



Case report

Actinomyces viscosus causing disseminated disease in a patient on methotrexate

Saad Habib^{a,*}, Abdul Hasan Siddiqui^b, Mohammed Azam^b, Faraz Siddiqui^b, Michel Chalhoub^b

^a Department- Internal Medicine, Staten Island University Hospital, 475 Seaview Avenue, Staten Island, NY, 10304, USA

^b Staten Island University Hospital, USA

ABSTRACT

Actinomycosis is an uncommon infectious disease caused predominantly by *Actinomyces israelii*. Actinomycosis usually involves the cervicofacial, thoracic, abdominal, and pelvic region, dissemination is uncommon. *Actinomyces viscosus* are commensal organisms that make up the normal flora of the oropharynx of humans and has rarely been reported to cause disease. Here, we report a unique case of disseminated actinomycosis with lung and brain lesions caused by *Actinomyces viscosus* resembling lung cancer with metastasis in a 74 year old male. Cultures from skin lesions confirmed *A. viscosus*. Although the patient was immunocompromised, antibiotic treatment with a penicillin-based regimen was effective.

1. Introduction

Actinomycetes are recognized anaerobic pathogens of man. They most commonly cause indolent, slowly progressive, locally invasive and destructive tissue disease of the orocervical region, chest, abdomen and pelvis with *Actinomyces israelii* being the most common etiologic agent [4,10]. Disseminated actinomycosis has rarely been reported due to *A. israelii* and *A. meyeri*. Facultative anaerobes such as *Actinomyces viscosus* (*A. viscosus*) in spite of being a commensal, human adults has rarely been reported to cause disease [1]. We present a rare case of disseminated actinomycosis caused by *A. viscosus* in a patient on methotrexate (see Figs. 1 and 2).

2. Case presentation

A 74 year old male with past medical history of Chronic Obstructive Pulmonary Disease, chronic smoking, psoriatic arthritis on methotrexate presented to the hospital with complaints of generalized weakness and difficulty to ambulate for few weeks prior to presentation. On physical examination, the patient was noted to have bilateral lower extremity edema and subcutaneous nodules over the chest, posterior neck, and right thigh. Computed Tomography (CT) chest showed the presence of left upper lobe lung mass measuring 4.5 cm × 2 cm along with multiple pulmonary nodules and multiple low-density circular structures scattered throughout the retroperitoneum, mesenteric and retroperitoneal fat. Further imaging with Magnetic Resonance Imaging (MRI) of the brain revealed scattered rim enhancing lesions

with surrounding edema throughout the brain, largest being 1.4 cm in the left frontal lobe. Two weeks prior to presentation, the patient was admitted to outside hospital for a fall. He was found to have a posterior neck abscess for which incision and drainage was done. Cultures from the neck abscess grew *A. viscosus*. In addition, imaging studies revealed a lung mass measuring 5 × 2.5 cm for which biopsy was deferred by the patient. Subsequently, he was discharged on oral amoxicillin for two weeks.

Based on the clinical presentation and work up, a possibility of disseminated infection vs metastatic process was considered. CT chest was compared to the previous CT chest done at outside hospital. A significant reduction in the size of the lung mass compared to the previous CT scan was found. Given the fact that patient was on amoxicillin for two weeks, the significant reduction in the size of the lung mass on the CT scan compared to the previous imaging was more suggestive of an infectious etiology. A consensus was established that the multiple lesions found on imaging including brain lesions were secondary to a disseminated infection rather than metastatic disease. The patient was started on intravenous penicillin and methotrexate was held. The patient was eventually discharged home on intravenous penicillin G 4 million units every four hours then later to 1 million unit continuous infusion every one hour for six months. A follow-up imaging with CT chest and abdomen in subsequent visits showed complete resolution of lung mass within four weeks and significant improvement in all the other lesions. Subsequently, full resolution of all chest, abdomen and brain lesions were seen with an overall six months of antibiotic therapy.

* Corresponding author.

E-mail addresses: ssaadhabib@gmail.com (S. Habib), drsiddiqui07@gmail.com (A.H. Siddiqui), mohdazam7492@gmail.com (M. Azam), drfarazsiddiqui@gmail.com (F. Siddiqui), chalhoubmichel@gmail.com (M. Chalhoub).

