Disseminated Infection Due to *Actinomyces meyeri*: Case Report and Review

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*Actinomyces meyeri* is rarely isolated in cases of actinomycosis. We present a case of disseminated actinomycosis due to *A. meyeri*: the patient had an abscess of the lung, osteomyelitis of the tibia, and multiple skin abscesses. Cure was achieved with surgical debridement and administration of intravenous penicillin, followed by oral penicillin, for 1 year. A concomitant gram-negative bacillus, *Actinobacillus actinomycetemcomitans*, was also isolated. Review of the literature revealed only 26 well-documented cases of infection with *A. meyeri*. Male adults are mainly affected, and alcoholism is frequently the underlying condition in these patients. Associated bacteria were isolated in two-thirds of these cases. In contrast to other species of *Actinomyces*, *A. meyeri* often causes pulmonary infection and shows a tendency for hematogenous dissemination. Even though multiple organs are involved, the outcome for these patients is excellent when penicillin is administered for several months and surgical procedures are performed when necessary.

The term *actinomycete*, which is of Greek origin, means ray fungus; however, actinomycosis is due to a gram-positive anaerobic bacterium. Bollinger [1] described the microorganism in 1877 as the causative agent of lumpy jaw in cattle and named it *Actinomyces bovis* [1]. In 1891, Wolff and Israel [2] grew a similar microorganism from a pulmonary abscess and named it *Actinomyces israelii* [2]. Although most cases of human actinomycosis are due to *A. israelii*, less common agents are *Actinomyces naeslundii*, *Actinomyces viscosus*, *Actinomyces odontolyticus*, *Actinomyces meyeri*, and *Propionibacterium propionicum*.

*A. meyeri* was first isolated from a lung abscess in 1911 by Meyer, who described it as a *Streptothrix* [3]. The microorganism was then reclassified as *Actinobacterium meyeri* in 1938 [4]; it was finally classified in the order Actinomycetales in 1977 [5]. This rare actinomycete has been recovered from a minority of patients, and the spectrum of infections it causes is not well known, although it shares all the microbiological characteristics of the organisms in this order. We describe a case of disseminated actinomycosis due to *A. meyeri*.

**Case Report**

A 47-year-old man presented to our hospital in mid-December 1993 with a painful effusion of the left knee. The collection was drained for analgesic purposes, and no analysis of the fluid was done at that time. Three weeks later his symptoms recurred, and red, firm, painless subcutaneous nodules appeared on the trunk and the extremities. On 16 January 1994, the patient became febrile and was admitted. His medical history was significant only for alcohol abuse and smoking (he reported that he consumed 60 g of ethanol and smoked three packs of cigarettes daily). He had not brushed his teeth for 15 years.

Physical examination revealed a temperature of 38.2°C; there were no enlarged lymph nodes. Examination of the trunk and extremities revealed red, firm, painless subcutaneous nodules, some of which had started to form abscesses with fistulization to the skin (figure 1). The left knee was swollen and painful on mobilization but showed no sign of effusion. The patient had dentogingival disease, with numerous caries and stumps.

Laboratory studies revealed that an inflammatory process was present. The erythrocyte sedimentation rate was markedly increased (117 mm/h; normal rate, 3–8 mm/h). The WBC count was 13.3 × 10^9/L (normal count, 4–10 × 10^9/L) with a left shift (14% band forms; normal, <8%), indicating leukocytosis. The platelet count was also elevated (466 × 10^9/L; normal count, 150–300 × 10^9/L). The RBC count (117 mml/h; normal rate, 3–8 mm/h). The WBC count was 13.3 × 10^9/L (normal count, 4–10 × 10^9/L) with a left shift (14% band forms; normal, <8%), indicating leukocytosis. The platelet count was also elevated (466 × 10^9/L; normal count, 150–300 × 10^9/L). The RBC count (2.95 × 10^12/L; normal count, 4.5–5.5 × 10^12/L). The serum γ-glutamyltransferase level was markedly increased (159 U/L; normal level, 40 U/L) and the levels of aspartate aminotransferase and alanine aminotransferase were slightly increased at 42 U/L (normal level, 50 U/L) and 51 U/L (normal level, <40 U/L), respectively; these abnormalities in liver function were attributed to his alcoholism.

