New Microbiologica, 42, 1, 55-60, 2019, ISN 1121-7138

CASE REPORT

A case report of esophageal actinomycosis in an immunocompetent patient and review of the literature

Valeria Palmitessa[1], Renato Cuppone[2], Rosa Monno[3], Luciana Fumarola[3], Antonio Lippolis[1]

[1]*Laboratory of Microbiology and Virology, National Institute of Gastroenterology "S. De Bellis", Research Hospital, Castellana Grotte, Bari, Italy;*
[2]*Department of Gastroenterology, National Institute of Gastroenterology "S. De Bellis", Research Hospital, Castellana Grotte, Bari, Italy;*
[3] *Department of Basic Medical Science, Neuroscience and Sense Organs, University of Bari Aldo Moro, Bari, Italy*

SUMMARY

Actinomycosis is a rare, chronic and slowly progressive granulomatous disease caused by *Actinomyces spp.*, a Gram-positive anaerobic bacterium that rarely affects the esophagus. Although this infection is uncommon, it has been reported in both immunocompromised and immunocompetent individuals.
The infection is often misdiagnosed because it can mimic other pathological conditions (like neoplasms and candidiasis), and *Actinomyces* is difficult to isolate because it requires specific growth conditions. However, actinomycosis has a favorable course if the microbiological diagnosis is timely.
We report a case of esophageal actinomycosis in an immunocompetent 23-year-old man. The patient was admitted with symptoms of gastro-esophageal reflux disease (GERD), that had subsequently worsened. Histological and microbiological investigations revealed the presence of *Actinomyces spp*. A review of the literature regarding the clinical features, diagnosis, and management of this infection is also discussed.

Received October 08, 2018 *Accepted* January 5, 2019

INTRODUCTION

Actinomyces spp. comprises 47 species, 20 of which are relevant to human disease, including *A. israelii*, *A. viscosus*, *A. meyeri*, *A. naeslundii*, *A. odontolyticus*, *A. neuii*, *A. turicensis* and *A. radingae*. They are non-motile, non-spore forming, pleomorphic Gram-positive rods, with a morphology ranging from branching to non-branching rods, and some have very short or even coccoid cells (Könönen, Wade, 2015). They are normal inhabitants of the human mouth, vaginal mucosa, and intestinal tract. Some *Actinomyces spp*. are responsible for human diseases (Boyanova *et al.*, 2015).
Actinomycosis is classified in distinct clinical forms according to the anatomical site infected. The most common infections caused by *Actinomyces* are cervicofacial suppurative infections (50% of all actinomycosis cases); thoracic actinomycosis (15-20% of cases) and abdominopelvic actinomycosis (about 20% of cases). Rare sites of actinomycosis include the central nervous system, bones, muscle tissue, and prosthetic joints (Wong *et al.,* 2011). However, the pathogenesis of this infection is via an interruption of tissue integrity due to a lesion, invasive surgical practices or inflammation and tissue corrosion, promoting the penetration of these microorganisms (Wong *et al.,* 2011). Although some authors have reported that actinomycosis is more common in immunocompromised patients, an increase in the number of cases described in immunocompetent individuals has recently been observed (Wong *et al.,* 2011; Valour *et al.,* 2014). This can be explained by the fact that actinomycoses are often underestimated because its clinical symptoms mimic those of other diseases and these pathogens are not always isolated (Valour *et al.,* 2014). The components of the immune response are crucial in the control of actinomycosis. Immunosuppression due to steroids, chemotherapy, HIV, and lung and renal transplants, are conditions that can promote the infection. Other risk factors involved in the acquisition and progression of actinomycosis include age (20-60 years), male gender, poor oral hygiene, diabetes, alcoholism, local tissue damage caused by trauma, recent surgery, irradiation (Brook, 2008). Recently, *Actinotignum schaalii* (formerly *Actinobaculum schaalii*) has been considered the agent of actinomycosis principally involving the urinary tract (Lotte *et al.,* 2016).
We report the uncommon case of symptomatic esophageal actinomycosis caused by *Actinomyces odontolitycus* in an immunocompetent 23-year-old man.

Key words:
Actinomyces, *Actinomyces odontolyticus*, Esophageal actinomycosis.

