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Don't Let Its Name Fool You: Relapsing Thoracic Actinomycosis Caused by *Pseudopropionibacterium propionicum* (Formerly *Propionibacterium propionicum*)

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 71-year-old
Final Diagnosis: Thoracic actinomycosis caused by Propionibacterium propionicum
Symptoms: Fever
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Diagnostic/therapeutic accidents

Background: *Pseudopropionibacterium propionicum* was called *Propionibacterium propionicum* until a recent taxonomy change in 2016. Diseases caused by *P. propionicum* resemble actinomycosis and thus differ dramatically from the infectious syndromes caused by common cutaneous *Propionibacterium* spp. However, if treating physicians are not familiar with *P. propionicum* and its clinical presentations, it is possible for them to regard it as a skin contaminant such as *Cutibacterium acnes* (formerly *Propionibacterium acnes*).

Case Report: A 71-year-old man with past surgical history of right pneumonectomy was admitted with right chest wall abscess and right empyema. The chest wall abscess was drained surgically, and the empyema was drained via a chest tube. The abscess culture took 5 days to grow beaded branching Gram-positive rods, and 15 days to identify them as *P. propionicum*. The patient received 17 days of ceftriaxone and 4 weeks of doxycycline. However, he experienced a relapse of the chest wall abscess and right empyema 4 months after discontinuation of doxycycline. Cultures from the chest wall abscess and empyema grew *P. propionicum* again. We treated him with ceftriaxone for 6 months followed by minocycline for 7 months along with adequate drainage.

Conclusions: It is important to recognize that *P. propionicum* can cause thoracic actinomycosis and will likely require the prolonged treatment course typical for actinomycotic disease, which is 2 to 8 weeks of intravenous antibiotic therapy followed by 6 to 12 months of oral antibiotic therapy.

MeSH Keywords: Actinomycosis • Classification • Empyema, Pleural • Propionibacterium

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/919775

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Background

Pseudopropionibacterium propionicum is an anaerobic Gram-positive rod that was initially named *Actinomyces propionicus* and subsequently renamed *Arachnia propionica* before being reclassified into the genus *Propionibacterium* in 1988 [1] and more recently into the genus *Pseudopropionibacterium* [2]. At the same time, most cutaneous *Propionibacterium* species were reclassified into the new genus *Cutibacterium*, whereas *Propionibacterium propionicum* was placed into a different genus as a taxonomic outlier. Of great clinical importance, diseases caused by *P. propionicum* resemble actinomycosis and thus differ dramatically from the infectious syndromes caused by common cutaneous *Propionibacterium* spp. [3]. Although few cases of disease due to *P. propionicum* have been reported, in the present case report we describe an infection that manifested as recurrent empyema necessitans that relapsed after 6 weeks of antibiotic therapy, which are clinical findings that typify thoracic actinomycosis. We suspect that unfamiliarity with the relevant microbial nomenclature and the resultant failure to recognize the clinical implications of recovering *P. propionicum* from the site of infection, and its potential to cause thoracic actinomycosis contributed to the initially unsuccessful therapy and a complicated clinical course.

Case Report

A 71-year-old man was admitted to our hospital with a right chest wall abscess and fever for 2 days. His past medical history included coronary artery disease, hypertension, atrial fibrillation, and gastroesophageal reflux disease. He had a history of penicillin allergy that manifested as skin rash.

Two years prior to the current admission to our hospital, the patient underwent a right pneumonectomy via posterolateral thoracotomy for complications of a right upper lobe abscess caused by methicillin-resistant *Staphylococcus aureus*. The pneumonectomy was complicated by a bronchopleural fistula (BPF) and empyema. Surgical closure of the BPF was unsuccessful and resulted in recurrent BPF and the need for drainage of several chest wall abscesses.

The patient was admitted to our cardiothoracic surgery service 5 months after the last chest wall drainage with recurrent right-sided empyema with cultures that grew *Mucor* sp. After initiation of therapy with liposomal amphotericin B and thoracoscopic drainage of the empyema, the patient underwent successful closure of the proximal right mainstem bronchus stump to control the BPF. Liposomal amphotericin B was continued postoperatively for 4 weeks and followed by posaconazole for 8 months, with therapy completed 3 months before the current admission. He did well clinically for 1 year, with all surgical incisions completely healed and no detection of *Mucor* sp. in culture or stains.

