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Case Report

ACTINOMYCOSIS: A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Actinomycosis is a rare infectious disease caused by Actinomycosis spp., anaerobic Gram-positive bacteria. Clinicians must be aware of typical clinical presentations of cervicofacial, thoracic, abdominopelvic and other forms of the disease. It is a suppurative and granulomatous inflammation with formation of multiple abscesses and sinus tracts that may discharge sulphur granules. Bacterial cultures and pathology are the cornerstone of diagnosis. Patients with actinomycosis require prolonged (6 to 12 months) high doses of penicillin G/amoxicillin, but the duration be shortened to 3 months in patients in whom optimal surgical resection of infected tissue has been performed. This article is intended to review the aetiology, clinical features, diagnosis and management of actinomycosis with a case report.

INTRODUCTION

Actinomycosis is a rare, subacute to chronic granulomatous disease caused by Actinomycocisspp., filamentous, Gram-postive,anaerobicbacteria.More than 30 species of actinomyces have been described. The most common ones are Actinomycesisraeli, A.gerensceriae, A.naeslundii, A.viscosus, A.turicensis, and A.meyeri. Actinomyces belong to the human commensal flora of the oropharynx, gastrointestinal tract and urogenital tract which becomes invasive through a mucosal lesion[3][2]. Actinomycosis infection could be polymicrobial and associated with other bacteria, named "companion microbes" [4] like Aggregatibacter actinomycetemcomitans, Eikenellacorodens which contributes to initiation and development of infection by inhibiting host defences or reducing oxygen tension.[2][10]

Actinomycosis is classified into distinct clinical forms according to the anatomical site infected: orocervicofacial, thoracic, abdominopelvic, central nervous system, musculoskeletal and disseminated.[1]

Oro cervicofacial actinomycosis is the most common form of the disease and comprises 50% of the reported cases. Dentalcaries, dental manipulation or trauma to the mouth can be triggering factors and even arise spontaneously in patients with poor dental hygiene.[3]The infection is charcaterised in initial stages by fever and chronic painful or painless soft tissue swelling(lumpy jaw) of the perimandibular region,which often forms sinus tracts and discharge purulent material containing sulphur granules.[2]Regional lympahadenopathy is typically absent until later stages[2]. Infection may also extend into local structures such as bone (periostitis and actinomycotic osteomyelitis). Firm woody consistency in later stages may mimic malignancy.

Thoracic actinomycosis accounts for 15-20%of cases. Usually results from aspiration of oropharyngeal secretions, but also occur after oesophageal perforation, local spread from cervicofacial or abdominal infection or from hemetagenous spread. It manifests as fever, chills, productive cough and pleural pain. It commonly presents as a pulmonary infiltrate or mass, which, if left untreated can involve pleura, pericardium and chest wall leading to the formation of sinuses that discharge sulphur granules.

Abdominopelvic actinomycosis makes upto 20% of cases. Patients typically have history of perforated acute appendicitis, gastrointestinal perforation, previous surgery, neoplasia and foreign bodies in gastrointestinal and genitourinary tract with

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or without erosion. Through he mucosal barrier. Patients may present with non-specific symptoms such as fever, weightloss, abdominal pain and sometimes a palpable mass. Pelvicactinomycosis is associated with prolonged use (>2 years) of intrauterine contraceptive devices.

Central nervous system infection usually arises from hematogenous spread, direct extension of orocervicofacial infection or following penetrating head injury. The disease presents as brain abscess, meningitis or meningoencephalitis, actinomycoma, subdural empyema and epidural abscess.[11]

Musculoskeletal infections are usually caused by spread from adjacent soft tissue, local trauma or hematogenous spread. The diagnosis of actinomycosis of any site is crucial. Blood investigation shows features of anemia, mildleucocytosis, raised ESR and C reactive protein values. Alkaline phosphatase concentration may bevraised in hepatic actinomycois.

Imaging features are usually non-specific in the early stages of infection but cross section imaging of CT and MRI scan provide accurate anatomical localisation which can aid tissue sampling.

Suppurative diagnosis is by demonstrating Gram positive filamentous organisms and sulphur granules on histological examination. Sulfur granules are round or oval basophilic masses with a radiating arrangement of eosinophlic terminal "clubs". Although the presence of sulphur granules is useful in making the diagnosis, they are not always recovered in culture confirmed cases of actinomycosis. In a study of 181 cases of actinomycosis, one to three granules were present in 56% of the cases and only one granule was present in 26%,none was present in seven cases.[8]And also granules are not specific to actinomycosis, because they are seen in Nocardiosis. Chromomycosis and Botryomycosis. A species specific fluroscentanibody allows rapid identification by direct staining, even after fixation of formalin.[9]

Definitive diagnosis could be made by direct isolation of the organism from a clinical specimen or sulfurgranules. But, the failure rate of isolation is high (>50%) because of previous antibiotic treatment, overgrowth of concomitant organisms or inadequate methodology.[5] The clinical specimens used are pus, tissue or sulphur granules. Swabs are not used, as the initial sample cannot be analysed with microscopy.