Corresponding author:
Antonio Lippolis
E-mail: antonio.lippolis@irccsdebellis.it

CASE REPORT

A 23-year-old man with a history of gastro-esophageal reflux disease (GERD), characterized by marked laryngeal signs such as a dry cough and arytenoid edema, who showed worsening of the disease despite treatment with antacids and proton pump inhibitors (Na alginate 300 mg plus alginic acid 365 mg, every 12 hours, pantoprazole, 40 mg each morning for the first 4 weeks and then 20 mg once daily for an additional 4 weeks) was referred to our hospital for dyspeptic disorders, epigastric pain,

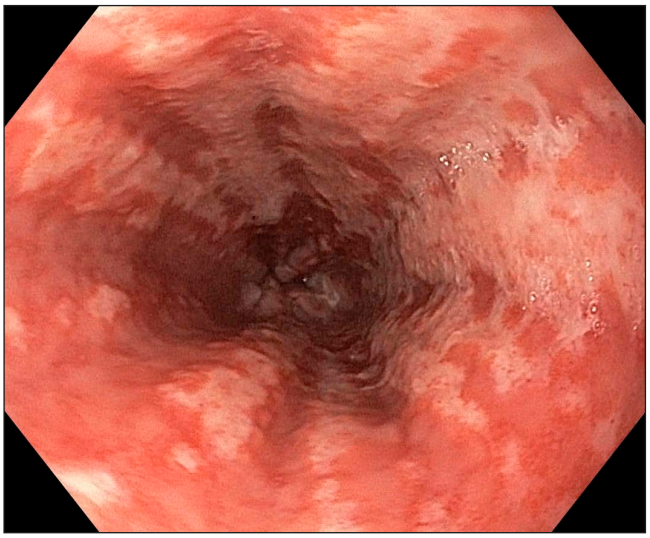

Figure 1 - *EGD image showing extensive longitudinal areas of erosion with hyperemic margins partially covered by membranes.*

pharyngodynia and dysphagia to solid and liquid foods. Intermittent fever up to 38.5°C was also present, as well as recurrent episodes of tonsillitis and otitis treated with amoxicillin-clavulanic acid. Laboratory investigations showed a normal complete blood count with normal leukocyte formula, normal biochemistry panel and negative serology for HIV, HBV and HCV.

Esophago-gastro-duodenoscopy (EGD) was performed and revealed extensive longitudinal areas of erosion with hyperemic margins that became confluent in the distal esophagus (*Figure 1*). During the EGD, several biopsy samples were collected for histological and microbiological examination. Taking into account that esophageal lesions presenting as discrete plaque-like lesions may also be due to *Candida* infection of the esophageal mucosa, the patient was empirically treated with amoxicillin-clavulanic acid 875 mg/125 mg twice daily for one week and fluconazole 100 mg twice daily for two weeks, associated with proton pump inhibitors (esomeprazole 40 mg twice daily for six weeks) and discharged from hospital.

Histological examination of one of the esophageal biopsy specimens revealed the presence of a single large clump with numerous branching filamentous organisms resembling sulfur granules, consistent with the diagnosis. Also, mucosal ulceration and a dense inflammatory infiltrate were evident (*Figure 2*).

PAS staining did not show hyphae or fungal spores compatible with *Candida spp*. For the microbiological investigation, the sample was grossly homogenized and Gram staining was performed. The presence of Gram-positive filamentous bacilli was observed. The sample was cultured on Columbia agar supplemented with 5% sheep's blood, Chocolate agar supplemented with VITOX incubated at 35°C in 10% CO_2 and on MacConkey agar, Mannitol Salt agar and Sabouraud agar supplemented with chloramphenicol, incubated in aerobic conditions. Biopsy samples were also cultured on Schaedler agar (supplemented with sheep's blood) and Schaedler plus KV agar (supplemented with lysed horse's blood hemin, Kanamyicin 0.1g/L, Vancomycin 0.0075 g/L and vitamin K) and incubated at 35°C in an anaerobic atmosphere (all the media were purchased from Thermo Fisher Scientific, Oxoid Microbiology Products, Hampshire, UK). After five days of incubation, white non-hemolytic colonies were observed only on Columbia and Schaedler agar. Gram staining of colonies showed the presence of Gram-positive microorganisms whose morphology resembled that of the previously described filamentous bacilli. Biochemical identification of these colonies was performed with the VITEK 2 automated identification