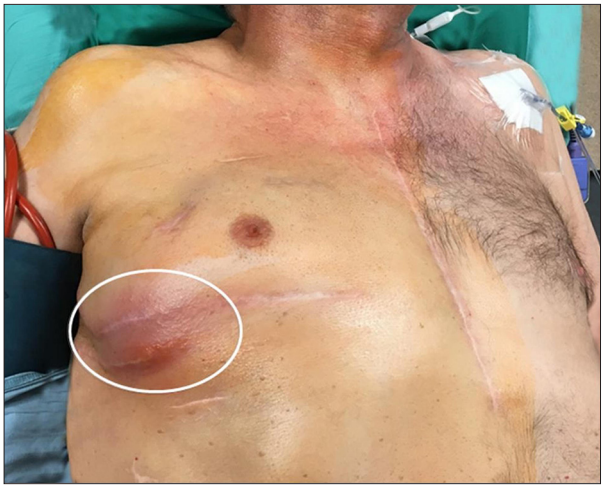

Figure 1. Preoperative photograph of the chest wall, demonstrating large chest wall abscess (circled). Note multiple healed thoracotomy and sternotomy scars.

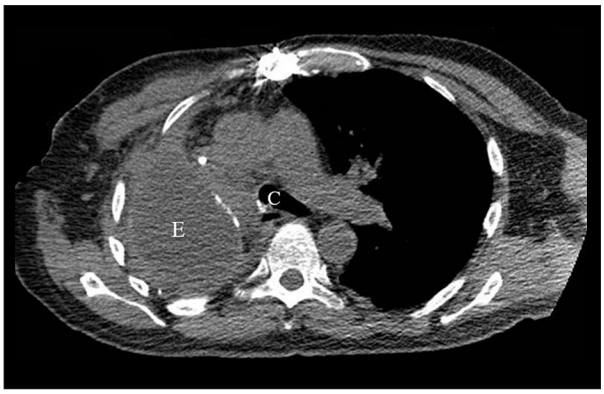

Figure 2. CT demonstrating fluid collection in the upper chest, consistent with empyema (E). Absence of air bubbles in the area of carina (C) suggests absence of bronchopleural fistula. Chest wall abscess was located lower and is not seen on this image.

On presentation on the current admission, he was afebrile and other vital signs were normal. His physical examination was remarkable for a fluctuant, right-sided chest wall swelling with blanching erythema, most consistent with an abscess (Figure 1). His initial laboratory data included a white blood cell (WBC) count of 15.6×10^9/L, hemoglobin level 9.5 g/dL, platelet count of 558×10^9/L, BUN 10 mg/dL, and creatinine 0.7 mg/dL. A chest computed tomography (CT) scan showed right pleural effusion and chest wall soft tissues changes consistent with an abscess and suggestive of empyema necessitans (Figure 2).

Incision and drainage (I&D) of the chest wall abscess yielded thick, white purulence with many polymorphonuclear

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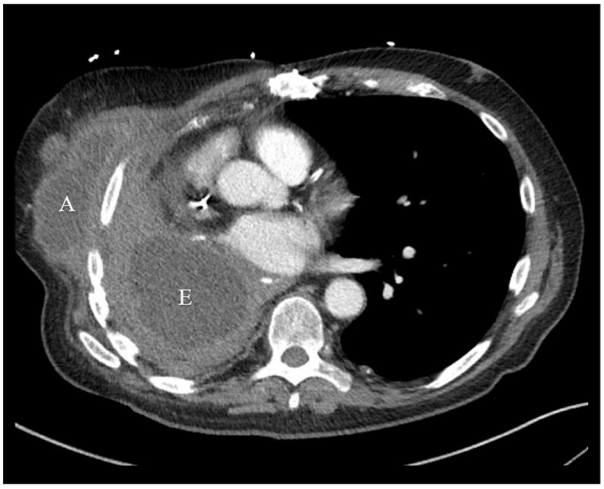

Figure 3. CT demonstrating empyema (E) and recurrent chest wall abscess (A). Their nearly contiguous location is consistent with empyema necessitans.