Actinomyces are slow growing organisms, cultured on selective agar medium at 37 C anaerobically for upto 3 weeks.[1][12][10] The organism is identified by colony morphology on agar and biochemical profiling. Serological assays have been developed but sensitivity and specificity need to be improved. Polymerase chain reaction, 16s rRNA sequencing, Fluroscence insitu hybridisation, mass spectrometry and Matrix associated laser desorption ionisation time of flight (MALDI-TOF) are new molecular genetic methods.[7]

All forms of actinomycosis is initially treated with high doses of (18-24 million units a day) of intravenous penicillin G over 2 to 6 weeks followed by oral penicillin V at a dose of 2-4g/day for 6 to 12 months. Though the risk of actinomycetes developing penicillin resistance is minimal, other alternative to penicillin are Ceftriaxone (2g IV/IM q 12-24h) ,Imipenem/Cilastin (500mg-1000mg IV q8h) Clindamycin(600 mg IV q8h)

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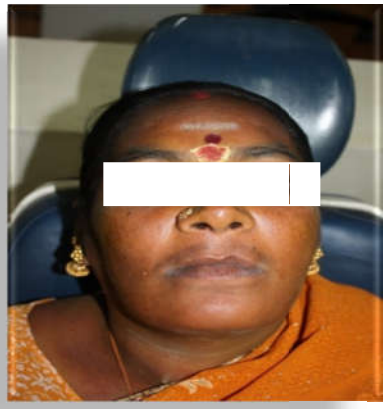
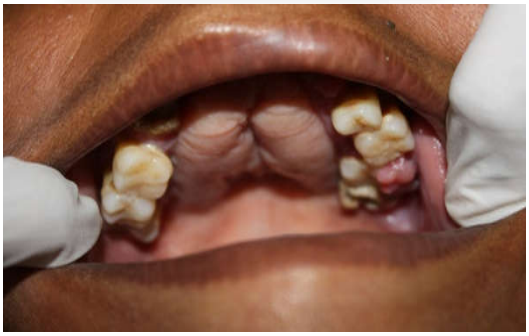
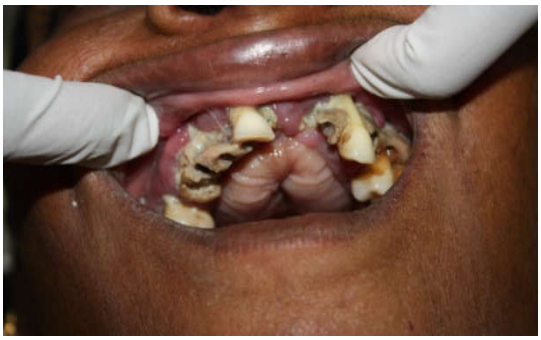
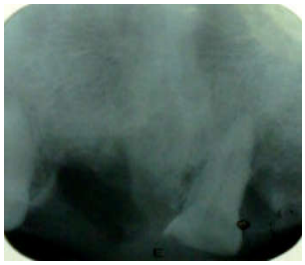
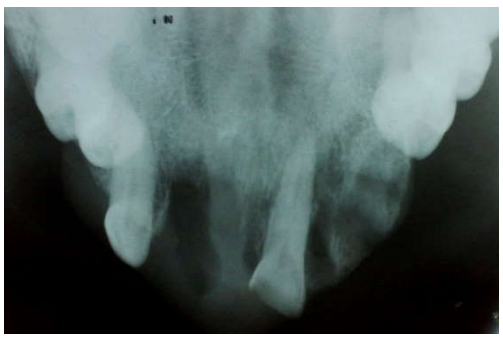
Amoxycillin/Clavulanicacid (500 mg q8h), Doxycycline (100 mg oral / IV q12h) Surgical resection of infected tissue may be necessary in some cases though initially treated with aggressive antimicrobial therapy.