A chest radiograph revealed an infiltrative mass in the middle lobe of the right lung, which was confirmed by a chest CT scan (figure 2). The patient refused to undergo CT-guided needle biopsy. Conventional tomograms of the left knee demonstrated a significant osteomyelitic process in the proximal left tibia (figure 3). An ultrasonogram of the abdomen showed slight splenomegaly, and the liver was described as normal in size.

One of the subcutaneous nodules was biopsied, and the histological findings were suggestive of disseminated actinomycosis: macroscopically, no sulfur granules were seen, but microscopically, chronic purulent inflammation in association with radiating microorganisms was observed. Three stains (gram, periodic acid-Schiff, and Grocott-Gomori methenamine-silver...
nitrate) were positive. The organisms were subsequently cul­tured and identified as *Actinomyces* on the basis of the results of gas-liquid chromatography, which demonstrated the presence of large amounts of succinic acid in prereduced anaerobi­cally sterilized broth [5]. The species was identified as *A. mey­eri* with use of the Rapid ANA II system (Innovative Diagnostic Systems, Atlanta). A gram-negative bacillus also was recovered from the same subcutaneous abscess and was identified as *Acti­nobacillus actinomycetemcomitans* by means of biochemical tests.

Treatment with intravenous penicillin G (20 million units per day for 8 weeks) was started and was then switched to oral penicillin V (3 million units per day for 12 months). All the subcutaneous abscesses disappeared within 2 weeks, and the pulmonary lesion regressed within 4 months. The parameters indicative of inflammation normalized within 4 weeks. Multiple dental extractions were done under general anesthesia. The patient underwent surgical debridement and synovectomy of the left knee, and 1 year later he underwent surgical reconstruc­tion of this joint. All tissue biopsy specimens were sterile, and as the parameters that had indicated inflammation were within normal limits at that time, the antibiotic therapy was stopped. The patient continues to do well.