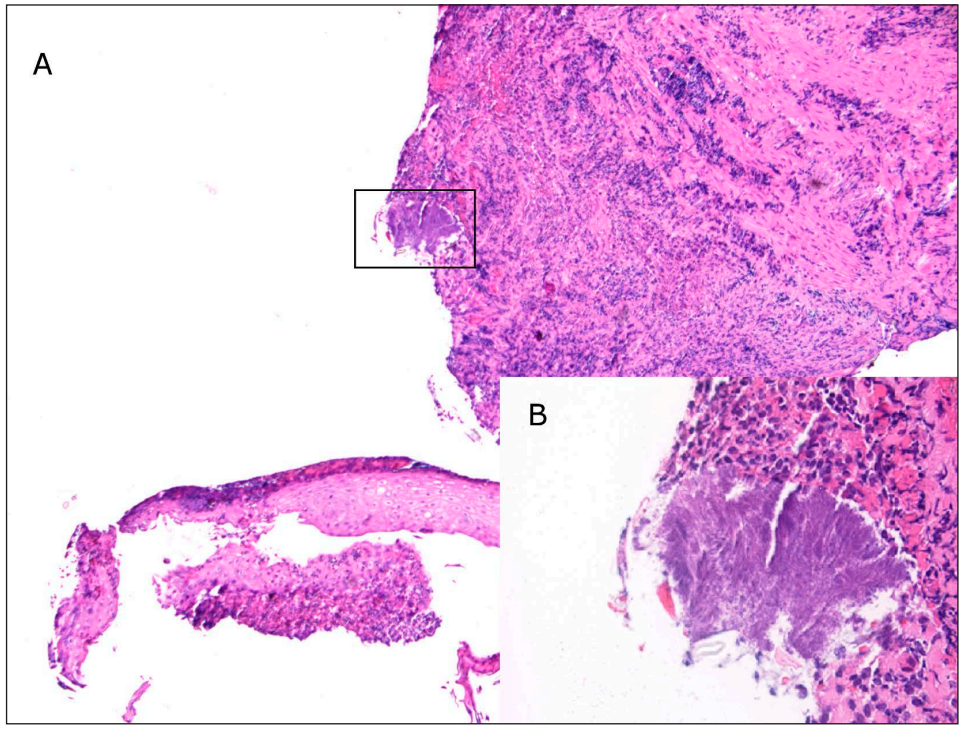

Figure 2 - *Esophageal biopsy. (A) Histological examination of a hematoxylin and eosin (H&E) stained biopsy specimen showing diffusely inflamed esophageal mucosa. Magnification: ×40. (B) In the right bottom box, sulfur granules suggestive of Actinomyces colonies are evident.*

system and an ANC card (bioMérieux, Inc., Durham, NC). The strain was identified as *Actinomyces odontolyticus* (Bionumber 2160004000101). Therefore, the diagnosis of esophageal actinomycosis made on the basis of histological data was confirmed by microbiological data. The patient was treated with amoxicillin-clavulanic acid (500 mg every eight hours for two weeks), which resulted in complete regression of the clinical symptoms and complete healing of the esophageal erosions.

DISCUSSION

Actinomycosis is a rare, often underestimated infection caused by microaerophilic anaerobic Gram-positive rods belonging to the *Actinomyces* genus. As human commensals, these organisms have a low degree of pathogenicity and generally cause infection only if the continuity of the mucosal barrier is interrupted due to trauma or invasive surgical practices that could promote penetration of the organism, leading to infections at virtually any site in the body. However, actinomycosis occurs most frequently at the oral, cervical, or facial sites, usually manifesting as a soft swelling, abscess, or massive lesion that can often be mistaken for a neoplasm. The incidence of all forms of actinomycosis is thought to have declined in recent years, especially in developed countries, as a result of better oral hygiene and susceptibility to a broad range of antibiotics (Brook, 2008).

Our patient suffered from GERD treated with antacids, which likely caused the erosion of the esophageal mucosa, allowing the *Actinomyces* commensals of the oral cavity to colonize this anatomical site. Although *Actinomyces* is a rare occurrence of esophageal disease it should be considered in the differential diagnosis. In our review of the clinical literature from 1953 to now, we found only 27 cases of esophageal actinomycosis (*Table 1*).