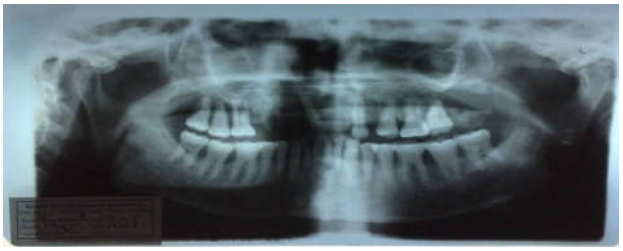


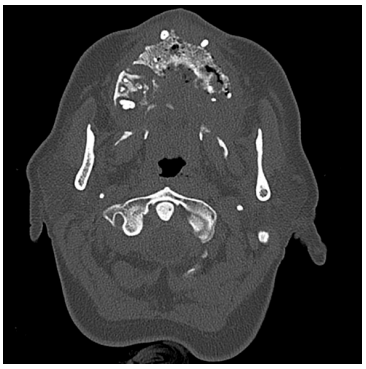


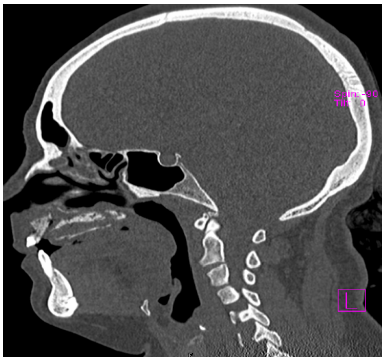


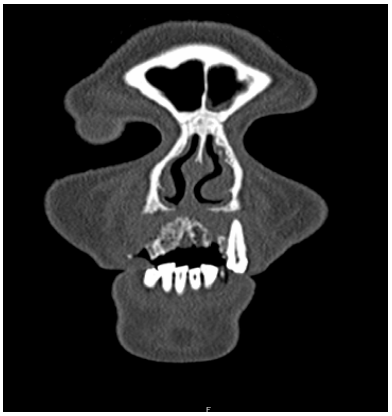


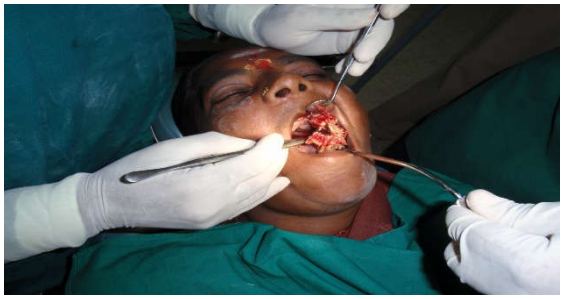












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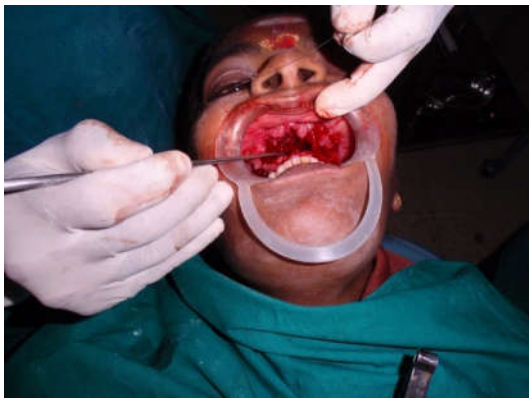

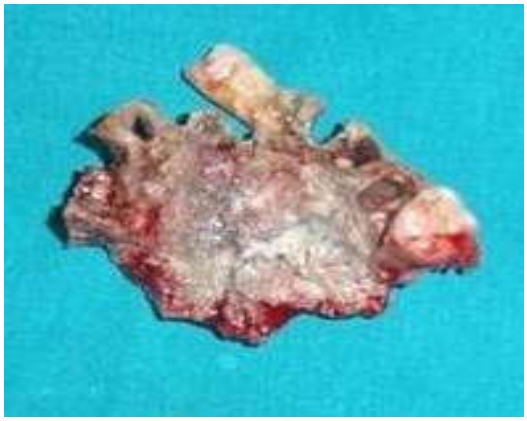

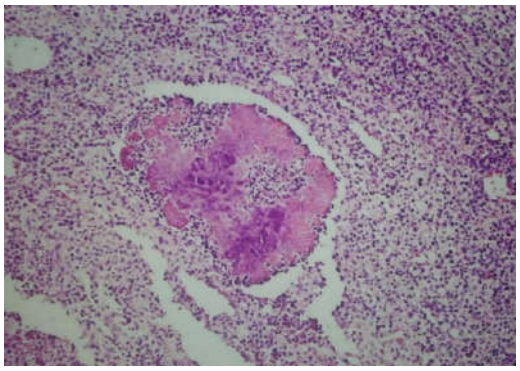

Histopathology

References

1. VKWong, TDTurmezei, VC Weston Actinomycosis: clinical review 20/0
2. Florentvalour, agathesenechal, celinedupieux, Judithkarsenty, sebasteinlustig Actinomycosis: Etiology, clinicalfeatures, diagnosis, treatment and management-Review 2014
3. M.Volante, A.M.Contucci, M.Fantoni, R.Ricci, J.Galli: Cervicofacial actinomycosis: still a difficult diagnosis: 2005
4. Moniruddin ABM, Begum H, Nahar K Actinomycosis: An update 2010
5. Bennhoff DF. Actinomycosis: diagnostic and therapeutic considerations and a review of 32 cases:1984
6. Miller PH, Wiggs LS, Miller JM. Evaluation of API An-IDENT and Rapid ANA II systems for identification of actinomyces species from clinical
7. Garner O, Mochon A, Branda J, *et al*. Multicentre evaluation of mass spectrometric identification of Actinomyces species from clinical specimens *J clin Microbial*. 1995
8. Brown JR. Human actinomycosis. A study of 181 subjects. *Hum Pathol* 1973
9. Smego RA Jr; FogliaG. Actinomycosis. *Clin infect Dis* 1998
10. Mandell GL, Bennett JE, Dolin R, editors. Mandell Douglas and Bennett's principles and practice of Infectious diseases 7thed.Philadelphia,PA: Churchill Livingstone ELSEVIER:2010
11. Smego RA Jr. Actinomycosis of the CNS: Rev Infect Dis 1987
12. Russo TA: Agents of Actinomycosis

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