Discussion

The literature from 1960 to 1995 was searched with use of MEDLINE and *Index Medicus* to identify reports of human cases of actinomycosis due to *A. meyeri*. Because of the taxo­nomic changes over the years, we used the terms *Actinomyces meyeri*, *Actinobacterium meyeri*, and *Streptothrix meyeri*. We found 26 well-documented cases of human infection with *A. meyeri* (table 1). Before 1960 a few cervicofacial and pulmo-
Table 1. Summary of 26 cases of actinomycosis due to *Actinomyces meyeri* that have been reported between 1960 and 1995.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Case no.</th>
<th>Sex of patient</th>
<th>Age (y)</th>
<th>Pathological findings</th>
<th>Treatment</th>
<th>Duration of antibiotic therapy (mo)</th>
<th>Coinfecting organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>[7]</td>
<td>2</td>
<td>M/16</td>
<td></td>
<td>Emphyema, hemotogenous spread to bone marrow</td>
<td>Clindamycin, rib resection, chest-tube drainage</td>
<td>6</td>
<td>None</td>
</tr>
<tr>
<td>[8]</td>
<td>3</td>
<td>M/40</td>
<td></td>
<td>Pneumonia, osteomyelitis of tibia, skin abscess</td>
<td>Penicillin</td>
<td>3</td>
<td>None</td>
</tr>
<tr>
<td>[9]</td>
<td>4</td>
<td>M/49</td>
<td></td>
<td>Chronic osteomyelitis of tibia and fibula, skin abscesses</td>
<td>Penicillin</td>
<td>4</td>
<td>None</td>
</tr>
<tr>
<td>[9]</td>
<td>5</td>
<td>M/34</td>
<td></td>
<td>Spondylitis, skin abscesses, suspected periosteitis</td>
<td>Penicillin, surgical drainage</td>
<td>4</td>
<td>None</td>
</tr>
<tr>
<td>[10]</td>
<td>6</td>
<td>M/58</td>
<td></td>
<td>Pyomyositis of calf, pleuropulmonary actinomycosis</td>
<td>Penicillin, surgical drainage</td>
<td>NA</td>
<td>None</td>
</tr>
<tr>
<td>[12]</td>
<td>8</td>
<td>M/44</td>
<td></td>
<td>Pneumonia, brain abscess, multiple skin abscesses</td>
<td>Amoxicillin</td>
<td>12</td>
<td><em>A. actinomycetemcomitans</em></td>
</tr>
<tr>
<td>[13]</td>
<td>9</td>
<td>M/46</td>
<td></td>
<td>Pneumonia, skin abscess</td>
<td>Penicillin, surgical debridement</td>
<td>4</td>
<td><em>Capnocytophaga species</em></td>
</tr>
<tr>
<td>[PR]</td>
<td>10</td>
<td>M/47</td>
<td></td>
<td>Pneumonia, skin abscesses, osteomyelitis of the tibia</td>
<td>Penicillin, surgical debridement</td>
<td>12</td>
<td><em>A. actinomycetemcomitans</em></td>
</tr>
<tr>
<td>[14]</td>
<td>11</td>
<td>F/70</td>
<td></td>
<td>Pneumonia</td>
<td>Penicillin</td>
<td>NA</td>
<td>None</td>
</tr>
<tr>
<td>[16]</td>
<td>12</td>
<td>M/49</td>
<td></td>
<td>Pneumonia, empyema (6 mo later)</td>
<td>Clindamycin, tetracycline, chest-tube drainage</td>
<td>6</td>
<td><em>A. actinomycetemcomitans</em>, <em>B. ureolyticus</em>, <em>Peptostreptococcus prevosii</em> <em>Eikenella corrodens</em></td>
</tr>
<tr>
<td>[15]</td>
<td>13</td>
<td>M/55</td>
<td></td>
<td>Asymptomatic mass in lung</td>
<td>Lobectomy</td>
<td>3</td>
<td>None</td>
</tr>
<tr>
<td>[16]</td>
<td>14</td>
<td>M/13</td>
<td></td>
<td>Pneumonia with cervical extension</td>
<td>Penicillin</td>
<td>3</td>
<td>None</td>
</tr>
<tr>
<td>[17]</td>
<td>15</td>
<td>M/45</td>
<td></td>
<td>Bilateral pneumonia</td>
<td>Ceftiraxone, penicillin</td>
<td>12</td>
<td><em>Actinomyces israelii</em></td>
</tr>
<tr>
<td>[18]</td>
<td>16</td>
<td>M/40</td>
<td></td>
<td>Liver abscess</td>
<td>Penicillin, clindamycin, percutaneous drainage</td>
<td>1</td>
<td><em>Streptococcus milleri</em></td>
</tr>
<tr>
<td>[19]</td>
<td>17</td>
<td>M/30</td>
<td></td>
<td>Liver abscess</td>
<td>Penicillin + clindamycin, then doxycycline</td>
<td>3</td>
<td><em>F. nucleatum</em></td>
</tr>
<tr>
<td>[20]</td>
<td>18</td>
<td>F/40</td>
<td></td>
<td>Liver abscess</td>
<td>Penicillin + clindamycin, then penicillin; percutaneous drainage</td>
<td>NA</td>
<td><em>Streptococcus anginosus</em></td>
</tr>
<tr>
<td>[21]</td>
<td>19</td>
<td>M/50</td>
<td></td>
<td>Chronic osteomyelitis of tibia</td>
<td>Penicillin, soquestrectomy</td>
<td>4</td>
<td><em>Bacteroides species</em>, <em>Streptococcus mitis</em> <em>Propionibacterium acnes</em></td>
</tr>
<tr>
<td>[22]</td>
<td>20</td>
<td>M/50</td>
<td></td>
<td>Osteomyelitis of first metatarsus</td>
<td>Antibiotics, surgical debridement</td>
<td>NA</td>
<td>None</td>
</tr>
<tr>
<td>[23]</td>
<td>21</td>
<td>F/46</td>
<td></td>
<td>Spinal epidural abscess</td>
<td>Pefloxacin + rifampin</td>
<td>NA</td>
<td>None</td>
</tr>
<tr>
<td>[24]</td>
<td>22</td>
<td>F/29</td>
<td></td>
<td>Recurrent breast abscess</td>
<td>Ampicillin, doxycycline, surgical debridement</td>
<td>4</td>
<td>Diverse anaerobes</td>
</tr>
<tr>
<td>[24]</td>
<td>23</td>
<td>F/36</td>
<td></td>
<td>Recurrent breast abscess</td>
<td>Tetracycline, doxycycline, surgical debridement</td>
<td>4</td>
<td>Diverse anaerobes</td>
</tr>
<tr>
<td>[25]</td>
<td>24</td>
<td>M/64</td>
<td></td>
<td>Cervical abscess</td>
<td>Antimycobacterial therapy (three agents)</td>
<td>3</td>
<td><em>A. actinomycetemcomitans</em></td>
</tr>
<tr>
<td>[26]</td>
<td>25</td>
<td>M/24</td>
<td></td>
<td>Cervicofacial actinomycosis</td>
<td>Penicillin, surgical debridement</td>
<td>9</td>
<td>None</td>
</tr>
<tr>
<td>[27]</td>
<td>26</td>
<td>F/28</td>
<td></td>
<td>Brain abscess</td>
<td>Penicillin, neurosurgical drainage</td>
<td>2</td>
<td><em>Streptobacillus moniliformis</em></td>
</tr>
</tbody>
</table>

