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Review Article

Cervicofacial Manifestation and Clinical Significance of *Actinomyces Israelii*

Aws Mahmmod Ahmed*[1], Zahraa Amer Hashim[2], Zahraa Sedeeq Qasim[3], Karam A. Aldabbagh[4]

[1] *Oral and Maxillofacial Surgery Department, College of Dentistry, University of Mosul, Mosul, Iraq*
[2,3,4] *College of Pharmacy, University of Mosul, Mosul, Iraq*
* Corresponding author: awsalghazal@uomosul.edu.iq

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ABSTRACT: *Actinomyces israelii* is a common organism of the oropharynx and may cause infection when it is introduced to the deeper tissue sites following soft tissue trauma. Around 50% of actinomycosis cases occur in the oral and maxillofacial areas and may cause failure of dental implants. Other parts of the body, such as the respiratory system and the abdomen, may also be affected. Actinomycosis has mostly been misdiagnosed as malignant rather than an infectious disease due to its variable clinical presentation. Actinomyces lesions contain clusters of neutrophils, plasma, and multinucleated giant cells with macrophages and foamy cytoplasm. These clusters (1-2 mm diameter) discharge a yellowish exudate of aggregates of organisms and calcium phosphate throughout sinus tracts known as "sulfur granules". Because of its similarity to other infectious and inflammatory diseases, the diagnosis of actinomycosis is challenging, and tissue biopsy and histopathology remain the gold standard for its diagnosis. Management is achieved with long-term pharmacological and surgical intervention, with a promising success rate if commenced at an early stage. In this review, we aimed to provide a deep insight into the different aspects of *Actinomyces israelii* infection of oral and non-oral involvement.

Keywords: Cervicofacial; Implant failure; Imaging; Sulfur granules

INTRODUCTION

As a disease, actinomycosis was reported around 150 years ago; however, it is still not well known to the majority of clinicians. It has mostly been misdiagnosed as malignant rather than an infectious disease due to its variable clinical presentation [1]. The bacterially caused infection is rare, with a slow progression rate. Inflammation generated by *A. israelii* is characterized by being suppurative and granulomatous with abscess formation, fibrosis, and sinus drainage that form characteristic but not pathognomonic "sulfur granules." In 1877, the pathologist Otto Bollinger discovered *Actinomycetes bovis* in cattle, and very soon after that, James Israel discovered *Actinomycetes israelii* in humans. A single actinomycete bacterium is rod-shaped, while its colonies form fungus-like, branched networks of hyphae. This phenotyping has led to the false assumption that *A. israelii* was a fungus, and hence the term actinomycetes "ray fungus) came from (-actis in Greek means ray or bean-like and mykes means fungus) [2,3].

Figure (1): *Actinomycetes*: branched networks of hyphae [2]

There are about 38 different species of actinomycetes; the most common species are *Actinomycetes israelii, Actinomycetes bovis, Actinomycetes viscous, Actinomycetes meyeri,* and others. *Actinomycetes israelii* is the most important and prevalent species that is isolated in human infection and almost found in almost all clinical presentations of actinomycosis [4].

Epidemiology, etiology, and risk factors

Normally, *A. israelii* is part of the endogenous microbiota of the oral cavity, gastrointestinal tract, and female genital tract. The external environmental reservoir of the pathogen has not been discovered, and no human-to-human spreading of disease has been documented. An exception is when a person is subject to human bites or trauma caused by a fist fight resulting in oral colonization. All age groups can be affected; however, it is rarely seen in children or in elderly patients (> 60 years), while

individuals aged between 20-60 are mostly affected by the disease [2]. Males are more likely to be infected (male-to-female ratio is 3:1), which might be due to their inferior oral hygiene and the higher incidence of oral trauma. Few cases of actinomycosis are diagnosed annually in developed countries, as sporadic cases in Iraq and around the world, which might be due to the confusion with other diseases. Factors such as improved oral hygiene, availability and activity of antibiotics, and the prompt commencement of treatment once the infection is assumed [5].

The fact that human *Actinomycetes* are often isolated alongside other normal commensal bacteria such as *Streptococci* (including *β-haemolytic Streptococci* and *S. pneumoniae*), *Staphylococci* (including *S. aureus*), *Bacteroides,* or *Enterobacteriaceae,* and many other bacterial species is reported to be advantageous to the former species. To explain more, the concomitant presence of commensal bacteria has been shown to synergistically strengthen the invasiveness of *A. israelii*, hampering host defense and reducing local oxygen tension, accounting for the early symptom of the disease and also for future treatment failure [6].