As seen, all age groups (range from 19 to 83) can be affected. Like actinomycoses that affect other anatomic sites, esophageal infections mainly affect males: the esophageal actinomycosis cases included 20 males and 6 females (in one case the gender was not reported). By analyzing the rare cases of esophageal actinomycosis reported in the literature, it is easy to see that these infections are associated with comorbidity conditions that involve impairment of the immune system. However, cases of actinomycosis are reported not only in immunocompromised (59.3%) but also in immunocompetent (40.7%) subjects. Among 16 immunodeficient patients, 7 were HIV positive, 7 had neoplastic diseases and had therefore undergone chemotherapy, and 2 were affected by an autoimmune disease that required the administration of immunosuppressants. As far as the symptoms of esophageal actinomycosis are concerned, in most cases patients reported severe dysphagia and/or odynophagia, which in some patients caused anorexia, resulting in weight loss. In only a few cases, the symptoms were further aggravated by vomiting. In our case, too, the patient reported dysphagia to solid and liquids, dyspepsia, heartburn, and fever. In rare cases, symptoms including epigastric pain were reported.

In all cases, the EGD highlighted erosion areas with varying degrees of hyperemic margins (in some cases areas of deep ulceration were also highlighted) covered by necrotic portions or eschar. In our case, EGD images showed extensive longitudinal areas of erosion with hyperemic margins partially covered by membranes. At a first EGD observation, actinomycotic infections of the esophagus mimic candidiasis. In many cases, antifungal therapy is administered as a first emergency therapeutic approach to ward off worsening of the putative *Candida spp*. involvement. In many cases, an initial improvement in clinical conditions has also been observed following this treatment. The diagnosis can be difficult, and involves making a distinction among neoplastic conditions, malignant hemopathy, and other infections. The diagnosis of actinomycosis is based on different approaches that vary according to the anatomical site affected by this microorganism. However, the gold standard for the diagnosis of actinomycosis is histological examination and bacterial culture of biopsy specimens. A definitive diagnosis of actinomycosis in the esophagus can be difficult because the radiologic findings are non-specific and computed tomography may demonstrate a thickened esophageal wall in early stage infections.

From the study of the literature included in this review, in all of the 27 cases examined, including the one we report, histological examination of the biopsy specimens had been performed, and always highlighted areas of inflammation of the mucous membranes characterized by the presence of aggregates of Gram-positive filamentous bacteria. It should be noted that, in many cases, histological examination revealed the presence of characteristic sulfur granules. However, the presence of granules does not always confirm *Actinomyces* infection. In fact, *Actinomyces* is sometimes observed as a contaminant in esophageal biopsies, being transported from the oral mucosa. In these cases, it is superficial and not associated with important inflammatory reactions. Instead, in our biopsies the actinomycotic granule was deeply located and partially surrounded by a dense granuloma-like inflammatory infiltrate. Likewise, in our case, histological analysis revealed the presence of a few granules. Our patient had previously been treated with amoxicillin-clavulanic acid, which may have caused a reduction in bacterial proliferation.

However, isolation and biochemical identification of the *Actinomyces* strains that caused esophagitis was conducted only in a few of the reported cases. This is because these microorganisms are difficult to cultivate and require particular growth conditions. In our clinical case, biochemical isolation and identification was made possible by good teamwork between the gastroenterologist and laboratory staff. *Actinomycetes* are β-lactam-susceptible and only occasionally resistant. In those cases, with protracted colonization resulting in necrotic colonized tissues, treatment includes surgery and/or long-term parenteral then oral antibiotics, but some 1-4 week regimens or oral therapy alone were curative (Sudhakar, Ross, 2004). Our patient was successfully treated with amoxicillin-clavulanic acid, even if this therapy is not the most frequent therapeutic regimen. Recently, two-thirds of patients with actinomycosis were treated with amoxicillin (Bonnefond, *et al.,* 2016).

In conclusion, esophageal actinomycosis must be also suspected in immunocompetent subjects and in subjects, like our patient, previously treated with proton pump inhibitors. This treatment may promote actinomycosis colonization of the esophageal mucosa, the reservoir of this bacterium being the oral cavity; the condition is also associated with poor dental hygiene.

Table 1 - *Review of the literature. Summary of published cases of esophageal actinomycosis.*
M, male, F, female; IC, immune-competent; ID, immune-deficient; IS, immune-suppressed; EGD, Esophagus-gastro-duodenoscopy; HE, histopathological examination; IBI, isolation and biochemical identification; RF, radiographic findings; A, non-reported data.