NOTE: NA = not available; PR = present report.
Pulmonary infection was present. Because most of the patients had dental disease, direct hematogenous spread from the oral cavity was often the source of the hematogenous dissemination [32]. It has been postulated that pulmonary infection is often the source of the hematogenous dissemination [32]. This assumption is supported by the observation that in eight of 10 cases of disseminated actinomycosis due to A. meyeri, pulmonary infection was present. Because most of the patients had dental disease, direct hematogenous spread from the oral cavity also may have caused disseminated infection as well as localized actinomycosis [9, 22, 24].

Actinomycetes are oral saprophytes that particularly affect periodontal lesions and carious teeth [30]. As with other actinomycoses, dentogingival disease is a major risk factor for the development of infections due to A. meyeri: 18 (69%) of the 26 patients in this review had poor dentition or a recent history of tooth extraction. Alcoholics are at risk for pulmonary actinomyces because they often have poor dentition, and they are prone to aspiration of oral secretions. In our series, 11 patients (42%) were alcoholics; 10 of these patients had pulmonary actinomycosis.

Actinomycosis can develop at the site of surgical procedures [30]; previous surgery at the site of infection due to A. meyeri was noted in two cases [21, 23]. However, it remains unclear whether infection occurred as a result of direct contamination or as a result of hematogenous spread of the oropharyngeal flora secondary to intubation maneuvers. Two-thirds (65%) of the infections with A. meyeri were polymicrobial, and up to five different bacteria have been cultured from an infected site (table 1). It is of interest that all of these associated pathogens, except one, also belonged to the human oropharyngeal flora. Anaerobic bacteria or facultative anaerobic bacteria predominated in these infections, and A. actinomycetemcomitans, an aerobic gram-negative bacillus that classically is isolated along with Actinomyces species, was recovered in only five cases. Hypothetical mechanisms by which associated bacteria may enhance the pathogenicity of Actinomyces species include reduced oxygen tension and anaerobiosis-induced inhibition of phagocytes [30].