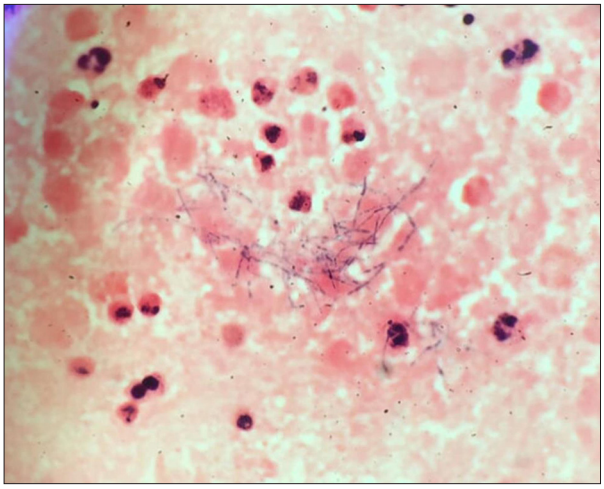

Figure 4. Gram stain of right pleural effusion. Beaded branching Gram-positive rods were seen.

neutrophils but no microorganisms seen on Gram stain. No obvious fistulous communication with the pleural space was found, and intraoperative flexible bronchoscopy confirmed the absence of recurrent BPF. Image-guided percutaneous tube thoracotomy was performed to drain the empyema. As no organisms were seen on examination of the pleural fluid, the patient was empirically treated with vancomycin, cefepime, metronidazole, and liposomal amphotericin B while awaiting results of pending cultures. He remained afebrile during his hospital stay, and vancomycin, cefepime, and metronidazole were discontinued on the third hospital day, as aerobic cultures from both the chest wall abscess and the right pleural effusion were negative at 48 h after the procedure. With a presumptive diagnosis of a relapse of his pleural space infection due to *Mucor* sp., the patient was started on posaconazole and discharged on the ninth day of hospitalization. Although all the abscess cultures were negative at the time of discharge, 3 days later anaerobic cultures from the right pleural effusion grew Gram-positive rods that were subsequently identified as *P. propionicum* by 16S rRNA sequencing. He received ceftriaxone for 17 days followed by doxycycline for 4 weeks. A chest X-ray taken at the time of transition to doxycycline demonstrated a stable right pleural effusion. The chest tube was removed 2 months after discharge and he was clinically stable and asymptomatic when seen in the Infectious Diseases clinic 4 months after discharge and ~3 months after discontinuation of doxycycline. The wound at the site of abscess drainage was managed with packing and healed.

Four weeks after his visit to the Infectious Diseases clinic, the patient presented to the Emergency Department with recurrence of the right chest wall abscess. His WBC count was 13.1×10^9/L, hemoglobin level 13.3 g/dL, and platelet count 295×10^9/L. A chest CT scan demonstrated the previously visualized loculated fluid collection in the subcutaneous tissues of the right lateral chest wall and right pleural space (Figure 3). Repeated I&D of the chest wall abscess and a chest tube insertion were performed. A Gram stain of the right pleural effusion revealed beaded Gram-positive rods (Figure 4), and the patient's empiric antibacterial therapy was changed to ceftriaxone. Anaerobic cultures from the pleural effusion and the chest wall abscess started to grow after 5 and 8 days in culture, respectively. Analysis by 16S rRNA sequencing identified the Gram-positive rods from the chest wall abscess and pleural effusion as *P. propionicum*.

The chest wall sinus tract at the site of the abscess I&D was managed with packing until it closed in 2 months. The chest tube was removed at that time. Ceftriaxone was continued for 6 months before switching to oral antibiotics. Initial therapies with amoxicillin and then cephalexin were complicated by skin rash, which prompted replacement with minocycline to complete a 7-month course of oral antibiotic treatment. After discontinuation of therapy, he has been doing well clinically without signs of recurrent infection for three months.

Discussion

We report a case of recurrent empyema necessitans caused by *P. propionicum* after an initial surgical drainage and a course of antibiotics. The presentation and course resembled thoracic actinomycosis, but the causative agent was *P. propionicum*. We suspect that early recognition of the virulence of *P. propionicum* as a potential pathogen would have prompted a therapeutic plan more consistent with that for actinomycotic infection and more likely to have prevented the relapse that our patient experienced.