Via a point of entry, Actinomyces can establish an infection of the deep tissue. Such entry points include mucosal lesions, periodontal pockets, or endodontic pathways with dental caries, invasive dento-maxillofacial measures, trauma, or radio necrosis being the main causes [7]. Individuals with impaired or attenuated immunity, such as diabetic and cancer patients, alcohol abusers, and steroid recipients, are at higher risk of developing the chronic infection. The route of infection in pulmonary actinomycosis is mainly due to aspiration of oropharyngeal secretions containing *Actinomycetes,* and this is mostly obvious in alcoholics and patients suffering from severe gastroesophageal reflux disease (GERD). The use of an intrauterine contraceptive device has been found to increase the incidence of genitourinary actinomycosis, and it is closely related to the duration of the use of the device [6, 8].

Pathology and pathogenesis

In order to establish the disease and owing to its low pathogenicity, *Actinomycetes* species require a disruption of the mucosal barrier [9]. Following the invasion of the local mucosa, the organism blowouts to the nearby tissues slowly but progressively. This results in the formation of a chronic pyogenic lesion with sinus drainage and fibrosis. The infection may spread hematologically to other vital organs at any stage of the infection course, but lymphatic distribution is uncommon [10]. Hardening of the fibrotic walls results in a wooden mass that is mostly misdiagnosed as a neoplastic mass [11]. The bacteria grow as clusters of twisted filaments embedded between neutrophiles in a rosette shape. Actinomyces lesions also contain plasma and

multinucleated giant cells with macrophages and foamy cytoplasm. These clusters (1-2 mm diameter) discharge a yellowish exudate of aggregates of organisms and calcium phosphate throughout sinus tracts known as "sulfur granules" (originally known as "Drusen") [12].

Clusters containing sulfur granules are also formed by other bacteria like *Nocardia, Streptomyces* and *Staphylococcus* in botryomycosis therefore, a differential staining is required to distinguish these from actinomycosis caused by *A. israelii*. For instance, Gram staining can show the filamentous branches of A. *israelii* at the periphery of the granules which is highly suggestive of actinomycosis. Also, immunofluorescent staining offers high specificity for the diagnosis of Actinomycetes [13].

Clinical manifestations

Actinomycosis can invade any anatomical parts of the body, leading to different acute/chronic findings. Generally speaking, actinomycosis can be classified according to the anatomical part into [14]:

Cervicofacial actinomycosis

Among the reported cases, *A. israelii* is the most common implicated pathogen, accounting for up to 70% of the cases [15,16]. The mandibular area is the most targeted part of the cervicofacial area (submandibular region, ramus, and angle) in half of the cases; however, other structures can also be affected [16,17]. such as the submental, retromandibular spaces, temporomandibular joint, and cheek. For this reason, any mass or relapsing infection in the areas of the head or neck should be thought of as actinomycosis [18]. Clinically, patients mostly complain of acute soft swelling, which is usually painful. In this case, the presence and composition of the synergistic normal flora play a role (for instance, *Staphylococcus aureus* or *beta-hemolytic Streptococci*). Also, actinomycosis patients may present with a chronic painless mass in the submandibular or paramandibular region (characteristic of "Lumpy jaw syndrome"). In this case, multiple abscesses and draining sinus are characteristic, and the discharge of yellow sulfur granules can occur in up to 25% of cases [15]. Other accompanied signs and symptoms may include fever, lethargy, periapical infection, and leukocytosis. Extended infection may involve other parts such as the tongue and salivary glands, the sinus and ears, the pharynx, masseter muscle, thyroid, larynx, trachea, or thorax [19]. Bacteria can disseminate from the adjacent soft tissue to the mandibular bone or other bones, causing osteomyelitis. If the cervical spine or cranial bone is infected, CNS invasion is possible, and bacteriemia may result if treatment is delayed or restricted. Cases of dental implant failure have also been documented. For instance, a 43-year-old

female presented 1 month after a dental implant operation with hyperplastic peri-implant soft tissue accompanied by suppuration, bleeding, and implant mobility. Following implant removal and histology examination, an acute ulcerative inflammatory reaction and Actinomyces colonies were revealed [20].