Author / date	*Age / gender*	*Co-morbid conditions*	*Immune conditions*	*Presenting symptom and clinical features*	*Diagnosis*	*RF and / or EGD findings*	*HE*	*Identification*
Fukuda S *et al.* 2018	60/M	Hepatocellular carcinoma	ID	Odynophagia	EGD HE	Esophageal ulcer	Thickened of the esophageal wall, presence of sulfur granules	A
Rodriguez-Lago *et al.*, 2017	30/M	Allergic asthma, hyperuricemia	IC	Dysphagia to solids	EGD HE	Thin longitudinal striations, as well as faint rings in the distal third.	Eosinophilic esophagitis in addition to surface colonies of *Actinomyces*	A
Pillappa *et al.*, 2016	55/F	Diabetes mellitus, hypertension, hyperlipidemia, cerebral aneurysm, and ventral hernia repairs	IC	Dysphagia to both solids and liquids and weight loss	EGD HE	Submucosal mass with partial luminal obstruction involving the lower third of the esophagus, with a small mucosal ulceration with purulent material	Fibroinflammatory lesion with areas of abscess with sulfur granules and Gram positive filamentous microorganisms	A
Nagaraju *et al.*, 2014	28/F	Systemic lupus erythematosus hepatitis B infection end-stage renal disease	ID	Epigastric pain, odynophagia	EGD HE	Extensive necrotic areas with membranes in the esophagus	Strips of necrotic slough with actinomycotic colonies and bacterial clumps	A
Korkmaz *et al.*, 2013	56/M	A	IC	Odynophagia, dysphagia, heartburn	RF EGD HE	White plaques predominantly in the mid esophagus, multiple shallow ulcers and an irregular, malignant-appearing ulcer in the distal esophagus	Discrete sulfur granules	A
Hyun *et al.*, 2013	47/M	Diabetes mellitus	IC	Dysphagia	EGD HE IBI	esophageal ulcers covered with thick exudate	Deep purple fragmentated sulfur granules within ulcers debritus with neutrophilic exudate. Sulfur granules showed Gram positive coccoid form and rare remnants of filaments	A
Chandrasekhara *et al.*, 2012	68/F	Mediastinal radiation for recurrent non-small cell lung cancer	IC	Dysphagia, odynophagia	RF EGD HE	Irregular exudative stricture with eschar formation	Necrotic material with filamentous Gram positive bacteria consistent with *Actinomyces*	A
Murchan *et al.*, 2010	53/M	Hiv-positive, hepatitis C, esophageal candidiasis	ID	Epigastric pain, odynophagia	EGD HE	Areas of deep ulceration in the distal esophagus	Numerous filamentous Gram positive organisms	A
Welling *et al.*, 2009	27/M	End stage renal disease secondary to lupus	IS	Odynophagia, epigastric pain swallowing	RF EGD HE	Ulcerated lesion in the esophagus	Ulcerated squamous mucosa with sulfur granules and branching filamentous Gram-positive bacteria	A
Ho *et al.*, 2006	76/M	Squamous cell carcinoma of the esophagus	ID	A	HE	A	A	A
Chou *et al.*, 2006	41/M	History of heavy alcohol use, esophageal candidiasis	IC	Odynophagia, dysphagia	EGD HE	Multiple well-defined, round ulcers scattered in the distal esophagus	Sulfur granules and filamentous Gram-positive bacteria	A