<https://doi.org/10.1016/j.rmcr.2018.08.009>

Received 8 August 2018; Received in revised form 13 August 2018; Accepted 13 August 2018

2213-0071/ © 2018 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

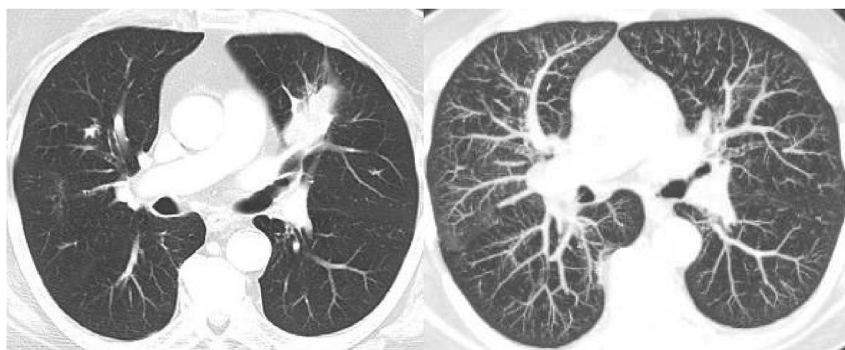


Fig. 1. Computed Tomography of chest showing left lung mass (left) and resolution of lung mass (right).

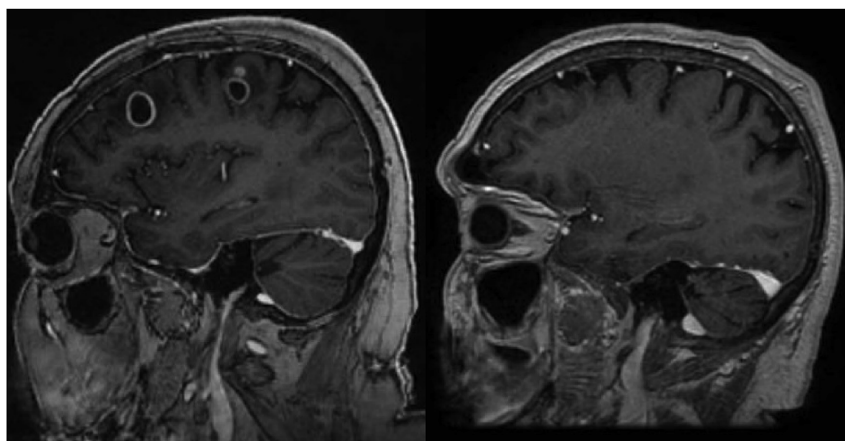


Fig. 2. Magnetic Resonance Imaging of brain showing rim enhancing lesions (left) and resolution of lesions (right).

3. Discussion

Actinomyces viscosus are normal flora of teeth in human adults and is usually known to cause disease in canines and other animals. Only a few cases have been reported in humans where it was mostly associated with subacute infections including cervicofacial, pulmonary, perioperative endophthalmitis, endocarditis & branchial cyst infections but never a disseminated infection. Two serotypes of *A. viscosus* have been described, serotype 1 and serotype 2. Serotype 1 was isolated from hamster and animals whereas serotype 2 from humans [1,2,7,9]. To the best of our knowledge, our case is the first reported case of a disseminated actinomycosis secondary to *A. viscosus* in existing literature. Methotrexate could be a reason for the dissemination as its use is associated with immunosuppression [12]. Disseminated actinomycosis has been seen usually with *A. israelii*, *A. meyeri*, *A. odontolyticus* [4,6,8]. Most actinomycotic infections are polymicrobial and include other flora such as *Actinobacillus*, *Fusobacterium*, and *Peptostreptococcus* species [6].