Pulmonothoracic actinomycosis

It accounts for about 15-20 of all cases reported. The mode of transmission is usually through aspiration of oropharyngeal secretions containing the pathogen. Rarely, infection may result due to esophageal perforation, introduction from an abdominal site, or bloodborne from a distal lesion [21]. Common patient presentation is with nonspecific symptoms such as generalized weakness and malaise, chest pain accompanied with cough which is productive cough, weight loss, and fever. Blood test reveals anemia, mild leukocytosis, and elevated ESR (erythrocyte sedimentation rate). Pulmonary lesion exists either as a mass lesion or as pneumonitis from which dissemination of the infection may occur to involve the pleura and chest wall. In case of deteriorated cases with no specific diagnosis or no effective treatment, complications ensue, including the formation of fistula and drainage of sulfur granules with pleural thickening, pleural effusion, or emphysema [22].

Abdominal pelvic actinomycosis

Gastrointestinal actinomycosis comprises around 10-20% of the cases. Infection is established weeks, months, or even years following the introduction of actinomycetes into the deeper tissue. Breakdown of the gastrointestinal mucosa by surgery for cases of appendicitis, perforated colonic diverticulitis, or other emergency surgery of the lower intestinal tract is the main route of the pathogen invasion. Lesions start in the ileocecal region and extend to other organs such as the liver, spine, and the abdominal wall. Patients are mostly presented with nonspecific signs and symptoms such as fever, GI disturbance, loss of appetite, with weight loss [12].

Pelvic actinomycosis has also been recognized and recommended to be considered in women with a history of intrauterine device (IUD) with abdominal pain or pelvic mass [23, 24]. Factors that predispose to getting infected with actinomycetes include: use of IUDs for over 8 years, use of vaginal pessaries, uterine prolapse, and septic abortion. Patients are mostly presented with abdominal pain, weight loss, vaginal discharge, mild fever with anemia, and leukocytosis. Unilateral or bilateral tubo-ovarian abscesses at surgery are found in around 90% of the patients. Other organs' involvement is common by adhesion or compression, such as the liver, ureters, urinary bladder, and the bowel [11].

Actinomycosis of the central nervous system

A recently diagnosed form of actinomycosis is that of the CNS, which may be bloodborne from thoracic or internal infection or may arise from direct spread of a cervicofacial lesion. Patients are often presented with brain lesions with symptoms of headache, increased intracranial pressure, focal seizures, hemiparesis, aphasia, ataxia, abnormal reflexes, and other symptoms depending on the affected site of the brain [25, 26].

Actinomycosis of the skin and bone

Wounds that are contaminated with saliva or dental plaque materials following human bites or as a result of fist-fight trauma can develop cutaneous actinomycosis. Presentation is fairly similar to the cervicofacial form. Direct extension from adjacent soft tissue foci can cause osseous involvement, resulting in periostitis and bone destruction. Most frequently involved bones include the mandible, ribs, and spine [27,28].

Diagnosis of actinomycosis infections

Because lesions of actinomycosis are commonly confused with different pyogenic masses or neoplasia, this has led to difficulty in diagnosing cases of actinomycosis, as only 10% of cases are accurately diagnosed [11, 29].

Laboratory Studies

1. Complete blood count: nonspecific findings such as mild leukocytosis and normochromic anemia may present [30].

2. Biochemical tests: show normal results, except alkaline phosphate levels are elevated in hepatic actinomycosis. Erythrocyte sedimentation rate (ESR) and C-reactive protein are also elevated, representing the nonspecific inflammatory nature of the disease. Serological methods of detection lack specificity and sensitivity and therefore cannot be relied on for the diagnosis of actinomycosis [14, 22, 31].

3. Cultivation and direct identification: The isolation of infectious microorganisms from clinical specimens is important for definitive diagnosis in most cases. Specimens that cannot be used for diagnosis include swabs, sputum, and urine samples, while draining sinuses, deep needle aspirates, or biopsy specimens are considered ideal. The anaerobic transport system is required to get the sample to the microbiology laboratory for subsequent processing. To start with, Gram-staining, beaded, branched, non-spore-forming, and Gram-positive filamentous rods can be seen because of the polymicrobial nature of the disease. This aids in the diagnosis of actinomycosis and the choice of adjunctive antibiotics [32, 33]. The presence of sulfur granules can give a definitive diagnosis of actinomycosis. This can be performed by crushing the granules between two glass slides, then staining with 1% blue solution. The slide is then