Of 26 infections with A. meyeri, we found that 10 (38%) were disseminated (defined as the involvement of two distant organs). The lungs, the skin, the long bones, the liver, the brain, and the muscles were the sites involved, in order of frequency. Disseminated actinomycosis has been described on rare occasions [30], but according to one study, dissemination may actually occur more frequently [31]. The propensity of A. meyeri to disseminate is difficult to explain because this organism does not differ from other actinomycetes in its microbiological characteristics. It has been postulated that pulmonary infection is often the source of the hematogenous dissemination [32]. This assumption is supported by the observation that in eight of 10 cases of disseminated actinomycosis due to A. meyeri, pulmonary infection was present. Because most of the patients had dental disease, direct hematogenous spread from the oral cavity also may have caused disseminated infection as well as localized actinomycosis [9, 22, 24].

Cervicofacial actinomycosis, which accounts for approximately one-half of cases of actinomycosis, is the commonest form and results from contiguous extension of disease from the oral cavity [30]. Such infection due to A. meyeri has been reported for only two (8%) of 26 patients, but, for two reasons, we suspect that it occurs more often. In the majority of cases, the diagnosis of actinomycosis is based on the clinical history, on typical histological findings, or on culture of Actinomyces species, and identification of the exact microorganism is rarely required in clinical practice [25]. In addition, many cases of lumpy jaw due to A. meyeri will not appear in the literature, while a case of systemic infection due to the same microorganism will certainly be published.

Pulmonary actinomycosis accounts for 15% of all cases of actinomycosis [30] and generally is caused by aspiration of oral secretions when dental disease is present. Infections with A. meyeri involved the lung in 13 (50%) of 26 cases. It is unclear why this clinical presentation was so preponderant, although the relative rarity of reports of the cervicofacial form of the infection probably causes a statistical bias.

Abdominal actinomycosis is reported in about 20% of cases, and liver involvement is infrequent [30]. We found four cases of liver abscess due to A. meyeri. Three were isolated lesions that were probably the result of the seeding, via the portal vein, of an occult focus of infection along the gastrointestinal tract [18–20]. No abdominal site other than the liver was infected by A. meyeri.

Actinomycosis of the CNS is rare; A. meyeri was recovered in two cases of brain abscess, which is the most common clinical presentation of CNS actinomycosis [30].

Like other actinomycetes, A. meyeri is susceptible to most antibiotics, and penicillin remains the most cost-effective drug for treating infections due to this organism. Clindamycin, tetracyclin, erythromycin, or ceftriaxone may be alternatives [30]. Because actinomycosis leads to the formation of scarred, amorphous tissue into which the penetration of antibiotics is poor, a prolonged course of antibiotic therapy (6–12 months) has been recommended [30], but the duration of therapy remains controversial. In our series, the mean duration of antibiotic therapy was 6 months (range, 1–12 months) (table 1). It is interesting that no recurrence of infection was observed among 10 patients treated for 3–4 months. The associated bacteria were generally susceptible to the antibiotics used against A. meyeri. Five strains of the associated organisms had variable susceptibility, and one was resistant to the antibiotic regimens used, but even so, cure was achieved. These data are insufficient to conclude whether the presence of coinfectants may lead to treatment failure. The outcome was favorable for all but one of the 26 patients treated for A. meyeri actinomycosis [6].

Conclusion

Our patient had disseminated actinomycosis due to A. meyeri, which involved the lung, bone, and skin. Cure was achieved
with a regimen consisting of an 8-week course of intravenous penicillin followed by a 12-month course of oral penicillin, in addition to surgical debridement of an osteomyelitic focus.

Infections with *A. meyeri* are rare; however, they are probably underdiagnosed because identification of the exact *Actinomyces* species is not necessary for adequate treatment. Dentogingival disease and alcoholism are important risk factors that permit the development of pulmonary infection via aspiration of mouth flora, including *A. meyeri*. Hematogenous seeding to distant organs occurs particularly frequently with *A. meyeri*, compared with other actinomyces, and often originates from a pulmonary focus of infection that should be carefully sought. Treatment with penicillin for several months, coupled with surgical procedures when necessary, results in a good prognosis, even when localizations are multiple.

Acknowledgments

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References


