr>
<tr>
<td>Anidulafungin</td>
<td>0.12</td>
</tr>
<tr>
<td>Caspofungin</td>
<td>0.12</td>
</tr>
<tr>
<td>Micafungin</td>
<td>0.12</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>0.25</td>
</tr>
<tr>
<td>Posaconazole</td>
<td>0.06</td>
</tr>
<tr>
<td>5 Fluycytosine</td>
<td>0.12</td>
</tr>
<tr>
<td>Voriconazole</td>
<td>2.0</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>&gt;256</td>
</tr>
</tbody>
</table>

*Susceptibility testing was performed using the Sensititer YeastOne YO10 panel (Trek Diagnostic Systems, West Sussex, UK).

Appendix Figure 1. Sequence of D1–D2 region of 28S rDNA of Candida auris isolate FSMC57608 (referred to as candida-FSH; GenBank accession no. SRP156632), Australia, 2015, and other C. auris strains (GenBank accession nos. JQ219331–2, KM000828, KM000830, KU321688), with mutations highlighted. Our isolate is similar to JQ219331–2 and different from the other 3 isolates at 5 sites.
Appendix Figure 2. Maximum-parsimony phylogenetic tree of all single-nucleotide polymorphisms (SNPs) in core genome constructed by using MEGA version 7.0 (https://www.megasoftware.net/). Numbers shown on phylogenomic tree branches are numbers of SNPs found with Snippy version 4.0 (http://www.vicbioinformatics.com/software.snippy.shtml) compared with other reference strains from around the world. Scale bar indicates nucleotide substitutions per site.
Appendix Figure 3. Erg11 sequence comparison demonstrating single-nucleotide polymorphisms (SNPs) V125A and F126L in FSMC57608 (GenBank accession no. SRP156632), Australia, 2015. MS3054 is a clade I Candida auris isolate from India used for comparing the Erg11 sequence. FSMC57608 is wild type at amino acid positions 132 and 143.