examined microscopically, looking for characteristic features of Actinomycetes [34-36]. Sulfur granules stained with hematoxylin–eosin in the discharge from the sinus tract show the diagnostic "sun ray appearance" [37]. Identification of Actinomycetes by culture methods requires the immediate culturing under anaerobic conditions and a prolonged incubation period of 48 h or even longer. Almost two to three weeks are required for isolation and definitive identification of *Actinomycetes* [38]. However, the culture method of identifying or verifying the infection is weakly reliable. Studies report that positive culture for actinomycosis can be obtained in only 35% of all cases following standard culture techniques [39]. Other enriched media which can be used for *Actinomycetes* isolation are brain, heart infusion broth. For selective isolation of the organism, semi-selective media like mupirocin-metronidazole blood agar can be used, which increases the isolation rate to 86% by inhibiting over overgrowth of concomitant faster-growing anaerobes [40].

Advanced techniques such as immunofluorescence microscopy, nucleic acid probes, and DNA detection by polymerase chain reaction are being applied for sensitive, specific, and rapid results [11].

Imaging studies

Chest X-ray examination, CT scan of head, chest, abdomen, and pelvic, and magnetic resonance imaging (MRI) do not provide a specific diagnosis, especially at the early stage of the disease, as other inflammatory diseases such as ulcerative Colitis and Chron's disease and neoplasia may have similar imaging findings. Radiological imaging allows a more accurate definition of the dimensions and extension of the infection at the later courses of actinomycosis [41, 42].

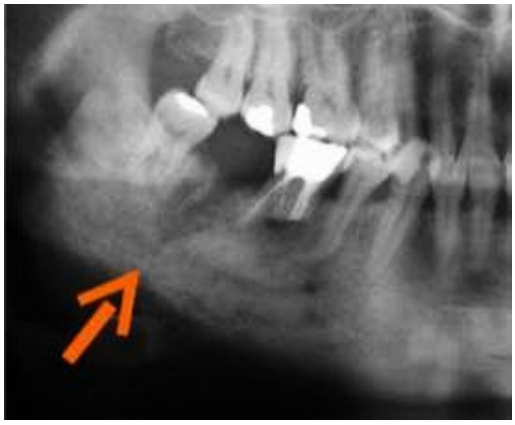

Figure (2): X-ray image showing lumpy jaw syndrome characteristic of actinomycosis[11].

FNAC and open biopsy

Clinical materials for the diagnosis of actinomycosis can be efficiently obtained by fine needle aspiration collection (FNAC). Tissue biopsy and histopathology remain the gold standard for actinomycosis diagnosis. Surgery may be required for diagnostic purposes (e.g., thoracotomy with open lung biopsy) [43, 44].

Management

Pharmacological treatment

Actinomyces infection is usually discovered late with severe, extensive infection and is often invasive, associated with significant purulence or fistulous tracts, which is why the mainstay of the treatment is prolonged courses of antibiotics. For instance, clinical experience has shown that a prolonged course (6-12 months) of high doses of antibiotics as penicillin, offers a good cure rate of actinomycosis. However, individualization of the adopted treatment regimen is the modern approach since a number of factors, such as site of infection, severity and patient's response, and compliance, affect the exact antibiotic regimen [11]. The most preferred protocol for actinomycosis is high-dose (18-24 million units a day) of intravenous penicillin G for 2-6 weeks, followed by penicillin V orally at a dose of 2-4 g per day for 6-12 months [11,45, 46].

The risk of *Actinomycetes* developing resistance to penicillin is minimal. Resistance to penicillin usually indicates the presence of resistant companion bacteria, which require modification of the protocol by adding a drug that is effective against these pathogens. If the patient is hypersensitive to penicillin, tetracycline, vancomycin, and erythromycin can be used as alternative choices, while cephalosporins such as ceftriaxone, amoxicillin/clavulanic acid, and imipenem are considered drugs of choice in co-infection not responding to penicillin.

Studies documented that the growth of *in vitro* growing oral *A. israelii* was shown to be decreased by a variety of nonconventional interventions such as licorice-based formulations, curcumin, cinnamon oil, resazurin, riboflavin, protoporphyrin IX, and light irradiation [47, 48].

