# Pleural empyema caused by Actinomyces turicensis

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### **Abstract**

Actinomyces turicensis was first identified in 1995. To the best of our knowledge, pleural empyema caused by A. turicensis has never been reported. In the case reported herein, a patient with pleural empyema was treated surgically, and in the bacterial samples, A. turicensis was isolated.

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Actinomyces turicensis was first reported in 1995 and is emerging as an important cause of infections [1]. Common clinical manifestations of A. turicensis infection include anogenital or urinary tract infections, breast abscess, and so on [2-8]. To the best of our knowledge, pleural empyema caused by A. turicensis has never been reported in the literature. In the case reported herein, a patient with pleural empyema was treated surgically, and in the bacterial samples, A. turicensis was isolated among other pathogens. A 51-year-old man was diagnosed with pleural empyema. He was a worker in the building industry. He was a heavy smoker, but he quit smoking 3 months before. He was a heavy alcohol consumer, but he declined drug abuse. He suffered chronic obstructive pulmonary disease and sleep apnoea syndrome. He was on treatment with methylprednisolone (16 mg per day) for about two months because of suspicion of extrinsic allergic alveolitis. Upon admission, the white blood cell count was  $17.42 \times 10^3/\mu L$  (93.4% neutrophils), the C-reactive protein level was 251.4 mg/L, and the procalcitonin level was 0.11 µg/L. A chest computed tomography scan revealed loculated left pleural effusion. The PCR test of the nasopharyngeal swab was negative for severe acute respiratory syndrome coronavirus 2.

Diagnostic thoracocentesis was performed, and purulent fluid was retrieved. An empirical treatment (intravenous amoxicillin/clavulanic acid, 4 g per day) was initiated. The next day, the patient was scheduled for surgery.

Debridement and decortication were performed.

The cultures of the liquid retrieved by thoracocentesis resulted in growth of A. turicensis (ceftriaxone-sensitive, ampicillin-sensitive, clindamycin-resistant, and amoxicillin/clavulanic acid-sensitive), Fusobacterium necrogenes, and Micromonas micros (multisensitive). The presence of A. turicensis was confirmed by 16S sequencing.

The cultures of the perioperative pleural samples resulted in growth of A. turicensis and M. micros. Intravenous ornidazole (I g per day) was added for a duration of 16 days.

The initial intravenous treatment was modified to an oral treatment with amoxicillin/clavulanic acid for a total duration of 6 months. The patient was discharged after 3 weeks of hospital stay in good general condition.

Actinomyces species are commensal flora of the mucosa of the oropharynx, gastrointestinal tract, and female genital tract [1–3]. The breach of the different mucosal barriers (resulting from trauma, surgery, or foreign bodies) offers an entry to the deeper planes and results in infection [2]. In the case reported herein, the most likely pathogenetic mechanism was inhalation of oropharyngeal secretions as the patient was an alcohol

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abuser and had poor oral hygiene. In addition, the underlying lung disease and the prolonged treatment with corticosteroids should be also considered as predisposing risk factors. The occupation of the patient should be also taken into account as he was likely exposed to inhalation of dust particles.

The following types of infections attributed to A. turicensis have been reported: breast abscess, pilonidal abscess, pyometra, spontaneous peritonitis without abscess formation, appendicitis, perianal abscess, meningitis, otogenic brain abscess, prostatic abscess, necrotizing soft-tissue infections, endocarditis of the eustachian valve, bacteraemia with or without predisposing risk factors, and so on [2–8]. A. turicensis is frequently isolated in a mixture of other microorganisms (e.g. Prevotella bivia, Peptostreptococcus spp., Peptoniphilus harei, and so on), as it was the case of our patient.

Identification by conventional laboratory methods is difficult; for that reason, molecular methods, such as the I6S rRNA sequence analysis, are often needed [9]. The I6S rRNA gene sequence contains hypervariable regions that constitute specific signatures used to identify bacteria [9]. On the other hand, it also permits to reclassify bacteria into completely new species. In the case of *A. turicensis*, the difficulty in its identification is attributed to the slow growth of this pathogen. The antibiotic agents of choice are ß-lactam, especially when combined with ß-lactamase inhibitors [10]. An initial intravenous administration is recommended for two to six weeks and then orally for 6 to 12 months. High resistance to metronidazole and ciprofloxacin has been reported [10].

In our experience, there was no difference in clinical presentation, diagnosis, and treatment in that particular case, in comparison with other actinomycotic empyemas (due to Actinomyces odontolyticus and Actinomyces meyeri) that we have already treated. Advanced molecular diagnostic workup is the gold standard to identify these difficult-to-isolate pathogens. Treatment of this type of empyema should be the same as for any other typical pleural empyema. Antimicrobial susceptibility

has to be taken into account to administer an efficient longterm antibiotherapy, taking also into account that polymicrobial infections are frequent in that particular setting.

## **Transparency declaration**

The authors have no conflict of interest nor source of funding to declare.

#### References

- [I] Wust J, Stubbs S, Weiss N, et al. Assignment of Actinomyces pyogenes-like (CDC coryneform group E) bacteria to the genus Actinomyces as Actinomyces radingae sp. nov. and Actinomyces turicensis sp. nov. Lett Appl Microbiol 1995;20:76–81.
- [2] Sabbe LJ, Van De Merwe D, Schouls L, et al. Clinical spectrum of infections due to the newly described Actinomyces species A. turicensis, A. radingae, and A. europaeus. J Clin Microbiol 1999;37:8–13.
- [3] Könönen E, Wade WG. Actinomyces and related organisms in human infections. Clin Microbiol Rev 2015;28:419–42.
- [4] Attar KH, Waghorn D, Lyons M, et al. Rare species of actinomyces as causative pathogens in breast abscess. Breast J 2007;13:501–5.
- [5] Oh HB, Abdul Malik MH, Keh CH. Pilonidal abscess associated with primary Actinomycosis. Ann Coloproctol 2015;31:243–5.
- [6] Kocsis B, Tiszlavicz Z, Jakab G, et al. Case report of Actinomyces turicensis meningitis as a complication of purulent mastoiditis. BMC Infect Dis 2018;18:686.
- [7] Barnes A, Kaur A, Augenbraun M. An unusual presentation of prostatic abscess due to Actinomyces turicensis and Peptostreptococcus. Cureus 2020:12:e8665.
- [8] Panwar K, Duane TM, Tessier JM, et al. Actinomyces turicensis necrotizing soft-tissue infection of the thigh in a diabetic male. Surg Infect (Larchmt) 2019;20:431–3.
- [9] Hall V, O'Neil GL, Magee JT, et al. Development of amplified 16S ribosomal DNA restriction analysis for identification of Actinomyces species and comparison with pyrolysis-mass spectrometry and conventional biochemical tests. | Clin Microbiol 1999;39:2255–61.
- [10] Steininger C, Willinger B. Resistance patterns in clinical isolates of pathogenic Actinomyces species. J Antimicrob Chemother 2016;71: 422-7