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The organism currently referred to as *P. propionicum* has previously had other names. Based on the presence of diaminopimelic acid in its cell wall and its ability to produce propionic acid from glucose, taxonomists changed its original name, *Actinomyces propionicus* [4], to *Arachnia propionica* in 1969 [5]. Data from 16S rRNA sequencing prompted its temporary reclassification into the genus *Propionibacterium* [1] until its current designation into the genus *Pseudopropionibacterium* [2].

P. propionicum has been implicated in several clinical diseases, including lacrimal canaliculitis [6,7], cervicofacial infections [8,9], tympanomastoiditis [10], pulmonary infections [11,12], osteomyelitis [13,14], pelvic abscess related to intra-uterine device (IUD) [15], psoas abscess [16], and brain abscess [17,18]. However, despite its genetic association with the genus *Propionibacterium*, *P. propionicum* can produce disease that mirrors that of *Actinomyces israelii*, which is chronic, granulomatous infection characterized by abscess formation, tissue fibrosis, and the presence of draining sinuses [7,19], as illustrated by our patient. The failure to immediately recognize the clinical implications of recovering *P. propionicum* from patient samples reflects the confusing taxonomy and the challenges inherent in recovering this organism from clinical samples.

*Cutibacterium (*formerly *Propionibacterium) acnes,* the most well-known species in the genus *Cutibacterium*, resides in the normal flora of human skin and mucosal surfaces and is frequently considered a contaminant when recovered from clinical specimens. We suspect that the isolation of *P. propionicum* from our patient was misconstrued as a contaminant and not recognized as an organism with the potential to cause actinomycosis, as both organisms at the time were members of the genus *Propionibacterium*. Had the beaded, branching Gram-positive rods of *P. propionicum* or sulfur granules been seen in purulent material obtained from the abscess or empyema fluid of our patient, the correct diagnosis of actinomycosis would have been made, despite unfamiliarity with *P. propionicum*.

Further complicating management of this patient was the challenge of identifying *P. propionicum* in culture, which, similar to *A. israelii*, grows very slowly and is difficult to identify. In this case, the initial pleural fluid sample grew pinpoint colonies anaerobically at 5 days, during an extended anaerobic culture that was prompted by a Gram stain consistent with *Actinomyces* or a similar organism. These evolved to colonies large enough to analyze with MALDI-ToF (Bruker Daltonics BioTyper) after another 10 days, but returned an unacceptable score (1.47) even with full extraction of the organism with the proper identification of *P. propionicum*. 16S ribosomal sequencing was required for definitive identification, which was made 22 days after specimen submission. A sample obtained at the initial I&D was not sent for histologic exam and may have otherwise been informative, although the organism load might have been inadequate to provide an early diagnosis. Most laboratories will have difficulty with culture and identification of *P. propionicum*, and, because this is not unique among agents that cause actinomycosis, a high index of clinical suspicion for actinomycosis is essential for the laboratory to pursue the atypically extensive effort necessary for successful identification of the causative agent.

Changes in taxonomy sometimes have unintended consequences. As in this patient, changes in taxonomy and reporting by the microbiology laboratory can be confusing to clinicians [20], particularly those in specialties other than infectious diseases. Reclassification of the genus *Propionibacterium* was recently performed, which reclassified *P. acnes* and other cutaneous *Propionibacterium* spp. into the genus *Cutibacterium*, and reclassified *Propionibacterium propionicum* as *Pseudopropionibacterium propionicum* [2]. These changes might reduce the chance of mistaking *P. propionicum* for a skin contaminant. However, it will take time for clinicians to become familiar with these changes in taxonomy.

Antimicrobial susceptibilities of *P. propionicum* are similar to those of *Actinomyces* spp. It is uniformly susceptible to penicillins and other beta-lactam antimicrobials, and generally susceptible to minocycline and clindamycin [21], but it is uniformly resistant to metronidazole.

Conclusions

It is important to recognize that *P. propionicum* can cause thoracic actinomycosis and will likely require the prolonged treatment course typical for actinomycotic disease; *i.e.,* 2 to 8 weeks of intravenous antibiotic therapy followed by 6 to 12 months of oral antibiotic therapy [22,23].

Conflict if interests

None.

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