Cash *et al.*, 2005	56/M	Asthma, bronchospasm	IC	Eight-week history of dyspepsia, dysphagia, and epigastric tenderness.	EGD HE	Ulceration in the lower third of the esophagus, more extensive confluent ulceration in the middle third	Acute on chronic inflamed granulation tissue	A
Fernandez Moreno *et al.*, 2005	80/F	A	IC	A	EGD HE	Ulceration in the middle third of the esophagus	Inflamed granulation tissue with *Actinomyces* colonies	A
Kosseifi *et al.*, 2005	61/M	Non-small cell distal tracheal carcinoma, endotracheal excision, chronic obstructive pulmonary disease	ID	Persistent dysphagia	EGD HE	Stricture in the middle esophagus and an ulcer	Acute erosive esophagitis with ulcer debris and reactive squamous mucosa	A
Kahn *et al.*, 2005	56/F	A	IC	Dysphagia, odynophagia	RF EGD	Stricture in the middle esophagus	A	A
Abdalla *et al.*, 2004	61/M	Non-small cell distal tracheal carcinoma	ID	Severe dysphagia and odynophagia	EGD HE IBI	Deep ulcer	Squamous mucosa with an ulcer and numerous sulfur granules containing abundant finely elongated hyphal forms	*Actinomyces* spp.
Sudhakar Ross, 2004	83/F	Alzheimer disease	IC	Exertional dyspnea and anemia	EGD HE	Several small esophageal ulcers with surrounding erythema.	Fibrinopurulent exudates with sulfur granules	A
Arora *et al.*, 2003	37/M	AIDS	ID	Chest pain	RF EGD HE	Necrotic material and extensive ulceration in association with candida.	Numerous filamentous bacteria which were not acid fast and the presence of discrete sulfur granules	A
Yagi *et al.*, 2003	19/M	Chemo-resistant nasal NK/T cell lymphoma, perforation of soft palate	ID	A	HE	A	Inflammation with actinomycotic colonies	A
Lee *et al.*, 2001	41/M	AIDS, esophageal candidiasis	ID	Odynophagia	EGD HE	esophageal ulcer with yellow exudate	Numerous sulfur granules containing abundant fine elongate hyphal forms	A
Benítez Roldán *et al.*, 2000	41/M	Colon cancer gastric hemorrhage due to Mallory-Weiss syndrome	ID	Copious vomiting	EGD HE	Ulcers with plaques	Filamentous Gram positive rods	A
Nair, Pitchumoni, 1999	37/M	AIDS, renal cell carcinoma	ID	Odynophagia and dysphagia, weight loss	EGD HE	Ulcer with exudative coating in the middle esophagus	A	A
Ng *et al.*, 1997	55/M	Disseminated pancreatic adenocarcinoma revealed after a three month	IC	Epigastric pain weight loss	EGD HE IBI	Erythematous nodule at middle esophagus	Clumped bacteria colony overlying an inflammed esophageal squamous epithelium confirmed by culture	*Actinomyces viscosus*
Vikram *et al.*, 1994	A	esophagus squamous cell carcinoma	ID	A	A	A	A	A
Poles *et al.*, 1994	42/M	CMV esophagitis, AIDS	ID	Dysphagia, odynophagia	EGD HE	A	A	A
Poles *et al.*, 1994	29/M	CMV esophagitis, AIDS	ID	Vomiting, dysphagia anorexia	EGD HE	A	A	A
Spencer *et al.*, 1993	47/M	AIDS, Oral candidiasis	ID	Odynophagia, dysphagia to both solids and liquids, abdominal pain weight loss	RF EGD HE	Ulcers with plaques scattered diffusely throughout the esophagus	Sulfur granules, Gram positive filamentous branching bacteria	A

Conflict of interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and publication of this article.

Compliance with ethical standards

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from the patient for the publication of this case report.

References

Abdalla J., Myers J., Moorman J. (2005). Actinomycotic infection of the oesophagus. *J Infect.* **51**, E39-43

Arora A.K., Nord. J, Olofinlade O., Javors B. (2003). Esophageal Actinomycosis: A Case Report and Review of the Literature. *Dysphagia.* **18**, 27-31.

Benítez Roldán A., López-Cepero Andrada J., Amaya Vidal A., Castro Aguilar-Tablada T., Ruiz Campos J.L. (2000). [Acute necrotizing esophagitis]. *Gastroenterol Hepatol.* **23**, 79-81.

Bonnefond S., Catroux M., Melenotte C., Karkowski L., Rolland L., et al. (2016). Clinical features of actinomycosis: A retrospective, multicenter study of 28 cases of miscellaneous presentations. *Medicine* (Baltimore). **95**, e3923.

Boyanova L., Kolarov R., Mateva L., Markovska R., Mitov I. (2015). Actinomycosis: a frequently forgotten disease. Future Microbiol. **10**, 613-628.

Brook I. (2008). Actinomycosis: diagnosis and management. *South Med J.* **101**, 1019-1023.

Cash J.W., McConville C., O'Rourke D., Harding T. (2005). Oesophageal ulceration: more than meets the naked eye? *Gut.* **54**, 127-133.

Chandrasekhara V., Zhang L., Floyd B.N., Kochman M.L. (2012). A rare cause of esophageal strictures: Actinomyces. *Gastrointest Endosc.* **75**, 1111-1112.