Non-pharmacological treatment

Surgical interferences such as tooth extraction, abscess drainage, and bone necrosis removal are performed for subacute or chronic voluminous lesions [24, 45, 49-53]. In general, surgical intervention in the treatment of actinomycosis is indicated in the following conditions:

1. Extensive necrotic tissue and bone involvement.

2. Presence of sinus tracts or fistulas.
3. If malignancy can't be excluded.
4. If empyema or large abscesses are difficult to drain by percutaneous aspiration.
5. Failure of response to medical treatment with antibiotics alone.
6. Surgery could be useful in controlling symptoms such as hemoptysis in thoracic actinomycosis.
7. Relieving obstruction, such as an actinomycotic lesion, compresses the ureter.
8. Removal of the IUCD is crucial in a patient with IUCD-associated actinomycosis.

Prevention

Regular dental care and good oral hygiene are important. Increasing the risk of actinomycetes infection associated with IUCD infection should be kept in mind by both the patient and physicians [11,54].

CONCLUSIONS

It is challenging to deal with actinomycosis. Therefore, to minimize the risk of delayed diagnosis and subsequent consequences, actinomycosis should be well-thought-out in the differential diagnosis of any inflammatory lesion of subacute or chronic nature.

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Authors' Contribution

Ahmed AM., Hashim ZA., Qasim ZS., and Aldabbagh KA contributed to conceptualization, validation, and writing the original draft. Ahmed AM., Hashim ZA., Qasim ZS., and Aldabbagh KA are responsible for formal analysis, methodology, and project administration. Ahmed AM., Hashim ZA., Qasim ZS., Aldabbagh K. Reviewed and edited the manuscript. Ahmed AM., Hashim ZA., Qasim ZS., and Aldabbagh KA contributed to the investigation, software development, validation, and visualization. Ahmed AM., Hashim ZA., Qasim ZS., and Aldabbagh KA are involved in data curation, resources, and review & editing. All authors have read and approved the final manuscript.

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The authors declare that there are no conflicts of interest regarding the publication of this manuscript.

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Declaration of Generative AI and AI-assisted technologies

No artificial intelligence tools were used. The authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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المظاهر العنقية الوجهية والأهمية السريرية لـ **Actinomyces *Israelii***

أوس محمود أحمد[1]، زهراء عامر هاشم[2]، زهراء صديق قاسم[3], كرم الدباغ[4]

1 قسم جراحة الفم والوجه والفكين، كلية طب الأسنان، جامعة الموصل، الموصل، العراق
2، 3، 4 كلية الصيدلة، جامعة الموصل، الموصل، العراق

الملخص

فطر **Actinomyces *Israelii*** هو كائن حي شائع يصيب البلعوم الفموي، وقد يسبب عدوى عند دخوله إلى الأنسجة العميقة بعد رضوض الأنسجة الرخوة. تحدث حوالي 50% من حالات داء الشعيات في مناطق الفم والوجه والفكين، وقد تُسبب فشل زراعة الأسنان. كما قد تُصاب أجزاء أخرى من الجسم، مثل الجهاز التنفسي والبطن، بهذا المرض. غالبًا ما يُشخص داء الشعيات خطأً على أنه خبيث وليس مرضًا معديًا نظرًا لاختلاف أعراضه السريرية. تحتوي آفات داء الشعيات على مجموعات من العدلات والبلازما وخلايا عملاقة متعددة النوى مع خلايا بلعمية وسيتوبلازم رغوي. تُفرز هذه المجموعات (بقطر 1-2 مم) إفرازات صفراء من تجمعات الكائنات الحية وفوسفات الكالسيوم في جميع أنحاء الجيوب الأنفية، تُعرف باسم "حبيبات الكبريت". نظرًا لتشابهه مع الأمراض المعدية والالتهابية الأخرى، يُمثل تشخيص داء الشعيات تحديًا، وتُعتبر خزعة الأنسجة والفحص النسيجي المعيار الأمثل لتشخيصه. يتم تحقيق العلاج من خلال تدخل دوائي وجراحي طويل الأمد، مع نسبة نجاح واعدة إذا بدأ في مرحلة مبكرة. في هذه المراجعة، هدفنا إلى تقديم نظرة متعمقة على الجوانب المختلفة لعدوى **Actinomyces *Israelii***، سواءً من خلال الفم أو غير الفم.

الكلمات المفتاحية: منطقة العنق والوجه؛ فشل الزرعات؛ التصوير؛ حبيبات الكبريت