There is no clinical characteristic of *A. viscosus* infection that can distinguish it from other actinomyces species. However, microbiologically *A. viscosus* is a gram-positive, non-acid fast, facultative anaerobe that grows as acute-angle branching filamentous rods. Unlike other species which are catalase-negative and indole-positive, *A. viscosus* colonies are catalase-positive and indole-negative [1]. The diagnosis is frequently made by histopathological examination of excised tissue, which has a characteristic “sulfur granule” appearance grossly and forms white to gray colonies. Gram stain shows branching gram-positive rods that grow anaerobically. “Sulfur granules” contain filamentous or club-shaped structures which are Gram-positive but non-acid fast. This feature differentiates actinomycosis from nocardiosis [3,8].

Prolonged antibiotic therapy with penicillin or amoxicillin for six to

twelve months is required since it has poor drug penetration into fibrotic lesions. Adjunct therapy with surgical or percutaneous drainage or excision are done when indicated [6]. In penicillin-allergic patients, tetracyclines, erythromycin, clindamycin, cephalosporins, and Chloramphenicol can be used as alternatives [5]. Even though this patient was immunocompromised and the lesions were disseminated, antibiotic treatment proved to be effective. Actinomycosis usually shows a good response to antibiotic therapy. However, careful treatment and follow-up are required, especially in immunocompromised patients.

In summary, we describe a case initially presumed as lung cancer with brain metastasis in a patient who presented with generalized weakness, later confirmed as an uncommon disseminated infection due to *A. viscosus*. Our case not only highlights an unusual presentation of rare infection caused by *A. viscosus* but also signifies the importance of multidisciplinary involvement in the management of complicated cases and is, therefore, worth reporting in the field of medical literature. Physicians should be aware of the various species and presentations of Actinomycosis infection.

Disclosures

Consent was obtained by the patient. No conflict of interest by any authors. All authors have declared that no financial support was received from any organization.

References

- [1] R.H. Eng, M.L. Corrado, D. Cleri, C. Cherubin, E.J. Goldstein, Infections caused by *Actinomyces viscosus*, *Am. J. Clin. Pathol.* 75 (1) (1981) 113–116.
- [2] C. Adeniyi-Jones, J.A. Minielly, W.R. Matthews, *Actinomyces viscosus* in a branchial cyst, *Am. J. Clin. Pathol.* 60 (5) (1973) 711–713.
- [3] L.V. Holde, E.P. Cata, W.E.C. Moore, *Anaerobe Manual*, fourth ed., Virginia Polytechnic Institute, Blacksburg, 1977.

- [4] L. Legum, K. Greer, S. Glessner, Disseminated actinomycosis, *South. Med. J.* 71 (1978) 463–465.
- [5] K.A. Hamed, Successful treatment of primary actinomyces viscosus endocarditis with third-generation cephalosporins, *Clin. Infect. Dis.* 26 (1998) 211–212.
- [6] Hyun Jung Park, Ki-Ho Park, Sung-Han Kim, Heungsup Sung, Sang-Ho Choi, Yang Soo Kim, Jun Hee Woo, Sang-Oh Lee, A case of disseminated infection due to actinomyces meyeri involving lung and brain, *Infect. Chemother.* 46 (4) (2014 Dec) 269–273.
- [7] Mary A. Gerencser, John M. Slack, Identification of human strains of actinomyces viscosus, *Appl. Microbiol.* 18 (1) (1969 Jul) 80–87.
- [8] H. Takeda, Y. Mitsuhashi, S. Kondo, Cutaneous disseminated actinomycosis in a patient with acute lymphocytic leukemia, *J. Dermatol.* 25 (1) (1998) 37–40.
- [9] J. Scarano, M.S. Ruddat, A. Robinson, Actinomyces viscosus postoperative endophthalmitis, *Diagn. Microbiol. Infect. Dis.* 34 (2) (1999 Jun) 115–117.
- [10] K.G. Julian, L. de Flesco, L.E. Clarke, L.J. Parent, Actinomyces viscosus endocarditis requiring aortic valve replacement, *J. Infect.* 50 (4) (2005) 359–362.
- [12] L. Genestier, R. Paillot, S. Fournel, C. Ferraro, P. Miossec, J.P. Revillard, Immunosuppressive properties of methotrexate: apoptosis and clonal deletion of activated peripheral T cells, *J. Clin. Investig.* 102 (1998) 322–328.