Chou F.T., Cheng K.S., Chiang I.P. (2006). Esophageal actinomycosis. *Adv Ther.* **23**, 623-626.

Fernández Moreno N., Ubiña Aznar E., Rivera Irigoín R., Fernández Pérez F., García Fernández G., et al. (2005). [Esophagus actinomycosis causing upper gastrointestinal bleeding]. *Rev Esp Enferm Dig.* **97**, 533-534.

Fukuda S., Watanabe N., Domen T., Ishioka M., Sawaguchi M., et al. (2018). A case of esophageal actinomycosis with a unique morphology presenting as a refractory ulcer. *Clin J Gastroenterol.* **11**, 38-41.

Ho L., Seto J., Jadvar H. (2006). Actinomycosis mimicking anastomotic recurrent esophageal cancer on PET-CT. *Clin Nucl Med.* **31**, 646-647.

Kim H.S., Cheon J.W., Kim M.S., Jung C.K., Kim K.R., et al. (2013). [A Case of Esophageal Actinomycosis in a Patient with Normal Immunity]. *Korean J Gastroenterol.* **61**, 93-96.

Kahn J.E., Boccara O., Girszyn N., Bernier M., Blétry O., et al. (2005). [A terrible diagnosis and hard to swallow!...]. *Rev Med Interne.* **26**, S230-3.

Könönen E., Wade W.G. (2015). Actinomyces and related organisms in human infections. *Clin Microbiol Rev.* **28**, 419-442.

Korkmaz H., Kutluana U., Bugdaci M.S., Kucukosmanoglu I. (2013). A rare presentation of esophageal actinomycosis in an immunocompetent patient. *Endoscopy.* **45**, E181-2.

Kosseifi S.G., Dittus K., Nassour D.N., Shaikh M.A., Young M.F. (2005). Actinomycosis Esophagitis in a Patient with Persistent Dysphagia. *South Med J.* **98**, 662-664.

Lee S.A., Palmer G.W., Cooney E.L. (2001). Esophageal actinomycosis in a patient with AIDS. *Yale J Biol Med.* **74**, 383-389.

Lotte R., Lotte L., Ruimy R. (2016). *Actinotignum schaalii* (formerly *Actinobaculum schaalii*): a newly recognized pathogen-review of the literature. *Clin Microbiol Infect.* **2**, 28-36.

Murchan E.M., Redelman-Sidi G., Patel M., Dimaio C., Seo S.K. (2010). Esophageal Actinomycosis in a Fifty-Three-Year-Old Man with HIV: Case Report and Review of the Literature. *AIDS Patient Care STDS.* **24**, 73-78.

Nagaraju S.P., Kirpalani D.A., Bhabhe A.S., Prasad R., Shah H, et al. (2014). Esophageal actinomycosis in a patient with end-stage renal disease. *Hemodial Int.* **18**, 544-546.

Pillappa R., O'Brien T.F., Sullivan J.L., Weksler B. (2016). Esophageal Actinomycoses Mimicking Malignancy. *Ann Thorac Surg.* **101**, 1967-1970.

Rodríguez-Lago I., Calderón Á., Cazallas J., Camino M.E., Barredo I., et al. (2017). First case report of oesophageal actinomycosis in a patient with active eosinophilic oesophagitis. *Gastroenterol Hepatol.* **40**, 404-406.

Sudhakar SS, Ross JJ. (2004). Short-term treatment of actinomycosis: two cases and a review. *Clin Infect Dis.* **38**, 444-447.

Valour F., Sénéchal A., Dupieux C., Karsenty J., Lustig S., et al. (2014). Actinomycosis: etiology, clinical features, diagnosis, treatment, and management. *Infect Drug Resist.* **7**, 183-197.

Welling R.D., Cardona D.M., Thompson W.M. (2009). Oesophageal actinomycosis a case report and review of radiographic findings. *Radiol Case.* **3**, 44-48.

Wong V.K., Turmezei T.D., Weston V.C. (2011). Actinomycosis. *Bmj.* **343**, d6099.

Yagi T., Fujino H., Hirai M., Inoue T., Sako M., et al. (2003). Esophageal actinomycosis after allogeneic peripheral blood stem cell transplantation for extranodal natural killer/T cell lymphoma, nasal type. *Bone Marrow Transplant.* **32**, 451-453.